Ventilatory and heart rate responses to hypoxia and hypercapnia in long distance runners

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Summary

Six male long distance runners were examined by an isocapnic progressive hypoxia test and CO_2 rebreathing test, both before and after 2 years of endurance training.

The results of ventilatory and heart rate responses to hypoxia were analyzed by the hyperbola equation and ventilatory response to hypercapnia by the linear regression equation, respectively:
$\dot{V}_E = \dot{V}_0 + A_{VE}/(P_{ETO2} - C_{VE})$, $HR = HR_0 + A_{HR}/(P_{ETO2} - C_{HR})$, and $\dot{V}_E = S(P_{ETCO2} - B)$. where $\dot{V}_E$ and HR are observed ventilation and heart rate, $\dot{V}_0$ and HR_0 the horizontal asymptotes in ventilation and heart rate for infinite end-tidal Po_2 (P_{ETO2}), A_{VE} and A_{HR} the slope constants indicating the magnitude of hypoxic sensitivity, C_{VE} and C_{HR} the vertical asymptotes in P_{ETO2} for infinite ventilation and heart rate, S the slope of the line expressed as change in ventilation per unit change in end-tidal Pco_2 (P_{ETCO2}), and B the extrapolated intercept on the abscissa (P_{ETCO2} axis).

1) Running record improved in every athlete after 2 years of endurance training.

2) Simultaneously, hypoxic ventilatory sensitivity decreased in all the athletes after the training.

3) Hypoxic heart rate sensitivity, A_{HR}, also decreased in all the athletes

except one after the training.

4) However, the training did not elicit significant influence on hypercapnic ventilatory sensitivity, S.

Introduction

Since Byrne-Quinn et al. (1971) first reported the blunted ventilatory responses to hypercapnia and hypoxia in athletes, a number of investigations studying the chemical control of ventilation have been carried out in athletes of various sports (Godfrey et al., 1971; Rebuck and Read, 1971; Leitch et al., 1975; Miyamura et al., 1976; Saunders et al., 1976; Scoggin et al., 1978; Martin et al., 1978; Blum et al., 1979; Hughson, 1980; Ohkuwa et al., 1980; Yoshida and Seki, 1980; Mahler et al., 1982; Ohyabu et al., 1982 and 1984; Honda et al., 1983; Kelley et al., 1984). The reported results, however, are conflicting. Some agreed with the initial findings, but others could not confirm significant changes in ventilatory chemosensitivities.

The disagreement from these results may be accounted for by the difference in the kinds of sports, training period, degree of exercise or athletic performance of the subjects studied. Moreover, there is evidence indicating that blunted chemosensitivities found in elite athletes may be related to familial or genetic influence (Rebuck and Read, 1971; Leitch et al., 1975; Scoggin et al., 1978).

To assess the effect of physical training on the ventilatory chemosensitivities, therefore, a study which will exclude other possible influences seems called for in a homogeneous, uniform group of athletes. In the present experiments, follow-up study examining the effect of endurance training on the ventilatory response to hypercapnia and hypoxia was conducted in the college long distance runners.

Materials and Methods

Subjects

Six male long distance runners, aged 18–19 years, were studied. The physical characteristics and running records of the subjects are shown in

Table 1. Physical characteristics and running time of the long distance runners.

		Before training	After trainig
Age	(yrs)	18.8±0.4	20.8±0.4
Weight	(kg)	58.8±4.4	58.9±4.9
Height	(cm)	171.5±5.4	171.9±5.7
$\dot{V}_{O_2}$ at rest	($ml \cdot min^{-1}$)	299±77	380±41
$\dot{V}_E$ at rest	($l \cdot min^{-1}$)	10.26±2.91	10.29±0.95
Running records			
10000 M (n=2)		32 min 12.1 sec±11.7 sec	31 min 29.9 sec±7.3 sec
5000 M (n=4)		15 min 27.2 sec±31.4 sec	15 min 0.1 sec±29.1 sec

Values are mean ± SD.

Table 1. All the runners were physically very active and participated in at least 3 hrs of endurance training every day during a 2-year period. Furthermore, they had already been training for 6 to 7 years during the high-school period. The present observations started just in the beginning of freshman. Informed consent was obtained from all the subjects with explanation of the experimental procedures but not of the purpose.

Experimental procedure

The experiments were conducted at least 3 hrs after the last meal, and 30 min rest was required just before the test.

