a careful reassessment of the differential diagnosis is an important element in the management of chronic severe asthma. Vocal cord dysfunction syndrome (VCDS) may certainly masquerade as refractory asthma, although the prevalence of this situation is unclear and may not be as high as implied by Drs. Pacht and St. John. In addition, coexisting asthma and VCDS have been described. VCDS, as well as other extrapulmonary conditions that may contribute to asthma pathogenesis, severity, or both, such as gastroesophageal reflux and chronic sinusitis, need to be ruled out before experimental asthma therapy is considered.

Richard B. Moss, MD,
Division of Allergy-Immunology & Respiratory Medicine,
Stanford University, Stanford, California

Pulmonary Hypertension and Right Heart Failure

To the Editor:

Aessopos and colleagues state in the January issue (CHEST 1995; 107:50-3) that recurrent respiratory tract infection was found very often in their patients. It is not surprising because all but one had undergone splenectomy. Therefore, pneumococcal vaccine prophylaxis should have been given.

Calcium channel blockers especially vasodilators like nifedipine and nicardipine certainly evoke the therapeutic idea along with antithrombotic agents in this condition.1,2 Cor pulmonale is the natural sequela to chronic pulmonary hypertension. Do the authors consider this and prescribe angiotensin-converting enzyme inhibitors and diuretics?

This communication is intended to seek clarification, not to criticize an otherwise excellent educational article.

Saeed Ahmad, MBBS, FCCP,
Cardio-Diagnostic Clinique,
Fairmont, West Virginia

References

To the Editor:

We thank Dr. Ahmad for his interest and comments about our article, "Pulmonary Hypertension and Right Heart Failure in Patients With β-thalassemia."

In our report (CHEST 1995; 107:50-3), we emphasize the finding of pulmonary hypertension (PH) as a previous unrecognized complication of β-thalassemia. Regarding treatment, we did not think it necessary to expand our discussion on the treatment of PH per se. We mainly commented on therapeutic interventions related to β-thalassemia, which can modify the development or alleviate the symptoms of PH.

As far as the pathogenesis of PH in β-thalassemia is concerned, we think that it is of multifactorial origin, the respiratory tract infections being one of them. Prophylaxis for splenectomized β-thalassemic individuals has been suggested in the literature and is performed on our patients as a standard policy for the last decade. However, three of our patients were splenectomized before the introduction of the established protocol and did not receive pneumococcal vaccination. We did not include the above information in the article; therefore, we appreciate your comments.

Athanassios Aessopos, MD,
First Department of Medicine,
University of Athens,
Athens, Greece

Reprint requests: Dr. Aessopos, University of Athens, Laikon Hospital, Athens, Greece

Is Not the Treatment Program of Dyspnea Management Strategies Effective for COPD?

To the Editor:

We read the paper by Sassi-Dambron and coworkers concerning the treatment of dyspnea in COPD patients (CHEST 1995; 107:724-29). Although the authors concluded that the treatment program of dyspnea management strategies is not sufficient to produce improvement of dyspnea in COPD patients, we believe that the treatment program may work out if the study protocol was designed well. First, the dyspnea sensation is not appropriately assessed by the authors. Because the Borg category scale is based on the psychophysics Stevens' power law, ie, perceived sensation (S) = K-stimuli1,1 an assessment of dyspnea by Borg scale may be valid only when the scale is standardized by proper stimuli. It has been reported that the unstandardized Borg scale is not reproducible with days in patients with COPD,2 suggesting the assessment of dyspnea by Borg scale alone may not be reliable between the treatment intervals. For the comparison of dyspnea sensation between the intervals, the individual data should be standardized by appropriate stimuli including ventilation, oxygen uptake, and walk distance.3,4 As shown in the table, the consistent reduction of standardized dyspnea score (Borg scale or VAS [visual analog scale]/walk distance) during a 6-min walk (6MW) was found in the treatment group, but not in the control group. Although the calculations do not represent the accurate value reflecting individual measurements, the differences in the standardized dyspnea score between treatment group and control group is relatively clearly observed when compared with the differences in the assessment of Borg scale or VAS alone (Table 1).

Second, an untreated and treatment crossover design may al-

Table 1—Standardized Dyspnea Scores

<table>
<thead>
<tr>
<th>Variables</th>
<th>% Change of Posttreatment Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VAS-6MW (VAS)</td>
<td>Treatment: −10.9</td>
</tr>
<tr>
<td>Control: −1.5</td>
<td></td>
</tr>
<tr>
<td>Borg-6MW (Borg scale [BS])</td>
<td>Treatment: −7.7</td>
</tr>
<tr>
<td>Control: −2.2</td>
<td></td>
</tr>
<tr>
<td>6MW (m)</td>
<td>Treatment: +2.2</td>
</tr>
<tr>
<td>Control: −0.7</td>
<td></td>
</tr>
<tr>
<td>VAS-6MW/6MW (VAS/m)</td>
<td>Treatment: −12.8</td>
</tr>
<tr>
<td>Control: −0.9</td>
<td></td>
</tr>
<tr>
<td>Borg-6MW/6MW (BS/m)</td>
<td>Treatment: −9.3</td>
</tr>
<tr>
<td>Control: −1.8</td>
<td></td>
</tr>
</tbody>
</table>

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