Novel method for measuring effects of gas compression on expiratory flow

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Maximal Expiratory Flow and Forced Expiratory Volume in the First Second of a Forced Expiratory Maneuver (FEV1) are routinely used for evaluation of respiratory function in clinical practice. A unique relationship exists between maximal expiratory flow, lung volume, and intrathoracic pressure generated during a forced expiratory maneuver. The maximal expiratory flow is determined by the lung elastic recoil pressure, the upstream frictional pressure loss, and the relationship between cross-sectional area and transmural pressure at the choke point (9, 23). However, the lung elastic recoil pressure is the primary determinant of the maximal expiratory flow (21) and is influenced by the absolute lung volume (7). In normal subjects, at lung volumes near total lung capacity (TLC), expiratory flow increases with efforts that are more forceful. However, below 75% of TLC, an increase in effort does not result in an increase in flow, and it may even paradoxically reduce the flow. For lung volumes <75% of TLC, the major determining factor in maximum achievable expiratory flow is the lung volume (21).

In a typical clinical pulmonary function laboratory, flow is measured with a flowmeter at the mouth level, and FEV1 is obtained by integration of this flow. However, when the volume change during a forced maneuver is measured simultaneously by expired volume using a pneumotachograph and by a volume displacement body plethysmograph, the expired volume may differ considerably from the volume change measured with body plethysmograph. This difference in volume change is primarily because of compression of intrathoracic gas. According to the basic laws governing gas flow in a rigid tube, gas with an access to an opening does not undergo dynamic compression unless the particle velocity of gas in the tube exceeds 0.3 of the speed of sound. However, in an airway that is nonrigid, the governing process of flow limitation is the wave speed concept. At high and mid lung volumes, as flow increases to equal that of wave speed, flow limitation will occur regardless of the driving pressure gradient (6, 15, 20). It is at this point that gas compression may occur, even in areas of lung with access to open airways. In contrast, at lower lung volumes, maximal flow is primarily determined by the coupling of viscous pressure losses and airway mechanical properties. Furthermore, dynamic narrowing of airways during a forced expiratory maneuver in subjects with flow limitation can create areas of trapped gas in the lung (20) and result in gas compression.

Figure 1 represents a forced maneuver in a normal subject and a subject with chronic obstructive pulmonary disease (COPD) using simultaneous measurements obtained with pneumotachograph at the mouth level and body plethysmograph. The data in Fig. 1 demonstrate that volume change measured at the mouth level is not a true representation of the absolute change in lung volume. This is consistent with data from other investigators (4, 5). A flow-volume loop of a patient with severe COPD is shown in Fig. 1B. This patient’s measured TLC was 10.23 liters, and his predicted TLC was only 6.69 liters. Interestingly, this subject’s forced vital capacity (FVC) was only 2.71 liters. At 50% of vital capacity (130% of predicted TLC in this subject), expiratory flow was only 0.4 l/s. At this same flow, the volume detected by the body box is only 90% of predicted TLC. The flow-volume loop generated by using plethysmograph shows a flow of 1 l/s higher than mouth flow at 50% of FVC. In contrast, in a subject with normal lung function (Fig. 1A), there is no appreciable differen-

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ence between exhaled volume and volume measured with the plethysmograph, and any minor gas compression may be of abdominal gas and airway compression. Interestingly, as the subject relaxes at the end of the FVC maneuver and gas compression is reduced, the lung volume (recorded with plethysmography) increases while there is continuing expiratory flow. Therefore, correlating maximal expiratory flow to the volume measured at the mouth level is a distorted portrayal of the relationship between maximal expiratory flow and absolute lung volume in subjects with expiratory flow limitation (10).