The experimental arrangements used in the progressive hypoxia and CO_2 rebreathing test, following the methods of Weil et al., (1970) and Read (1967), respectively, are shown in Fig. 1. In the hypoxia test, the subjects,

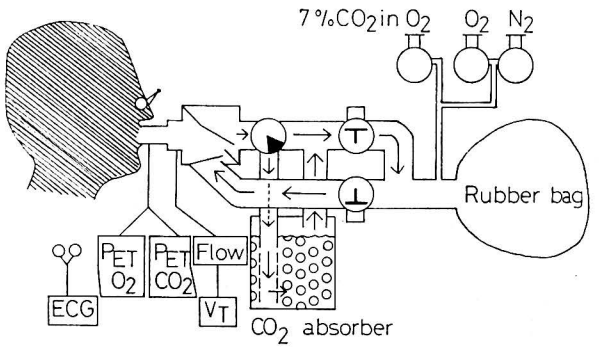

Fig. 1. A schematic drawing of the experimental set-up.

holding a mouth-piece in place, breathed room air at first, then started rebreathing into a bag filled with approximately 18 l of room air.

By adjusting the amount of O_2 inflow from an O_2 cylinder and of CO_2 absorbed by a CO_2 absorber in the by-pass circuit, end-tidal Po_2 and Pco_2 (P_{ETO2} and P_{ETCO2}) were set at 100 mm Hg and 5 mm Hg higher than the control value in room air breathing, respectively. These P_{ETO2} and P_{ETCO2} values were maintained for at least 3 min in order to obtain a steady state ventilatory condition. Then, while maintaining the P_{ETCO2} level constant, P_{ETO2} was progressively lowered by turning off the O_2 supply from the O_2 cylinder. The rate of P_{ETO2} fall was approximately 10 mm Hg per min. The rebreathing was terminated when P_{ETO2} decreased to 40 mm Hg. In the hypercapnic test, the subjects started rebreathing into the bag filled with approximately 7 l of 7% CO_2 in O_2. The rebreathing was terminated when P_{ETCO2} increased to about 70 mm Hg.

A probe with a hot wire respiratory flowmeter (Minato Medical Science Co., Ltd., Tokyo, Japan) inserted between the rebreathing bag and the mouth-piece was carried out to detect the breath-by-breath respiratory flow, which was integrated to tidal volume (V_T). At the same time, ECG was recorded by a cardiac telemeter (SAN-EI Cardiosuper 2E31A). The average heart rate was then obtained from every 8 R-R interval.

The expired air at rest was collected for 5 min. The amounts of air, O_2 and CO_2 concentrations were measured by a dry gasometer, and O_2 and CO_2 analyzer (San-ei Expired Gas Monitor 1H21, Tokyo), respectively. After this, the oxygen consumption was calculated.

Results

Data analysis

In the hypoxia test, ventilatory and heart rate responses were analyzed by a modified hyperbolic equation (Weil et al., 1970), which Lloyd and Cunningham (1963) originally used to evaluate ventilatory responses to steady state hypoxia.

$$\dot{V}_E = A_{VE}/(P_{ETO2} - C_{VE}) + \dot{V}_0$$

The same hyperbola equation was also applied to the heart rate response by our previous communication (Ohyabu et al., 1984) as:

$HR = A_{HR}/(P_{ETO2} - C_{HR}) + HR_O$, where $\dot{V}_E$ and HR are minute ventilations and heart rate, $\dot{V}o$ and HRo are horizontal asymptotes in ventilation and in heart rate for infinite P_{ETO2}, A_{VE} and A_{HR} are the slope constants of the hyperbola expressing the degree of hypoxic sensitivity of the subjects, and C_{VE} and C_{HR} are vertical asymptotes in P_{ETO2} for infinite $\dot{V}_E$ or HR.

To obtain the above three parameters in the equation, C_{VE} and C_{HR} were first determined by a curve-fitting procedure using a microcomputer (Apple II), i.e., for seeking the C_{VE} and C_{HR} values which gave the best correlation coefficients between $\dot{V}_E$ and $1/(P_{TEO2} - C_{VE})$ and HR and $1/(P_{ETO2} - C_{HR})$.

Then, from the linear regression between $\dot{V}_E$ and $1/(P_{ETO2} - C_{VE})$ or HR and $1/(P_{ETO2} - C_{HR})$, $\dot{V}o$ and A_{VE} or HRo and A_{HR} were determined as the intercept at the ordinate and the slope of the regression line, respectively (Fig. 2).

