MAXIMAL RESPIRATORY PRESSURES AND PULMONARY FUNCTION IN MALE RUNNERS

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ABSTRACT
To determine the effects of long term exercise on respiratory muscle strength, maximal inspiratory (P₂ max) and expiratory (Pₑ max) pressures, pulmonary volumes and capacities and anthropometric parameters were measured in a group of 101 male runners aged 16 to 58 years. The runners exhibited significantly (p < 0.05) lower Pₑ max (202 ± 41 cm H₂O and significantly greater residual lung volumes (RV) (2.08 ± 0.49 L) than predicted values for normal subjects of similar height and age. Forced vital capacities were not different (p > 0.05) from values reported for normal non-smoking populations. These data suggest that running may cause a non-pathological increase in RV, perhaps mediated by reductions in expiratory muscle strength. Additionally, current RV regression equations developed for normal subjects may be inappropriate for use in running populations.

Key words: Maximal respiratory pressures, Residual lung volume, Forced vital capacity, Exercise

INTRODUCTION
Previous studies of training programmes examining the respiratory muscles have shown that these muscles respond to exercise by increasing in strength and endurance (Leith and Bradley, 1976; Martin and Stager, 1981; Keens et al, 1977), similar to other skeletal muscles. Whether these changes occur in the respiratory muscles as a consequence of long term endurance exercise (e.g. running) has not been well documented (Cordain et al, 1982; Robinson and Kjeldgaard, 1982). Furthermore, it is unclear if changes in respiratory muscle strength can alter pulmonary volumes and capacities. In an attempt to answer these questions, we measured maximal inspiratory (P₂ max) and expiratory (Pₑ max) pressures, pulmonary volumes and capacities, and anthropometric measurements in a large group of male runners.

METHODS

Subject Selection
The subjects consisted of 101 Caucasian, male runners between 16 and 58 years of age who had been participating in a running programme for at least one year with a minimum running frequency of three times per week. Subjects were selected from local high school and university cross-country teams as well as from area running clubs. All subjects were non-smokers (for at least five years) and to be free of known respiratory disease. Prior to the initiation of this study, approval was obtained from the Colorado State University Human Research Committee and informed consent was obtained from each subject.

Anthropometry
Prior to pulmonary measurements, anthropometric data were gathered. Measurements included chest girth (GC), distance from the jugular notch to the sternal notch (DJS) and the bicipical diameter (DBA). Measurements were made to the nearest 0.1 cm with a fibreglass anthropometric tape. Each landmark was measured in a standardised manner so that no more than 1% difference existed between repeated trials. Height was measured with a medical-grade stadiometer to the nearest 0.1 cm.

Pulmonary Determinations
All subjects were required not to have exercised within 12 hours of reporting to the laboratory for pulmonary function testing, since forced vital capacities (FVC) have been shown to be reduced, temporarily, following an acute exercise bout (Maron et al, 1979) whereas residual lung volumes (RV) have been shown to be temporarily increased (Girandola et al, 1977). All measurements were made while the subjects were seated and wore noseclips. Subjects made three FVC manoeuvres into a 13.5L spirometer (Warren E. Collins, Braintree, MA) with the largest value being used for data reduction. Residual lung volumes were determined using the closed circuit single breath, oxygen dilution technique (Wilmore, 1969) in conjunction with a modified 10L Stead-Wells spirometer (Warren E. Collins, Braintree, MA) and a nitrogen analyser (Ohio Medical Products, Madison, WI). The spirometer was filled with 7.0 litres of oxygen, and the seated, nose-clipped subjects took three or four normal breaths into a three-way mouthpiece that was opened to room air. After the subjects were asked to exhale maximally, the three-way valve was switched to the spirometer and the subjects were instructed to inspire and expire at two-thirds of their maximum, at a rate of one breath every three seconds. After equilibrium was reached (approximately 6-9 breaths), the subjects were instructed to take a maximal inspiration followed by a maximal expiration. The valve was then turned back to room air and the nitrogen reading at this point was recorded. Three trials were taken, with an interval of 20 minutes between trials, to allow the subject’s lungs to be cleared of the 100% oxygen. Maximal inspiratory pressure measurements were made at RV and maximal expiratory measurements were made at total lung capacity (TLC) following the technique described by Black and Hyatt (1969). The mouthpiece, which occluded the airway, was constructed of thick, non-resilient plastic to prevent the pressures from being dissipated. A digital manometer (Validyne, Model PS 309, Northridge, CA) was interfaced to an IBM personal computer and a peak-detecting algorithm was designed to monitor and record maximal pressures. Pressures were measured consecutively until no further increases occurred, and the highest value was used for data reduction. Predictions of FVC (Berglund et al, 1963; Cotes et al, 1966; Goldman and Becklake, 1959; Kory et al, 1961; Morris et al, 1971; Needham et al, 1954) and RV (Boren et al, 1966; Goldman...
TABLE I
Subject characteristics by age

