we stressed that misattribution and panic were probably the prime reason for presentation to the emergency department. The role of asthma is more complex. I agree that we have no data about the incidence of asthma in a nonhyperventilating group of patients presenting in the same way, and I am sure that a control group would have strengthened our findings, but I doubt that the incidence of asthma in any control group would be as high as we found in these patients. Asthma is becoming increasingly difficult to define and is now recognized that standard measures of airflow obstruction such as peak flow can be normal in the presence of considerable mucosal edema and inflammation. Although we cannot prove that asthma directly contributed to the hyperventilation in our patients, there is a considerable body of literature showing marked hypocapnia in mild and moderate asthmatics, and we have long argued that it is the very mild and often previously undiagnosed asthmatics who suffer from the most profound hyperventilation. The reasons for this remain to be determined. I agree with Dr. Singer about the usefulness of attempting to reproduce symptoms by forced overbreathing, and this was discussed at length in my review.

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Improving Percutaneous Dilational Tracheostomy

To the Editor:

This is in response to the letter by Dr. Sandifer that appeared in CHEST (January 1997). He pointed out that in the technique of percutaneous dilational tracheostomy, it was an advantage to have a tracheostomy tube with an oblique tip, as this would make entry into the trachea easier and less traumatic. This, of course, is true and would be an advantage. However, as Dr. van Heurn pointed out in his response to the letter, in the literature, the incidence of tracheal stenosis after percutaneous dilational tracheostomy is relatively low. The percentage of tracheal stenosis reported by van Heurn et al is well below the reported percentages in open, conventional tracheostomies, and is operator-dependent. I personally, have done 117 percutaneous dilational tracheostomies and in over 90% of them have used a single cannula, flexible Shiley tracheostomy tube with an internal diameter of 7 or 8 mm. In these cases, overdistention was usually achieved using a 36F dilator. This was sufficient and not too traumatic, since the tracheostomy tube, although it did not have an oblique tip, was flexible, not made of rigid plastic. None of the patients who lived long enough for long-term follow-up developed tracheal stenosis. Unfortunately, our film demonstrating the technique was an exception; we used a rigid, plastic Shiley tracheostomy tube. Percutaneous dilational tracheostomy continues to be improved and made easier and safer. The first improvement was endoscopic visualization of the procedure. The oblique tracheostomy tube tip is also an improvement and parallels the oblique tip of the standard endotracheal tube. I have no doubt that there are other improvements to be made.

Van Heurn and his colleagues are to be congratulated on their excellent study of the late complications of percutaneous dilational tracheostomy.

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Fiberoptic Bronchoscopy After Recent Acute Myocardial Infarction

Stress for the Heart?

To the Editor:

We read with interest the retrospective analysis of Dweik and colleagues (September, 1996), in which the authors state that bronchoscopy is safe after recent acute myocardial infarction (AMI). In 20 patients reviewed between 1986 and 1994, who underwent flexible fiberoptic bronchoscopy (FFB) while hospitalized for AMI, they found no adverse event. In a recent study, we had investigated the influence of FFB on hemodynamics, right ventricular function, and plasma atrial natriuretic peptide (ANP) concentration in 14 critically ill, mechanically ventilated patients who were deeply sedated and paralyzed. We observed a prompt and significant increase in pulmonary artery pressure and cardiac index (median [range]: 4.25 [3.1-5.7] to 4.85 [4.3-6.9] L/min/m²; p<0.01) within 3 min during FFB. Right ventricular end-diastolic volume increased, as well as stroke volume and stroke work index, from 8.2 [4.7-15.7] to 13.3 [2.4-41.3] g/m²/m²; p<0.01. Plasma ANP concentration rose significantly during FFB. We concluded that FFB induces a "hyperdynamic situation" as well as changes in ventricular function in reaction to acute pulmonary hypertension.

In summary, we hypothesize from our results that the cardiac system mobilizes stroke work to prevent FFB-induced stress. We therefore believe that FFB might induce disadvantageous reactions due to an increase of myocardial oxygen consumption in patients hospitalized after AMI. We wish to emphasize the statement of the accompanying editorial comment that the indications to bronchoscopy should be extremely rigorous in these patients; the maneuver should be accompanied by extensive monitoring of hemodynamic parameters.

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