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METABOLIC ALKALOSIS INDUCED BY PRE-EXERCISE INGESTION OF $NaHCO_3$ DOES NOT MODULATE THE SLOW COMPONENT OF VO_2 KINETICS IN HUMANS

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Seven healthy physically active nonsmoking men, aged 22.4 ± (SD) 1.8 years performed two 6 min bouts of cycling at 40% VO_{2max} (sub-lactate threshold / low power output exercise) and 87% VO_{2max} (supra-lactate threshold / high power output exercise) at 70 rev · min^{-1}, separated by 20 minutes rest, on two occasions: once as a control experiment (test C) and on a different day at ~ 1,5 h after ingestion of 250 mg (3 mmol) · (kg body weight)$^{-1}$ of $NaHCO_3$ (test A). At the onset of low and high power output exercise performed after ingestion of $NaHCO_3$, antecubital venous blood pH and HCO_3^- were significantly elevated ($p < 0.05$). Moreover, blood pH and HCO_3^-, tested at every minute of low and high power output exercise, was significantly higher ($p < 0.05$) in test A than in test C. No difference was found in plasma lactate concentration $[La]_{pl}$ during low power output exercise between A and C tests. In the terminal phase of the high power output exercise (87% VO_{2max}) the level of $[La]_{pl}$ rose more rapidly in test A than in test C, reaching in the sixth minute of cycling 8.27 ± 1.11 and 6.76 ± 0.68 mmol · l^{-1} ($p < 0.01$) in test A and C, respectively. No significant differences were found in the rate of VO_2 measured breath-by-breath between A and C tests, both during low and high power output exercise. The slow component of VO_2 kinetics (expressed by difference betwen VO_2 measured at the 6th minute of exercise minus the VO_2 reached at the 3rd minute), occuring only during exercise corresponding to 87% VO_2, was not significantly different in C and A tests (0.373 ± 0.050 and 0.339 ± 0.078 l O_2, respectively). The total VO_2 consumed throughout the six minute cycling at power output of 40 and 87% VO_{2max} performed in control conditions and after ingestion of $NaHCO_3$ was not significantly different. We have demonstrated that significantly reduced exercise acidemia accompanied by a significantly elevated level of $[La]_{pl}$ accumulation, did not affect the slow component of the VO_2 kinetics and the magnitude of oxygen uptake during exercise corresponding to 40 and 87% of VO_{2max}.

Key words: *acid-base balance, bicarbonate, metabolic alkalosis, oxygen uptake kinetics, exercise*

INTRODUCTION

In the initial phase of a low power output exercise (below the onset of blood lactate accumulation) the increase of oxygen uptake follows a monoexponential process, reaching steady state within about 3 minutes (1, 2). However, when high intensity exercise is performed, even at a constant power output, no steady state in oxygen uptake can be reached (2). After finishing the fast phase of increase, which normally takes place within 3 minutes, VO_2 slowly increases

until the end of exercise. The slow increase in VO_2 has been called 'the slow component of the VO_2 kinetics' (for review see 3). The appearance of the slow component of VO_2 kinetics which may be expressed by the difference between VO_2 measured at the 6th minute of exercise minus the VO_2 reached at the 3rd minute (ΔVO_2^{6-3}) (2) reflects an increase in the VO_2/power output ratio and a decrease in muscle efficiency.

Despite the fact that the slow component of the VO_2 kinetics is a well documented phenomenon, the physiological mechanisms responsible for the progressive increase in VO_2 at a constant power output are not fully understood. Understanding the physiological mechanisms controlling this process seems to be of great importance especially for improving exercise tolerance of patients, limited by oxygen transport to the muscle and its utilization, in whom the slow component of VO_2 uptake appears already at extremely low power output.

One may argue that the increase in the pulmonary VO_2/power output ratio has its origin in the decrease of mechanical efficiency of the active muscles, or is simply being caused by additional factors, such as an increase in the cost of ventilation and cardiac output (4) and/or involvement of additional groups of muscles. In light of the study by Poole *et al.* (5), in which muscle VO_2 was measured simultaneously with pulmonary VO_2, the increase in pulmonary VO_2/power output ratio is accompanied by an increase in muscle VO_2/power output ratio. These authors have shown that during constant load high power output exercise, 86 percent of the slow component of pulmonary VO_2 arises from changes within the exercising limbs (5). Thus a major part of the slow component of total body VO_2 measured during cycling seems to have its origin in exercising muscles.

It has been reported that the magnitude of the slow component of VO_2 kinetics positively correlates with the degree of blood lactate concentration (6—9). Moreover, it has been recently suggested that acidosis play a major regulatory role in the mechanism(s) responsible for the slow component of VO_2 kinetics (10, 11).

