Partial and Maximal Expiratory Flow-Volume Curves in Normal and Asthmatic Subjects Before and After Inhalation of Metaproterenol*

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The effects of deep inspiration upon expiratory flow rates and response to inhaled metaproterenol were studied in normal and asthmatic subjects using partial (PEFV) and maximal (MEFV) expiratory flow volume curves. Routine pulmonary function tests and specific conductance were also measured. Prior to administration of metaproterenol, 18 of 24 normal subjects and 11 of 24 asthmatic subjects (p 0.05) had higher flow rates on MEFV than on PEFV curves. The II volume history responsive asthmatic subjects showed better lung function and more density-dependence of expiratory flow than the other 13 asthmatic subjects; furthermore, the effect of lung inflation was significantly larger in the volume history responsive asthmatic subjects than in the volume history responsive normal subjects. Responses to inhaled metaproterenol were much larger on PEFV than MEFV curves; nevertheless, differences between normal and asthmatic subjects in metaproterenol responsiveness were less using PEFV curves. Thus, the use of PEFV curve measurement did not facilitate the detection of individual asthmatic responses to inhaled metaproterenol.

Deep inspiration (total lung capacity volume history) has been shown to increase specific conductance and maximal isovolume flow rates in many normal persons and some asthmatic patients. Other asthmatic patients may fail to decrease, or may actually increase, bronchomotor tone after a total lung capacity (TLC) maneuver. Volume history effects diminish with time after a deep inspiration.

Partial expiratory flow volume (PEFV) curves initiated from a volume below TLC, but with maximal effort, minimize volume history effects. Flow rates from PEFV studies can be compared to those generated on maximal expiratory flow volume curves (MEFV) initiated from TLC. By comparing isovolume flow rates, one can infer the presence of airway hysteresis if partial flow rates are lower. Other factors, such as reduction in lung elastic recoil post-inspiration and lung non-homogeneity, tend to increase flow on PEFV relative to MEFV curves. Zamal and coworkers found that expiratory flow was higher on MEFV than PEFV curves in only three of 20 adult asthmatic subjects (with spontaneous bronchospasm). By comparison, Orehek et al. found a decrease in airway resistance following inspiration to TLC in over 50 percent of asthmatic subjects with carbachol-induced bronchoconstriction.

In normal subjects, measurements of specific airway conductance (SGaw) are more sensitive for detecting response to inhaled beta agonist drugs than are measurements of forced expiratory volume in one second (FEV). This observation could be explained if the deep inspiration preceding measurement of FEV reduced bronchomotor tone, thereby limiting the possible change from the inhaled beta agonist. Partial flow volume curves (which are not preceded by an inspiration to TLC) eliminate this problem. In a preliminary report, Parry and coworkers described response to isoproterenol using PEFV and MEFV curves in obstructed and nonobstructed subjects; increments in flow rates after administration of a bronchodilator were much greater on the partial curves in both groups. Barnes and colleagues found PEFV curves to be more reproducible than airway resistance measurements for studying bronchodilator dose-response relationships.

We studied PEFV and MEFV maneuvers in age-matched groups of normal and asthmatic subjects in order to address several questions: 1) What proportion of normal and asthmatic subjects (with spontaneous rather than provoked bronchospasm) increase expiratory flow rates after a preceding deep inspiration? The proportions of asthmatic subjects showing bronchodilatation after a TLC volume history have varied considerably in previous studies. 2) Is there a correlation between severity of asthma and the ability to increase flow rates after a deep inspiration? 3) Is there a relationship between the site of airflow limitation (as assessed by helium-oxygen [He-O2] studies) in asthmatic subjects and the ability to increase airflow after a deep breath? 4) Are partial flow volume curves useful

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in demonstrating bronchodilator response in normal and asthmatic subjects, compared to standard methods?

