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CARDIORESPIRATORY RESPONSES TO SLIGHT EXPIRATORY RESISTIVE
LOADING DURING STRENIOUS EXERCISE AT SEA LEVEL

A DISSERTATION SUBMITTED TO THE GRADUATE DIVISION OF THE
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By

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I would like to take this opportunity to thank the following friends, without whose generous support, this project would not have been possible.

Thank you, Richard Smith, my good friend and mentor, for your wit and wisdom. And, thank you for allowing me to draw on your vast knowledge of pulmonary physiology. Thank you for your timely editing and your pertinent recommendations. Those great conversations really made all the hard work fun. Thanks, also, to my dissertation committee members: Dave Lally, John Claybaugh, Dave Jameson, and Jon Pegg, whose time and advice were invaluable and quite appreciated; to Causey Whittow, whose sage recommendation to, "Do a little bit every day, Larry," really helped me to get the job underway; to Dr. Hanna, whose statistical advice was invaluable; to Charles Matsuda, our benign department chair, at Kapi'olani Community College, who allowed me to conduct my research here at KCC; to Ron Dunn, whose technical assistance from the office next-door was indispensable; to Jeff Hall, my indulgent office mate, who spent months on end, crowded by equipment, into a corner, thank you, Buddy; to Stephen Wehrman and Ken Mito, those
generous Merlins, who opened their respiratory storage room to me "carte blanche;" to my stalwart subjects, especially Cliff Riggsbee, the "Ironman," who used his considerable influence to help me gather the premier endurance athletes of Hawaii for this study; to Rick Greco, my old lifeguard buddy, who visited just long enough to help me establish the protocols and to become the "desaturation king;" to Mr. and Mrs. Robert and Peggy Kodoma Dinman, whose gracious scholarship helped make scholastic life a little smoother; to Charles Daniels, who has been my guardian angel (as Dot Fee called him) and chief counselor these past five years in Hawaii; to Michael English, my able assistant, whose tireless effort and bottomless coffee pot kept the project well oiled; to Joan Cebrick, who was the spark for this dream; and to Dot Fee and Leo Fee, for their unflagging loyalty and encouragement. I sure wish you were here for this!
ABSTRACT

At sea level we determined the cardiorespiratory and performance effects of a slight expiratory resistive load (ERL) which, at mild altitude, had been effective in mitigating exercise-induced hypoxemia (EIH). Paired \( \text{VO}_2\text{max} \) bicycle ergometer tests, (ERL vs control) were performed by 28 highly-fit (\( \text{VO}_2\text{max} = 63.4 \pm 1.36 \)) athletes (age = 33.5 ± 7.3).

There was significant EIH in both control and ERL tests at 75, 80, 85, and 90% maximum power output (\( \text{PO}_{\text{max}} \)) and at \( \text{VO}_2\text{max} \) when compared to resting \( \text{SaO}_2 \); but no difference in \( \text{SaO}_2 \) between control and ERL at any level of intensity. Peak-expiratory mouth pressures (\( \text{P}_{\text{ao}} \)) were greater (\( p<0.05 \)) with ERL at 75, 80, 85, 90 % \( \text{PO}_{\text{max}} \) and \( \text{VO}_2\text{max} \); with ERL, \( \text{P}_{\text{ao}} \) was increased compared to control by 0.20, 0.35, 0.41, 0.49, and 0.73 cm H\(_2\)O, respectively. Concomitantly, minute ventilation (\( \text{V}_E \)) was greater with ERL vs control (\( p<0.05 \)) by 4.1, 4.9, 4.5, and 5.8 L min\(^{-1} \) at 80, 85, 90 % \( \text{PO}_{\text{max}} \) and \( \text{VO}_2\text{max} \). The increase in \( \text{V}_E \) was largely due to a trend
toward increased tidal volume ($\Delta \text{ml} = 121, 198, 154, \text{and} 103, \text{from} 75 \text{to} 90 \% \text{PO}_{\text{max}}$). There was a small, non-significant increase in $\text{VO}_2$ (averaging $3.0 \text{ml}\cdot\text{kg}^{-1}\cdot\text{sec}^{-1}$) with ERL from 75-90 \% \text{PO}_{\text{max}}. With ERL, heart rate (HR) was consistently lower ($\geq 2.0 \text{BPM}$) throughout, although not significantly so. However, $\text{O}_2$ pulse ($\text{VO}_2$/HR) was significantly greater with ERL by 9.5, 9.0, 7.9, 7.1 and 7.0 \% at 75, 80, 85, 90 \% \text{PO}_{\text{max}} \text{and} \text{VO}_2_{\text{max}}$. With ERL, athletes attained greater ($p \leq 0.05$) $\text{PO}_{\text{max}} = 352.0 \pm 9.9 \text{vs} 345.7 \pm 9.5 \text{watts}$, and higher ($p \leq 0.05$) $\text{VO}_2_{\text{max}} = 63.4 \pm 1.36 \text{vs} 60.3 \pm 1.26 \text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$.

Subsequently, in a subset ($n = 12$), FRC was determined by He-dilution during steady-state 75 \% \text{PO}_{\text{max}}. With ERL, $P_{\text{ao}}, V_E$, and $V_T/T_e$ were greater ($p \leq 0.05$) and FRC was elevated ($p \leq 0.05$) 0.67 $\pm$ 0.29 L. End-inspiratory lung volume (EILV) with ERL was greater than control ($p \leq 0.05$), 82.2 $\pm$ 1.1 vs 72.5 $\pm$ 1.3 \% of TLC, respectively.

We conclude that during heavy to intense exercise, surprisingly small ERL (ranging from 1.27 to 2.94 cm $\text{H}_2\text{O}$) may cause $V_T$ to
increase and FRC to shift upward, reducing the potential for airflow limitation. That $O_2$ pulse was higher with ERL at workloads identical to that of control, would suggest that venous return was augmented, perhaps in response to reduced pulmonary vascular resistance associated with increased FRC. The significantly improved $VO_{2\text{max}}$ with ERL may be due in part to the increased inspiratory work of breathing that exercising at elevated FRCs may entail; however, it would appear that some fraction of the increased $VO_{2\text{max}}$ observed with ERL was delivered to the working skeletal muscle which produced significantly greater $PO_{\text{max}}$. We conclude that during strenuous exercise, slight expiratory resistive loading enhances performance in highly-trained athletes.
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</tr>
<tr>
<td>CPAP</td>
<td>continuous positive airway pressure</td>
</tr>
<tr>
<td>CPEP</td>
<td>continuous positive expiratory pressure</td>
</tr>
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<td>EELV</td>
<td>end-expiratory lung volume, i.e., dynamic FRC</td>
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<td>EIH</td>
<td>exercise-induced hypoxemia</td>
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<td>EILV</td>
<td>end-inspiratory lung volume</td>
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<td>ERL</td>
<td>expiratory resistive loading</td>
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<td>FEV&lt;sub&gt;1.0&lt;/sub&gt;</td>
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<td>IVPF</td>
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<td>IRV</td>
<td>inspiratory reserve volume</td>
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<td>MEFV</td>
<td>maximal expiratory flow-volume (curve)</td>
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<td>P&lt;sub&gt;ao&lt;/sub&gt;</td>
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$P_E$  
expiratory pressure

PEEP  
positive end-expiratory pressure

$P_{eff}$  
effective pleural pressure ($P_{pl}$ at max flow)

PLB  
pursed lips breathing

$P_{max}$  
maximum power output

$P_{pl}$  
pleural pressure

P-V  
pressure-volume

R  
respiratory exchange ratio ($VCO_2/VO_2$)

RV  
residual volume

$SaO_2$  
artrial oxygen saturation

TLC  
total lung capacity

$V_{A/Q_c}$  
alveolar ventilation to perfusion ratio

VC  
vital capacity

$V_E$  
minute ventilation

$V_{eq O2}$  
$V_E/VO_2$

$V_{eq CO2}$  
$V_E/VCO_2$

$VO_2$  
oxygen consumption

$VCO_2$  
carbon dioxide production
\( \text{VO}_{2\text{max}} \) maximal aerobic capacity/oxygen consumption

\( V_s \) spirometer volume

\( V_T \) tidal volume

COPD chronic obstructive pulmonary disease

CPAP continuous positive airway pressure

CPEP continuous positive expiratory pressure

EELV end-expiratory lung volume, i.e., dynamic FRC

EIH exercise-induced hypoxemia

EILV end-inspiratory lung volume

ERL expiratory resistive loading

\( \text{FEV}_{1.0} \) forced expiratory volume (1.0 sec)

FL flow limitation

FRC functional residual capacity

F-V flow-volume

FVC forced vital capacity

\( [\text{He}_i] \) initial concentration of helium

\( [\text{He}_f] \) final concentration of helium

IVPF isovolume pressure-flow (curve)

IRV inspiratory reserve volume

**xx**
<table>
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<tr>
<th>Abbreviation</th>
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<tr>
<td>MEFV</td>
<td>maximal expiratory flow-volume (curve)</td>
</tr>
<tr>
<td>P_{ao}</td>
<td>peak-expiratory mouth pressure</td>
</tr>
<tr>
<td>P_E</td>
<td>expiratory pressure</td>
</tr>
<tr>
<td>PEEP</td>
<td>positive end-expiratory pressure</td>
</tr>
<tr>
<td>P_{eff}</td>
<td>effective pleural pressure (P_{pl} at max flow)</td>
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<tr>
<td>PLB</td>
<td>pursed lips breathing</td>
</tr>
<tr>
<td>P_{O_{max}}</td>
<td>maximum power output</td>
</tr>
<tr>
<td>P_{pl}</td>
<td>pleural pressure</td>
</tr>
<tr>
<td>P-V</td>
<td>pressure-volume</td>
</tr>
<tr>
<td>R</td>
<td>respiratory exchange ratio (VCO_2/VO_2)</td>
</tr>
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<td>RV</td>
<td>residual volume</td>
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<tr>
<td>SaO_2</td>
<td>arterial oxygen saturation</td>
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<td>TLC</td>
<td>total lung capacity</td>
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<td>V_{A/Q_c}</td>
<td>alveolar ventilation to perfusion ratio</td>
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<td>VC</td>
<td>vital capacity</td>
</tr>
<tr>
<td>V_E</td>
<td>minute ventilation</td>
</tr>
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<td>V_{eq O_2}</td>
<td>V_E/VO_2</td>
</tr>
<tr>
<td>V_{eq CO_2}</td>
<td>V_E/VCO_2</td>
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\( \text{VO}_2 \) oxygen consumption
\( \text{VCO}_2 \) carbon dioxide production
\( \text{VO}_{2\text{max}} \) maximal aerobic capacity/oxygen consumption
\( V_{sp} \) spirometer volume
\( V_T \) tidal volume
CHAPTER I

INTRODUCTION

"A remarkable feature of the maximum expiratory flow-volume envelope is that it is virtually impossible to penetrate it."
(John B. West, Respiratory Physiology.)

"Conscious attempts to modify breathing during general physical activities, such as running, are usually doomed to failure and probably are of no benefit in terms of performance. In fact, conscious manipulation of breathing would be detrimental to the exquisitely regulated physiological adjustments to exercise."
(McArdle, Katch, and Katch, Exercise Physiology.)

I. Exercise-Induced Hypoxemia (EIH)

A. Previous Studies of EIH

Airflow limitation and hypoventilation, both due to the mechanical constraints of the pulmonary system, have been prominently implicated in EIH (Bye et al., 1983). Our interest in EIH began with our asking the question, is there any physiological benefit to controlling expiratory airflow, as athletes are wont to do
during heavy exercise. Does control of expiration affect, either positively or negatively, airflow limitation? A search of the literature revealed that many studies (which will be enumerated presently) had been conducted in which expiratory resistive loading (ERL) was employed during exercise; however, there is not a single study in which ERL has produced cardiorespiratory benefit in healthy, exercising subjects. ERL, in the form of pursed lips breathing (PLB), has proven effective in mitigating hypoxemia associated with chronic obstructive pulmonary diseases (COPD), such as emphysema. In COPD patients, PLB is also instrumental in increasing minute ventilation ($V_E$), tidal volume ($V_T$), and decreasing breathing frequency (BF), and arterial carbon dioxide ($PaCO_2$) (Mueller et al., 1970). We felt that there may be some parallel between the extraordinary expiratory efforts of COPD and those of highly-fit endurance athletes who are able to elicit EIH during strenuous exercise. At moderate altitude (1520 m), we were able to mitigate EIH by slightly restricting expiratory airflow in highly-fit endurance athletes during strenuous treadmill running.
Exercise-induced arterial oxygen desaturation, or hypoxemia, (EIH) at altitudes above sea level has been demonstrated in both athletes (Squires and Buskirk, 1982) and nonathletes (Maresh et al., 1983). Desaturation may be greater, and may be demonstrated at lower oxygen consumptions (VO$_2$) in athletes (Saltin, 1967). There is also a direct relationship between an individual's maximal oxygen consumption (VO$_{2\text{max}}$) and his/her ability to desaturate (Williams et al., 1986). Tucker and co-workers (1984), at 1520 m, found that during heavy to maximal exercise testing, arterial oxygen saturation (% SaO$_2$) decreased from 93.3 to 84.5 % toward the end of maximal runs. These measurements of % SaO$_2$ are consistent with the control values at moderate altitude (1520m) (Fee et al., 1992; McClaran et al., 1992). In the latter studies, ERL during graded treadmill testing to VO$_{2\text{max}}$ was compared to conventional exercise-testing conditions (control). Slight ERL mitigated EIH in highly-fit athletes during strenuous exercise, in both studies, significantly improving the % SaO$_2$ over a wide range.
of percentages of \( VO_{2\max} \). At \( VO_{2\max} \), Fee et al. (1992) found that ERL was associated with % a \( SaO_2 \) values of 89.7 ± 1.12 vs 86.6 ± 1.25 % (control); similarly, McClaran et al. observed 87.1 ± 1.1 (ERL) vs 84.9 ± 1.3 % \( SaO_2 \) (control). In both of these studies, utilization of the Hans Rudolph low-resistance three-way breathing valve, which is conventionally used in exercise testing, represented "control," whereas, ERL was effected by reducing the internal diameter of the expiratory port from the standard 28.6 to 22.2 mm with a washer (I.D. = 22.2 mm).

