Enhanced ventilatory and exercise performance in athletes with slight expiratory resistive loading

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Fee, Lawrence L., Richard M. Smith, and Michael B. English. Enhanced ventilatory and exercise performance in athletes with slight expiratory resistive loading. J. Appl. Physiol. 83(2): 503–510, 1997.—We determined the cardiopulmonary and performance effects of slight (1.5–3.0 cmH2O) expiratory resistive loading (ERL). Twenty-eight highly fit athletes (age = 33.5 ± 1.3 yr) performed paired VO2peak cycle ergometer tests (control vs. ERL). End-expiratory lung volume was separately determined in a subset of subjects (n = 12) at steady-state 75% maximum power output (POmax) and was found to increase (0.67 ± 0.29 liter) with ERL. In the VO2peak tests, peak expiratory flow at the mouth, mean inspiratory flow, minute ventilation, and O2 pulse were greater with ERL at every intensity level (i.e., 75, 80, 85, and 90% POmax). Increased minute ventilation was largely due to a trend toward increased tidal volume (P < 0.05 at 80% POmax). O2 uptake was greater with ERL at every intensity level (18). FL at effort-independent slope of the maximal expiratory flow-volume (MEFV) curve (18). FL over a significant portion of the VT has been demonstrated over the range of intense to maximal exercise, developing at 50–75% V˙O2max (18). FL begins to occur at 50–75% V˙O2max (18). FL at end-expiratory lung volume (EELV) with exercise (13). When EELV is lowered, exercise expiratory tidal flow shifts closer to the MEFV envelope (3), increasing the potential for FL and reducing the maximal predicted Ve (16). Furthermore, at lung volumes below resting FRC, airway closure in dependent lung regions adds to maldistribution of inspiratory gas flow (13).

In healthy subjects, EELV has been observed to progressively decrease by ~0.7 liter during heavy exercise. EELV remains depressed until exercise becomes intense and then gradually returns to near-resting FRC at VO2max (17, 18). Despite this tendency of EELV to recover to its near-resting FRC position, a progressively greater portion of the VT becomes flow limited as exercise becomes more intense (18, 19).

In principle, expiratory resistive loading (ERL) might experimentally elevate EELV to a volume at which expiratory flow remains entirely within the MEFV curve (1) and away from the FL experienced at lower lung volumes (19). One frequently observed consequence of ERL is to increase V˙E and mean inspiratory flow (Vt/Ti) (11, 14, 24). However, concomitant with the increases in EELV associated with ERL, typically there have been adverse alterations, such as compromised Ve (11, 24) and mean expiratory flow (Vt/Te) (22, 24) and decreased cardiac output (perhaps secondary to reduced venous return in patients with chronic obstructive pulmonary disease due to elevated intrathoracic pressures) (23). However, our preliminary work (5) suggested that the degree of ERL employed in previous studies may have been too great (5.0–40.0 cmH2O) (7, 10, 11, 14, 22, 24) to elicit cardiorespiratory benefit. In a prior study, with only slight reductions in the internal diameter of the expiratory port of the Hans Rudolph low-resistance three-way valve, we observed mitigated exercise-induced hypoxemia (EIH) in highly fit, endurance-trained athletes at mild altitude (5). Because our former EIH-ERL study suggested that one of our experimental resistors offered beneficial resistance to expiration during strenuous exercise, we chose to conduct a sea-level study utilizing the same expiratory flow resistor used at altitude (5). In the present graded peak aerobic capacity (VO2peak) study, we measured peak expiratory pressure at the mouth (Pepeak) and changes in cardiorespiratory variables in highly fit endurance athletes. In a companion study, we determined exercise EELV in a representative subset of athletes (n = 12) with and without the experimental expiratory orifice during heavy, steady-state exercise at 75% of maximum power output (POmax).

METHODS

Subjects. All subjects (n = 28) were actively competitive male (n = 23) and female (n = 5) cyclists and/or triathletes (age = 33.5 ± 1.3 yr, VO2peak = 63.6 ± 1.3 ml·kg−1·min−1). They were studied with their informed consent, and all procedures were approved by the Human Experimentation

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Committee of the University of Hawaii at Manoa. Selected physical characteristics and lung volumes of the subjects are given in Table 1. All paired testing with a given subject was completed within 7 days. Subjects were asked to refrain from strenuous activity on the day before testing. All 28 subjects performed paired \( V_\text{O2peak} \) tests. Subsequently, EELV at steady-state 75% \( P_{\text{Omax}} \) was determined in a random subset of the \( V_\text{O2peak} \)-tested subjects (n = 12); 11 men and 1 woman. Their mean age (34.3 ± 2.3 yr), \( V_\text{O2peak} \) (68.7 ± 1.5 ml·kg\(^{-1}\)·min\(^{-1}\)), residual volume (1.52 ± 0.11 liters), vital capacity (5.31 ± 0.39 liters), and total lung capacity (7.17 ± 0.26 liters) differed only slightly from the \( V_\text{O2peak} \)-tested group.

