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RELATIONSHIP BETWEEN BLOOD LACTATE AND HYPERVENTILATION DURING HIGH-INTENSITY CONSTANT-LOAD EXERCISE IN HEAT

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ABSTRACT: The purpose of this study was to examine the relationship between hyperventilation and increase in blood lactate during high-intensity constant-load exercise in heat and normal conditions. Seven male volunteers exercised for 10 min on a cycle ergometer at 80%$\dot{V}O_2$max in heat (40°C, 50% relative humidity: HT) and normal conditions (20°C, 50% relative humidity: CON). Oxygen uptake, carbon dioxide output, ventilation, blood lactate and blood electrolytes (K^+, Na^+, Cl^-) were measured in HT and CON. We found that ventilation was significantly higher during exercise in HT compared with CON ($p < 0.05$) and RER tends to be higher in HT than in CON. Blood lactate was significantly higher at 3 min during exercise in HT compared with CON (5.96 ± 0.57 mEq·l^{-1} 5.00 ± 0.28 mEq·l^{-1}, $p < 0.05$). Change in strong ion difference [$\Delta SID = (\Delta K^+ + \Delta Na^+) - (\Delta Cl^- + \Delta La^-)$], which affects ΔHCO_3^- in blood significantly, was lower at 5 min during exercise in HT compared with in CON ($p < 0.05$). These results suggest that hyperventilation during exercise in heat would induce lower HCO_3^- in blood and consequently would result in an increase in blood lactate at an earlier time during high-intensity exercise in heat. It was concluded that hyperventilation during short-term high-intensity exercise in heat is temporarily associated with an increase in blood lactate.

KEY WORDS: blood lactate, hyperventilation, blood electrolyte, strong ion difference, heat

INTRODUCTION

It has often been observed that blood lactate in exercise increases in a hot environment more than it does in a cool environment. The interpretation of this increase was that the hypoxia in the active muscle was induced by attenuation of the muscle blood flow during exercise due to an increase in the skin blood flow in heat stress, resulting in augmented anaerobic metabolism and increased lactate production. However, direct measurements of blood flow in the active limb in humans using a thermodilution technique has indicated that the blood flow during exercise does not change in heat stress [16,18].

It has also been observed that intramuscular glycogen utilization during prolonged exercise increases in the heat [3,4,5,9]. The increase in glycogen utilization is derived from both the aerobic and the anaerobic energy supply system activated by heat stress. Thus, it is supposed that increased muscle lactate accumulation observed in humans [6, 20] and dogs [14] during exercise in the heat comes from accelerated anaerobic glycolysis, especially in prolonged exercise. However, in constant-load exercise for 5 min, there is no difference in glycogen utilization and lactate accumulation in muscle between environment conditions of 40°C and 20°C [17]. Therefore, the increase in blood lactate during short-term intense exercise in the heat cannot be attributable to metabolic change of active muscle.

During exercise in the heat, skin blood flow and sweat rate increase to regulate body temperature by removing heat from the body [15]. Pulmonary ventilation is also one of the thermolytic responses to control core temperature in panting mammals [8]. Humans are not panters. However, hyperventilation occurs in humans when core temperatures are passively increased by 1.0°C above normothermic body temperatures [7]. Recently Hayashi et al. have reported that minute ventilation ($\dot{V}E$) increases with hyperthermia during prolonged submaximal exercise with body warming [10].

Hyperventilation in heat stress can induce excessive output of carbon dioxide (CO_2excess) and a reduction in end tidal CO_2 pressure (P_{ETCO_2}) in exercise. CO_2excess output by artificial hyperventilation is derived from blood at first and then muscle since the enzyme reaction between CO_2 and HCO_3 is slow in muscle [12]. If respiratory alkalosis in the blood occurs by hyperventilation in the heat, HCO_3^- decreases. The lactate ion, as an anion, could diffuse from muscle cell to the blood to compensate the decrease of HCO_3^- by

hyperventilation during exercise in the heat since the whole electrolyte in blood should be neutral electrically. Therefore, we hypothesized that blood lactate during short-term intense exercise in the heat increases to counteract the changes of electrolyte balance in the blood by hyperventilation.