The absolute magnitudes of hypoxic response in ventilation and heart rate at P_{ETO2} 40 mm Hg were also determined as $\Delta\dot{V}_{40}$ and ΔHR_{40}, respectively, which were calculated as follows:

$\Delta\dot{V}_{40} = A_{VE}/(40 - C_{VE})$,

$\Delta HR_{40} = A_{HR}/(40 - C_{HR})$.

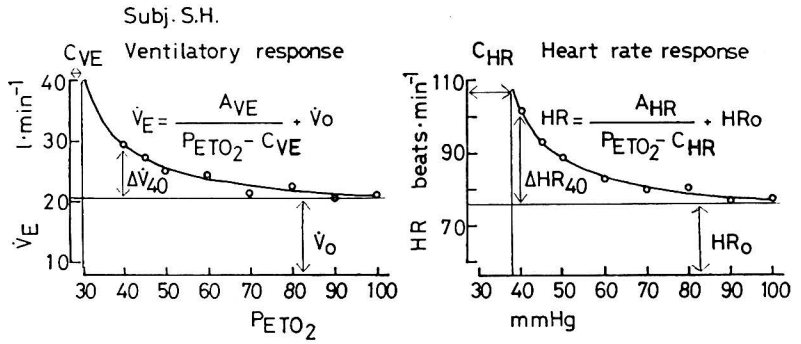

Fig. 2. Ventilatory and heart rate responses are plotted against end-tidal P_{O2}(P_{ETO2}). Data were obtained from subj. S.H. Both responses are shown to adequately expressed by a hyperbola equation described in the figure.

In the hypercapnic test, ventilatory response was evaluated by the slope of the regression line between P_{ETCO_2} and $\dot{V}_E$.

The equation traditionally used to relate ventilation and P_{ETCO_2} is as follows:

$\dot{V}_E = S(P_{ETCO_2} - B)$, where B is the extrapolated intercept on the abscissa (P_{ETCO_2} axis) and S is the slope of the line expressed as change in ventilation ($\dot{V}_E$, $l \cdot min^{-1}$ BTPS) per unit change in P_{ETCO_2} (P_{ETCO_2}, mm Hg),

Hypoxic responses

As shown in Table 1, body weight and height before and after training for 2 years were not found to be significant by different. $\dot{V}o_2$ at rest after training was larger than that of 2 years ago, but no significant difference was seen after training.

As can be expected, the running records improved in every athlete as a result of the endurance training (Fig. 3).

As seen in Table 2, the slopes of the ventilatory response to hypoxia (A_{VE}) were 482.0 ± 364.0 and 186.2 ± 251.8 $l \cdot min^{-1} \cdot mm$ Hg, before and after training for 2 years, respectively. A_{VE} in every athlete after endurance training was smaller than that of 2 years earlier. Furthermore, $\Delta\dot{V}_{40}$ significantly decreased after the training (Fig. 4). The slope of the heart rate response to hypoxia (A_{HR}) were 1013.9 ± 669.0 and 646.6 ± 347.1 beats$\cdot min^{-1} \cdot$mm Hg, before and after the 2 years training, respectively (Fig. 5).

A_{HR} in all the athletes with the exception of one subject after the train-

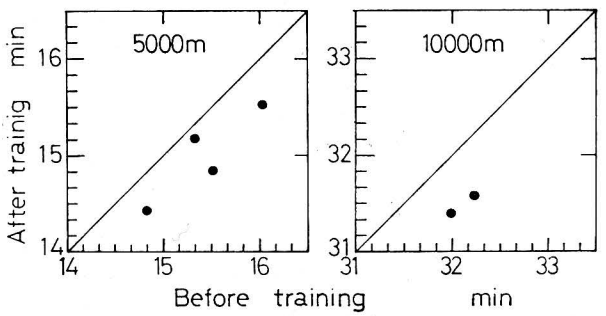

Fig. 3. Comparison of running time before and after training.

Table 2. Ventilatory and heart rate responses to hypercapnia and hypoxia.