If indeed the magnitude of the slow component of the VO_2 kinetics is influenced by the degree of acidemia, then shifting the blood acid-base balance into alkalosis should be reflected in a decrease of the magnitude of the slow component of the VO_2 kinetics. To our knowledge no data has been published showing the influence of changes in the acid-base balance on the magnitude of the slow component in VO_2 kinetics.

This is why in the present study we have evaluated the effect of pre-exercise metabolic alkalosis on the rate of VO_2 uptake at the onset of exercise of low and high power output in humans and on the magnitude of the slow component in the VO_2 kinetics measured breath-by-breath during exercise corresponding to 87% VO_{2max}.

METHODS

Subjects

Seven healthy, physically active nonsmoking males (means ± SD.; age, 22.4 ± 1.8 years; body weight 75.8 ± 4.3 kg; height 183.3 ± 7.8 cm, body fat 12.1 ± 1.8% of B.W.) participated in this study. The maximum oxygen uptake (VO_{2max}) of the subjects was 50.4 ± 4.0 ml·min^{-1}·kg^{-1}. An interview of a medical history and physical examination were completed before the study. Moreover basic blood tests for hemoglobin (Hb), hematocrit value (Ht), erythrocyte (E), leukocyte (L), sodium (Na^+), potassium (K^+) and creatinine (Cr) were performed (see *Table 1*). The subjects abstained from a fatiguing physical activity one day before and on the day of the experiments. All of them were experienced in laboratory exercise tests.

Table 1. Subject hematocrit value (Ht), hemoglobin concentration (Hb), erythrocyte count (E), leukocyte count (L), sodium (Na^+), potassium (K^+) and cretinine concentration (Cr) in antecubital venous blood.

Subject	Ht (vol. %)	Hb (g%)	E (mln·mm^{-3})	L (1000·mm^{-3})	Na^+ (mmol·l^{-1})	K^+ (mmol·l^{-1})	Cr (μmol·l^{-1})
A	40.4	13.9	4.65	8.2	144.5	4.29	76.0
B	43.8	14.5	5.26	6.5	142.5	4.52	85.3
C	46.7	16.3	5.30	5.6	145.0	4.06	81.3
D	50.2	16.9	5.58	7.0	144.5	3.82	95.2
E	47.0	16.0	5.40	6.7	143.5	3.62	110.9
F	45.5	15.6	5.19	6.7	145.5	4.47	91.2
G	45.3	15.2	5.29	6.6	147.0	4.37	96.8

Procedures

Preliminary tests

At about two weeks before performing the constant power output tests, all subjects performed incremental tests until exhaustion on a cycle ergometer. Before the experiments were started, the ergometer was calibrated according to the manual. During the incremental test, the subjects cycled at a pedalling rate of 70 rev·min^{-1}. After 3 minutes of cycling at an initial power output of 30 watts, the power output was then increased by 30 W every 3 minutes, until exhaustion. During the test, gas exchange variables were measured continuously (breath-by-breath). One minute before starting the first bout of exercise and at the end of each step (the last 15 seconds), blood samples were taken for plasma lactate concentration $[La]_{pl}$. The tests were stopped when the subjects could no longer maintain the required pedalling rate and power output.

The constant power output tests

The subjects performed two constant power output exercises (each of six min. duration) separated by a 20 minute pause. The power output during the first bout of exercise was planned to correspond to 40% VO_{2max} and the second bout — the high power output exercise — was planned to be performed at power output corresponding to 80% VO_{2max}. The calculations of individual power output corresponding to 40 and 80% VO_{2max} were based on the linear relationship of VO_2/power output data obtained during incremental tests, taking into consideration only data below the stage of sustained increase in $[La]_b$ concentration (see 12). For this calculation, values of VO_2 reached during the 3rd minute of each stage of incremental test were applied.

The low power output exercise, corresponding to 40% VO_{2max}, in each subject was below the lactate threshold established during the incremental test, whereas the power output corresponding to 80% VO_{2max} was above the lactate threshold.

At one minute before starting the constant power output exercise and at the end of each minute of cycling, blood samples (1 ml each) were taken for determination of blood gases and plasma lactate concentration. Starting from 2 minutes prior to exercise, gas exchange variables were measured continuously using the breath-by-breath system. During the experiments the room temperature and the relative humidity of air was about 22°C and 50%, respectively.

All exercise tests were performed on the same cycloergomer Ergo-line 800s the Netherlands. Care was taken to provide on each occasion the same cycling position by adjusting the height of the saddle.