To verify this hypothesis, we examined the relationship between hyperventilation and the increase in blood lactate, and the changes in blood electrolytes including blood lactate during 10 min high-intensity constant-load exercise in the heat compared with that in a cooler environment.

MATERIALS AND METHODS

Subjects. The subjects were seven healthy males who trained for five or six days in a week. Their mean±SD age, height, weight and maximal oxygen intake ($\dot{V}O_{2max}$) were 20.7±1.4 years, 174.1±4.9 cm, 68.1±4.3 kg and 47.3±5.8 ml·kg^{-1}·min^{-1}. Each subject was informed of the purpose of the study, the experimental procedure and the risks associated with the experiments before consent was obtained. Furthermore, they were instructed to refrain from heavy exercise, alcohol intake and smoking on the day before experiments. This study was approved by the Ethical Committee of the Graduate School of Education, Hokkaido University.

$\dot{V}O_{2max}$ test

$\dot{V}O_{2max}$ was determined during an incremental exercise test in an environmental chamber (FJC-5500S, Fujiika Sangyo) which was regulated for the $\dot{V}O_{2max}$ measurement at 20ºC, 50% relative humidity. A cycle ergometer (75IIx, Combi) was used in the experiments. Subjects pedalled at 60 rpm. After a warm-up at 0 W for four minutes subjects performed the test the load of which was increased at a rate of 20 W·min^{-1} to exhaustion.

Constant-load exercise test

The subject was weighed, and moved to the environmental chamber with a rectal probe. The subject rested in a chair for 30 min, and then gas parameters were measured for 5 min. Then the subject performed a constant-load test at 80%$\dot{V}O_{2max}$ for 10 min under a control environmental trial (20ºC, 50% relative humidity; CON) and a hot environmental trial (40ºC, 50% relative humidity; HT). After the end of exercise, the subject sat on a chair for 10 min recovery, and then body weight was measured again without a rectal probe. Each subject completed two experiments separated by at least 7 days in random order.

Measurements

$\dot{V}E$, O_2 uptake ($\dot{V}O_2$), CO_2 output ($\dot{V}CO_2$) and P_{ETCO_2} were measured breath by breath using a respiratory gas analyser (Aeromonitor AE-300s, Minato) throughout the experiments. These were averaged and recorded for each 20-s period. $\dot{V}E$ was measured by a hot-wire flow meter that was fixed to the subject's mask. The flow meter was calibrated with a syringe of known volume (2.0 l). Sample gas was drawn continuously from the front mask for determination of the fractional concentrations of O_2 and CO_2 by a paramagnetic sensor and an infrared absorption analyser, respectively. The gas analyser

TABLE I. COMPARISON OF BODY MASS, RECTAL TEMPERATURE (Trec), OXYGEN UPTAKE ($\dot{V}O_2$), %$\dot{V}O_{2max}$, BLOOD LACTATE DURING CONSTANT-LOAD EXERCISE TEST IN CONTROL TRIAL (CON) AND HOT TRIAL (HT).

		CON	HT
Body mass	pre	67.70 ± 4.64	67.30 ± 4.31
(kg)	post	67.45 ± 4.64	66.56 ± 4.33
	Δkg	0.25 ± 0.06	0.74 ± 0.18 *
Trec	rest	36.62 ± 0.38	36.98 ± 0.15
(ºC)	peak	37.08 ± 0.40	38.02 ± 0.33 *
	ΔºC	0.46 ± 0.06	0.87 ± 0.30 *
$\dot{V}O_2$	(ml·min^{-1})	2671 ± 432	2657 ± 467
%$\dot{V}O_{2max}$	(%)	82.8 ± 6.1	82.1 ± 6.1
Blood lactate	rest	1.01 ± 0.25	0.91 ± 0.27
(mmol·l^{-1})	peak	9.61 ± 1.51	9.96 ± 1.13

Note: Values are means ± SD. N = 7 subjects., * Significant difference from CON values.

