
3 Larsson L, Marth PA, Oldham G. Detection of tuberculostearic acid in mycobacteria and nocardiae by gas chromatography and mass spectrometry using selected ion monitoring. J Chromatogr 1979; 163:221-24

To the Editor:

I thank Dr Pang for his comments on our article. My personal feeling is that it would be hard to envisage Actinomycetes species, which normally colonize the oropharynx, amidst the anaerobic organisms gaining entry into the pleural space. If anything, they should interfere more with results of sputum analysis for tuberculostearic acid, which has not been shown.1 Organisms of the other important member of Actinomycetales, namely, Nocardia, can be present in specimens due to environmental contamination. However, it is not very likely that they would be present in our specimens of pleural fluid, which were aspirated under sterile technique and stored in very clean, if not completely sterile, tubes.

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REFERENCE

1 French GL, Chan CY, Cheung SW, Oo KT. Diagnosis of pulmonary tuberculosis by detection of tuberculostearic acid in sputum using gas chromatography-mass spectrometry with selected ion monitoring. J Infect Dis 1987; 156:356-62

Variability in Aerosol Output of the DeVilbiss 646 Jet Nebulizer

To the Editor:

We read with interest the article by Hollie et al.,1 which appeared in the November 1991 issue of Chest. In that article the authors reported on extreme variability in aerosol output of the DeVilbiss 646 jet nebulizer, which could lead to problems in both medical care and bronchoprovocation testing.

When we started using the DeVilbiss 646 nebulizer for bronchoprovocation testing ten years ago following the protocol of Chai et al.,2 we also observed the strong dependence of the nebulizer output on the orientation of the straw and its vertical position. Since that observation, we have always oriented the straw in the 12 o’clock position and repeatedly checked that it was firmly seated. However, after prolonged use, the straw fitted more loosely, just as described by Hollie et al.2

The remedy was to fix the straw on the jet port with an adhesive by wetting the lower part of the jet tube with acetone with use of a fine brush and then plugging in the straw quickly. The proper orientation of the straw must have been determined previously from an analysis of the nebulizer output and must be checked after fixing the straw.

In our laboratory, the aerosol is generated during five nebulizations for 0.6 s at an oxygen pressure of 2×104 dynes/sq cm, with a standard output of 15 mg of saline per nebulization. One can easily account for small systematic differences in output by applying appropriate factors in the computation of provocative concentrations. Whenever possible, we use a single nebulizer within one study. The output is checked at least every two months by determining the weight difference over ten nebulizations; this maneuver is repeated ten times. It may be estimated that a variation coefficient of 8 percent or less within the ten repeated weighings will suggest, with a probability of about 90 percent, that the deviations from the mean output for five nebulizations will be 20 percent or less. In our experience, any significant change in output is reflected in a noticeably altered noise during nebulization.

The accuracy that can be achieved is illustrated by recent data on the circadian rhythm of airway responsiveness.3 From a residual analysis it can be inferred that the variability of provocative concentrations is within ±0.5 doubling concentration.

In summary, when the procedures described above are followed, the DeVilbiss 646 nebulizer is an accurate and reliable tool for bronchoprovocation testing.

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REFERENCES


Volume Adjustment of Maximal Expiratory Flow Rates of Flow-Volume Loops

To the Editor:

A problem limiting the use of flow-volume loops is the wide variation of predicted values for maximal expiratory flow rates. In patients with interstitial lung disease with a purely restrictive pattern, maximal expiratory flow rates are low due to volume loss. This decrease in flow-volume rates compared with prediction tables may be misinterpreted as airflow limitation. Flow standardized to lung size by division by lung volume is interpreted as a variable reflecting the presence or absence of airflow limitation. The best example of this is the ratio of FEV1 to vital capacity (VC). The FEV1 is a volume, but it is numerically equal to the average flow during the first second of a forced expiration. It may be evaluated as a flow related to lung size. The lung size used may be total lung capacity, but since VC may be measured from the flow-volume loop, VC is a more practical measurement of lung size.

We investigated the relation of maximal expiratory flow rates to lung size by dividing maximal expiratory flow rates measured at 75 percent, 50 percent, and 25 percent of VC by VC. In all, 201 healthy subjects, 166 patients with chronic obstructive lung disease, and 30 patients with interstitial lung disease entered the study. Spirometry was performed and flow-volume loops were drawn in a volume-displacement body plethysmograph (Fenyes and Gut, Basel, Switzerland). The index of maximal expiratory flow rate divided by VC was decreased in patients with chronic obstructive lung disease (p<0.001). It was increased in patients with interstitial lung disease (p<0.001). The mean values and confidence limits of the index are summarized in Table 1.
Table 1—Maximal Expiratory Flow Rates Divided by Vital Capacity*