The flow-volume relationship is dependent on the magnitude of thoracic gas compression. During a force maneuver, intrathoracic pressure may reach well above 100 cmH$_2$O. This high pressure can cause significant compression of intrathoracic gas in areas of lungs that are upstream of the choke points. For ease of calculation, we assume a barometric pressure of 1,000 cmH$_2$O. Therefore, a 100-cmH$_2$O intrathoracic pressure will compress the alveolar gas by 10%. Assuming that lung volume of a normal adult man at $50\%$ of vital capacity is 3 liters, a 0.3-liter reduction in absolute lung volume resulting from this gas compression would occur if this volume of gas was trapped. If the slope of the flow-volume loop were about $0.6$ l/s, a difference of $0.6$ l/s would be generated because of this gas compression. Therefore, given a typical normal flow of 4 l/s, a variability of $15\%$ (0.6 l/s) in flow will be seen because of thoracic gas compression. This degree of

Fig. 1. Flow vs. volume loops and volume vs. time of forced expiratory maneuvers in a normal subject and in a subject with chronic obstructive pulmonary disease (COPD) using both expired mouth flow (thin line) and volume change measured with a volume body plethysmograph (thick line). A: flow-volume loop in a normal subject measured using both mouth flow and plethysmograph. Although the peak expiratory flow (PEF) is slightly delayed when measured by plethysmograph, both methods show 0 flow at residual volume (RV). In contrast, as shown in B, the flow and volume change recorded with mouth flow is appreciably different from the parameters recorded with plethysmograph. This difference is the result of gas compression. The effect of gas compression is clearly demonstrated at the end of the forced vital capacity (FVC) maneuver. Although the volume obtained from integration of mouth flow stays close to RV, the plethysmograph volume decreases below RV. As the subject relaxes at the end of the FVC maneuver and gas compression is reduced, the volume (recorded with plethysmograph) increases from 95% of TLC to 105% of TLC while there is continuing expiratory flow. In this COPD subject, the measured and the predicted TLC are 10.23 and 6.69 liters, respectively. In both A and B, the x-axis represents volume change both by body plethysmograph [presented as %total lung capacity (TLC)] and integration of mouth flow (thin horizontal line labeled as FVC). C: graph of volume vs. time from the same maneuver in A. The volume change obtained from mouth flow is very similar to that obtained by plethysmograph. D: volume vs. time graph from the same maneuver is shown in B. Interestingly, at the first second of this maneuver, the volume obtained from mouth flow (thin line) is <1 liter, but plethysmograph shows a 2.5-liter volume change. Similar to B, at the end of the maneuver and subsequent to subject relaxation, the plethysmograph volume change reverses.
variability is well above the clinically significant change in volume and flow seen in response to a bronchodilator in subjects with expiratory flow limitation.

Variables affecting gas compression have been studied both in normal subjects and in individuals with expiratory flow limitation. In a study of normal subjects, Jaeger and Otis (12), showed that magnitude of gas compression increased with increasing airway resistance, exhalation flow rate, and lung volume. Large lung volume and expiratory flow limitations are hallmarks of subjects with COPD. Therefore, in subjects with COPD, gas compression may cause significant distortion of the flow-volume relationship and most likely may result in underestimation of the ventilatory capacity (11). In contrast to expired volume, the volume change measured with plethysmograph is an overestimate of expired volume in an FVC maneuver (11).

In this study, we present a novel method, the no-compression method (NCM), to measure the effects of thoracic gas compression on FEV1. This method involves simultaneous measurement of the volume using a volume-displacement body plethysmograph and mouth flow with a pneumotachograph. The NCM allows us to estimate FEV1 corrected for the effect of gas compression. We present a description of the theory underlying the NCM. Furthermore, to examine the effect of gas compression, we tested the NCM in normal, asthmatic, and COPD subjects.

### Glossary

- FEV1: FEV1 obtained by mouth flow
- PFEV1: FEV1/predicted FEV1
- NFEV1: FEV1 obtained by the NCM
- PNFEV1: NFEV1/predicted FEV1
- DFEV1: (NFEV1 − FEV1)/FEV1
- Rle: Expiratory lung resistance during quiet breathing
- EPPEF: Esophageal pressure at the peak expiratory flow
- EPpeak: Maximum esophageal pressure during the expiratory maneuver
- PEF: Peak expiratory flow
- PTP: Transpulmonary pressure
- FEV1-Dif: Difference between the lowest and highest FEV1 values during same session/lowest FEV1 at that session
- NFEV1-Dif: Difference between the maneuvers with lowest and highest NFEV1 from same session/lowest NFEV1 at that session

### METHODS

We studied 11 healthy subjects, 10 subjects with asthma, and 65 subjects with moderate to severe COPD resulting from emphysema. The subjects’ anthropometric and lung function data are shown in Table 1. Asthmatic and COPD subjects had diagnoses of illness based on the criteria of the American Thoracic Society (1). All subjects were clinically stable at the time of the study. The asthmatic and COPD subjects had stopped the use of short-acting bronchodilator for ≥8 h and long-acting bronchodilator for >24 h before lung function tests. The protocol for all subjects was approved by the Institutional Review Board, and each subject gave written consent. All COPD subjects had ceased smoking ≥3 mo before the beginning of this evaluation.

