The detection of collapsible airways, which may be a component of asthma and emphysema, has important therapeutic implications. We describe a patient with significant airways collapse contributing to his airflow limitation and discuss how collapsible airways can be assessed by the volume difference between what exists the lung as determined by a spirometer and the volume compressed as measured by the plethysmograph. More simply, a large volume difference between the slow and forced vital capacity (SVC-FVC), easily obtained from spirometry, may be used as a surrogate index of airway collapse.

(Chest 1985; 856-59)

Herein we present a patient as an example of airflow limitation due largely to airways collapse. The point of this case is that in patients who present with evidence of airflow limitation without hyperinflation, airways collapse is a possibility. Further we will demonstrate how this cause of airflow limitation can be assessed with flow-volume relationships derived from either a body plethysmograph or more simply from spirometry.

**Case Report**

A 66-year-old man was referred for a chronic nonproductive cough of about 1-year duration. The cough has been more frequent in the early evening hours and is often triggered by cold air, deep breathing, and perfume. There is no significant associated nasal or sinus symptoms. His medical history is notable for childhood “asthma,” hypertension, insulin-dependent diabetes mellitus, coronary artery bypass surgery, and a benign nasal polyp. He smoked a pack of cigarettes a day for 30 years but quit over 20 years ago. He is a restaurant owner with no current or past toxic fume or dust exposure although he has had significant second-hand cigarette smoke exposure.

He appeared healthy but anxious on examination. He was afebrile with a pulse 84/min, BP of 160/90 mm Hg, and a respiratory rate of 16/min. There was a paroxysmal cough. The skin, nodes, thyroid, heart, abdomen, and extremities were unremarkable. There was a right nasal polyp. The chest wall was normal and the lung examination was significant for a prolonged expiratory phase with diffuse expiratory wheezes.

Chest radiograph showed the previous sternotomy with clear lung fields and a normal sized heart. Computed tomography of the sinuses was unremarkable except for the nasal polyp. The results from a fiberoptic laryngoscopy were normal. His pulmonary function test was performed with care to avoid prior treatment with bronchodilator or steroids.

**Pulmonary Function**

The spirometry (Table 1) showed a reduced forced expiratory volume in 1 s (FEV₁) and forced vital capacity (FVC) (both are 60% of predicted) and yet the FEV₁/FVC ratio is 70% or 104% of predicted and the specific conductance (SGaw) is 86% predicted. There is evidence of some element of reversible airflow limitation based on a significant bronchodilator response to albuterol with improvement in SGaw of 58% and in FEV₁ of 20% from baseline. Moreover, his lung volume decreased (Fig 1) with bronchodilator therapy (isovolume shift) with a reduction in total lung capacity (TLC) of 220 mL, functional residual capacity (FRC) of 340 mL, and residual volume (RV) of 100 mL; hence, flows at absolute volume are improved.

In assessing airway collapse, the flow volume loops in which the volume measured at the mouth and that which is measured in the plethysmograph were superimposed (Fig 2). When the flow difference between the two curves at any particular volume are compared, it can be appreciated that there is compression of alveolar gas that is greater than seen in normal subjects. This is also confirmed by the large discrepancy between the slow vital capacity (SVC) and FVC of 1,200 mL (Table 1). This later finding is not explained by poor patient effort as there were significant gas compression and good test reproducibility; in addition, the American Thoracic Society end-of-test criteria were met. Lastly, evidence for collapsible airways is noted from the flow volume loops since the flow during eupnea exceeds that at forced expiration (Fig 3).

**Discussion**

Airflow limitation intrinsic to the lung may be due to reversible or irreversible airways disease or to a combination of both. One form of “irreversible”

**Key words:** airway collapse; asthma; gas compression; obstructive lung disease

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airflow limitation is collapsible airways, which has been shown to be a component of asthma (probably due to inflammation) or to emphysema (due to the loss of supporting structure of the airways). The distinction of a purely collapsible airways disease from that of asthma is important because the treatment of the former may include the use of purse lip breathing or nasal positive pressure ventilation whereas in the latter, pharmacologic approaches are used.

There are a number of ways to assess collapse of airways. One method is to compare the maximal expiratory flow volume (MEFV) measured at the mouth with that by the plethysmograph. Ingram and Schilder\(^2\) compared the MEFV relationships in which the volume displaced was measured simultaneously by both a spirometer and a plethysmograph. They showed that even in normal subjects, the change in volume measured by the spirometer (\(\Delta V\)) at most lung volumes was less than the thoracic gas volume measured by the plethysmograph (\(\Delta TGV\)). The difference in these volumes (\(\Delta TGV-\Delta V\)) represents the compression of gas in the thorax. In our patient (Fig 2), the volume remaining in the lung (\% VC) was less when \(\Delta TGV\) was measured than when \(\Delta V\) was measured for any given flow. More recently, Coates and coworkers\(^3\) showed that the flow rate at 50% of VC derived from the volume measured at the mouth (\(V_{\text{max},50,m}\)) was lower than the flow rate at 50% of VC derived from volume measured by the plethysmograph (\(V_{\text{max},50,p}\)) by 8% in normal subjects. In

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21710/)  
**Figure 1.** Flow-volume relationship for the presenting patient: prebronchodilator (solid line) and postbronchodilator (dash line). Flow and volume are plotted at absolute volume by determining FRC in the body box prior to maximal inhalation to TLC. Note that there is an increase in flow at any given absolute volume (isovolume shift).

