Respiratory Compensation Point during Incremental Exercise as Related to Hypoxic Ventilatory Chemosensitivity and Lactate Increase in Man

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Abstract: The pulmonary ventilation–O₂ uptake (VE–VO₂) relationship during incremental exercise has two inflection points: one at a lower VO₂, termed the ventilatory threshold (VT); and another at a higher VO₂, the respiratory compensation point (RCP). The individuality of RCP was studied in relation to those of the chemosensitivities of the central and peripheral chemoreceptors, which were assessed by resting estimates of hypercapnic ventilatory response (HCVR) and hypoxic ventilatory response (HVR), respectively, and the rate of lactic acid increase during exercise, which was estimated as a slope difference (Δslope) between a lower slope of VCₐO₂–VO₂ relationship (ΔVCₐO₂: CO₂ output) obtained at work rates below VT and a higher slope at work rates between VT and RCP. Twenty-two male and sixteen female subjects underwent a 1 min incremental exercise test until exhaustion, in which VT, RCP and Δslope were determined. All measures were normalized for body surface area. In the males, the individual difference in RCP was inversely correlated with those of HVR and Δslope (p<0.05), and in the females, similar tendencies persisted, while the correlation did not reach statistically significant levels (0.05<p<0.1). There was no significant correlation between RCP and HCVR in either sex. A multiple linear regression analysis showed that 40 to 50% of the variance of RCP was accounted for by those of HVR and Δslope, both of which were related linearly and additively to RCP, this relation being manifested in the males but not in the females without consideration of the menstrual cycle. These results suggest that the individuality of RCP depends partly on the chemosensitivity of the carotid bodies and the rate of lactic acid increase during incremental exercise. [Japanese Journal of Physiology, 50, 449–455, 2000]

Key words: heavy exercise, respiratory compensation point, exercise hyperpnea, carotid body, lactic acidosis.

During incremental exercise in normal humans, pulmonary ventilation (VE) linearly increases with increasing O₂ uptake (VO₂), but the increment of VE against VO₂ becomes steeper at two VO₂ points [1]. The first inflection point, occurring at a lower VO₂, is termed the ventilatory threshold (VT) [2], above which various kinds of ventilatory stimuli such as a fall in arterial pH due to lactic acidosis, hyperkalemia and augmentation of arterial PCO₂ oscillation are newly induced [3–6]. With further increases in the ventilatory stimuli as exercise becomes heavier, a steeper increase in VE against VO₂ occurs. The VO₂ point of the onset of this VE augmentation is termed the respiratory compensation point (RCP), above which the VE augmentation (hyperventilation) induces a fall in arterial PCO₂, with resulting constraint of further falls of arterial pH due to severer lactic acidosis [7]. Peripheral chemoreceptors have been implicated as the site at which the ventilatory stimuli act because of the absence of compensatory hyperventilation and subsequent fall in PCO₂ during heavy exercise in carotid body–resected patients [8, 9].
We hypothesized that, in individuals with greater carotid body chemosensitivities, compensatory hyperventilation would begin at lower work rates at which smaller changes in the concentration of ventilatory stimuli to the carotid bodies would be associated. The present study was undertaken to test this hypothesis. In addition, involvement of central chemosensitivity in the individual variance of RCP was also investigated. Peripheral chemosensitivity was assessed by hypoxic ventilatory responsiveness (HVR) and central chemosensitivity by hypercapnic ventilatory responsiveness (HCVR), both at rest. Since it has been reported that HVR [10–12] and HCVR [13] increase with increasing exercise intensity, and that the exercise estimates of HVR depend on the resting estimates of HVR [11, 12, 14], the individual differences in exercise estimates of chemosensitivities at varying work rates were assumed to be predicted from those in the resting estimates.