### Table 1. Anthropometric and lung function measurements of study subjects

<table>
<thead>
<tr>
<th>All</th>
<th>Normal</th>
<th>Asthma</th>
<th>COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of subjects</td>
<td>86</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>PEFR, l/s</td>
<td>4.4 ± 2.3</td>
<td>8.2 ± 2.4</td>
<td>5.8 ± 1.7</td>
</tr>
<tr>
<td>EPpeak, cmH2O</td>
<td>126 ± 59</td>
<td>95 ± 34</td>
<td>117 ± 52</td>
</tr>
<tr>
<td>EPPEF, cmH2O</td>
<td>31 ± 21</td>
<td>46 ± 15</td>
<td>39 ± 20</td>
</tr>
<tr>
<td>TLC, %predicted</td>
<td>125 ± 20</td>
<td>106 ± 16</td>
<td>114 ± 18</td>
</tr>
<tr>
<td>FVC, %predicted</td>
<td>77 ± 22</td>
<td>92 ± 15</td>
<td>84 ± 20</td>
</tr>
<tr>
<td>SVC, %predicted</td>
<td>85 ± 18</td>
<td>92 ± 23</td>
<td>87 ± 15</td>
</tr>
<tr>
<td>Rl,c, cmH2O l−1 s−1</td>
<td>16 ± 12</td>
<td>3.2 ± 0.7</td>
<td>9.5 ± 8</td>
</tr>
<tr>
<td>FEF25−75, %predicted</td>
<td>45 ± 26</td>
<td>90 ± 14</td>
<td>66 ± 22</td>
</tr>
<tr>
<td>NFEV1, %predicted</td>
<td>59 ± 25</td>
<td>100 ± 14</td>
<td>79 ± 20</td>
</tr>
<tr>
<td>DFEV1</td>
<td>45 ± 42</td>
<td>10 ± 16</td>
<td>24 ± 20</td>
</tr>
</tbody>
</table>

Values are means ± SD. M, male; F, female; COPD, chronic obstructive pulmonary disease; PEF, peak expiratory flow; EPpeak, peak esophageal pressure during a forced expiratory maneuver; EPPEF, esophageal pressure at PEF; TLC, total lung capacity; FVC, forced vital capacity; SVC, slow vital capacity; Rl,c, expiratory lung resistance; FEF25−75, forced expired volume in 1st second; NFEV1, FEV1 obtained using no compression method; DFEV1, (NFEV1 − FEV1)/FEV1.

subjects seated in an air-conditioned volume-displacement plethysmograph. The frequency response of this plethysmograph is adequate up to 10 Hz, and the box volume measurement is linear up to 25 l/s (19). Volume measurements were obtained by integrating, with respect to time, the pressure difference across a pneumotachograph measured by an MP45 Validyne (Northridge, CA) pressure transducer (±2 cmH2O) located in the wall of the plethysmograph. The plethysmograph flow is calculated by knowing the resistance of the pneumotachograph. The flow signal was then integrated to obtain volume. The characteristics of this type of pneumotachograph are described elsewhere (16). Flow at the mouth level was measured by a no. 3 Fleisch pneumotachograph connected to an MP45 Validyne pressure transducer (±2 cmH2O). Esophageal pressure was measured by a 10-cm-long thin latex balloon positioned in the lower one-third of the esophagus at ~38–45 cm from the nostril. The balloon was connected to a pressure transducer (±352 cmH2O; Statham 131). The balloon was filled with ~1 mL air. We calculated the PTP as the difference between mouth and esophageal pressure. Placement of the balloon was considered correct if PTP remained constant while subjects made gentle respiratory efforts against a small ori.

