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Delayed appearance of blood lactate with reduced frequency breathing during exercise

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Summary. The purpose of the present study was to investigate the blood lactate (LA^-) responses to hypoventilation induced by reduced frequency breathing (RFB) during recovery from exercise. Five male subject performed 16 4 min cycling bouts alternating with 16 min rest periods. Exercise intensities were chosen at power outputs corresponding to 30% $\dot{V}_{O_{2max}}$ at 2 mMLA^-, $\dot{V}_{O_2}$ at 4 mMLA^-, and 90% $\dot{V}_{O_{2max}}$ in each subject. Breathing frequency was voluntarily controlled starting 10 s before each 3rd min of exercise and maintained throughout the rest of the exercise period. Four different breathing patterns at each exercise intensity were used: normal breathing (NB), breathing every 4 s, breathing every 8 s, and maximal RFB. Except for the NB trials, subjects held their breath at functional residual capacity during each breathing interval. The concentration difference of LA^- between the 3rd min sample and the 4th min sample was defined as the lactate change during exercise (ΔLA^-ex), and that between the 4th min sample and the sample at the 3rd min after the end of the exercise as the lactate change during recovery (ΔLA^-rec). An ANOVA showed significant ($p<0.05$) differences in breathing procedures only in ΔLA^-rec. ΔLA^-rec seemed to increase as compared to NB only at $\dot{V}_{O_2}$ at 4 mMLA^- and 90% $\dot{V}_{O_{2max}}$, while ΔLA^-ex remained unchanged as compared to NB in spite of reduced $\dot{V}_A$. These results might indicate that RFB inhibited lactate removal from working muscles during exercise.

Key words: Reduced frequency breathing — Hypoventilation — Lactate removal — Human

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Introduction

It is well known that alveolar ventilation ($\dot{V}_A$) is maintained by regulating both tidal volume and breathing frequency to supply an adequate amount of oxygen to working muscles. In these two controlled variables, breathing frequency is easily suppressed by many factors such as aquatic environment, cadence of exercise mode, psychological state etc. Therefore, in athletic training situations, it has been thought that reduced frequency breathing (RFB) can be used to create alveolar hypoventilation induced arterial hypoxemia and to enhance lactate production in working muscles (Counsilman 1977).

However, studies concerning RFB during swimming (Dicker et al. 1980; Holmer and Gullstrand 1980) and cycling (Craig 1979) failed to show enhanced lactic acidosis. Craig (1979); Dicker et al. (1980), and Holmer and Gullstrand (1980) reported alveolar oxygen pressures (P_AO_2) of 77—88 Torr (10.2—11.7 kPa) during exercise with RFB, inferring that this reduction in P_AO_2 was insufficient to cause a marked reduction in arterial oxygen saturation (SaO_2). Recently, Yamamoto et al. (1987b) reported that there was a large increase in the alveolar-arterial difference for oxygen pressure (A-aDO_2) and a decrease in SaO_2 as low as 88% by direct arterial sampling, but no increase in blood lactate concentration (LA^-) with RFB, during exercise.

Although there was no increase in blood LA^-, however, it remains possible that there was an increase in LA^- in the working muscle, but that the movement of LA^- into the blood was suppressed while the exercise continued (Graham et al. 1986), and that a rise in blood LA^- may not have occurred until after the exercise had ceased. In the present study, therefore, we have sought to deter-

mine whether there is a rise in blood LA^- during the post-exercise recovery period.

Methods

Subjects. The subjects were five healthy males ranging from 21—27 years in age, 170.5—183.1 cm in height, and 55.6—82.0 kg in weight. Before the experiment, each subject signed a written consent from after being informed of its purpose and risks.

Procedures. On the first day of the experiment, each subject performed an initial incremental exercise test which consisted of cycling until exhaustion on a Monark bicycle ergometer with an incremental load increase by 15 W every 4 min, starting at 0 W. Maximal oxygen uptake ($\dot{V}_{O_{2max}}$) and LA^- responses were measured.