Cooper et al. [15] divided the CO2 output–O2 uptake ($V_{CO2}/V_{O2}$) relationship up to RCP during incremental exercise into two linear components. The intersection point of the two linear components per se has been described as $V$-slope AT (AT: anaerobic threshold, expressed in terms of $V_{O2}$ level) and a more reliable, non-invasive estimate of AT [7]. It has been argued that the lower linear component with a lower slope ($S_1$) in the $V_{CO2}/V_{O2}$ relationship reflects the aerobic metabolism (respiratory quotient) of the working muscles and the upper linear component with a higher slope ($S_2$), aerobic metabolism plus “excess CO2” derived from bicarbonate buffering of lactic acid taking place intramuscularly and extracellularly [16] if the body CO2 stores are stable [15, 17]. Therefore, it would be assumed that the difference between $S_2$ and $S_1$ reflects an “approximate” rate of lactic acid increase occurring intramuscularly and extracellularly, although it does not reflect the “total” amount of lactic acid increase in the body fluids, a small portion of which is buffered by the non-bicarbonate buffer system [18]. In the present study, the difference of $S_2$ and $S_1$ ($\Delta$slope) was also examined and used as an estimate of the rate of lactic acid increase at the work rates below RCP. The aim of this was to explore the relationship between the rate of lactic acid increase and RCP during incremental exercise (i.e., whether the greater the former is, the lower the latter is).

METHODS

Subjects. Twenty-two male students and sixteen female students volunteered for this study after giving written, informed consent. Their physical characteristics are shown in Table 1. Body surface area was calculated from the height and weight measurements using calculation formulas for Japanese men and women [19]. The subjects were all healthy non-smokers, of which 15 subjects were training as short and middle-distance runners, 4 subjects as long-distance runners in after-class activities, and 18 subjects were taking part in recreational sports. Because of the varying sports training status, physical fitness levels that were evaluated as maximum $O_2$ uptake differed greatly among the subjects (Table 1). No special consideration for menstrual cycle was given for the female subjects, to whom the phases of the menstrual cycle on the day of the study were not inquired.

Measurements. During the tests, the subjects breathed through a respiratory mask (dead space, 200 ml) to which a hot-wire flowmeter was fixed in order to measure respiratory flow. Respiratory gas was continuously sampled (200 ml/min) from a nostril and introduced into a CO2-O2 gas analyzer (MG-360, Nihonkohden Medical Co., Japan), with which the CO2 and O2 concentrations were analyzed through infrared absorption and zirconium oxide reaction, respectively. Signals from the flowmeter and gas analyzer were fed into a signal processor (RM-200, Respirimonitor, Nihonkohden Medical Co., Japan) and processed breath-by-breath to obtain pulmonary ventilation ($V_E$), $O_2$ uptake ($\dot{V}_{O2}$), CO2 output ($\dot{V}_{CO2}$), ventilatory equivalents for $O_2$ and CO2 ($V_{E/O2}$ and $V_{E/CO2}$, respectively) and end-tidal $P_{O2}$ and $P_{CO2}$. Heart rate (HR) was monitored through an electrocardiogram. Arterial oxygen saturation ($S_{O2}$) was measured using a finger oximeter (Pulsox 8, Canon Co., Japan).

Tests. Three tests were given to the subjects in the following order. Before the tests, the subjects underwent a training session in order to familiarize them with the equipment for the tests.

Transient hypoxic test for HVR assessment. This was based on the method of Shaw et al. [20]. A reser-voir bag containing pure N2 was connected to the respiratory mask. After $V_E$ and HR reached a steady state during air breathing, the inspired gas was switched to N2 and three to eight breaths of N2 were imposed on the subjects, followed by air breathing. Subsequent hypoxic transients with a different number of N2 breaths were resumed after end-tidal $P_{O2}$, $S_{O2}$ and HR returned to air-breathing levels. If $S_{O2}$ levels during N2 breathing approached 70%, the subject was switched back to room air. The highest $V_E$ (actually calculated as the mean value of the two highest consecutive $V_E$) and the lowest $S_{O2}$ achieved during the period of N2 breathing were obtained for each hypoxic transient. Then, using the data from all six hypoxic
transients, a linear regression of the highest \( \dot{V}E \) and the lowest \( \Delta S_{O_2} \) was analyzed for each subject by the least-squares method. The slope of the regression line, \( \Delta \dot{V}E/\Delta S_{O_2} \), was designated as hypoxic ventilatory responsiveness (HVR).