First, a plot of the expiratory flow rate, and lung volume. Large lung volume and expiratory flow limitations are hallmarks of subjects with COPD. Therefore, in subjects with COPD, gas compression may cause significant distortion of the flow-volume relationship and most likely may result in underestimation of the ventilatory capacity (11). In contrast to expired volume, the volume change measured with plethysmograph is an overestimate of expired volume in an FVC maneuver (11).
exhitory flow-volume loop with units of liters per second on the y-axis and liters on the x-axis. Subsequently, the software inverts the exhitory flow signal between TLC and RV, plots box volume signal (liters) on the x-axis and inverts exhitory flow signal (s/l) on the y-axis, and generates a graph (Fig. 2B). This new graph contains a very steep negative slope at the beginning of the maneuver that reverses to a positive shallow slope during the effort-independent portion (<80% TLC) of the FVC maneuver. Subsequently, the software uses the following algorithm to calculate the computed time (\( T \)).

\[
Z(n) = \sum_{n=1}^{N} \frac{1}{\bar{V}(t_n + t_{n-1})} \cdot V_{box}(t_n + t_{n-1})
\]

where \( \bar{V} \) is mouth flow, \( t \) is the time at which each data point is recorded, \( n \) is the index of data arrays that is collected, and \( V_{box} \) is the volume measured by the body plethysmograph.

This computed time (\( Z \)), a time based on volume and flow, is the mouth transit time for increments of box volume. Likewise, mouth transit time is smaller at the start of the forced maneuver (at TLC) than at the end of the maneuver (at RV). As an individual becomes more obstructed near the end of a forced maneuver, the likelihood of gas compression is higher. By summing each computed time point, the software reconstructs a time line that represents volume changes based on exhitory mouth flow and body plethysmograph volume.

After generating the computed time, the software calculates the subject’s NFEV (Fig. 2C). The backward extrapolation technique is used to determine the start time for the NFEV calculation. The software uses the computed time for this determination instead of the standard linear time. The start time is determined by assuming that peak flow had begun since the expiration began at TLC. Volume-time curves are shown in Fig. 2D. These curves are recorded at the mouth and are based on mouth-flow and box-volume measurements, as described. To estimate the magnitude of gas compression, we used the following equation:

\[
\text{DFEV}_1 = \frac{\text{NFEV}_1 - \text{FEV}_1}{\text{FEV}_1}
\]

where \( \text{DFEV}_1 \) is the difference between \( \text{FEV}_1 \) and \( \text{NFEV}_1 \), and NFEV and FEV are absolute values of forced expired volume (liters) in first second as measured by the NCM and standard method.

Measuring variability and reproducibility of NFEV. To compare variability of the NFEV with that of the FEV within same testing session, we analyzed the baseline FEV and NFEV data for all subjects with three FVC maneuvers. We calculated the difference between FEV of FVC maneuvers with the highest and lowest FEV and compared this difference with the difference in NFEV for the same maneuvers. FEV-Dif was defined as mean difference in FEV between maneuvers with the highest and the lowest FEV divided by the lowest FEV. NFEV-Dif is defined as the mean difference of NFEV between maneuvers with the highest and lowest FEV divided by the lowest NFEV.

Furthermore, to evaluate reproducibility of NFEV measurements over a 6-mo interval, we calculated the NFEV for baseline and a 6-mo follow-up in COPD patients randomized to the medical arm of lung volume reduction surgery (LVRS). The Houston Veterans Affairs Medical Center LVRS is a randomized trial with a control arm of medical therapy and an intervention arm of bilateral LVRS in patients with severe emphysema. Individuals enrolled in this study had lung function measurement at baseline and at 6 mo follow-up.

Statistics. We used STATA version 7 (College Station, TX) for data analysis. Demographic and baseline lung function data were analyzed by descriptive statistics and presented as means ± SD. The relationships between DFEV (as an index of magnitude of gas compression) and other parameters of lung function were evaluated by stepwise multiple regression analysis. We used Student’s t-test and ANOVA when appropriate to compare FEV and NFEV values for the same-session comparison and between-session comparisons. A \( P \) value <0.05 was considered significant.