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21710/)  
**Figure 2.** Maximal effort flow-volume curve as measured at the mouth (flow vs \(\Delta V\), solid line) vs that measured at the chest wall by body plethysmograph (flow vs \(\Delta TGV\), dash line). \(\Delta V\)=volume exiting the lung measured by spirometer; \(\Delta TGV\)=volume measured by plethysmograph. Volume shift or difference between the curves represents the air volume in the chest that is being compressed.

Table 1—Pulmonary Function Test Results

<table>
<thead>
<tr>
<th></th>
<th>Predicted</th>
<th>Baseline</th>
<th>Base % Predicted</th>
<th>After Albuterol</th>
<th>% Change</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Volumes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TLC, L</td>
<td>7.02</td>
<td>6.20</td>
<td>88</td>
<td>5.98</td>
<td>-4</td>
</tr>
<tr>
<td>FRC, L</td>
<td>4.15</td>
<td>3.28</td>
<td>79</td>
<td>2.94</td>
<td>-10</td>
</tr>
<tr>
<td>RV, L</td>
<td>2.08</td>
<td>2.08</td>
<td>100</td>
<td>1.98</td>
<td>-5</td>
</tr>
<tr>
<td>SVC, L</td>
<td>4.94</td>
<td>4.11</td>
<td>83</td>
<td>3.96</td>
<td>-4</td>
</tr>
<tr>
<td><strong>Spirometry</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVC, L</td>
<td>4.94</td>
<td>2.89</td>
<td>59</td>
<td>3.20</td>
<td>11</td>
</tr>
<tr>
<td>FEV(_1), L</td>
<td>3.33</td>
<td>2.03</td>
<td>61</td>
<td>2.43</td>
<td>20</td>
</tr>
<tr>
<td>FEV(_1)/FVC, %</td>
<td>67</td>
<td>70</td>
<td>104</td>
<td>76</td>
<td>9</td>
</tr>
<tr>
<td><strong>Conductance</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raw, cm H(_2)O/L/s</td>
<td>1.69</td>
<td>2.53</td>
<td>150</td>
<td>1.68</td>
<td>-34</td>
</tr>
<tr>
<td>SGaw, L/s/cm H(_2)O</td>
<td>0.14</td>
<td>0.12</td>
<td>86</td>
<td>0.19</td>
<td>58</td>
</tr>
</tbody>
</table>
Significant alveolar gas compression results from a multitude of factors, including obstructive lung diseases, muscular effort, a greater volume of gas in the chest to be compressed, and a more compressible gas in the chest, e.g., breathing helium or at high altitude. Since it is obvious that gas compression is based on muscular effort during a maximal expiratory maneuver, one sees some degree of compression in all normal subjects. Recently, Stoller and coworkers were able to show that a modified expiratory technique, consisting of an initial maximal expiratory effort followed by a “relaxed expiration” for as long as possible, increased the FVC in patients with airflow obstruction and increased the likelihood of meeting end-of-test spirometric criteria. However, if the MEFV curves as derived from ΔV and ΔTGV are identical, it is due to a suboptimal patient effort. For example, Charan and colleagues showed that when increased gas compression occurs due to increased muscular effort as during a forced expiration, the difference between ΔTGV and ΔV widens once a threshold in esophageal pressure is reached. As a corollary then, if a body plethysmograph is used to measure flow volume relationships in a patient with obvious severe airflow limitation and gas compression, the lung volumes may be significantly lower than expected for any set flow and result in a negative effort dependence.

These studies explain the large difference of 1,200 mL between the SVC and FVC in our patient. During the slow or partial maneuver (SVC), there was less thoracic gas compression and therefore greater amounts of airflow and volume measured. In contrast, during an MEFV maneuver (FVC), there is greater airways compression and collapse and thus a smaller volume is measured. Hence, the difference between the SVC-FVC can be used as a surrogate index of airway collapse that is more easily obtained from simple spirometric results.

In summary, airways collapse can be an important contribution to airflow limitation that is often not appreciated. Collapsible airways may be inferred from the comparison of expiratory flow loops performed by plethysmography and may be more obvious in patients with significant gas compression. This variable of thoracic gas compressibility is further increased in cases of airflow limitation due to airways collapse. Recognition and identification of patients in which airways collapse is an important element of their airflow limitation has potentially important therapeutic implications.

REFERENCES