**CO\(_2\)** rebreathing test for HCVR assessment. This was based on the method of Read [21]. After \( \dot{V}E \) and HR reached a steady state during air breathing, the subjects rebreathed a 7% CO\(_2\)–93% O\(_2\) gas contained in a reservoir bag for 4 min, during which end-tidal \( P_{CO_2} \) and \( \dot{V}E \) were measured. A regression line of the end-tidal \( P_{CO_2} - \dot{V}E \) relationship was calculated and the slope (\( \Delta \dot{V}E/\Delta P_{CO_2} \)) was defined as hypercapnic ventilatory responsiveness (HCVR).

**Incremental exercise test.** After 2 min unloaded cycling on a bicycle ergometer, the subjects exercised at a 1 min incremental work load until exhaustion. The increment of the load was 25 W/min at the pedalling rate of 70 rpm for the male subjects and 15 W/min at 50 rpm for the female subjects. During the test, \( \dot{V}O_2 \), \( \dot{V}CO_2 \), \( \dot{V}E/\dot{V}O_2 \), \( \dot{V}E/\dot{V}CO_2 \), and end-tidal \( P_{CO_2} \) were measured.

Based on changes in \( \dot{V}E/\dot{V}O_2 \) and \( \dot{V}E/\dot{V}CO_2 \) and end-tidal \( P_{CO_2} \) with changing \( \dot{V}O_2 \) associated with the work rate, ventilatory threshold (VT) was identified as the \( \dot{V}O_2 \) point at which transition of \( \dot{V}E/\dot{V}O_2 \) from falling to rising phases occurred before transition of \( \dot{V}E/\dot{V}CO_2 \) from falling to rising phases, and that of end-tidal \( P_{CO_2} \) from increasing and then leveling off (i.e., isocapnic buffering) to decreasing occurred. The \( \dot{V}O_2 \) point at which the latter two transitions occurred was defined as respiratory compensation point (RCP) [7, 22]. Changes in end-tidal \( P_{CO_2} \) during isocapnic buffering phase (corresponding to the \( \dot{V}O_2 \) range between VT and RCP) were ascertained to be virtually zero, in actual being 0.95±0.25 (mean±SEM) mmHg. Maximum \( O_2 \) uptake (\( \dot{V}O_{2_{\text{max}}} \)) was determined as the average value of breath-by-breath data of \( \dot{V}O_2 \) during the last 1 min of the exercise test.

The \( \dot{V}CO_2/\dot{V}O_2 \) relationship was divided into two linear components, which had a lower slope (\( S_1 \)) and a higher slope (\( S_2 \)), respectively. \( S_1 \) was calculated using the \( \dot{V}CO_2/\dot{V}O_2 \) plots within the range of \( \dot{V}O_2 \) from the start of loaded cycling to VT, and \( S_2 \), using those from VT to RCP by the least-squares method. For the \( S_1 \) calculation, the data immediately after the start of the loaded cycling in which the respiratory gas exchange ratio (\( \dot{V}CO_2/\dot{V}O_2 \)) transiently fell were excluded. The difference between the two slopes (\( \Delta \text{slope} \)) was used as an estimate of the rate of lactic acid increase per \( \dot{V}O_2 \) increase at the work rates below RCP.

**Statistics.** The results are presented as means±SEM. Correlations between two variables were tested using Pearson’s correlation coefficients. Comparisons in the measurements between male and female subjects were analyzed using Welch’s test for unpaired samples. The level of \( p < 0.05 \) was considered to be significant.

## RESULTS

Mean results of the measurements and the variability among the subjects are shown in Table 1 for each sex. Since it has been reported that HVR and HCVR are affected by body surface area [23], all variables studied were corrected for body surface area. Correlation analysis was performed to examine how the variability of RCP can be explained by those of HVR, HCVR and \( \Delta \text{slope} \). The analysis was made on each sex, because there were sex differences in RCP and HVR (Table 1) even after the correction for body size. Higher HVR in females than in males has been observed by some investigators, while the inverse relation has also been reported by others (see Aitken et al. [23]).

