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. Please include a cover letter with a complete list of authors (including full first and last names and highest degree), corresponding author’s address, phone number, fax number, and e-mail address (if applicable). Specific permission to publish should be cited in the cover letter or appended as a postscript. CHEST reserves the right to edit letters for length and clarity.

**Communications to the editor**

_Treating Refractory Cough With Aerosols of Mepivacaine_

To the Editor:

I read with interest the article, "Nebulized Lidocaine in the Treatment of Refractory Cough," by Dr. Trochtenberg (CHEST 105:1592-93).

Since 1975, I have treated with aerosols of either lidocaine hydrochloride or mepivacaine hydrochloride (1 to 2%) propelled by a compressed air device a series of more than 500 patients with dry cough, which had preexisted for periods that varied from several weeks to several months, or even years. Previous conventional medical treatments of these patients had been unsuccessful; their ages ranged from 18 months to 90 years. Patients received immediate relief and tolerated the aerosol treatments well. No significant side effects occurred, and the chronic cough was eradicated after 10 to 15 treatment sessions, although a minority of patients needed more treatments and concomitant therapy with macrolide antibiotics and budesonide or ipratropium inhalers.

Most of these patients were suffering from infectious viral tracheitis,1 or tracheitis due to _Mycoplasma pneumoniae_, and the cough was the residual symptom that resisted conventional medical treatment. The infection alters, or desquamates, the epithelial cells of the trachea and leaves the vagus nerve endings exposed. The dry cough is part of a vicious cycle initiated by irritation or stimulation of the nerve endings that form the efferent component of the reflex arc that is perpetuated, in turn, by further epithelial irritation provoked by the violent action of coughing. Lidocaine or mepivacaine serve equally well to anesthetize the vagus nerve endings to arrest the cough and permit the reepithelialization of the mucosa.

We have classified three types of patients with persistent dry cough: (1) those without previous respiratory disease, (2) those with obstructive disease, and (3) those with restrictive disease.

Refractory dry cough of patients without previous respiratory problems is frequently found in children and at times can lead to emesis; it can also occur in adolescents and adults. Auscultation and radiologic examination of the thorax in these cases usually show normal findings.

Dry cough complicated by tracheitis can occur in patients with obstructive or restrictive diseases that have been stabilized until the development of the cough. In both these conditions, cough is usually the first sign of relapse and often is the last sign to disappear when a new stable phase is established.

Two special indications for aerosol treatment are cough syncope triggered by frequent violent coughing episodes and preoperative preparation for abdominal surgery in patients with cough, to prevent suture failure, wound opening, and postoperative hernia.

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**REFERENCES**


To the Editor:

Dr. Almansa-Pastor’s letter confirms both the long-term safety and efficacy of nebulized lidocaine hydrochloride for refractory cough. Nebulized lidocaine provides rapid relief (often within 5 min) and a sustained duration of action (usually 6 to 8 h). It mixes with albuterol and/or ipratropium, allowing combination therapy with a single nebulizer session. I have not had the opportunity of using lidocaine in children, but I suspect a reduction in dose would be wise to avoid toxic serum levels of the drug.

Dr. Almansa-Pastor implicates the vagus nerve in the pathogenesis of chronic cough, particularly when caused by _Mycoplasma pneumoniae_ tracheitis. It seems reasonable to invoke a vagal trigger in the patient described in my article, as his underlying disease process was severe erosive gastroesophagitis. Unfortunately, my patient (and some of Dr. Almansa-Pastor’s) failed to respond to either atropine or ipratropium. Furthermore, research done on sheep with tracheal edema and airway hyperresponsiveness (from either ARDS or elevated vascular pressures) failed to display a response to atropine.1-3 Non-sympathetic-non-cholinergic neurohumoral pathways may also play a role in airway irritation and cough. Clearly more work is needed in this area to elucidate the relationship among vagal activation, tracheal injury, and cough.

Dr. Almansa-Pastor describes broad classes of patients with chronic cough: those with airway obstruction, restrictive defects, or no previous disease (ie, idiopathic). I have found it useful to think of patients with chronic cough as having either primary airway inflammation (whether obstructive or restrictive disease) or an extrapulmonary trigger. In the patients with airway inflammation, aggressive treatment of the underlying pulmonary disease is required. The cough is often an asthma equivalent, and it may respond to inhaled bronchodilators, steroids, cromolyn, and nedocromil. Systemic steroids or cytotoxic drugs may be required in severe disease to halt the inflammatory process and to allow a cessation of nebulized lidocaine. Extrapulmonary triggers are typically difficult to treat, particularly if the trigger is an anatomic lesion (such as mediastinal fibrosis, neoplasm, or stroke). Peptic ulcer disease or gastroesophageal reflux may require surgical intervention. These