Effects of CO₂ Inhalation Prior to Maximal Exercise on Physical Performance

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Summary In the untrained subjects, inhalation of 4.5–6.0% CO₂ prior to maximal treadmill exercise does not affect physical performance and maximum oxygen uptake, while blood lactate levels during recovery have a tendency to greater decrease in CO₂ breathing than that in the room-air breathing. It was suggested that CO₂ inhalation immediately prior to maximal exercise as applied here is not a useful tool in increasing physical performance.

Key words: CO₂ inhalation, physical performance, blood lactate.

It is well known that, during intensive exercise, lactic acid (LA) is formed and accumulates in the working muscle. Concomitant with lactate accumulation there is a rise in concentrations of intracellular hydrogen ions. In a previous study performed by HermanSEN and Osnes (1972), changes in muscle pH were evaluated repeatedly after continuous and intermittent maximal exercise. Muscle pH was found to decrease to about the same level in both continuous and intermittent exercise, thus lending support to the hypothesis that muscle fatigue during maximal exercise is, in part, caused by the accumulation of hydrogen ions within the working muscle. On the other hand, Graham et al. (1980) have reported that during exhaustive bicycle exercise there were significant decreases in the blood lactate and in the feeling of local fatigue of legs during 4% CO₂ breathing as compared with room-air breathing. These phenomena were confirmed by EhRSAM et al. (1982) while inspired CO₂ concentration and work loads were different, more or less, from those of Graham et al. (1980). Reduced feeding of local fatigue in the exercising legs might be due to the decrease in the concentration of hydrogen ions because lactate production is inhibited during metabolic and respiratory acidosis (Rizzo et al., 1976; Jones et al., 1977; FujitsuKA et al., 1980; Sutton et al., 1981). It may be possible to assume that physical performance could be improved by the inhibition
of lactate production during maximal exercise even if the subject inhaled CO₂ prior to exercise but not during exercise. However, there are no available data whether or not physical performance and blood lactate were affected by CO₂ inhalation immediately prior to maximal exercise. The purpose of this study, therefore, was to examine the effect of acute induced respiratory acidosis prior to exercise on aerobic work capacity and blood lactate levels of untrained subjects in maximal treadmill running.

Sixteen healthy male students, aged 18–21 years, participated in this study as subjects. All subjects gave their consent after the nature of the experiments had been explained. The subjects came to the laboratory three times. On the first day, each subject was familiarized only with the apparatus and testing procedure. In order to ascertain the effect of CO₂ inhalation prior to maximal treadmill running on physical performance, double determinations were carried out for each subject on separate days, generally 1 week apart: on one day, after CO₂ inhalation and on the other day, after inhalation of room air (placebo); these two tests for each subject were conducted arbitrarily. The subjects were divided into two groups. The first 8 subjects (hereafter called A group) and the second 8 subjects (hereafter called B group) were studied to ascertain the difference in concentration of inhalation CO₂ prior to exercise; the A group inhaled 4.5% CO₂, 21% O₂, N₂ balance, and the B group inhaled 6.0% CO₂, 21% O₂, N₂ balance.

Each individual was weighed prior to testing. After electrocardiographic leads had been placed, the subject warmed-up on the treadmill at a speed of 120 m/min for 30 min at a constant temperature and humidity (25 ± 1°C db, 60 ± 3% RH) in a climatic chamber. This warm-up was followed by rest periods of 5–10 min; during rest periods the subject inhaled CO₂ gas mixture or room air from a Douglas bag of about 200 l. The inhalation time was 3–7 min for the CO₂ gas mixture and 4–9 min for the room air. The difference in the inhalation time of CO₂ gas mixture may be mainly due to individual difference of CO₂ sensitivity (IRSIGLER, 1976). After 1–1.5 min of inhalation of CO₂ or room air, maximal exhaustive exercise was performed using stepwise incremental loading on a treadmill with a constant grade of 8.6%. The initial speed was chosen so that the subjects could run for 4 to 8 min before they were exhausted. In most cases, the initial speed was found to be 110 to 130 ml/min. The subjects were allowed to breathe room air during warm-up, exercise, and recovery.

Oxygen uptake during exercise was determined by the Douglas bag technique. Expired gas was collected into a Douglas bag every minute until exhaustion. The collected gas volume was measured with a wet-gasometer, and gas analysis was performed with an oxygen analyzer (Morgan, England) and an infrared CO₂ analyzer (Capnograph, Holland). Heart rate during exercise was continuously monitored and output from the amplifier was connected to a pen recorder (Fukuda, Japan). In order to obtain venous blood, a 21-gauge butterfly needle with a sampling vinyl tube was inserted into the antecubital vein as quickly as possible after exhaustive exercise. About 5 ml of venous blood was withdrawn into a

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As described previously, GRAHAM et al. (1980) and EHRSAM et al. (1982) have reported that there was a distinctly reduced feeling of local fatigue in the legs with CO2 breathing, although the work loads were identical to room-air exercise. We speculate that this could be due to a number of factors including decrease of muscle lactate, since it has been shown that blood lactate was reduced when the subject continuously breathed CO2 gas mixture during exercise (RIZZO et al., 1976; GRAHAM et al., 1980; MCCARTNEY et al., 1983). If so, it is possible to infer that the inhalation of hypercapnic gas prior to exercise may be a valuable tool in the improvement of physical performance. In the present study, average value and standard deviations of treadmill endurance time in room air and CO2 breathing prior to exercise were 6.90 ± 1.33 and 6.63 ± 1.31 min for the A group, and 5.84 ± 0.98 and 6.02 ± 1.08 min for the B group, respectively. No significant difference of endurance time and feeling of local fatigue in the legs were noticed between CO2 and room air in both groups. As shown in Table 1, furthermore, there are no significant differences in maximum heart rate, maximum pulmonary ventilation, maximum oxygen uptake, and maximum oxygen uptake per kg of body weight between CO2 and room air, whereas respiratory exchange ratio in the A group was higher in the CO2 inhalation than that in the room air (p < 0.05). These results indicate that the CO2 inhalation prior to exercise as applied here does not affect endurance and maximum aerobic power.

It was found that blood lactate during recovery was lower in CO2 breathing than that in the room-air breathing, but not significant statistically (Fig. 1). We have

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<th>Physical performance and physiological parameters in maximal treadmill exercise in the A and B groups.</th>
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<td><strong>Group A</strong></td>
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<td><strong>Room air</strong></td>
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<td>Endurance time (min)</td>
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Values are mean ± S.D. HRmax, maximum heart rate; VEmax, maximum pulmonary ventilation; VO2max, maximum oxygen uptake; VO2max/BW, maximum oxygen uptake per kg of body weight; R, respiratory exchange ratio. Significant level of difference between CO2 and room-air breathing represented by asterisk. *p < 0.05.
recently observed, however, that in athletes blood lactate level during recovery is significantly ($p < 0.01$) lower in 8% CO$_2$ breathing immediately prior to 200 m sprint run as compared with ambient air breathing, though record of 200 m sprint run and feeling of local fatigue did not change by CO$_2$ inhalation (MIYAMURA et al., 1989). These results infer that blood lactate during recovery decreases by CO$_2$ inhalation prior to exercise and the degree of decrement in blood lactate is relative to the concentration of inspired CO$_2$. In these experiments, we could not measure blood pH and CO$_2$ storage before and after maximal exercise. However, it was suggested that lower lactate concentration obtained in the CO$_2$ inhalation may be due to inhibition of glycolysis by respiratory acidosis induced by CO$_2$ inhalation prior to exercise, even though the subjects were allowed to breath room air during warm-up, exercise, and recovery.

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