RESULTS

Baseline pulmonary function data are shown in Table 1. The mean \( \text{FEV}_1 \) was 45% of predicted values, whereas mean \( \text{NFEV}_1 \) was 59% of predicted values. The DFEV was notably larger in COPD and asthma subjects compared with normal subjects. In a multiple linear regression analysis, the DFEV
Table 2. Lung function parameters in 23 COPD patients (medical arm of LVRS) at baseline and 6 mo follow-up

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>6 Months</th>
<th>Paired t-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEF, l/s</td>
<td>3±0.88</td>
<td>3±0.84</td>
<td>P&gt;0.91</td>
</tr>
<tr>
<td>EPpeak, cmH2O</td>
<td>153±49</td>
<td>146±58</td>
<td>P&gt;0.68</td>
</tr>
<tr>
<td>EPFRC, cmH2O</td>
<td>23±20</td>
<td>30±19</td>
<td>P&gt;0.12</td>
</tr>
<tr>
<td>TLC, %predicted</td>
<td>134±18</td>
<td>136±14</td>
<td>P&gt;0.44</td>
</tr>
<tr>
<td>RV, %predicted</td>
<td>258±46</td>
<td>259±50</td>
<td>P&gt;0.86</td>
</tr>
<tr>
<td>FVC, %predicted</td>
<td>57±16</td>
<td>58±14</td>
<td>P&gt;0.70</td>
</tr>
<tr>
<td>SVC, %predicted</td>
<td>72±16</td>
<td>76±14</td>
<td>P&gt;0.06</td>
</tr>
<tr>
<td>Rrs, cmH2O·1⁻¹·s⁻¹</td>
<td>20±10</td>
<td>21±12</td>
<td>P&gt;0.33</td>
</tr>
<tr>
<td>FEV1, %predicted</td>
<td>27±7</td>
<td>27±7</td>
<td>P&gt;0.80</td>
</tr>
<tr>
<td>NFEV1, %predicted</td>
<td>42±9</td>
<td>43±9</td>
<td>P&gt;0.67</td>
</tr>
<tr>
<td>DFEV1, %change</td>
<td>63±8</td>
<td>66±9</td>
<td>P&gt;0.72</td>
</tr>
</tbody>
</table>

Values are means ± SD. LVRS, lung volume reduction surgery; RV, residual volume. P values >0.05 considered not statistically significant.

correlated significantly with FEV1, Rls, TLC, and PEF (P values <0.0001). Additionally, slow vital capacity was higher than FVC.

Variability of FEV1 and NFEV1 within the same testing session as measured by FEV1-Dif and NFEV1-Dif was 13 ± 1.3% and 2.9 ± 2.4%, respectively. Student’s t-test showed a significant difference between FEV1-Dif and NFEV1-Dif (P < 0.0005). To measure the reproducibility of the NCM between different testing sessions, we analyzed the data from the medical arm of the LVRS in 23 subjects with severe COPD. These data are presented in Table 2. NFEV1 and FEV1 measured at baseline were not significant after 6 mo. Data indicate that NFEV1 and other parameters of lung function are reproducible over different test sessions.

Figure 3 is the graphical presentation of FEV1 and NFEV1 from a normal subject, a subject with asthma, and a subject with COPD. In Fig. 3, there are six graphs each showing a plot of a volume vs. time of an FVC maneuver. Data for a normal subject at baseline and after inhalation of 180 mg albuterol are shown in Fig. 3, A and B, respectively. The plot shows that, in spite of the high intrathoracic pressure, the magnitude of DFEV1 (i.e., magnitude of gas compression) is negligible because of low Rls. This observation is consistent with the known factors affecting gas compression (13). Figure 3, C and D, shows the relationship between volume and time during a forced expiratory maneuver in a subject with asthma. Interestingly, although PEF increased 60%, reduction of Rls to 50% of baseline has reduced the magnitude of gas compression from 8 to only 1%. Furthermore, the intrathoracic pressure did not change appreciably in response to a considerable increase in PEF. This can be explained in part by the reduction of Rls. The data from a patient with COPD before and after bronchodilator inhalation are presented in Fig. 3, E and F. In this subject, although Rls decreased ~60% from baseline, the magnitude of gas compression only decreased from 80 to 57%.