On the second day of the experiment, the subjects performed sixteen 4 min exercise bouts alternating with 16 min rest periods on a Monark bicycle ergometer. Exercise intensities were chosen at power outputs corresponding to 30% $\dot{V}_{O_{2max}}$, $\dot{V}_{O_2}$ at 2 mmol · l^{-1} of LA^- ($\dot{V}_{O_2}$ at 2 mMLA^-), $\dot{V}_{O_2}$ at 4 mmol · l^{-1} of LA^- ($\dot{V}_{O_2}$ at 4 mMLA^-), and 90% $\dot{V}_{O_{2max}}$ for each subject, determined from the results of the initial incremental exercise. Breathing frequency was voluntarily controlled, starting 10 s before each 3rd min of exercise and maintained throughout the rest of the exercise period. Four different breathing patterns were used: normal breathing (NB), breathing every 4 s, breathing every 8 s, and reduced frequency of breathing as low as possible. Except for the NB trials, subjects held their breath at functional residual capacity (FRC) during each breathing interval because breath holding at FRC has been reported to increase A-aDO_2 and to induce a marked arterial hypoxemia (Yamamoto et al. 1987b). Subjects came to the laboratory at 9:00 AM and performed 16 successive exercises in one day. For each subject, 4 × 4 blocks of exercise intensities and breathing procedures were assigned, using a randomly selected standard greco-latin square. Effect of exercise intensities, breathing procedures, and exercise intensity — breathing procedure interactions were examined by three-way ANOVA.

Variables measured. Pulmonary ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}_{O_2}$), carbon dioxide output ($\dot{V}_{CO_2}$), respiratory exchange ratio (R), and end-tidal oxygen and carbon dioxide pressures were measured on a real time breath-by-breath basis (Yamamoto et al. 1987a), using a Fleisch type pneumotachograph Model i/a 7321 (DYNASCIENCE, USA), a differential pressure transducer MP45-871 (Validyne, USA) and a 1H26 polarographic oxygen and infrared carbon dioxide analyzer (San-Ei-Sokki, Japan). Analog-digital conversion was performed by hardware interruption form a 12-bit real time A/D converter PCN-2198 (Neolog-Denshi, Japan). Breath-by-breath signal processing was performed by a personal computer (PC-9801Vm2, NEC, Japan) and mean values during the 4th min of the exercise period were reported. Alveolar carbon dioxide pressure (P_ACO_2) was estimated from the end-tidal value. $\dot{V}_A$ and P_AO_2 were estimated as follows (Jones and Campbell 1982):

$$\dot{V}_A = \dot{V}_E \times \frac{P_ACO_2 - P_ECO_2}{P_ACO_2}$$

$$P_AO_2 = 0.2093 \times (P_B - 47) + P_ACO_2 \times \left(0.2093 + \frac{0.7904}{R}\right)$$

where P_ECO_2 and P_B represent the carbon dioxide pressure of mixed expired air and barometric pressure respectively.

Before the experiment, saline filled catheters (TOP 18G TOP-KASEI, Japan) were inserted into a right or left dorsal hand vein. One cc polyethylene syringes were used to collect heated arterialized venous blood samples (Forster et al. 1972) anaerobically at the 3rd and 4th min of exercise and 3 min after the end of exercise. Immediately following sample collection the syringes were sealed and stored anaerobically on ice until analyzed, within 10—15 min.

LA^- was measured by an automated lactate analyzer (MODEL 23L, Yellow Springs Instrument, USA). The concentration difference between the 3rd and 4th min samples was defined as the lactate change during exercise (ΔLA^-ex), and the difference between 4th min exercise sample and that taken 3 min after the end of the exercise as the lactate change during recovery (ΔLA^-rec).