Table 2 shows that RCP correlated significantly and inversely with HVR and \( \Delta \text{slope} \) in the male subjects and similar tendencies persisted in the female subjects, although the correlation with HVR and \( \Delta \text{slope} \),

### Table 1. Mean results and variability of the measurements.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Male (n=22)</th>
<th>Female (n=16)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong> (year)</td>
<td>22±1</td>
<td>21±1</td>
</tr>
<tr>
<td><strong>Weight</strong> (kg)</td>
<td>64±2</td>
<td>50±2</td>
</tr>
<tr>
<td><strong>Height</strong> (cm)</td>
<td>172±1</td>
<td>160±2</td>
</tr>
<tr>
<td><strong>Body surface area (m(^2))</strong></td>
<td>1.75±0.02</td>
<td>1.59±0.03</td>
</tr>
<tr>
<td><strong>( \dot{V}O_{2_{\text{max}}} )</strong> (l min(^{-1}) m(^{-2}))</td>
<td>1.74±0.05</td>
<td>1.26±0.05</td>
</tr>
<tr>
<td><strong>VT</strong> (l min(^{-1}) m(^{-2}))</td>
<td>0.70±0.04</td>
<td>0.56±0.03</td>
</tr>
<tr>
<td><strong>RCP</strong> (l min(^{-1}) m(^{-2}))</td>
<td>1.35±0.05</td>
<td>1.05±0.05</td>
</tr>
<tr>
<td><strong>HVR</strong> (l min(^{-1}) m(^{-2}) (%))</td>
<td>0.18±0.02</td>
<td>0.26±0.02</td>
</tr>
<tr>
<td><strong>HCVR</strong> (l min(^{-1}) m(^{-2}) mmHg(^{-1}))</td>
<td>0.40±0.53</td>
<td>0.61±0.32</td>
</tr>
<tr>
<td><strong>( \Delta \text{slope} )</strong></td>
<td>0.33±0.02</td>
<td>0.31±0.02</td>
</tr>
</tbody>
</table>

Values are means±SEM and ranges in parentheses. \(^6\) Rate of excess \( CO_2 \) output (l min\(^{-1}\)) per \( \dot{V}O_2 \) increase (l min\(^{-1}\)). Gender difference at \(* p<0.05\) and \(** p<0.01\).
respectively, did not reach significant levels (0.05 < 
\( p < 0.1 \)). Figures 1 and 2 illustrate the distributions of 
RCP-HVR relation and RCP-\( \Delta \)slope relation, respec-
tively, in both sexes. Combining the data of both 
sexes, significant correlation was seen in RCP-HVR 
and RCP-\( \Delta \)slope relations. On the other hand, there 
was no significant correlation between RCP and 
HCVR for either sex or in the combination of both 
sexes (Table 2).

To test whether the two variables (HVR and 
\( \Delta \)slope) were linearly and additively related to the 
RCP variability (i.e., whether relationship among the 
three variables can be described as 
\[ RCP = a[HVR] + b[\Delta \text{slope}] + \text{constant} \]), a multiple linear regression 
analysis was applied. As shown in Table 3, in the 
male subjects, the partial correlation for each the 
HVR and \( \Delta \)slope was significant and the magnitudes 
of the correlation coefficient were similar in both 
variables; the results indicating that HVR and \( \Delta \)slope 
were related independently and at similar rates to RCP 
variation. In the female subjects, however, the results 
of the partial correlation were not significant. Com-
paring the results of both sexes, results were signifi-
cant. The results of \( r^2 \) (Table 3) indicated that 40 to 
50% of the variability of RCP could be explained by 
a multiple linear regression model with HVR and 
\( \Delta \)slope, although the results of the female subjects 
did not reach significant levels (\( p=0.07 \)).

**DISCUSSION**

The multiple linear regression analysis between the 
variance of RCP and those of HVR and \( \Delta \)slope demonstrated that the latter two variables were related 
independently and at similar rates to RCP variation in 
the male subjects, while this relationship did not hold 
true for the female subjects. In the present study, the

![Fig. 1. Relationship between HVR and RCP of individual subjects. Closed circles indicate males (n=22), and open circles, females (n=16).](image1)

![Fig. 2. Relationship between \( \Delta \)slope and RCP of individual subjects. For explanation, see Fig. 1.](image2)

<table>
<thead>
<tr>
<th>Sex</th>
<th>n</th>
<th>HVR</th>
<th>HCVR</th>
<th>( \Delta )slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>22</td>
<td>-0.423*</td>
<td>0.365</td>
<td>-0.617**</td>
</tr>
<tr>
<td>Female</td>
<td>16</td>
<td>-0.461</td>
<td>0.060</td>
<td>-0.464</td>
</tr>
<tr>
<td>All</td>
<td>38</td>
<td>-0.566**</td>
<td>0.250</td>
<td>-0.423**</td>
</tr>
</tbody>
</table>

* \( p < 0.05 \) and ** \( p < 0.01 \).

