Effect of inspiratory threshold loading on ventilatory kinetics during constant-load exercise

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Rationale of the ventilatory adaptation to exercise load, especially the time constants (τ), which represent the time to reach 63% of the steady-state response of the ventilatory parameters.

Although the neural and humoral mechanisms involved in ventilatory control are considered to be the major drivers of exercise hyperpnea, evidence has emerged that mechanical respiratory factors (receptor afferents from the diaphragm, abdominal and intercostal muscles, and lung) may also contribute to phase 2 regulation. For example, Sergysels et al. (23), Hesser and Lind (10), and Ramonatxo et al. (20) showed a progressive divergence during incremental exercise between mouth occlusion pressure (P0.1), which represents the respiratory neuromuscular command, and ventilatory response, which reflects an increased respiratory drive in response to higher respiratory impedance. These results are consistent with the notion of a compensatory reaction to a mechanical load during exercise of even moderate intensity. Moreover, Ramonatxo et al. (19) showed that an inspiratory resistive load during phase 3 of moderate exercise caused an increase in the total inspiratory neural output and that at least a part of the extra work imposed by the added resistance was carried out by respiratory muscles other than the diaphragm, particularly the abdominal accessory and intercostal muscles. Similarly, Poon et al. (16) showed that a reduction in effective inspiratory resistance by assisted ventilation elicits a compensatory decrease in inspiratory neural output with little change in chemical drive and exercise ventilatory response. This was assumed to reflect a compensatory reduction in ventilatory drive, with steady-state VE and end-tidal PCO2 restored to their control levels.

However, the role of respiratory mechanical factors on ventilatory response during phase 2 of constant-load exercise is not well understood. Prioux et al. (17) showed that inspiratory neural output as assessed by mouth occlusion pressure was higher and ventilatory response was lower during an exercise with increments of 4 min as opposed to 1 min, 30 s. They thus suggested that change in the respiratory system impedance during phase 2 of constant-load exercise could affect the ventilatory drive.

Based on the above findings, we hypothesized that afferents of the neuromechanical ventilatory factors and consequently of the neuromechanical loop would serve as a feed-forward component of the ventilatory response. Because an experiment with heart and heart-lung transplant patients showed that when one control mechanism is missing, another one replaces it (1), one way to evaluate the contribution of neuromechanical factors to ventilatory control is to increase the respiratory system work,
i.e., the stimulation of receptors from the lung and the thoracopulmonary system. This can be accomplished by applying an external inspiratory load during exercise. To verify our hypothesis, we studied the effects of an added inspiratory load on the $V_{O2}$, $V_{CO2}$, and $V_{E}$ kinetics during constant-load exercise in normal subjects.

**MATERIALS AND METHODS**

**Subjects**

We studied 14 healthy men (mean age 22.6 ± 3.2 yr, height 182.5 ± 5.7 cm, and weight 77.9 ± 7.1 kg). The local Review Board for Research on Human Subjects approved the protocol. All subjects gave informed consent to participate in a loaded breathing experiment after receiving a detailed description of the experimental procedures. However, they were unaware of the purpose of the study. The inclusion criteria were the following: age between 20 and 35 yr, free of cardiac and pulmonary disease, and between 7 and 10 h of physical exercise per week.

**Protocol**

The study was carried out on two different days. On the first day, lung volumes and flows were assessed by plethysmography at rest. The subjects then performed an incremental load exercise, so that their ventilatory threshold and maximal oxygen uptake ($V_{O2,max}$) could be determined.

On the second day, which was 2 to 6 days after the first laboratory visit, the subjects performed two constant-load tests. Each test was conducted at the same time of day to limit circadian evolution. On the day of the tests, subjects were asked to not participate in other exercise until after the experiment.

**Plethysmography**

Vital capacity (VC), forced expiratory volume in 1 s (FEV$_1$), and total lung volume capacity were measured for each subject according to standard techniques and procedures (American Thoracic Society) ($V_{max}$ 229 series Sensormedics).

**Maximal Incremental Exercise**

Each subject performed an incremental cycling exercise to volitional exhaustion. Pedaling frequency was set at 70 rpm. The time constant of heart rate dependence at 70 rpm and the plateau for $V_{O2}$ were considered as criteria of $V_{O2,max}$.