Results

Figure 1 shows the relationship between $\dot{V}_E$ and $\dot{V}_A$ during exercise. At each exercise intensity, of course, the decrease in $\dot{V}_E$ was the primary measure of the hypoventilation induced by RFB. Since, however, as shown in Fig. 1, $\dot{V}_A$ decreased linearly with $\dot{V}_E$, in the following figures all other data have been plotted against $\dot{V}_A$.

Figure 2 shows $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$ and R in relation to $\dot{V}_A$. With all the variables plotted against $\dot{V}_A$ in Fig. 2, there were significant ($p < 0.01$) differences between the experimental variables obtained for the different experimental conditions (breathing procedure). Significant ($p < 0.01$) differences were also observed in all the variables with exercise intensity. The linear decrease in $\dot{V}_{CO_2}$ with reduction in $\dot{V}_A$ was greater than the corresponding decrease in $\dot{V}_{O_2}$. Consequently, R decreased as $\dot{V}_A$ decreased.

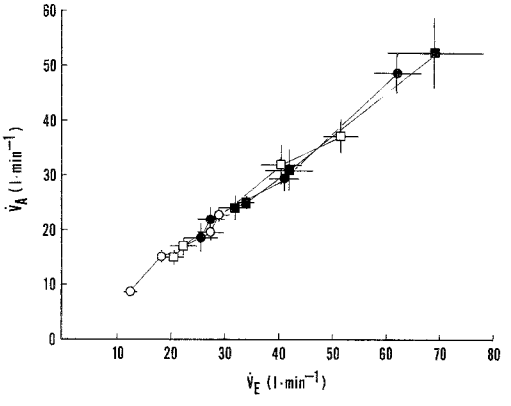

Fig. 1. The relationship between pulmonary ventilation ($\dot{V}_E$) and alveolar ventilation ($\dot{V}_A$). Open circles, open squares, closed circles and closed squares represent the values at 30% $\dot{V}_{O_{2max}}$, $\dot{V}_{O_2}$ at 2 mMLA^-, $\dot{V}_{O_2}$ at 4 mMLA^- and 90% $\dot{V}_{O_{2max}}$, respectively. Vertical and horizontal bars express S.E.M. The same symbols will be used in Fig. 2—4

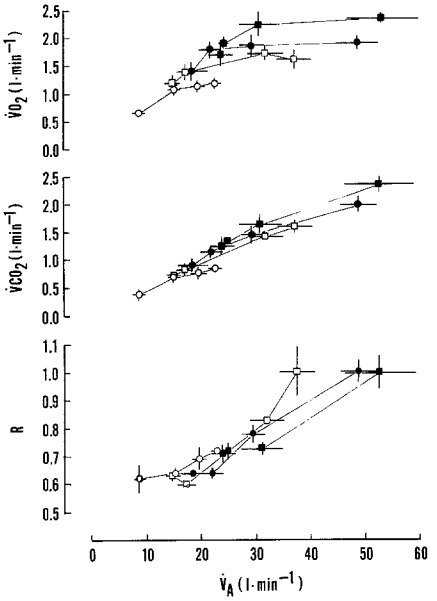

Fig. 2. Oxygen uptake ($\dot{V}_{O_2}$), carbon dioxide output ($\dot{V}_{CO_2}$), and respiratory exchange ratio (R) in relation to $\dot{V}_A$

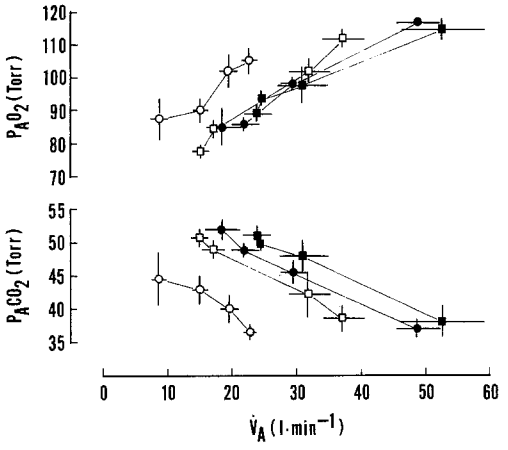

Fig. 3. Estimated alveolar oxygen pressure (P_AO_2) and alveolar carbon dioxide pressure (P_ACO_2) in relation to $\dot{V}_A$

