stimulates the synthesis of SP. We have shown that the IV administration of levodopa significantly improved the swallowing reflex in patients with aspiration pneumonia, and that amantadine, a drug that acts by releasing dopamine from dopaminergic nerve terminals, could lower the risk of pneumonia to about 20% in patients with stroke. Therefore, amantadine may also be beneficial in preventing pneumonia in patients with stroke. This drug should be useful to reduce the risk of aspiration pneumonia in elderly patients with hypotension or airway hyperreactivity, for which the use of ACE inhibitors would be relatively risky.

Katsutoshi Nakayama, MD
Mutsuo Yamaya, MD
Hideidata Sasaki, MD, FCCP
Tohoku University School of Medicine
Sendai, Japan

Correspondence to: Hidetada Sasaki, MD, FCCP, Professor and Chairman, Department of Geriatric Medicine, Tohoku University School of Medicine, Aoba-ku, Sendai 980-8574, Japan

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Electrocautery in Endobronchial Therapy

To the Editor:

Dr. van Boxem et al (October 1999) should be complimented on their efforts to promote electrocautery and on their analysis of the relative cost of endobronchial treatments. My personal experience, in an institution that employed electrocautery, Nd-YAG laser, and brachytherapy, supports their conclusion that the cost of electrocautery is small in comparison to other endobronchial techniques. However, I believe that the reasons why electrocautery did not catch on, after our initial reports of its success, are more complicated than those which they proposed.

We began using electrocautery to treat endobronchial disease when no other endobronchial method was available. In the early 1980s, patients seeking treatment often were referred to us because of the lack of any other options for rapid treatment. Our initial experience grew simply from an apparent need and a lack of alternatives. At the same time, the Nd-YAG laser was being readied for release. Publications from academic institutions supported the effectiveness of the Nd-YAG laser. Our second article on electrocautery was published in 1985, just after the release of the Nd-YAG for clinical use, and our last report followed in 1985. Despite the benefits experienced, there was little enthusiasm for electrocautery. By that time, the laser decade had begun for the pulmonary community in the United States. After Nd-YAG lasers were released for clinical use, they rapidly became widely available. Courses for physicians on laser therapy began, as well as promotions by equipment manufacturers. Locally, the laser and its magic caught on like wildfire. Physicians and patients alike were enamored of the new technology. Referrals decreased as new lasers were installed. Some of the original authors of our work were never to use electrocautery again. Nationally, no academic institutions studied electrocautery.

The paradigm of endobronchial therapy for the next decade was set by the favorable view that physicians, patients, academic programs, and equipment manufacturers focused on the Nd-YAG laser technology. Electrocautery was left for another time, its role unconfirmed and uncertain. Maybe the time for serious assessment has come.

Robert G. Hooper, MD, FCCP
Scottsdale, AZ

To the Editor:

We thank Dr. Hooper, in his letter above, for the positive comments on our article, and the additions to the reasons why electrocautery did not become a popular technique in endobronchial therapy. We agree that the assessment of bronchoscopic electrocautery was seriously handicapped at that time by the popularity and magic of laser. Unfortunately, Dr. Hooper practiced during that particular period when Nd-YAG laser was so immensely popular, and the magic of laser was so difficult to resist, that the necessity for randomized trials and further in-depth investigations were not considered necessary.

I personally believe that even in the field of medicine there is some influence by current fashion from time to time, despite our persistent devotion to hard, objective data in randomized, placebo-controlled, clinical trials. Costs of treatment and equipment were perhaps not such critical issues in earlier days. Although we also agree that it is time for serious assessment of well-designed, randomized, phase III trials, performed by unprejudiced physicians, to give definite answers to the questions that remain, we currently lack any prospect of support to conduct such a trial. The tragedy is that while some health insurance companies in The Netherlands are severely cutting costs of managed care, specialists, they do cover expenses for alternative medicine.

I thank Dr. Hooper for his article back in 1985 in CHEST, which ultimately convinced me, in 1989, after observing that Nd-YAG laser was not frequently used in The Netherlands despite many centers having the facility, that other factors may be equally important for the clinical practice. While it does not always “sell” very well, I am lucky to practice in The Netherlands, where the attitude is “thrifty” and sober.