	Before training	After training
Ventilatory responses		
S ($l \cdot min^{-1} \cdot mmHg^{-1}$)	1.14± 0.55	1.30± 0.54
B (mmHg)	29.0 ±15.0	37.1 ± 6.5
A_{VE} ($l \cdot min^{-1} \cdot mmHg$)	482.0 ±364.0	186.2 ±251.8
C_{VE} (mmHg)	17.6 ± 8.7	26.2 ±14.2
$\dot{V}_0$ ($l \cdot min^{-1}$)	11.0 ± 5.5	13.1 ± 5.6
$\Delta\dot{V}_{40}$ ($l \cdot min^{-1}$)	19.6 ± 8.1	9.5 ± 6.2*
Heart rate responses		
A_{HR} (beats$\cdot min^{-1} \cdot$mmHg)	1013.9 ±669.0	646.6 ±347.1
C_{HR} (mmHg)	10.0 ±13.2	11.0 ±12.5
HR_0 (beats$\cdot min^{-1}$)	53.8 ±13.0	54.4 ± 7.5
ΔHR_{40} (beats$\cdot min^{-1}$)	31.9 ±13.2	21.2 ± 6.2

Values are mean ± SD. * $p<0.05$

S is the slope of the line expressed as change in ventilation per unit change in P_{ETCO_2}, B is the extrapolated intercept on the abscissa, A_{VE} and A_{HR} the slope constants indicating hypoxic sensitivity, C_{VE} and C_{HR} the vertical asymptotes in P_{ETO_2}, $\dot{V}_0$ and HR_0 the horizontal asymptotes in ventilation and heart rate, and $\Delta\dot{V}_{40}$ and ΔHR_{40} the incremental ventilation and heart rate at P_{ETO_2} 40 mmHg, obtained from the hyperbola equation.

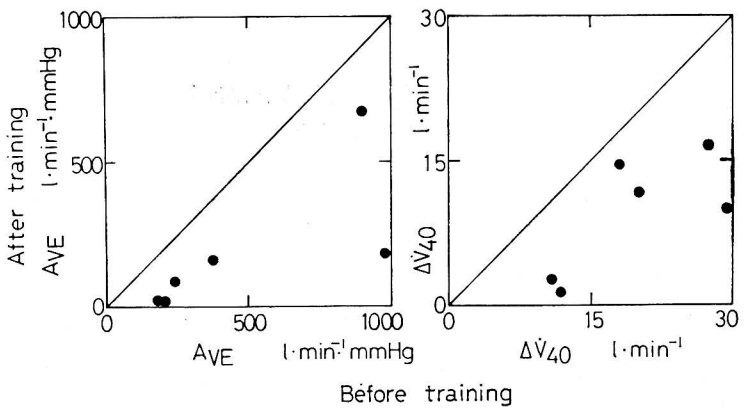

Fig. 4. Comparison of hypoxic ventilatory sensitivity in terms of hyperbola slope (A_{VE}) and of magnitude at P_{ETO_2} 40 mm Hg ($\Delta\dot{V}_{40}$) before and after training.

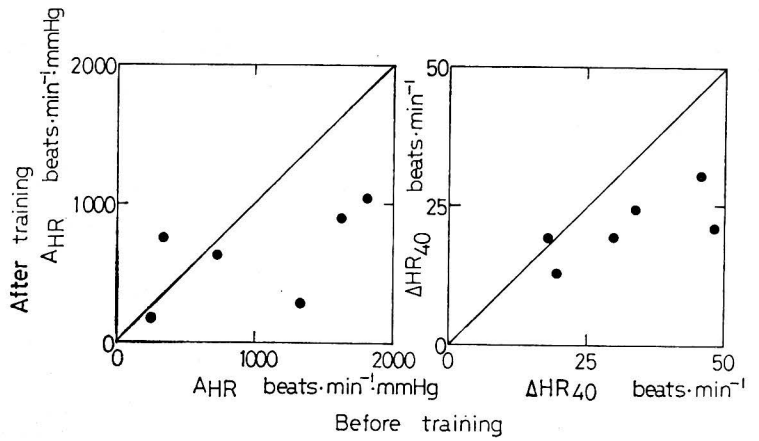

Fig. 5. Comparison of hypoxic heart rate sensitivity in terms of hyperbola slope (A_{HR}) and of magnitude at P_{ETO2} 40 mm Hg (ΔHR_{40}) before and after training.

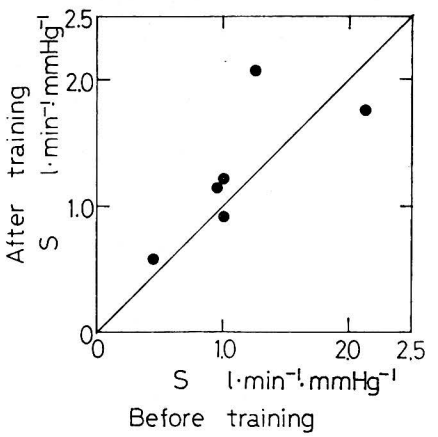

Fig. 6. Comparison of CO_2-ventilation response slope, S, before and after training.

ing was smaller than that of 2 years previously.

