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THE INFLUENCE OF THERMOREGULATORY MECHANISMS ON POST-EXERCISE HYPOTENSION IN HUMANS

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SUMMARY

1. To examine the influence of the rate of heat loss on the magnitude of post-exercise hypotension, subjects were exposed to three different environmental conditions during recovery from bicycle exercise.

2. When subjects recovered in warm conditions both core temperature (measured in the external auditory meatus) and mean skin temperature were significantly elevated 60 min after the cessation of exercise. This attenuation of heat loss was associated with a significant reduction in post-exercise mean arterial pressure.

3. In contrast, when subjects recovered in neutral or cool conditions both core temperature and mean arterial pressure had returned to baseline levels 60 min after exercise.

4. These results indicate that persistence of peripheral vasodilatation during recovery from exercise, and therefore the magnitude of post-exercise hypotension, is dependent upon thermoregulatory-induced changes in skin blood flow.

INTRODUCTION

The decreases in systolic and diastolic blood pressure which follow an acute bout of muscular exercise are well documented in both hypertensive (Wilcox, Bennett, Brown & MacDonald, 1982; Bennett, Wilcox & MacDonald, 1984) and normotensive (Kaufman, Hughson & Schaman, 1987; Coats, Conway, Isea, Pannarale, Sleight & Somers, 1989) individuals and have been reported to persist for up to 12·7 h (Pescatello, Fargo, Leach & Scherzer, 1991). Several mechanisms have been proposed to account for this post-exercise hypotension including the release of vasodilator metabolites from the exercising muscle (Kaufman *et al.* 1987), resetting of the baroreflex control of sympathetic tone (Bennett *et al.* 1984; Somers, Conway, Le Winter & Sleight, 1985), and inhibition of muscle sympathetic nervous activity (Floras, Sinkey, Aylward, Seals, Thoren & Mark, 1989). A further possibility relates to the need to dissipate heat accumulated during the exercise period.

Muscular work causes an increase in the body core temperature (T_C) that persists following exercise, the magnitude of which is determined by the relative intensity of the activity performed (Saltin & Hermansen, 1966). Claremont, Nagle, Redden &

Brooks (1975) have reported that this elevation in T_C can persist for more than 60 min following exercise. Since Van Beaumont & Bullard (1963) observed a marked decrease in sweat production rate at the cessation of exercise (which was independent of T_C), the heat load present at the end of exercise must be dissipated by mechanisms in addition to evaporative cooling. One possible avenue is an increase in convective heat loss via an elevation in skin blood flow (SkBF).

Increases in SkBF are associated with a marked increase in cutaneous venous volume which, unless displaced centrally by muscle movement or venoconstriction, can cause a fall in blood pressure subsequent to decreases in thoracic blood volume, central venous pressure (CVP), stroke volume (SV) and cardiac output ($\dot{Q}$) (Rowell, 1977). Zelis, Mason & Braunwald (1969) observed an increase in forearm blood flow following leg exercise. This increase was due to augmented SkBF, possibly resulting from an elevated T_C. The present study therefore used three different post-exercise environmental conditions to examine the hypothesis that exaggerating or restricting convective heat loss mechanisms would influence the magnitude of post-exercise hypotension.

METHODS

Subjects

Eleven healthy male subjects from the Department of Human Movement and Recreation Studies at the University of Western Australia volunteered to take part in the study. The subjects had a mean (±S.D.) age of 21·6±2·2 years, weight of 80·4±9·1 kg, height of 182·6±5·6 cm, and maximal aerobic power ($\dot{V}_{O_2,max}$) of 4·73±0·88 l min^{-1}. All were habitually active, possessed at least a moderate level of fitness, had normal electrocardiograms and no medical history of central or peripheral cardiovascular disease. Each subject gave written informed consent to the procedures, which had been approved by the Human Rights Committee of The University of Western Australia.

Procedure

Subjects attended a climatically controlled laboratory (20·8±0·8 °C and 57·3±5·9% relative humidity) on four separate occasions. They were requested to abstain from food and caffeinated drinks for at least 3 h prior to each session and from physical activity for the preceding 12 h. The effects of circadian variation were controlled by conducting each session at the same time of day for a given subject.