**Materials and Methods**

Twenty four non-smoking normal subjects (13 men, 11 women) with mean age 33.3±9.5 were studied; the asthmatic group consisted of 24 subjects (12 men, 12 women) with mean age 30.3±11.4 (p = NS). Four of the asthmatic subjects were light smokers (<10 pack-years); all of these subjects had asthma prior to the onset of cigarette smoking. All subjects gave informed consent to participate in the study. The asthmatic subjects all had a history of episodic dyspnea or bronchospasm but were selected by evidence of reversible airflow obstruction (≥15% improvement in FEV1, post-bronchodilator).

Forced vital capacity (FVC) and its subdivisions were determined using a 13.5 L water-sealed spirometer (Warren E Collins Inc, Braintree, MA). Slow vital capacity (VC) and expiratory reserve volume (ERV) were also determined with this spirometer. Thoracic gas volume at functional residual capacity (VTG) and airway resistance (Raw) were measured by body plethysmographic study. Airway resistance was expressed as specific conductance (SGaw=Raw−1/VTG). Residual volume (RV) was calculated by subtracting ERV from VTG. Total lung capacity was calculated by adding FVC or VC (which ever was larger) to RV. Single breath diffusing capacity for carbon monoxide (DLco) was measured by the technique of Ogilvie and associates.

Maximal and partial expiratory flow volume (MEFV, FEV) curves were obtained using a rolling-seal spirometer (Cardiopulmonary Instruments, model 220, Houston, TX). Results were recorded on an X-Y plotter (Cardiopulmonary Instruments, model 750). The sequence of flow-volume testing was as follows: after three minutes of tidal breathing (avoiding any coughing or deep breaths), each subject inspired to 60 to 70 percent of VC, exhaled maximally to RV, immediately inspired to TLC, and again immediately expired maximally to RV. An isovolume point of comparison for the curves generated on this maneuver was defined at 25 percent of the complete VC (25 percent above RV on the MEFV curve); flow rates at this volume on the PEFR curve and the MEFV curve were termed Vmax−p and Vmax−c, respectively. Flow volume maneuvers as described above were performed at least three times, with three min allotted between maneuvers. The relationship between Vmax−p and Vmax−c was expressed as the ratio Vmax−p/Vmax−c; thus a Vmax−p/Vmax−c ratio <1 would indicate reduction in bronchomotor tone following inflation of the lung. Results were averaged from three technically satisfactory maneuvers.

Routine pulmonary function tests, PEFR, and MEFV curves were repeated 10 min after four slow inspiratory capacity inhalations of 0.65 mg of metaproterenol (total = 2.6 mg) from a metered dose inhaler. Absolute changes in FEV1, FVC, SGaw, Vmax−p, and Vmax−c were calculated as post-bronchodilator value minus pre-bronchodilator value. Percent change from baseline for the above variables was calculated as absolute change × 100 divided by the pre-bronchodilator value.

Maximum expiratory flow volume curves were performed before and after breathing an 80 percent helium/20 percent oxygen mixture until expired N2 concentration (measured by mass spectrometry) was ≤2 percent. Volume history was standardized by three inflations to TLC prior to performance of MEFV curves. Three technically satisfactory room air and He-O2 curves were obtained in all subjects; MEFV maneuvers were discarded if the vital capacity was less than 95 percent of the largest vital capacity recorded. Curves selected for analysis were those exhibiting best effort (highest product of FEV1×FVC). Air and He-O2 curves were superimposed at TLC. Density dependence was quantitated by ΔVmax=(Vmax(He-O2)−Vmax(air))/Vmax(air)×100 where Vmax is the flow rate at 50 percent of the vital capacity.

Spirometric results were compared to the predicted normal data of Morris et al. Mean values and standard deviations were calculated by standard equations. Mean results were compared using Students' t-test analysis. The paired t-test was used, where appropriate. The frequency of Vmax−p/Vmax−c ratios <1 was compared with the frequency of ratios ≥1 using chi-square analysis. Linear regression analysis was done using the least squares method. A p value ≤.05 was considered statistically significant.