B. EIH Schema

A number of investigators have been successful in partitioning the several physiologic mechanisms responsible for exercise-induced hypoxemia (EIH), or \( O_2 \) desaturation, in superior endurance athletes performing at very heavy workloads. These mechanisms include: 1) insufficient erythrocyte transit time through the pulmonary capillary (Bye et al., 1983; Dempsey et al., 1982; Dempsey et al., 1984; Powers and Williams, 1987), 2) alveolar ventilation to
perfusion ($V_A/Q_c$) mismatch (Bayly et al., 1983; Gledhill et al., 1977; Wagner, 1992), 3) shunting (Lilienthal et al., 1946; Wagner, 1977), 4) interstitial edema, especially at altitude (Maron et al., 1979; Younes and Burks, 1985), 5) respiratory muscle fatigue (Roussos and Macklem, 1977) and 6) hypoventilation. The latter (hypoventilation) may be due to inappropriately low levels of ventilation in the face of hypercapnia and metabolic acidosis (Dempsey et al., 1984; Johnson et al., 1992) and/or air flow limitation (FL), due to the mechanical constraints of the pulmonary system (Bye et al., 1983). Bye et al. (1983) have suggested that the most important determinant is FL, and a factor which may prevent further compensatory hyperventilation (Bye et al., 1983; Dempsey et al., 1984).

C. Exacerbation of ELH at Altitude

In a study by Gale and coworkers (1985), $V_A/Q_c$ mismatch increased during heavy exercise at moderate altitude (1524 m) when compared to sea level, and the authors suggested that this
mismatch was the consequence of nonuniform hypoxic pulmonary vasoconstriction (Gale et al., 1985). Wagner (1992) attributes $V_A/Q_c$ mismatch during heavy exercise to interstitial pulmonary edema. With the considerable rise in pulmonary arterial pressure which accompanies heavy exercise (Bevegard et al., 1960), the potential for interstitial pulmonary edema increases (Wagner and Gale, 1991), a problem which is not only accentuated at altitude (Hultgren, 1978), but perhaps unique to altitude (Brower and Permutt, 1991).

Additionally, at the moderate altitude (1520 m) of the previous EIH studies (Fee et al., 1992; McClaran et al., 1992) the barometric pressure ($P_{Bar}$) was approximately 635 Torr. This reduced the inspired $O_2$ from a sea level value of 149 to 123 Torr. This would certainly contribute to impaired oxygen transfer across the alveolar-capillary membrane, which has been proposed as a mechanism for EIH (Tucker et al., 1984). Studies with ERL similar to Fee's et al., (1992) and McClaran's et al., (1992) have not been repeated at sea level, so the relative contributions of altitude and ERL in mitigating hypoxemia cannot be assessed.
II. Respiratory Flow Limitation in Strenuous Exercise

Aside from altitude, Bye et al. (1983) have proposed that the most important determinant in the precipitation of EIH is FL. FL sets limits on exercise by limiting ventilation to relatively low levels (Bye et al., 1983). The mechanical constraint imposed by FL exerts a limit on $V_E$ during heavy to maximal exercise (Jensen et al., 1980). FL during exercise hyperpnea occurs when portions of the expiratory tidal flow-volume (FV) envelope become tangent to the flow-limiting (effort-independent) slope of the maximal expiratory flow-volume (MEFV) curve (Johnson et al., 1991). FL over a significant portion of expiratory $V_T$ flow has been demonstrated over the range of intense-to-maximal exercise, that is, developing at $\geq 83\%$ of maximal aerobic capacity ($VO_{2\text{max}}$) in young (21 ± 1 yr) competitive endurance athletes (Johnson et al., 1992). In exceptionally fit older (69 ± 1 yr) athletes this occurs in the range of 50 to 75 % $VO_{2\text{max}}$ (Johnson et al., 1991). FL at lower intensities in fit, older athletes is due to loss of elastic recoil of the lung (Frank et al., 1957).
A. Expiration Pressures during Exercise

When exercising athletes reach FL, pleural pressure ($P_{pl}$) has met or exceeded effective pleural pressure ($P_{eff}$), that is, the minimal pressure to drive maximal expiratory flow; $P_{eff}$ is the pleural pressure at which expiratory flow reaches a plateau on the isovolume pressure-flow (IVPF) curve (Beck, 1991). Exceeding $P_{eff}$ represents a waste of energy, and can lead to a decrease in expiratory flow from compression of airways (Ingram and Schilder, 1966; Mead et al., 1967). In fact, it is estimated that at greater than 40% of maximal expiratory effort, there is no further increase in expiratory ventilation (i.e., $P_{eff}$ is < 40% maximal expiratory effort) (Younes, 1991). Normal, healthy subjects rarely exceed $P_{eff}$ during exercise hyperpnea (Beck et al., 1991; Olafsson and Hyatt, 1969); however, very fit athletes, regardless of age (Johnson et al., 1991; Johnson et al., 1992), often do go beyond $P_{eff}$ during strenuous exercise.
B. Relationship between Flow Limitation and End-Expiratory Lung Volume (EELV)

i. Lung Volume Excursions during Exercise

EELV has been extensively studied during exercise and the weight of evidence has supported the conclusion that in healthy, young subjects (Younes, 1991) EELV (or functional residual capacity, FRC) decreased on average 0.7 L in response to exercise (Henke et al., 1988; Kagawa and Kerr, 1970; Kiers et al., 1980; Klas and Dempsey, 1989), decreased even at very low exercise intensity (Lind and Hesser, 1984, Henke et al., 1988), and remained depressed until exercise became intense. These reductions in EELV are the result of increased active expiration (Henke et al., 1988). During heavy exercise (200-300 W), decreases in EELV have been observed to average 0.8 L (Henke et al., 1988; Sharratt et al., 1987). EELV gradually returns to near-resting FRC only at or near VO_{2max} (Johnson et al., 1991; Johnson et al., 1993). Despite the strategy that returns FRC to near resting values, a progressively greater
portion of the $V_T$ becomes flow limited as exercise becomes more intense (Johnson et al., 1991; Johnson et al., 1992).

ii. Flow Limitation at Lower Lung Volumes

One circumstance which affects the development of FL is that FRC or EELV changes with exercise (see above). When EELV is lowered, $V_T$ is shifted closer to the MEFV envelope (Bye et al., 1983), increasing the potential for FL and reducing the maximal predicted minute ventilation ($V_{E_{\text{max}}}$) (Beck et al., 1991). Therefore, the critical determinant of $V_{E_{\text{max}}}$ becomes the placement of $V_T$ with respect to EELV (Beck et al., 1991). At lower lung volumes, the upper limit of ventilation is reduced because of lower maximal flows (Jensen et al., 1980).

Exercise-induced reductions in FRC caused decreased dynamic lung compliance ($C_{\text{dyn}}$) (decreases of 10-60 % of resting levels) in moderate to heavy exercise (Gilbert and Auchincloss, 1969; Henke et al., 1988; Younes and Kivinen, 1984). This reduction in $C_{\text{dyn}}$ may have been due to displacement of the lung to the lower, alinear
portion of pressure-volume curve (Henke et al., 1988). Reduced EELV during heavy exercise increases the likelihood of airway narrowing and/or closure in the dependent regions of the lung (Engle, 1986), which may contribute to the nonuniformity of inspiratory gas flow distribution (Henke et al., 1988). At low lung volumes, airway closure during expiration has been demonstrated to occur in short lengths (2-3 cm) of the trachea; while at higher lung volumes these "choke points" move upstream to the lobar or segmental bronchi (Smaldone and Bergofski, 1976). During graded exercise, this nonuniformity contributes significantly to the widening alveolar-arterial O₂ gradient (P[A-a] O₂) (Gledhill et al., 1978; Torre-Bueno et al., 1985), which has ranged from 20-30 Torr in average subjects, and up to 40 Torr in elite athletes when at or near VO₂max (Dempsey et al., 1985). Rising alveolar to arterial oxygen gradient (P[A-a]O₂) indicates that gas exchange inefficiency has developed (Wagner, 1992).

Alternatively, Henke et al., (1988) have noted that breathing from a reduced FRC theoretically assists the diaphragm at the onset
of inspiration. They also suggest that the near-optimal position of the diaphragm in generating force is below resting FRC.

iii. Respiratory Responses to Progressive Flow Limitation

Pellegrino and colleagues (1993) substantiated a direct correlation between FL and increases in EELV. That is, as FL begins to become pronounced, EELV moves upward in the direction of resting FRC. They conjectured that dynamic airway compression may elicit a reflex mechanism which causes expiration to end prematurely. Garrard and Lane (1977) have also observed a linear relationship between the degree of expiratory obstruction to airflow and EELV.

The reflex elevation of EELV that develops as exercise intensity approaches its maximum evolves only in response to severe FL. When FL is less than severe, there is virtually no elevation in EELV. This phenomenon was observed in two separate athlete groups. FL was experienced in both groups, comprising around 24 % of $V_T$ in the younger (25 ± 1 yr) group of competitive elite athletes and FL
occurred at \( \geq 83 \% \) \( V_{O2\text{max}} \) (Johnson et al., 1992). In an older (69 ± 1 yr) group of exceptionally fit athletes, this degree of FL was observed from 50 to 75 \% \( V_{O2\text{max}} \) (Johnson et al., 1991). Despite this obvious limitation to ventilation, EELV remained well below resting level. FL seems to be tolerated or undetected until it becomes extreme, namely, at \( V_{O2\text{max}} \), at which point 42 \% and 61 \% (younger vs older, respectively) of \( V_T \) met the limit imposed by the effort-independent segment of the MEFV curve (Johnson et al., 1992). It was only when faced with severe FL that EELV returned to near (although still below) resting FRC levels (Johnson et al., 1991; Johnson et al., 1992).

C. Available Strategies for Averting Exercise-Related FL

Shifting EELV upward and away from the flow-limiting constraints of the MEFV curve, to a position where maximal expiratory flows are greater (Beck et al., 1991) and/or exerting greater expiratory pressure earlier (Johnson et al., 1992) (see discussion), before \( V_T \) flow falls into the effort-independent slope of the MEFV
curve (West, 1986) are the two proposed strategies available to avert FL in exercise.

i. Flow Limitation and Hyperinflation

Since hypothetically, EELV could be elevated to a volume at which the expiratory flow remains entirely within the MEFV curve (Beck et al., 1991) and away from the flow-limiting pressures reached at lower lung volumes (Johnson et al., 1992). One strategy to avert FL would be to move the entire $V_T$ away from the flow-limiting pressures reached at lower lung volumes (Johnson et al., 1992). When EELV is isolated as the sole factor determining the maximum amount of ventilation ($V_{E_{max}}$) that can be generated, there is a direct correlation between EELV and $V_{E_{max}}$ (Beck et al., 1991). When EELV increases, $V_{E_{max}}$ increases.

At higher lung volumes, airways in the lower (dependent) regions, which are susceptible to dynamic compression of the airways and begin to undergo FL earliest, are better ventilated...
(Hyatt et al., 1973), thus improving the V/Q relationship, and improving gas exchange (Wagner, 1992).

