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*Full Length Research Paper*

# Transient post-exercise hyper-perspiration of forehead area

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**Information about sweating responses during exercise recovery is insufficient and mostly controversial. In the present study, particular attention is given to sweating behavior at forehead area immediately after exercise. Simultaneous changes of some underlying factors are also addressed. Eight healthy young males (27.4±0.8 years old) volunteered for the study. They performed a constant work rate exercise (60 rpm at 50% of individual work load for VO2 peak) for 30 min. Sweating from the forehead area was measured by the weight gain of a covered filter paper disk. Simultaneous variations in tympanic temperature, heart rate and mean arterial pressure were obtained. Plasma concentrations for sodium, vasopressin, adrenalin and noradrenalin were also determined. Sweating rate increased up to the first sample taken immediately after exercise cessation ($P < 0.05$). All other variables measured increased during exercise and with exception of noradrenalin, which was maintained high. Other variables, on the whole, decreased during recovery period. At exercise, off-transient coincident with overshoot in sweating rate, tympanic temperature, noradrenalin and vasopressin were subject to a non significant elevation. These results suggest that, sweating from forehead area is still subject to increase at exercise off-transient. The effects of relevant factors studied here are contradictory and could not justify the increase seen in sweating at off-transient. More studies are required to elucidate the causative factor(s).**

**Key words:** Exercise off-transient, recovery, sweating rate, forehead.

## INTRODUCTION

Heat loss from the evaporation of sweat during exercise is an important way of controlling the internal temperature in humans. Whole body sweating rate (SR) increases linearly with an increase in the intensity of exercise (Nielsen et al., 1996). It has been reported that, exercise intensity does not affect the sweating threshold on the forearm (Taylor et al., 1988) or the sensitivity of whole body sweating (Saltin et al., 1971). In contrast, Kondo et al. (1998) reported that, the mean sweating rate increased with exercise intensity. However, despite controversies in some details, there is consensus among researchers about the incremental pattern of sweating during exercise.

A number of investigations are after SR pattern following exercise. Despite relatively well-characterized sweating responses during exercise, information about SR during subsequent recovery seems to be insufficient. In 1970, Saltin et al. reported that, after heavy exercise (75% of VO2max) at 30°C, incremental pattern of sweating may have continued at least 5 min into the recovery period. Later coming studies advocated decrease in SR by exercise termination (Wilson et al., 2004; Shibasaki et al., 2004). It has been claimed that, reductions in SR occurs at all three modes of recoveries, that is, inactive, loadless exercise (active) and passive

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**Abbreviations: SR,** Sweating rate; $P_{osm}$, plasma osmolality; $P_{AVP}$, plasma argenine vasopressin; $P_{ad}$, adrenalin; $P_{nad}$, noradrenalin; $T_{tym}$, tympanic temperature; **MAP,** mean arterial blood pressure; **HR,** heart rate; ELISA, enzyme-linked immunosorbent assay; **PEHP,** post exercise hyper-perspiration; **EDTA,** ethylenediaminetetraacetic acid.

exercise. It is suggested that, these are consistently occurring at supine (minimizing the role of baroreceptors) (Wilson et al., 2004 Shibasaki et al., 2004) or upright positions (Carter et al., 2002). Although, they did not focus on the transients of SR at exercise cut-off point, it may be inferred from the data reported by Carter et al. (2002) that, when recovery is in active form there is no significant increase in SR which immediately followed the exercise.

Many researchers consider internal and skin temperatures as the primary drive for sweating during exercise (Nielsen, 1969; Benzinger, 1959; Kondo et al., 1997). Nevertheless, it has been reported that, the rate of sweating is not totally dependent on factors such as baroreceptor activity, mechano and metaboreceptors, central command and humoral factors such as arginine vasopressin and catecholamines may be involved in variations seen in sweating during and after exercise (Wilson 2004; Carter et al., 2002; Shibasaki et al., 2003).