**Results**

**Routine Pulmonary Function Tests (Table 1)**

In the asthmatic group, abnormalities ranged from mild to severe. Compared to the normal subjects, FEV1, percent predicted FEV1, FVC, SGaw, ΔVmax−p, and RV were all significantly worse in the asthmatics. In addition, density-dependence of maximal expiratory flow (ΔVmax−p) was substantially lower in the asthmatic than in normal subjects (p<0.001). Total lung capacity and DLco did not differ significantly between the two groups.

In both groups, FEV1, percent predicted FEV1, FVC, percent predicted FVC, and SGaw all improved significantly after metaproterenol administration. Lung volumes decreased significantly post-broncho-

| Table 1—Pulmonary Function Results before and after Bronchodilator Administration |
|-----------------------------------|-------------|-------------|
| **Normal Subjects** | **Post-BD** | **Asthmatic Subjects** | **Post-BD** |
| FEV1 (L) | 3.34 ± .64 | 3.50 ± .70† | 2.21 ± .85‡ | 2.86 ± .99‡ |
| (percent predicted) | 98 ± 9 | 103 ± 10† | 60 ± 18‡ | 73 ± 20‡ |
| FVC (L) | 4.18 ± .77 | 4.26 ± .83† | 3.65 ± 1.1 | 4.15 ± 1.1† |
| FVC (percent predicted) | 101 ± 14 | 102 ± 14† | 84 ± 17† | 95 ± 14† |
| SGaw(cm−2·sec−1) | .19 ± .06 | .28 ± 1.14† | .08 ± .06§ | .15 ± .08‡ |
| RV (L) | 1.64 ± .47 | 1.54 ± .46 | 2.71 ± .83‡ | 2.02 ± .75‡ |
| TLC (L) | 5.85 ± .95 | 5.78 ± .97 | 6.52 ± 1.35 | 6.16 ± 1.30† |
| DLco(ml/min/mmHg) | 28.9 ± 5.6 | 27.1 ± 8.2 | 32.3 ± 18.0 | 32.3 ± 18.04 |

*BD = bronchodilator
†p<.05-.001 compared to pre-BD values
‡p<.001 compared to corresponding value in normals
dilator only in the asthmatic group.

Flow Volume Maneuvers

Mean results for normal and asthmatic subjects are shown in Table 2. Compared to results in the normal subjects, \(\dot{V}_{\text{max}}/p\) and \(\dot{V}_{\text{max}}/c\) were significantly lower in the asthmatic subjects, both pre- and post-metaproterenol.

The ratio \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c\) (pre-metaproterenol inhalation) was less than 1 in 18 of 24 normal and in 11 of 24 asthmatic subjects (p < .05). All normal and asthmatic subjects with \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c < 1\) pre-bronchodilator had an increase in the ratio post-metaproterenol administration. Among those asthmatic subjects showing \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c < 1\), the ratio was lower than in the corresponding group of normal subjects (.69 ± .11 vs .85 ± .09, p < .001).

To characterize asthmatic subjects who were able to increase their flow rates after a deep inspiration, physiologic variables were compared for asthmatic subjects with \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c\) ratios <1 and ≥1 (Table 3). The \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c < 1\) group had significantly higher FEV\(_1\), FVC, \(\Delta V_{\text{max50}}\), and significantly lower residual volume; there was no significant difference in SGaw or bronchodilator responsiveness. Using a least squares regression, \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c\) was inversely related to FEV\(_1\) (r = -.56, p < .01) and percent predicted FEV\(_1\) (r = -.47, p < .025) (Fig 1). The percent change in FEV\(_1\) post-bronchodilator had no linear dependence on \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c\) (r = -.02). Therefore, better lung function, but not response to bronchodilator, was correlated with lower ratios of \(\dot{V}_{\text{max}}/p/\dot{V}_{\text{max}}/c\).