Cycle ergometry. All tests were performed on an electrically braked cycle ergometer (model 800, Ergometrics) that was integrated with a Schiller AT-6 (CH6340), a multiplex system capable of monitoring electrocardiogram (ECG), testing pulmonary function, and controlling the exercise protocol. During an initial familiarization session, subjects were asked to choose a power output (PO, in W) that was comfortable, yet somewhat demanding, and to indicate at what cadence they normally cycled. These became the starting PO and the subjects maintained their fixed cadence at zero pedal resistance, followed by 1-min interval/transition, during which the subjects maintained their fixed cadence at zero pedal resistance, followed by the 8-min warm-up. Every test (i.e., those in the \( V_\text{O2peak} \) series or the steady-state at 75% \( P_{\text{Omax}} \) series) began immediately after the transition interval and commenced with the initial minute at the subject’s predetermined, fixed cadence and chosen PO. Group mean heart rate (HR) at starting PO was 131.0 ± 2.9 (SE) beats/min. Workload was increased by 10 W/min thereafter. Subjects were asked to keep their cadence within 1–2 revolutions/min (rpm) of the digital, instantaneous rpm readout at all times, which we strictly monitored along with current wattage.

ERL. During the control tests, subjects breathed through a three-way low-resistance valve (model 2700, Hans Rudolph). The three-way valve was mounted in such a way as to ensure a relatively consistent body position during the tests. ERL was effected by reducing the internal diameter of the expiratory port of the three-way valve from 28.6 to 22.2 mm at its junction with the gas-conducting tubing. Figure 1 regressions are derived from the pooled data of all subjects during \( V_\text{O2peak} \) testing and present the profiles of \( P_\text{Epeak} \) vs. \( V_\text{E} \) in without (control) and with ERL. The control data were best described by a first-order polynomial, whereas the ERL data were best described by a second-order polynomial. In all comparisons within this study the order of presentation of expiratory orifices was alternated, such that ERL was presented first in one-half of the subjects, and the subjects were naive to the intervention. \( P_\text{Epeak} \) was sampled from a sampling port hose barb in the housing of the three-way valve. A 0.5-m section of rigid plastic tubing (2 mm ID) connected the barb to a volumetric pressure transducer (model PT-5A, Grass), which was linear over 0.0–12.0 cmH\(_2\)O. Before each test the transducer was calibrated with a water manometer. The Grass transducer was interfaced with a strain-gauge coupler (model 2193, Harvard Apparatus). During all tests, \( P_\text{Epeak} \) traces were recorded on a 12-speed chart mover (model 486, Harvard Apparatus). The chart speed was maintained at 0.025 cm/s, except for single-breath traces (\( P_\text{E} \)), which were recorded at 5.0 cm/s.

\( V_\text{O2peak} \) testing. Each subject performed two graded \( V_\text{O2peak} \) cycle ergometer tests to exhaustion: one with ERL and one without (control). In each test, \( V_\text{O2peak} \) was considered the highest \( O_2 \) consumption (\( V_\text{O2} \)) maintained for a full minute. The higher of the two \( V_\text{O2peak} \) values was used to determine group mean \( V_\text{O2peak} \) (Table 1). \( P_{\text{Omax}} \) (in W) was considered to be the higher last full-minute wattage attained in either of the two \( V_\text{O2peak} \) tests. We did not validate the reproducibility in the \( P_{\text{Omax}} \) tests. We did, however, randomize the presentation of the orifices (control vs. ERL).

Steady-state testing. Subjects performed two graded cycle ergometer tests (with and without ERL) up to a steady state of 75% \( P_{\text{Omax}} \) at which point EELV was determined. The EELV test protocol began just as in the \( V_\text{O2peak} \) tests, except at 75% \( P_{\text{Omax}} \) the wattage was manually fixed and maintained for 4 min. With the assumption of steady state (28), metabolic data associated with EELV were collected and averaged in the 3rd min. At the beginning of the next (4th) min, we switched the subject to the closed-circuit \( H_2 \)-rebreathing system for 30 s.

