should be concerned because the cost can be formidable. Interventional radiology routinely uses the technology available be it ultrasound for thoracentesis, or computerized tomography for chest tube placement or transthoracic needle aspiration biopsy of the lung. There is little documentation that these technologic devices improve outcomes. Expenditures for diagnostic x-ray studies indeed are one of the fastest rising components of healthcare services.

Patients should be concerned because these three procedures have a potential for significant complications. Few radiologists, in my experience, are proficient in intubation, cardiopulmonary resuscitation, or chest tube insertions. Moreover, these procedures become an isolated event in the management of patients.

Clinicians should be wary because official radiology reports can encourage self-referral and trap the attending physician. Clinicians frequently "get run over by the bandwagon."9

The attitude that "imaging guided catheter techniques provide here-tofore unsurpassed precision and accuracy in performance of these procedures"10 should be tempered. I fear that interventional radiology is yet another example of fractionated healthcare and occasionally a detriment to the best interest of the patient. Interventional radiology will soon begin to face the rigors of technologic assessment, hospital credentialing committees, and peer review organizations. The concept of informed consent and other legal ramifications that radiologists have avoided in the past will need to be addressed. I have reservations that these practitioners "flounder along in an aimless fashion, never able to gain any accurate conception of disease . . . hitting now the malady and again the patient, he himself not knowing which." Their intrusion into the clinical arena prompts this caveat emptor.

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Laceration of the Cuff of an Endotracheal Tube During Percutaneous Dilatational Tracheostomy

To the Editor:

The technique of percutaneous dilatational tracheostomy described by Ciaglia et al.1 appears to be gaining popularity and has been the subject of several recent reports.2-4 It is well recognized that the cuff of the endotracheal tube is at risk of puncture during first placement of the guidewire needle, and it is recommended that the tube be withdrawn to a position immediately below the cords prior to insertion of the needle.

We wish to report a case in which the cuff was, not simply ruptured, but unwittingly torn such that two separate fragments were left in the trachea during an otherwise uneventful tracheostomy (Fig 1). The defect in the cuff was discovered only on withdrawal of the endotracheal tube, and bronchoscopy was needed to recover the fragments.

Figure 1. Lacerated endotracheal tube cuff with the two fragments.

We now remove the endotracheal tube from the trachea before the procedure begins, leaving only the tip between the cords, using the inflated cuff as an obturator above the cords in order to ventilate the lungs. This works satisfactorily and removes the cuff from risk of puncture and laceration.

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Bronchial Responsiveness to Methacholine in Insulin-Dependent Diabetic Patients With Autonomic Neuropathy

To the Editor:

The bronchomotor tone is determined by the synergism-antagonism of several systems: the parasympathetic system, the sympathetic system, and a third nonadrenergic-noncholinergic system. We can suppose that in diabetic patients with autonomic diabetic neuropathy demonstrated at the cardiovascular level, compromise of the vagal tone can be present. The data in the currently available literature are conflicting: a reduced bronchial response to hyperventilation of cold air and inhalation of methacholine (MTH) have been reported,5,6 but so has an increased reactivity to histamine.7 These discrepancies might be due to confounding factors, such as smoking habits and genetic differences of the studied individuals related to the type of diabetes. To evaluate the presence and type of alterations in the autonomic nervous system of the airways in diabetic disease, we studied the reactivity to a bronchial stimulating test with MTH in 20 insulin-dependent diabetic patients (aged 38 ± 14 years) without personal and/or familiar anamnesis of bronchial hyperreactivity. Patients were subdi-
Table 1—Baseline Respiratory Values and Response to MTH Inhalation*

