Liang,12 and in the United States by Wilt,13 it appears that this procedure can be done safely, and that significant amounts of dust can be removed. Whether this procedure, alone or in combination with pharmacologic intervention, halts or ameliorates the fibrotic process can only be answered by controlled, long-term studies.

Cases of silicosis continue to occur. The optimum approach to workers with silica exposure must be two-pronged: an emphasis on education of employers and workers with vigilance by regulatory agencies and an aggressive, collaborative effort by basic scientists and clinical researchers to find ways to halt progression, or even better, to reverse the onslaught of aggressive disease in those unfortunate workers who develop this process despite public health measures.

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The Importance of Ethnicity in the Diagnosis and Prognosis of Sarcoidosis

In the previous issue of CHEST, Torrington, Parker, and Shorr1 shed new light on facts, neglected for decades, about endobronchial sarcoidosis that should modify current approaches to its diagnosis. In addition, their analysis helps to explain the widely divergent yield of flexible fiberoptic bronchoscopy for detecting granulomas, regardless of whether endobronchial biopsies (38 to 85%), transbranochial biopsies (43 to 74%), or both (47 to 88%) are examined (Table 1). Remarkably, the yield in the 150 patients studied by Torrington and colleagues1 almost exactly duplicated that obtained by Armstrong et al2 in 101 patients studied 15 years earlier. While the ethnicity of the patients studied by Armstrong et al2 was not reported, Torrington and colleagues1 detected a striking difference in granuloma yields in whites compared with African-Americans that should prove helpful to the practicing physician.

In spite of a similar prevalence of mucosal abnormalities in white and African-American patients (56% and 54%, respectively), the granuloma yield from biopsy was strikingly different, regardless of whether mucosal biopsies, transbronchial biopsies, or both were examined. While the yield ranged from 38 to 52% in white patients, it ranged from 74 to 85% in African-Americans. The high yield from mucosal biopsy, particularly in African-Americans, supports the authors’ recommendations that endobronchial biopsies should be obtained in all patients with mucosal abnormalities. Previous reports by Armstrong et al2, Stjernberg et al,3 and Bjerner et al,4 in which granulomas were detected in biopsy specimens from sites that were grossly normal, support the recommendation to obtain endobronchial biopsies from apparently normal mucosa. Because of the low morbidity associated with endobronchial biopsies, we suggest that they should be obtained in all patients without a contraindication, even in whites in whom the yield was only 38%. The minimal morbidity associated with transbronchial biopsy in the series reported by Torrington and colleagues1 also suggests that this procedure should be performed at the time of the initial bronchoscopy.
In addition to differences in biopsy yield according to ethnicity, the clinical course of sarcoidosis also appears to be affected by ethnicity. We followed 99 African-Americans and 53 whites for a mean of 9.3 years after detection of pulmonary sarcoidosis.5 Even though the initial radiographic opacities had the same character, extent, and severity in these two groups, clinical recovery in patients with no extrapulmonary involvement was observed in 59% of whites, but only in 76% of African-Americans. In patients with extrathoracic disease, recovery was observed in 70% of whites, but only in 46% of African-Americans.

These differences suggest that although sarcoidosis is often less severe in whites than in African-Americans, it is also often more difficult to diagnose. We can therefore add sarcoidosis to the list of diseases, such as prostate cancer, diabetes mellitus, and hypertension, that show racial differences in important aspects such as norms used for disease screening,5 prevalence,7 and pathophysiology.8

What implications do these findings have for our management of patients with sarcoidosis? Some physicians advocate treating virtually all patients with stage II or III sarcoidosis with corticosteroids if they do not show signs of spontaneous recovery.9 A review of our experience,10,11 the results of several clinical trials,12-17 and questions raised by another physician18 have caused us to question the routine use of corticosteroids in such patients.

Our first concern arose from our inability to predict which patients with sarcoidosis were destined to have chronic, relapsing disease, and therefore would likely require prolonged corticosteroid treatment.5,10,19 Meanwhile, controlled studies from around the world consistently reported that prolonged corticosteroid therapy failed to prevent pulmonary fibrosis in patients with sarcoidosis.12-16,18 Even the meticulously controlled study performed by the British Thoracic Society17 found minimal differences (a 9% improvement in FEV1 and FVC) in patients randomized to receive prolonged treatment with corticosteroids in comparison with a control group. In the British study, patients who either improved spontaneously or developed early troublesome symptoms that required corticosteroid therapy were excluded from randomization.17