Preliminary test. The first session involved a preliminary test to examine each subject's maximal aerobic power ($\dot{V}_{O_2,max}$). Subjects were seated on an electronically braked bicycle ergometer (Orival 400, Lode, Holland) and started pedalling with the workload set at 100 W. This was increased by 25 W every 2 min until volitional exhaustion.

Inspired volume was measured using a Morgan turbine flow meter connected to a 292-B ventilation monitor (P. K. Morgan, Chatham, Kent). Expired gas was sampled by Ametek S3A/1 oxygen and CD3A carbon dioxide analysers connected in series (Ametek, Pittsburgh, PA, USA). These analysers were calibrated against a known gas mixture prior to and at the end of each test. An on-line IBM microcomputer (model No. 5160, IBM, Australia) continuously sampled the output from the two analysers and the ventilometer and calculated the volume of oxygen consumed ($\dot{V}_{O_2}$), the volume of carbon dioxide produced ($\dot{V}_{CO_2}$) and the respiratory exchange ratio (RER).

Experimental protocol. The three experimental sessions involved an identical test protocol. Prior to exercising the subjects lay supine for 30 min in a constant environmental temperature (20·8±0·8 °C and 57·3±5·9% relative humidity) during which time blood pressure (BP), heart rate (HR), core temperature (T_{EAM}) and mean skin temperature (T_{Sk}) were recorded every 5 min. Resting expired gases were collected and analysed at 15 and 25 min. Values of BP, HR, T_{Sk} and $\dot{V}_{O_2}$ were averaged to give baseline levels for these parameters.

Subjects then exercised in a constant environment (20·8±0·8 °C and 57·3±5·9% relative humidity) for 30 min on an electronically braked bicycle ergometer at a workload equivalent to 70% of their $\dot{V}_{O_2,max}$. During exercise T_{EAM} and T_{Sk} were recorded every 5 min. Oxygen consumption

was measured at 5, 15 and 25 min to determine the subject's work intensity and HR readings were taken at 10, 20 and 30 min. After 30 min the subject was allowed a 1 min cool down.

After exercise subjects entered an environmentally controlled chamber where they rested supine for 60 min in one of the three following conditions: (1) neutral temperature 21·4±0·5 °C, relative humidity 51·8±4·6%, (2) warm temperature 31·1±0·4 °C, relative humidity 53±4·6%, (3) cool temperature 17±0·8 °C, relative humidity 57·8±4·3%.

The order of presentation of these conditions was randomized and tests were conducted at least 48 h apart. During the post-exercise period BP was measured every 2 min for 12 min and then every 5 min from 15 to 60 min. HR, T_{EAM} and T_{Sk} were recorded every 5 min and $\dot{V}_{O_2}$ calculated at 5, 10, 15, 30, 45 and 60 min.

Measurements

Blood pressure. Blood pressures were measured by either microphonic sphygmomanometer ($n = 4$) (Bonn-SP, Medelic Int. Corp., Japan) or automated oscillometric blood pressure monitor ($n = 7$) (Dinamap Vital Signs Monitor 8100, Critikon, Australia). In both cases the cuff was placed over the brachial artery of the left arm. Systolic blood pressure (SBP) was recorded as phase 1 Korotkoff and diastolic blood pressure (DBP) as phase 5. Mean arterial pressure (MAP) was calculated using the formula:

$$\text{MAP} = \text{DBP} + \tfrac{1}{3} \text{ pulse pressure.}$$

One investigator was involved in the collection of all blood pressure values and the same blood pressure monitor was used on all three occasions for any given subject.

Core temperature. Core temperature was measured in the external auditory meatus (T_{EAM}) by placing a thermistor (Yellow Springs Instruments, OH, USA), embedded in a moulded plastic earpiece, approximately 1 cm inside the subject's left external meatus. This probe was connected to a Yellow Springs 46 TUC telethermometer, from which values were recorded. The thermistor was insulated from the environment by covering the entire auricle with a polyfoam earmuff. This procedure was completed as soon as the subject entered the laboratory, allowing for a 45 min stabilization period (i.e. 15 min while other probes and leads were positioned and a 30 min pre-exercise rest period). Baseline T_{EAM} was taken as the last reading prior to exercise and because a temperature gradient of up to 1 °C exists along the meatus (Cooper, Cranston & Snell, 1964), subsequent measurements are expressed as a change from this value.

