Tropical Pulmonary Eosinophilia

To the Editor:

I read with keen interest the article on airway hyperreactivity in tropical pulmonary eosinophilia by Drs Chhabra and Gaur, which appeared in the May 1986 issue of Chest,1 as well as the comments on that article by Drs Chitkara and Donath, which appeared in the January 1990 issue of Chest.2 It was rightly pointed out in the latter communication that the presence of measurable hyperresponsiveness in tropical pulmonary eosinophilia (TPE) might indeed be useful in elucidating the pathogenesis.

In addition to my findings in an article3 quoted in the letter by Drs Chitkara and Donath, we subsequently documented improvement in lung capacity in acute TPE within eight weeks following successful treatment with diethylcarbamazine.4 We also studied the bronchial response to inhaled isoprenaline in TPE and found that patients during the first episode of TPE of recent onset were on the whole unresponsive whereas those experiencing a relapse of the disease showed a significant response.5 The pathologic picture of TPE in the first few weeks is one of outpouring of histocytes, soon followed by eosinophils in alveolar, interstitial, peribronchial, and perivascular spaces and eosinophilic infiltration of the bronchioles with edema of the walls and blocking of the lumen with shedding of mucous membrane and clumps of eosinophils.6 We thought that these earliest changes in TPE may have acted as a mechanical hindrance to satisfactory bronchial response in our patients with primary TPE. It would be interesting to know whether or both patients of Drs Chhabra and Gaur had any previous eosinophilic episode. If these two hyperreactive patients with primary TPE were indeed studied during the first episode of TPE, then the more likely explanation for our finding would be that the bronchospasticity due to intense major basic protein activity and excessive release of leukotrienes7 is so severe in the patients with an acute attack of primary TPE that they, unlike those in relapse, may not show significant bronchial response to the 10 mg of aerosol isoprenaline commonly administered.

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Synchronous Pulmonary and Renal Cancer

To the Editor:

We read with interest the article by Libby et al,8 which appeared in the August 1990 issue of Chest. The authors correctly pointed out that controversy still exists as to the choice of which bronchoscope to use during laser phototherapy (LPT). The advantages of the rigid bronchoscope generally cited are ease of tissue removal by mechanical means, ease of ventilation, better airway control in the event of major endobronchial hemorrhage, and nonflammability. The authors also attempted to show that the rigid bronchoscope was superior in treatment of proximal lesions in the airways.

The authors mentioned some of the disadvantages of the rigid bronchoscope, but we feel that they did not give sufficient emphasis to others that may be equally important. They cited the inability to adequately treat lesions peripheral to the main-stem bronchi, particularly in the upper lobes. They also correctly indicated that it was always necessary to use general anesthesia with the rigid

REFERENCES

Nd:YAG Laser Bronchoscopy: Rigid or Fiberoptic Mode?

To the Editor:

We read with interest the article by Chan et al,1 which appears in the August 1990 issue of Chest. The authors correctly pointed out that controversy still exists as to the choice of which bronchoscope to use during laser phototherapy (LPT). The advantages of the rigid bronchoscope generally cited are ease of tissue removal by mechanical means, ease of ventilation, better airway control in the event of major endobronchial hemorrhage, and nonflammability. The authors also attempted to show that the rigid bronchoscope was superior in treatment of proximal lesions in the airways.

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