Dominance of forehead area over other regions of human body in many aspects of sweating is another issue worth noting here. Kondo et al. (1998) investigated regional differences in sweating during exercise. They described that, activated sweat glands and the sensitivity of sweating response on the forehead were higher than at the other sites. Forehead area enjoys some dominant characteristics in capacity for sweating. It has been shown that, in this region, density of sweat glands, glands capacity for sweating and maximal sweating rate are significantly higher than other regions (Kondo, 1998). Unfortunately, the few studies on sweating pattern during recovery period assess SR in other regions like forearm and chest (Wilson et al., 2004; Shibasaki et al., 2004; Carter et al., 2002). The idea behind the present investigation originated from a personal experience by one of the authors, which suggested a sensible increase in SR from forehead area at exercise cut-off. The present study was scheduled to try this observation.

## MATERIALS AND METHODS

### Subjects

Eight healthy males (27.4±0.8 years old, 71.4±3 kg weight and 174.1±1.7 cm height) participated in this study. All volunteers were familiarized with the experimental procedure and written informed consents were obtained.

### Test protocol

Subjects performed two exercise runs on separate days. All tests were conducted ~3 p.m. Pretest instructions included eating a light lunch, refraining from drinking any beverage from ~ 1:00 pm and no exercise on the day of experiment.

On the first meeting, each subject had his peak oxygen uptake ($VO_2$ peak) measured. $VO_2$ peak was determined by an incremental work rate protocol on a bicycle ergometer (ergo-metrics 800S Sensormedics, USA) interfaced with a personal computer (V max 29C Sensormedics, USA), as previously described by Whipp and Wasserman (1972). It was preprogrammed to begin at a load of 25 w for an initial 2 min warm up stage, increase to 50 w for the second stage and increases by 20 w each min until the subject achieved volitional fatigue. Subjects were requested to maintain a pedal cadence of 60 rpm for the duration of the test.

On the second meeting, the effect of exercise and subsequent recovery on sweating profile plus some relevant humeral changes such as plasma osmolality ($P_{osm}$), plasma argenine vasopressin ($P_{AVP}$), adrenalin ($P_{ad}$) and noradrenalin ($P_{nad}$) and nonhumoral changes such as tympanic temperature ($T_{tym}$), mean arterial blood pressure (MAP) and heart rate (HR) were studied. Particular attention was given to the changes occurring at the off-transient of exercise.

Initially, an indwelling cannula was inserted into a large superficial vein in the forearm. Then subjects rested in the sitting position for 30 min in the laboratory (28.4±0.4 ambient temperature, 45.5±0.03 relative humidity and natural convection). Later, 9 ml of blood sample was collected through the indwelling cannula at the end of the resting period; they performed a constant work rate exercise test (60 rpm at 50% of his determined $VO_2$ peak) for 30 min. Second and third blood samples were drawn at 15 and 30 min after the onset of exercise. During the recovery period, following exercise termination, blood samples were taken at 3, 15 and 30 min. These were immediately centrifuged at 3000 g and 4°C for 5 min and then $P_{osm}$, $P_{AVP}$, $P_{ad}$, and $P_{nad}$ were measured.

Sweating rate was measured during 3 min periods ending in 15 and 30 min after the onset of exercise and every consecutive 3 min, daringly 30 min into the recovery. Simultaneously, $T_{tym}$ heart rate and arterial blood pressure were also measured and recorded.

### Measurements

#### *Sweating rate*

Sweating rate from forehead area was measured by the weight gain of a filter paper disk (48 $cm^2$) placed on the skin. The outer exposed surface of the disks was covered by a waterproof tape to prevent evaporation. Filter paper disks were weighed using EK-500G beam balance, accurate to ±0.01 g.

#### *Tympanic temperature*

Change in $T_{tym}$ was measured using the Braun Pro 3000. Blood pressure was determined using a mercurial sphygmomanometer and adult cuff.

#### *Plasma sodium concentrations*

Plasma sodium concentrations were determined by eppendorf flame photometry (Efox 5054, instrumentation laboratory). Since sodium and its associated anions account for about 94% of the solute in the extracellular compartment, plasma osmolality could be roughly approximated as (Guyton and Hall, 1996):

$$P_{osm} = 2.1 \times P_{Na}$$

#### *Plasma adrenalin and noradrenalin*

$P_{ad}$ and $P_{nad}$ were determined by enzyme-linked immunosorbent assay (ELISA) from samples collected in ethylenediaminetetraacetic acid (EDTA) that were immediately centrifuged for 15 min at 4°C. Then, plasma samples collected were stored at -70°C until used.