<table>
<thead>
<tr>
<th>Years</th>
<th>n</th>
<th>Ht (cm)</th>
<th>Wt (kg)</th>
<th>FVC (L)</th>
<th>RV (L)</th>
<th>TLC (L)</th>
<th>PE max cm H₂O</th>
<th>P₁ max cm H₂O</th>
</tr>
</thead>
<tbody>
<tr>
<td>16-19</td>
<td>22</td>
<td>178.2 ± 5.7</td>
<td>70.1 ± 6.1</td>
<td>5.31 ± 0.53</td>
<td>1.76 ± 0.47</td>
<td>7.07 ± 0.86</td>
<td>24.9 ± 4.1</td>
<td>207 ± 43</td>
</tr>
<tr>
<td>20-29</td>
<td>27</td>
<td>180.0 ± 6.4</td>
<td>72.9 ± 9.7</td>
<td>5.50 ± 0.52</td>
<td>1.99 ± 0.53</td>
<td>7.49 ± 0.82</td>
<td>26.6 ± 4.7</td>
<td>204 ± 34</td>
</tr>
<tr>
<td>30-39</td>
<td>25</td>
<td>175.5 ± 6.1</td>
<td>73.1 ± 9.0</td>
<td>5.36 ± 0.42</td>
<td>2.13 ± 0.41</td>
<td>7.49 ± 0.67</td>
<td>28.4 ± 3.6</td>
<td>202 ± 46</td>
</tr>
<tr>
<td>40-49</td>
<td>19</td>
<td>177.8 ± 4.9</td>
<td>80.7 ± 10.6</td>
<td>5.46 ± 0.46</td>
<td>2.41 ± 0.33</td>
<td>7.87 ± 0.71</td>
<td>30.6 ± 3.1</td>
<td>204 ± 38</td>
</tr>
<tr>
<td>50-59</td>
<td>8</td>
<td>178.3 ± 5.2</td>
<td>77.9 ± 9.0</td>
<td>4.68 ± 0.71</td>
<td>2.51 ± 0.31</td>
<td>7.19 ± 0.73</td>
<td>34.9 ± 3.6</td>
<td>178 ± 46</td>
</tr>
<tr>
<td>All groups</td>
<td>101</td>
<td>177.9 ± 5.9</td>
<td>74.2 ± 9.5</td>
<td>5.36 ± 0.53</td>
<td>1.87 ± 0.49</td>
<td>7.23 ± 0.81</td>
<td>25.7 ± 4.9</td>
<td>202 ± 41</td>
</tr>
</tbody>
</table>

Values are means ± SD

and Becklake, 1959; Grimsby and Soderholm, 1963; Needham et al, 1954) were made from previously-reported equations. These data appear in Table IV.

Statistical Analyses

Differences between measured and predicted values for the pulmonary function parameters were determined using a repeated measures ANOVA. An all variable inclusive correlation matrix (Pearson's r) was developed to examine relationships among variables and stepwise multiple regression techniques were employed to develop the prediction equations for the present group of runners.

Validation

A similar, but independent group of subjects (n = 24) were assessed to determine the validity and generalisability of the prediction equations. Criterion measures of PE max, P₁ max, FVC, RV, TLC, and RV/TLC × 100 were obtained from each subject by using the previously described techniques. Predictions of these measures were then computed from the equations developed in this study. Repeated measures ANOVAs were used to determine if systematic differences existed between the criterion and predicted means.

RESULTS

Subject characteristics for the entire sample as well as by ages appear in Table I. Mean PE max exhibited no significant (P > 0.05) relationship (Table II) to age, suggesting that expiratory muscle strength does not decline significantly after maturity in runners. However, in the 50 to 59 year age group, a 13% reduction in mean PE max was noted. Conversely, mean P₁ max exhibited a trend for decreasing pressure with increasing age, as reflected by the significant relationship (p < 0.001, r = -0.43) between age and P₁ max. None of the pulmonary volumes and capacities measured in the present study showed significant relationships to either PE max or P₁ max.