In Fig. 3, estimated P_AO_2 and P_ACO_2 values are plotted against $\dot{V}_A$. It can be seen that P_AO_2 fell linearly and P_ACO_2 rose linearly as $\dot{V}_A$ was reduced in all four experimental conditions, although the plots for the 30% $\dot{V}_{O_{2max}}$ condition fall to the left of those for the other three conditions.

Figure 4 shows ΔLA^-ex and ΔLA^-rec in relation to $\dot{V}_A$. There were significant ($p<0.05$) differences between the breathing procedures only in ΔLA^-rec, while no significant ($p<0.05$) differences were found in exercise intensity in either ΔLA^-ex or ΔLA^-rec. ΔLA^-rec seemed to increase when $\dot{V}_A$ had fallen to approximately half of the NB values, but only at higher exercise intensities ($\dot{V}_{O_2}$ at 4 mMLA^- and 90% $\dot{V}_{O_{2max}}$).

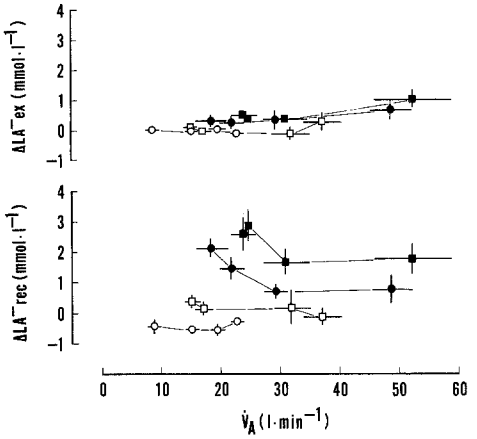

Fig. 4. The concentration difference in blood lactate over exercise periods (ΔLA^-ex) and over recovery periods (ΔLA^-rec) in relation to $\dot{V}_A$

Discussion

The results of the present study showed that hypoventilation induced by RFB led to decreased pulmonary $\dot{V}_{O_2}$ and increased LA^-, especially in the recovery phase from exercise. To compare the effects of hypoventilation clearly, each subject performed a normal exercise period of 3 min before controlling the breathing frequency to make the physiological responses to exercise near "steady-state". This was confirmed by real time breath-by-breath visual monitoring of the expired gas parameters.

Effect of RFB on Pulmonary $\dot{V}_{O_2}$

Decreases in P_AO_2 to below 90 Torr (12.0 kPa) and in pulmonary $\dot{V}_{O_2}$ to below 1 l · min^{-1} at $\dot{V}_A$ below 20 l · min^{-1} were observed during RFB. As shown in Fig. 1, the $\dot{V}_A/\dot{V}_E$ ratio remained almost constant during RFB, which indicates that there were no increase in the physiological dead space, which would have affected $\dot{V}_{O_2}$. A possible explanation for decreased pulmonary $\dot{V}_{O_2}$ will be provided.

It is well known that a reduction in lung volume occurs during breath holding with oxygen (Kloche and Rahn 1965; Mithoefer 1965) and with air (Mithoefer 1959a, 1959b, 1965). Mithoefer (1965) summarized the mechanisms of lung

volume reduction from the standpoint of alveolar gas exchange during breath holding. During breath holding, outward movement of CO_2 is reduced because of the increase in P_ACO_2, although the uptake of O_2 is less reduced because of the flat shape of the oxygen dissociation curve at normal alveolar tensions (Mithoefer 1965). $\dot{V}_{CO_2}$ reduction exceeds $\dot{V}_{O_2}$ reduction, and the lung volume is thereby gradually reduced. This phenomenon can be observed as decrease of R. Lanphier and Rahn (1963) reported that during breath holding with air at rest, R fell to 0.3 within 10 s from the onset of breath holding, and reached zero beyond 40 s. In the present study, $\dot{V}_{CO_2}$ decreased linearly with the reduction in $\dot{V}_A$, but was less than the decrease in $\dot{V}_{O_2}$, therefore, R reduced as $\dot{V}_A$ decreased (Fig. 1). It seems likely that lung volume during each breathing interval might have been restricted by the RFB of this study.