As can be determined from the isovolume pressure-flow (IVPF) curves of Bouhuys and Jonson (1967), increased EELV brings about a concomitant increase in $P_{eff}$, so that expiratory pleural pressures, which at lower lung volumes would exceed $P_{eff}$, and, hence, cause FL (Beck et al., 1991), are now within $P_{eff}$. At higher EELV, greater expiratory effort (pressure) can be exerted, and, consequently, greater levels of $V_E$ can be generated. And as lung volume increases the expiratory muscles are lengthened to better mechanical advantage, reaching their optimum length for power generation at total lung capacity (Berne and Levy, 1983). Another benefit of elevated EELV is that with hyperinflation airway resistance ($R_{aw}$) is reduced, since $R_{aw}$ is inversely related to lung volume (Briscoe and Dubois, 1958).
Disadvantages to Hyperinflation during Exercise

Breathing at elevated EELV during exercise, however, does tax the pulmonary system somewhat. With hyperinflation the inspiratory muscles operate at a mechanical disadvantage (Bye et al., 1983). Contributing to this "disadvantage" are compromising changes in 1) muscle length, which affects the length-tension relationship, 2) fulcrum/leverage arrangement of the muscles and their attachments, and 3) the radius of curvature of the muscles (Younes, 1991). With increased EELV, elastic work of breathing increases (Roussos et al., 1979; Yamashiro and Grodins, 1971), evidenced by increased inspiratory muscle work (Martin et al., 1980). In terms of energetics, breathing at higher lung volumes is disadvantageous since it requires a greater fraction of maximum inspiratory effort to generate a given inspiratory pressure at a higher lung volume (Younes, 1991). And, hyperinflation during exercise, even if it can meet the increased inspiratory energy demand and transcend FL, still presents the potential for reduced exercise performance due to respiratory muscle fatigue (Bye et al., 1983).
D. EELV Response to ERL, PLB, PEEP, Hyperbaria, and Asthma

The one frequently observed response to expiratory loading, whether in the form of expiratory resistive loading (ERL) (Garrard and Lane, 1977; Goldstein et al., 1975; Gothe and Cherniack, 1980; Grunstein et al., 1975; Hill et al., 1985; Pellegrino et al., 1993; Poon et al., 1987; Remmers and Bartlett, 1977), pursed lips breathing (PLB) (Barach et al., 1938), positive end-expiratory pressure (PEEP) (McIntyre et al., 1969; Kacmarek and Petty, 1988), hyperbaria (Demets and Anthonisen, 1973), or induced-bronchoconstriction (Martin et al., 1980), has been increased EELV.

Increased EELV in response to ERL has been a consistent finding in the literature; however, several investigators have observed this increase in EELV to be absent at maximal exercise (Goldstein et al., 1975) and at maximum ventilation (Garrard and Lane, 1977). In previous ERL studies, the resistance has ranged from 5 to 372 cm H₂O (Cerretelli et al., 1969; Remmers and Bartlett, 1977) and the protocols have entailed either resting or graded maximal or submaximal exercise. In a study by Garrard and Lane (1977), at rest
and during CO$_2$-stimulated hyperpnea, there was a direct correlation between amount of ERL and the rise in EELV.

Offering resistance to expiratory airflow has been observed to be an adaptive maneuver in patients with flow-limiting pathologies, and has been cited in the literature dating back the 1830's, when Laennec (1830) reported that emphysema patients intuitively used PLB to mitigate dyspnea and improve ventilation.

In the late 1940's, Barach and colleagues administered 20 cm H$_2$O of PEEP to healthy, normal subjects at rest and observed increases in EELV, BF, $V_E$, and $V_T$ (Kacmarek and Petty, 1988). PEEP, in its various forms, has been observed to increase EELV (FRC), by exerting positive pressure in the airways during either part of (expiratory positive airway pressure, EPAP) or during the entire breathing cycle (continuous positive airway pressure, CPAP) (Barach, 1973). The increase in EELV is due, at least partially, to the inflation of formerly non-patent airways of patients with COPD (Brewer et al., 1946). Enlargement of the bronchi has been demonstrated with PEEP pressures as low a 6 cm H$_2$O (Barach and Swenson, 1939). In laboratory animals, 40 cm H$_2$O of PEEP doubled
the cross-sectional diameter of the bronchi (Bickerman et al., 1952). PEEP has also been employed in high altitude aviation in conjunction with hyperoxia to effectively mitigate hypoxemia; 100 % O₂ was administered with PEEP ranging from 5 to 30.5 cm H₂O (Gagge et al., 1945).

Similarly, studies have been done by imposing expiratory load in the form of hyperbaria. Wood and Bryan (1978) had subjects perform graded exercise 0 to 10 atmospheres of ambient pressure (ATA), and observed a direct correlation between the increases in ATA and EELV. This relationship was consistent up to 10 ATA. However, increasing ATA reduced exercise tolerance and Vₑₑₘₐₓ.

Another example of increased EELV in response to increased expiratory resistance was observed in a study by Martin and coworkers (1980) in which EELV increased when bronchoconstriction was histamine-induced in asymptomatic asthmatic patients, raising airway pressure from 2.5 to 12.3 cm H₂O. In this study, the increase in airway resistance caused EELV to increase from 50.0 % to 74.4 % of total lung capacity.
i. Effect of ERL on Expiratory Airflow

In addition to increased EELV, the most prevalently observed consequences of ERL have been reductions in $V_E$ (Cain and Otis, 1949; Gothe and Cherniack, 1980; Hill et al., 1985; Poon et al., 1987; Wood and Bryan, 1978; Zechman et al., 1976) and mean expiratory flow (Pellegrino et al., 1993; Poon et al., 1987; Wood and Bryan, 1978). Other responses to ERL have been increased $V_T$ and increased $V_T/T_i$ (Gothe and Cherniack, 1980; Grunstein et al., 1975; Hill et al., 1985). With ERL vs control, previous investigators have observed an increase in both $T_i$ and $T_e$ (Poon et al., 1987); an increase in $T_e$ alone (Zechman et al., 1976); and no change in either $T_i$ or $T_e$ (Garrard and Lane, 1977).

E. Effect of ERL on Tidal Volume

The first available strategy to increase ventilation in response to increased ventilatory demand during graded exercise is to increase $V_T$ (Johnson et al., 1992). The benefit to this strategy is 20
limited since $V_T$ reaches a plateau at approximately 50-60% of vital capacity (Lind and Hesser, 1984; Younes and Kivinen, 1984). However, in addition to the normal expansion of $V_T$ with graded exercise, there has been observed a predictably greater increase in $V_T$ in response to ERL when compared to control (Grunstein et al., 1975; Hill et al., 1985; Poon et al., 1987).

III. Cardiovascular Ramifications of Changes in EELV

The flaccid pulmonary veins are exposed to the same compressive expiratory pressures as the membranous airways, so that it is not surprising that concomitant with the foregoing ventilatory changes with ERL there have typically been adverse alterations in left ventricular venous return. Normally, the positive $P_{pl}$ of expiration limits venous return to the left heart by compressing the pulmonary veins (Agostoni and Butler, 1991). There are flow-limiting segments where the pulmonary veins exit the lungs, which can limit venous return to the left atrium (Smith and Butler, 1975). However, if and when $P_{pl}$ exceeds $P_{eff}$, excessive
intrathoracic pressures are generated, which may impede venous return (Potter et al., 1971). Thus, during strenuous exercise, if and when expiratory $P_{pl}$ exceeds $P_{eff}$, in addition to causing airflow limitation, cardiovascular performance may also be hindered, secondary to reduced venous return. In COPD, this becomes evident with reductions in $O_2$ pulse ($VO_2/HR$) (Potter et al., 1971).

In addition, venous return to the right heart is reduced during expiration in proportion to the extent and duration of the positive pleural pressure; and, because of ventricular interdependence, reductions in right atrial filling would have commensurate, diminishing effects on left heart stroke volume (Agostoni and Butler, 1991). This situation may well be ameliorated by increasing lung volume during heavy exercise since elevated EELV is generally associated with decreased pulmonary vascular resistance (Wagner and Gale, 1991).
IV. ERL Utilized in Former Studies

Typically, the ERL that has been previously utilized has been 5 cm H$_2$O or greater. Some examples of ERL which have been employed are the following: 5-40 cm H$_2$O threshold ERL (Goldstein et al., 1975); 10 and 18 cm H$_2$O · L$^{-1}$ · s$^{-1}$ (Gothe and Cherniack, 1980); 12.3 cm H$_2$O · L$^{-1}$ · s$^{-1}$ (Martin et al., 1980); 22-40 cm H$_2$O of peak-expiratory pressure (Cerretelli et al., 1969); and 100 to 372 cm H$_2$O · L$^{-1}$ · s$^{-1}$ (Zechman et al., 1976). In the latter study using cats, these expiratory resistances represented 5-20 times the normal values. This is certainly an extreme case of unphysiologic expiratory airway resistance. In healthy humans, the cumulative total resistance of the respiratory system ($R_{rs}$) is generally taken to be 3 cm H$_2$O/L/sec (Mead and Agostoni, 1964), with resistance being somewhat greater during expiration than inspiration (England et al., 1982). The majority of this resistance is offered by the upper airways, and, in particular, by the glottis (Gautier et al., 1973), since all airflow must pass through the opening in the larynx, the glottis (England et al., 1982).
With the exceptions of the studies of Fee and colleagues (1992) and McClaran and coworkers (1992), in which EIH was mitigated, there has not been a previous study in which ERL has been employed with healthy exercising subjects in which there has been any cardiorespiratory improvement. The studies of Fee et al. (1992) and McClaran et al. (1992), however, do suggest that small amounts of ERL may be of cardiorespiratory benefit.

V. Selection of Breathing Pattern in Exercise

During strenuous exercise, maximal ventilatory capacity ($V_{E_{max}}$) is determined by the combination of the breathing frequency (BF), $V_T$, time of expiration $T_e$, and EELV which the subject selects (Jensen et al., 1980). Because $V_T$ plateaus at approximately 50-60 % of vital capacity (Lind and Hesser, 1984; Younes and Kivinen, 1984) during strenuous exercise, the critical determinant of $V_{E_{max}}$ becomes the placement of $V_T$ with respect to EELV (Beck et al., 1991). If during strenuous exercise EELV falls below resting FRC, $V_{E_{max}}$ will be significantly diminished in comparison to positioning
EELV above resting FRC (Jensen et al., 1980). Despite the physical constraint which a decreased EELV places on $V_{E\text{max}}$, and in effect exercise capacity, we have seen that during graded exercise, the exercising athlete "selects" and generally maintains an EELV significantly below resting FRC. Many authors have described this selection of EELV level as "optimal," because it optimizes diaphragmatic length (Henke et al., 1988) and it requires "minimal effort" (Dempsey et al., 1979), which would be reflected in lower $VO_2$ (McArdle et al., 1986).

VI. Statement of the Problem

We were perplexed by the fact that during strenuous exercise the respiratory system selects an EELV which apparently handicaps $V_{E\text{max}}$, and perhaps sets lower limits on exercise capacity. As we have seen from the work of Jensen et al. (1980), small upward movements in EELV significantly increase $V_{E\text{max}}$. The respiratory system, however, takes little notice of incipient expiratory FL as exercise progresses from heavy to intense (Johnson et al., 1991;
Johnson et al., 1992), deferring any compensatory response until expiratory FL has encompassed roughly half of $V_T$. Not until this stage does it return EELV close to resting FRC. Yet this may be too little too late.

In our former study, in which slight ERL mitigated EIH in highly-fit, endurance-trained athletes at mild altitude (Fee et al., 1992) and in the follow-up study by McClaran et al. (1992), we suspected that ERL may have effected an increase in EELV, thereby shifting the $V_T$ upward and away from the flow-limiting constraints of the MEFV curve, to a position where maximal expiratory flows are greater (Beck et al., 1991). Besides the mitigation of EIH, with ERL we observed a trend toward increased work capacity, as evidenced by an (albeit nonsignificant) increase in time to exhaustion. McClaran and colleagues (1992) observed increased ($p \leq 0.05$) $V_O2_{max}$ (in 14 of 18 subjects) with ERL.

Interpretation of this mitigation of EIH and the other data was compounded by the fact that both of these studies were conducted at mild altitude, where hypoxic vasoconstriction had no doubt affected the degree of EIH seen (Gale et al., 1985) and by the
absence of correlating expiratory pressure and lung volume measurements.

Other investigators have loaded expiration, typically with what we judge to have been unphysiologic resistances (from 5.0-372.0 cm H$_2$O), which in all likelihood overwhelmed the respiratory system. Others have employed ERL in protocols in which FL had not been a prospect, that is, either in subjects at rest or exercising at moderate workloads. We felt that choosing a model in which FL would potentially develop, that is, in highly-fit athletes performing at heavy to maximal workloads, ERL may improve cardiorespiratory and exercise performance at sea level.

