Contribution of Peripheral Chemoreceptor Drive in Exercise Hyperpnea in Humans

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Abstract. The peripheral chemoreceptors play a dominant role in the respiratory compensation of lactic acidosis during heavy exercise of humans. Our object was to determine the contribution of peripheral chemoreceptors to exercise hyperpnea during mild to moderate and heavy exercise above the anaerobic threshold. We used a hyperoxic suppression test in six normal male subjects. Inspired gas was abruptly changed without the subject's knowledge from air to pure oxygen for 5 to 6 breaths. The maximal ventilatory depression after O2 breathing was 5.5 ± 1.7 L/min (BTTS) at mild exercise, and the depression increased with increasing exercise intensity up to 12.8 ± 4.1 L/min (BTTPS). The relative contribution of the peripheral chemoreceptors to ventilation in terms of percentage of the maximal ventilatory depression was maintained, being 20% throughout the entire work ranges studied. The contribution of the peripheral chemoreceptors to total ventilation is hardly altered by lactic acidosis caused by heavy exercise above the anaerobic threshold according to our data. These results suggested that the peripheral chemoreceptors may not be solely responsible for excessive hyperventilation, or residual activities of peripheral chemoreceptors still exist after O2 breathing especially during heavy exercise above the anaerobic threshold.


Keywords: control of breathing, exercise hyperpnea, anaerobic threshold, O2 test, peripheral chemoreceptors

Introduction

The peripheral chemoreceptors were discovered more than fifty years ago, but its quantitative role in ventilatory regulation is still not resolved. Dejours et al. (1958) introduced the “single O2 breath test” in humans with inspiration of pure O2 and evaluated the contribution of the peripheral chemoreceptors to be approximately 10% at rest. The ventilation is depressed 8-10 sec after the O2 breath. This is consistent with blood transfers from the lungs to the carotid body: the so-called lung-to-carotid body circulation time. Dejours et al. (1958) also found a greater ventilatory response and a shorter time delay when the O2 breath test was used during exercise.

Similar studies confirmed that the peripheral chemoreceptors are more important during exercise than at rest (Weil et al., 1972; Wasserman 1976; Wasserman et al., 1979). Wasserman et al. (1976, 1979) observed that ventilation diminished greatly and persistently in response to continuous O2 breathing during exercise when exceeding the anaerobic threshold (AT) where a lactic acidosis develops. This result was ascribed to the hyperoxic blockade of peripheral chemoreceptor activity. Without the O2 chemodenervation the ventilation is augmented by the lactic acidosis imposing an extra stimulus to the peripheral chemoreceptors. For years it has thus been generally accepted that marked exercise hyperpnea during heavy exercise is mainly mediated by the peripheral chemoreceptors in humans. Consequently, it might be expected that the chemoreceptors contribute relatively more to hyperventilation at work rates above than below AT.

On the other hand, some investigators have disagreed with the concept that peripheral chemoreceptors are the only and unique organ for mediating excessive hyperventilation accompanying lactic acid accumulation. Stockley (1978) observed no significant differences in ventilatory response to short-lasting O2 breathing at rest and during several different intensities of exercise. However, the AT was not measured in his subjects. Jeyaranjan et al. (1987) assessed the contribution of the peripheral chemoreceptors during heavy exercise above AT, using abrupt O2 administration to suppress the
receptor activity. Only a 15% peripheral chemoreceptors contribution to ventilation was found, which was even less than the magnitude previously reported below the AT level (Weil et al., 1972; Wasserman, 1976). Jeyarajan et al. (1987) did not carry out the same test during mild to moderate exercise range below the AT.

The object of the present study was to clarify these controversies on the quantitative role of peripheral chemoreceptors in exercise hyperpnea. We systematically examined ventilatory response to O₂ breathing with different exercise intensities, ranging from below to above the AT. We also measured the lung-to-ear circulation time, which was previously shown to be equivalent to the lung-to-carotid body circulation time (Jain et al., 1972). This was done in order to evaluate the corresponding time course between ventilatory depression after O₂ breathing, and the hyperoxemia developing at the level of the peripheral chemoreceptors.

Methods

Six male subjects, ranging in age from 21 to 34 yrs, were studied. All subjects were informed of the risks and stresses associated with this study and gave their informed consent to participation. Each subject was asked to avoid strenuous physical activity for at least 24 hrs before the study and to refrain from eating and smoking for 3 hrs.

