Minimally Invasive Closure of Bronchopleural Fistulas

To the Editor:

We would like to congratulate Fruchter et al.1 for their ingenious, effective, and minimally invasive way of treating the very challenging condition of bronchopleural fistulas as reported in a recent issue of CHEST (March 2011). However, because we were cited in their work, we must make a correction. The misunderstanding is that our first publication was a case report of our first patient (53-year-old man with a right upper lobectomy) using this method. Later, Scordamaglio et al.3 published a review of our experience endoscopically treating airway fistulas (two bronchopleural and one tracheoesophageal), which included this same patient. Since then, five more fistulas in four other patients have been treated, making a total of seven fistulas treated in six patients (one patient with two fistulas), with five of them already closed. Although the technique used is similar to that in Fruchter et al.,1 there are small differences we believe are worth noting.

Unlike Fruchter et al.,1 we used the Occlutech Figulla ASD Occluder N (Occlutech International AB; Helsingborg, Sweden) because this device has a design that makes it more malleable. Fruchter et al.1 stated that “bronchography was performed to outline the anatomy of the fistula, in particular its length.” Because we treated only total fistulas, the “fistula length” was not a concern in our cases because we could see the end of the stump with the bronchoscope. In our cases, the diameter of the fistula was the most relevant measure for choosing occluder size. We estimated diameter by insufflating a ballooned catheter or an endobronchial blocker inside the fistula. Consequently, we have not used fluoroscopy or bronchography.

Finally, we agree with the authors’ statement that the procedure can be carried out in a bronchoscopy suite in patients under conscious sedation, which is the most remarkable distinction between the classic surgical procedure of treating this condition and this new minimally invasive method. Once again, we congratulate the authors for their valuable contribution.

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Pulmonary Graft Dysfunction and Elevated Pulmonary Pressures

To the Editor:

In a recent study, Fang and colleagues2 brought up some interesting points concerning elevated pulmonary pressures (ie, mean pulmonary arterial pressure [mPAP]) and primary graft dysfunction (PGD). In their study, they propose that elevated pulmonary pressures are associated with the development of PGD.

We congratulate the authors on their hard work and success. However, we do have some concerns:

- The authors do not clearly define their cutoff for elevated pulmonary pressure. According to the American College of Chest Physicians,3 most experts generally accept elevated pulmonary pressures to be an mPAP > 25 mm Hg with a pulmonary capillary or left atrial pressure < 15 mm Hg. The authors have a wide range of elevated pulmonary pressures for both the PGD and the non-PGD groups (35.5 ± 16.3 mm Hg and 29.6 ± 11.5 mm Hg, respectively). By the current American College of Chest Physicians

References