To examine this hypothesis we chose to create ERL by employing the same expiratory orifice which mitigated EIH in our altitude study, and to compare this ERL against the control Hans Rudolph valve (2700), in two series of comparisons: a VO$_{2\text{max}}$ series and a He dilution determination of EELV during steady-state heavy exercise. The aims of this study were:

a). To investigate the cardiorespiratory responses to ERL during graded maximal exercise.
b) To measure changes to peak-expiratory mouth pressure ($P_{ao}$) and expiratory pressure ($P_{E}$) waveform during ERL and control.

c) To determine the response of EELV to ERL during heavy, steady-state exercise.
CHAPTER II

METHODS

I. Subjects and Their Selection

Subjects were all actively competitive men (n=23) and women (n=5) cyclists and/or triathletes, age = 33.5 ± 7.3 SEM and VO$_{2\text{max}}$ = 63.4 ± 1.36 SEM. All were studied with their informed consent. All procedures were approved by the Human Experimentation Committee, University of Hawaii at Manoa. The physical characteristics and selected lung volumes of the subjects are given in Table 1.

II. Protocols

A. Bicycle Ergometry

All tests were performed on a Ergo-metrics 800 electrically braked bicycle ergometer which was integrated with the Schiller
Table 1. Subject characteristics. Residual volume, RV; vital capacity, VC; total lung capacity, TLC; forced vital capacity, FVC; forced expiratory volume in 1 s., FEV 1.0. (n = 28; women, n = 5; men, n = 23).

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>± SD</th>
<th>Range</th>
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<td>7.3</td>
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<td>Height (cm)</td>
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<td>7.4</td>
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<tr>
<td>Weight (kg)</td>
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<td>8.1</td>
<td>51.8-80.9</td>
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<tr>
<td>VO$_{2}$max (ml/kg /min)</td>
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<td>6.6</td>
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<tr>
<td>RV (liter)</td>
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<td>0.39</td>
<td>1.02-2.47</td>
</tr>
<tr>
<td>VC (liter)</td>
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<td>0.90</td>
<td>4.78-8.03</td>
</tr>
<tr>
<td>TLC (liter)</td>
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<td>1.10</td>
<td>5.66-10.35</td>
</tr>
<tr>
<td>FVC* (%)</td>
<td>111.30</td>
<td>12.8</td>
<td>91-137</td>
</tr>
<tr>
<td>FEV 1.0* (%)</td>
<td>112.10</td>
<td>14.4</td>
<td>89-133</td>
</tr>
</tbody>
</table>

* Percentage of normal predicted values: Schiller America: (Schiller derivations based on Knudson, Crapo, and Morris).
AT-6 (CH 6340), a multiplex system capable of ECG monitoring, pulmonary function testing, and controlling the exercise protocol. During an initial familiarization session, subjects were asked to choose a power output (PO) (watts) that was comfortable, yet somewhat demanding, and to indicate at what pedal cadence they normally cycled. These became the starting PO and the fixed cadence for all testing. Prior to all test protocols, subjects were given the same warm-up, which began at 135 w on the cycle ergometer and was increased 10 w·min⁻¹, for a total of 8 min. All protocol tests commenced with one minute at the subject's chosen, fixed cadence and PO, and was increased by 10 w·min⁻¹. Subjects were asked to keep their cadence within 1-2 RPM of the digital instantaneous RPM readout at all times.

B. Expiratory Resistive Loading (ERL)

During the control tests, subjects breathed through a three-way low-resistance valve (Hans Rudolph 2700). The three-way valve was mounted in such a way as to assure 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). Figure 1 presents relationship between peak-pressure at the mouth (P\textsubscript{ao}) and minute ventilation (V\textsubscript{E}), control vs ERL. In all comparisons from subject to subject, the order of presentation of expiratory orifices, ERL or control, was systematically alternated and the subjects were naive to the order of presentation. P\textsubscript{ao} was sampled from a sampling port hose barb in the housing of the three-way valve. A 0.5 m section of non-yielding plastic tubing (I.D.=2 mm) connected the barb to a Grass volumetric pressure transducer (P T 5 A), which exhibited linear fidelity over the range of 0.0 to 12.0 cm H\textsubscript{2}O. Before each test, the transducer was calibrated with a water manometer. The Grass transducer was integrated with a Harvard Apparatus strain gauge coupler (2193). During all tests, P\textsubscript{ao} traces were recorded on a Harvard Apparatus 12-speed Chart Mover (486). The chart speed was maintained at 0.025 cm·sec\textsuperscript{-1} except for single-breath traces of expiratory pressure (P\textsubscript{E}), recorded at 5.0 cm·sec\textsuperscript{-1}. 

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Figure 1. Peak-expiratory pressure at the mouth ($P_{ao}$) relationship to minute ventilation ($V_E$): control vs expiratory resistive loading (ERL) ($n = 28$).
C. Test Protocols

The study was carried out in two separate protocols: a graded VO$_{2\text{max}}$ series and a steady-state series (at 75 % PO$_{\text{max}}$) in which FRC (EELV) was determined.

i. Graded VO$_{2\text{max}}$ Testing

Each subject performed two graded VO$_{2\text{max}}$ bicycle ergometer tests, one with ERL and one with control, systematically alternated in presentation. PO$_{\text{max}}$ (watts) was considered to be the higher last full-minute wattage attained in either of the two VO$_{2\text{max}}$ tests.

ii. EELV Testing: Steady-State Series at 75 % PO$_{\text{max}}$

Subjects performed two graded bicycle ergometer tests up to a steady-state of 75 % PO$_{\text{max}}$, during which EELV was determined with ERL vs control. The EELV-determination test procedures
followed those of the VO$_{2\text{max}}$ testing. At 75 % PO$_{\text{max}}$, the wattage was manually fixed, and maintained there for 3 min. In the next (4th) minute, presuming steady state (Wasserman, 1978), we switched the subject into the closed-circuit He-rebreathing system for 30 s.

**iii. EELV Determination: He-dilution**

EELV was determined by a He-dilution technique in which two Hans Rudolph manual directional control, three-way Y-shaped valves (2100 C) were used in-line to switch from from the open-circuit gas evaluating system to the closed-circuit Collins Helium Residual Volume Apparatus (P-1300) rebreathing system with a13.5 L spirometer. Switches were made manually. Prior to each test the system was purged, the He analyzer calibrated with a known concentration of He-containing gas (5.0%), purged again, then loaded with 4 L O$_2$ and 0.6 L He. During the rebreathing (30 s), 100 % O$_2$ was infused to the system at a rate equal to the VO$_2$. The system was scrubbed of CO$_2$ by barium hydroxide lime, USP (Collins absorb-
ent granules). Final He concentrations were recorded when further gas mixing elicited no further changes in [He$_d$].

III. Measurements

A. Test Conditions

All tests were conducted under relatively stable Honolulu environmental conditions: 20-21°C, 758-765 mm Hg, and 50-65% relative humidity. Subjects' oral temperatures were taken pre- and post-exercise in all tests with Thermoscan® ear thermometer (HM-2) and no differences were observed.

B. Oximetry

$\text{SaO}_2$ was determined with a Satlite ear oximeter (OPS-200), which was internally "self-calibrated" prior to each exercise test. $\text{SaO}_2$ was measured only in the VO$_{2\text{max}}$ testing.
C. Electrocardiography

Each subject's electrocardiogram (ECG) and heart rate (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 Mitsuba monitor (710 A) interfaced with the AT-6 system. Before every exercise test the ECG monitor was calibrated with the Physio-dyne Heart Rate Calibrator (ECG-Cal).

D. Gas Analysis

Ventilatory variables were monitored via standard open-circuit spirometry. Fractional concentrations of $O_2$ ($F_{E}O_2$) were determined by a Ametek $O_2$ analyzer (S-3A/I) and fractional concentrations of $CO_2$ ($F_{E}CO_2$), were determined by a Ametek $CO_2$ analyzer (CD-3A).
E. Ventilatory Volumes

Inspired ventilation volume ($V_I$) was determined with a K. L. Engineering flow transducer (K 520), and $V_E$ was calculated from $V_I$ and the gas exchange ratio ($R$) by the following formula: $V_E = (V_I) \cdot (R)$.

F. Data Collection Program

Before each test, subject anthropomorphic information (height, weight, and age) was loaded into the data collection program, along with the ambient barometric pressure (ascertained from the hourly-updated National Weather Service phone recording), lab temperature and relative humidity (determined from Micronta thermometer/hygrometer, #63-844). The OCM-2 program generated breath-by-breath recordings of the following variables: $SaO_2$, HR, $R$, $V_E$, breathing frequency ($BF$), $V_T$, duration of inspiration ($T_i$), duration of the breathing cycle ($T_{tot}$), $T_i/ T_{tot}$, mean inspiratory flow ($V_T/ T_i$), mean expiratory flow ($V_T/ T_e$), $F_{E0}_2$, $F_{ECO_2}$, carbon dioxide
production (VCO₂), O₂ pulse (VO₂/HR), VO₂ ml·kg⁻¹·min⁻¹, ventilatory equivalent for O₂ (VE/VO₂), and ventilatory equivalent for CO₂ (VE/VCO₂). All dynamic ventilatory volumes were converted by the OCM-2 program into BTPS, and along with SaO₂ and HR were averaged to minute intervals. All data collected were stored on disc and written in hard-copy.

G. EELV and RV Measurements

EELV and residual volume (RV) determinations were derived from spirometry and the standard He-dilution equation: EELV = ([Heᵢ/Heᵣ]-1) · Vₛₚ (Guyton, 1991) and reflect allowances for O₂ consumed or added to the system. CO₂ was entirely removed from the system, and was verified by a volumetric plateau in the kymograph tracing. This plateau usually occurred after 3 min of mixing. Vital capacity (VC), forced vital capacity (FVC), and forced expiratory volume in 1 s. (FEV₁₀) were determined with the Schiller AT-6 pulmonary function testing equipment. Predicted
values were based on a composite derived by Schiller (Schiller Issue 07.1991) from the work of Knudson, Crapo, and Morris.

H. Data Collection

Measurements of expired fractional concentrations of $O_2$ and $CO_2$, and $V_I$, along with those of $SaO_2$ and HR (via Physio-dyne HR computer [ECG-HR3]), were integrated with a CompuAdd 433 (A000) computer and monitor (51109), which ran the Ametek Oxygen Uptake System, OCM-2 program.

IV. Statistical Analyses

A. Data Normalization

The criterion for intrasubject comparisons was $PO_{max}$, as determined during the paired $VO_{2max}$ tests. Since the majority of subjects attained different levels of $PO$ in each of their $VO_{2max}$ tests, $PO_{max}$ was ascribed to be the higher full-minute $PO$ of the
two VO$_{2\text{max}}$ tests. The virtue of this method is that it ensured that every intrasubject comparison was made at identical PO, or wattage levels. It also provided a criterion for normalizing the data into percentages of PO$_{\text{max}}$.

**B. Graded VO$_{2\text{max}}$ Test Series Comparisons**

Inherent in the fact that a given subject who accomplished two different PO levels is that he/she would have only one set of data at 100% and that there was no reference for comparison at that level. In many cases there is no reference for comparison at 95%, either. Therefore, the data were normalized with reference to PO$_{\text{max}}$ at 75% (heavy exercise), 80, 85, and 90% PO$_{\text{max}}$ (intense exercise). These data were analyzed using the SigmaStat® program. Repeated measures analyses of variance were performed, and where significant differences ($p \leq 0.05$) between control and treatment (ERL) were indicated, the Student-Newman Keuls post hoc test was performed to determine at what level(s) of PO$_{\text{max}}$ there was a
significant ($p \leq 0.05$) difference between control and treatment (ERL).

Because there were no comparable data at 95 and 100 \% $P_{O_{max}}$, and due to the fact that $VO_{2\max}$ may well be viewed as a discrete physiologic event, unto itself, we analyzed the $VO_{2\max}$ data using paired t-tests to determine if significant ($p \leq 0.05$) differences existed between control and treatment (ERL).

Selected data (Table 4) at $VO_{2\max}$ were analyzed by the Wilcoxon signed rank test (Ferguson and Takane, 1989).

C. EELV Test Series: Steady-State at 75 \% $P_{O_{max}}$

These data were analyzed using the SigmaStat\textsuperscript{®} program. The data were analyzed using paired t-tests to determine if there were significant ($p \leq 0.05$) differences between control and treatment (ERL) during steady-state at 75 \% $P_{O_{max}}$. 
CHAPTER III

RESULTS

1. Graded VO2max Test Series Comparisons

There was significant EIH in both control and ERL tests at 75, 80, 85, and 90% PO_{max} and at VO2_{max} (Figure 2) when compared to resting SaO2; however, there was no significant difference in SaO2 between control and ERL at rest, or at any level of intensity. SaO2 comparisons at VO2_{max} were 93.4 ± 0.98 % vs 94.3 ± 0.55 %, control vs ERL, respectively.

