Maximal Inspiratory Flow Rates in Patients With COPD*

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Objectives: To assess the relevance of maximal inspiratory flow rates (MIFR) in the assessment of airway obstruction in COPD.

Setting: University teaching hospital.

Participants: Ten consecutive COPD patients (O group; mean [± SD] age, 58.5 ± 8.3 years) and 10 matched healthy subjects (H group; mean age, 58.7 ± 7.4 years).

Measurements: Lung volumes, FEV1, specific airway conductance, single-breath lung diffusing capacity, MIFR, and maximal expiratory flow rates (MEFR).

Results: Mean FEV1/vital capacity (VC) was 74.7% in the H group and 37.8% in the O group (p < 0.001). Total lung capacity was higher (p < 0.001) in the O group compared with the H group. Lung diffusing capacity was less than half in the O group compared with the H group (p < 0.001). MEFR at all lung volumes were lower in the O group (p < 0.001). MIFR were comparable in the two groups, except at 25% inspired VC, where MIFR were lower in the O group (p < 0.05).

Conclusion: MIFR are less sensitive than MEFR to detect airway obstruction in COPD patients. Yet, the interest of MIFR lay in the possibility to separate intrinsic from extrinsic involvement of airways. A normal MIFR associated with low MEFR, as in the present study, suggests either a lack of parenchymal support, an increased collapsibility of the airways, or a reversible peripheral airway narrowing. A fixed, generalized airway narrowing would be associated with a decrease of both MIFR and MEFR.

Materials and Methods

We studied 10 male, consecutive patients with COPD (mean [± SD] age, 58.5 ± 8.3 years), who were treated in the pneumology division of our hospital. The diagnosis of COPD was based on the standard criteria of the European Respiratory Society and the American Thoracic Society.6,7 All patients were long-term regular cigarette smokers, with a smoking history of 57.4 ± 41.8 pack-years. None of them had chronic expectoration. All medication was stopped 24 h before the measurements. We also studied 10 healthy subjects (mean age, 58.7 ± 7.4 years) recruited from the staff or from hospital volunteers. The volunteers were all asymptomatic.

Abbreviations: DLco = diffusing capacity of the lung for carbon monoxide; MEFR = maximal expiratory flow rate; MEFR90 = MEFR measured at 50% of vital capacity; MIFR = maximal inspiratory flow rate; Pel = elastic recoil of the lung; Ppl = pleural pressure; RV = residual volume; sGaw = specific airway conductance; TLC = total lung capacity; VC = vital capacity.

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aximal expiratory flow rates (MEFR) measured from a flow-volume curve are currently used to assess expiratory flow limitation. However, except in studies on upper-airways abnormalities, maximal inspiratory flow rates (MIFR) are rarely recorded.1–5 There are only three articles, published some 30 to 40 years ago, that systematically assessed both MIFR and MEFR in patients with COPD.1–3 In the three studies, MIFR and MEFR were significantly less than in normal subjects, and MEFR were lower than MIFR.

In contrast to these data, it has been our experience that in patients with pulmonary emphysema, MIFR were often normal or close to normal. Since, to the best of our knowledge, the above studies1–3 are the only ones comparing MIFR and MEFR in COPD, we decided to repeat them. Taking the influence of gas compression into account, changes in lung volumes were measured by body plethysmography.
tomatic, in good health, and without any upper-respiratory infection in the preceding 3 months. Six of them were nonsmokers.

Slow vital capacity (VC) and FEV\(_1\) were measured with a water spirometer (Pulmonet III; SensorMedics; Yorba Linda, CA). Residual volume (RV) was measured by the He closed circuit method. MEFR and MIFR were recorded at the mouth, in this order, with a heated Fleisch No. 4 pneumotachograph (Fleisch; Lausanne, Switzerland). The pneumotachograph was connected to a differential pressure transducer (Statham PM 15 TC, 0.04 pounds per square inch; Oxnard, CA). Its output was amplified (Hewlett-Packard 8805 B carrier amplifier; Hewlett Packard; Waltham, MA) and recorded on a Gould TA 11 recorder (Gould; Valley View, OH). MEFR and MIFR were measured at 75%, 50%, and 25% of VC. Lung volume changes were measured in a pressure-corrected flow body plethysmograph (homemade). To take into account differences in body size, MEFR and MIFR were normalized by dividing them by the cubic height of the patient. Specific airway conductance (sGaw) was measured during panting (two cycles per second) by dividing airway conductance (the inverse of airway resistance) by the thoracic lung volume. Airway resistance was measured between 0 L/s and 0.2 L/s inspiratory and expiratory flow. The diffusing capacity of the lung for carbon monoxide (DL\(_{\text{CO}}\)) was measured by the single-breath method, using the Morgan benchmark transfer test.

Data are reported as mean (SD) and were compared using the Student's t test for independent variables. The linear correlation coefficient was calculated. A p value < 0.05 was considered significant.