Skin temperatures. Skin temperatures were recorded at four sites using Grant Squirrel 'u' thermistor probes (Grant Instruments, Cambridge, UK) held in position by air permeable woven surgical tape. The probes were attached to a Grant Squirrel data logger (Grant Instruments) for the collection of temperatures. Data from these sites were used to calculate mean skin temperature (T_{Sk}) according to the method of Ramanathan (1964).

Heart rate. HR was measured using bipolar pre-cordial ECG leads (CM5 configuration; Blackburn, Taylor, Okamatyo, Rautaharju, Mitchel & Kerkhoff, 1967) attached to a Cardiofax 6511 electrocardiograph (Nihon Kohden, Tokyo). Data from this apparatus were used to screen for any abnormalities and to calculate HR throughout each study.

Oxygen consumption. Expired gases were collected during the pre-exercise, exercise and post-exercise periods using a Douglas bag. Fractions of expired O_2 and CO_2 were analysed by a Beckman OM11 oxygen analyser (Beckman, Fullerton, CA, USA) and a Datex CD101 carbon dioxide analyser (Datex, Helsinki, Finland). Both analysers were calibrated with a known gas mixture at regular intervals during each testing session. Expired gas volume, under conditions of ambient temperature and pressure saturated ($V_{E_{ATPS}}$), was measured using a Tissot chain compensated gasometer (Warren E. Collins Inc., MA, USA). Results were then converted to standard temperature pressure dry ($V_{E_{STPD}}$) to calculate $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$ and RER.

Statistical analysis

Results are expressed as means±S.E.M. Differences between conditions during the pre-exercise and exercise periods were determined using one-way analysis of variance (ANOVA). Differences between post-exercise conditions were analysed using two-way ANOVA (repeated measures). *Post hoc* contrasts were used to identify significant differences between the cool, neutral and warm conditions. In addition, baseline and 60 min post-exercise values were compared using a Student's paired t test. Significant differences were recorded for $P < 0.05$.

TABLE 1. Pre-exercise (baseline) haemodynamic and thermoregulatory data under the three post-exercise conditions

	Recovery condition		
	Neutral	Warm	Cool
SBP (mmHg)	121·1 ± 3·2	121·9 ± 3·3	121·3 ± 3·1
DBP (mmHg)	63·7 ± 2·5	60·8 ± 2·5	61·4 ± 2·1
MAP (mmHg)	82·9 ± 2·2	81·2 ± 2·4	81·3 ± 1·6
HR (beats min^{-1})	59·5 ± 2·2	56·7 ± 2·7	55·8 ± 3·3
T_{EAM} (°C)	36·17 ± 0·11	36·47 ± 0·16	36·11 ± 0·12
T_{Sk} (°C)	31·92 ± 0·20	31·76 ± 0·24	31·47 ± 0·22
$\dot{V}_{O_2}$ (l min^{-1})	0·28 ± 0·02	0·31 ± 0·03	0·34 ± 0·02

Baseline values for all variables in the three post-exercise treatment conditions. Results are expressed as means ± S.E.M. No significant difference existed between the three conditions for any of the above variables.

TABLE 2. Exercise data under the three post-exercise conditions

	Recovery condition		
	Neutral	Warm	Cool
% $\dot{V}_{O_2,max}$	69·3 ± 2·0	70·7 ± 2·4	69·6 ± 2·3
HR (beats min^{-1})	159·7 ± 1·3	165·0 ± 1·9	160·3 ± 1·7
T_{EAM} (°C)	1·42 ± 0·20	1·59 ± 0·10	1·63 ± 0·10
T_{Sk} (°C)	0·93 ± 0·31	1·32 ± 0·28	1·19 ± 0·28

Values for all exercise variables under the three experimental conditions. Results are expressed as means ± S.E.M. T_{EAM} and T_{Sk} results are expressed as relative change from baseline levels. No significant difference existed between the three conditions for any of the above variables.

RESULTS

Pre-exercise resting variables and exercise variables

During the baseline period of 30 min there were no significant differences between the three treatment conditions for any of the measured variables (Table 1). In addition, no significant differences existed between variables measured during the exercise period (Table 2).