Ingestion of $NaHCO_3$

At about I week after performing the constant power output tests (control experiment), the subjects reported to the laboratory once again. On this occasion within about 30 minutes the subjects ingested 250 mg · kg B.W.$^{-1}$ (3 mmol · kg B.W.$^{-1}$) of $NaHCO_3$ placed in capsules containing 420 mg (5 mmol) of $NaHCO_3$ each. During ingestion of the capsules the subjects were allowed to drink up to 500 ml of water. At the beginning of ingestion and every 15 minutes during ingestion of $NaHCO_3$, blood samples were taken to determine the changes in the blood acid-base balance. *Fig. 1* presents the changes in antecubital blood HCO_3^- and pH during and after the

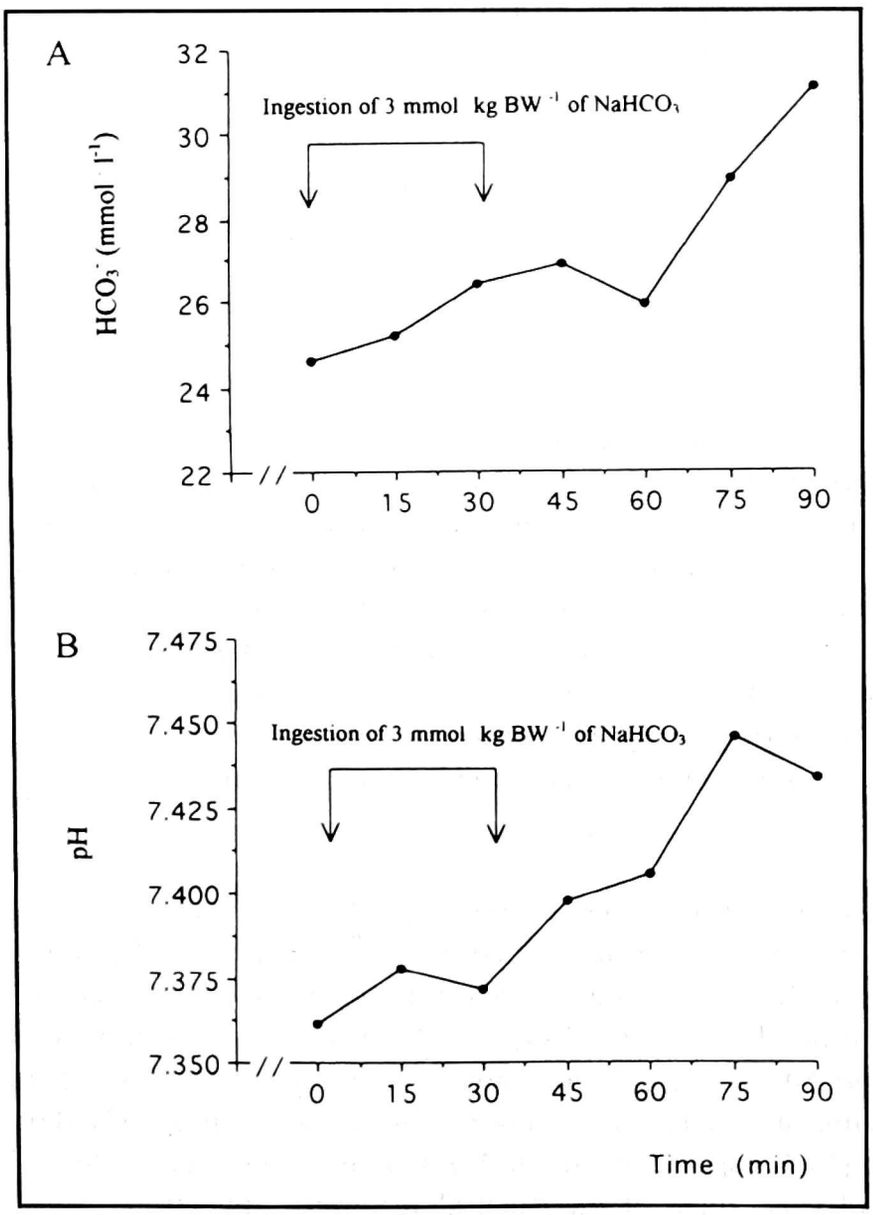

Fig. 1. Changes in antecubital venous blood HCO_3^- (panel A), and pH (panel B) during and after ingestion of 250 mg (3 mmol) · (kg body weight)$^{-1}$ of $NaHCO_3$.

ingestion period. When a significant alkalemia had occured, which normally happened about 1.5 hours after ingestion of $NaHCO_3$, the subjects performed the constant power output tests once again. Care was taken that the meal ingested up to one day before the control experiment was exactly repeated before the exercise performed after alkalization.

Gas exchange variables

The gas exchange variables were measured continuously breath-by-breath (Oxycon Champion Jaeger, Germany). Before and after finishing each test, gas analyzers were calibrated with certificated calibration gases. The first 20 s of this calibration were used to flush the analyzers, the last 10 s were used to measure the concentration of sample gas. The ventilation volume sensor was calibrated with a 3 l syringe. After at last six complete strokes, the average values of the last five strokes were used to calculate inspiratory and expiratory values. The measured values had to fall within 1% of the reference values. The repeated calibration procedures had shown very high stability of the breath-by-breath system over the duration of the test.