EELV determination: He dilution. EELV was determined by \( H_2 \) dilution. Two manual, directional control, three-way Y-shaped valves (model 2100 C, Collins) were used to switch from the open-circuit gas-evaluating system to the closed-circuit \( H_2 \) residual volume apparatus (model P-1300, Collins) rebreathing system with a 13.5-liter spirometer. Before each test the system was purged, and the \( H_2 \) analyzer was

<table>
<thead>
<tr>
<th>Subject characteristics</th>
<th>Mean ± SE</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>33.5 ± 1.3</td>
<td>21–51</td>
</tr>
<tr>
<td>Height, cm</td>
<td>172.0 ± 1.4</td>
<td>165–192</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>70.1 ± 1.5</td>
<td>51.8–80.0</td>
</tr>
<tr>
<td>( V_\text{O2peak} ), ml·kg(^{-1})·min(^{-1})</td>
<td>63.6 ± 1.3</td>
<td>53.6–78.2</td>
</tr>
<tr>
<td>RV, liters</td>
<td>1.45 ± 0.07</td>
<td>1.02–2.47</td>
</tr>
<tr>
<td>VC, liters</td>
<td>5.73 ± 0.17</td>
<td>4.78–8.03</td>
</tr>
<tr>
<td>TLC, liters</td>
<td>7.21 ± 0.21</td>
<td>5.66–10.35</td>
</tr>
<tr>
<td>FVC, %</td>
<td>111.3 ± 2.42</td>
<td>91–137</td>
</tr>
<tr>
<td>FEV(_{1.0} ), %</td>
<td>112.10 ± 2.72</td>
<td>89–133</td>
</tr>
</tbody>
</table>

Values are representative of 28 subjects (23 men and 5 women). \( V_\text{O2peak} \), peak aerobic capacity; RV, residual volume; VC, vital capacity; TLC, total lung capacity; FVC, forced vital capacity; FEV\(_{1.0} \), forced expiratory volume in 1 s. *Percentage of normal predicted values from Schiller (Schiller derivations based on Knudson, Crapo, and Morris).
calibrated with a known concentration of He-containing gas (5.0%), purged again, and loaded with 4 liters of O₂ and 0.6 liter of He. During the rebreathing (30 s), 100% O₂ was infused at a rate that maintained the system volume relatively constant. The system was scrubbed of CO₂ by barium hydroxide lime, US Pharmacopea (Collins absorbent granules). Final He concentrations were recorded when further gas mixing elicited no further changes in He concentration. All volumes were converted to BTPS.

Measurements. All tests were conducted under relatively stable environmental conditions: 20–21°C, 758–765 Torr, and 50–65% relative humidity. Barometric pressure was ascertained from the National Weather Service telephone recording, which was updated hourly. Laboratory temperature and relative humidity were determined from a thermometer/hygrometer (model 63–844, Micronta). Arterial O₂ saturation (SaO₂) was determined with an ear oximeter (model OPS-200, Satilite), which was internally self-calibrated before each exercise test. SaO₂ was measured only in the VO₂peak tests. Each subject's ECG and HR were continuously monitored during exercise testing with the Schiller AT-6 ECG system. Instantaneous reports of ECG and HR were given on the screen of a monitor (model 710 A, Mitsuba) interfaced with the AT-6 system. Before every exercise test the ECG monitor was calibrated with the Physio-dyne HR calibrator (ECG-Cal).

Ventilatory variables were monitored via standard open-circuit spirometry. Fractional concentrations of expired O₂ (FEO₂) and CO₂ (FECO₂) were determined by Ametek analyzers (models S-3A/I and CD-3A, respectively). Before every test, these analyzers were calibrated with two reference gases of the following concentrations: 21.03% O₂ and 5.02% CO₂ or 13.00% O₂ and 0.03% CO₂. Inspired ventilation volume (Vi) was determined with a flow transducer (model K520, K. L. Engineering). These measurements of FEO₂, FECO₂, and Vi and those of SaO₂, and HR (via Physio-dyne HR computer/ECG-HR3) were integrated with a CompuAdd 433 (model A000) computer and monitor (model 51109) that ran the Ametek O₂ uptake system OCM-2 program. The OCM-2 recorded SaO₂, HR, the respiratory exchange ratio (R), Ve, breathing frequency (f), VT, duration of expiration, inspiration, and breathing cycle (TE, Ti, and TT, respectively), Ti/TT, VT/TE, VT/Ti, FEO₂, FECO₂, VO₂, V CO₂ production (VCO₂), and O₂ pulse (VO₂/HR). Ve was determined from Vi and R, with differences in VO₂ and VCO₂ accounted for and corrected by the OCM-2 program. All dynamic ventilatory volumes were converted to BTPS and, along with SaO₂, and HR, were averaged to minute intervals by the OCM-2.