Response to Inhaled Metaproterenol (Table 4)

In the normal group, absolute increases in \(\dot{V}_{\text{max}}/p\) were much larger than absolute increases in \(\dot{V}_{\text{max}}/c\) (p < .001). Percent change from baseline \(\dot{V}_{\text{max}}/p\) was, on average, comparable to that of SGaw, but much greater than that of FEV\(_1\), FVC, or \(\dot{V}_{\text{max}}/c\). In the asthmatic subjects, absolute increases after metaproterenol were

Table 3—Characteristics of Asthmatic Subjects Grouped by Response to Deep Inspiration (Mean ± SD)

<table>
<thead>
<tr>
<th>(\dot{V}<em>{\text{max}}/p/\dot{V}</em>{\text{max}}/c&lt;1)</th>
<th>(\dot{V}<em>{\text{max}}/p/\dot{V}</em>{\text{max}}/c≥1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV(_1) (L)*</td>
<td>2.78 ± .61</td>
</tr>
<tr>
<td>FEV(_1)%</td>
<td>70.5 ± 14.6</td>
</tr>
<tr>
<td>SGaw(cm(^{-1})sec(^{-1}))*</td>
<td>.07 ± .04</td>
</tr>
<tr>
<td>(\Delta V_{\text{max}})%</td>
<td>39.8 ± 10.6</td>
</tr>
<tr>
<td>FVC (L)*</td>
<td>4.17 ± 1.11</td>
</tr>
<tr>
<td>RV (L)*</td>
<td>2.31 ± .73</td>
</tr>
<tr>
<td>(\Delta FEV_{1.0}) (percent from baseline)</td>
<td>30.8 ± 19.7</td>
</tr>
<tr>
<td>(\Delta SGaw) (percent from baseline)</td>
<td>1.52 ± 1.05</td>
</tr>
</tbody>
</table>

* = Prebronchodilator
† = p < .05-0.001
Table 4—Absolute and Percentage Changes in Physiologic Variables after Inhalation of Metaproterenol (Mean ± SD)

<table>
<thead>
<tr>
<th>Normal subjects</th>
<th>Asthmatic subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Delta \text{FEV}_t ), (L)</td>
<td>.15 ± .13</td>
</tr>
<tr>
<td>( \Delta \text{FVC} ), (L)</td>
<td>.08 ± .13</td>
</tr>
<tr>
<td>( \Delta \text{SGaw} ), (cm⁻¹ sec⁻¹)</td>
<td>.09 ± .11</td>
</tr>
<tr>
<td>( \Delta \text{V}_{max-p} ), (L/min)</td>
<td>42.8 ± 19</td>
</tr>
<tr>
<td>( \Delta \text{V}_{max-c} ), (L/min)</td>
<td>15.3 ± 17.5±</td>
</tr>
<tr>
<td>( \Delta \text{FEV} ), (percent from baseline)</td>
<td>5 ± 4t</td>
</tr>
<tr>
<td>( \Delta \text{FVC} ), (percent from baseline)</td>
<td>2 ± 3t</td>
</tr>
<tr>
<td>( \Delta \text{SGaw} ), (percent from baseline)</td>
<td>45 ± 50</td>
</tr>
<tr>
<td>( \Delta \text{V}_{max-p} ), (percent from baseline)</td>
<td>50 ± 24</td>
</tr>
<tr>
<td>( \Delta \text{V}_{max-c} ), (percent from baseline)</td>
<td>15 ± 16t</td>
</tr>
</tbody>
</table>

*p<.05-.001 compared to values in normals
†p<.05-0.001 compared to \( \Delta \text{V}_{max-p} \) (percent from baseline) in the same group
‡p<.001 compared to \( \Delta \text{V}_{max-p} \) (absolute change) in the same group

larger for \( \text{V}_{max-p} \) than for \( \text{V}_{max-c} \) (p<0.001). Percent changes in \( \text{V}_{max-p} \) and \( \text{SGaw} \) were comparable, but were greater than the mean percentage changes of \( \text{FEV}_t \), \( \text{FVC} \), and \( \text{V}_{max-c} \).

