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Exercise and Ventilatory Chemosensitivities

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Althouh physical training does not affect CO_2 chemosensitivity at rest, different kinds of physical training affect hypoxic ventilatory chemosensitivity at rest in different ways.

On the other hand, a number of studies indicated that the mechanism of exercise hyperpnea is related to hypoxic chemosensitivity.

Key words: Ventilatory chemosensitivity, Hypercapnia, Hypoxia, Athletes, Exercise hyperpnea

It is well known for years that blood gas is well maintained at its homeostatic level at rest as well as a number of different exercises with varying intensities. To accomplish this end, powerful negative feedback system to control alveolar-arterial O_2 and CO_2 pressures has been assumed to play an important role in athletes. Followings are a brief review summarizing the data accumulated in our laboratory and in the literatures.

Ventilatory chemosensitivities at rest in athletes

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 activities (Godfrey et al. 1971, Rebuck and Read 1971, Leitch et al. 1975, Miyamura et al. 1976b, Saunders et al. 1976, Scoggin et al. 1978, Martin et al. 1978, Kelley et al. 1984). The reported results, however, are conflicting, some agree with the initial findings, but others could not confirm significant changes in ventilatory chemosensitivities. The disagreement from these results may be accounted for by 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 influences (Rebuck and Read 1971, Leitch et al. 1975, Scoggin et al. 1978).

In our study (1989), 54 male track and field athletes and 18 male non-athletes were examined by CO_2 rebreathing test. We found that all the athletes did not show any significant difference in hypercapnic ventilatory sensitivity in comparison with that of non-athletes (Fig.1).

On the other hand, we (1989) found blunting in hypoxic ventilatory responses in long distance runners and sprinters (Fig.2). From the findings obtained from this experiment, we assumed that

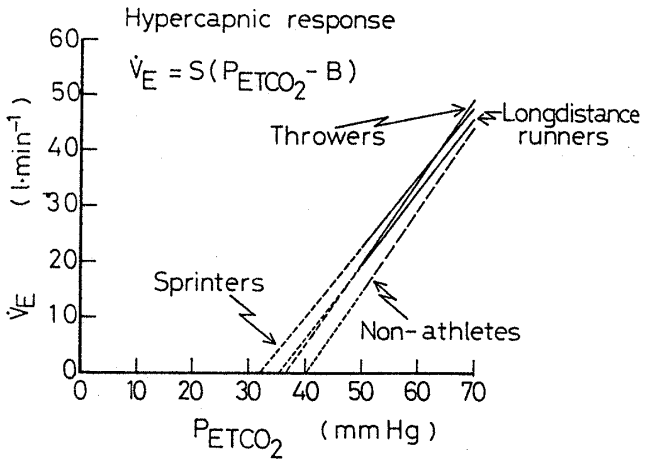

Fig. 1. Averaged ventilatory response curve during hypercapnia in different kinds of athletes and non-athletes (Ohyabu et al. 1989).

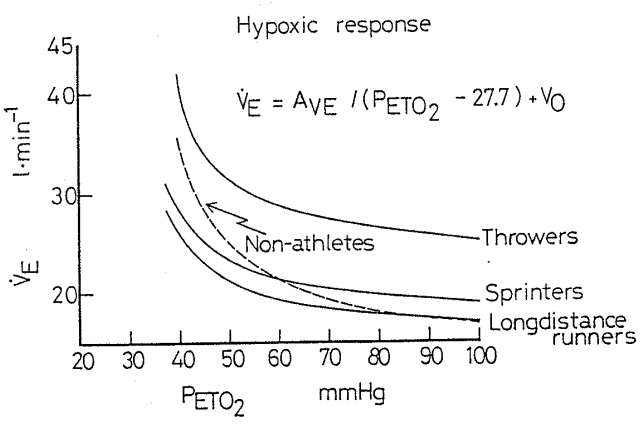

Fig. 2. Averaged ventilatory response curve during hypoxia in athletes and non-athletes (Ohyabu et al. 1989).

blunted hypoxic response formerly reported in the athletes may be attributed not only to possible familial factors as mentioned above, but also to these particular, aerobic or anaerobic power trainings.

In our previous studies (1982, 1984), we observed the increased hypoxic ventilatory sensitivity in heavy weight judo athletes and discussed the possibility of the underlying mechanisms as increased metabolism, muscle hypertrophy and obesity (Ohyabu et al. 1982, Nishibayashi et al. 1987).

From our previous findings, depressing and augmenting factors for hypoxic ventilatory response were assumed to exist. Among these depressed hypoxic response may have derived from the following influences:

1) aerobic power training (Byrne-Quinn et al. 1971, Scoggin et al. 1978, Ohyabu et al. 1984),

2) anaerobic power training (Martin et al. 1978),

3) lifelong sojourn in high altitude (Milledge and Lahiri 1967).

On the other hand, enhancing factors for hypoxic ventilatory response were proposed as follows:

1) obesity (Nishibayashi et al. 1987),

2) increasing body weight (Ohyabu et al. 1982, 1984),

3) short term sojourn in high altitude (Hayashi et al. 1982),

4) increased metabolism or muscle hypertrophy (Zwillich et al. 1975, Stockley and Bishop 1977, Honda et al. 1983, Ohyabu et al. 1984).

The effects of physical training on ventilatory chemosensitivities might have been derived in the integrated results of these complex influences.