Blood sampling and analysis

The intravenous catheter Int-Catheter Abbott Ireland (18G/1.2 × 45 mm) was inserted into the antecubital vein. The catheter was connected with the Extension Set with 'T' Adapter SL Abbott Ireland (a tube of 10 cm of lenght). The 1 ml samples of blood were taken anaerobically, usually within 5 seconds. Each time, immediately before taking the blood samples for analysis, 1 ml of blood volume were taken in order to eliminate the blood from the catheter and the T-set. One part of each sample was taken for detection of blood gases (PO_2 and PCO_2) and pH, using a heparinized 90 µl capillary. The second part (0.5 ml) was placed in 1.8 ml Eppendorf tubes containing 1 mg ammonium oxalate and 5 mg sodium fluoride and mixed for about 20 seconds. Subsequently, in order to separate plasma for performing lactate measurements, the blood samples were centrifuged. Samples of blood plasma (200 µl) were stored for further analysis at a temperature of minus 25°C.

PO_2 and PCO_2, as well as pH were determined using a Ciba-Corning analyzer 238 (England). The blood bicarbonate concentration (HCO_3^-) was calculated by this unit. Plasma lactate concentration was detected using Automatic Analyzer Biochemistry Kodak Ektachem XR 700 (USA). Serum sodium (Na^+) and potassium (K^+) concentration was determined using a flame photometer, (Ciba Corning, Model 480, England). Blood creatinine level was determined by the kinetics method based on reaction with picric acid using an automatic analyzer, (Express 550 CBI, England). Hemoglobin concentration (Hb), hematocrit value (Ht), erythrocyte count (E) and leukocyte count (L) were determined using an automatic hematological analyzer, Baker 9000 (USA). The percentage of body fat has been assessed according to Hassager *et al.* (13).

Statistical analysis

Values represent means ± S.D. Statistical significance was tested by a paired t-test. The chosen level of significance was $*p < 0.05$, $**p < 0.02$ and $***p < 0.01$.

RESULTS

Acid-base balance, plasma lactate concentration and VO_2 during the low power output exercise (107 ± 14 W), corresponding to 40% VO_{2max}

After ingestion of 250 mg · kg^{-1}BW of $NaHCO_3$, the pre-exercise values of antecubital blood HCO_3^- and pH, were significantly higher ($p < 0.01$) than in control conditions (*Fig. 2 A and Fig. 2 B*). This shift of the acid-base balance

