sarcoidosis, providing long-term follow-up. I find it impossible to account for the spectrum of systemic responses to *Histoplasma capsulatum*—PDH; seropositive, paucibacillary PDH, failing treatment with amphotericin B and responding to CST; and seropositive, otherwise typical sarcoidosis appearing or recurring in a setting of endemic histoplasmosis—other than by postulating a deficiency gradient in cellular immune response.7,8

Treatment: If sarcoidosis is a granulomatous response to an elusive, persistent antigen (unitarian view), the premise that its suppression would forestall the evolution of fatal or disabling pulmonary fibrosis is reasonable. Proof of principle would reside in demonstration of long-term efficacy. If, however, the granuloma, which is the defining characteristic of sarcoidosis, proves to be a regressive response to inefficient, antecedent, cell-mediated processing—probably to a variety of antigens—then its suppression might be inexpedient. I suspect that the belief that persons with sarcoidosis experience a 10% mortality, cited in several standard references, strongly influences the decision to intervene. However, sarcoidosis mortality in reporting population-based settings, which should resemble clinical practice, is approximately 0.5%. It follows under the (doubtful) assumption that, on balance, CST is beneficial long-term in individuals with pulmonary shadowing, sustained treatment of a large number of asymptomatic persons—most of whom would have a self-limited course—would be required to prevent one death. Hillerdal et al furnished CST in a population-based setting only to persons exhibiting progressive pulmonary shadowing, and reported a mortality of 0.8% (4 of 409 patients; mean age at death, 74 years). Johnston, who employed the same indication in a referral setting, furnished it in only 3% and reported a mortality of 0%. The redoubtable Professor Dame Margaret Turner-Warwick recommended observation for 6 to 12 months without treatment, hoping for spontaneous remission in persons with asymptomatic pulmonary shadowing and normal pulmonary function (written communication; March 30, 1999). Lacking evidence of sufficient benefit to offset the adverse effects of sustained CST in persons with pulmonary shadowing, the majority of whom would experience a favorable outcome in the absence of intervention, the framers of a joint statement on sarcoidosis were unable, absent compelling symptoms, to define its indications.11 I do not know to what extent these conservative indications have influenced physician's practice. Whether "substantial progress" has been made in understanding the nature of sarcoidosis, its etiology, or its treatment since publication of Dr. DeRemee's editorial. I leave to the reader and to time to judge.

Thanks also to Dr. Baum for his kind comments, and for drawing my attention to the two articles co-authored by Dr. Schwarz, with which I was not acquainted. In the first article,12 the authors report on the identification of acid-fast bacilli on tissue stains in a large proportion of patients with putative sarcoidosis. As Dr. Baum points out in his article,13 the clinical and radiographic delineation of these cases was insufficient to draw firm conclusions regarding the certainty of sarcoidosis, and the histologic findings were unsupported by culture confirmation. Additionally, other investigators of well-characterized sarcoidosis have not replicated this finding, and polymerase chain reaction evaluation has shown inconsistent results; most were negative. The provocative 1973 case series13 reported on individuals with respiratory disorders, the majority of which were misdiagnosed sarcoidosis, in which the correct diagnosis—fungal or mycobacterial disease—was delayed due to the confounding effect of histologic similarity. In addition, several cases exhibited an association with a fungal agent in which it appeared that the latter was a consequence of sarcoidosis (semi-invasive aspergillosis), CST (Candida neoformans), or fortuitous (cubane sporotrichosis). Case 5, in which a firm diagnosis of sarcoidosis was succeeded by tuberculosis, is a good example. I think, of the overlap between an immunologic vs an infectious response to *Mycobacterium tuberculosis*, paralleling the spectrum of PDH cited above. In summary, while I agree that sarcoidosis is etiologically heterogeneous, and an abnormal cellular response its underlying cause, the two articles lack persuasive evidence of the correctness of either proposition.

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Routine Chest Radiographs After Endoscopically Guided Percutaneous Dilatational Tracheostomy

To the Editor:

We read with great interest the article by Datta et al (May 2003) analyzing chest radiographs after bronchoscopically guided percutaneous dilatational tracheostomy (PDT). While we may instinctively agree with their conclusion that it is not necessary to perform a chest radiograph after an uneventful PDT, if guided by direct vision via bronchoscopy, we do not think

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that the conclusion can be safely made on the basis of only 60 chest radiographs that were retrospectively analyzed.