TABLE 2. COMPARISON OF BLOOD ELECTROLYTES, SODIUM ION (Na^+), POTASSIUM ION (K^+) AND CHLORIDE ION (Cl^-) DURING A CONSTANT-LOAD EXERCISE TEST IN CONTROL TRIAL (CON) AND HOT TRIAL (HT).

		rest	ex5	ex10	rec5	rec10
Na^+	CON	140.6 ± 1.5	144.4 ± 0.8	143.5 ± 2.1	139.8 ± 1.5	139.5 ± 0.8
	HT	140.7 ± 1.4	143.3 ± 1.9	142.6 ± 1.8	140.3 ± 1.5	140.1 ± 1.8
K^+	CON	4.3 ± 0.2	5.6 ± 0.2	5.1 ± 0.4	4.3 ± 0.2	4.4 ± 0.2
	HT	4.1 ± 0.2	5.6 ± 0.4	5.3 ± 0.2	4.2 ± 0.2	4.4 ± 0.3
Cl^-	CON	105.5 ± 1.0	110.7 ± 1.4	108.3 ± 2.1	105.0 ± 1.3	105.2 ± 1.0
	HT	106.7 ± 1.4	112.3 ± 1.5	111.2 ± 1.0 *	108.2 ± 1.3 *	108.5 ± 1.2 *

Note: Values are means ± SD. N = 7 subjects., * Significant difference from CON values.

was calibrated by known standard gases. The rectal temperature was measured using a thermometer (K730, Techno Seven). Twenty-five microlitres of blood was sampled using a capillary tube from the finger tip at rest before exercise, at 3, 6, 9 min during exercise, and at 3, 6, 9 min after exercise. The lactate concentration (La) in the sampled blood was determined by an automatic lactate analyser (1500 sport, YSI). The analyser was calibrated with a standard liquid (5 mM, YSI). Furthermore, 80 μl of blood was obtained using a capillary tube from the finger tip at rest before exercise, at 5 min during exercise, and immediately after the end of exercise, and at 5, 10 min after exercise. The Na^+, K^+, Cl^- concentrations in the sample blood were determined by an automatic blood electrolyte analyser (300F, i-Stat Corporation). The analyser was calibrated with a standard cartridge (EC8+, i-Stat Corporation). The criteria of $\dot{V}O_{2max}$ were i) more than 90% of maximum heart rate (HR_{max} = 220 − age), ii) levelling off of the increase of $\dot{V}O_2$ to less than 2 ml·kg^{-1}·min^{-1} of the pre-load value, iii) respiratory exchange ratio (RER) higher than 1.15. The criteria should include at least two of the above three conditions [11]. The intensity for the constant-load exercise was determined by the relationship between $\dot{V}O_2$ and work load during the $\dot{V}O_{2max}$ test. According to the method of [1], excess CO_2 output per unit of time (excess $\dot{V}CO_2$) was calculated by subtracting $\dot{V}O_2$ from $\dot{V}CO_2$ during the constant exercise test. The change of strong ion difference (SID) in the blood (ΔSID) was determined by the following formula [13].

$$\Delta SID = (\Delta Na^+ + \Delta K^+) - (\Delta Cl^- + \Delta La^-) \quad (1)$$

where, ΔNa^+, ΔK^+, ΔCl^- and ΔLa^- were calculated by subtracting the resting value.

Statistical analysis

Differences in variables obtained in two constant-load exercise tests were analysed using analysis of variance (ANOVA). Tukey's post hoc test was then conducted to assess the differences. A Student's paired t-test was used to assess the significance of differences in variables in the tests. The level of significance was $p<0.05$. Results are expressed as mean±standard deviation.

RESULTS

Body weight and rectal temperature

The body weight loss (Δkg) from pre-test to post-test was significantly greater in HT than that in CON. The peak rectal temperature was significantly higher in HT than that in CON. The change of rectal temperature (Δ°C) from pre-test to the peak value was significantly greater in HT than that in CON (Table 1).

