reversing oxygen desaturation after sputum induction seems, therefore, to be in contrast with a mechanism involving mediators release.

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Angiotensin-Converting Enzyme Inhibitors, Angiotensin II Receptor Antagonists, and Symptomless Dysphagia

To the Editor:

Symptomless dysphagia is one of the main causes of pneumonia in elderly people with stroke, and swallowing or the cough reflex can prevent aspiration pneumonia. The article by Nakayama and colleagues (May 1998), concerning angiotensin-converting enzyme (ACE) inhibitors and the swallowing reflex, was a fascinating and timely study. We previously reported that ACE inhibitors may cure symptomless dysphagia in hypertensive patients with stroke. ACE inhibitors and angiotensin II receptor antagonists (Ang II) are very important drugs for hypertensive patients. We investigated the prevention of symptomless dysphagia and treatment with different antihypertensive drugs in hypertensive patients with stroke. We investigated whether a correlation existed between patients with symptomless dysphagia with stroke and the elimination of low serum substance P concentration by ACE inhibitors and Ang II. We excluded the immunocompromised patients.

The subjects were 33 patients with hypertension, symptomless dysphagia, and history of stroke. They were divided into group A (32 patients; 13 men and 19 women) and group B (21 patients; 10 men and 11 women). We obtained informed consent from the patients or their families.

To determine the occurrence of symptomless dysphagia, we gave 1 mL technetium tin colloid (99mTc) to patients in groups A and B during sleep via a nasal catheter. At 9:00 AM the next day, we checked for symptomless dysphagia by imaging.

We gave all patients in group A an ACE inhibitor (imidapril hydrochloride), 5 to 10 mg qd orally, and all patients in group B received Ang II (losartan potassium), 50 to 100 mg qd orally. We measured serum substance P before and 12 weeks after administration. The mean serum substance P before drug administration was 26.5 pg/mL in group A and 26.36 pg/mL in group B. After 12 weeks, symptomless dysphagia improved in 23 of 32 patients in group A. In these 23 patients, the mean serum substance P was 82.91 pg/mL. In six patients, symptomless dysphagia did not improve (mean serum substance P, 50.62 pg/mL) and in four of them, serum substance P was not increased (mean serum substance P, 46.12 pg/mL). Imidapril hydrochloride was stopped in the remaining patients because of excessive cough (mean serum substance P, 109.83 pg/mL). On the other hand, in all 21 patients in group B, symptomless dysphagia did not improve, and in all of them, serum substance P did not increase (mean serum substance P, 30.49 pg/mL) after 12 weeks (p < 0.0001).

We concluded that ACE inhibitors have advantage over Ang II in prevention of symptomless dysphagia in patients with stroke.

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To the Editor:

We thank Dr. Arai et al for their interesting comments on our study. They report that the administration of angiotensin-converting enzyme (ACE) inhibitor could increase serum substance P (SP) and could improve the symptomless dysphagia in patients with hypertension and previous stroke, but that angiotensin II receptor antagonists (Ang II) could not. ACE inhibitors inhibit not only the activation of angiotensin II, but also the degradation of SP and bradykinin. We have shown, in a 2-year study, that ACE inhibitors could upregulate impaired swallowing reflex, and that they could also reduce the risk of pneumonia by about one third, compared with results of other antihypertensive drugs used in treatment of patients with hypertension and previous stroke. However, the effect of Ang II on the swallowing reflex has not yet been examined. Dr. Arai’s experience clearly showed that the effect of ACE inhibitors on improvement of symptomless dysphagia should be independent from the angiotensin II pathway. We agree that ACE inhibitors have an advantage over Ang II in preventing symptomless dysphagia in patients with stroke.

Despite the important antihypertensive effect, ACE inhibitors also cause a side effect of excessive cough in 5 to 10% of subjects, as shown in the comment by Arai et al. Therefore, ACE inhibitors would not be useful for patients who have developed hypotension or other side effects as a result of these drugs. Another way to increase SP is through the administration of dopamine, which
followed in 1988.3,4 Despite the benefits experienced, there was little enthusiasm for electrocautery. By that time, the laser became widely available. Courses for physicians on laser therapy were being readily offered to them because of the lack of any other options for rapid treatment. 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.2 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 realized 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 1988.3,4 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.

To the Editor:

Dr. van Boxem et al (October 1999)1 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.2 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 realized 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 1988.3,4 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.

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To the Editor:

We thank Dr. Hooper, in his letter above, for the positive comments on our article,1 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 by specialists, they do cover expenses for alternative medicine.

I thank Dr. Hooper for his article back in 1985 in CHEST,2 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 “thriftly” and sober.

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