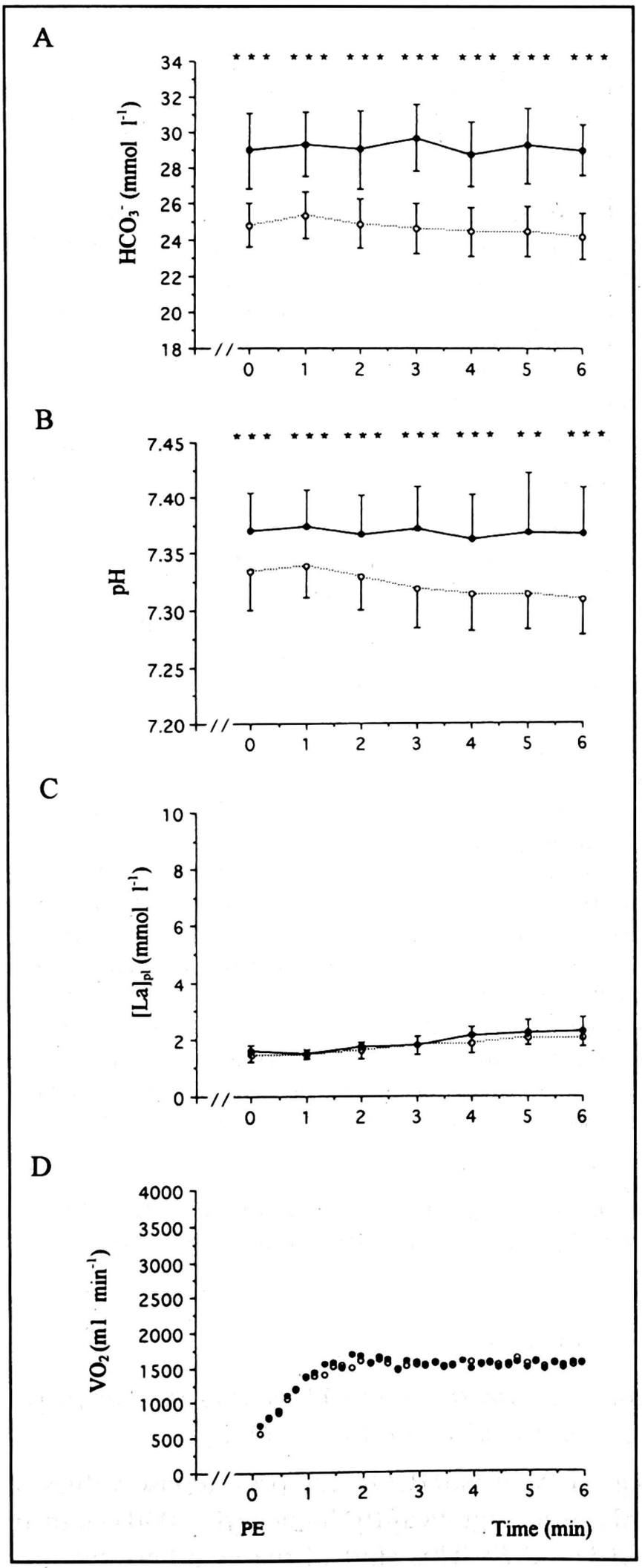

Fig. 2. Antecubital venous blood HCO_3^- (panel A), pH (panel B), plasma lactate concentration $[La]_{pl}$ (panel C), and oxygen uptake (panel D) during exercise corresponding to 40% VO_{2max} (○ — control, ● — after ingestion of $NaHCO_3$). PE — the pre-exercise values. Paired t-test $*p < 0.05$, $**p < 0.02$ and $***p < 0.01$. Data for HCO_3^-, pH, and $[La]_{pl}$ are presented as means ± SD for 7 subjects. Data for VO_2 are given as mean values for 7 subjects, for 36 ten-second intervals. Paired t-test have shown no significant difference in VO_2 at any of the 36 intervals as a results of ingestion of $NaHCO_3$.

into alkalosis was also present throughout the 6 minute exercise at power output corresponding to 40% VO_{2max} (see *Fig. 2 A and 2 B*). No difference in pre-exercise plasma lactate concentration has occured in alkalotic condition in relation to the control experiment. The plasma lactate concentrations measured during exercise performed in control and alkalotic conditions were not significantly different (*Fig. 2 C*). The rate of VO_2 at the onset of exercise as well as throughout 6-minute exercises was similar both in control and alkalotic conditions (*Table 2*), and no slow component in VO_2 was present during exercise corresponding to 40% VO_{2max} (*Fig. 2 D*). The comparison of VO_2

Table 2. Mean (± SD) values of the percentage of VO_{2max} (n = 7), reached during the low power output exercise (40% VO_{2max}) and during the high power output exercise (87% VO_{2max}) performed in control conditions and after ingestion of $NaHCO_3$ (alkalosis). The significance of differences between the control and alkalotic conditions were tested for each minute (n.s.; $p > 0.05$).

Low Power Output Exercise				High Power Output Exercise		
Time (sec)	**Control**	**Alkalosis**	*Signif. diff.*	**Control**	**Alkalosis**	*Signif. diff.*
0	12.0 (4.7)	10.0 (4.6)	*n.s.*	13.0 (4.1)	12.1 (2.5)	*n.s.*
60	25.0 (2.8)	26.2 (3.5)	*n.s.*	37.6 (6.4)	37.6 (5.2)	*n.s.*
120	39.3 (3.5)	40.9 (3.8)	*n.s.*	71.2 (6.1)	71.8 (4.7)	*n.s.*
180	41.0 (2.9)	41.4 (3.5)	*n.s.*	77.3 (6.0)	78.5 (5.1)	*n.s.*
240	41.0 (3.1)	40.5 (4.4)	*n.s.*	81.1 (5.8)	81.1 (5.4)	*n.s.*
300	40.8 (2.8)	40.4 (3.0)	*n.s.*	85.2 (6.9)	85.0 (5.6)	*n.s.*
360	40.6 (3.3)	40.8 (3.9)	*n.s.*	87.1 (6.6)	87.5 (6.6)	*n.s.*

expressed as mean values of 10-second intervals (using paired t-test) have shown no significant differences between control and alkalotic conditions. The total of oxygen consumed throughout 6-minute exercise in the control test has amounted to 8 685 ± 628 ml, it was not significantly different from the value reached after ingestion of $NaHCO_3$, which amounted to 8 770 ± 820 ml.

Acid-base balance, plasma lactate concentration and VO_2 during the high power output exercise (256 ± 24 W), corresponding to 87% VO_{2max}

The power output (256 ± 24 W), which during incremental test corresponded to 80% VO_{2max} (see methods), in the 6th minute of the constant power output exercise required ~ 87% of VO_{2max} (*see Table 2*).