**Constant-Load Exercise**

Each subject performed two cycling exercises with and without added inspiratory load (15 cmH$_2$O) in balanced random order. The subjects were warmed up with 5 min of unloaded cycling before each test to begin testing with stable ventilatory parameters and respiratory exchange ratio (QR). They then pedaled for 8 min at a power output corresponding to 40% of $V_{O2,max}$, followed by 10 min of recovery with unloaded cycling. A metronome and speed transducers linked to the computer were used to maintain a constant pedaling frequency at 70 rpm.

**Measurements**

Tests were performed in a laboratory maintained at 21°C. All the tests were performed on a friction-loaded cycle ergometer (model 828 E; Monark, Stockholm, Sweden) fitted with a strain gauge (MFG type; Interface, Scottsdale, AZ) and an incremental encoder to ensure accurate measurement of power output. Use of speed and force transducers (Electronic Informatics Pilat, Jonzieux, France) ensured that the targeted power output was maintained. The ergometer was calibrated immediately before the start of the test by using equipment supplied by the manufacturer. To measure the friction force, the strain gauge was calibrated with a known mass (10 kg) hung on the friction belt and in unloaded condition to give a zero value. To measure the flywheel inertia, the method proposed by Lakomy (13) was used. Briefly, the inertia was simply determined from the linear relationship obtained between the free acceleration of the flywheel and the corresponding friction load.

Breath-by-breath data for $V_{O2}$, $V_{CO2}$, and $V_E$ were collected continuously throughout the tests and measured with an automatic gas exchange system (CPX Medical Graphics, St. Paul, MN), including a zirconium cell for $O2$ analysis, an infrared cell for $CO2$ analysis, and a heated pneumotachograph (Fleish No. 3; Godart Slatham, Amsterdam, Holland). The $CO2$ and $O2$ analyzers were calibrated before each test with a bottle of known composition (12% $O2$ and 5% $CO2$). The pneumotachograph was calibrated using a 3-liter syringe. The two-way valve was reinforced with a mica part to prevent losses in the inspiratory and expiratory circuits through valve deformation. The total dead space was 100 ml. For the constant-load exercise with added inspiratory load, a threshold valve system (threshold model IMT 730; EU-Respirtonics, Health Scan Asthma, Allergy Producer, NJ) was inserted on the inspiratory circuit of the valve. This type of threshold valve maintains a constant resistance whatever the ventilation level (8); in other words, the increase in the work rate of breathing due to the added inspiratory resistance is independent of ventilation. Breath-by-breath data for $V_{O2}$, $V_{CO2}$, and $V_E$ tidal volume ($V_T$), respiratory frequency (in breaths/min), inspiratory time (TI; in s) and expiratory time (in s) were collected continuously throughout testing. Heart rate was measured using an electrocardiogram including a standard bipolar electrode placement.

**Data Analysis**

Nonlinear regression techniques were used to fit the $V_{O2}$, $V_{CO2}$, and $V_E$ data after exercise onset with an exponential function. An iterative process ensured that the sum of the squared error between the modeled $V_{O2}$, $V_{CO2}$, and $V_E$ and the measured $V_{O2}$, $V_{CO2}$, and $V_E$ was minimized. The mathematical model consisted of an exponential term that represents phase 2. The exponential term constrained to start only at the "inflection point" of the response thanks to a time delay and an amplitude (29)

$$\Delta Y(t) = Y_b + A_1 [1 - e^{-t/\tau} / e^{t/\tau}]$$

where $\Delta Y(t)$ is the parameter variation ($V_{O2}$, $V_{CO2}$, and $V_E$), $Y_b$ is the unloaded cycling baseline value, $A_1$ is the asymptotic values for the exponential term, $\tau$ is the time constant. The values of the measured $V_{O2}$, $V_{CO2}$, and $V_E$ that were greater than three standard deviations from the modeled $V_{O2}$, $V_{CO2}$, and $V_E$ were considered outliers and were removed, in line with a previous work (4). These outlier values were assumed to be due to shallow breaths or breath-holding and represented < 1% of the total data collected.