DISCUSSION

The effects of gas compression on forced expiratory flow and volume have been studied previously (10, 12, 13). However, these studies did not provide a methodology for the correction of these measurements for gas compression. In the present study, we presented a novel method (NCM) for measuring the effects of gas compression on maximal expiratory flow in normal, asthmatic, and COPD subjects. In subjects with expiratory flow limitation, a forced maneuver results in gas compression and dynamic gas trapping. With this method, we measured the amount of gas compression and dynamically trapped gas during a forced expiratory maneuver. The method provided a measure of FEV1 corrected for the effect of gas compression. The method requires a volume displacement body plethysmograph and specialized software. Our results demonstrated that, in subjects with expiratory flow limitation, the NCM provided accurate information about the relationship between maximal expiratory flow and true lung volume.
Our data and those of others (11–13) show that gas compression can significantly distort the relationship between flow and volume during an FVC maneuver (Fig. 1B). Such distortion is magnified in subjects with expiratory flow limitation like COPD patients.

As shown in Table 2, the NCM provides reproducible measurements. The test variability over time was negligible in patients with moderate to severe COPD. In subjects with expiratory flow limitation, FEV₁ is affected by the underlying airway disease and the effect of gas compression. Therefore, variability in efforts used in performing FVC maneuvers at the same testing session or different testing sessions may result in a false increase or decrease in recorded FEV₁. This effect has been shown to be the result of gas compression (13). NCM reproducibility in successive measurements over time indicates that NFEV₁ is a reliable measurement. NFEV₁ is corrected for the effect of gas compression; therefore, compared with FEV₁, NFEV₁ can generate more reproducible and less variable measurements. This is indicated by comparing the FEV₁ and NFEV₁ obtained from multiple maneuvers during the same session. Our data showed FEV₁-Dif to be significantly larger than NFEV₁-Dif (P < 0.0005).

In normal subjects, the lung function data using expired mouth volume were similar to those using NCM. This finding was expected because airflow limitation and high effort are required to cause appreciable gas compression. As shown in Fig. 1A, the amount of expired air is the same when measured at the mouth and with the plethysmograph. This is typical for healthy young subjects, where the RV is determined by the balance between expiratory muscle effort and the outward recoil of the respiratory system. In addition, because of dynamic compression of airways, air trapping is less likely in normal subjects. In the normal subjects, factors affecting the gas compression, Rₑ and TLC, were within normal limits (Table 1). Therefore, it is not surprising that NEFV₁ and FEV₁ were not statistically significantly different in this group.

In asthmatic subjects, NFEV₁ was significantly larger than FEV₁. In addition, the Rₑ was higher than in normal subjects. This can explain the larger gas compression in asthmatic subjects (DFEV₁ = 24 ± 20%) compared with normal subjects (DFEV₁ = 10 ± 16%). Furthermore, the mean PEF in our normal subjects is much higher than PEF in asthmatic and COPD subjects. However, thoracic gas compression estimated with the NCM was negligible. This observation demonstrates the significant effect of Rₑ on the generation of gas compression and air trapping and the sensitivity of the NCM in estimating the effect of gas compression. The COPD subjects in this study showed the highest Rₑ and TLC among the study groups. Therefore, not surprisingly, the gas compression was highest in this group. The relationship between Rₑ and gas compression should be evaluated with care. A normal Rₑ can be accompanied by large dynamic compression, provided the effort is very large. In addition, some groups of patients can have normal Rₑ but severe flow limitation.

In summary, we presented a novel method for measuring the effects of gas compression on expiratory flow (NCM). Our data demonstrated that the NCM can be used to reasonably estimate the gas compression during a forced expiratory maneuver. Our data showed that the NCM produced an index of pulmonary function that was reproducible in repeated measure (NFEV₁).

Furthermore, the NFEV₁ is sensitive to factors affecting magnitude of gas compression. However, future work should include the investigation of the sensitivity of this new method to changes in Rₑ, TLC, and effort in a larger sample of subjects with and without underlying obstructive airway disease.

REFERENCES