Oxygen uptake

There was no significant difference in mean $\dot{V}O_2$ during exercise between CON and HT (2671±432 ml·min^{-1} vs 2657±467 ml·min^{-1}). The exercise intensity in CON and HT calculated by $\dot{V}O_2$ in constant-load exercise tests was 82.8±6.1% $\dot{V}O_{2max}$, 82.1±6.1% $\dot{V}O_{2max}$ respectively (Table 1).

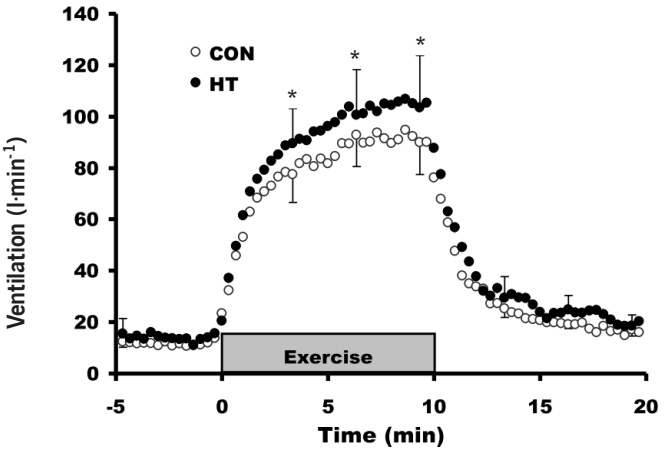

FIG. 1. COMPARISON OF MINUTE VENTILATION ($\dot{V}E$) DURING A CONSTANT-LOAD EXERCISE TEST IN CONTROL TRIAL (CON) AND HOT TRIAL (HT).
Note: Values are means ± SD. N = 7, * Significant difference from CON values

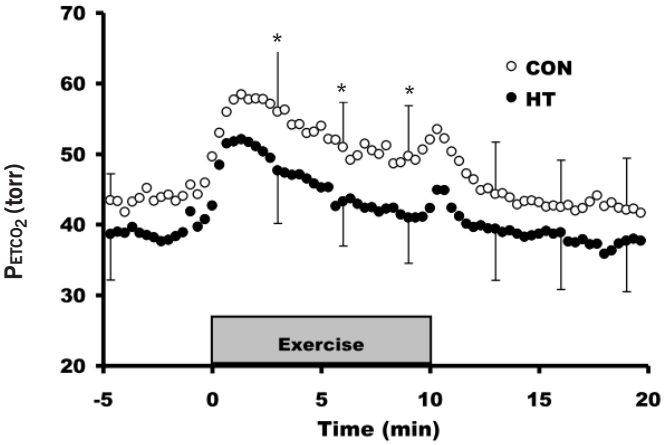

FIG. 2. COMPARISON OF END TIDAL CO_2 PRESSURE (P_{ETCO_2}) DURING A CONSTANT-LOAD EXERCISE TEST IN CONTROL TRIAL (CON) AND HOT TRIAL (HT).
Note:Values are means ± SD. N = 7 Subjects, * Significant difference from CON values.