Ventilatory response to hypercapnia during moderate exercise

The results obtained by Asmussen and Nielsen (1956), Deours (1964) and Duffin et al. (1980) have generally been accepted that the slope of the CO_2 -ventilation response curve is not significantly different during rest and exercise. However, contradictory data have been reported by different authors, e. g. increased responsiveness by Cunningham et al. (1963), Weil et al. (1972) and Miyamura et al. (1976a) whereas decreased responsiveness by Clark and Godfrey (1969), and Miyamura et al. (1976b) during

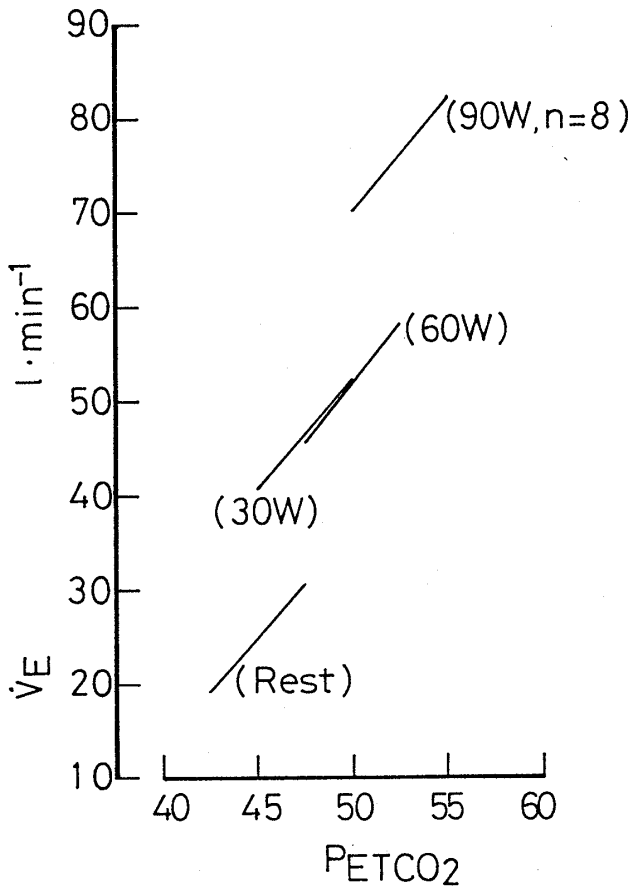

Fig. 3. The mean ventilatory response curves at rest and during exercise (30, 60, 90W) (Ohyabu et al. 1989).

exercise as compared with rest. Accordingly, a consensus of opinion seems still not to have been established.

We (1985) have also tried to explore this problem by applying external dead space. The CO_2-ventilation response curves thus obtained were generally shifted upwards in parallel from rest to exercise as shown in Fig.3. Similar results have also been reported by Asmussen and Nielsen (1975), Dejours (1964), Cunningham (1974), Martin et al. (1978) and Duffin et al. (1980).

Ventilatory response to hypoxia during moderate exercise

A number of studies indicated that the mechanism of exercise hyperpnea is related to hypoxic chemosensitivity. Asmussen and Nielsen (1956) has found that exercise hyperpnea decreased by O_2 inhalation. Honda et al. (1979) showed the decreased exercise hyperpnea in patients with bilateral carotid chemoreceptor resections. Furthermore, Weil et al. (1972) and Martin et al. (1978) demonstrated an augmentations of ventilatory sensitivity to hypoxia during moderate exercise in normal humans. These results suggested that contribution of the periopheral chemoreceptors is greater during exercise than at rest.

We (1988) also confirmed that ventilatory response to hypoxia was enhanced during exercise (Table 1).

A number of investigators have proposed possible mechanisms to explain involvement of the peripheral chemoreceptors in exercise hyperpnea. Yamamoto (1960) pointed out that increased CO_2 production by exercise will make greater arterial P_{CO2} oscillation in synchronization with the respiratory cycle, and that this would stimulate ventilation even with the mean arterial P_{CO2} maintaining unchanged. In a recent review, Linton et al. (1984, 1985) and Band et al. (1985) reported that arterial plasma potassium rose rapidly at the start of exercise, and that this may have stimulated ventilation via activation of the peripheral chemoreceptors.

Table 1. Ventilatory response to hypoxia during moderate exercise in normal healthy subjects (Ohyabu et al. 1988)

	Rest	Exercise
$A_{\dot{V}E}$ ($l \cdot min^{-1} \cdot mmHg$)	306.5±229.1	1,082.9±928.3*
$\dot{V}_O$ ($l \cdot min^{-1}$)	4.7±2.9	16.2±9.0**
$\Delta\dot{V}_{E50}$ ($l \cdot min^{-1}$)	8.9±5.6	38.4±22.1*

Values are mean ± SD. n=12 (one male and 11 female)
*and**: The differences comparad to the values at rest are significant at the 5 and 0.1 % levels, respectively. Paired t test was conducted.
$A_{\dot{V}E}$: The slope constant of the hyperbola expressing the degree of hypoxic sensitivity.
$\dot{V}_O$: Horizontal asymptote in ventilation for infinite end-tidal P_{CO2}.
$\Delta\dot{V}_{E50}$: The absolute magnitude of hypoxic response in ventilation at P_{ETO2} 50 mmHg.

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