Except for the 50 to 59 year age group, no trend was apparent for the expected decline in FVC with increasing age, as reported in other large scale studies of normals. Forced vital capacity showed no significant (p > 0.05, r = -0.17) relationship to age. Similar to studies of normals, there was an age-related increase in both RV and RV/TLC × 100. Residual lung volumes were significantly related to age (p < 0.001, r = 0.48).

TABLE III
Multiple regression equations for the prediction of various pulmonary parameters (n = 101)

<table>
<thead>
<tr>
<th>Equation</th>
<th>R</th>
<th>R²</th>
<th>SEE</th>
</tr>
</thead>
<tbody>
<tr>
<td>PE max (cm H₂O) = -0.43 (A) + 215*</td>
<td>0.13</td>
<td>0.017</td>
<td>41.0</td>
</tr>
<tr>
<td>P₁ max (cm H₂O) = 0.99 (A) - 161</td>
<td>0.43</td>
<td>0.185</td>
<td>25.0</td>
</tr>
<tr>
<td>FVC (L) = 0.043 (H) - 0.008 (A) - 2.06</td>
<td>0.50</td>
<td>0.250</td>
<td>0.47</td>
</tr>
<tr>
<td>RV (L) = -0.020 (A) + 0.0306 (H) - 3.99</td>
<td>0.61</td>
<td>0.372</td>
<td>0.39</td>
</tr>
<tr>
<td>TLC (L) = 0.0732 (H) + 0.0132 (A) - 6.21</td>
<td>0.56</td>
<td>0.314</td>
<td>0.68</td>
</tr>
<tr>
<td>RV/TLC × 100% = 0.248 (A) + 18.2</td>
<td>0.59</td>
<td>0.348</td>
<td>4.00</td>
</tr>
</tbody>
</table>

A = age in years; H = height in cm
* p < 0.24, all other equations p < 0.001

Simple and multiple regression equations employing age or height as predictors of pulmonary function variables appear in Table III. None of the three anthropometric chest dimensions were identified as significant predictors of any pulmonary function variable when forward stepwise multiple regression techniques were applied to the data. Of the six equations reported in

TABLE IV
Validation of prediction equations (n = 24)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Criterion</th>
<th>Prediction</th>
<th>Error of Estimate</th>
<th>SEE</th>
</tr>
</thead>
<tbody>
<tr>
<td>PE max (cm H₂O)</td>
<td>201.0</td>
<td>195.6</td>
<td>5.40</td>
<td>41.0</td>
</tr>
<tr>
<td>P₁ max (cm H₂O)</td>
<td>-132.0</td>
<td>-127.3</td>
<td>4.70</td>
<td>25.0</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>5.28</td>
<td>5.33</td>
<td>0.05</td>
<td>0.47</td>
</tr>
<tr>
<td>RV (L)</td>
<td>1.94</td>
<td>2.10</td>
<td>0.16</td>
<td>0.39</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>7.27</td>
<td>7.37</td>
<td>0.10</td>
<td>0.68</td>
</tr>
<tr>
<td>RV/TLC × 100%</td>
<td>26.7</td>
<td>27.1</td>
<td>0.40</td>
<td>4.00</td>
</tr>
</tbody>
</table>

All error of estimate values p > 0.05
Table III, only the equation for $P_E$ max was not a significant
(p = 0.24) predictor of pulmonary function. The equations
reported in Table III were cross-validated within a similar
but independent group of runners (n = 24), and no
significant differences ($p > 0.05$) were demonstrated
between any of the observed measurements and those
predicted from the equations in Table III. Also, all of the
error of estimates fell within one SEE of each equation.
These validations may be seen in Table IV.

![Diagram](image)

**Fig. 1: Relationship of FVC to age in normal non-smokers and in runners.**

Predicted values for FVC were calculated from six
equations reported for normals. Observed values were
significantly larger ($p < 0.05$) than values predicted from
five of six regression equations (Table V). Only in the study
by Morris et al (1971), who evaluated FVC in a large sample
of non-smokers, was there no difference between observed
and predicted values. Observed values for RV were
significantly larger ($p < 0.05$) than predicted for four
previously reported RV regression equations, while observed $P_E$ max values were significantly lower ($p < 0.05$)
than those reported for normals by Black and Hyatt (1969).
These results may also be seen in Table V.