Post-exercise variables

Blood pressure

Relative to baseline, SBP was significantly depressed 60 min after the cessation of exercise under the warm recovery condition (115·4 ± 2·7 *versus* 121·9 ± 3·3 mmHg). Mean arterial pressure (MAP) was also significantly lower 60 min after exercise compared to baseline (76·5 ± 2·0 *versus* 81·2 ± 2·4 mmHg) under the warm recovery condition ($P < 0·05$). No such differences were evident under the neutral or cool recovery conditions. No significant differences between baseline and post-exercise measures of DBP were evident under any of the three conditions (Fig. 1). In the post-exercise period no significant differences were recorded between any of the conditions

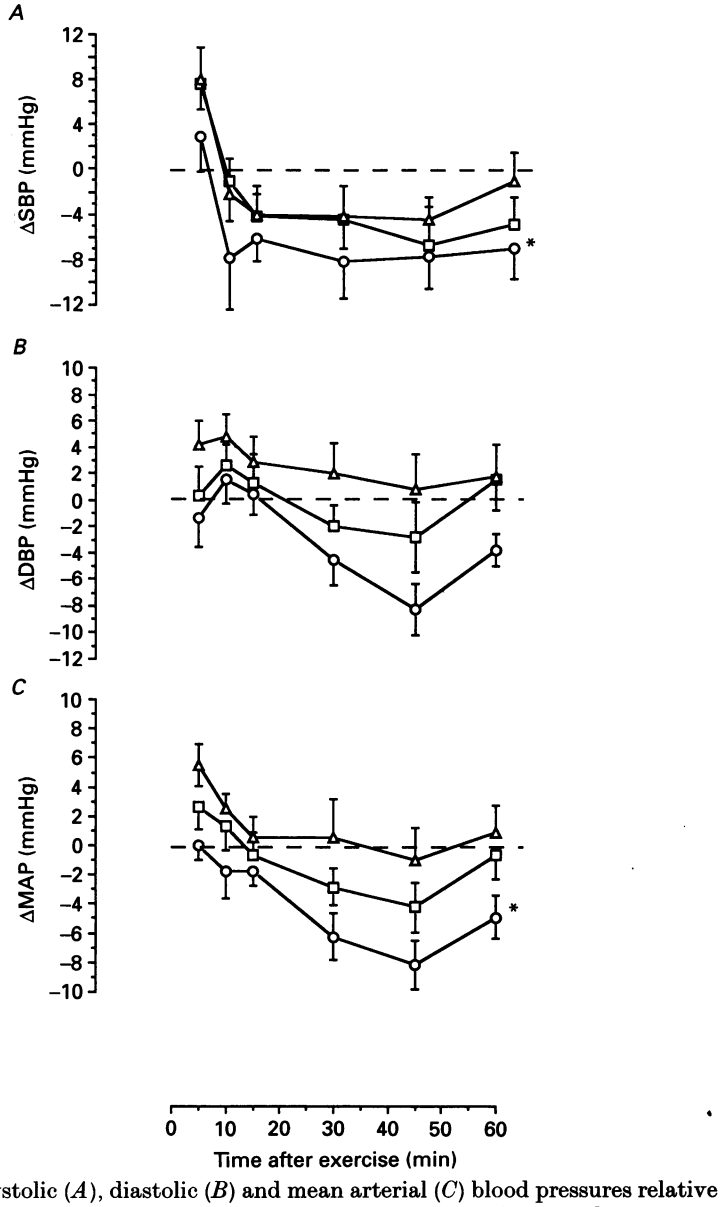

Fig. 1. Changes in systolic (*A*), diastolic (*B*) and mean arterial (*C*) blood pressures relative to pre-exercise baseline values (dashed line) under neutral (□), cool (△) and warm (○) recovery conditions. Results are expressed as means ± S.E.M. * $P < 0.05$ for Student's *t* test between baseline and 60 min post-exercise values. Changes in mean arterial pressure after exercise under the warm condition were significantly different from those under the cool condition ($P = 0.0296$), whilst differences between post-exercise diastolic blood pressures were evident between both warm and neutral ($P = 0.0268$) and warm and cool ($P = 0.0158$) conditions (two-way ANOVA).

for SBP. However, MAP was significantly lower in the warm condition compared to the cool condition during this time ($P = 0{\cdot}0296$) and DBP was significantly lower under the warm condition than both the neutral ($P = 0{\cdot}0268$) and cool ($P = 0{\cdot}0158$) conditions (Fig. 1).