Peak expiratory pressures at the mouth (P_{ao}) (cm H2O) were greater (p<0.05) with ERL at 75, 80, 85, and 90% PO_{max} and at VO2_{max} (Table 2). Under control conditions P_{ao} were in the range of 1.07 to 2.38 cm H2O; with ERL P_{ao} ranged from 1.27 to 2.94 cm H2O. With ERL in the present study, the range of P_{ao} which we employed was considerably lower than that which had been employed in 43
Figure 2. Arterial oxygen saturation (SaO₂) at rest and during graded maximal exercise; control vs expiratory resistive loading (ERL). Percentage of power output (% PO<sub>max</sub>). *Significantly (p ≤ 0.05) different from resting values (both control and ERL). All values ± SEM.
Table 2. Corresponding values for peak-expiratory pressure at the mouth (P<sub>ao</sub>) and minute ventilation (V<sub>E</sub>), at progressive levels of exercise intensity: control vs expiratory resistive loading (ERL). (n = 28). Δ = change; PO<sub>max</sub> = power output (watts); maximal oxygen consumption = VO<sub>2max</sub>.

<table>
<thead>
<tr>
<th>% PO&lt;sub&gt;max&lt;/sub&gt;</th>
<th>P&lt;sub&gt;ao&lt;/sub&gt; (Control)</th>
<th>P&lt;sub&gt;ao&lt;/sub&gt; (ERL)</th>
<th>Δ P&lt;sub&gt;ao&lt;/sub&gt; (cm H&lt;sub&gt;2&lt;/sub&gt;O)</th>
<th>V&lt;sub&gt;E&lt;/sub&gt;&lt;sup&gt;^&lt;/sup&gt; (l/min) (Control)</th>
<th>V&lt;sub&gt;E&lt;/sub&gt;&lt;sup&gt;^&lt;/sup&gt; (l/min) (ERL)</th>
<th>ΔV&lt;sub&gt;E&lt;/sub&gt; (l/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>75</td>
<td>1.07</td>
<td>1.27</td>
<td>0.20*</td>
<td>82.6</td>
<td>85.9</td>
<td>3.3</td>
</tr>
<tr>
<td>80</td>
<td>1.23</td>
<td>1.58</td>
<td>0.35*</td>
<td>93.1</td>
<td>97.2</td>
<td>4.1*</td>
</tr>
<tr>
<td>85</td>
<td>1.47</td>
<td>1.88</td>
<td>0.41*</td>
<td>104.6</td>
<td>109.5</td>
<td>4.9*</td>
</tr>
<tr>
<td>90</td>
<td>1.84</td>
<td>2.33</td>
<td>0.49*</td>
<td>119.9</td>
<td>124.4</td>
<td>4.5*</td>
</tr>
<tr>
<td>VO&lt;sub&gt;2max&lt;/sub&gt;</td>
<td>2.21</td>
<td>2.94</td>
<td>0.73*</td>
<td>147.7</td>
<td>153.5</td>
<td>5.8*</td>
</tr>
</tbody>
</table>

† SEM ± 0.113; ^ SEM ± 3.42. *Significant (p<0.05) difference.
previous studies, which had been in the range of 5-372 cm
H₂O (Cerretelli et al., 1969; Goldstein et al., 1975; Gothe and
Cherniack, 1980; Martin et al., 1980; Zechman et al., 1976).

Concomitant with these values of P_{ao}, minute ventilation (V_E)
(L·min⁻¹) was increased (p≤0.05) at 80, 85, 90% PO_{max}, and at
VO_{2max} with ERL vs control (Table 2, Figure 3). The increase in V_E
is contrary to the findings of all other loaded expiration studies
(Cain and Otis, 1949; Gothe and Cherniack, 1980; Hill et al., 1985;
Poon et al., 1987; Wood and Bryan, 1978; Zechman et al., 1976).

Tidal volume (V_T) was consistently greater and significantly so
(p≤0.05) at 80 % PO_{max} with ERL (Figure 4). The increase in V_T with
ERL averaged 144 ml from 75-90% PO_{max}, but was nearly identical
with control values at VO_{2max}. Neither time of inspiration (T_i)
or
time of expiration (T_e) changed. Data for V_T and other cardiores-
piratory variables at 75, 80, 85, 90% PO_{max}, and at VO_{2max} are
given in Table 3. The expansion of V_T seen in response to ERL in our
study is consistent with the observations of previous investigators.
Figure 3. Minute ventilation ($V_E$) vs percentage of maximum power output ($\% P_{O_{max}}$): control vs expiratory resistive loading (ERL). ($n = 28$). * Significantly ($p \leq 0.05$) different. All values ± SEM.
Figure 4. Tidal volume ($V_T$) vs percentage of maximum power output (% $PO_{max}$): control vs expiratory resistive loading (ERL). ($n = 28$). * Significantly ($p \leq 0.05$) different. All values ± SEM.
Table 3. Cardiorespiratory variables during graded maximal (VO$_{2\text{max}}$) exercise tests: control vs expiratory resistive loading (ERL). (n = 28). Minute ventilation (V$_E$); tidal volume (V$_T$); relative oxygen consumption (VO$_2$ ml·kg$^{-1}$·min$^{-1}$); heart rate (HR); oxygen pulse (VO$_2$/HR); time of expiration (T$_e$); mean expiratory flow (V$_T$/T$_e$); time of inspiration (T$_i$); duty cycle (T$_i$/T$_{tot}$); mean inspiratory flow (V$_T$/T$_i$); and breathing frequency (BF) *Significant (p≤0.05) difference.

<table>
<thead>
<tr>
<th>Percentage of PO$_{\text{max}}$</th>
<th>75</th>
<th>80</th>
<th>85</th>
<th>90</th>
<th>VO$_{2\text{max}}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>V$_E$ (L·min$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>control</td>
<td>82.6</td>
<td>93.1</td>
<td>104.6</td>
<td>119.9</td>
<td>147.7 ± 4.1</td>
</tr>
<tr>
<td>ERL</td>
<td>85.9</td>
<td>97.2*</td>
<td>109.5*</td>
<td>124.4*</td>
<td>153.5 ± 4.5*</td>
</tr>
</tbody>
</table>

(75-90 %, SEM = ± 3.42)

| V$_T$ (ml)                      |     |     |     |     |                   |
| control                         | 2535| 2617| 2717| 2815| 2857 ± 82.7       |
| ERL                             | 2656| 2815*| 2871| 2918| 2864 ± 79.7       |

(75-90 %, SEM = ± 84.7)

| VO$_2$ (ml·kg$^{-1}$·min$^{-1}$) |     |     |     |     |                   |
| control                         | 44.0| 48.0| 51.0| 54.5| 60.3 ± 1.26       |
| ERL                             | 46.8| 51.3| 54.3| 57.6*| 63.4 ± 1.36*      |

(75-90 %, SEM = ± 1.53)

49
Table 3. (continued)

<table>
<thead>
<tr>
<th>Percentage of $PO_{max}$</th>
<th>75</th>
<th>80</th>
<th>85</th>
<th>90</th>
<th>$VO_2max$</th>
</tr>
</thead>
<tbody>
<tr>
<td>75 80 85 90</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**HR**
- **control**
  - 153.7
  - 161.5
  - 166.7
  - 171.7
  - 180.6 \(†\)
- **ERL**
  - 150.8
  - 159.3
  - 164.8
  - 169.2
  - 177.0 \(†\)

(75-90 %, SEM = ± 2.85) (†Wilcoxon signed rank test)

**$O_2$ pulse (VO$_2$/HR)**
- **control**
  - 20.1
  - 20.9
  - 21.4
  - 22.2
  - 24.1 \(†\)
- **ERL**
  - 21.9 *
  - 22.6 *
  - 23.1 *
  - 23.8 *
  - 25.8 *\(†\)

(75-90 %, SEM = ± 0.81) (†Wilcoxon signed rank test)

**$T_e$ (sec)**
- **control**
  - 0.71
  - 0.63
  - 0.56
  - 0.48
  - 0.32 ± 0.02
- **ERL**
  - 0.74
  - 0.67
  - 0.57
  - 0.48
  - 0.30 ± 0.02

(75-90 %, SEM = ± 0.03)

**$V_T/ T_e$ (L·sec$^{-1}$)**
- **control**
  - 2.97
  - 4.35
  - 5.15
  - 6.28
  - 8.50 ± 0.30
- **ERL**
  - 3.75 *
  - 4.36
  - 5.19
  - 6.39
  - 8.91 ± 0.34 *

(75-90 %, SEM = ± 0.34)
Table 3. (continued)

<table>
<thead>
<tr>
<th>Percentage of $PO_{\text{max}}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>75</td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>1.16</td>
</tr>
<tr>
<td>1.14</td>
</tr>
</tbody>
</table>

$(75-90 \%$, SEM = ± 0.03$)$

$T_i (\text{sec})$

| control | 0.62 | 0.64 | 0.65 | 0.68 | 0.73 † |
| ERL     | 0.61 | 0.63* | 0.64 | 0.67 | 0.72 † |

$(75-90 \%$, SEM = ± 0.01$)$ ($†$Wilcoxon signed rank test)

$V_T/ T_i (L \cdot \text{sec}^{-1})$

| control | 2.22 | 2.44 | 2.67 | 2.97 | 3.47 ±0.09 |
| ERL     | 2.35* | 2.60* | 2.84* | 3.11* | 3.60 ± 0.10* |

$(75-90 \%$, SEM = ± 0.08$)$

$B_F (\text{br} \cdot \text{sec}^{-1})$

| control | 33.1 | 36.0 | 38.8 | 43.2 | 52.4 ± 1.73 |
| ERL     | 32.8 | 34.9 | 38.6 | 43.2 | 54.1 ± 1.62* |

$(75-90 \%$, SEM = ± 1.33$)$
(Grunstein et al., 1975; Hill et al., 1985, Poon et al., 1987). $PO_{max}$, but was greater ($p < 0.05$) with ERL at $VO_{2max}$ (Table 3). Breathing frequency (BF) was unchanged from 75-90 %, where it was $54.1 \pm 1.6$ vs $52.4 \pm 1.7$ SEM breaths/min (control) (Table 3). This varies from previous investigators who observed decrements in BF with ERL during exercise (Gothe and Cherniack, 1980; Hill et al., 1985). BF was actually significantly higher ($p \leq 0.05$) with ERL at $VO_{2max}$, which no one has previously reported.

With control vs ERL, neither duration of inspiration ($T_i$) nor duration of expiration ($T_e$) were changed at any level of intensity. With ERL vs control, previous investigators have observed an increase in both $T_i$ and $T_e$ (Poon et al., 1987); an increase in $T_e$ alone (Zechman et al., 1976); and no change in either $T_i$ or $T_e$ (Garrard and Lane, 1977). The prolongation of $T_e$ observed by Goldstein and coworkers (1975) was proportionate to the amount of ERL, which ranged from 0 to 30 cm $H_2O$. In light of this, it may be that the ERL in our study was too slight to elicit this lengthening of $T_e$. In our study ($T_i/T_{101}$) was lower ($p \leq 0.05$) with ERL at 80 % $PO_{max}$. Mean
inspiratory flow ($V_T/T_i$) was greater ($p<0.05$) with ERL at 75, 80, 85, and 90% $P_{O_{\max}}$, and at $V_{O_{2_{\max}}}$ (Figure 5). This is consistent with the data of Grunstein and coworkers (1975). Mean expiratory flow ($V_T/T_e$) was greater ($p<0.05$) with ERL at 75 % $P_{O_{\max}}$.

Heart rate (HR) was consistently lower ($\geq 2.0$ BPM) throughout with ERL (Figure 6), but not significantly so. $V_{O_{2}}$ (ml·kg$^{-1}$·min$^{-1}$) was non-significantly higher ($\leq 3.2$ ml·kg$^{-1}$·min$^{-1}$) from 75-85 % $P_{O_{\max}}$ with ERL, and greater ($p<0.05$) with ERL at 90 % $P_{O_{\max}}$ and at $V_{O_{2_{\max}}}$. It should be noted that the nonsignificantly elevated $V_{O_{2}}$, when combined with the nonsignificantly lowered HR, produced an O$_2$ pulse ($V_{O_{2}}$/HR) which was significantly greater by 9.5, 9.0, 7.9, and 7.1%, with ERL at 75, 80, 85, 90% $P_{O_{\max}}$ and at $V_{O_{2_{\max}}}$ (Figure 7).

