plained by COPD alone. This lower limit equation (FVC% PRED = 1.0*FEV1/FVC%) applies well to the authors’ data (Fig 1). The equation identifies 8 to 9% of the men as having an unusually low FVC. In contrast to these working men of Lefante et al, a much greater percentage of patients with COPD admitted for inpatient pulmonary rehabilitation had an unusually low FVC (H. M. Thomas, unpublished data). FVC% PRED was less than FEV1/FVC% in 30% of the 317 rehabilitation patients studied, suggesting that they had complicating abnormalities (eg, respiratory muscle weakness or asthma) or were unable to maintain expiration long enough during spirometric testing to generate a larger FVC.

To better define patients’ abnormal pulmonary function, we need confidence band spirometric data relating reduction in vital capacity to severity of airflow obstruction for uncomplicated COPD and uncomplicated asthma. The present study describing a working population is a step in the right direction.

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REFERENCES

To the Editor:

We appreciate Dr. Thomas’s interest, but we cannot agree that his rule of thumb applies well to the data shown in the figure. Our equations allow upward adjustment of the observed FVC in percent predicted (FVC% PRED) in proportion to the reduction of observed FEV1/FVC. The line added by Dr. Thomas identifies about nine individuals as having FEV1/FVC > 0.65 and FVC% PRED ranging from < 70% to down to about 45%. No one would doubt that these individuals show restrictive abnormality, but what about the more numerous individuals with mild obstruction just above his line? The mathematical adjustment for obstruction will be small because the obstruction is mild, and their adjusted FVCs will still be below the lower limit of normal (however it is reckoned). Those individuals also show physiological restriction, since their obstruction does not account for the subnormal FVC.

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Desensitization to Trimethoprim-Sulfamethoxazole Following Lung Transplantation

To the Editor:

We are writing to report a series of four patients who, following lung transplantation, were successfully desensitized to trimethoprim-sulfamethoxazole (TMS) for prophylaxis against pneumocystis pneumonia. The patients were four women, ages 45-50, who underwent transplantation at the University of Colorado Health Sciences Center between March 1993 and December 1995. Indications for transplantation were bronchiolitis obliterans associated with rheumatoid arthritis, α-1-antitrypsin deficiency, primary pulmonary hypertension, and idiopathic pulmonary fibrosis. Previous allergic reactions to TMS ranged from urticarial rashes of varying anatomic distributions to leukopenia.

Each patient’s immunosuppressive regimen consisted of IV methylprednisolone and azathioprine. All of the patients were successfully desensitized on the first attempt without untoward effects using an oral protocol3,4 (Table I). Subsequent to an initial desensitization, patient no. 3 experienced acute rejection of the transplant, which was successfully treated with intensive immunosuppressive therapy and plasmapheresis. During this episode, TMS therapy was interrupted for 9 days. Because the patient was unable to take oral medications, we reintroduced TMS intravenously in graded doses over 8 hours without difficulty. Our doses were chosen based on those reported for oral desensitization over 8 hours.2 All patients have remained on maintenance therapy with one double-strength TMS (Bactrim, Roche Laboratories, Nutley, NJ) tablet three times a week for a mean of 18 months (range, 8-26 months) without adverse effects.

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REFERENCES

Table 1—Characteristics of Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Initial Reaction to Sulfamethoxazole or TMS</th>
<th>Indication for Transplant</th>
<th>Protocol for Desensitization</th>
<th>Reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>50</td>
<td>Leukopenia</td>
<td>Primary pulmonary hypertension</td>
<td>22 day oral</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>48</td>
<td>Hives</td>
<td>Idiopathic pulmonary fibrosis</td>
<td>8 hour oral</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>45</td>
<td>Confluent macular rash on palms and soles</td>
<td>Bronchiolitis obliterans</td>
<td>22 day oral</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>47</td>
<td>Morbilloid rash on arms and legs</td>
<td>α-1-antitrypsin deficiency</td>
<td>8 hour IV (see text)</td>
<td>None</td>
</tr>
</tbody>
</table>

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