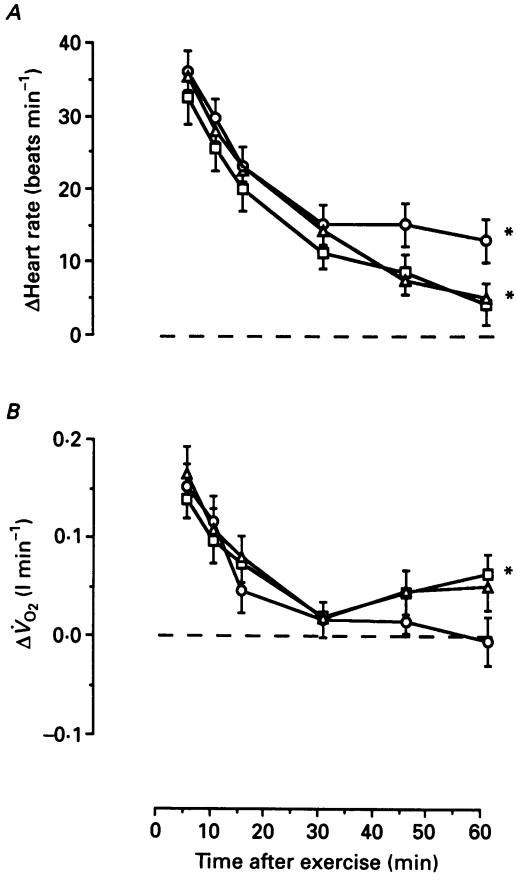

Fig. 2. Changes in heart rate (*A*) and $\dot{V}_{O_2,max}$ (*B*) relative to pre-exercise baseline values (dashed line) under the neutral (□), cool (△) and warm (○) recovery conditions. Results are expressed as means ± S.E.M. * $P < 0{\cdot}05$ for Student's *t* test between baseline and 60 min post-exercise values.

Heart rate

Compared to baseline levels, 60 min post-exercise HR values were significantly elevated under both the warm ($76{\cdot}0 \pm 4{\cdot}2$ *versus* $56{\cdot}7 \pm 2{\cdot}7$ beats min^{-1}) and cool ($60{\cdot}6 \pm 2{\cdot}0$ *versus* $55{\cdot}8 \pm 3{\cdot}3$ beats min^{-1}) conditions ($P < 0{\cdot}05$), whilst no significant difference was present under the neutral condition (Fig. 2*A*). When the three post-exercise conditions were compared, no significant differences in HR were evident. However, a linear time/treatment interaction effect was present between the warm and cool conditions indicating that the decrease in HR under warm conditions was slower than that in the cool environment (Fig. 2*A*).

Oxygen consumption

Post-exercise $\dot{V}_{O_2}$ measurements, recorded 60 min after the cessation of exercise, were not significantly different to baseline values under either the cool or warm condition. However, post-exercise $\dot{V}_{O_2}$ was significantly higher than the baseline

