paper was published in the Polish medical literature,\(^1\) which at that time, obviously limited its readership.

We studied 15 COPD patients with severe airways obstruction (vital capacity=2.7±1.2 L, FEV\(_1)=1.2±0.6\) L). Pulmonary artery catheterization was performed using the Grandjean microcatheter technique.\(^2\) After completion of baseline pressure and flow (Fick method) recordings, patients were given 60 pg of IB from a metered-dose inhaler. Control measurements were performed 30 min after the inhalation. The results are shown in Table 1.

No changes in the pulmonary arterial pressure, cardiac output, or arterial blood gases were found. We concluded that IB given in therapeutic doses to COPD patients does not affect pulmonary hemodynamics. I hope that our findings contribute to the knowledge of pathophysiologic mechanisms of anticholinergic bronchodilators.

Jan Zielinski, MD, PhD, FCCP,
Department of Respiratory Medicine,
Institute of TB and Lung Diseases,
Warsaw, Poland

REFERENCES

To the Editor:

We thank Dr. Zielinski for his interest in our work. Unfortunately, we were not aware of his report on the lack of pulmonary hemodynamic effects of the administration of ipratropium bromide. Although Sackner et al\(^1\) and Chapman et al\(^2\) with their colleagues performed hemodynamic studies after ipratropium bromide administration, as both the above authors derived the pulmonary hemodynamic measurements only indirectly, the results obtained by Dr. Zielinski may be considered more definitive. These also confirm what is commonly believed to be the absence of appreciable effect of ipratropium bromide on pulmonary hemodynamics.

Kumar Ashutosh, MD, FCCP,
Pulmonary Disease Section,
Department of Veterans Affairs,
Syracuse, New York

REFERENCES

Collapsible Airways Contributing to Airflow Limitation

To the Editor:

I read with interest the pulmonary function test of the month appearing in the March issue (CHEST 1985; 107:856-59). First I'd like to point out that "airways collapse" is in fact a finding in all normal subjects. The evidence for this is the effort-independence of expiratory flow, which has been well-recognized to be due to dynamic compression of the airways,\(^1\) ie, airways collapse. I also contend that the severity of dynamic vs "fixed" airway obstruction cannot be assessed by measuring the thoracic gas compression. According to Boyle's law, the only factor that determines the volume compression is the intrathoracic pressure generated during the forced expiratory maneuver (P*V=constant). Coates et al\(^2\) showed this elegantly by demonstrating the correlation between the measured and calculated volume using Boyle's law. The ability to generate intrathoracic pressure is related to the strength and speed of muscle contraction, and the flow resistance. But it is irrelevant whether the resistance is due to dynamic or static obstruction, since either can result in higher intrathoracic pressure and compressed intrathoracic volume. The statement that the measurement of compressed volume measurement is both sensitive and specific for increased airflow limitation due to airway compression and collapse is thus unfounded.

Finally, I'm not aware of any evidence that therapeutic implications exist in determining the presence of airway compression. For this particular patient, bronchodilators may improve his symptoms given his response on a pulmonary function test. If he would breathe more comfortably by pursing his lips, he'd probably be doing it. And I'd wait for more clinical studies before I'd ever consider the use of a continuous positive airway pressure mask on patients with COPD or asthma.

David C. Chao, MD,
Barlow Respiratory Research Center,
Los Angeles

REFERENCES

To the Editor:

We thank Dr. Chao for his comments and interest in our recent article on thoracic compression artifact due to collapsible airways. We agree with him that some degree of airways collapse is a finding in all normal subjects. We had noted this in our article by stating that some degree of compression is found on pulmonary function testing in all normal subjects. Also, if the maximal expiratory

Table 1—Control Measurements*

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Before IB</th>
<th>After IB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, min(^{-1})</td>
<td>93±15</td>
<td>90±17</td>
</tr>
<tr>
<td>PPA, mm Hg</td>
<td>26.7±11.4</td>
<td>26.0±10.5</td>
</tr>
<tr>
<td>CI, L/min·m(^{-2})</td>
<td>4.39±2.76</td>
<td>4.27±2.52</td>
</tr>
<tr>
<td>TPR, dynes·s·cm(^{-5})</td>
<td>382±275</td>
<td>379±228</td>
</tr>
<tr>
<td>PaO(_2), mm Hg</td>
<td>59±14</td>
<td>60±15</td>
</tr>
<tr>
<td>PaCO(_2), mm Hg</td>
<td>41.3±9.5</td>
<td>41.1±9.9</td>
</tr>
</tbody>
</table>

*PPA=mean pulmonary arterial pressure, CI=cardiac index, TPR=total pulmonary resistance.