At one minute prior to the exercise corresponding to 87% VO_{2max}, performed after ingestion of $NaHCO_3$, blood acid-base balance was significantly shifted into alkalosis when compared to the control conditions ($p < 0.05$) (*Fig. 3A and Fig. 3 B*). This shift of the acid-base balance into alkalosis was also present throughout the 6 minute exercise at power output corresponding to 87% VO_{2max}. The plasma lactate concentration measured one minute prior to

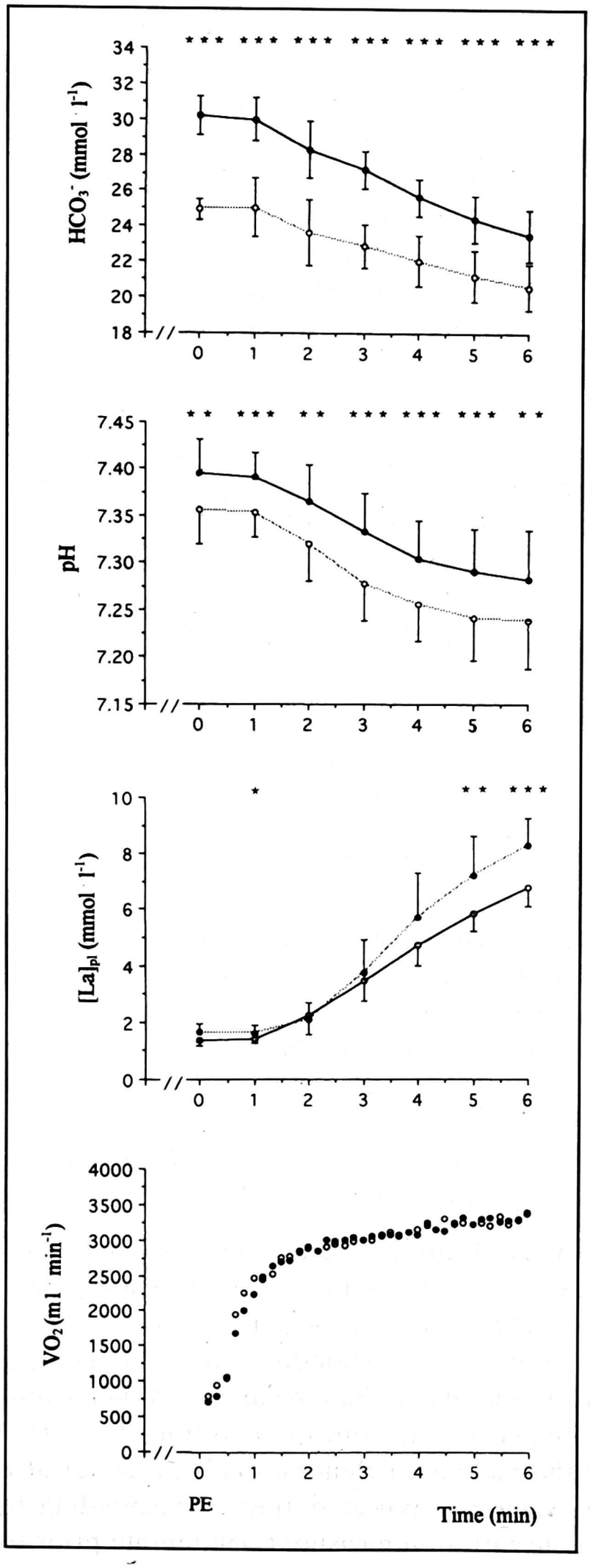

Fig. 3. Antecubital venous blood HCO_3^- (panel A), pH (panel B), plasma lactate concentration $[La]_{pl}$ (panel C), and oxygen uptake (panel D) during exercise corresponding to 87% VO_{2max} ○ — control, ● — after ingestion of $NaHCO_3$). PE — the pre-exercise values. Paired t-test $*p < 0.05$, $**p < 0.02$ and $***p < 0.01$. Data for HCO_3^-, pH, and $[La]_{pl}$ are presented as means ± SD for 7 subjects. Data for VO_2 are given as mean values for 7 subjects, for 36 ten-second intervals. Paired t-test have shown no significant difference in VO_2 at any of the 36 intervals as a results of ingestion of $NaHCO_3$.