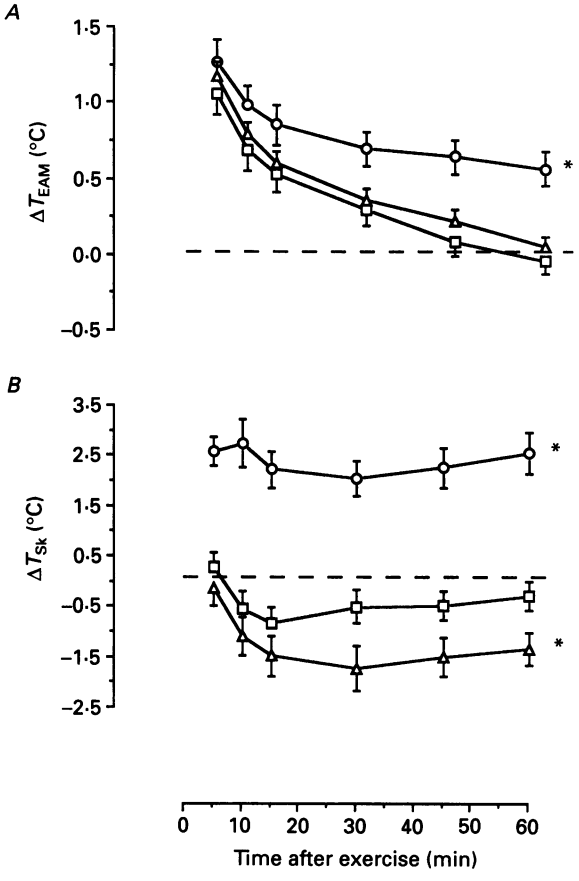

Fig. 3. Changes in T_{EAM} (*A*) and T_{Sk} (*B*) relative to pre-exercise baseline values (dashed line) under the neutral (□), cool (△) and warm (○) recovery conditions. Results are expressed as means ± S.E.M. $*P < 0{\cdot}05$ for Student's *t* test between baseline and 60 min post-exercise values. Changes in T_{EAM} after exercise under the warm condition were significantly different from those under both the neutral ($P = 0{\cdot}0136$) and cool ($P = 0{\cdot}0007$) conditions. Changes in T_{Sk} after exercise under the warm condition were significantly different from those under both neutral ($P = 0{\cdot}0001$) and cool ($P = 0{\cdot}0001$) conditions (two-way ANOVA).

value recorded under the neutral condition ($0{\cdot}34 \pm 0{\cdot}02$ *versus* $0{\cdot}28 \pm 0{\cdot}02$ l min^{-1}) ($P < 0{\cdot}05$). No significant differences existed between $\dot{V}_{O_2}$ values recorded under each of the three conditions during the recovery period (Fig. 2*B*).

Core temperature (T_{EAM})

Under the warm condition, T_{EAM} values were significantly higher 60 min post-

exercise compared to resting measures (36·72 ± 0·09 *versus* 36·47 ± 0·16 °C) ($P > 0·05$). No significant differences existed between baseline and post-exercise T_{EAM} values under the neutral or cool conditions. Post-exercise T_{EAM} values were significantly higher under the warm condition than either the neutral ($P = 0·0136$) or cool ($P = 0·0007$) conditions. A linear time–treatment interaction indicated that the decline in T_{EAM} under the warm condition was significantly less than that under the cool and neutral conditions (Fig. 3*A*).

Mean skin temperature (T_{Sk})

Relative to baseline levels, T_{Sk} was significantly elevated 60 min post-exercise under the warm condition (34·09 ± 0·30 *versus* 31·76 ± 0·24 °C) and significantly reduced at the same point under the cool condition (30·10 ± 0·19 *versus* 31·47 ± 0·22 °C) ($P < 0·05$). There was no significant difference between baseline and post-exercise T_{Sk} values under the neutral condition. Under the warm condition, post-exercise T_{Sk} values were significantly higher than those recorded under the neutral ($P = 0·0001$) and cool ($P = 0·0001$) conditions (Fig. 3*B*).

DISCUSSION

The hypotensive effects of an acute bout of exercise have been described previously (Hannum & Kasch, 1981; Wilcox *et al.* 1982; Bennett *et al.* 1984; Somers *et al.* 1985; Floras *et al.* 1989). These studies indicate that light to moderate intensity aerobic exercise of 20–30 min duration is sufficient to produce post-exercise hypotension, which has been reported to last for up to 12·7 h (Pescatello *et al.* 1991). This response seems to be caused by a decrease in total peripheral resistance (TPR) which, in turn, is due to persistence of the peripheral vasodilatation produced by exercise (Coats *et al.* 1989). Since sweat production rate decreases rapidly at the cessation of exercise (Cable & Green, 1990), even in warm ambient conditions (Van Beaumont & Bullard, 1966), it is likely that the principal route of heat loss in the period following exercise is convection from the skin. The present study was therefore designed to investigate the possibility that increased peripheral blood flow after exercise is in part due to thermoregulatory demands of exercise-induced elevation in T_C.