The subjects were seated on a cycle ergometer and breathed room air through a face mask (Ramp exercise test), or a mouth piece (Oxygen breath test), which was connected to a hot-wire flow meter, and to rapid responding O₂ (zirconia) and CO₂ (infrared) analyzers. These devices were incorporated into a data acquisition and processing system (RM 280, MINATO). The respiratory flow, O₂ and CO₂ signals were real time treated in order to obtain tidal volume (Vₚ), respiratory frequency (fₛ), minute ventilation (Vₑ), end-tidal O₂ (ETO₂) and CO₂ (ETCO₂) concentration, inspiratory O₂ and CO₂ concentrations, O₂ uptake (V̇O₂), CO₂ output (V̇CO₂) and the ventilatory equivalents for O₂ and CO₂ (V̇E/V̇O₂, V̇E/V̇CO₂) on a breath-by-breath basis. All data were stored on a floppy disk and those necessary for control were also displayed on a color monitor throughout the experiment. The flow signal was also fed into an analog computer in which Vₚ, Tᵢ, Tₑ and Vₑ were calculated and recorded on a 6-channel pen recorder (RECTI-HORIZ-8K, NEC-Sanei). These systems were carefully calibrated before each study.

Ramp exercise test

This test was performed in order to determine the AT and the peak oxygen uptake. Subjects were seated on a cycle ergometer (Road Corival 400 with a ramp slope controller) and breathed room air through the face mask.

After 4 min of constant 20 Watt (W) loaded pedaling (ca. 60 rpm), each subject performed a ramp exercise test up to exhaustion, at an incremental work rate (1 W/sec). AN was determined either by the V-slope method (Beaver et al., 1986) or by the starting point of consistent increase in the V̇E/V̇O₂ without a concomitant augmentation in the V̇E/V̇CO₂ (Davis et al., 1979).

Oxygen breath test

This test was performed on a separate occasion. The subjects were seated on the cycle ergometer and breathed through a tube system consisting of mouthpiece, flow meter probe, respiratory valve, and a three-way stopcock. The three-way stopcock was kept open to room air and was connected to a 20 L Douglas bag containing pure oxygen. The dead space volume of this system was about 150 ml. The inspiratory line, including respiratory valve, was properly heated with a tape heater in order to prevent water deposition, as well as to eliminate temperature difference between air and test gas (O₂). Inspiratory air was humidified. As the sensitivity of the flow sensor to pure O₂ was higher than that to air by approximately 6%, the respiratory volume was corrected by this ratio during O₂ inhalation.

Three different work rates were examined below and above AT, i.e., work intensities corresponding approximately to 50, 80, and 130% of V̇O₂ for the AT level, respectively (Fig. 1). Each subject performed constant-load exercise at these three different work rates in separate runs. Following a 4-min warming up with 20 W, the work load was increased up to the precalculated level and maintained during the entire period of one experimental run. The subjects exercised for at least 4 min with air breathing, which was long enough to attain a steady state below AT. The inspired air was then abruptly replaced by pure oxygen for 5-6 breaths (about

![Fig. 1 Experimental protocol of oxygen breath test during constant load exercise below (50%, 80%) and above (130%) the anaerobic threshold. A given exercise intensity was performed twice in each subject.](image-url)
15 sec) and then switched back to room air. Care was taken so that the subjects did not become aware of the switch to O₂. After confirming the restoration of the mixed expired gas composition, which took about 3 min following the first O₂ test, the same procedure were repeated twice.

Breath-by-breath data was continuously recorded throughout the experimental run. Minute ventilation, PetCO₂ and Peto₂ for ten breaths before and fifteen breaths after the oxygen breathing were used for the subsequent analysis. The experiment run with a given exercise intensity was performed twice in each of the six subjects, with at least 2 hrs of rest between successive runs.

**Lung-to-ear circulation time**

Arterial oxygen saturation (Sao₂) was measured by an ear oximeter (Ohmeda Biox III), and was continuously recorded. At the end of each oxygen test, inspiratory gas was switched to N₂ instead of O₂ for three breaths. The time delay from the commencement of N₂ breathing to the point where the desaturation started, as well as that to the point of its maximal fall, were measured from the chart recording and defined as Lung-to-ear-start (LE(S)), and Lung-to-ear-maximum (LE(M)), respectively (Fig. 2).