exercise was not significantly different in control and alkalotic conditions. However, during exercise performed after ingestion of $NaHCO_3$, starting from the 5th minute of exercise plasma lactate concentration rose significantly faster than in the control conditions (see *Fig. 3 C*). The plasma lactate level in the 6th min of cycling performed after ingestion of $NaHCO_3$ was by 1.5 mmol $\cdot$ l^{-1} higher than in control conditions. Oxygen uptake at the onset of exercise corresponding to 87% VO_{2max} rose very rapidly and no difference could be seen between the rate of VO_2 in control and alkalotic conditions (see *Table 2*). After completing the fast phase of the increase in VO_2, no steady state was reached, but a slow continuous increase in VO_2 occured in both control and alkalotic conditions. Comparison of VO_2 expressed as mean values of 10-second intervals (using paired t-test) have shown no significant differences between control and alkalotic conditions (*Fig. 3 D*). The difference in VO_2 between the 6th and the 3rd minute of exercise, representing the magnitude of the slow component of VO_2 kinetics, amounted to 373 $\pm$ 50 vs. 339 $\pm$ 78 ml, in control and alkalotic conditions respectively. The magnitude of the slow component of VO_2 kinetics determinant in control and alkalotic conditions was not significantly different. The VO_2 reached in the 6th minute of cycling in the control conditions was not significantly different from the value reached in the alkalotic conditions: 3320 $\pm$ 258 *v.s* 3334 $\pm$ 247, respectively. The total of oxygen consumed throughout 6-minute exercise in the control test has amounted to 16 760 $\pm$ 1 437 ml, it was not significantly different from the value reached after ingestion of $NaHCO_3$, which amounted to 16 838 $\pm$ 1 421 ml.

DISCUSSION

It is well documented that, when a constant high power output exercise accompanied by blood lactate accumulation is performed, there is a progressive increase of the VO_2/power output ratio, called the slow component of VO_2 kinetics (2, 3, 7). Although the magnitude of the slow component of VO_2 kinetics is closely related to the degree of blood lactate accumulation (6—9), the influence of acidemia on the slow component of VO_2 remains unclear.

In the present study, we have examined the influence of pre-exercise metabolic alkalosis on the magnitude of the slow component of the VO_2 kinetics in humans during 6 minutes cycling at constant power output corresponding to 87% VO_{2max} (above the onset of blood lactate accumulation). The main finding of our study is that a significant shift of the blood pH into alkalosis, developed by ingestion of 250 mg $\cdot$ kg^{-1} BW of $NaHCO_3$ (see *Fig. 3 B*), had no effect on the magnitude of the slow component of VO_2 kinetics (see *Fig. 3 D* and *Table 2*). Moreover, as illustrated by the data presented at *Fig. 3 C*, plasma lactate concentration $[La]_{pl}$ in the alkalotic condition rose

significantly faster than in the control experiment. In the final state of exercise corresponding to 87% VO_{2max} performed after ingestion of $NaHCO_3$, $[La]_{pl}$ was 1.5 mmol $\cdot 1^{-1}$ higher than in control exercise; however, such elevation of plasma lactate concentration had no influence on the magnitude of the slow component of VO_2 kinetics, which amounted to 373 ± 50 vs. 339 ± 78 ml, in control and alkalotic conditions, respectively. The presented findings suggest that modification of the blood pH in the range as ilustrates the *Fig. 3 B* has no direct influence on the magnitude of the slow component of VO_2 kinetics.

An increase in the rate of blood lactate accumulation during exercise performed after ingestion of $NaHCO_3$ in humans been already reported (14). However, the physiological mechanism responsible for the faster rate of $[La]_b$ accumulation during exercise performed in alkalosis has not been found. A number of factors have been suggested to explain the increase of $[La]_b$ concentration in patients during alkalosis, including increased lactate production from red cells, decreased carrier-mediated lactate transport from blood to liver cells, decreased lactate consumption by diminished hepatic gluconeogenesis from lactate (see 15) and acceleration of lactate diffusion from the muscle cells (16, 17). In our experiment the pre-exercise $[La]_{pl}$ concentration in alkalotic and in control conditions did not differ. No difference in $[La]_{pl}$ level between alkalotic and control conditions was seen during low power output exercise corresponding to 40% VO_{2max}. Thus or study does not support the suggestion that the faster rate of $[La]_{pl}$ accumulation in alkalosis is due to intensification of release of lactate from erythrocytes. It seems most likely that the cause of acceleration of blood lactate concentration accompanying alkalosis is due to intensification of glycolysis and/or increase in the rate of lactate efflux from the muscle cells.

It may be asked why the slow component VO_2 is accompanied by pronounced blood lactate accumulation, as reported in several independent observations. Indeed a number of studies have shown that $[La]_b$ accumulation during constant power output exercise (2, 3, 7) is accompanied by the increase in the VO_2/power output ratio. Moreover, recently Żołądź *et al.* (12) have shown that, during incremental test onset of blood lactate accumulation is accompanied by a pronouced increase in the VO_2/power output ratio. Thus indeed the blood lactate accumulation occuring during intense exercise seems to be associated with the decrease of muscle efficiency. However, the mechanism by which $[La]_b$ accumulation may influence the VO_2/power output ratio remains unclear.

