Table 1—Methacholine Test Results and Degree of BHR

<table>
<thead>
<tr>
<th>PC_{20}, Methacholine</th>
<th>Mean in Positive</th>
<th>&lt;8 mg/mL, No.</th>
<th>&lt;25 mg/mL, No.</th>
<th>Negative, No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic HIV patients</td>
<td>8.7±9</td>
<td>1</td>
<td>2</td>
<td>21</td>
</tr>
<tr>
<td>AIDS patients</td>
<td>14.2±10</td>
<td>6</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>Injected-drug users</td>
<td>13±10</td>
<td>3</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>Non-drug users</td>
<td>4.5±3</td>
<td>6</td>
<td>16</td>
<td>11</td>
</tr>
<tr>
<td>Prior HIV-related lung disease</td>
<td>14.6±11</td>
<td>3</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>No HIV-related lung disease</td>
<td>9.5±9</td>
<td>6</td>
<td>11</td>
<td>15</td>
</tr>
</tbody>
</table>

partially explain the different results. On the other hand, the influence of asthma was negligible in our study. In our series, the number of injected-drug users was larger (61.3% vs 13.6%) and they showed more mild, but not moderate, BHR increase than those who did not use drugs. Even when the results are difficult to compare because of the different methodologies, different risk factors for HIV infection, and a possible control bias, they are probably similar to those of Wallace et al. and Moscato et al.6 The increase in moderate BHR in asymptomatic HIV patients is difficult to explain but may be in concordance with our previous results.8 However, these findings are of uncertain clinical significance at the moment and only a follow-up of these patients could show if they are exposed to a higher risk for pulmonary infections or clinically relevant BHR.

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REFERENCES


To the Editor:

Chiner et al have suggested that their experience with nonspecific airway hyperresponsiveness (AHR) in patients with HIV disease was similar to ours.1 Yet, they found a significant difference in the proportion of their patients with HIV disease who demonstrated AHR compared with a control group of healthy volunteers (20.5% vs 4.3%), whereas our results suggest no increase prevalence of AHR in HIV-infected subjects compared with matched controls (19.3% vs 12.9%). We agree that differences in sampling and methodology make it extremely difficult to compare results from the two studies.

The proportion of control subjects with AHR in the study of Chiner et al was substantially lower than ours, suggesting that these groups differed between the two studies. We obtained detailed demographic and medical history information from both HIV-infected and control subjects. In an attempt to remove bias due to factors other than HIV disease, we matched each control to an HIV-infected cohort member according to age, gender, race, smoking status, prior asthma, and pretest FEV1. Four pairs were excluded from the analysis because matching for all six variables was not considered adequate. Chiner et al did not provide the data to assess comparability of factors other than HIV disease between their subject and control groups. In addition, it would be important to know the proportion of injecting drug users included in their control group, given that the majority of HIV-infected subjects were users.

In the study of Chiner et al, the proportion of HIV-infected subjects who had previous pulmonary disorders was very large compared to that of our HIV-infected cohort members. The majority of their HIV-infected subjects seem to have had either “non-HIV-related lung disease” or “prior HIV-related lung disease,” including 18 who had either Pneumocystis carinii infection or tuberculosis. Only one of our HIV-infected cohort members had a prior episode of pneumonia. Other reports2,3 have suggested that AHR may occur subsequent to pulmonary opportunistic infection, especially P carinii pneumonia. Perhaps this phenomenon has contributed to the higher prevalence of AHR among the HIV-infected subjects compared to the controls in the work of Chiner et al.

Finally, the methodology used in performing the methacholine challenge, and presumably the dose administered, differed substantially between the two studies. How these differences in technique might have contributed to differences in the results is not immediately apparent.

The issue of whether airway injury is a complication of HIV remains open. Further studies, especially in persons with more advanced HIV disease and prior pulmonary infection, are needed.

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REFERENCES

Usefulness of the Flow Volume Loop

To the Editor:

We have read with interest the article by Guntupalli et al (February 1997) on the usefulness of the flow volume loop (FVL) in emergency and ICU settings. The selection of cases where the FVL can easily help to establish the level of flow impairment was well chosen by the authors. In emergency situations, the FVL is easily helpful in identifying certain abnormalities quickly by visual pattern recognition. With respect to this, we were particularly interested in the two cases of variable upper airway obstruction: case 1 with bilateral vocal cord paralysis (BVCP) and case 5 with vocal cord dysfunction (VCD). The clinical description of BVCP was adequate, with a patient being able to speak almost normally but having tremendous difficulty breathing in, especially on the slightest effort. Therefore, the FVL will show a severely reduced forced inspiratory flow but almost normal expiratory flow, with the exception of an amputated peak flow, and usually a plateau following the peak flow (Fig 1). Contrary to the loop shown by Guntupalli et al, there usually is no restrictive pattern if the patient has normal lungs. In the patient shown, one can see that she did not inhale completely from residual volume to total lung capacity because the inspiratory flow is clearly not zero at the y-intercept. Thus, the FVL shown is compatible with, but not typical for, BVCP. We previously published a typical loop in a patient with BVCP, with a perfectly normal FEV\textsubscript{1} and FVC.\textsuperscript{2}

A second point relates to the FVL pattern of the VCD illustrated by case 5. Again, the loop shown in Figure 10 is compatible with, but not typical for, VCD. It is also possible that the expiratory curve is perfectly normal with the inspiratory curve being completely flat,\textsuperscript{3,4} identical to the one obtained in BVCP. A further differential diagnosis with identical FVL to VCD is variable upper airway obstruction above the larynx due to pharyngeal constriction without concomitant VCD. Such a case has been described by Nagai et al (Fig 2).\textsuperscript{5} The only difference between the FVL in VCD and in BVCP is the amputated peak flow in the latter. In our opinion, the authors should have mentioned the lack of specificity of the FVL in both their cases in this otherwise excellent article.

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REFERENCES


To the Editor:

We appreciate the comments of Dr. Bollinger regarding two of the cases described in our article.\textsuperscript{1}

As Dr. Bollinger correctly points out, the flow volume loop in case 1 (the patient with bilateral vocal cord paralysis) is suggestive of a restrictive-type lung defect. Although there are clinical reasons why this patient could have such a restrictive lung defect (metastatic disease to the lung with or without radiation-induced charges), we do not have further information, such as lung volume measurements, to confirm the presence of these restrictive lung conditions.

For case 5 (the patient with vocal cord dysfunction), we agree that the appearance of the flow volume loop is nonspecific, as he suggests. This was included in the broader differential at the end of the article.