To conduct the test, plasma samples were brought out of freezer

and were placed at room temperature until changed into liquid. All kit reagents also were taken out of refrigerator half hour before measurement to reach room temperature (~ 25°C) and were mixed by gentle inversion before use.

Three hundred microliters of standard, controls or plasma extract were pipetted into the bottom of the appropriate tubes and then $P_{ad}$ and $P_{nad}$ were measured by ELISA using kit (adrenalin, and noradrenalin IBL Hamburg ELIZA kits).

#### *Plasma arginine vasopressin ($P_{AVP}$)*

$P_{AVP}$ was determined by radioimmunoassay. Processing and storage of samples were the same as stated earlier. Plasma samples were extracted using Sep-Pack C18 cartridge (Waters associates, Milford, MA, USA) as previously described (Paolo and Seccia, 2000). Three hundred microliter standards, controls or plasma extract were pipetted to the bottom of the appropriate tubes and then $P_{AVP}$ was measured by radioimmunoassay using kit (vasopressin RIA, IBL Hamburg RIA kit).

#### Statistical analysis

The average response of the different physiological variables was compared using analysis of variance (ANOVA) with repeated measures. In the event of statistical significance ($P < 0.05$), a Tukey's test was used to identify significant differences. Results obtained during exercise and recovery period were assessed for possible correlation between the changes in the humeral and non-humeral factors and forehead perspiration by the Pearson correlation test ($P < 0.05$). All values are presented as means ± SE.

## RESULTS

Eight healthy males (27.4±0.8 years old, 71.4±3 kg weight and 174.1±1.7 cm height) participated in this study. The subjects' $VO_2$ peak and maximum heart rate were 47.39±4.01 ml/min/kg and 182±3 beats/min, respectively.

### Sweating profile

There was an increase in SR during exercise. Significant differences were observed between control and mean values during and after exercise ($P < 0.05$). It was finally fully recovered in 7$^{th}$ recovery sample ending in 21$^{st}$ min after exercise. Comparison of final SR during exercise and its first measurement into recovery demonstrated a significant increase ($P < 0.05$) (Figure 1a). Results achieved for other variables, categorized as non-humeral and humeral factors are given further as

### *Non-humeral factors*

**Tympanic temperature:** As can be seen in Figure 1, tympanic temperature increased significantly during exercise, (37.36±0.05°C, p = 0.03) and continued to be so up to the first post exercise sample (37.38±0.06°C, p = 0.02). Then, it recovered to the baseline values (Figure 1b).

**Heart rate:** With the onset of exercise, heart rate increased significantly and then remained so to the end of exercise ($P < 0.05$). Significant decrease occurred on transition from exercise to recovery ($P < 0.05$). Then, it underwent a gradual decline being still significantly higher than control until 12 min post-exercise ($P < 0.05$) (Figure 1c).

**Mean arterial pressure (MAP):** There was a no significant increase in MAP during exercise. On transition to post exercise period, it displayed a classic post exercise hypotension and then was kept so during 30 min into the recovery (Figure 1d). There were significant positive correlations between SR and tympanic temperature ($r = 0.401$), heart rate ($r = 0.598$) and MAP ($r = 0.280$) during recovery period ($p < 0.05$).

### *Humeral factors*

**Osmolality:** There was significant increase in $P_{osm}$ during exercise (311.6±2.5 mosm/kg H2O, p= 0.00), followed by a significant decrease after exercise (290.3±1.9 mosm/kg H2O, p= 0.01) and then recovered to baseline values (Figure 2a).

**Arginine vasopressin:** Significant increases in $P_{AVP}$ were observed during exercise. It increased to still higher level in the first sample taken 3 min after exercise (3.73±0.20 pg/ml, p= 0.00) and then fell to lower levels recovering to values not significantly different from the baseline (Figure 2b).