EELV and resting residual volume measurements were determined by standard He dilution and reflect allowances for O₂ consumed or added to the system. CO₂ was presumed to be entirely removed from the system. Vital capacity (VC), forced VC, and forced expiratory volume in 1 s were determined with the Schiller AT-6 pulmonary function testing equipment. Predicted values were based on a composite derived by Schiller (manual issue 07.1991) from the work of Knudson, Crapo, and Morris.

Data analysis. Because the majority of subjects attained different levels of PO in each of their VO₂peak tests, POmax was defined to be the higher full-minute PO of the two VO₂peak tests. The virtue of this method is that it ensured that every intraindividual comparison was made at identical PO or wattage levels. It also provided a criterion for normalizing the data into percentages of POmax.

Inherent in the fact that if a given subject accomplished two different POmax levels is that he/she would have only one set of data at 100% POmax and that there was no reference for comparison at that level. Also, in many cases there is no reference for comparison at 95% POmax. Therefore, the data were normalized with reference to POmax at 75 (heavy exercise), 80, 85, and 90% POmax (intense exercise) by interpolation.

Statistics. All data were analyzed using the SigmaStat program. Repeated measures analyses of variance were performed on the VO₂peak data, and where significant differences (P ≤ 0.05) between control and treatment (ERL) were indicated, the Student-Newman-Keuls post hoc test was performed to determine at what level(s) of POmax there was a significant (P ≤ 0.05) difference. Steady-state data were analyzed using Bonferroni’s pairwise multiple comparisons to determine whether there were significant (P ≤ 0.05) differences between control and treatment (ERL).

RESULTS

Graded VO₂peak test series comparisons. Mean test duration was 13.1 ± 0.73 min. There was slight but statistically significant EIH in control and ERL tests at 75, 80, 85, and 90% POmax (Fig. 2) compared with resting sea-level SaO₂; although SaO₂ with ERL tended to be greater, there was no significant difference in SaO₂ between control and ERL at rest or at any level of intensity. PEpeak was greater (P ≤ 0.05) with ERL at every workload (Table 2). Ve with ERL was greater (P ≤ 0.05) at 80, 85, and 90% POmax (Table 2, Fig. 3). VT was consistently greater with ERL over the comparison

| Table 2. PEpeak vs. Ve at progressive levels of exercise intensity |
|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| %POmax              | Control            | ERL                | Δ                   | Control            | ERL                | Δ                   |
| 75                  | 1.1 ± 0.3          | 1.3 ± 0.3          | 0.3 ± 0.2           | 82.6 ± 8.9         | 85.9 ± 9.5         | 3.3 ± 3.4           |
| 80                  | 1.2 ± 0.3          | 1.6 ± 0.3          | 0.4 ± 0.4           | 93.1 ± 7.1         | 97.2 ± 7.1         | 4.1 ± 4.1           |
| 85                  | 1.3 ± 0.3          | 1.9 ± 0.3          | 0.6 ± 0.4           | 104.6 ± 10.5       | 109.5 ± 10.4       | 4.9 ± 4.9           |
| 90                  | 1.8 ± 0.3          | 2.3 ± 0.3          | 0.5 ± 0.5           | 119.9 ± 12.4       | 124.4 ± 8.1        | 4.5 ± 4.5           |

Values are representative of 28 subjects. PEpeak = peak expiratory pressure; Ve, minute ventilation; POmax, maximum power output; ERL, expiratory resistive loading. *SE ± 0.113; †SE ± 3.42. ‡Significant (P ≤ 0.05) difference.
range but significantly greater (P ≤ 0.05) only at 80% PO\textsubscript{max}, where it was greater by 200 ml (Fig. 4). The increase in VT with ERL averaged 144 ml from 75 to 90% PO\textsubscript{max}. This trend toward increased VT at 80, 85, and 90% PO\textsubscript{max} in concert with relatively high f accounted for the increase in VE. The increase of VT in response to ERL in our study is consistent with the observations of previous studies (13, 24). From 75 to 90% PO\textsubscript{max}, f was unchanged. This varies from the results of previous investigators who observed decrements in f with ERL during exercise (11, 14). VT/TI was greater (P ≤ 0.05) with ERL at every workload (Fig. 5). VT/TE tended to be greater with ERL and significantly (P ≤ 0.05) greater at 75% PO\textsubscript{max}. HR was consistently lower (≥2.0 beats/min) throughout with ERL (Fig. 6) but statistically lower only at 75% PO\textsubscript{max}. Relative VO\textsubscript{2} was nonsignificantly higher (≥3.2 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) from 75 to 85% PO\textsubscript{max} with ERL and significantly greater (P ≤ 0.05) with ERL at 90% PO\textsubscript{max}. The clear tendencies for elevated VO\textsubscript{2} and lowered HR produced an O\textsubscript{2} pulse that was significantly greater with ERL at every workload (Fig. 7).