Except for absolute increase in \( \text{V}_{max-p} \) and \( \text{SGaw} \), all changes in physiologic variables after metaproterenol were greater in asthmatic than in normal subjects. To evaluate the ability of different tests to separate normal from asthmatic responses to metaproterenol inhalation, we determined the number of asthmatic subjects with bronchodilator responses (expressed as percent change from baseline) greater than those observed on the corresponding test of 95 percent of the normal group. Such responses occurred in 24 (\( \text{FEV}_t \)), 17 of 24 (\( \text{FVC} \)), seven of 24 (\( \text{SGaw} \)), 13 of 24 (\( \text{V}_{max-p} \)) and 17 of 24 (\( \text{V}_{max-c} \)) asthmatic subjects. Since our asthmatic group was selected on the basis of percent change in \( \text{FEV}_t \), ±15 percent, it was expected that this test would provide the best separation. To further analyze our data, we divided our asthmatic subjects into a group with \( \text{FEV}_t \), ≥70 percent of predicted and a group with \( \text{FEV}_t <70 \) percent of predicted (pre-bronchodilator). Nevertheless, we still found no advantage in using partial flow volume curves to separate normal and asthmatic responses in either group.

**DISCUSSION**

A total lung capacity (TLC) volume history could affect flow rates through several mechanisms. Using the equal pressure point model, \( \text{V}_{max} = \text{Pst}/\text{Rus} \), where \( \text{Pst} \) is lung elastic recoil pressure and \( \text{Rus} \) is upstream segment resistance. A deep inspiration is known to decrease \( \text{Pst} \) and thereby decrease the driving pressure for flow, as well as decrease external traction on the airways and airway diameter. Alternatively, a post inspiratory decrease in bronchomotor tone would decrease resistance. The net change in \( \text{V}_{max} \) would therefore depend on the relative magnitude of the effects. Flow rates on complete curves initiated from TLC would be greater than flow rates on partial curves, if airway resistance on the former decreased more than elastic recoil pressure. Thus, \( \text{V}_{max-p}/\text{V}_{max-c} <1 \) provides evidence of airway hysteresis, but \( \text{V}_{max-p}/\text{V}_{max-c} =1 \) does not rule out the effect.

Conversely, other effects might cause flow rates on PEFV curves to be greater. According to the analysis of Melissinos and coworkers, faster compartments in a nonhomogeneous lung should increase flow on PEFV curves relative to MEFV curves, since any given volume on the partial curve is nearer the start of exhalation. In their study, seven of seven bronchitis patients, three of five normal smokers and no nonsmoking normal subjects out of 14 had evidence of time dependent emptying of different lung compartments. Nonhomogeneous emptying could therefore have affected our results in asthmatic subjects, but not in the normal subjects.

In our study, 18 of 24 normal and 11 of 24 asthmatic subjects increased flow rates after a deep inspiration (\( \text{V}_{max-p}/\text{V}_{max-c} <1 \)). The results in the normal subjects corroborate recent data and indicate that normal, nonprovoked airways have bronchomotor tone which can be reduced by deep inspiration. Furthermore, the proportions of asthmatic individuals exhibiting airways hysteresis has varied in different studies. The reasons for these differences between studies are unclear, but may depend on the population of asthmatic subjects studied. In the present study, the response to a deep breath was most impaired in severe asthmatic subjects.

Administration of metaproterenol blocked the increase in flow rates due to a TLC volume history (Table 2). Baseline airway tone prior to inspiration to TLC would presumably determine the magnitude of possible change after inspiration to TLC. Bronchodilatation (from metaproterenol inhalation) by markedly decreasing tone could blunt the effect of deep inspiration. In contrast, bronchoprovocation (from histamine or methacholine), by increasing baseline airway tone, would increase the possible change that could occur after a TLC volume history. In this regard, it is of interest that the 11 asthmatic subjects with pre-bronchodilator \( \text{V}_{max-p}/\text{V}_{max-c} <1 \) had a lower mean ratio \( \text{V}_{max-p}/\text{V}_{max-c} <1 \) than did the corresponding group of 18 normal subjects (69 ± 11 vs 85 ± 0.9, p<0.001). To our knowledge, this has not been demonstrated previously, but might be expected as asthmatic subjects have higher baseline bronchomotor tone; those asthmatic subjects retaining the ability to bronchodilate after a deep inspiration would have a larger possible change in bronchomotor tone.