**RESULTS**

Table 1 presents physical and functional data in both healthy and COPD subjects. Age and height were comparable in both groups. Spirographic tests were within predicted limits for healthy subjects, but sharply decreased in COPD patients. The FEV\(_1/VC\) was 74.7% in healthy subjects and 37.8% in COPD patients (p < 0.001). Total lung capacity (TLC) and RV/TLC ratio were significantly higher in the latter compared with the former group. DL\(_{\text{CO}}\) was less than half in COPD subjects compared with healthy ones (p < 0.001). sGaw was lower in obstructive patients than in healthy ones (p < 0.001).

Within the investigated limits of airflows (± 0.2 L/s), inspiratory and expiratory conductance were similar. The average expiratory and inspiratory flow-volume curves for healthy and COPD subjects are presented in Figure 1. Values of MEFR and MIFR at different lung volumes are shown in the Table 1. MEFR at all lung volumes were significantly lower in the COPD group (p < 0.001). MIFR were comparable (p > 0.05) in the two groups, except at 25% inspired VC, where MIFR were lower in COPD subjects than in healthy ones (p < 0.05). As shown in Figure 2, a highly significant correlation was found in normal and COPD subjects between sGaw and MIFR measured at 50% of VC (MIFR\(_{50}\)). There was also a significant correlation between sGaw and MEFR\(_{50}\).

**Table 1—Physical and Functional Data Expressed as Mean (SD) in Healthy Volunteers and COPD Patients**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Healthy Volunteers</th>
<th>COPD Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>58.7 (7.4)</td>
<td>58.5 (8.3)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.75 (0.06)</td>
<td>1.73 (0.05)</td>
</tr>
<tr>
<td>TLC, L</td>
<td>6.00 (0.66)</td>
<td>7.88 (1.22)</td>
</tr>
<tr>
<td>VC, L</td>
<td>4.70 (0.56)</td>
<td>3.78 (1.32)</td>
</tr>
<tr>
<td>RV/TLC, %</td>
<td>29.0 (4.6)</td>
<td>51.7 (14.6)</td>
</tr>
<tr>
<td>DL(_{\text{CO}}) S, mL L(^{-1}) min(^{-1}) mm Hg(^{-1})</td>
<td>30.9 (3.1)</td>
<td>12.8 (7.2)</td>
</tr>
<tr>
<td>FEV(_1/VC), %</td>
<td>74.7 (5.1)</td>
<td>37.8 (13.1)</td>
</tr>
<tr>
<td>sGaw, cm H(_2)O(^{-1}) s(^{-1})</td>
<td>0.27 (0.06)</td>
<td>0.08 (0.04)</td>
</tr>
<tr>
<td>MEFR(_{50}), Ls(^{-1})m(^{-3})</td>
<td>1.25 (0.22)</td>
<td>0.51 (0.34)</td>
</tr>
<tr>
<td>MIFR(_{50}), Ls(^{-1})m(^{-3})</td>
<td>0.85 (0.22)</td>
<td>0.20 (0.16)</td>
</tr>
<tr>
<td>MEFR(_{25}), Ls(^{-1})m(^{-3})</td>
<td>0.23 (0.11)</td>
<td>0.06 (0.04)</td>
</tr>
<tr>
<td>MIFR(_{25}), Ls(^{-1})m(^{-3})</td>
<td>0.85 (0.16)</td>
<td>0.68 (0.11)</td>
</tr>
<tr>
<td>MEFR(_{75}), Ls(^{-1})m(^{-3})</td>
<td>0.94 (0.20)</td>
<td>0.81 (0.17)</td>
</tr>
<tr>
<td>MIFR(_{75}), Ls(^{-1})m(^{-3})</td>
<td>0.79 (0.18)</td>
<td>0.72 (0.22)</td>
</tr>
</tbody>
</table>

\*DL\(_{\text{CO}}\) = single-breath DL\(_{\text{CO}}\); MEFR\(_{75}\) = MEFR at 75% of VC; MIFR\(_{75}\) = MIFR at 25% of VC; MEFR\(_{25}\) = MIFR at 25% of VC; MIFR\(_{50}\) = MIFR at 50% of VC; MIFR\(_{75}\) = MIFR at 75% of VC.

\(\dagger p < 0.05.\)

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in the COPD patients only \((r = 0.92)\). No correlation was found between sGaw and MIFR at 50% of VC in either COPD patients alone or in both COPD and healthy subjects (Fig 3).