**TABLE V**

<table>
<thead>
<tr>
<th>Source</th>
<th>Observed</th>
<th>Predicted</th>
<th>Per cent of Predicted</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Berglund et al, 1963</td>
<td>5.36</td>
<td>5.13</td>
<td>104.5</td>
<td>0.23</td>
</tr>
<tr>
<td>Goldman et al, 1959</td>
<td>5.36</td>
<td>5.09</td>
<td>105.3</td>
<td>0.27</td>
</tr>
<tr>
<td>Needham et al, 1954</td>
<td>5.36</td>
<td>4.71</td>
<td>113.8</td>
<td>0.65</td>
</tr>
<tr>
<td>Kory et al, 1961</td>
<td>5.36</td>
<td>5.04</td>
<td>106.3</td>
<td>0.32</td>
</tr>
<tr>
<td>Morris et al, 1971</td>
<td>5.36</td>
<td>5.35</td>
<td>99.8</td>
<td>0.01*</td>
</tr>
<tr>
<td>Cotes et al, 1966</td>
<td>5.36</td>
<td>5.07</td>
<td>105.7</td>
<td>0.29</td>
</tr>
<tr>
<td>RV (L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Goldman et al, 1959</td>
<td>2.08</td>
<td>1.88</td>
<td>111.0</td>
<td>0.20</td>
</tr>
<tr>
<td>Needham et al, 1954</td>
<td>2.08</td>
<td>1.92</td>
<td>108.6</td>
<td>0.16</td>
</tr>
<tr>
<td>Boren et al, 1966</td>
<td>2.08</td>
<td>1.50</td>
<td>139.1</td>
<td>0.58</td>
</tr>
<tr>
<td>Grimby et al, 1963</td>
<td>2.08</td>
<td>1.55</td>
<td>134.2</td>
<td>0.53</td>
</tr>
<tr>
<td>$P_E$ max (cm H$_2$O)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black &amp; Hyatt, 1969</td>
<td>202.0</td>
<td>236.0</td>
<td>85.6</td>
<td>-34.0</td>
</tr>
<tr>
<td>$P_I$ max (cm H$_2$O)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black &amp; Hyatt, 1969</td>
<td>-130.0</td>
<td>-126.0</td>
<td>103.2</td>
<td>-4.0*</td>
</tr>
</tbody>
</table>

* $p > 0.05$, all other differences $p < 0.05$

**DISCUSSION**

The results of the present study demonstrate that runners
have $P_I$ max values similar to values reported by others
(Table VI), but have values for $P_E$ max lower than observed
in other studies of normals. These data suggest that
running programmes of longer than one year in duration
may decrease maximal expiratory pressures. Ringqvist
(1966) suggested that changes in airway resistance served
as a major stimulus for respiratory muscle hypertrophy.

**TABLE VI**

<table>
<thead>
<tr>
<th>Source</th>
<th>n</th>
<th>$P_I$ max</th>
<th>$P_E$ max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black &amp; Hyatt, 1969</td>
<td>36</td>
<td>-124 ± 22</td>
<td>233 ± 42</td>
</tr>
<tr>
<td>Cook et al, 1964</td>
<td>17</td>
<td>-133 ± 39</td>
<td>237 ± 45</td>
</tr>
<tr>
<td>Ringqvist, 1966</td>
<td>82</td>
<td>-130 ± 32</td>
<td>237 ± 46</td>
</tr>
<tr>
<td>Present group</td>
<td>101</td>
<td>-130 ± 28</td>
<td>202 ± 41</td>
</tr>
</tbody>
</table>

Values are means ± SD

Since airway resistance is related inversely and
curvilinearly to lung volumes (Briscoe and Dubois, 1958),
then airway resistance will be reduced when subjects
breathe at high lung volumes (West, 1984). Pyorala et al
(1968) pointed out that endurance athletes maintain lower
and deeper rhythms of breathing, both at rest and at
exercise than compared to normals. Therefore, the reduced
$P_E$ max demonstrated in the present runners may be
resultant from long-term reductions in airway resistance
from breathing at high lung volumes both at rest and during
exercise. It is relevant to point out that as FRC is reduced
during exercise in normal subjects breathing is
accomplished at lower lung volumes; however, the mean
lung capacity increases, hence the average resistance is
likely to be lower over the entire breath. Maximal
inspiratory pressure was similar to values reported in
normals, indicating that running provides no additional
stimulus for inspiratory muscular strength.

In the present study, all of the subjects were
non-smokers, whereas in several previous studies of $P_E$ max
(Black and Hyatt, 1969; Cook et al, 1964; Ringqvist, 1968),
as well as in studies of RV (Goldman and Becklake, 1958;
Needham et al, 1954; Boren et al, 1966; Cook et al, 1964),
subjects included both smokers and non-smokers. Smoking
could increase expiratory muscle strength by inducing a
muscular training effect brought about by increased
expiratory flow resistance or by chronic coughing.
Therefore, it is possible that differences in $P_E$ max, and
perhaps RV, demonstrated in the present group of runners
relative to normals, resulted from differences in smoking
habits.