<table>
<thead>
<tr>
<th>Subjects</th>
<th>( \dot{V}_{\text{max75%/VC}} )</th>
<th>( \dot{V}_{\text{max50%/VC}} )</th>
<th>( \dot{V}_{\text{max25%/VC}} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (n = 201)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>1.74</td>
<td>1.301</td>
<td>0.703</td>
</tr>
<tr>
<td>SD</td>
<td>0.52</td>
<td>0.346</td>
<td>0.212</td>
</tr>
<tr>
<td>CL</td>
<td>1.66-1.81</td>
<td>1.25-1.35</td>
<td>0.64-0.71</td>
</tr>
<tr>
<td>COLD (n = 166)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.82</td>
<td>0.455</td>
<td>0.238</td>
</tr>
<tr>
<td>SD</td>
<td>0.428</td>
<td>0.255</td>
<td>0.12</td>
</tr>
<tr>
<td>CL</td>
<td>0.737-0.904</td>
<td>0.405-0.504</td>
<td>0.215-0.262</td>
</tr>
<tr>
<td>ILD (n = 30)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>2.12</td>
<td>1.67</td>
<td>0.903</td>
</tr>
<tr>
<td>SD</td>
<td>0.68</td>
<td>0.75</td>
<td>0.30</td>
</tr>
<tr>
<td>CL</td>
<td>1.87-2.38</td>
<td>1.39-1.95</td>
<td>0.72-1.02</td>
</tr>
</tbody>
</table>

*COLD = chronic obstructive lung disease; ILD = interstitial lung disease; \( \dot{V}_{\text{max}} \) = forced expiratory flow when the lung is at a given percentage of its vital capacity; VC = vital capacity; SD = standard deviation; CL = confidence limits with 0.05 risk.

Our preliminary study suggests that maximal expiratory flow rate divided by VC can be used as a predictive value in evaluating flow-volume loops.

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REFERENCE


Chronic Cough Caused by Endobronchial Sutures

To the Editor:

I was fascinated by the case report by Shure,1 which appeared in the November 1991 issue of Chest. Dr Shure reported chronic cough caused by endobronchial sutures in adults. We have dealt with a similar situation.

The patient presented at the age of 13 years with marked wheezing on exercise. A chest x-ray film showed an air-trapping lesion in the left main-stem bronchus. An adenoma was seen at bronchoscopy and resected at thoracotomy, and the boy progressed well. Two years later he developed an intractable cough, which was not associated with abnormalities on examination or at radiography; however, there was minor reduction in FEV1. A trial of bronchodilators and inhaled corticosteroids was made without success. At bronchoscopy, granulation tissue was seen at the site of the anastomosis, and the presence of a silk suture was obvious. The suture could not be removed by forceps. It was vaporized by laser treatment. Examination of a biopsy specimen revealed granulation tissue only, without recurrence of the tumor. The boy's cough resolved quickly and has not recurred during a two-year follow-up period.

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REFERENCE

1 Shure D. Endobronchial suture: a foreign body causing chronic cough. Chest 1991; 100:1193-96

To the Editor:

Dr Mitchell describes an interesting case of symptomatic endobronchial suture occurring remote from the surgical procedure, similar to the cases described in my recent report. Dr Mitchell's successful removal (by vaporization) of the suture with a laser is similar to that reported by Unger.1 While lasers can certainly eliminate suture material, it will be interesting to see what can be achieved with the wider use of endoscopic scissors, rather than conventional biopsy forceps, in the treatment of this condition. If future experience is similar to that described in my series, this technique offers a lower risk and less expensive approach to the problem.

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REFERENCE


Surgical Correction of Posttraumatic Ventricular Septal Defect via the Right Atrium

To the Editor:

I was somewhat disappointed by the article by Sisto et al,1 which appeared in the November 1991 issue of Chest. Their article reports on four patients seen in their institution between 1983 and 1989, in whom a transatrial repair of a posttraumatic ventricular septal defect (VSD) was undertaken. They discussed four patients; however, they did not inform us whether there were other patients during that same time period with a similar diagnosis. They at no point in the article compare the transatrial repair of a VSD with other methods of repair.

Patient data are not available in the article. We are not told of the patients' ages nor of any other medical conditions. Perhaps the most bothersome part of the article is that although they state that surgery is indicated when the QP:QS ratio is greater than 2.1, they give no indication as to the results of medical therapy in a similar group. Although this is not a study article comparing two different groups, it would be difficult for me to imagine that their results would be poor considering that they state that their patients "were treated expectantly without any medication" and underwent elective cardiac catheterization. As Dr Daggett has pointed out in the past,2,3 these patients appear to be self-selected and one would expect them to do well. My question remains, however, if they were doing well without medication, was the indication for surgery based solely on the cardiac catheterization data?

My congratulations to Dr Sisto and his group for their four successful cases of posttraumatic VSD repair via the right atrium.

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REFERENCES


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