Subjects attained 1.8% greater PO\textsubscript{max} (352.0 ± 9.9 vs. 345.7 ± 9.5 W; Fig. 8) and 4.8% higher VO\textsubscript{2peak} (63.0 ± 1.4 vs. 60.1 ± 1.3 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}, both P ≤ 0.05) with ERL (Fig. 9).

Steady state at 75% PO\textsubscript{max} comparisons. Mean test duration was 17.0 ± 0.1 min. Table 3 presents the essential cardiorespiratory data, comparing control with ERL during steady-state exercise at 75% PO\textsubscript{max}. VE is somewhat higher than that observed at 75% PO\textsubscript{max} in the VO\textsubscript{2peak} studies. This disparity can be explained in part by a ventilatory drift (4.96 ± 1.35 and 4.12 ± 1.19 l/min in control and ERL, respectively) that we observed during the 4 min of cycling leading to steady state.

Breathing patterns. In the steady state and in graded maximal exercise testing, PE traces suggest that there were frequently three noticeable differences in breathing patterns between control and ERL. Several ex-

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Fig. 3. VE vs. %PO\textsubscript{max} with (■) and without ERL (control, ○). Values are means ± SE; n = 28. *Significantly (P ≤ 0.05) different from control.

Fig. 4. Tidal volume (VT) vs. %PO\textsubscript{max} with (■) and without ERL (○). Values are means ± SE; n = 28. *Significantly (P ≤ 0.05) different from control.

Fig. 5. Mean inspiratory flow (VT/TI) vs. %PO\textsubscript{max} with (■) and without ERL (○). Values are means ± SE; n = 28. *Significantly (P ≤ 0.05) different from control.

Fig. 6. Heart rate (HR) vs. %PO\textsubscript{max} with (■) and without ERL (○). Values are means ± SE; n = 28. *Significantly (P ≤ 0.05) different from control.
amples of these differences in pressure profile are shown in Fig. 10. With ERL there was 1) a sharper initial rise in \(P_E\), 2) a prolonged plateau of \(P_E\), and 3) a more abrupt decline in \(P_E\) at end expiration than in control.

**DISCUSSION**

In the range of exercise intensity of this study, it is probable that the athletes experienced various degrees of expiratory FL (19). FL over a significant portion of expiratory VT has been demonstrated over the range of intense to maximal exercise (the range of our study) and, specifically, developed at \(83\%\) of \(\dot{V}O_2\max\) in young (25 ± 1 yr) competitive elite athletes (19). In exceptionally fit older (69 ± 1 yr) athletes, this occurred at 50–75\% \(\dot{V}O_2\max\) (18). FL at lower intensities in fit, older athletes is due to loss of elastic recoil of the lung (6). With losses in elastic recoil, the static lung recoil pressure at any given lung volume is lower, so greater effort (Ppl) is required during expiration (18). In our graded \(\dot{V}O_2\max\) study, we suspect that slight ERL may have somewhat attenuated the effects of FL during heavy exercise and effected measurable increases in exercise performance and in several cardiorespiratory variables; in particular, we observed increased \(P_{Omax}\), \(V_E\), and \(O_2\) pulse, which are reported here for the first time. Also, the increase in \(V_E\) is contrary to the findings of all other loaded expiration studies (10, 11, 14, 24) of which we are aware.

Prior ERL studies. In previous ERL studies in which there were no cardiorespiratory benefits, there were three potentially limiting factors: 1) the degree of ERL employed, 2) the aerobic capacity of the subjects, and 3) the relative physical demand of the protocol, since in healthy subjects FL can occur only during strenuous exercise (3, 18, 19). One or several of these factors in previous studies may have been inappropriate to elicit cardiorespiratory benefit.