**Catecholamine:** With the onset of exercise, $P_{ad}$ and $P_{nad}$ increased dramatically and then were kept significantly higher than baseline to the final sample, taken 30 min after exercise. No significant change occurred on transition from exercise to rest (Figure 2c, d). There were significant positive correlations between SR and $P_{AVP}$ ($r= 0.820$), $P_{ad}$ ($r= 0.370$) and negative correlation between SR and $P_{nad}$ ($r= -0.451$) during recovery period ($p < 0.05$).

**Off-transient:** Due to the fact that SR and relevant changes at exercise off-transient constituted the main goal of the present study, results obtained for this critical period are collectively approached here. Figure 3 provides a pictorial demonstration of the results for this step. As can be seen, at this point, there is a non significant increase in SR (24.15%) ($P < 0.05$). This

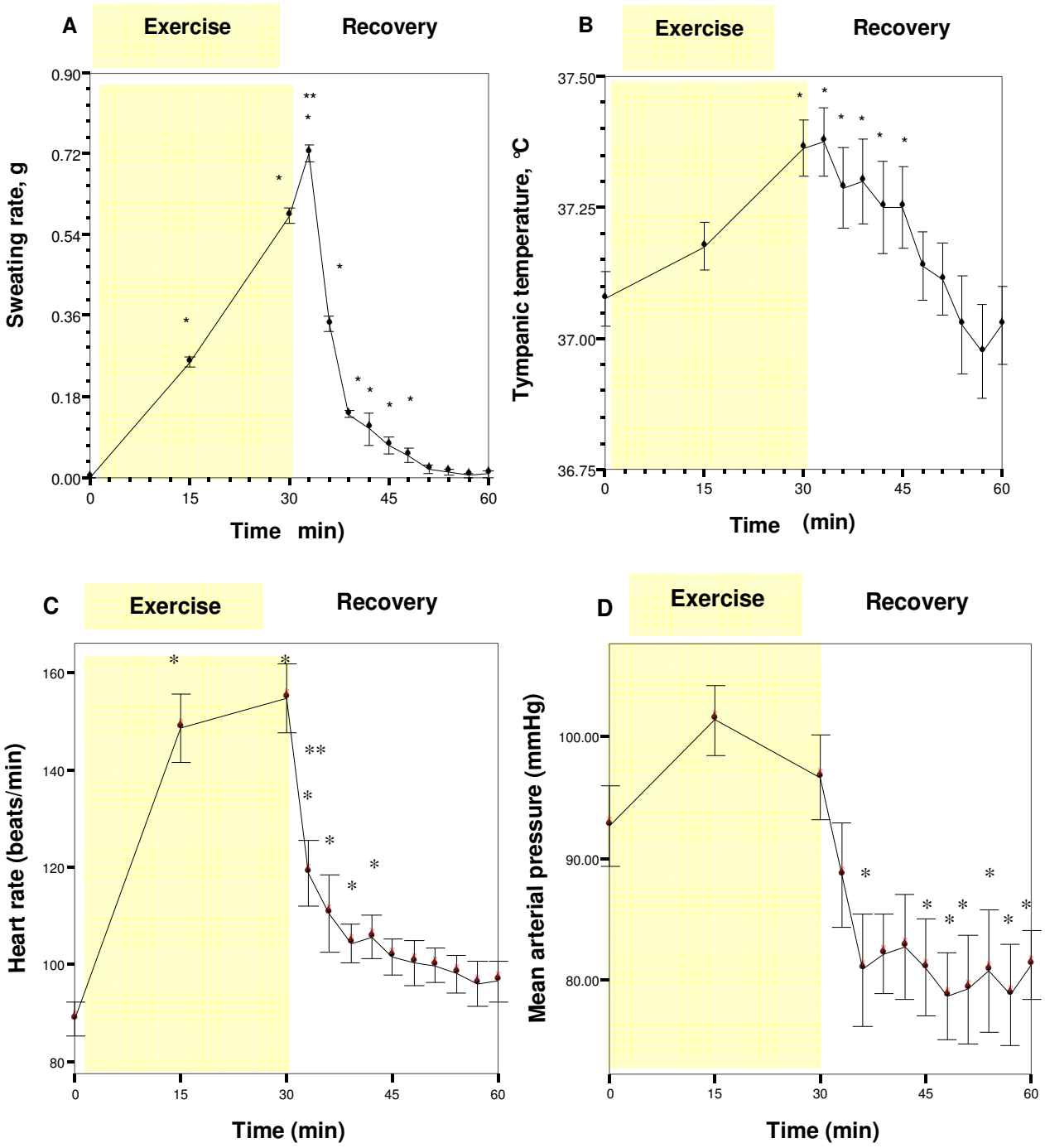


**Figure 1.** Effect of exercise (50% VO2peak) and its cessation on sweating rate, tympanic temperature, heart rate and mean arterial pressure. Subjects started to exercise at 0 min, the values at which are considered as controls. All values are mean ± S.E. *Significant difference with control; **significant changes at exercise off-transient.

coincided with a similar but smaller change in $T_{tym,}$ not gaining significance. In contrast, HR, MAP and $P_{osm}$ diminished significantly at this point. A non-significant elevation in $P_{ad,}$ $P_{nad}$ and $P_{AVP}$ occurred at off-transient of exercise. The relevant values were 0.51, 4.3 and 7.66 %, respectively ($P < 0.05$).

## DISCUSSION

The main finding of the present study, not reported so far, is the significant enhancement of SR in forehead area immediately after a moderate exercise with inactive recovery. As described in the introduction section, none