The single breath oxygen dilution technique has been
shown to underestimate RV by approximately 6 per cent
when compared with other multiple breath techniques
(Sterk et al, 1980), particularly in smokers and others with
airway obstruction. However, in the present study only
non-smokers were tested, and the measured mean RV was
greater than estimated values from four prediction
equations (Table V). Therefore, it is likely that the elevated
RV demonstrated in runners with the present technique
would be slightly larger should other multiple breath
techniques have been employed.
In one of the few longitudinal studies to examine respiratory strength changes with running programmes, Robinson and Kjeldgaard (1982) reported a 14.4% increase in expiratory pressures in a group of 11 adults (8 females and 3 males) at the end of 20 weeks. No changes occurred in maximal inspiratory pressures with running training. The maximal inspiratory pressure data of the present study is in agreement with the notion that running causes little change in inspiratory muscle strength. However, it is difficult to compare the expiratory strength changes observed by Robinson and Kjeldgaard (1982) with the present data, since values for both men and women were combined in the earlier study. 

Women comprised 8 of 11 subjects in that study and, since women have maximal inspiratory pressure 30% lower than males (Ringqvist, 1966), the mean value for P_{E max} even after 20 weeks of training, was considerably lower than that observed in the present study (156 vs 202 cm H_{2}O). Additionally, the values of the previous study were taken after five months of training, whereas the subjects in the present study had trained for a minimum of one year.

Flow-volume and volume-time curves illustrate that the limitation to RV in healthy subjects is primarily static where a balance exists between opposing chest wall and lung elastic recoil forces (Leith and Mead, 1967). Thus a reduction in expiratory muscle strength would be expected to result in an increased RV. In support of this runners in all age groups in the present study exhibited a significantly larger RV than predicted, while exhibiting a significantly lower P_{E max} than predicted. Other cross-sectional studies that examined individuals who have been engaged in aerobic activities for many years, show that these endurance athletes maintain elevated values for RV and FRC (Pyorala et al, 1968; Eriksson et al, 1978; Kaufmann et al, 1974; Magel and Anderson, 1969; Maksud et al, 1971). Both Pyorala et al (1968) and Maksud et al (1971) speculated that an elevated FRC would tend to minimise fluctuations in alveolar gas tensions during the slower and deeper pattern of breathing exhibited by endurance athletes.

Short-term (6-12 weeks) longitudinal studies have shown that RV decreases in subjects undertaking an aerobic training programme (Bachman and Horvath, 1968; Girandola, 1976; Girandola and Katch, 1973; Katch, 1971; Katch et al, 1969; Lynch et al, 1968; Swenson and Zauner, 1967; Wilmore et al, 1970), and it has been hypothesised that these reductions are resultant from increased strength of the respiratory expiratory muscles (Cordain et al, 1982; Girandola and Katch, 1973). This reduction in RV with aerobic training may only be a transitory effect. Swenson and Zauner (1967) demonstrated a 25% reduction in RV in 10 sedentary men after two weeks of conditioning; however, after eight weeks of training, only a 9.4% reduction was noted. Thus, it may be that RV is initially reduced by increased expiratory muscle strength, but eventually, after a year or more of aerobic training, reductions in airway resistance, brought about by breathing at high lung volumes, serve to reduce expiratory muscle strength, thereby increasing RV.

In the present study observed FVC was not different from that predicted by the equation of Morris et al (1971) whose sample consisted entirely of healthy, non-smokers. However, observed FVC values were significantly larger than five predicted values (Table V) in which equations were developed from both smokers and non-smokers. These data suggest that running causes little or no change in FVC in healthy, non-smoking populations. Although exercise may not cause increases in FVC, it may be responsible for slowing the decline in FVC normally associated with aging. Pyorala et al (1968) noted that the decrease in FVC occurred less rapidly in a group of former champion endurance athletes. The results of the present study also support this observation with differences occurring after age 33.

Since the current study is of a cross-sectional nature, it is difficult to determine more precisely whether differences in pulmonary function between runner and normals occur as an adaptation to exercise or simply are a result of selection. Additionally, a control group was not used in this study; therefore, the interpretation of the treatment effects were somewhat compromised. Should systematic errors have occurred in the measurement of the pulmonary variables, it would be inappropriate to compare the present results with other studies. Further longitudinal studies of greater than one year, evaluating pulmonary changes in runners, are needed to determine more precisely mechanisms responsible for the larger RV demonstrated in these individuals.

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