**Table 3. Respiratory variables during steady state at 75% \(\dot{V}O_2\max\)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>ERL</th>
<th>(\Delta)</th>
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<tbody>
<tr>
<td>(P_{Epeak}), cmH_2O</td>
<td>1.1 ± 0.13</td>
<td>1.5 ± 0.17</td>
<td>0.4 ± 0.01</td>
</tr>
<tr>
<td>EELV, liters</td>
<td>2.45 ± 0.16</td>
<td>3.11 ± 0.24</td>
<td>0.67 ± 0.29*</td>
</tr>
<tr>
<td>EILV, liters</td>
<td>5.22 ± 0.22</td>
<td>5.92 ± 0.28</td>
<td>0.70 ± 0.28*</td>
</tr>
<tr>
<td>IRV, liters</td>
<td>1.95 ± 0.27</td>
<td>1.25 ± 0.17</td>
<td>-0.70 ± 0.28*</td>
</tr>
<tr>
<td>(V_E), l/min</td>
<td>98.7 ± 2.9</td>
<td>105.9 ± 4.4</td>
<td>7.2 ± 2.9</td>
</tr>
<tr>
<td>(V_T/TE), l/s</td>
<td>4.42 ± 0.23</td>
<td>4.98 ± 0.36</td>
<td>0.56 ± 0.25</td>
</tr>
<tr>
<td>(V_T/TI)</td>
<td>2.64 ± 0.06</td>
<td>2.76 ± 0.09</td>
<td>0.12 ± 0.06</td>
</tr>
<tr>
<td>(V_T), ml</td>
<td>2778 ± 110.5</td>
<td>2808 ± 127.8</td>
<td>30.3 ± 61.1</td>
</tr>
<tr>
<td>(f), breaths/min</td>
<td>36.01 ± 1.62</td>
<td>38.24 ± 1.86</td>
<td>2.22 ± 10.0</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>154.8 ± 3.7</td>
<td>156.0 ± 4.7</td>
<td>1.2 ± 1.6</td>
</tr>
<tr>
<td>(V_{O2}), ml·kg⁻¹·min⁻¹</td>
<td>52.0 ± 1.1</td>
<td>52.8 ± 1.4</td>
<td>0.8 ± 0.7</td>
</tr>
<tr>
<td>(T_E), s</td>
<td>0.65 ± 0.04</td>
<td>0.59 ± 0.04</td>
<td>-0.06 ± 0.04</td>
</tr>
<tr>
<td>(T_i), s</td>
<td>1.11</td>
<td>1.01</td>
<td>-0.10</td>
</tr>
<tr>
<td>(T_i/T_T)</td>
<td>0.66</td>
<td>0.64</td>
<td>-0.02</td>
</tr>
</tbody>
</table>

Values are means ± SE; \(n = 12\). EELV, end-expiratory lung volume; EILV, end-inspiratory lung volume; IRV, inspiratory reserve volume; \(V_T/TE\), mean expiratory flow; \(V_T/TI\), mean inspiratory flow; \(f\), breathing frequency; \(V_T\), tidal volume; HR, heart rate; \(V_{O2}\), relative \(O_2\) consumption; \(T_E\), time of expiration; \(T_i\), time of inspiration; \(T_i/T_T\), duty cycle. *Significantly different (\(P < 0.05\), Bonferroni’s pairwise multiple comparisons).
First, the ERL employed in previous studies appears to have been overbearing, having ranged from 5.0 to 40.0 cmH₂O (8, 11, 22, 24). Second, to our knowledge, there has not been a study employing ERL in which the subjects have had superior aerobic capacity. Typically, the subjects’ mean VO₂max values have been 40 ml·kg⁻¹·min⁻¹ (8, 22). Healthy subjects in these ranges of aerobic capacity would not place the ventilatory demand on the pulmonary system, which would precipitate FL (19). The third factor precluding cardiorespiratory benefit in response to ERL is that tests have been resting tests or low-intensity submaximal exercise tests. Such protocols would not precipitate FL, even in highly fit subjects (19).

Respiratory responses to ERL. Neither Ti nor Te changed in response to ERL. Previous investigators have observed an increase in Ti and Te (24) or no change in Ti or Te with ERL (7). A prolongation of Te observed by Goldstein and co-workers (8) was proportional to the amount of ERL, which ranged from 5.0 to 30.0 cmH₂O, far more than the ERL employed in the present study.

EELV typically increases in response to expiratory loading (7, 8, 11, 22), increasing up to 1.71 ± 0.67 liters in response to extreme ERL (7). Not unexpectedly, in the steady-state (75% POmax) comparisons, EELV was increased with ERL. However, what was surprising, in view of previous studies employing ERL, was the magnitude of increase in EELV (0.67 ± 0.29 liter) in response to so little increase in P_{E, peak} (0.4 ± 0.1 cmH₂O) with ERL vs. control. Johnson and colleagues (20) observed a consistently slightly higher EELV (120 ± 10 ml, P < 0.01) with the two-tracer-gas method than with the He-dilution method employed in this study. This disparity was greatest during heavy exercise: 158 ± 40 and 209 ± 24 ml with equilibration times of 7 ± 1 and 20 s, respectively. Because this consistently slight and directional error would have been common to control and ERL trials in our study, we surmise that the observed differences in EELV with ERL were not significantly affected by the He method.