**DISCUSSION**

We have found in a group of COPD patients, compared with a matched group of healthy volunteers, a decrease of MEFR and sGaw but normal or near-normal MIFR. Since single-breath DLco was markedly reduced, it is likely that pulmonary emphysema was present in a significant degree in these patients. Indeed, single-breath DLco is considered to be a good reflection of pulmonary emphysema. A further argument in favor of emphysema is the higher TLC in COPD patients \((+30\%)\) as compared with the healthy volunteers. The decrease of MEFR is a typical finding in COPD patients and is an expression of flow limitation. Two mechanisms are advanced to explain expiratory flow limitation in COPD patients: an intrinsic mechanism, *ie*, decrease of the caliber of the small airways (inner diameter of < 2 mm) by inflammation, fibrosis, and mucus plugging; and an extrinsic mechanism, one due to lack of parenchymal support of the peripheral airways as a consequence of disruption of the elastic network of the lung. In most COPD patients, the two mechanisms coexist, though there are rare subjects with exclusive intrinsic or extrinsic impairment of airways. Both peripheral airway narrowing and lack of support also result in a decrease of sGaw. This may explain the close correlation we found between sGaw and MEFR\(_{50}\) in normal and COPD subjects (Fig 2). A third mechanism explaining the reduction of MEFR was previously advanced by Leaver et al: an enhanced collapsibility of the flow-limiting airways, in keeping with the wave-speed theory.

MIFR at a given volume depends on airways caliber, as well as strength and speed of shortening of the inspiratory muscles. Since we have found normal or near-normal values for MIFR, we can conclude that neither an impairment of the shortening of the inspiratory muscles nor a substantial narrowing of airways caliber was operating in our COPD patients. Therefore, how can the difference between

![Figure 2. Relationship between sGaw and MEFR at 50% of VC (MEFR\(_{50}\) divided by the cubic height) in healthy subjects (open circles) and COPD patients (closed circles). The coefficient of correlation \(r\) is highly significant \((p < 0.001)\). See Figure 1 for abbreviation.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21953/)

![Figure 3. The absence of relationship between sGaw and MIFR at 50% of VC in healthy subjects (open circles) and COPD patients (closed circles). See Figure 1 for abbreviation.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21953/)
MEFR and MIFR be explained? The basic difference between these indexes is related to the different mechanical forces acting during inspiration and expiration. During forced expiration, pleural pressure (Ppl) has a dual action: it is, together with the elastic recoil of the lung (Pel l), a component of alveolar pressure, the driving pressure during expiration. It is also the main outside component of the transmural pressure of the intrathoracic airways. A large increase in Ppl results in negative transmural pressure and intrathoracic airway compression, leading to flow limitation. When the latter is reached, Ppl is neutralized as a driving pressure, the primary driving pressure for expiration then being Pel l. If the latter is reduced, the MEFR will decrease. A peripheral airway narrowing or an increased collapsibility of the flow-limiting airways will amplify the narrowing of the compressed airways, both resulting in a further decrease of MEFR. In contrast, during forced inspiration, flow limitation does not occur; the transmural pressure of the intrathoracic airways is strongly positive over the largest part of the bronchial tree, keeping the intrathoracic airways wide open. In addition, the driving pressure (Ppl – Pel l) is much larger than Pel l. Therefore, neither a reduction of Pel l (lack of support of the intraparenchymatous airways) nor an increased collapsibility of the intrathoracic airways will modify the MIFR. An airway narrowing at low flow rates, evidenced by a decrease of airway conductance, would be associated with a normal or near-normal MIFR (high flow rates), as the result of a high positive transmural inspiratory pressure that restores the airways caliber to a normal or near-normal size. One expects a clear-cut decrease of MIFR only if there is a marked, generalized airways narrowing not yielding to an increase in transmural pressure (low compliance airways, fixed airway obstruction), either anatomic (bronchiolitis obliterans) or functional (bronchial asthma). In a study of 14 asymptomatic asthmatics, inhalation of histamine resulted in decreased FEV₁ (average decrease, 18%). There was a decrease of MEF₅₀ of 0.9 L/s (−40% with respect to control values) and of MIF₅₀ of 1.1 L/s (−19%).

Detailed assessment of MIFR in COPD patients was rarely done before. In three reports in COPD patients, MIFR and MEFR were both significantly lower than in normal subjects. In all three articles, MEFR values were lower than MIFR. This pattern is compatible with a generalized airway obstruction, with or without associated pulmonary emphysema. In one of these articles, in patients with chronic bronchitis, there was, as in our study, a decrease of MEFR, but no decrease of MIFR. Unfortunately, these studies were published some time ago, before generally recognized criteria for the diagnosis of pulmonary emphysema became available. In our patients, a lack of parenchymal support, associated probably with a reversible peripheral airways narrowing, was responsible for the decrease of MEFR and near-normal values of MIFR. An extreme example in a patient with severe emphysema (DLCO, 15% of predicted) is presented in Figure 4.

In conclusion, MIFR is less sensitive than MEFR in detecting airway narrowing. Yet, the interest of MIFR in COPD is in the possibility of separating intrinsic from extrinsic involvement of the airways, marked airways narrowing from lack of support. Normal MIFR associated with low MEFR, as in the present study, suggests either a lack of parenchymal support, an increased collapsibility, or reversible narrowing of the airways. A fixed, generalized airways narrowing would be associated with a decrease of both MIFR and MEFR.

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