Model parameters were determined with an iterative procedure by minimizing the sum of the mean squares of the differences between the estimated $V_{O2}$, $V_{CO2}$, and $V_E$ based on the model and the measured $V_{O2}$, $V_{CO2}$, and $V_E$. Iterations continued until successive repetitions reduced both the sum of the residuals by < $10^{-6}$ and the correlation coefficient of the relationship between residuals and time by < $10^{-4}$. To assess the validity of the model parameters, coefficients of variation were computed using the bootstrap method (3, 9). Briefly, this consisted of resampling the original data set with replacements to create a number of "bootstrap replicate" data sets of the same size as the original data set. For each replicate set, model parameters were estimated following the same procedures as for original data. This operation was repeated 1,000 times, and the estimated parame-
Table 1. Pulmonary function data and maximal oxygen uptake of the subjects

<table>
<thead>
<tr>
<th>Subjects</th>
<th>VO₂max, m³/min·¹⁻¹ kg⁻¹</th>
<th>VC, liters</th>
<th>VC, %</th>
<th>FEV₁, liters</th>
<th>FEV₁/VC, %</th>
<th>TLC, liters</th>
<th>TLC, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>55.8</td>
<td>5.4</td>
<td>102.5</td>
<td>4.5</td>
<td>90.7</td>
<td>8.5</td>
<td>106.1</td>
</tr>
<tr>
<td>SD</td>
<td>6.4</td>
<td>0.8</td>
<td>9.3</td>
<td>0.3</td>
<td>8.9</td>
<td>1.2</td>
<td>15.5</td>
</tr>
</tbody>
</table>

Values are mean ± SD for 14 human subjects. VO₂max, maximal oxygen consumption; VC, vital capacity, %, percent predicted (18); FEV₁, force expired volume in 1 s; TLC, total lung capacity, %, percent predicted (18).

Results

All subjects completed the three exercises tests. All were within the normal predicted ranges for VC and FEV₁. The plethysmographic results are given in Table 1. The mean value of VO₂max was 55.8 ± 6.4 ml/kg (Table 1). The two constant-load exercises were performed at power outputs below the ventilatory threshold. The mean power output was 128 ± 13 W.

No significant relationships were identified between residuals and time in either experimental condition, suggesting random distribution of the residuals and a model adapted to describe the kinetics of the ventilatory parameters under both conditions. Model adjustment to the parameter kinetics led to coefficients of determination at 0.89 ± 0.01. The Fisher test indicated a high degree of significance of the model for all the coefficients of determination at 0.89 ± 0.01) (5). Figure 1 shows the mean VO₂, VCO₂, and VE responses pattern for all subjects to be more representative during exercise with and without inspiratory load. As shown in Figs. 1 and 2, an increase in VO₂, VCO₂, and VE was observed during the constant-load exercise with added inspiratory load compared with exercise without load. During the 0-W baseline exercise, basal VO₂ increased significantly by 3.7% with added load (0.82 ± 0.21 vs. 0.85 ± 0.19 l/min; P < 0.01), VCO₂ increased significantly by 14.7% (0.61 ± 0.29 vs. 0.70 ± 0.17 l/min; P < 0.01), and VE by 12.9% (17.9 ± 1.2 vs. 20.2 ± 1.1 l/min; P < 0.001). There was a significant increase in VE/VCO₂ with added load (21.8 ± 0.54 vs. 23.7 ± 0.62 l/min; P < 0.001) but not in VE/VCO₂ (29.3 ± 0.7 vs. 28.9 ± 0.7 l/min).

The steady-state value of VO₂ during phase 3 of the constant-load exercise increased significantly (+1.5%) during exercise with added load (2.00 ± 0.18 vs. 2.03 ± 0.19 l/min; P < 0.05), as did VCO₂ by +2.5% (1.98 ± 0.17 vs. 2.03 ± 0.18 l/min; P < 0.01) and VE by +6.7% (46.0 ± 7.1 vs. 49.1 ± 6.9; P < 0.001). The breathing pattern during exercise with and without added inspiratory load is shown in Table 2. With load, we observed a significant increase in tidal volume (VT) (P < 0.01) and T₁ (P < 0.01), and a significant decrease in respiratory frequency (P < 0.01) and mean inspiratory flow (VT/T₁).

Statistical Analysis

For all the subjects, ventilation and the pulmonary gas exchange τ were expressed as the means ± SD.