<table>
<thead>
<tr>
<th>Sex</th>
<th>n</th>
<th>Partial correlation coefficient</th>
<th>( r^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>22</td>
<td>-0.533*</td>
<td>-0.678**</td>
</tr>
<tr>
<td>Female</td>
<td>16</td>
<td>-0.395</td>
<td>-0.399</td>
</tr>
<tr>
<td>All</td>
<td>38</td>
<td>-0.590**</td>
<td>-0.462**</td>
</tr>
</tbody>
</table>

\( r^2 \): coefficient of determination of a multiple linear regression model as described by 
\[ RCP = a[HVR] + b[\Delta \text{slope}] + \text{constant} \]. * \( p < 0.05 \) and ** \( p < 0.01 \).

<table>
<thead>
<tr>
<th>Sex</th>
<th>n</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>22</td>
<td>0.815**</td>
</tr>
<tr>
<td>Female</td>
<td>16</td>
<td>0.708**</td>
</tr>
<tr>
<td>All</td>
<td>38</td>
<td>0.785**</td>
</tr>
</tbody>
</table>

* \( p < 0.05 \) and ** \( p < 0.01 \).
individual difference in HVR during exercise was assumed to be predicted by that in the resting estimate of HVR \[11, 12, 14\]. A lower correlation between resting and exercise estimates of HVR in women than in men \[12\] might partly explain the lower partial correlation between RCP and HVR in our female subjects. In addition, no special regard of the menstrual cycle was paid to the female subjects. The luteal phase of the menstrual cycle has been reported to produce greater ventilatory response at rest and during exercise \[24, 25\], greater HVR \[24–26\] and a lower extent of blood lactate increase during heavy exercise \[26, 27\] compared to the follicular phase. Therefore, in the luteal phase, a decrease in arterial pH associated with a given increase in blood lactate during heavy exercise (i.e., at a given level of Δslope) might be smaller due to a greater fall in arterial \(PCO_2\) as a result of greater ventilatory response during exercise \[26, 27\] as compared with the follicular phase. This sequence might lead to a higher level of RCP in the luteal phase. However, previous studies have failed to find a dependency of RCP on the menstrual cycle \[26\] or on blood progesterone concentrations at similar levels of blood lactate increase during heavy exercise \[28\]. At any rate, heterogeneity in the phases of menstrual cycle in our female subjects might have caused a lower partial correlation between RCP and Δslope. Further studies are required as to whether relationship between RCP and each of HVR and Δslope depends on sex and menstrual cycle in woman.

With some reservations for woman, the present study indicates that 40 to 50% of the RCP variance was describable in a multiple linear regression model with HVR and Δslope (Table 3). This implies that, in the subjects with higher HVR, RCP indicating the onset of compensatory hyperventilation for metabolic acidosis during exercise could occur at lower levels of work rate even if the Δslope indicating an approximate rate of lactic acid increase during moderate to heavy exercise was the same. It follows that in individuals with higher HVR, the fall in pH due to lactic acidosis at a given level of heavy work rate would be restricted to a smaller extent because compensatory hyperventilation could start at lower levels of work rate as compared with individuals with lower HVR. Rausch et al. \[29\] have evidenced the importance of the carotid chemoreceptor sensitivity in constraining the fall of arterial pH by demonstrating a more rapid restoration of arterial pH toward the normal level during heavy exercise while breathing 12% \(O_2\) and a slower restoration while breathing 80% \(O_2\) as compared to restoration while breathing air.