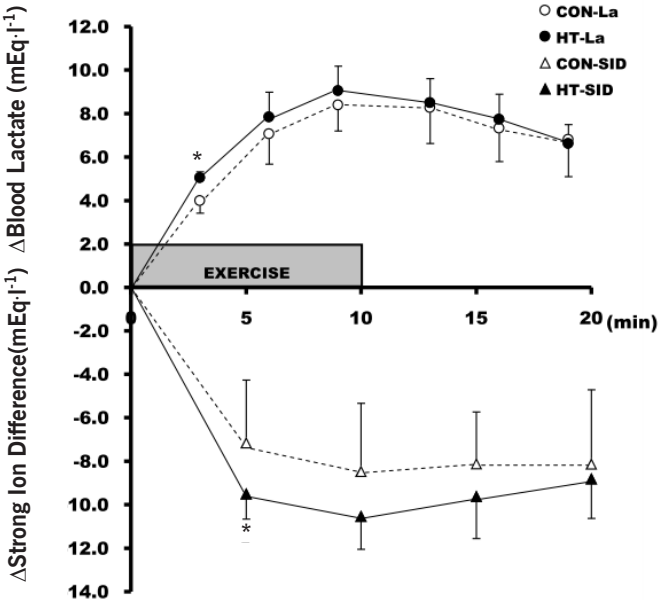

FIG. 3. COMPARISON OF END BLOOD LACTATE AND STRONG ION DIFFERENCE (SID) DURING A CONSTANT-LOAD EXERCISE TEST IN CONTROL TRIAL (CON) AND HOT TRIAL (HT).
Note:Values are means ± SD. N = 7 Subjects, * Significant difference from CON values.

$\dot{V}E/\dot{V}O_2$, P_{ETCO_2} and respiratory exchange ratio

$\dot{V}E$ was significantly higher at 3, 6, 9 min during exercise in HT than in CON (Fig. 1). $\dot{V}E/\dot{V}O_2$, which is an index of hyperventilation, was also significantly higher at 3, 6, 9 min during exercise in HT compared with in CON. P_{ETCO_2} increased immediately after the start of exercise, and then decreased gradually in both conditions. P_{ETCO_2} was significantly higher in 3, 6 min during exercise in HT compared with CON (Fig. 2). Respiratory exchange ratio (RER) decreased immediately after the start of exercise, increased for about 3 min and then decreased gradually until the end of exercise in both conditions. RER was significantly higher during exercise in HT compared with CON.

Blood lactate and blood electrolyte

La was significantly higher at 3 min during exercise in HT compared with CON (5.00±0.57 $mEq \cdot l^{-1}$ vs 5.96±0.28 $mEq \cdot l^{-1}$, $p<0.05$). However, there was no significant difference in La at 6, 9 min during exercise and during the recovery period between the two trials (Fig. 3). Table 2 shows the values of Na^+, K^+ and Cl^- in blood in CON and HT. The peak values of Na^+, K^+ and Cl^- were observed at 5 min during exercise, and then all values decreased in both conditions. There were no significant differences in Na^+, K^+ at rest, during exercise and during the recovery period. However, Cl^- was significantly higher at 10 min during exercise and at 5, 10 min during recovery in HT compared with CON. ΔSID was calculated by using the data of Na^+ and Cl^- and ΔLa^- during exercise and recovery respectively although a time lag of 1 min existed between ΔLa^- and others. ΔSID showed the lowest value at 10 min during exercise and then increased gradually during the recovery period in CON and HT. ΔSID was significantly lower at 5 min during exercise in HT compared with CON (Fig. 3).

DISCUSSION

In this study, the La was significantly higher at 3 min during high-intensity constant-load exercise in HT. However, there was no significant difference in the La after 6 min during the exercise between CON and HT.

It has been thought that the increase in La during exercise in heat would be due to the decrease in the blood flow to active muscle by the increased skin blood flow for thermoregulatory heat transport. It has also been reported that muscle glycogen utilization increased due to augmented core temperature and dehydration during prolonged exercise in heat stress [2]. In HT, however, the oxygen deficit in the early period during exercise did not increase compared with that in CON, and also mean $\dot{V}O_2$ during exercise did not alter in both conditions. Moreover, there was no significant difference in the La peak during the exercise in CON and HT. From these results, it seems that the hypoxia in active muscle does not induce an increase in La during the constant-load exercise test used in this study in heat stress.

Recently, Saunders et al. examined the activity of enzymes which regulate carbohydrate metabolism in the early period of exercise by using 70%$\dot{V}O_{2max}$ cycling exercise for 5 min at 20ºC and 40ºC [17]. They observed that the activity of glycogen phosphorylase, which is a rate-limiting enzyme of glycogenolysis, is activated sufficiently at 1 min of exercise and the enzyme activity is not different at 1 and 5 min during exercise in both conditions, resulting in no difference in lactate level in the muscle and in the blood. Therefore, the La increase observed in HT is not attributable to the metabolic change of muscle in heat stress.