**Data sampling and analysis**

We compared the time delays for attaining the maximal depression in Sao₂ by N₂ breathing (LE(M)) and those for nadir ventilation by the O₂ test, defined as the Lung-ventilation-minimum (LV(M)) (Fig. 2). The two variables (LE(M)) and (LV(M)) were very similar except in one subject. As this subject had exceptionally long LV(M) compared with LE(M), the ventilatory response was determined at the breathing cycle closest to the LE(M).

The magnitude of peripheral chemoreceptor activity was calculated in absolute terms as the difference between the control ventilation and the minimum ventilation observed after O₂ inhalation. The relative value of the response in percentage of control ventilation was also calculated. This was assumed to be the relative contribution of the peripheral chemoreceptors to exercise hyperpnea. Statistical analysis was performed by Student t-test. Differences were considered significant, when the p-value was less than 0.05.

**Results**

The physical characteristics of the subjects and the results of the ramp exercise tests are shown in Table 1.

![Diagram](image)

**Fig. 2** Changes in ventilatory response and Sao₂ during O₂ and N₂ breathing in one subject. Broken horizontal line indicates control Vₑ. The time delay from the commencement of O₂ breathing to nadir ventilation and from the initiation of N₂ breathing to the beginning and maximal depression of Sao₂ were defined as LV(M), LE(S) and LE(M), respectively. Note that LV(M) is approximately the same as LE(M).

### Table 1 Physical characteristics of subjects and results of incremental exercise testings

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>V₀₂ at anaerobic threshold (ml/min/kg)</th>
<th>V₀₂ at peak exercise (ml/min/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MT</td>
<td>23</td>
<td>73</td>
<td>175</td>
<td>3.6</td>
<td>47.8</td>
</tr>
<tr>
<td>TK</td>
<td>34</td>
<td>64</td>
<td>173</td>
<td>3.6</td>
<td>41.8</td>
</tr>
<tr>
<td>SH</td>
<td>23</td>
<td>67</td>
<td>174</td>
<td>3.6</td>
<td>51.0</td>
</tr>
<tr>
<td>TH</td>
<td>21</td>
<td>64</td>
<td>176</td>
<td>3.6</td>
<td>56.8</td>
</tr>
<tr>
<td>YM</td>
<td>21</td>
<td>61</td>
<td>167</td>
<td>3.6</td>
<td>48.2</td>
</tr>
<tr>
<td>TA</td>
<td>22</td>
<td>60</td>
<td>169</td>
<td>3.6</td>
<td>52.8</td>
</tr>
<tr>
<td>Mean</td>
<td>24</td>
<td>65</td>
<td>172</td>
<td>3.6</td>
<td>49.7</td>
</tr>
<tr>
<td>SD</td>
<td>5.0</td>
<td>4.7</td>
<td>3.6</td>
<td>3.3</td>
<td>5.1</td>
</tr>
</tbody>
</table>
Table 2  Effects of O2 and N2 breathing on ventilatory activities during exercise

<table>
<thead>
<tr>
<th>Condition of Exercise</th>
<th>$\dot{V}O_2$ (ml/min/kg)</th>
<th>Time to the lowest $\dot{V}E$ after O2 test (L/min)</th>
<th>Control $\dot{V}E$ (L/min)</th>
<th>Maximal $\dot{V}E$ decrement after O2 test (L/min)</th>
<th>Maximal $\dot{V}E$ decrement in % after O2 test (%)</th>
<th>Time delay for the start of Sao2 drop (LE(S)) (sec)</th>
<th>Time delay for maximal Sao2 drop (LE(M)) (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>50% AT</td>
<td>13.5</td>
<td>16.3</td>
<td>27.5</td>
<td>5.5</td>
<td>19.9</td>
<td>10.0</td>
<td>16.7</td>
</tr>
<tr>
<td>± 1.1</td>
<td>± 2.8</td>
<td>± 4.1</td>
<td>± 1.7</td>
<td>± 5.5</td>
<td>± 1.2</td>
<td>± 1.2</td>
<td>± 1.3</td>
</tr>
<tr>
<td>80% AT</td>
<td>18.7</td>
<td>14.1*</td>
<td>37.0**</td>
<td>7.6**</td>
<td>20.5</td>
<td>8.5**</td>
<td>14.8**</td>
</tr>
<tr>
<td>± 1.3</td>
<td>± 2.6</td>
<td>± 4.7</td>
<td>± 1.8</td>
<td>± 5.1</td>
<td>± 1.2</td>
<td>± 1.2</td>
<td>± 1.5</td>
</tr>
<tr>
<td>4 min with 29.2</td>
<td>10.7**</td>
<td>56.8**</td>
<td>10.7*</td>
<td>19.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>130% AT</td>
<td>± 2.3</td>
<td>± 1.3</td>
<td>± 4.2</td>
<td>± 9.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 min with 30.0</td>
<td>11.2</td>
<td>59.9*</td>
<td>12.8</td>
<td>21.3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>130% AT</td>
<td>± 2.2</td>
<td>± 1.6</td>
<td>± 4.1</td>
<td>± 5.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 min with 30.6</td>
<td>9.9*</td>
<td>62.0*</td>
<td>11.4</td>
<td>19.0</td>
<td>5.7**</td>
<td>11.8**</td>
<td></td>
</tr>
<tr>
<td>130% AT</td>
<td>± 2.2</td>
<td>± 1.5</td>
<td>± 3.0</td>
<td>± 5.9</td>
<td>± 1.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD. (n=6). *p<0.05, **p<0.01; significantly different from the value of the preceding work rate.