The Fisher test was used to determine the model’s degree of significance. Quality of the adjusted model was assessed by the coefficient of determination (r²) obtained between the modeled VO₂, VCO₂, and VE and the measured VO₂, VCO₂, and VE. The random distribution of the model according to time was checked using linear and nonlinear regressions. The Shapiro-Wilk test assessed the normality of the distribution of the parameters. Paired Student’s t-tests were used to compare the model parameters between the two experimental conditions. The relationship between VE and VCO₂ was assessed by Pearson’s correlation coefficients in the two experimental conditions. Differences were declared to be significant for P < 0.05.
Moreover, the correlation between V˙O2 max and exercise with and without load, respectively, and between 8.6 s without load to 60.9
0.001) for exercise with load compared with without load, as shown in Fig. 2. The V˙O2 increased by 41.3% from 43.1 ± 8.6 s without load to 60.9 ± 14.1 s with load; V˙CO2 increased by 40.1% (60.3 ± 17.6 vs. 84.5 ± 18.1 s) and V˙VE by 44.6% (59.4 ± 16.1 vs. 85.9 ± 17.1 s). The following ratios V˙O2/V˙E (0.72 ± 0.07 vs. 0.71 ± 0.06 s) and V˙CO2/V˙E (1.01 ± 0.05 vs. 0.98 ± 0.07 s) remained stable.

Figs. 3 and 4 show the individual values of V˙VE in relationship to both V˙CO2 and V˙O2 in the unloaded (Figs. 3A and 4A) and loaded (Figs. 3B and 4B) conditions. In all subjects, V˙VE was higher than V˙O2 in the two conditions. The V˙VE approximated V˙CO2, except in two subjects in unloaded condition and three in loaded condition for whom in both conditions, V˙VE was slightly lower than V˙CO2. The correlations between V˙VE and V˙O2 were 0.64 (P < 0.001) and 0.62 (P < 0.001) for exercise with and without load, respectively, and between V˙VE and V˙CO2 they were 0.72 (P < 0.001) and 0.83 (P < 0.001). Moreover, the correlation between V˙O2 max and V˙O2 was significant (r = 0.92; y = −1.36x + 119.45).

DISCUSSION

The most important finding of this study was the significant increase in V˙O2, V˙CO2, and V˙VE with inspiratory load during phase 2 of the constant-load exercise. The increased resistance thus significantly slowed the kinetics of these ventilatory parameters (Fig. 1).

Methodological Considerations

Several authors (2, 11) have used a procedure of measuring the kinetics of individual respiratory parameters two or three

times to decrease the variability inherent to breath-by-breath measurement of gas exchange. In the present study, this method was not applied, in accordance with the work of Carra et al. (4). To measure V˙VE, respiratory parameters must be stable before exercise begins, i.e., during unloaded cycling. By continuously monitoring the respiratory exchanges and the respiratory quotient, we were able to ascertain this stability. Moreover, a sudden switch to the desired power (<2 s) was necessary to fit the phase 2 kinetics. Thus the subjects were able to pedal at the chosen power output at the very beginning of constant-load exercise to achieve an optimal adaptation. We chose 40% of V˙O2 max so that the subjects perform the exercise

Table 2. Ventilatory parameters during steady state phase 3 of the constant-load exercise

<table>
<thead>
<tr>
<th>Subjects</th>
<th>V˙E, l/min</th>
<th>V˙T, liters</th>
<th>F, breaths/min</th>
<th>T1, s</th>
<th>T1/T, s</th>
<th>V˙E/T, l/s</th>
<th>V˙E/V˙CO2</th>
<th>QR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without load</td>
<td>46.0±7.1</td>
<td>2.6±0.5</td>
<td>18.8±3.1</td>
<td>1.5±0.3</td>
<td>1.7±0.4</td>
<td>23±0.3</td>
<td>0.88±0.08</td>
<td></td>
</tr>
<tr>
<td>With load</td>
<td>49.1±6.9</td>
<td>3.0±0.6†</td>
<td>16.9±2.2</td>
<td>1.8±0.4*</td>
<td>2.4±0.4†</td>
<td>14±0.3†</td>
<td>25±0.3†</td>
<td>0.92±0.07†</td>
</tr>
</tbody>
</table>

Values are means ± SD for 14 human subjects. V˙E, minute ventilation; V˙T, tidal volume; F, respiratory frequency; T1, inspiratory time; T1/T, expiratory time; and QR, respiratory quotient. *P < 0.05; †P < 0.01; ‡P < 0.001.

