erative day 10. The patient returned to the clinic weekly and at her last visit (8 weeks postsurgery) she was no longer oxygen-dependent at rest or with exercise. Her FEV₁ was 1,000 mL. Her PaCO₂ was 47 mm Hg and she felt significantly less dyspneic.

Although these results (Table 1) are from a single patient, it is clear that volume reduction surgery can work for patients with severe impairment, disability, and handicap. We have not categorically deprived patients the opportunity to participate in our program on the basis of a single physiologic parameter (either FEV₁ or PaCO₂). We believe it is unwise to do so until we understand their significance in context of the proposed surgery.

**REFERENCES**


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**Treat Patients Who Have Nocturnal Asthma With Anti-inflammatory Drugs First**

*To the Editor:*

We read with interest the article by D’Alonzo and colleagues (CHEST 1995; 107:406-12) on the effect of the terbutaline prodrug bambuterol in asthmatics. They compared a once-daily morning with an evening administration of one tablet of 20 mg, focusing on nocturnal bronchodilatation. They found that the two treatment regimens produced similar bronchodilatation at 4 AM, but preferred an evening dosage, which resulted in larger bronchodilatation at 7 AM. D’Alonzo et al, however, do not further discuss the role of β-agonists such as bambuterol in the pharmacotherapy of nocturnal asthma.

Several research groups have shown that the presence of nocturnal symptoms is related to the degree of airway responsiveness. Airway inflammatory processes are known to influence airway responsiveness. One might thus hypothesize that patients with asthma with more active airway inflammation are likely to have nocturnal symptoms. Indeed, cellular activation during the day, as measured in BAL fluid, is higher in asthmatics with than without nocturnal symptoms. In addition, inflammatory cell numbers in BAL fluid are increased at night in subjects with nocturnal asthma, but not in normal subjects. Therefore, it seems obvious to use anti-inflammatory drugs in the treatment of nocturnal asthma.

Previously, we have compared the effects of the inhaled corticosteroid budesonide (0.4 µg bid) and bambuterol (20 mg at 8 pm) on lung function and symptoms of nocturnal asthma in a placebo-controlled study. Bambuterol produced a significant nocturnal bronchodilatation and reduction of histamine airway responsiveness, and improvement of nocturnal wheeze, dyspnea, and quality of sleep. In comparison with bambuterol, however, treatment with budesonide resulted in a larger improvement in airway responsiveness at 4 AM (2.1 vs 0.8 doubling concentrations) and a greater beneficial effect on nocturnal dyspnea and wheeze.

In conclusion, information so far suggests that nocturnal symptoms of asthma are a reflection of more severe airway inflammation, and we advocate the use of anti-inflammatory drugs as first-line treatment. Long-acting inhaled or oral β-agonists should be considered as second-line treatment drugs.

**REFERENCES**


2 Martin RJ, Cicotto LC, Ballard RD. Factors related to the nocturnal worsening of asthma. Am Rev Respir Dis 1990; 141:33-8


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**Consider Anti-CMV Therapy**

*To the Editor:*

We are writing in response to the editorial by Hyland and colleagues (CHEST 1995; 107:595-97) regarding the influence of simultaneous cytomegalovirus (CMV) infection on the outcome of Pneumocystis carinii pneumonia (PCP). Pathology records at St Paul’s Hospital were computerized in 1992. We have therefore searched the computerized pathology database from July 1, 1992, to May 5, 1995, and have found that CMV inclusions were present in 5 of 208 patients with PCP (2.4%) who underwent bronchoscopy. CMV cultures were not routinely performed, as positive cultures were not thought to be of clinical significance in the absence of inclusion bodies. Of these five patients, only one died from the pneumonia without CMV treatment, having had corticosteroid treatment. This patient had very advanced disease, with Kaposi’s sarcoma, cryptospo-