Both the mean $\dot{V}O_2$ at AT (23.8 ± 3.3 ml/min/kg), and the ratio of the AT to the peak $\dot{V}O_2$ in % (47.7 ± 2.9%) were within the normal ranges.

Oxygen breath test

Figure 2 illustrates the effects of O2 and N2 breathing during exercise (80% AT) examined in one subject. When O2 inhalation started, $F_i$O2 abruptly increased well above 30%, and $Vr$ gradually declined. $Vr$ also decreased and revealed a nadir; then returned to the initial value. The control value of $\dot{V}E$ was obtained by averaging the last ten breaths before the O2 inhalation and was depicted as a broken horizontal line. During N2 breathing, $F_i$O2 immediately went down and Sao2 began to decrease with several seconds delay (i.e., the lung-to-ear circulation time), then reached a nadir and again returned to the control level.

Timing of lung-to-ear circulation and of maximal ventilatory depression

The lung-to-ear circulation time, estimated by the interval from the commencement of N2 breathing to the point where the desaturation started (LE(S)), became significantly shorter, from 10.0 to 5.7 seconds (sec), with increasing exercise intensities (Fig. 2 and Table 2). The intervals for a maximum fall in Sao2 after N2 inhalation (LE(M)) and the intervals from the start of O2 inhalation to the point of the maximum fall in ventilation (LV(M)) also showed significant shortening with increasing exercise intensities (Fig. 2 and Table 2). Both (LE(M)) and (LV(M)) revealed almost identical values, when compared at the same work rate (16.3 ± 2.8 vs. 16.7 ± 1.3 in 50%AT, 14.1 ± 2.6 vs. 14.8 ± 1.5 in 80%AT and 9.9 ± 1.5 vs. 11.8 ± 0.9 in 130%AT, respectively). Subject to subject variation of the timing and breath number for reaching the maximal fall of ventilation were noted.

Contribution of peripheral chemoreceptors

The maximal depression of ventilation, which appeared at the 4th to 8th breath after O2 inhalation, was determined in each run and presented in Table 2 and Fig. 3. The absolute magnitude of decrement of ventilation was significantly greater with increasing exercise intensity, from 5.5 L/min to 12.8 L/min. On the other hand, the magnitude relative to the control were almost the same (ca. 20%) among the different exercise intensities.

Discussion

Contribution of peripheral chemoreceptors

We found that the amount of ventilatory depression by the O2 test became significantly greater with increasing exercise intensities. On the other hand, its relative magnitude remained at approximately 20% irrespective of the different work rates. Contribution of the peripheral chemoreceptors during exercise, as estimated by O2 test under different exercise intensities, varied among investigators from 10% to 25% (Dejours et al., 1958; Wasserman, 1976; Stockley, 1978; Jeyaraman et al., 1987). It seems essential, therefore, to perform a systematic examination with exercise ranging from moderate to severe work rates to accurately determine this contribution. In the present study, we used normal young males as subjects, and determined AT (ventilatory anaerobic threshold) as well as intensities of exercise both below and above AT. We were particularly careful that the manipulation of the O2 test was not noticed by the subjects. We found that the level of 20% as the relative contribution of the peripheral chemoreceptors was almost the same as that previously reported by Wasserman (1976) for mild to moderate exercise below AT, but was greater than those reported by Stockley
hyperventilation above AT, and the other is that residual activities of peripheral chemoreceptors still exist after O₂ breathing especially during heavy exercise above AT.