Finally, a recent re-evaluation of our data demonstrated that relapse was much more common in patients treated with corticosteroids (74%) than in patients not treated (8%), suggesting that not only might corticosteroids not be beneficial in the long-term management of sarcoidosis, but that they might be detrimental.11 In addition, Izumi20 performed a retrospective analysis of 101 asymptomatic patients with sarcoidosis and no extrathoracic disease who were allocated to receive either no treatment (71 patients) or oral corticosteroids (30 patients). After 10 years of follow-up, abnormal chest radiographs were noted in 8 (27%) of the treated group, but only in 2 (2.8%) of the untreated group. Such findings are consistent with the findings of Haslam,21 who reported that patients with the highest pretreatment bronchoalveolar lymphocytosis showed the least abnormality on chest radiographs 3 years later and postulated that the granulomatous reaction of sarcoidosis might be a beneficial response to help control or eliminate an unknown etiologic agent.

In summary, the study by Torrington and colleagues1 highlights the value of endobronchial biopsy in the diagnosis of sarcoidosis, and confirms important differences in diagnostic efficiency according to ethnicity. Because of the milder disease and the relatively better prognosis of sarcoidosis in whites, efforts both to obtain a histologic diagnosis and to begin early steroid therapy may be replaced by careful observation in this group, unless symptoms prove troublesome. Additional controlled studies to determine if, in fact, corticosteroids are harmful in the long-term outcome of patients with sarcoidosis appear warranted.

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Table 1—Yield of Granulomas from Endobronchial and Transbronchial Biopsies in Sarcoidosis

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients (n)</th>
<th>Ethnicity*</th>
<th>Abnormal Bronchial Mucosa (%)</th>
<th>Granuloma Yield from Biopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>W</td>
<td>Endobronchial (%)</td>
<td>Transbronchial (%)</td>
</tr>
<tr>
<td>Stjernberg et al6</td>
<td>29</td>
<td>W</td>
<td>50</td>
<td>41</td>
</tr>
<tr>
<td>Armstrong et al2</td>
<td>101</td>
<td>Not specified</td>
<td>57</td>
<td>57</td>
</tr>
<tr>
<td>Bjerner et al7</td>
<td>62</td>
<td>W</td>
<td>27</td>
<td>45</td>
</tr>
<tr>
<td>Torrington et al4</td>
<td>150</td>
<td>W (39%/AA 61%)</td>
<td>55</td>
<td>70</td>
</tr>
<tr>
<td>Torrington et al1</td>
<td>59</td>
<td>W</td>
<td>56</td>
<td>58</td>
</tr>
<tr>
<td>Torrington et al1</td>
<td>91</td>
<td>AA</td>
<td>54</td>
<td>55</td>
</tr>
</tbody>
</table>

*W=white; AA=African-American.
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'Circa Menstrual’ Rhythmicity and Asthma

Premenstrual asthma (PMA) is a recognized clinical entity. The question of whether PMA is an under- or over-recognized condition has not been adequately addressed. Large-scale longitudinal studies have just not been undertaken. Smaller studies, however, seem to suggest that 30 to 40% of female asthmatics experience a premenstrual worsening of symptoms. Rees1 studied an unsampled series of 81 female patients of reproductive age with asthma and found that 27 (33%) showed a “clear tendency for attacks during the week or ten days prior to the onset of menses with a peak incidence during the two to three days before the menses.” Gibbs and colleagues2 reported the results of a questionnaire returned by 91 women with asthma. Thirty-six (40%) answered “Yes” when asked: “Does your asthma ever seem worse before the menstrual period?” Eliasson et al3 found that 19 (33%) of 57 women with asthma reported significant worsening of total pulmonary symptom scores during the premenstrual period, the menstrual period, or both. These and similar questionnaire studies rely on patient impressions and recollection. As a result, they suffer from a number of inherent biases. Several studies have attempted to use objective data to study the problem. Hanley4 noted statistically significant reduction of peak flow rate at the time of menstruation in those women whose asthma flared up compared to subjects who were unaffected. A similar finding was noted in the study of Gibbs and coworkers.5 In both studies, however, clinically significant deterioration was infrequent. Gibbs noted that the “falls in peak expiratory flow were usually modest and of a degree that would not be expected to result in increased breathlessness.” Gibbs suggested that a heightened awareness of symptoms during the premenstrual period, rather than a demonstrable reduction in pulmonary function, may play a role. In a similar vein, Juniper et al6 discussed altered perception of asthma symptoms as a possible explanation for PMA.

Several investigators have looked at the influence of the menstrual cycle on airway responsiveness. Juniper et al7 examined changes in airway respon-