<table>
<thead>
<tr>
<th></th>
<th>Diabetics With Neuropathy</th>
<th>Diabetics Without Neuropathy</th>
<th>Normal Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEF L/s</td>
<td>6.86 ± 3.25</td>
<td>9.14 ± 10.60</td>
<td>6.6 ± 5.30</td>
</tr>
<tr>
<td>PEF Δ%</td>
<td>1.56</td>
<td>8.2 ± 11.40</td>
<td>6.1 ± 3.20</td>
</tr>
<tr>
<td>FEF25% L/s</td>
<td>115.00 ± 32.92</td>
<td>74.6 ± 3.41</td>
<td>74.2 ± 2.15</td>
</tr>
<tr>
<td>FEF50% L/s</td>
<td>4.33 ± 1.56</td>
<td>4.98 ± 0.99</td>
<td>4.4 ± 2.15</td>
</tr>
<tr>
<td>FEF75% L/s</td>
<td>1.56 ± 0.47</td>
<td>2.20 ± 0.44</td>
<td>1.8 ± 0.39</td>
</tr>
<tr>
<td>MEMF L/s</td>
<td>3.63 ± 1.19</td>
<td>4.48 ± 2.80</td>
<td>3.6 ± 1.18</td>
</tr>
<tr>
<td>MEMF Δ%</td>
<td>77.66 ± 9.07</td>
<td>69.60 ± 8.16</td>
<td>67.0 ± 3.27</td>
</tr>
</tbody>
</table>

*Response to MTH inhalation is expressed as final decrease (Δ%), calculated as final value minus baseline value.

provided into two groups of 10 each on the basis of the presence or absence of autonomic neuropathy evaluated according to the classic tests proposed by Ewing and Clarke.4 We administered the same test to a group of 20 nondiabetic control subjects. On the basis of our data we assumed that a dose of MTH greater than 120 mg when inhaled by normal subjects always induces a statistically significant reduction (>25 percent) of the peak expiratory flow (PEF), the maximum forced mean expiration (MEMF), and the forced expiratory fraction at 25 percent, 50 percent, and 75 percent (FEF25%, FEF50%, and FEF75%, respectively), and indexes of involvement of the medium and small airways; on the other hand, one does not observe the involvement of the large airways (FEV1, and forced vital capacity).

The basal values of PEF, MEMF, and FEF25%, FEF50%, and FEF75% were not significantly different in the three studied groups (Table 1). The inhalation of MTH caused a decrease in those measurements by more than 25 percent in diabetic patients without autonomic neuropathy and in the control subjects at doses between 20 and 120 mg. In diabetic patients with autonomic neuropathy, we did not observe any significant alteration of the involvement indexes of medium and small airways when 120 mg was inhaled.

Our results, even though preliminary, show that the vagal tone of airways can be reduced in the presence of diabetic autonomic neuropathy. Efferent vagal fibers that innervate the bronchial tree could be involved by diabetic neuropathy, contributing to the fall of the vagal tone.

Actinobacillus actinomycetemcomitans Pneumonia With Possible Septic Embolization

To the Editor:

We read with interest the report by Yuan et al.1 which appeared in the May 1992 issue of Chest. They described a case of Actinobacillus actinomycetemcomitans pneumonia with chest wall involvement. We report a similar case with possible septic embolization.

A 56-year-old male smoker presented with 6 months of worsening nonproductive cough, left pleuritic chest pain, and a 100-lb (45-kg) weight loss. Chest radiography revealed a left upper lobe mass with postobstructive atelectasis. Outpatient bronchoscopy and percutaneous lung biopsy specimens were nondiagnostic. The patient was hospitalized after the onset of fever and new erythematous papular lesions on the left palm, and the first, third, fourth, and fifth fingers. These lesions soon became pustular (Fig 1). Physical examination was unremarkable except for marked periodontitis and a short systolic murmur. Blood cultures held for 10 days were negative.

At thoracotomy, the left upper lobe was noted to be necrotic, constricted, and adherent to the mediastinum. Histopathology revealed chronic nongranulomatous inflammation. Cultures from both the lung mass and a hand pustule grew moderate amounts of A actinomycetemcomitans. Smaller amounts of Actinomyces meyeri and Fusobacterium species were also recovered. Surface echocardiography performed to look for a source of embolization showed no vegetations. A left subclavian arteriogram was normal. The patient responded to 6 weeks of intravenous antibiotic therapy and is doing well 2 years later.

Hand infection due to A actinomycetemcomitans is rare.2-3 In a review by Kaplan et al.,4 clinically apparent embolization to the limbs in the absence of endocarditis was not reported. Our evaluation did not suggest a diagnosis of endocarditis in this patient. We hypothesize that he had transient polymicrobial septic embolization to the hand from the area of chest infection. We would appreciate other clinicians’ insightful comments.

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REFERENCES


FIGURE 1. Pustular lesions on left palm and first, third, fourth, and fifth fingers.

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