This study confirms the presence of post-exercise hypotension when recovery conditions are warm and heat loss is attenuated. However, when subjects were rested in a cool environment the difference between baseline and post-exercise blood pressures was not significant. Under neutral recovery conditions MAP was significantly lower than its pre-exercise level between 30 and 45 min post-exercise, but had returned toward its baseline level by 60 min. These results indicate that manipulation of the temperature gradient between the core and skin after an acute bout of exercise can influence post-exercise blood pressures.

This finding may be explained by the interplay between the effects of T_C and skin temperature on skin blood flow. The vasoactive status of peripheral blood vessels is dependent upon both skin temperature, which influences vasoconstrictor outflow to the skin and T_C, which activates the vasodilator system (Rowell, 1983). Blood flow to the skin is highest when both core and skin temperatures are elevated (Roddie &

Shepherd, 1956). Under warm conditions in the present study, T_{Sk} and T_C (measured as T_{EAM}) were significantly higher than baseline levels 60 min post-exercise and were significantly greater than values recorded under neutral and cool conditions during the last 30 min of recovery (Fig. 3*A* and *B*). These elevations in T_C and T_{Sk} were associated with a significant reduction in MAP during recovery (Fig. 1*C*). Under the cool and neutral conditions both T_C and T_{Sk} had returned toward baseline levels by 60 min post-exercise (Fig. 3*A* and *B*) and MAP was not significantly different from its baseline level (Fig. 1*C*). Thus, elevation of T_{Sk} under warm recovery conditions may sustain peripheral vasodilatation, causing a reduction in TPR and therefore a decrease in MAP following exercise. Conversely, recovery in a neutral or cool environment may abolish peripheral vasodilatation and re-establish pre-exercise blood pressures.

We did not measure cardiac output, skin or muscle blood flow in this study. It is therefore not possible to establish differences in the distribution of blood flow induced by each of the post-exercise conditions. However, assuming that pre- and post-exercise supine stroke volumes are similar (Cleroux, Kouame, Nadeou, Coulombe & Lacourciere, 1992) it can be calculated that a 24% decrease in TPR occurred after 60 min of recovery in the warm condition, with a 7% decrease in the neutral condition and an 8% decrease in the cool condition at the same point. These decreases in TPR are unlikely to be the result of vasodilatation in visceral vascular beds since elevated heart rates, such as those evident in this study are paralleled with vasoconstrictor outflow to the viscera (Rowell, 1974). Conversely, a decrease in TPR consequent to thermoregulatory cutaneous vasodilatation provides a plausible explanation for the influence of post-exercise environmental conditions on MAP in this study since elevated skin temperatures reflect increases in peripheral blood flow (Roddie, 1983). Indeed, Zelis *et al.* (1969) reported an increase in forearm skin blood flow following leg exercise whilst blood flow to the muscles of the same region did not change. Further research involving measurement of $\dot{Q}$, skin and muscle blood flow will be necessary to confirm this speculation.

A limitation of the present study was that a control group was not used to establish the BP responses caused by each of the environmental conditions independent of prior exercise. This raises the question of how much of the reported changes occurred due to exercise and how much can be apportioned to the environmental conditions to which subjects were exposed. Rowell, Brenglemann & Murray (1969) recorded the haemodynamic changes in resting supine subjects in response to maximal whole-body heating. They reported that MAP decreased transiently but recovered after approximately 20–30 min. However, in some cases MAP was reduced for periods up to 60 min (Rowell *et al.* 1969). The present study did not use environmental temperatures as severe as those used in the above study (31 *versus* 40·5 °C respectively) but MAP was still significantly reduced 60 min after exercise in the warm condition to levels even greater than those reported by Rowell *et al.* (1969). This would suggest that the post-exercise hypotension recorded in this condition must be dependent, in part, on the preceding exercise.

In conclusion the present study found that in a warm recovery environment both T_{EAM} and T_{Sk} are significantly elevated relative to baseline levels 60 min after the cessation of exercise and this is associated with a significant reduction in MAP.

Conversely, when recovery conditions are cooler there is a faster decrease in T_{EAM} which, when associated with a significant reduction in T_{Sk}, abolishes the hypotensive effects of exercise. It seems, therefore, that thermoregulatory mechanisms do play a role in the persistence of peripheral vasodilatation leading to hypotension during the recovery from exercise.

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