nonresponsive. In our patients, the decrease in bronchial responsiveness was related to the decrease in cardiothoracic index (r = 0.72, p<0.01) and in radiologic score for lung edema (r = 0.61, p<0.05), but not to any change in spirometric values.

We think that chronic lung congestion might cause airway changes (ie, muscular hypertrophy*), which were not reversible in a short time (15 days in the study by Pison et al® and 2 months in the study by Nishimura et al®). Serial measurements of bronchial responsiveness for prolonged time after MVR could help to elucidate this point.

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REFERENCES

To the Editor:
We appreciate the opportunity to respond to the comments of Dr Rolla and his colleagues.

They showed that bronchial hyperreactivity (BHR) was still observed 35 months after mitral valve replacement (MVR) in patients with mitral valve disease. In our study, BHR was evaluated two months after MVR. Decreased BHR after MVR in some surgically treated patients is consistent both in our results and in those of Rolla et al.

We evaluated the BHR 12 months after MVR (Table 1) and found that BHR was significantly reduced in four of six patients, two of whom became nonresponders, although the parameters of pulmonary function testing were not significantly changed.

There are many factors involved in the change of severity in BHR after MVR. We also think that the persistence of BHR at two and 12 months after MVR in our study and at 35 months in the study by Rolla et al might have resulted from airway remodeling induced by long-standing congestion, adding to the increased vagal tone associated with pulmonary congestion and the geometric change due to bronchial edema in mitral valve disease (Y. Nishimura, H. Maeda, A. Hashimoto, H. Nakamura, Y. Hashimoto, et al, unpublished data).

Prolonged follow-up study will be necessary for us to elucidate the pathogenesis of BHR in patients with mitral valve disease, as proposed by Dr Rolla and his colleagues. Again, we would like to thank Dr Rolla and his colleagues for their helpful comments.

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Table 1—Bronchial Reactivity to Inhaled Methacholine and Pulmonary Function Testing before and 12 Months after MVR*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, yr</th>
<th>Sex</th>
<th>Methacholine Inhalation Test</th>
<th>Pulmonary Function Test</th>
<th>Vmax,s, L/s</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Brs, cm H2O/L/s</td>
<td>Log PD20, Grs, log units</td>
<td>Log Dmin, log units</td>
</tr>
<tr>
<td>1 Pre</td>
<td>57</td>
<td>F</td>
<td>5.2</td>
<td>1.382</td>
<td>0.507</td>
</tr>
<tr>
<td>Post</td>
<td>47</td>
<td></td>
<td>4.7</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>2 Pre</td>
<td>63</td>
<td>F</td>
<td>4.9</td>
<td>1.500</td>
<td>0.636</td>
</tr>
<tr>
<td>Post</td>
<td>47</td>
<td></td>
<td>4.7</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>3 Pre</td>
<td>40</td>
<td>M</td>
<td>5.0</td>
<td>0.133</td>
<td>-1.675</td>
</tr>
<tr>
<td>Post</td>
<td>47</td>
<td></td>
<td>4.7</td>
<td>0.473</td>
<td>0.069</td>
</tr>
<tr>
<td>4 Pre</td>
<td>42</td>
<td>M</td>
<td>5.4</td>
<td>1.397</td>
<td>-0.578</td>
</tr>
<tr>
<td>Post</td>
<td>5.3</td>
<td></td>
<td>1.425</td>
<td>0.409</td>
<td>2.09</td>
</tr>
<tr>
<td>5 Pre</td>
<td>44</td>
<td>M</td>
<td>3.6</td>
<td>0.593</td>
<td>-0.538</td>
</tr>
<tr>
<td>Post</td>
<td>3.5</td>
<td></td>
<td>0.134</td>
<td>-0.302</td>
<td>3.38</td>
</tr>
<tr>
<td>6 Pre</td>
<td>53</td>
<td>F</td>
<td>10.4</td>
<td>0.473</td>
<td>-1.125</td>
</tr>
<tr>
<td>Post</td>
<td>14.3</td>
<td></td>
<td>0.268</td>
<td>-1.481</td>
<td>1.68</td>
</tr>
<tr>
<td>7 Pre</td>
<td>65</td>
<td>F</td>
<td>5.9</td>
<td>1.351</td>
<td>-0.866</td>
</tr>
</tbody>
</table>

*Pre = preoperative period; Post = postoperative period; NR = no response during inhalation; Brs = baseline respiratory resistance; PD20 = cumulative dose producing a 35 percent decrease in respiratory conductance; Dmin = minimum cumulative dose required to start to decrease respiratory conductance from baseline; VC = vital capacity; Vmax,s = maximal expiratory flow at 25 percent of VC.
†Died suddenly eight months after MVR.
‡p<0.05 compared with value for preoperative period.

Communications to the Editor

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Positioning vs Vibrations in Chest Physiotherapy

To the Editor:

I enjoyed the interesting article by Stiller et al., which appeared in the December 1990 issue of Chest.

The question was asked whether positioning or vibrations was the effective component of treatment. Research on vibrations has shown as many positive as negative outcomes, which is why vibrations are no longer performed routinely. Accurate positioning, however, is used effectively for the treatment of atelectasis, ventilation-perfusion mismatch, and breathlessness. Positioning can be incorporated into a patient's management plan 24 h a day, and it is cheap.

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REFERENCES

Breakage of Forceps Valve Cover and Its Introduction into a Patient

An Unusual Complication of Fiberoptic Bronchoscopy

To the Editor:

We would like to report an unusual complication that occurred while performing fiberoptic bronchoscopy using Olympus BF-P20 equipment (Olympus Corp, Lake Success, NY). After obtaining the bronchial brush biopsy specimen, a black object, apparently a foreign body, was visualized in the bronchus and was removed by suction. The object had a rubbery consistency, and it exactly matched a hole found in the rubber forceps valve cover of the bronchoscope (Fig 1).

The forceps valve cover is marketed as "semi-disposable," and there are no clear recommendations on the frequency of replacement. Replacement at our institution normally occurs after ten to 30 uses, especially when suctioning becomes suboptimal. The deterioration of the rubber could be due to multiple sterilizations with glutaraldehyde.

Cromolyn for Cough Due to Angiotensin-Converting Enzyme Inhibitor Therapy

Preliminary Observations

To the Editor:

Although estimates of incidence vary, cough is now recognized as a common complication of angiotensin-converting enzyme (ACE) inhibitor therapy. By virtue of the study design, the report by Sebastian et al., which appeared in the January 1991 issue of Chest, provides the best estimate available of the incidence of this troubling side effect in male patients.

The problem was first seen with enalapril, but is now recognized with all of the ACE inhibitors, including several not yet released for use in this country. Cough is severe enough to require cessation of therapy in about 10 percent of patients, and discontinuation of ACE inhibitor therapy is the only treatment suggested by Sebastian et al. For hypertensive patients, alternative drugs can usually be found, but for patients with severe congestive heart failure, ACE inhibitors are less easily replaced because of their potent "afterload reduction" effect. In these cases, a treatment for cough associated with ACE inhibitors would be of great value. In a review of 115 articles on the subject of cough due to ACE inhibitor therapy, 1