**Physiological denervation of peripheral chemoreceptor by O₂ test**

In this study, we used a similar technique as Stockley (1978). The subjects breathed pure oxygen for six breaths and we confirmed that PETO₂ was elevated up to 500 mmHg. This is a level high enough to suppress peripheral chemoreceptors activities in cats under various Paco₂ and pH conditions (Fitzgerald and Parks, 1971). Pokorski and Lahiri (1983) also reported that the stimulation of arterial chemoreceptors in cats by acid was virtually abolished by hyperoxia. In intact man, the stimulatory effect of different conditions of Paco₂ to ventilation was probably abolished by O₂ administration at rest (Miller et al. 1974) and during moderate-intensity exercise (Ward and Bellville, 1988). In case of heavy exercise, whether the peripheral chemoreceptors are completely silenced by high oxygen are controversial. The results of Cunningham and MacFarlane (1985) would suggest that peripheral chemosensitivity is abolished by high oxygen. But Rausch et al. (1991) cannot rule out small residual component of the carotid bodies by O₂ breathing in ventilatory compensation to the metabolic acidosis of exercise.

The possible involvement of plasma potassium (K⁺) in exercise hyperpnea has been also proposed by a number of investigators. Potassium stimulates the arterial chemoreceptors (Linton and Band, 1985) and increases ventilation in the anaesthetized cat by direct stimulation of these chemoreceptors (Band et al., 1985). Whether or not the stimulation of K⁺ is abolished by hyperoxia is still in dispute. Several reports (Burger et al., 1988; Paterson and Nye, 1991) showed that the effect of potassium was essentially abolished by an abrupt switch to 100% oxygen in the cat. On the other hand, Sneyd et al. (1988) showed in cats that the stimulation by potassium was independent of the background inspired P0₂. However, there were some differences in methodology between their experiments and the present study. We used a method similar to that of Burger et al. (1988), and assume that physiological denervation of the peripheral chemoreceptor was well achieved by our O₂ test below the AT level. However, we cannot completely rule out the small residual activities of peripheral chemoreceptors when the O₂ test was applied during exercise above the AT level.

**Timing of ventilatory depression after O₂ breathing and possible involvement of central chemoreceptors mechanism**

Maximal ventilatory depression in response to the O₂ test appeared to occur with a time delay of 16.3 and 14.1
sec in exercise at 50 and 80% AT, respectively, and this
time delay was progressively shortened in heavy exercise,
9.9 - 11.2 sec at 130% AT. In parallel with the time
profile of ventilatory depression, initiation of arterial
desaturation measured at the ear lobe was 10.0, 8.5 and
5.7 sec after \( N_2 \) breathing in exercise at 50, 80 and 130%
AT, respectively. Jain et al. (1972) confirmed that
pulmonary artery-ear lobe circulation time was 4.1 to 6.6
sec in resting humans, and that this duration was nearly
equal to the injection-respiratory response times for
phenylbiguanide and sodium cyanide. The lung-to-ear
circulation times in the present study were slightly longer
than that reported by Jain, possibly due to differences in
methods. They injected Evans Blue dye directly as a
bolus into the pulmonary artery, whereas we measured the
lung-to-ear circulation time from the commencement of
\( N_2 \) breathing.

MacDonald et al. (1990) reported that the prediction of
peripheral chemoreceptor activity with the transient
\( O_2 \)-switching technique of Dejours might underestimate
its contribution during heavy exercise above the AT. This
is due to some acidic stimulation of the central
chemoreceptors to the Haldane effect, and to local
acidosis consequent to the hypoxia-induced cerebral
hypoperfusion. At our exercise intensities, any humoral
information originating from the lung will take
approximately 10-15 sec to reach the central
chemosensitive regions (Whipp, 1980). In our study,
\( LV(M) \) and \( LE(M) \) showed almost identical below the AT.
During heavy exercise above the AT, \( LV(M) \) showed
significantly shorter than \( LE(M) \) (9.9 vs 11.8 sec:
p<0.01), which indicates that some stimuli for ventilation
might be involved within the time required to attain
minimum ventilation with complete denervation of
peripheral chemoreceptor by high oxygen.

