Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Acute Pulmonary Effects of Aerosolized Pentamidine

To the Editor:

In the October 1990 issue of Chest, Dr Chan and his colleagues in the Toronto Aerosolized Pentamidine Study Group reported cough in 65 percent and bronchospasm in 24 percent of their patients after the use of aerosolized pentamidine as secondary prophylaxis for Pneumocystis carinii pneumonia (PCP).

In our laboratory, 22 patients with acquired immunodeficiency syndrome received aerosolized pentamidine mesylate followed by aerosolized pentamidine isethionate every four weeks as secondary prophylaxis for PCP. Baseline pulmonary function tests, expressed as percentage of reference value (mean ± SD), were as follows: vital capacity, 94.4 ± 16.9 percent; FEV1, 94.0 ± 18.9 percent; maximal midexpiratory flow, 77.7 ± 29.3 percent; and diffusing capacity, 71.5 ± 19.2 percent. At each aerosolized pentamidine session, spirometry was performed before treatment (T0), 10 min after salbutamol inhalation (T1), and after aerosolized pentamidine treatment (T2). A total of 104 sessions were analyzed during a seven-month period. Despite beta-agonist administration, bronchospasm, defined as a reduction in FEV1 of 15 percent or greater following treatment, occurred in two patients (9 percent) after aerosolized pentamidine mesylate (−19.5 and −84.4 percent of reference value). However, bronchospasm was not detected in these patients or in 20 others after aerosolized pentamidine isethionate. The mean change in FEV1 was −51 ± 26 ml (−1.5 ± 0.7 percent of reference value) between T2 and T0.

We confirm that beta-agonist administration prevents bronchospasm. The absence of this adverse reaction in our patients is probably due to the nebulizer (Respirgard II; Marquest, Europe Medical, Bourg en Bresse, France), which generates smaller particles than the Fisons nebulizer (Fisons, New Bedford, Mass).

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REFERENCES

To the Editor:

We are pleased that other centers currently engaged in aerosolized pentamidine prophylaxis for Pneumocystis carinii pneumonia are also noting the development of bronchospasm with aerosolized pentamidine and the alleviation of symptoms with use of a beta-agonist. As a follow-up to the original randomized controlled trial, we have noted that if aerosolized salbutamol solution is administered concurrently with aerosolized pentamidine, the protection from bronchospasm is less than 100 percent.

We also agree with Doré et al that because of different delivery efficiency, particle size generation, and concentration of pentamidine solution, use of different nebulizers can have a significant effect on the incidence of bronchospasm.

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Subpleural Mononuclear Cell Infiltration in Nonspecific Pleuritis

To the Editor:

In an article that appeared in the November 1990 issue of Chest, Nagata et al reported that they could find few previous reports of the pathology of nonspecific pleuritis, particularly the subpleural mononuclear cell infiltration on which they concentrated. I would like to draw their attention to an account they appear to have overlooked. Although this report describes the infiltrate as focal, the illustrations of its being limited to the subpleural fibrofatty interface are very similar to those of Nagata et al. Indeed, this was the characteristic feature of all four of our cases of "cryptogenic bilateral fibrosing pleuritis." Thus, the incidence of this pattern of disease in idiopathic cases of nonspecific pleuritis can be raised from the two of four reported by Nagata et al to six of eight.

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REFERENCES

To the Editor:

We are grateful for Dr Corrin’s comment. As he pointed out, we interpret the pleural histology of the cases of Buchanan et al as that of subpleural mononuclear cell infiltration, which we reported in

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