Recently Stringer *et al.* (11), have suggested that the slow component of VO_2 kinetics is just caused by blood lactic acid accumulation causing a shift of the oxyhemoglobin dissociation curve to the right and increasing oxygen transport to the muscle and to promote aerobic metabolism during heavy exercise.

If indeed metabolic acidosis may influence the rate of VO_2 during high power output exercise in such a way as suggested by Stringer *et al.* (11), then alkalization of blood should slow down the rate of VO_2 and decrease the magnitude of slow component of oxygen uptake. However, our data do not support such a scenario. During exercise corresponding to 87% VO_{2max} performed after ingestion of $NaHCO_3$, we observed a significantly higher blood pH than in control conditions (*Fig. 3 B*), but no difference in VO_2 throughout 6 minutes of cycling (see *Fig. 3 D* and *Table 2*).

Another possible explanation of the close relationship between metabolic acidosis and the slow component of VO_2 kinetics has been described by Capelli *et al.* (10). These authors have postulated that acidosis may contribute to the upward drift in oxygen uptake. This may be caused by intensification of the rate of mitochondrial respiration in the muscle by increase in the free ceratine, as already suggested by Mahler (18). The rise in free creatine may be caused by the shift in the equilibrium of the creatine kinase reaction due to H^+ accumulation (see 19). In our study we did not measure the intramuscular H^+ concentration. However, on the basis of data presented by Costill *et al.* (20), who found significantly higher muscle pH after a series of repeated bouts of exercise performed after ingestion of $200\,mg \cdot kg^{-1}$ B.W. of $NaHCO_3$, when compared to control conditions, we can guess that in our study, during exercise corresponding to 80% VO_{2max} performed after ingestion of 250 mg (3 mmol) · (kg body weight)$^{-1}$ of $NaHCO_3^-$, muscle pH was significantly higher than in the control test.

The influence of increase of $[La]_b$ concentration on the rate of VO_2 during exercise has been evaluated in experiments in which lactate was infused during exercise. Ryan *et al.* (21) for example have shown that infusion of sodium L(+)-lactate during exercise performed at 50% VO_{2max} causing an increasing of (L−) from 3.92 to $5.25\,mmol \cdot l^{-1}$ (respectively for control and infusion conditions) was accompanied by an increase in VO_2 of $129\,ml \cdot min^{-1}$. On the other hand, a recent study by Poole *et al.* (22) in which animal model was applied does not support the suggestion that lactate mediates the slow component of VO_2 kinetics. In our study the significantly faster rate of $[La]_b$ accumulation present during exercise performed after ingestion of $NaHCO_3$ had no influence on the magnitude of the slow component of the oxygen uptake (see *Fig. 3 D*). Moreover, it has been demonstrated that an increase of blood lactate concentration induced by infusion of epinephrine does not influence the level of VO_2 during exercise (23, 24).

In the light of our study, the manipulation with blood pH in the range as illustrates *Fig. 2 B* and *Fig. 3 B* has no direct influence on the magnitude of the slow component of VO_2 kinetics and the amount of VO_2 required to perform the exercise at power output corresponding to 40 and 87% of VO_{2max}. We believe that the increase in the VO_2/power output ratio (the slow component of

VO_2 kinetics) observed at high power output exercise is a consequence of recruitment of type II muscle fibres, being less efficient in terms of VO_2/power output ratio than type I (see 25, 26), but the increase in blood lactate accumulation is just a metabolic consequence of intensification of glycolysis in the glycolytic type II muscle fibres. This seems to be in accordance with early findings showing a positive linear relationship between the magnitude of the slow component of VO_2 kinetics and the level of blood lactate accumulation reported in several studies (6—9). One should also consider a decrease of muscle efficiency developed by exercise induced hyperthermia (27, 28), as a possible cause of the slow component of the VO_2 kinetics.

In conclusion, the results of our study confirmed the early findings showing that the appearance of the slow component of VO_2 kinetics is accompanied by $[La]_b$ accumulation and a decrease in blood pH. However, we demonstrated that pre-exercise ingestion of $3\ mmol \cdot kg^{-1}$ of B.W. of $NaHCO_3$, causing a significant shift of blood pH into alkalosis, did not influence the rate of oxygen uptake at the onset of exercise. Moreover, significantly reduced exercise developed acidemia accompanied by a significantly higher level of $[La]_{pl}$ accumulation, when compared to control conditions, did not affect the magnitude of the slow component of the VO_2 kinetics. Thus or results clearly demonstrate that the changes of blood pH and enhancement of $[La]_{pl}$ concentration had no direct influence on the magnitude of the slow component of VO_2 kinetics.

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Local Ethical Committee approval was obtained for this study.

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