P_{E, peak} may have been a bit lower in steady state than in the graded exercise tests (which increased 10 W/min), because by the 4th min at 75% POmax a more comfortable breathing pattern had been established, with a little less emphasis on the initial expiratory effort.

The cumulative total resistance of the respiratory system is ~3 cmH₂O·l⁻¹·s, with resistance being somewhat greater during expiration than inspiration (4). The majority of this resistance is offered by the upper airways and, in particular, by the glottis (8). Normally, a great deal of expiratory glottal resistance is progres-

Fig. 10. Single-breath expiratory pressure at mouth (P_{E}) vs. time without (A) and with ERL (B). Values ≥ 0.0 cmH₂O are expiratory pressure tracings; values ≤ 0.0 cmH₂O are incomplete inspiratory pressure tracings. Max, maximum.
sively lost during graded exercise in response to increased airflow rates (26). This phenomenon has been observed during hyperventilation (15) and exercise hyperpnea (4). In our study the aperture chosen for ERL created increasingly higher PE\textsubscript{peak} (Fig. 1), inasmuch as glottal braking presumably diminished with increasing V\textsubscript{E} (15).

The ERL presented during the steady-state exercise at 75% PO\textsubscript{max} apparently offered sufficient stimulus to shift EELV upward and away from the FL, which occurs during heavy to maximal exercise. VT/TE was unchanged with ERL, in contrast to previous studies in which reductions in VT/TE were observed with ERL (22, 24). Although these larger ERL values (5 and 8 cmH\textsubscript{2}O, respectively) did increase EELV, their thwarting effects on expiratory flow probably contributed to a reduced V\textsubscript{E}. The ERL employed here at steady state (1.5 ± 0.2 cmH\textsubscript{2}O) represents quite modest ERL compared with that utilized in prior studies (7, 10, 11, 14, 22, 24) and did not affect VC adversely. In two of these studies, ERL and VC were inversely related (10, 24).

Johnson and colleagues (19) suggested that exerting greater effort earlier in the expiratory phase (when airflow is still effort dependent) would be one strategy by which to mitigate FL. With ERL, this strategy appears to be at work, as observed in the sharp rise in expiratory pressure at the commencement of the expiratory phase in the single expiratory breath tracings (Fig. 10). Also, there was a pattern of prolonged elevation (plateau) of Pe\textsubscript{w} with ERL (Fig. 10). This may have served to distend the airways and may have effectively prolonged the effort-dependent stage of expiration as well.

V\textsubscript{O\textsubscript{2peak}} test series. In addition to the normal expansion of VT to a plateau of 50–60% of VC (30) with graded exercise, there is a further increase in VT with ERL (14, 24). Throughout our comparison V\textsubscript{O\textsubscript{2peak}} tests, VT was greater and significantly greater (P ≤ 0.05) at 80% PO\textsubscript{max} with ERL vs. control (Fig. 4), with little or no change in F with ERL. Further expansion in VT, after it has reached a plateau, is limited by FL, as described by the descending profile/envelope of the MEFV curve (3), and is relatively fixed for a given EELV (12). Because we did observe increases in VT in response to ERL, we infer that FL was ameliorated.

Because in these graded V\textsubscript{O\textsubscript{2peak}} tests we employed the same resistor used in the EELV series, we presumed that there had been an increase in EELV in response to ERL in the graded V\textsubscript{O\textsubscript{2peak}} series and that the increased VT (Table 3) was correspondingly shifted to higher lung volumes. We believe that this evident increase in VT due to the probable increase in EELV explains the significantly increased V\textsubscript{E} at 80, 85, and 90% PO\textsubscript{max} (Fig. 3). In the steady-state comparisons, VT was not significantly greater with ERL. We suspect that at this level of intensity, i.e., 75% PO\textsubscript{max}, FL had not yet become a problem. As mentioned above, in the V\textsubscript{O\textsubscript{2peak}} tests, VT was significantly greater.

ERL provokes greater inspiratory effort (10, 11). The increased end-inspiratory lung volume (Table 3) offers circumstantial evidence of increased inspiratory effort with ERL. However, opting to do more inspiratory work appears to be a beneficial trade-off for our athletes. Although breathing at hyperinflated lung volumes may predispose the inspiratory muscles to fatigue (3), in our study, it does not appear that ERL presented any such liability. Fatigability of respiratory muscles has been shown to be a function of the percentage of respiratory effort expended vs. the maximal capacity for muscular effort (29). Our highly trained endurance athletes very likely had developed superior respiratory muscle strength, so that absolute increases in respiratory muscle effort would represent smaller percentages of maximum capacity for muscular effort, making them less susceptible to respiratory muscle fatigue than less fit subjects (9).