FIG. 2. Means values of the V˙O2, V˙CO2, and V˙VE during constant-load exercise with and without inspiratory load. Note how respiratory load induced faster V˙O2, V˙CO2, and V˙VE (**P < 0.001).

FIG. 3. Individual values of the minute V˙E in relation to V˙CO2 during constant-load exercise without (A) and with (B) added inspiratory load. Values of V˙CO2 are shown on the x-axis, and values of V˙E are shown on the y-axis.

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Does an Added Inspiratory Load Change the \( V_O2, V_{CO2}, \) and \( V_E \) Kinetics?

The aim of our study was to define the contribution of the neuromechanical loop to ventilatory control during exercise by the addition of an inspiratory load. Breathing with a load increases the work of the respiratory muscles and, thereby, the impedance of the thoracopulmonary system. This in turn causes increased stimulation of the receptors of the respiratory muscles, lungs, and airways. The main result was that the added threshold load significantly increased the \( \tau \) of ventilation and the respiratory exchanges. This reflects slower kinetics of these parameters and consequently a poorer adaptation during constant-load exercise. This result may be surprising, because the increase in the respiratory mechanical factors caused by an inspiratory load would add neural drive (19) and may serve as a feed-forward component of the ventilatory response to exercise, thus serving to reduce and not to prolong the ventilatory time-constants.

Some mechanisms can be advanced to explain the slower kinetics of \( V_O2, V_{CO2}, \) and \( V_E \) with load. An added threshold load during baseline and constant-load exercise induces changes in 1) respiratory homeostasis i.e., a ventilation without variations to maintain \( P_{ACO2} \) and \( pH \) at resting values; 2) the intrathoracic pressures and thus in cardiac output or right ventricular strain; and 3) information from the respiratory muscles, as well as from lung and airways mechanoreceptors.

Changes in respiratory homeostasis. During the 5-min, 0-W baseline exercise, no differences in the values of \( V_E/V_{CO2} \), \( P_{ETCO2} \), and \( \text{QR} \) were noted between the loaded and unloaded exercise. On the other hand, during \( \text{phase 3} \), the mean \( V_E/V_{CO2} \) ratio was higher and the mean \( P_{ETCO2} \) was lower for loaded exercise. Only the \( \text{QR} \) was slightly higher during exercise with load. However, the constant-load exercises were performed under the ventilatory threshold, and it seems unlikely that anaerobiosis would occur during loaded exercise. Thus the slower ventilatory kinetics do not seem due to changes in homeostasis.

Changes in intrathoracic pressure. Wasserman et al. (25) proposed the cardiodynamic hyperpnea hypothesis. For these authors, a neural mechanism originating in the heart and lungs may cause increased ventilation consequent to a rapid increase in cardiac output or right ventricular strain. Thus changes in intrathoracic pressures might slow the increase in cardiac output, and the decreased rate of \( CO2 \) delivery to the lungs would increase \( V_O2, V_{CO2}, \) and \( V_E \). However, addition of an inspiratory resistive load induces a more negative esophageal pressure at peak inspiration compared with control (19). The more negative intrathoracic pressure modifies the venous return. However, the results are contradictory; a more negative thoracic pressure could decrease (21, 27) or increase (5) the venous return and thus modify differently the cardiac output. A slower increase in cardiac output consequent to a decreased venous return could partly explain the slower \( \tau \) with respiratory load instead of a resistive load. Indeed, the studies of Ramonatxo et al. (19) and D’Urzo et al. (7), which used resistive loads, found either a significant decrease in \( V_E \) during steady-state exercise or no change during incremental exercise. The \( V_O2, V_{CO2}, \) and \( V_E \) measured without added inspiratory threshold load were 43, 59.3, and 60.2 s, respectively.

Comparison with the Literature

Our results showed a significant increase in \( V_E \) during steady-state \( \text{phase 3} \) exercise with inspiratory load. The \( V_E \) increase may have been due to the use of a threshold load below the ventilatory threshold and reach a steady state (\( \text{phase 3} \)) during the exercise with and without inspiratory threshold load. Above this threshold, a slow component of \( V_O2 \) could appear.