In the asthmatic group, a significant correlation was present (before metaproterenol) between \( \text{V}_{max-p}/\text{V}_{max-c} \)

Flow-Volme Curves Before and After Metaproterenol (Bryan, Faltaeh)
and the severity of airway disease (as assessed by FEV₁). The ratio was not related to the subsequent response to inhaled metaproterenol. Fish et al. found no relationship between the ability to increase flow rates after a deep inspiration and the degree of obstruction (flow rates) in a group of mild asthmatic subjects (baseline FEV₁ = 88.4 ± 3 percent of predicted [mean ± SEM]) before or after inhalation of methacholine. However, this asthmatic group had much milder obstruction than our asthmatic subjects, as well as a smaller range of abnormality. In addition, induced bronchoconstriction may not be totally equivalent to the de novo obstruction present in our asthmatic subjects. Also, Orehek et al. found that asthmatic subjects who did not decrease airway resistance following a deep breath had more severe asthma (frequency of attacks of bronchospasm) compared to asthmatic patients with similar pulmonary function who did respond to a deep inspiration. Gaynard et al. also showed that asthmatic patients with higher baseline specific airway resistance (SRaw) had greater increases in SRaw following a deep inspiration.

In our study, asthmatic subjects showing Vmax-p/Vmax-c < 1 also had more density dependence of flow than the remainder of the asthmatic subjects. In a previous study, all of the asthmatic subjects with Vmax-p/Vmax-c < 1 responded to He-O₂ inhalation. Thus, in volume history responsive asthmatic subjects, large airways may contribute more to flow limitation than peripheral airways during MEFV maneuvers. This does not indicate that sites of flow limitation are, on average, normal in asthma. On the contrary, consistent with previous studies, the lower mean ΔVmax in our entire asthmatic group (compared to the normal group) suggests more peripheral sites of flow limitation. However, within the asthmatic group, sites of flow limitation appear to be more central in the volume history responsive subjects.

The more central site of airflow limitation in the volume history responsive asthmatic group during MEFV maneuvers may reflect reduction in peripheral airway resistance by the TLC volume history prior to forced exhalation. This is compatible with the observation of Burns and colleagues that deep inspiration increased SGaw in a group of asthmatics, but did not cause dead space hysteresis. These investigators reasoned that an increase in large airway caliber after a deep breath should be reflected by dead space hysteresis, whereas an increase in SGaw (after lung inflation) would indicate hysteresis of the total airway. Since deep inspiration apparently did not dilate the large airways of their asthmatic subjects (no dead space hysteresis), the increase in SGaw was presumably due to dilatation of more distal airways.

Experimental evidence suggests that, in normal lungs, the peripheral airways are more responsive than the central airways to lung inflation. However, previous studies have suggested that peripheral airway disease becomes more severe as airflow obstruction (assessed by FEV₁ and Vmax) from asthma worsens. If bronchodilatation after deep inspiration should occur in the peripheral airways, then airway hysteresis could be impaired in proportion to the severity of peripheral airway disease. This is compatible with our observations that airflow obstruction was generally worse in asthmatic patients with a Vmax-p/Vmax-c ratio $\geq 1$ and that Vmax-p/Vmax-c correlated inversely and significantly with FEV₁. An alternate interpretation for the inverse relationship of Vmax-p/Vmax-c and FEV₁, in asthma would be that, rather than failure to show hysteresis, asthmatic subjects with worse lung function simply had higher flow rates due to more inhomogeneity in their lungs. The fact that the asthmatic subjects with Vmax-p/Vmax-c $<$ 1 had greater FEV₁, but not SGaw could also suggest that the ability to increase flow rates after a TLC volume history may be a determinant of FEV₁ rather than vice versa.