Thomas G. Sutedja, MD, FCCP
Academic Hospital Free University
Amsterdam, The Netherlands

Correspondence to: Thomas G. Sutedja, MD, FCCP, Academic
IV Epoprostenol in Gaucher’s Disease

To the Editor:

We read with interest the article by Bakst et al (October 1999), reporting the use of continuous IV epoprostenol in a patient with Gaucher’s disease with pulmonary hypertension. The effective use of this therapeutic modality is encouraging. Pulmonary hypertension in patients with Gaucher’s disease may be secondary to the effects of interstitial lung involvement or intercapillary plugging by Gaucher cells, or primary-like, in association with enzyme replacement therapy. Enzyme therapy may ameliorate pulmonary disease, including improvement in secondary pulmonary hypertension, as was initially observed in the cited case. Later reappearance of pulmonary hypertension may be associated with enzyme treatment.

We have a similar case of a 34-year-old woman who had undergone splenectomy, who presented with hypoxia and a tricuspid incompetence (TI) gradient of 27 mm Hg. After 3 years on enzyme therapy with improvement in hypoxemia, clubbing, and pulmonary function tests, the TI gradient dropped to 20 mm Hg (within the normal range). After 2 more years of treatment, she developed elevation of TI gradient to 37 mm Hg. We also documented another patient with Gaucher’s disease, who started enzyme therapy with a TI gradient within the normal range, which then rose above 30 mm Hg with treatment with the placental derivative, alglucerase, and then returned to the normal range after withdrawal. Challenge with the recombinant form, imiglucerase, resulted in elevation of the TI gradient above 30 mm Hg, and treatment withdrawal again resulted in return to the normal range.

Continuous use of enzyme replacement (and in very high dosages) in the case by Bakst et al1 could have been the cause of the reemergence of pulmonary hypertension, and enzyme withdrawal might be considered to wean her from lifelong dependence on epoprostenol. On the other hand, the success of epoprostenol may be of greater importance to patients who have other life-threatening features of Gaucher’s disease and who cannot, therefore, terminate enzyme therapy. We applaud the pioneering efforts of Bakst et al, as like them, we are concerned with the increased number of patients with Gaucher’s disease who develop pulmonary hypertension. In addition, we concur that echocardiography should be included in routine follow-up of all Gaucher patients, treated and untreated, despite the fact that, in recent guidelines for diagnosis and monitoring by the International Collaborative Gaucher Group registry, this procedure was unfortunately omitted.

Deborah Elstein, PhD
Ari Zimran, MD
Shaare Zedek Medical Center
Jerusalem, Israel

Correspondence to: Ari Zimran, MD, Shaare Zedek Medical Center, P. O. Box 3235, Jerusalem 91031; Israel; e-mail: zimran@md2.huji.ac.il

Corticosteroid Therapy in Acute Asthma

To the Editor:

We read with interest the meta-analysis by Rodrigo and Rodrigo (August 1999) evaluating 16 randomized control trials on the effect of steroids in acute asthma. Their conclusion, based on effect size (ES) estimates of pulmonary function, was that systemic steroid effects do not seem to manifest themselves until ≥ 6 h after use. This is consistent with what is known about the time course of steroids in asthma, including our evaluation of IV steroid effects at 1 h. We recently finished evaluating the effect of IV steroids in acute asthmatic patients over 2 h using a randomized, double-blind, placebo-controlled trial, and noted a significant increase in peak flow measurements in the steroid group over 2 h. The ES estimate of peak flow was small, however, at 0.32 at the 2-h time point, when adjusting for baseline peak flow rates. (It was unclear from their methods section how the 95% confidence intervals were calculated, i.e., what standard error was used, so we could not include them here.) This ES of 0.32, assuming normality, suggests that the average asthmatic patient’s peak flow in the steroid group would be > 62% of the peak flows of asthmatic patients in the control group. Assuming this a study of high quality as graded by Rodrigo and Rodrigo, this result probably would not have changed their conclusion due to the small ES estimates for the other studies using systemic steroids under 6 h. It is unclear to us why our study showed a slight

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To the Editor:

We appreciate the comments by Drs. Elstein and Zimran. In light of their work suggesting a role for enzyme replacement in the pathogenesis of pulmonary hypertension in Gaucher’s disease, we did indeed discontinue enzyme therapy; however, during this interval off enzyme replacement, our patient experienced worsening hepatomegaly, which subsided upon reinstitution of replacement therapy.

Lewis J. Rubin, MD, FCCP
UCSD Healthcare
San Diego, CA

Correspondence to: Lewis J. Rubin, MD, FCCP, Professor of Medicine, Division of Pulmonary/Critical Care Medicine, UCSD Healthcare, 200 West Arbor Dr, San Diego, CA 92103-8372, e-mail: ljrubin@ucsd.edu

Chest 117;6/JUNE, 2000 1821