The increase in end-inspiratory lung volume with ERL to 77.9 ± 2.9% VC vs. 66.3 ± 4.2% VC (control) would not appear to have required a great increase in inspiratory effort, since 77.9% VC is still within the range of optimum compliance (i.e., 20–80% VC), i.e., within the range in which the elastic work of breathing is at a minimum (30). With ERL there still remained 1.25 ± 0.17 liters of inspiratory reserve volume (Table 3). We surmise that the observed increases in V\textsubscript{E} associated with ERL were not at the expense of extraordinary inspiratory effort but rather more probably due to a moderate and manageable increase in inspiratory effort. Our subjects did not complain of increased work of breathing with ERL. In fact, anecdotally, many of the athletes in our study, without solicitation, commented that "it felt easier" or "I felt stronger this time (or last time)," unknowingly referring to the ERL test. It is also likely that the increase in EELV with ERL reduced the energy requirement for expiration. With ERL, EELV was elevated from 16.7 ± 2.9 to 28.0 ± 3.4% VC and to a more favorable position on the compliance curve.

There was a nonsignificant increase in V\textsubscript{O\textsubscript{2}} with ERL from 75 to 85% PO\textsubscript{max}, which became significant (P ≤ 0.05) at 90% PO\textsubscript{max}. A partial explanation for this elevated V\textsubscript{O\textsubscript{2}} during ERL would be the increased energy requirement to perform increased inspiratory work. However, the athletes’ performances were not compromised by this increased energy demand, and it would appear that some fraction of the increased V\textsubscript{O\textsubscript{2peak}} observed with ERL was delivered to the working skeletal muscle, which produced (P ≤ 0.05) greater PO\textsubscript{max} (Fig. 9).

Effective P\textsubscript{pl}. When exercising athletes reach FL, P\textsubscript{pl} has met or exceeded effective P\textsubscript{pl} (Peff), which is the minimal pressure to drive maximal expiratory flow, as described by isovolume pressure-volume curves (1). Exceeding Peff represents a waste of energy and can lead to a decrease in expiratory flow from dynamic compression of airways (21). However, during breathing at elevated lung volumes, as the athletes in our study did with ERL, the Peff is raised (2), and this can move the entire VT away from the flow-limiting pressures reached at lower lung volumes (3), thereby enabling the athlete to exert more effective expiratory pressure. Ordinarily, only a small fraction of maximum expiratory effort is required to generate maximum expiratory flow (29). In fact, it is estimated that at
>40% of maximal expiratory effort, there is no further effect on expiratory ventilation (29). At heavy workloads \( (V_e \geq 120 \text{ l/min}) \) the cost of breathing increases markedly because of the expiratory work done against FL (17). In our study the strategy/response to ERL may have been to effectively divert effort from "wasted" expiratory work, where expiratory Ppl > Peff to greater inspiratory activity. With this strategy, FL may have been prevented and, consequently, Ve increased.

Breathing at elevated EELV in response to ERL had significant cardiovascular implications as well. In the graded \( V_{O2\text{peak}} \) comparisons, \( O_2 \) pulse was greater (P \( \leq 0.05 \)) from 75 to 90% PO\(_{max}\) (Fig. 7), yet \( S_aO_2 \) was only \( \sim 1\% \) greater with ERL (Fig. 1). Because there is little reason to suspect that ERL caused decreased mixed venous \( O_2 \) content and because these comparisons with and without ERL were made at identical workloads, increased \( O_2 \) pulse suggests increased stroke volume, and without ERL were made at identical workloads, increased \( O_2 \) pulse suggests increased stroke volume, probably secondary to augmented venous return with ERL. Besides the deleterious ventilatory effects of having Ppl exceed Peff, excessive Ppl, which is likely to occur in very strenuous exercise (3, 18, 19), can impede venous return (25). Furthermore, elevated EELV, as was observed in our study in response to ERL, is associated with decreased pulmonary vascular resistance (27). We suggest that increased stroke volume, in concert with increased Ve, very likely contributed to the increases in \( V_{O2\text{peak}} \) and PO\(_{max}\).

In conclusion, we infer from our findings that increased EELV in response to slight ERL during strenuous exercise served to attenuate airflow and blood flow limitations, which otherwise may have been the product of excessive Ppl developed during active expiration at lower lung volumes. We further conclude that appropriate ERL during strenuous exercise can enhance ventilatory and exercise capacity.

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