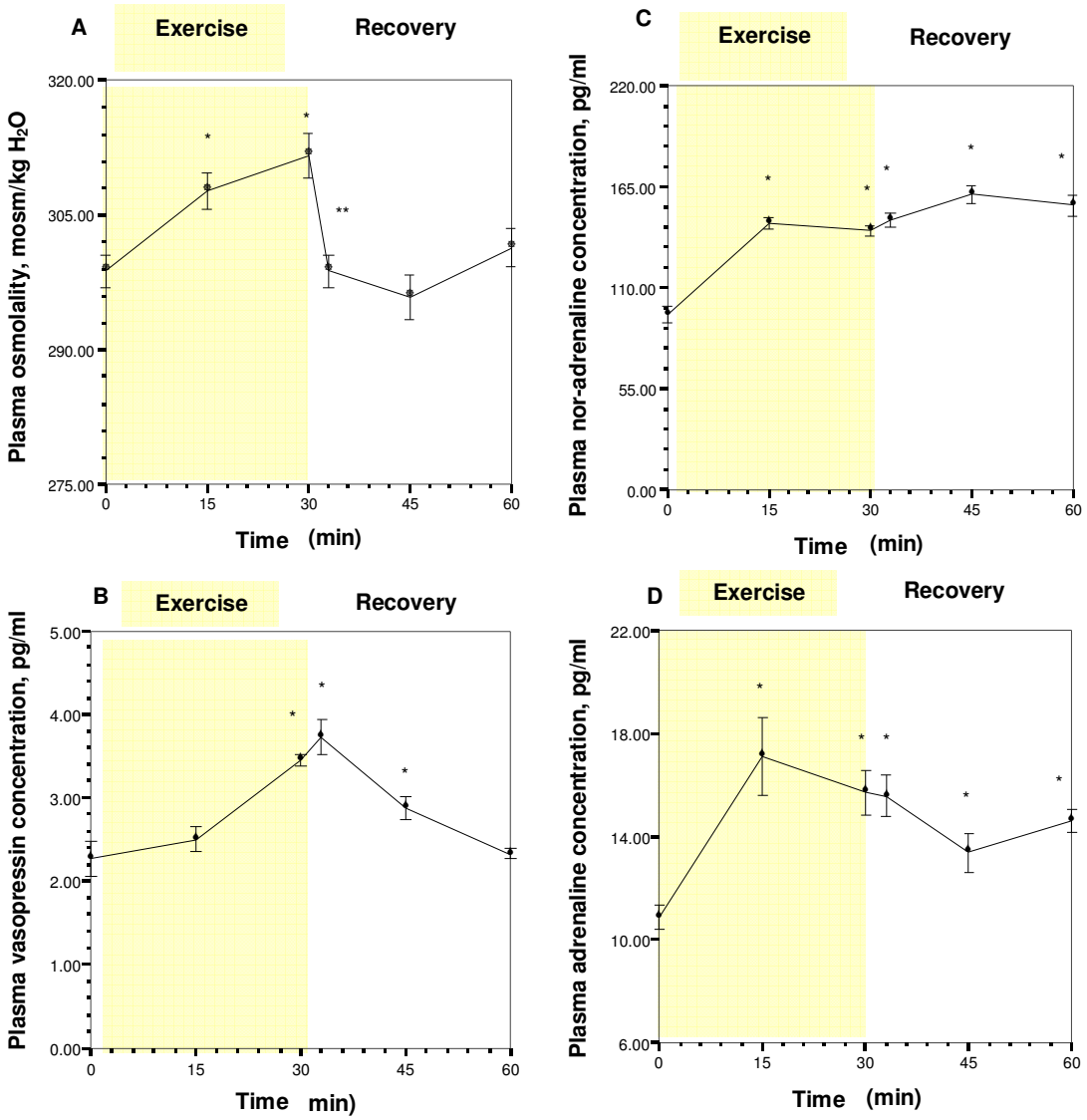


**Figure 2.** Change in plasma osmolality, plasma levels of arginine vasopressin, adrenalin and noradrenalin during exercise (50% VO2peak) and after its cessation. Other considerations are as described in the legend for Figure 1.

of the previous studies focused specifically on the off-transient changes in SR after exercise. They also did not monitor sweating from forehead area. The differences of behavior in different locations of the body have been explained before (Kondo et al., 1998). It can be seen in Figures 1a and 3 that, sweating undergoes a sharp increase by exercise cessation ($P < 0.05$). As a new term to be introduced, we tend to call this phenomenon "post exercise hyper-perspiration" (PEHP) of forehead region. When explained as percentage of changes relative to sweating rates prior to exercise cessation, on average, PEHP attained 24.15% increases (Figure 3). Figure 3 also depicts coincidental changes in some humoral and non-humoral factors, which may contribute to SR at this stage.

It is difficult indeed, to justify this phenomenon in connection with these factors. Core temperature, represented by $T_{tym}$, which is assumed to be one of the most important positive factors involved in perspiration (Nadel et al., 1971; Gisolfi and Robinson, 1970; Nadel et al., 1971), is accompanied with a trivial and non-significant increase. $P_{ad}$ and $P_{nad}$ are both increased non-significantly at this period. Kjaer (1992) however, reported significant increments in these factors post-exercise. These catecholamines have been shown to enhance sweating (Guyton and Hall, 1996). Their increase may be as result of activation of sympathetic system via baroreflex. This might have originated from the abrupt undershoot in MAP which could be caused by diminished cardiac output due to rapid fall in HR (Figure