Subjects attained 1.8 % greater $P_{O_{\max}}$ with ERL, 352.0 $\pm$ 9.9 vs 345.7 $\pm$ 9.5 watts ($p<0.05$) (Figure 8), and 5.1 % higher $V_{O_{2_{\max}}}$, 63.4 $\pm$ 1.36 vs 60.3 $\pm$ 1.26 ml·kg$^{-1}$·min$^{-1}$ ($p<0.05$) (Figure 9).
Figure 5. Mean inspiratory flow ($V_T / T_i$) vs percentage of maximum power output ($\% \text{PO}_{\text{max}}$): control vs expiratory resistive loading (ERL). ($n = 28$). * Significantly ($p \leq 0.05$) different. All values ± SEM.
Figure 6. Heart rate (HR) vs percentage of maximum power output (% PO_{max}): control vs expiratory resistive loading (ERL). (n = 28).

* Significantly (p≤0.05) different. All values ± SEM.
Figure 7. Oxygen pulse (VO₂max/HR) vs percentage of maximum power output (% PO_max): control vs expiratory resistive loading (ERL). (n = 28). * Significantly (p≤0.05) different. All values ± SEM.
Figure 8. Maximum power output ($P_{O_{max}}$) control vs expiratory resistive loading (ERL). $n = 28$. *Significantly ($p \leq 0.05$) different. All values ± SEM.
Figure 9. Maximal oxygen consumption ($VO_2max$): control vs expiratory resistive loading (ERL). All values ± SEM.
ERL had no effect (p>0.05) on R, expired fractional concentrations or ventilatory equivalents for O₂ or CO₂.

II. Steady-State at 75 % \(PO_{max}\) Comparisons

Table 4 presents the essential cardiorespiratory data, comparing control to ERL during steady-state exercise at 75 % \(PO_{max}\). The remainder of the data is presented in Table 6, of the Appendix.

A. ERL and EELV

During steady state at 75 % \(PO_{max}\), \(P_{ao}\) was 0.44 cm H₂O greater with ERL: 1.57 ± 0.17 vs 1.13 ± 0.13 cm H₂O (control). EELV was elevated (p<0.05) 0.67 ± 0.29 L with ERL: 3.11 ± 0.27 (ERL) vs 2.45 ± 0.16 L (control). Increases in EELV in response to ERL have been widely observed in previous studies (Garrard and Lane, 1977; Goldstein et al., 1975; Gothe and Cherviack, 1980; Grunstein et al., 1975; Hill et al., 1985; Pellegrino et al., 1993; Poon et al., 1987; Remmers and Bartlett, 1977).
B. End-Inspiratory Lung Volume

With ERL, end-inspiratory lung volume (EILV) was significantly greater than control: 5.92 ± 0.41 vs 5.22 ± 0.37 L (± SEM), respectively. Of this difference (0.7 L) in EILV, 0.67 was due to an increase in EELV, and .03 L due to increased V_T (see data in Table 4). These EILV volumes represent 82.2 % ± 1.46 % of TLC vs 72.5 % ± 1.23 % (± SD), ERL vs control; and, 77.5 % ± 0.98 % vs 65.9 % ± 1.11 % VC (± SD), ERL vs control.

C. The Effect of ERL on V_E

V_E was greater (p≤0.05) with ERL. The ERL employed here at steady-state (1.57 ± 0.17 cm H_2O) represents quite a modest expiratory load in comparison to the ERL which has been utilized in prior studies, in which ERL has ranged from 5 to 372 cm H_2O (Cerretelli et al., 1969; Goldstein et al., 1975; Gothe and Cherniack, 1980; Martin et al., 1980; and Zechman et al., 1976). In fact, there has been a direct correlation demonstrated between the amount of
Table 4. Respiratory variables during steady-state 75% maximal power output. Peak-expiratory pressure at the mouth, \( P_{ao} \); end-expiratory lung volume, \( EELV \); minute ventilation, \( V_E \); tidal volume, \( V_T \); breathing frequency, \( BF \); heart rate, \( HR \); relative oxygen consumption, \( VO_2 \) \( ml^{-1}kg^{-1}min \); \( O_2 \) pulse, \( VO_2/HR \); time of expiration, \( T_e \); mean expiratory flow, \( V_T/T_e \); time of inspiration, \( T_i \); duty cycle, \( T_i/T_{tot} \); and mean inspiratory flow, \( V_T/T_i \). All values are \( \pm \) SEM. \( n = 12 \). *Significantly different \( (p \leq 0.05) \) †Wilcoxon signed rank test.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>ERL</th>
<th>( \Delta )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( P_{ao} ) (cm ( H_2O ))</td>
<td>1.13 ± 0.13</td>
<td>1.57 ± 0.17</td>
<td>0.44 ± 0.01*</td>
</tr>
<tr>
<td>( EELV ) (L)</td>
<td>2.45 ± 0.16</td>
<td>3.11 ± 0.24</td>
<td>0.67 ± 0.29*</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 ) (ml)</td>
<td>2778 ± 110.5</td>
<td>2808 ± 127.8</td>
<td>30.3 ± 61.1</td>
</tr>
<tr>
<td>( BF ) (Breaths/min)</td>
<td>36.0 ± 1.6</td>
<td>38.2 ± 1.86</td>
<td>2.2 ± 1.1</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>( VO_2 ) (( ml^{-1}kg^{-1}min ))</td>
<td>52.0 ± 1.1</td>
<td>52.8 ± 1.4</td>
<td>0.7 ± 0.7</td>
</tr>
<tr>
<td>( O_2 ) pulse (( VO_2/HR ))</td>
<td>24.6†</td>
<td>24.7†</td>
<td></td>
</tr>
</tbody>
</table>

61
Table 4. (continued)

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>ERL</th>
<th>Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_e$ (sec)</td>
<td>0.65 ± 0.04</td>
<td>0.59 ± 0.04</td>
<td>0.06 ± 0.04</td>
</tr>
<tr>
<td>$V_{T/T_e}$ (L/sec)</td>
<td>4.42 ± 0.23</td>
<td>4.98 ± 0.36</td>
<td>0.57 ± 0.25</td>
</tr>
<tr>
<td>$T_i$ (sec)</td>
<td>1.11†</td>
<td>1.01†</td>
<td></td>
</tr>
<tr>
<td>$T_f/T_{tot}$</td>
<td>0.66†</td>
<td>0.64†</td>
<td></td>
</tr>
<tr>
<td>$V_{T/T_i}$ (L⋅sec⁻¹)</td>
<td>2.64 ± 0.06</td>
<td>2.76 ± 0.09</td>
<td>0.12 ± 0.06</td>
</tr>
</tbody>
</table>
ERL employed and commensurate reductions in $V_E$ (Zechman et al., 1976; Poon et al., 1987; Goldstein et al., 1975). The small amount of ERL used in the present study may have been too small to cause the adverse effects on $V_E$ seen in previous studies.

On the other hand, the resultant increase in $V_E$ with the slight ERL observed in our study suggests that the Hans Rudolph "low resistance" three-way valve, which is conventionally used in exercise testing, may offer too little resistance. The valve may prevent the normal impedance to expiration which is offered by the lips, teeth, etc. during everyday exercise/training.

**D. ERL and Mean Expiratory Flow**

$V_T/T_e$ was greater ($p<0.05$) with ERL. This may suggest that expiratory flow was improved by ERL, however, it would be difficult to conjecture during which stage of expiration flow was increased (i.e., during the effort-independent or the effort-dependent segment, see Breathing Patterns below).
None of the other cardiorespiratory variables were affected by ERL at 75 % $P_{O_{max}}$ steady state, these include: $V_T$, HR, BF, $V_T/T_e O_2$ pulse (Table 5), $T_i$, $T_e$, $T_i/T_{tot}$, $V_T/T_i$, $F_{E}O_2$, $F_{E}CO_2$, $R$, and ventilatory equivalents for $O_2$ and $CO_2$ (Appendix, Table 6).

III. Breathing Patterns

In both the steady-state and in graded maximal exercise testing, the single-breath expiratory pressure ($P_E$) traces suggest that there were three noticeable differences in breathing patterns, with ERL (Figure 10): there was 1) a prolonged plateau of $P_E$, 2) a sharper initial rise, and 3) a more abrupt decline in $P_E$ at end-expiration.
Control

Subject B.H.
74.8\% Max Power output

Subj ect M.J.
75.2\% Max Power output

Subject L. J.
92.2\% Max Power output

Figure 10. Single-breath expiratory pressure at the mouth ($P_E$) vs time.
CHAPTER IV

DISCUSSION

I. Arterial Oxygen Saturation

In this study, at sea level, there was significant EIH in both control and ERL tests at 75, 80, 85, and 90% PO\textsubscript{max} and at VO\textsubscript{2max} (Figure 2) when compared to resting SaO\textsubscript{2}; however, there was no significant difference between control and ERL at rest, or at any level of intensity. The degree of arterial desaturation was far milder at sea level than that which was observed at moderate altitude, 1520 m, in graded VO\textsubscript{2max} tests. In previous studies at 1520 m, under control conditions at VO\textsubscript{2max}, SaO\textsubscript{2} was observed to be 84.5 % (Tucker et al., 1984), 86.6 % (Fee et al., 1992), and 84.9 % (McClaran et al., 1992). In the present study, at VO\textsubscript{2max}, SaO\textsubscript{2} was (93.4 ± 1.0, control; 94.3 ± 0.6 %, ERL). We suspect that in this study there were several conditions, aside from the higher P\textsubscript{1}O\textsubscript{2}, which may have precluded all but mild EIH.
The most obvious difference between this study and our former study is altitude. At sea level, $V_A/Q_e$ mismatch (Wagner, 1992), secondary to interstitial pulmonary edema (Bevegard et al., 1960) is no longer a potential contributing factor in EIH. Also, at sea level, $V_A/Q_e$ mismatch, which has been observed to increase during heavy exercise at moderate altitude (1520 m) as a consequence of nonuniform hypoxic pulmonary vasoconstriction (Gale et al., 1985) was no longer an influence at sea level.

Another difference, the change in mode of exercise from treadmill running at altitude, to bicycle ergometry in this study at sea level may have affected arterial saturation ($\% \text{SaO}_2$) levels. There is a direct relationship between an individual’s $VO_{2\text{max}}$ and his/her ability to desaturate (Williams et al., 1986); and, since there is less oxygen-consuming muscle mass involved in biking than there is in treadmill running, $VO_{2\text{max}}$ (or, more properly, $VO_{2\text{peak}}$) determined on the bicycle is normally 6-11 $\%$ lower than on the treadmill (McArdle et al., 1986).

Another difference was that in the present study we included a 8-minute warm-up period prior to all graded exercise tests.
Dempsey and colleagues (1982) found that when a group of highly fit athletes (who had demonstrated EIH in short-term, sudden-onset, heavy, that is, 80-85 % $\text{VO}_{2\text{max}}$, exercise) were given a long, easy warm-up first, they didn't desaturate in the heavy exercise bout.

Despite our initial disappointment in failing to produce all but mild EIH at sea level, there were some very encouraging changes in exercise performance and in several cardiorespiratory variables in response to ERL, in particular, increased $V_{E}$, $O_{2}$ pulse, $\text{VO}_{2\text{max}}$, and $P_{O_{max}}$, which are reported here for the first time.

II. Methodological Limitations of Prior ERL Studies

In previous ERL studies, we feel that there were three potentially limiting factors present which account for the adverse alterations in cardiorespiratory variables. These factors are: 1) the degree of ERL; 2) the aerobic capacity of the subjects; and 3) the relative physical demand of the protocol. Variously, one or several of these factors affected these studies.
A. ERL Chosen in Previous Studies

The most influential factor was that, with rare exception, the ERL employed in previous studies has been unphysiologic. Even in studies where the expiratory resistance has been described as "small," for example, in a study by Poon and co-workers (1987) (expiratory resistance = 8 cm H$_2$O · L$^{-1}$ · s$^{-1}$), the resistance appears to have been too great to elicit all but limiting cardiorespiratory effects.

B. Aerobic Capacity of the Subjects

Given the fact that FL can occur during strenuous exercise in highly fit athletes, and presupposing that moderate ERL may shift EELV upward and away from FL, then to elicit any potentially beneficial cardiorespiratory alterations with ERL during exercise the subject pool would have to have a relatively high aerobic capacity and the protocol would need to be one in which the physical demands were rather strenuous.
To our knowledge, there hasn't been a study employing ERL in which the subjects have had superior aerobic capacity. Typically the subjects' mean \( VO_{2\max} \) values have been below 45 ml·kg\(^{-1}\)·min\(^{-1}\). The following studies of ERL offer representative subject mean values for \( VO_{2\max} \): 27.3 in Pellegrino et al., (1993); 39.9 in Goldstein et al., (1975); and \( \leq 45.0 \) in Faulkner et al., (1977). Subjects in these ranges of aerobic capacity would not place the ventilatory demand on the pulmonary system which would precipitate FL (Johnson et al., 1992).