Figure 4 illustrates the \( \dot{V}_E \) and \( \dot{V}_O_2 \) relationship
obtained in this study in order to understand
schematically the changes of peripheral chemoreceptor
contribution to exercise hyperpnea. The thick line
indicates the actual magnitude of exercise ventilation.
Clearly, the curve bends upwards at the point of AT and
\( \dot{V}_E \) augmentation is intensified above the AT region. The
broken line below AT level represents the magnitude of
exercise ventilation after physiological denervation by \( O_2 \)
breathing. Because the values are approximately 20%
less than that of the thick line at both the 50 and 80% AT
levels, further extension of this line beyond the AT point
may be considered to express the predicted magnitude of
exercise hyperpnea after physiological denervation of
the peripheral chemoreceptors. Thus the difference between
the thick and broken lines beyond the AT point may be
ascribed to peripheral chemoreceptor activity. However,
the actual amount of ventilatory decline by the \( O_2 \) test
shown by perpendicular solid lines is substantially less
than the predicted value.

Other possible mechanisms than peripheral
chemoreceptors for inducing hyperventilation during
exercise above the anaerobic threshold

Although Wasserman et al. (1973) proposed that
metabolic acidosis due to lactic acid accumulation
stimulates the peripheral chemoreceptors and elicits
excessive ventilation during heavy exercise, other
investigators claimed that the anaerobic threshold
determined from the blood lactate accumulation is not
necessarily the same as that defined by the change in
ventilation (Green et al., 1983; Simon et al., 1983). Pan
et al. (1986) reported that hyperventilation during high-
intensity exercise in ponies was not dependent on arterial
acidosis nor on the carotid chemoreceptors, and
Jeyarajan et al. (1989) reported in humans that lactic

![Fig. 4](image_url)

**Fig. 4** Comparison of the amount of predicted ventilatory
contribution of the peripheral chemoreceptor activity with
that of experimentally determined ventilatory depression
by \( O_2 \) breathing, illustrated in the region above AT. In
reflecting excessive hyperventilation above AT, the solid
line connecting the magnitude of exercise hyperventilation
becomes steeper beyond the AT point. The broken line
obtained by connecting the nadir ventilation in the \( O_2 \) test
at 50 and 80% AT levels was assumed to predict the
contribution of peripheral chemoreceptor activity. The
predicted values were found to be substantially larger than
the amount of ventilatory depression experimentally obtained
by the \( O_2 \) test at 130% AT.
acidosis was not an essential determinant of the hyperventilatory response to heavy exercise in the majority of individuals. Moreover, the evidence of a normal ventilatory response of Patients with Mc Ardle's syndrome (Hagberg et al., 1982) who can not develop lactic acidosis during heavy exercise. This may lead us to consider other stimuli than the peripheral chemoreceptors for inducing hyperventilation during heavy exercise above the AT.

Yamamoto et al. (1992) reported that there was some correlation between changes in the activity of the autonomic nervous system and the appearance of AT during incremental exercise. They found that parasympathetic nervous system was markedly reduced below AT and that sympathetic nervous system showed threshold-like increase above AT. It was also reported that the threshold-like change of opioids such as β-endorphin during exercise has also been demonstrated (McMurray et al., 1987). Similar to the change of the autonomic nervous control system during exercise, the ventilatory threshold during ramp exercise may also appear with the augmented activity of sympathetic nervous system, like other threshold-like phenomena.

In this study, the relative contribution of the peripheral chemoreceptors to the total ventilation during heavy exercise above AT was the same as that below AT, which suggests that the relative contribution of the peripheral chemoreceptor drive to the total ventilation in humans is not altered in heavy exercise above AT. These results indicated possible implication that the peripheral chemoreceptors may not be solely responsible for excessive hyperventilation under such conditions and that excessive hyperventilation during heavy exercise and lactic acid accumulation occur only coincidentally. This is like many other metabolic and hormonal factors, which increase nonlinearly during ramp exercise. Further studies are necessary to clarify either residual activities of peripheral chemoreceptors still exist after O2 breathing or some amount of central chemoreceptor activity for enhancing ventilation might be involved during O2 breathing, especially during heavy exercise.

References


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