The authors acknowledge that the incidence of pneumothorax after PDT has been reported as 0 to 3%, and paratracheal placement as 0 to 6%. Any study would therefore need several hundred patients to convincingly exclude the usefulness of a postprocedure chest radiograph. Their own study found two significant complications (3.3%), one tension pneumothorax, and one pneumomediastinum, both suspected clinically, but diagnosed radiologically.

We performed a similar retrospective audit in 2001. Preprocedure and postprocedure chest radiographs were analyzed in 221 patients who underwent bronchoscopically guided PDT performed between 1996 and 2001 in the James Cook University Hospital ICU. We found that the overall complication rate (Table 1) for PDT was low (8.59%). The chest radiograph itself detected only the following four complications (Table 2): tube malrotation in one case; a self-limiting pneumomediastinum that had been noticed clinically before the radiograph had been performed and did not require treatment; and two areas of consolidation that were noticed clinically before the radiograph had been performed and did not require treatment; and two areas of consolidation that required bronchoscopy, but which may have not been related to the PDT. There were no pneumothoraces observed during the study period.

Studies of non-endoscopically guided PDT have shown the incidence of pneumothorax, pneumomediastinum, and paratracheal tube insertion to be up to 12%,2,3 compared to studies of bronchoscopically guided PDT, which have not shown these complications.4 Our audit confirmed the low rate of complications and, similarly to the study by Datta et al.,1 questioned the value of the routine postprocedure chest radiograph. In 221 procedures, the postprocedure chest radiograph served only to review the angulation of one tracheostomy. However, given the low rate of complications, together with the potentially life-threatening nature of potential complications, we are currently continuing this study prospectively and have gathered data on a further 80 patients to date. We agree with Datta et al.1 that more data are required before we can agree that after the performance of uncomplicated, endoscopically guided PDTs in adult patients there is no need to perform a routine postprocedure chest radiograph.

Table 1—All Recorded Complications After PDT*†

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total (n = 221)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding</td>
<td>12 (5.42)</td>
</tr>
<tr>
<td>Consolidation</td>
<td>2 (0.9)</td>
</tr>
<tr>
<td>Surgical emphysema</td>
<td>2 (0.9)</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>0</td>
</tr>
<tr>
<td>Pneumomediastinum</td>
<td>1 (0.45)</td>
</tr>
<tr>
<td>Tube malposition</td>
<td>2 (0.9)</td>
</tr>
<tr>
<td>Total</td>
<td>19 (8.59)</td>
</tr>
</tbody>
</table>

*Values given as No. (%).

Table 2—Complications After PDT Detected Only by Chest Radiograph

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total (n = 221)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumothorax</td>
<td>0</td>
</tr>
<tr>
<td>Pneumomediastinum</td>
<td>1</td>
</tr>
<tr>
<td>Consolidation</td>
<td>2</td>
</tr>
<tr>
<td>Tube malposition</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>4 (1.8%)</td>
</tr>
</tbody>
</table>

Inhaled β-Adrenoreceptor Agonists and Left Ventricular Systolic Function

To the Editor:

We read with interest the article in CHEST by Au et al (June 2003)1 on the risk of inhaled β-adrenoreceptor agonists in patients with left ventricular systolic dysfunction. However, we were astonished not to see tobacco consumption among the recorded risk factors for death or degradation. Tobacco use remains the most preventable cause of death and disability in the United States.2

Far and away the most important cause of lung cancer is exposure to tobacco smoke through active or passive smoking.3 It also is a leading cause of COPD. Tobacco smoking also has been involved in terms of the rate of cardiac death.4 In 2000, Envangelsita et al5 demonstrated, using a retrospective analysis of Veterans Affairs hospital records, that noncompliance with smoking restriction was a main risk factor for multiple hospital readmissions of patients with heart failure.

We would urge the authors of any study dealing with lung-related or heart-related morbidity or mortality to include tobacco usage as part of the variables assessed.

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