Inspiratory load was applied with a threshold valve, which maintains a constant-load, whatever the ventilation level (8); in other words, the pressure imposed at each breath by this device is flow independent and thus more constant than linear inspiratory resistance. We used this type of valve because it has been already used in an attempt to simulate the conditions of restrictive and obstructive lung disease. Moreover, this valve reproduces the conditions encountered in certain sports like scuba diving.

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load during phase 2. Furthermore, changes in intrathoracic gas volume results in a shift in the ventilation-perfusion match, which could affect kinetics of VO2, VCO2, and VE.

Changes in feed-forward mechanisms from respiratory muscles, lung, and airways. The added respiratory load increased the respiratory muscle work and stimulated receptors from respiratory muscle, lung, and airways. Among them, lung stretch receptors are known to be particularly influenced by changes in the mechanical properties of lungs, presumably because they are located in the airways’ wall and are affected by the pull of the lung parenchyma on the airways. This effect could be due to a change in compliance of the unit where the receptor is located (reviewed by Widdicombe in Ref. 30). The role of the pulmonary stretch receptors is to protect the lungs against hyperinflation; they exert the well-established Breuer-Hering inflation reflex, whereby increases in lung volume inhibit respiratory activity. Because we showed a significant increase in VT and a decrease in Vg/Ti during exercise with inspiratory load, it seems reasonable to think that this type of load stimulated the pulmonary stretch receptors. Consequently, their “defense” mechanism of inhibiting inspiratory activity might explain the poorer adaptation to exercise, as indicated by the longer \( \tau \).

Other important receptors that could play a role are from the respiratory muscles. Indeed, during exercise with inspiratory load, there is a higher stimulation of the diaphragm, which contains a great number of Golgi tendon organs. These organs, in series with the muscle fibers, are stretch receptors with slow-adapting response. The work of Laporte and Lloyd (14) indicated that afferent messages originating from Golgi receptors trigger an inhibition of the \( \alpha \)-motoneurons of corresponding muscles via interneurons. Thus stimulation of Golgi tendon organs, located mainly in the diaphragm, exerts an inhibiting effect on ventilation and may thus explain the slower ventilatory kinetics during exercise with inspiratory load.

Moreover, some studies (reviewed by Jammes in Ref. 12) have shown that breathing and airway resistance can be altered by activation of thinly myelinated and nonmyelinated afferents whose endings are in the diaphragm. A mechanical stimulation of these afferents produces a reflex inhibition of phrenic motoneurones, slightly alters respiratory frequency, and reduces airways resistance. Even though all of these effects are small, we can suggest that increased activation of these afferents by inspiratory resistance may exert an inhibitory influence on breathing and could account for the slower ventilatory kinetics.

Furthermore, with load, the recruitment of the external and parasternal intercostal muscles is high, which may be at the origin of an increase in the discharge of the neuromuscular spindles. These afferents could act by their projections at the cortical level and generate the respiratory feeling of increased constraint. Responses to loads in conscious subjects at rest are thus, in part, behavioral. This feeling, which is responsible for a cortical arousal, induces a modification in ventilatory control and may thereby account for the slower ventilatory kinetics.

This study was the first to show that mechanical factors from the lung and respiratory muscles play a role in the control of exercise hyperpnea during phase 2 of constant-load exercise. The 45% increase in the \( \tau \) with a threshold load of 15 cmH2O shows that the influence of respiratory feedback is important.

Our results agree with Poon’s model of ventilatory control that combines chemical and mechanical feedback (15, 16). According to this model, ventilatory flow is regulated by the respiratory centers to minimize the opposing effects caused by the imbalance between the humeral and mechanical respiratory constraints. A mechanical limitation induced by an inspiratory load explains the deceleration in the ventilatory kinetics.

In conclusion, our main finding was the increase in \( \tau \) during constant-load exercise with inspiratory load compared with unloaded exercise, i.e., a poorer adaptation to effort. These changes indicated that respiratory mechanical factors play a role in ventilatory control. This role is probably minor in normal conditions during constant-load exercise but becomes more important with exercise intensity. Any increase in the load imposed on the respiratory system, whether from sports activities (diving), pollution masks, or pathologies, is likely to involve identical deterioration.

REFERENCES