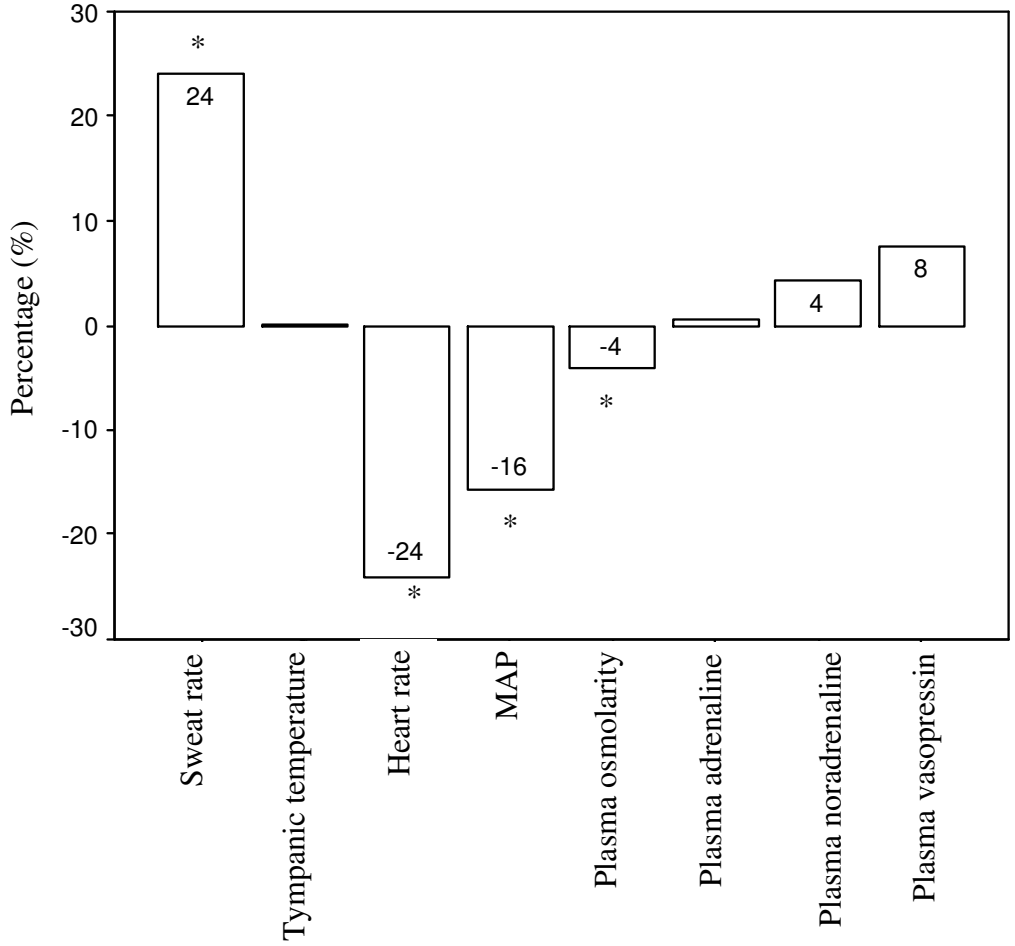


**Figure 3.** Variations seen for all variables studied at off-transient of exercise explained as percentage of changes.*Inter protocol significant differences.

3) as well as decrease in cardiac contractility (Kjaer, 1992; Hagberg et al., 1987). Nevertheless, Brown et al. (1993) found no significant changes in cardiac output stroke volume or total peripheral resistance during exercise recovery.

Arginine vasopressin was shown to inhibit sweat glands (Maeda and Miyauchi, 1996). Therefore, increase in $P_{AVP}$, shown in the Figure 3 seems to negate the increase seen in SR. The paradoxical behavior of these factors presents a complicated state, which demands more studies in order to be clarified.

The information provided by Kaufman et al. (1987) and some other researchers (Harris and Holly, 1987; Hill et al., 1989) might be used here to justify the increase in SR shown in our study. They demonstrated post exercise cutaneous vasodilation after both dynamic (Kaufman et al., 1987) and static (Harris and Holly, 1987; Hill et al., 1989) forms of exercise. This is attributed to increase in skin temperature (Kaufman et al., 1987). Marked skin blood flow could result in enhanced sweat secretion thus, explaining PEHP found in the present study. Skin blood flow and temperature on the forehead area was shown to be significantly higher than chest and forearm at all exercise intensity (Kondo et al., 1996). To our assumption, the enhanced skin blood flow at this point could be partly due to rapid redistribution of blood from exercising muscles. The truth of this assumption needs to be investigated. On the contrary, (Hori et al; 1978), and Johnson et al; 1981) demonstrated either a reduced or unchanged mean skin temperature in recovery from exercise, which would indicate a propensity toward cutaneous vasoconstriction rather than dilation. They have tried to justify this by the increase in circulating catecholamines (Kjaer, 1992), the trend of which is demonstrated in our results as well. The enhanced sweating from forehead area might be effective in brain cooling. The possibility of which is brought up by Cabanac et al. (1987).

## Conclusions

In conclusion, our results demonstrated a transient but

dramatic enhancement of sweating from forehead area just after 30 min of moderate intensity exercise. Indeed, the present investigation led to the discovery of a new physiological response, not reported before. Further studies are required to provide more qualitative and quantitative understanding of this new observation and to elucidate the possible causative factor(s).

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