hygiene, bypassing the oropharynx, is an inherent limitation in the use of silver-coated tubes and subglottic suctioning devices. Due to the absence of any significant survival benefit from the use of subglottic drainage and silver-coated tubes, we continue to prevent pneumonia in ventilated patients with SDD, which is associated with a significant 8% reduction in mortality.

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Financial/nonfinancial disclosures: The authors have reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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DOI: 10.1378/chest.09-0113

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Exercise-Induced Bronchospasm

Coding and Billing for Physician Services

To the Editor:

I read with interest the article by Pohlig 1 in a recent issue of CHEST (January 2009) outlining coding and billing for exercise-induced bronchospasm (EIB). I was concerned about a statement that the author made concerning diagnostic evaluation for EIB. The author states that exercise testing and/or eucapnic voluntary hyperventilation (EHV) testing have significant logistical drawbacks and therefore, methacholine challenge is the “preferred” method for diagnosis of EIB. In the article, 1 the author writes: “Therefore, methacholine or histamine challenges are more sensitive and preferred for the average individual.”

I strongly disagree with the author’s statement. A 2007 workgroup report on EIB from the American Academy of Allergy, Asthma & Immunology 2 stated that methacholine challenge is a “suboptimal” test for the documentation of EIB. In addition, one of the studies by Rundell et al 3 that the author uses to support her statement in fact states that direct challenges such as methacholine are less sensitive than physical challenges such as exercise or EHV testing. That same article by Rundell et al 3 was comparing EHV testing to field-exercise testing in elite cold weather athletes and did not support the author’s statement about methacholine challenge testing. It also does not apply to “average individuals” as the author writes, because the study by Rundell et al 3 was a study of elite athletes. Many authors have stated 4,5 that methacholine challenge is not sensitive and specific to the bronchoconstriction associated with exercise, and that in reality it is a less preferred test for the documentation of EIB than EHV testing or exercise.

The author also states that EHV testing may “overdiagnose” EIB; however, no evidence is provided to support that statement. It is true that many of the prevalence studies related to EIB have included populations of athletes and that EHV testing might be best suited for that population. However, I am not aware of any data to support the author’s statement about overdiagnosis, as one would need a “gold standard” diagnostic test to which one could compare EHV, and that test is not available at this time.

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DOI: 10.1378/chest.09-0113

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Chronic Necrotizing Pulmonary Aspergillosis or Invasive Pulmonary Aspergillosis

To the Editor:

We read with interest the article by Sakkour et al 1 in CHEST (February 2008), who reported the case of a 56-year-old woman with COPD and multiple pulmonary nodules. We congratulate the authors on finding pulmonary aspergillosis associated with
marijuana smoking in a patient without clinical evidence of an immune deficiency. However, two aspects of this report require further discussion.

First, instead of invasive pulmonary aspergillosis (IPA), we believe the precise diagnosis should be chronic necrotizing pulmonary aspergillosis (CNPA). To start with, the patient does not show any clinical symptoms of IPA, nor are her COPD symptoms getting any more severe. The occurrence of IPA in COPD patients will intensify symptoms like shortness of breath. Next, the CT scan of her chest shows no evidence that her left upper lobe nodule is progressing any further. This suggests that the nodule is of a slow progressive nature.

In addition, the findings of the two CT scan-guided biopsies were both nondiagnostic and revealed predominantly fibrous tissue. In clinical practices, the diagnosis of CNPA commonly stands on the presence of multiple cultures that are positive for Aspergillus organisms, chest radiographs with abnormal findings, and test findings for bronchoscopy biopsy specimens that are consistent with tissue invasion.6

Last, the authors’ diagnosis rests mainly on the results of a biopsy performed with video-assisted thoracoscopic surgery, showing casing granulomas and granulomatous pleuritis, with evidence of Aspergillus species and thrombosed vessels in the center of one of the nodules. However, Yousem3 has also reported 4 of 10 persons with conditions resembling necrotizing granulomatous pneumonia centered around a central zone of infantile necrosis of the parenchyma, resulting from angoeinvasive Aspergillus. This is incredibly similar to the histopathology reported by Sakkour et al.1

In conclusion, in contrast to IPA, CNPA is a chronic process that progresses slowly over months to years. IPA, on the other hand, is a severe and commonly fatal disease that is seen in immunocompromised patients. Therefore, we incline to believe that the diagnosis in this case should be CNPA.

Second, we totally agree with the authors in regarding marijuana smoking as the main factor for pulmonary aspergillosis in this patient. Nonetheless, we believe that the fluticasone therapy the patient received (since the report did not specify the dosage of fluticasone, we can only take the recommended dosage of fluticasone for a severe pulmonary patient into consideration) is also one of the key factors for the occurrence of pulmonary aspergillosis in this patient. Reports4,5 have shown that therapy with not only oral steroids, but also with inhaled steroids might promote IPA or CNPA in COPD patients.

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DOI: 10.1378/chest.09-0137

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Does Biofilm Formation Play a Role in Ventilator-Associated Tracheobronchitis?

To The Editor:

I enjoyed reading “Ventilator-Associated Tracheobronchitis” by Craven et al4 in a recent issue of CHEST (February 2009). It may be useful to determine whether this condition includes biofilm formation. The authors mentioned contiguous biofilm sites including the endotracheal tube and oral cavity. Dental plaque is a form of biofilm that supports the growth of pulmonary pathogens and is a probable source of pneumonia. Oral hygiene decreases rates of ventilator-associated pneumonia in some groups.2

Pseudomonas and Staphylococcus aureus are biofilm formers.2,3 Biofilm colonies anchor to mucosal surfaces or foreign bodies and are composed of layers of slow-growing bacteria embedded in the glyocalyx (exopolysaccarides). The close proximity of bacteria facilitates chemical quorum sensing when the colony achieves high density, triggering the production of virulence factors and/or launching free-living bacteria to infect other sites such as the alveolae. Glyocalyx interferes with antibiotic penetration, and slow growth makes bacteria resistant to growth-dependent antibiotic killing. Killing bacteria in biofilm requires antibiotic concentrations 10 to 1,000 times that needed to kill free-living bacteria. Conceivably, topical or aerosolized antibiotics could achieve local concentrations high enough to suppress biofilm formation and avoid exposing bacteria to sub-minimum inhibitory antibiotic concentrations if the biofilm was not fully mature. Other novel therapies that can be directed toward the formation of biofilms or are capable of breaking the chemical bonds of the biofilms may become available.3,5

The exopolysaccarides in biofilm cannot be visualized by conventional light microscopy.4 Detection requires scanning electron or laser microscopy. Fluorescent in situ hybridization is required to identify specific bacteria.3 These techniques have demonstrated the presence of biofilm and clarified the pathophysiology of ear, nose, and throat infections, including chronic sinusitis, otitis media with effusion, and adenotonsillitis.3 Perhaps it is time to apply the same techniques to determine whether biofilm formation is part of the pathophysiology of ventilator-associated tracheobronchitis.

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