When both responses were evaluated in terms of $\Delta\dot{V}_{40}$ and ΔHR_{40}, ventilatory response in all subjects after the 2 year regimen was still smaller than before, as was the heart rate response with the exception of one participant.

Hypercapnic response

The slopes of the ventilatory response to hypercapnia (S) were 1.14 ±

0.55 $l \cdot min^{-1} \cdot mm\ Hg^{-1}$ before and 1.30 ± 0.54 $l \cdot min^{-1} \cdot mm\ Hg^{-1}$ after the training period. S failed to show any significant difference after the 2 years (Fig. 6).

Discussion

Follow-up studies observing the effect of physical training on CO_2-ventilatory responsiveness are controversial. Saunders et al. (1976) first noted in swimmers that no significant relationship existed between the slope of CO_2-ventilation response curve and training period. The same conclusion was obtained by Hughson (1980) who conducted 14 weeks of aquatic studies with rats. On the other hand, Blum et al. (1979) found a significant depression of the CO_2-response slope on 5 normal humans after physical conditioning.

Recently, Kelley et al. (1984) reported even enhanced CO_2 chemosensitivity in varsity student rowers after 7 months of training. They supposed that the reason for this unusual result was that the subjects were novices at the outset and the period of training was relatively short.

In the present experiment, we found no difference in CO_2 response after two years of endurance training. This result basically agrees with the observation by Mahler et al. (1982) in 20 marathon runners. Both groups of investigators compared their athletes' CO_2-ventilation slopes with those of non-athletes, and found no significant difference.

We assume that extremely low CO_2-response in some champion athletes (Rebuck and Read, 1971; Leitch et al., 1975; Saunders et al., 1976) may have a cause of familial or genetic origin, and that endurance training does not affect CO_2 chemosensitivity. The increased CO_2 response in rowers reported by Kelley et al. (1984) can not easily be reconciled with out data. The only obvious differences between the two studies were that Kelley's subjects were raw beginners at their sport and their training period was only 7 months, whereas in our study the runners were already highly experienced from their previous high school training and the experimental duration was 2 years.

We found blunting in hypoxic ventilatory response after two years of endurance training. To our knowledge no comparative follow-up studies have yet been reported. From the findings obtained from this experiment, we assume that blunted hypoxic response formerly reported in the athletes may be attributed not only to possible familial factors as mentioned above, but also to this particular form of physical exertion, that is, endurance training.

Blunted hypoxic response but not CO_2 response resembles the characteristic features of ventilatory chemosensitivities seen in high altitude natives (Millege and Lahiri, 1967). Such highlanders are known for their high physical performance with low ventilatory activities. It is thought that their capability of O_2 utilization in the peripheral tissues has improved because of lifelong exposure to hypoxia (Lahiri et al., 1969).

Endurance training used in the present study calls for tremendously extended physical activities, probably accompanied by continued hypoxic stress to the tissue metabolism. In this respect, short-lasting sprint training, in contrast, may not result in hypoxic blunting. In fact, we even observed enhanced hypoxic response in heavyweight judo athletes (Ohyabu et al., 1982 and 1984; Honda et al., 1983). Blunted response to hypoxia was not accompanied by depressed CO_2 response in the present study. Although both hypoxic and hypercapnic ventilatory activities are demonstrated via the same respiratory centers and muscles, their chemoreceptors and afferent pathways are largely different. Therefore, a dissociation between hypoxic and hypercapnic responsiveness is very conceivable. Scoggin et al. (1978) also reported the same characteristic features in 5 long distance runners.

The heart rate response to hypoxia in 6 subjects after endurance training was also depressed in all but one. It is well known that the effect of vagally mediated inflation reflex increases the heart rate (Coleridge and Coleridge, 1979). Therefore, the decreased heart rate response seen in our subjects, whose hypoxic ventilatory activity was blunted, might be expected. Also, a number of neural cells and their networks in both the respiratory and

cardiovascular centers converge and are interrelated with each other in brain-stem reticular activating systems (Koepchen 1983). The high synergistic nature of the different system functions in the living organism might have made it possible that both respiratory and circulatory functions adapted to the same direction at a similar magnitude.

In summary we conclude that acquired attenuation in hypoxic ventilatory and heart rate responses by endurance training may be responsible for facilitating the exercise performance in long distance runners.

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