Flow volume curves measured with a spirometer as opposed to volume displacement plethysmography ignore the effect of gas compression during forced exhalation. If compressive effects differed on our partial and MEFV flow volume maneuvers, Vmax-p and Vmax-c would not have been measured at the same lung volume. To our knowledge, there are no data comparing PEFV and MEFV curves measured simultaneously with a spirometer and a volume displacement plethysmograph.

In agreement with the work of Parry et al. absolute changes (after metaproterenol inhalation) were greater for Vmax-p than for Vmax-c, particularly in normal subjects. Percentage changes in Vmax-p were also much larger than those observed for that of FEV₁, FVC, and Vmax-c, but were comparable to SGaw in both groups (Table 4). Since Vmax-p and SGaw measurements are not preceded by a TLC maneuver, these tests may be more sensitive to pharamacologic change in bronchomotor tone because they avoid the effects of the TLC volume history. In asthma, due to larger responses to inhaled bronchodilators, this may be less of an advantage.

Absolute changes in Vmax-p after metaproterenol inhalation did not differ significantly in normal and asthmatic subjects; furthermore, percent changes in Vmax-p were less useful than changes in Vmax-c in differentiating individual normal and asthmatic responses to metaproterenol inhalation. Avoidance of the TLC volume history increased the magnitude of response to metaproterenol, but minimized the differences between the groups. Thus, after metaproterenol inhalation, percent change in Vmax-p was 3.33 times greater than percent change in Vmax-c in the normal subjects, but only 1.6 times greater in the asthmatic group. In contrast, previous studies have indicated that
PEFV curves are more useful for quantitating the airway response to inhaled bronchoconstrictor agents. In summary, a TLC volume history improved expiratory flow in many normal subjects without induced bronchoconstriction, and in some asthmatic patients with spontaneous bronchospasm. Asthmatic subjects who had improved expiratory flow rates after lung inflation had higher FEV₁ values and more density dependence of expiratory flow. In both normals and asthmatic subjects, average change in Vₘₚ and SGaw were comparable after metaproterenol inhalation and were much larger than changes in FEV₁, FVC, and Vₘ₋C. Partial flow volume curves are useful for studying alterations in bronchomotor tone, but appear to offer no added advantage in separating normal from asthmatic individuals on the basis of response to inhaled metaproterenol.

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APPENDIX

In Table 2, the mean values for Vₘ₋p (40.8 L/min) and Vₘ₋c (43.6 L/min) in the asthmatics might appear inconsistent with the mean Vₘ₋p/Vₘ₋c ratio of 1.0; the same would appear true after bronchodilators. The results have, in fact, been verified. The ratio of the mean flow rates does not necessarily equal the mean of the ratios, particularly in a group with a wide range of flow rates. For example, two of our asthmatics (subjects 22 and 23) had values for Vₘ₋p, Vₘ₋c and Vₘ₋p/Vₘ₋c of 18 L/min, 12 L/min, 1.5 and 78 L/min, 102 L/min and 0.76 respectively. The mean Vₘ₋p/Vₘ₋c ratio for these subjects is 1.13; however Vₘ₋p averages 96 L/min and Vₘ₋c 114 L/min. In the normal subjects, the ratios of mean flow rates is similar to the mean of the ratios since the range of expiratory flow rates was more homogeneous in that group. It should not be inferred that the differences between the asthmatics and normals were an artifact of high Vₘ₋p/Vₘ₋c ratios in a few severe asthmatics. As can also be seen in table 2, the frequency of Vₘ₋p/Vₘ₋c ratios <1 differed significantly between the groups pre and post-metaproterenol and within the groups pre and post-metaproterenol.

REFERENCES