C. Relative Physical Demand of the Protocol

The third factor precluding cardiorespiratory benefit in response to ERL has been that tests have either been resting tests or low-intensity submaximal exercise tests, neither of which protocol would elicit FL in healthy, highly-fit subjects (Johnson et al., 1992).
III. Steady-State Responses to ERL

A. EELV Responses to ERL

Not unexpectedly, in the steady-state (75 % P_{Omax}) comparisons, EELV was increased with ERL. However, what may have been a bit surprising, in view of previous studies employing ERL, was the magnitude of change in response to so little ERL. In our study, during steady-state, the P_{aO} comparison was 1.57 ± 0.17 (ERL) vs 1.13 ± 0.13 cm H_{2}O (control), a difference of only 0.44 ± 0.09 cm H_{2}O; yet, the comparison in EELV was 3.11 ± 0.27 vs 2.45 ± 0.16 L, ERL vs control, respectively, a difference of 0.67 ± 0.29 L. EELV typically increases in response to expiratory loading (Garrard and Lane, 1977; Goldstein et al., 1975; Gothe and Cherniack, 1980; Grunstein et al., 1975; Hill et al., 1985; Pellegrino et al., 1993; Poon et al., 1987; Remmers and Bartlett, 1977). In the Garrard and Lane study (1977), the experimental expiratory resistance was created by reducing the expiratory internal diameter to 5 mm (no pressure measurements were made). (N.B. This expiratory diameter
was only 1/4 that of our resistor.) In their protocol, resting data was collected, while the subjects rebreathed, so that carbon dioxide progressively increased ventilation. At maximum ventilation, FRC was observed to increase by $1.71 \pm 0.67$ L. In the study by Pellegrino and coworkers (1993), a threshold resistance of 5 cm H$_2$O at low ventilation levels ($V_E = 54.5$ L·min$^{-1}$) brought about an increase in EELV of 0.15.

**B. Previous Studies**

In previous studies, however, the amount of ERL generally was far greater than the peak-expiratory pressure ($P_{ao}$) which we employed. In these studies, ERL ranged from 5 to 372 cm H$_2$O (Gerretelli et al., 1969; Goldstein et al., 1975; Gothe and Cherniack, 1980; Martin et al., 1980; and Zechman et al., 1976). We suspected that the ERL used in the former studies had been physiologically overwhelming. An indicator of this is that as the amount of ERL progressively increased, the $V_E$ invariably decreased proportion-
ately (Zechman et al., 1976; Poon et al., 1987; Goldstein et al., 1975).

C. Rationale for ERL Choice

i. Empirical Basis

It should be noted that at the outset of this line of inquiry (in the altitude ERL study), we had chosen several expiratory orifices that we felt approximated the degree of physiologic back-pressure which athletes generate in restricting expiratory airflow during strenuous exercise, and therefore, we do not find it strange that so little ERL should have such a dramatic effect on EELV, and on some of the other cardiorespiratory variables as well. This acute elevation of EELV may have been a heightened response in this group of inveterate athletes, considering that significantly elevated EELV (FRC) has been seen as a chronic adaptation to years of endurance athletic participation (Erikson et al., 1978; Kaufmann et al., 1974).

We were also encouraged to employ this small amount of ERL since it had been this precise amount of ERL which had successfully
mitigated EIH in two previous studies at altitude (Fee et al., 1992; McClaran et al., 1992).

ii. Physiological Analog to Slight ERL: Glottal "Breaking"

There is also a definite physiological phenomenon which would suggest that the slight degree of ERL which we used in our studies may have been physiologically appropriate. The case in point is glottal "breaking" and the response of the glottal aperture to increased airflow.

The cumulative total resistance of the respiratory system ($R_{rs}$) is generally taken to be 3 cm H$_2$O/L/sec (Mead and Agostoni, 1964), with resistance being somewhat greater during expiration than inspiration (England et al., 1982). The majority of this resistance is offered by the upper airways, and in particular by the glottis (Gautier et al., 1973), since all airflow must pass through the opening in the larynx, the glottis (England et al., 1982). During quiet breathing, part of this differential in $R_{rs}$ between inspiration and expiration is due to the fact that the glottis aperture widens during
inspiration and then narrows during expiration (Proctor, 1964). A great deal of this expiratory resistance offered by the glottis, however, is progressively lost during graded exercise, in response to concomitant factors, which are: 1) increased airflow rates and 2) increased thoracic gas volume (that is, end-inspiratory lung volume) increase (Stanescu et al., 1972).

During panting maneuvers (Stanescu et al., 1972) and during exercise hyperpnea (England et al., 1982), a positive correlation has been observed between glottal aperture diameter and airflow rate during expiration. Therefore, as ventilation increases, expiratory breaking by the glottis diminishes. Hyatt and Wilcox, (1961) found that $R_{rs}$ was reduced 43% due to glottal aperture widening during hyperventilation.

The other stimulus for removal of expiratory glottal breaking is increased thoracic gas volume, with glottal aperture having been observed to be at its widest at 100% TLC (Stanescu et al., 1972). End-inspiratory lung volume (EILV) has been observed to approach 100% TLC during maximal exercise (Henke et al., 1988).

The foregoing suggests that the small amount of ERL which we employed may have been in the range of that which was lost due to
progressive glottal widening. This loss of glottal expiratory airflow braking would decrease pressure in the airways, which would lower the transmural pressure threshold for dynamic airway compression in those airway segments which are most susceptible to collapse. It may be significant that the aperture chosen for ERL herein created increasingly higher $P_{ao}$'s at the highest $V_E$'s (Figure 1), when glottal braking is minimal.

D. Slight ERL as Stimulus to Increased EELV

The ERL presented during the steady-state exercise at 75% $P_{O_{max}}$ (1.57 vs 1.13 cm H$_2$O, ERL vs control, respectively) may have offered sufficient stimulus to shift EELV upward, and away from the FL which occurs during heavy to maximal exercise. Averting FL would certainly enhance $V_E$ (Jensen et al., 1980). This adjustment may have both conscious and reflex components.

Gottfried and colleagues (1973) have observed that small changes in resistance to airflow are readily perceived and discriminated within 14 ± 2 % of baseline resistance. In the
steady-state series, the increase in $P_{ao}$ was 0.44 cm H$_2$O, which represents a 28.0 % increase in $P_{ao}$, with ERL vs control, which would certainly appear to be a great enough difference in expiratory resistance to be consciously perceived by the subjects.

Reflexively, obstruction to expiratory airflow may be sensed by extrathoracic tracheal stretch receptors which mediate a prolongation of $T_e$ (Remmers and Bartlett, 1977). In our study, $T_e$ was unchanged (see Appendix), which concurs with the observations of Garrard and Lane, (1977), who observed neither $T_i$ nor $T_e$ to change with ERL.

**E. ERL and Enhanced Inspiratory Effort**

ERL does provoke greater inspiratory effort (Goldstein et al., 1975; Gothe and Cherniack, 1980) which, in turn, would entail increased elastic work of breathing (Yamashiro and Grodins, 1971). It may be that with ERL, however, opting to do more inspiratory work would be a beneficial tradeoff for the athlete. Although Roussos et al. (1979) have noted that breathing at hyperinflated
lung volumes greatly disadvantages the inspiratory muscles, decreasing efficiency and increasing vulnerability to fatigue, in our study, it doesn't 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 ($P_{\text{mus}}$) (Younes, 1991). Our highly-trained endurance athletes would very likely have developed superior respiratory muscle strength (Lieth and Bradley, 1976), so that absolute increases in respiratory muscle effort would represent smaller percentages of $P_{\text{mus}}$, making them less susceptible to respiratory muscle fatigue than less highly-fit subjects. However, increased exercise duration with our ERL in place will be necessary to test this presumption.

i. End-Inspiratory Lung Volume and Inspiratory Effort

During steady-state 75 % $P_{\text{O}_{\text{max}}}$ conditions, end-inspiratory volume (EILV) was 72.5 % ± 1.23 % vs 82.2 % ± 1.46 % of total lung capacity (TLC), control vs ERL, respectively (Table 5), presenting
circumstantial evidence of increased inspiratory effort. This increase in EILV with ERL, increasing lung volume to 82.2 % TLC, would not appear to have required a great increase in inspiratory effort, since 82.2 % TLC would represent 77.5 % of vital capacity (See Table 5), still within the 20 to 80 % of vital capacity range, which is the lung volume range of optimum compliance, and the range in which the elastic work of breathing is at a minimum (Younes, 1991).

F. Effect of Increased Inspiratory Effort on $V_E$

Ordinarily, increasing inspiratory effort alone has little effect on $V_{E_{max}}$; in fact, doubling of average maximal inspiratory pressure ($P_{I_{max}}$) normally results in increased $V_{E_{max}}$ of only 7 % (Younes, 1991). At steady-state, in the present study, the increase in $V_E$ was 7.25 %, that is, 105.9 ± 2.92 (ERL) vs 98.7 ± 4.38 L·min⁻¹ (control); however, it is doubtful that the increased inspiratory effort even approached $P_{I_{max}}$, since a subject would have to be operating at an EILV approaching TLC to exert $P_{I_{max}}$ (Younes, 1991). As can be seen
Table 5. Lung volumes and capacities during steady-state exercise at 75 % \( \text{PO}_{\text{max}} \). End-expiratory lung volume, EELV; tidal volume, \( V_T \); end-inspiratory lung volume, EI\( L \)V; total lung capacity, TLC; percentage of TLC, % TLC; percentage of vital capacity, % VC; and inspiratory reserve volume, IRV. TLC, EI\( L \)V, % TLC, % VC and IRV were taken or derived from tables 1 and 5.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>ERL</th>
</tr>
</thead>
<tbody>
<tr>
<td>EELV (L)</td>
<td>2.45</td>
<td>3.11</td>
</tr>
<tr>
<td>( V_T ) (L)</td>
<td>2.78</td>
<td>2.80</td>
</tr>
<tr>
<td>EI( L )V* (L)</td>
<td>5.23</td>
<td>5.92</td>
</tr>
<tr>
<td>% TLC</td>
<td>72.5</td>
<td>82.2</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>7.21</td>
<td>7.21</td>
</tr>
<tr>
<td>IRV (L)</td>
<td>1.98</td>
<td>1.29</td>
</tr>
<tr>
<td>% VC</td>
<td>65.9</td>
<td>77.5</td>
</tr>
</tbody>
</table>
from Table 5, with ERL there still remained 1.3 L of inspiratory reserve volume (IRV), which was considerably below from TLC. We surmise that the observed increases in $V_E$ associated with ERL were not at the expense of extraordinary inspiratory effort but rather more probably due to a moderate and manageable enhancement of inspiratory effort. Our subjects did not complain of increased work of breathing with ERL, but, in fact, anecdotally they found it easier.

G. Mean Expiratory Flow

Mean expiratory flow ($V_T/T_e$) was greater ($p<0.05$) with ERL, this is in contrast to previous studies, in which reductions in $V_T/T_e$ were observed in consequence to ERL (Pellegrino et al., 1993; Poon et al., 1987; Wood and Bryan, 1978). In these studies, the amounts of ERL were 5, 8, and 5-10 cm H$_2$O, which may have been too great, that is, although these large ERLs did increase EELV, their thwarting effects on expiratory flow may have contributed to the reductions in $V_E$. The increased $V_T/T_e$ which was associated with the slight ERL of our study may have been the consequence of
elevated EELV, since maximal expiratory flows are greater at increased EELV (Beck et al., 1991).

None of the other cardiorespiratory variables at steady-state were different with ERL (see Tables 3, and 6 of the Appendix). At 75 % PO\textsubscript{max} subjects may have been below threshold in exhibiting limitation in cardiorespiratory variables, which underwent limitation at higher levels of intensity during the graded VO\textsubscript{2max} tests.

IV. VO\textsubscript{2max} Test Series

A. Tidal Volume Response to ERL

The first available strategy for increasing $V_E$ in response to increases in ventilatory demand associated with graded exercise is to increase $V_T$ (Johnson et al., 1992). $V_T$ progressively increases with the increasing ventilatory demand of graded exercise, and reaches a plateau at approximately 50-60 % of vital capacity (Lind and Hesser, 1984; Younes and Kivinen, 1984). In addition to the
normal expansion of $V_T$ with graded exercise, there is a predictably greater increase in $V_T$ in response to ERL (Grunstein et al., 1975; Hill et al., 1985, Poon et al., 1987). Throughout our comparison $VO_{2max}$ tests, $V_T$ was greater, and significantly greater ($p \leq 0.05$) at 80 % $PO_{max}$ with ERL vs control (Figure 2), with little or no change in breathing frequency (BF), [except at $VO_{2max}$, where BF was higher ($p \leq 0.05$) with ERL (Table 2)]. The significance of this increase in $V_T$ lies in the fact that, normally, further expansion in $V_T$, after it has reached a plateau, is limited by the mechanical constraints of the pulmonary system (that is, FL as described by the descending profile/envelope of the MEFV curve) (Bye et al., 1983), and is relatively fixed for a given FRC/EELV (Hanson et al., 1982). Because we did observe increases in $V_T$ in response to ERL, we conclude that to some degree these mechanical constraints were ameliorated. By shifting EELV upward and away from the flow-limiting consequences of lower lung volumes, $V_T$ could increase, as seen here.
B. Relationship between Tidal Volume, EELV, and \( V_E \)

Since we employed the same resistor in these graded \( \text{VO}_2\text{max} \) tests as in the EELV series, we presumed that there had been an increase in EELV in response to ERL, and that the increased \( V_T \) (Table 3) was correspondingly shifted to higher lung volumes in the graded \( \text{VO}_2\text{max} \) series, as well. We think that this evident increase in \( V_T \) and the probable increase in EELV explains (Beck et al., 1991) the significantly increased \( V_E \) at 80, 85, and 90 % \( \text{PO}_\text{max} \), and at \( \text{VO}_2\text{max} \).