It is, therefore, unclear why there was a difference in La at 3 min during exercise in both conditions. In this study, $\dot{V}E$ and $\dot{V}E/\dot{V}O_2$, which is an index of hyperventilation, increased significantly in HT compared with CON. When lactate production increases in active muscle, hydrogen ions (H^+) dissociated from the lactate increase in muscle and blood during exercise above the anaerobic threshold, resulting in metabolic acidosis. It has been suggested that the hyperventilation which is observed during exercise above the anaerobic threshold occurs to eliminate CO_2 resulting from the buffering by the bicarbonate system for increased H^+ in blood. This phenomenon is called respiratory compensation.

It is possible that metabolic acidosis occurred during the 80% $\dot{V}O_{2max}$ constant-load exercise because the La increased more than 5 $mEq \cdot l^{-1}$ in both conditions. However, $\dot{V}E$ over the course in HT was significantly higher than in CON, despite there being no difference in La after 6 min during exercise in both conditions. Therefore, hyperventilation during exercise in heat would not be derived only from the respiratory compensation related to La accumulation.

Recently, Hayashi et al. reported that during exercise in heat, $\dot{V}E$ increases with core temperature [10]. Hyperventilation during exercise observed in this study is attributable to the influence of heat stress. Although hyperventilation with hyperthermia increases heat loss, the heat loss by breathing is considerably smaller than body surface heat loss from sweating, which is effective heat dissipation from the head. Therefore increased ventilation in hyperthermic humans could contribute to selective brain cooling [19].

Hyperventilation during exercise in heat occurs simultaneously with the decrease of P_{ETCO_2} and the increase of RER. The increase in excess CO_2 output with hyperventilation induces the decrease in HCO_3^- in blood, resulting in respiratory alkalosis. It was thought that the relationship between the decrease in HCO_3^- and the increase in lactic acid in blood should be explained by the notion of non bicarbonate buffering and bicarbonate buffering of lactic acid. Recently, however, the change in the amount of HCO_3^- in blood is considered equivalent to the change in strong ion difference (SID) under the condition which is no change in CO_2 pressure. Moreover, under the condition of lactic acid production in active muscles, SID should be taken as follows [13]:

$$\Delta SID = (\Delta Na^+ + \Delta K^+) - (\Delta Cl^- + \Delta La^-)$$

In this study, the difference in strong cations, ΔNa^+ at 5 min during exercise between HT and CON was -1.3 $mEq \cdot l^{-1}$ and ΔK^+ was 0.2 $mEq \cdot l^{-1}$, the difference in anions, ΔCl^- was 0.5 $mEq \cdot l^{-1}$,

ΔLa was 0.8 mEq·l^{-1} (data from 6 min during exercise); as a result, ΔSID decreased 2.4 mEq·l^{-1} in HT compared with CON.

If ΔHCO_3^- is equivalent to ΔSID, the decrease of SID in HT would include the decrease of HCO_3^- by hyperventilation in heat. Because the whole electrolyte in blood is neutral electrically and the changes of Na^+, K^+ are approximately parallel, it is necessary for some anions to change to compensate the decrease of HCO_3^-. Therefore the diffusion of lactate ion as an anion from muscle cell to blood might increase in the early period of exercise.

In this study, ΔSID at 10 min of exercise and recovery decreased in HT compared with CON. From the result, hyperventilation would affect ΔSID during the later part of exercise and recovery. However, from the later part of exercise to the recovery period, La as a component of ΔSID showed no difference under both conditions. SID might be regulated by La during the early stage of exercise since it is more labile from a cell than other electrolytes. After that time, however, SID regulation by La would not be necessary because other electrolytes begin to move. Therefore there was no difference in La after 6 min of exercise. The results of former studies that La did not increase during exercise in heat might not be considered as a result of the change of SID.

CONCLUSIONS

It was concluded that hyperventilation during short-term high-intensity constant-load exercise in heat is temporarily associated with an increase in La.

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