Findley et al. (1983) reported that the differences between measured SaO_2 and that predicted from P_AO_2 increased when lung volume at breath holding reduced below closing capacity. Craig et al. (1971) and Weenig et al. (1974) also reported that A-aDO_2 increased when the closing capacity exceeded FRC. This increase in A-aDO_2 can be attributed to an increased ventilation-perfusion inequality resulting from airway closure in the lower lung. Considering the facts that: 1) RFB in this study was accompanied by breath holding at FRC, 2) there was a strong possibility of lung volume reduction, suggested by the decreased R, and 3) there was no increase in physiological dead space effect, it could be said that the decreased $\dot{V}_{O_2}$ shown with RFB in this study was partly due to a mechanically induced limitation in ventilation-perfusion matching for oxygen to diffuse in each alveoli. Because of the limited information, however, understanding of the complete mechanism of decreased $\dot{V}_{O_2}$ must await further research.

Effects of RFB on LA^-

Although decreased pulmonary $\dot{V}_{O_2}$ does not directly result in a decreased muscular $\dot{V}_{O_2}$ (by decreased blood oxygen stores), increased LA^- was observed during recovery from exercise.

Adams and Welch (1980) reported that there was a slight but significant ($p<0.05$) increase in LA^- during exercise at 55% $\dot{V}_{O_{2max}}$ in a hypoxic condition (oxygen fraction of inspired air = 17%) and a large increase at 90% $\dot{V}_{O_{2max}}$ compared with the same level of exercise in a normoxic condition. Hogan et al. (1983), using the same hypoxic condition, found no significant ($p>0.05$) increases in LA^- until reaching 4 mMLA^- during incremental exhaustive exercise. At a work rate of 195 W, however, LA^- increased significantly ($p=0.001$) over that with same rate of work in normoxic conditions ($LA^- = 5.5$ mM). Also in this study, ΔLA^-rec rose significantly ($p<0.05$), especially at $\dot{V}_{O_2}$ at 4 mMLA^- and at 90% $\dot{V}_{O_{2max}}$, when $\dot{V}_A$ decreased to half the NB values. However, ΔLA^-ex showed no increases, as shown in the previous studies of RFB (Holmer and Gullstand 1980; Hsieh and Hermiston 1982; Yamamoto et al. 1987b). These results clearly indicate a delay in the appearance of LA^- consistent with the inhibition of the movement of accumulating LA^- out of the working muscles during exercise with RFB, in spite of accentuated anaerobic glycolysis in working muscles.

It has also been reported that hypercapnia or respiratory acidosis reduces LA^- (Ehrsam et al. 1982; Graham et al. 1980, 1982). Marked hypercapnia reported in previous studies of RFB (Craig 1979; Dicker et al.1980; Holmer and Gullstrand 1980; Yamamoto et al. 1987b) was also observed in the present study (Fig. 3). Recently, Graham et al. (1986) reported that, using a perfused canine gastrocnemius-plantaris model, respiratory acidosis (4% carbon dioxide) reduced lactate efflux from working muscles. Hirche et al. (1975), Mainwood and Worsley-Brown (1975), and Seo (1984) have also indicated that the permeability of the membrane to lactate increased with increment in extracellular pH. It might be considered that the consistent inhibition of lactate efflux from working muscles due to hypercapnia-induced extracellular acidosis occurred during exercise with RFB.

In conclusion, it seems likely that RFB during exercise does enhance muscle lactate production when the oxygen supply to the working muscles becomes decreased, but that this effect cannot be gauged from changes in LA^-.

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