In the range of exercise intensity of this study, it is probable that under control conditions the athletes experienced expiratory air flow limitation (FL) (Johnson et al., 1992). FL over a significant portion of expiratory \( V_T \) flow has been demonstrated over the range of intense-to-maximal exercise (the range of our study), and specifically, developing at \( \geq 83 \% \) of \( \text{VO}_2\text{max} \) in young \((25 \pm 1 \text{ yr})\) competitive elite athletes (Johnson et al., 1992). In exceptionally fit older \((69 \pm 1 \text{ yr})\) athletes, this occurred in the range of 50 to 75
% VO₂max (Johnson et al., 1991). FL at lower intensities in fit, older athletes is due to loss of elastic recoil of the lung (Frank et al., 1957). With losses in elastic recoil, the static lung recoil pressure at any given lung volume is lower, so that greater effort (pleural pressure) is required during expiration (Johnson et al., 1991). It is on account of this schema that maximal expiratory airflow rates decline with age (Levitzky, 1984). Since the mean age in our study was 33.5 ± 7.3 yr, we may well expect FL to have occurred somewhat below 83 % of VO₂max, which was the intensity at which FL was observed in the Johnson (1992) study.

i. Averting Flow Limitation with Increased EELV

Increasing EELV is a strategy by which FL can be averted (Johnson et al., 1992) and this may be one explanation for improved V̇E with ERL in the VO₂max series. This strategy naturally occurs as exercise intensity progresses from heavy to maximal intensity, that is, the depressed EELV, that develops in moderate to heavy exercise (Henke et al., 1988; Kagawa and Kerr, 1970; Kiers et al.,
1980; Klas and Dempsey, 1989;) returns to near-resting level during the progressively greater FL that accompanies maximal intensity exercise (Johnson et al., 1991; Johnson et al., 1993). This response may be as much weakness as strategy, since expiratory muscles progressively lose their ability to maintain a reduced EELV as the respiratory rate increases (Younes, 1991). In any case, it appears that this tardy rise in EELV seen with maximal exercise comes about only in response to severe FL. When FL is less than severe, there is little elevation in EELV without ERL.

V. Breathing Pattern Selection

In other studies of loaded breathing, the spontaneously chosen breathing pattern wasn't necessarily appropriate to minimize $O_2$ consumption (Killian et al., 1982) but rather one that minimized the sense of effort (Jones, 1991). Anecdotally, we might add that in our study many of the athletes, without solicitation, commented that "it felt easier" or "I felt stronger this time (or last time)," unknowingly referring to the ERL $VO_{2\text{max}}$ test.
A. Advantageous Modifications in Breathing Patterns with ERL

i. Abrupt Curtailment of Expiration with ERL and/or FL

Beck et al. (1991) have speculated that expiratory resistance, for example, like that sensed in the COPD patient, may bring about a derecruitment of expiratory muscle activity, causing inspiration to begin abruptly before expiration is completed. In normal subjects, Pellegrino and colleagues (1993) observed a direct relationship between FL and increases in EELV and have suggested that dynamic compression of the airways, and the resistance to expiration that it offers, may stimulate a reflex mechanism which causes expiration to end prematurely. This curtailment of expiration causes an "auto-PEEP" effect, in which airway pressure still exceeds atmospheric at end-expiration (Beck et al., 1991). This appears to be evident in our $P_E$ single-breath pressure traces (Figure 6), in which there was a more abrupt decline of pressure at end-expiration with ERL vs control.
ii. Expiratory Pressure Plateau with ERL

Along with an apparently sharper initial rise in expiratory pressure, there was also a pattern of prolonged elevation (plateau) of expiratory pressure. This may serve as yet another strategy for averting FL. It would appear likely that this protracted duration of airway pressure helped resist FL caused by airway collapse. Johnson and colleagues (1992) have suggested that exerting greater effort earlier in the expiratory phase (when the airflow generation is still effort-dependent) would be one strategy to mitigate FL. In our study, this elevated plateau in $P_E$ associated with slight ERL may have splinted the airways, and effectively prolonged the effort-dependent stage of expiration. If so, the main effect may have been on airways in the lower, dependent regions, which are susceptible to dynamic compression and begin to undergo FL earliest (Hyatt et al., 1973).
VI. Increased $P_{\text{eff}}$ with Increased EELV

When exercising athletes reach FL, pleural pressure ($P_{\text{pl}}$) has met or exceeded effective pleural pressure ($P_{\text{eff}}$), which is the minimal pressure to drive maximal expiratory flow, as described by isovolume pressure-volume (IVPV) curves (Beck et al., 1991). Exceeding $P_{\text{eff}}$ represents a waste of energy, and can lead to a decrease in expiratory flow from dynamic compression of airways (Ingram and Schilder, 1966; Mead et al., 1967). However, while breathing at elevated lung volumes, as the athletes in our study did with ERL, the $P_{\text{eff}}$ is raised (Bouhuys and Jonson, 1967) and it can move the entire $V_T$ away from the flow-limiting pressures reached at lower lung volumes (Bye et al., 1983), thereby enabling the athlete to exert greater effective expiratory pressure.

VII. Shift from Expiratory to Inspiratory Effort

Ordinarily, only a small fraction of maximum expiratory effort ($P_{E_{\text{max}}}$) is required to generate maximum expiratory flow (Younes, 89
1991). In fact, it is estimated that above 40% of maximal expiratory effort, there is no further effect on expiratory ventilation (Younes, 1991). At heavy workloads (those exceeding a $V_E$ of 120 L·min$^{-1}$) the cost of breathing increases markedly due to the expiratory work done against FL (Johnson et al., 1993). In our study, the strategy/response to ERL may have to effectively divert effort from "wasted" expiratory work, where expiratory $P_{pl} > P_{eff}$, to greater inspiratory activity. With this strategy, FL may have been prevented, and consequently, $V_E$ increased. The inherent logic of this increased inspiratory effort is that inspiration never becomes truly flow limited (Beck et al., 1991), whereas, expiratory FL would be quite probable in our study, given the range of intensity studied and the mean age of our subjects (Johnson et al., 1991; Johnson et al., 1992)

A. Energy Cost with Enhanced Inspiratory Effort

There was a non-significant increase in $VO_2$ (averaging 3.1 ml·kg$^{-1}$·min$^{-1}$) with ERL from 75-85% $PO_{max}$, becoming

90
significantly ($p \leq 0.05$) greater at 90% $P_{O_{\text{max}}}$ and at $V_{O_{2\text{max}}}$. The obvious explanation for this elevated $V_{O_{2}}$ during ERL would be the increased energy requirement to perform the increased inspiratory work. With ERL at $V_{O_{2\text{max}}}$, this additional oxygen consumption amounts to an additional 5.2% in energy expenditure. The cost of breathing during maximal exercise has been estimated to be from 10-16% of total $V_{O_{2}}$ (Aaron et al., 1992; Johnson et al., 1993), in which case, the increased $V_{O_{2}}$ observed at $V_{O_{2\text{max}}}$ in our study would represent a 20-33% increase in cost of breathing compared to control, if the entire increased energy expenditure was accounted for by the respiratory musculature. In any event, it appears, as shall be discussed presently, that the athletes' performances weren't compromised by this increase energy demand.

Another indicator of enhanced inspiratory effort, and consistent with previous ERL studies (Grunstein et al., 1975; Hill et al., 1985; McClaran et al., 1991), was that $V_{T}/T_{i}$ increased ($p \leq 0.05$) at all levels of exercise with ERL (Figure 3).
B. Significance of Increased Maximal Oxygen Consumption

At the several levels of intensity observed in this study, one might justifiably argue that the evidently substantial increases in VO₂ totally reflect the increased cost of breathing attendant to increased inspiratory work. But at VO₂max the argument takes on another character. That VO₂max was significantly (p ≤ 0.05) higher (63.4 vs 60.3 ml·kg⁻¹·min⁻¹, ERL vs control) may appear to be of equivocal benefit in light of the increased inspiratory work of breathing that exercising at elevated FRCs may entail; however, it would appear that some fraction of the increased VO₂max observed with ERL was delivered to the working skeletal muscle which produced significantly (p ≤ 0.05) greater PO₂max (345.7 ± 9.5 vs 352.0 ± 9.9 watts).

VIII. Cardiovascular Implications of Increased EELV with ERL

Breathing at elevated EELV, in response to ERL, had significant cardiovascular implications, as well. In the VO₂max comparisons O₂...
pulse was greater ($p \leq 0.05$) from 75-90 % $P_{O_{max}}$. The conclusion from these data is that cardiac stroke volume must have increased with ERL, which is very likely the cause of increased $VO_{2_{max}}$ and $PO_{max}$. Besides the deleterious ventilatory effects of having $P_{pl}$ exceed $P_{eff}$, excessive $P_{pl}$ (which is likely to occur in very strenuous exercise [Bye et al., 1983; Johnson et al., 1991; Johnson et al., 1992]), can impede venous return (VR) (Pick et al., 1982; Potter et al., 1971). Elevated EELV, as was observed in our study in response to ERL, is associated with decreased pulmonary vascular resistance (PVR) (Wagner and Gale, 1991). Reduced PVR may have accounted for the trend toward lowered HR (>2.0 BPM) during 75-90 % $PO_{max}$ despite the slight, elevations in $VO_{2}$ with ERL. Since these comparisons were made at identical workloads, increased $O_{2}$ pulse would suggest increased stroke volume, secondary to augmented VR.

Another mechanism which may have contributed to augmented stroke volume may have been the more rapid onset of inspiration, which was evidenced in the single-breath tracings of this study. Right ventricular filling is augmented by abrupt, transient increases in right ventricular venous return which occur with the
onset of inspiration (Vermiere and Butler, 1968), and the right heart is more rapidly and completely filled by brief, strenuous inhalations during exercise (Agostoni and Butler, 1991).

IX. Conclusions

We hope that we have invited some suspicion that perhaps in highly-fit athletes the respiratory system while encumbered by a mouthpiece and valve errs during strenuous exercise in selecting an EELV which is below resting FRC. We infer from our findings that increased EELV in response to slight ERL during strenuous exercise served to attenuate both airflow and bloodflow limitations which may be the product of excessive pleural/intrathoracic pressures that developed during active expiration at lower lung volumes. We further conclude that ERL during strenuous exercise can enhance exercise capacity.
APPENDIX

Table 7. Cardiorespiratory data from steady-state 75 % P_{O_{max}}. Ventilatory equivalent for oxygen and carbon dioxide (V_E/V_O2) and (V_E/C_O2), fractional concentration of expired O_2 and CO_2 (F_EO_2), and (F_ECO_2), and respiratory exchange ratio (R). There were no significant differences in any of these values when comparing control to expiratory resistive loading (ERL). All values ± SEM. †Wilcoxon signed rank test.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>ERL</th>
</tr>
</thead>
<tbody>
<tr>
<td>VeqO_2</td>
<td>26.7 ± 0.8</td>
<td>28.1 ± 0.6</td>
</tr>
<tr>
<td>VeqCO_2</td>
<td>25.9 ± 0.8</td>
<td>26.7 ± 0.7</td>
</tr>
<tr>
<td>F_EO_2 (%)</td>
<td>16.35 ± 0.13</td>
<td>16.57 ± 0.13</td>
</tr>
<tr>
<td>F_ECO_2 (%)</td>
<td>3.88†</td>
<td>4.04†</td>
</tr>
<tr>
<td>R (VCO_2 / VO_2)</td>
<td>1.03 ± 0.01</td>
<td>1.05 ± 0.02</td>
</tr>
</tbody>
</table>
REFERENCES


Tucker, A., J. Stager, and L. Cordain. Arterial O\textsubscript{2} saturation and maximum O\textsubscript{2} consumption at moderate-altitude runners exposed to sea level and 3,050 m. JAMA, 252, 2867-2871, 1984.