Other investigators have also studied the influence of the chemosensitivity of the carotid bodies on the onset of hyperventilatory responses, detectable as VT and RCP, during incremental exercise. Hogan et al. \[30\] reported that, as compared to VT during exercise at 15-W increment every 3 min while breathing air, there was a shift to higher levels of VT with reduced chemosensitivity by breathing 60% \(O_2\), and there was no change with an elevated chemosensitivity by 17% \(O_2\). At such a slower rate of work incrementation, VT has been assumed to approximately equal RCP \[5\]. On the other hand, Henson et al. \[31\] failed to demonstrate an inhibitory effect of dopamine, which is an inhibitory neuromodulator in the carotid bodies, on ventilatory responses during 1-min incremental exercise (i.e., infused dopamine did not diminish the ventilatory responses or delay the onset of compensatory hyperventilation) in spite of having an inhibitory effect on the hypoxic ventilatory response assessed at rest. Henson et al. \[31\] therefore postulated that either the carotid bodies respond differently to hypoxia than to acute metabolic acidosis and/or hyperkalemia that are induced during heavy exercise, or the carotid bodies are not the sole mediators of compensatory hyperventilation during heavy exercise.

Agreeing with Henson’s postulation \[31\], we suppose that factors other than HVR and Δslope might be involved in the 50 to 60% RCP variance. They are marked increases in \(K^+\) and catecholamine concentrations, \(pH/PCO_2\) oscillation of the arterial blood and the \(O_2\)-labile component estimated by Dejours’ \(O_2\) test, which likely result in increased activities of the carotid bodies during exercise \[3–6\]. Increased plasma concentrations of \(K^+\) \[14, 32\] and catecholamines \[33\] have also been considered to contribute to increases in HVR during exercise. In the present study, the resting estimate of HVR was used as an index of the exercise estimate of HVR based on previous studies \[11, 12, 14\] showing that individuals with higher HVR at rest tended to have higher HVR during exercise, although this tendency (correlation between both) was lower for women, as mentioned above.

Besides the carotid chemoreceptor mediation, central command mediation might be involved in RCP determination, such that an augmented central command signal possibly due to increased muscle fatigue at heavy exercise might produce hyperventilation \[34\]. On the contrary, less involvement of the chemosensitivity of the central chemoreceptors was found in the present study (Table 2).

Recently, Oshima et al. \[22\] reported a positive correlation between RCP and \(\dot{V}O_{2\text{max}}\), in which the subjects with higher \(\dot{V}O_{2\text{max}}\) had higher levels of RCP. After Oshima et al. \[22\], a correlation analysis
between $\bar{V}O_2_{\text{max}}$ and each of RCP, HVR, HCVR and $\Delta$slope was performed on the subjects of the present study. The results are shown in Table 4, indicating that RCP correlated positively with $\bar{V}O_2_{\text{max}}$ in both sexes, while HVR and $\Delta$slope tended to correlate weakly and inversely with $\bar{V}O_2_{\text{max}}$. The inverse correlation between $\bar{V}O_2_{\text{max}}$ and $\Delta$slope might be associated with a training effect of diminishing lactate increase during heavy exercise [35]. On the other hand, regarding the correlation between HVR and $\bar{V}O_2_{\text{max}}$, the previous studies are controversial: some studies have shown an inverse correlation [36, 37], but others no correlation [11, 38, 39].

Thus, the positive correlation between $\bar{V}O_2_{\text{max}}$ and RCP found by Oshima et al. [22] and us appears to be accounted for partly by reduction in the chemosensitivity of the carotid bodies and/or lactic acidosis with increasing $\bar{V}O_2_{\text{max}}$. These alterations would make it possible to perform higher intensities of exercise without excessive hyperventilation in response to metabolic acidosis, and hence, intense breathlessness during heavy exercise [40].

In conclusion, the present study demonstrated that the individual difference in the onset of compensatory hyperventilation for lactic acidosis during incremental exercise, detectable as RCP, was dependent partly on the hypoxic ventilatory chemosensitivity of the carotid bodies and the rate of lactic acid increase during exercise. That is, earlier onsets of compensatory hyperventilation could be seen in individuals with higher chemosensitivities of the carotid bodies, even at the same rate of lactic acid increase during exercise, and in individuals with greater rates of lactic acid increase, even with the same chemosensitivity of the carotid bodies. This relation was manifested in the male subjects but not in the female subjects, where consideration of the menstrual cycle was not given. Based on the present results, the higher RCP seen in the subjects with higher $\bar{V}O_2_{\text{max}}$ was ascribed partly to lower levels of HVR and/or lactic acid increase during heavy exercise.

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