observed in our study was unnecessary. In retrospect, we agree. However, this was the practice at our institution during the period of study (from 2000 to 2001), and was consistent with the manufacturer’s guidelines at that time for the use of enoxaparin in patients with an ACS.

Drs. Spinler and Dobesh question whether incorrect enoxaparin dosing had an effect on bleeding. Despite one in five patients in our study receiving an enoxaparin dose that was > 10% or < 10% of the recommended 1 mg/kg dose, major bleeding did not occur in any of the 17 patients who received a dose > 1 mg/kg. Consequently, enoxaparin dose was not included as a variable in the logistic regression model that assessed determinants of bleeding. The authors also state that our criteria for major bleeding were more liberal than those used in the ESSENCE and TIMI 11B trials, which may have accounted for the higher rate of bleeding in our study (4%) than that observed in the clinical trials (1 to 2%). All patients who had major bleeding had a decrease in hemoglobin > 30 g/L, which would qualify as a major bleed, irrespective of the criteria used. Indeed, there are no accepted standard criteria for major bleeding. Furthermore, we never stated the intent to formally compare rates of bleeding across studies.

Drs. Spinler and Dobesh question the lack of a comparator anticoagulant strategy. The prespecified objectives of our study were to assess dosing practices in patients with an ACS who received enoxaparin, not to compare treatment practices with different anticoagulants. The authors also question our use of a creatinine clearance cut-off of < 25 mL/min in our description of the study population. However, this cut-off was used only to describe patients, whereas in the regression analyses, creatinine clearance was a continuous variable when assessed as a predictor of bleeding. Furthermore, the creatinine clearance cut-off of < 30 mL/min, which the authors claim is widely recognized to adjust low-molecular-weight heparin (LMWH) dosing, has never been validated as the cut-point at which LMWH dosing should be adjusted because of concerns of excessive anticoagulation due to impaired renal clearance of LMWH.

Drs. Spinler and Dobesh question why cardiac catheterization or bypass surgery were not included as predictors of bleeding. As stated in our article, our institution does not support cardiac catheterization; therefore, these procedures could not have been assessed as predictors of bleeding. The authors also state there is a “disconnect” between patient care in our study and that from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml). Correspondence to: James Douketis, MD, St. Joseph’s Hospital, Room F-541, 50 Charlton Ave East, Hamilton, ON L8N 4A6, Canada; e-mail: jdoeket@nuhealth.ca

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Assessment of Ventilation During the Performance of Elective Endoscopic-Guided Percutaneous Tracheostomy

To the Editor:

We read with great interest the article by Ferraro et al (July 2004)1 assessing ventilation during elective endoscopic-guided percutaneous tracheostomy, and we have some questions and comments. We agree that bronchoscopic guidance has increased the safety of the procedure and may prevent complications.2,3 We do not agree, however, that the presence of the bronchoscope inside the lumen of the endotracheal tube and trachea produce clinically important airway obstruction leading to hypoventilation, hypercarbia, and hypoxemia.

First, the period during which the bronchoscope is present (ie, from the moment of puncture until dilatation), is relatively short, especially when the Ciaglia Blue Rhino technique is used (Cook; Son, the Netherlands). Indeed, Ferraro et al1 show that the duration of this procedure (from incision of the skin to insertion of the tracheostomy tube) is only 2.45 ± 1.36 min (mean ± SD). Keeping in mind that 1 min of this time period is used for the

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single one-step dilator to dilate the trachea wall, the time period during which the bronchoscope is present in the airways is even shorter.

Second, in our experience, hypoventilation during the procedure is only present during the short period of dilation of the trachea. To demonstrate this, we present end-tidal CO₂ levels in five consecutive patients in our clinic. In these patients, we used the Ciaglia Blue Rhino technique. To assess ventilation during the procedure, we withdrew the endotracheal tube with the cuff just above the vocal cords; the cuff was then re-inflated. Tidal volumes were kept constant, and pressure limits were adjusted. For bronchoscopic guidance, we used a tracheal intubation fiberscope (Olympus LF-GP; Olympus Winter & Ibe; Hamburg, Germany). As can be seen in Figure 1, during the 5 min when the scope was present in the airways (indicated by the arrow), there was no rise in end-tidal CO₂ levels. Furthermore, even dilation does not result in a rise in end-tidal CO₂ levels. Based on our experience, we prefer this technique above one in which a potential dangerous procedure must be performed (changing the endotracheal tube).

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To the Editor:

We thank Dr. Dongelmans and his coworkers for their interest in our study (July 2004) and for sharing their data. We agree that the use of a small-sized scope (≤ 4 mm) generally may allow adequate ventilation around the bronchoscope during a percutaneous tracheostomy, but these smaller scopes also do not permit secretions to be adequately cleared from view. Nevertheless, a separate operator is required to maintain the withdrawn endotracheal tube with the cuff just above the vocal cords, a very proximal scope position that provides little margin for safety against extubation. We proposed our procedure because changing the endotracheal tube is safe when using a tube exchanger, even in cases of difficult airways, and ensures airway control during the whole procedure.

The following are not clear from the data reported by Dongelmans:

1. Which are the clinical and respiratory baseline characteristics of their five patients?
2. How do the end-tidal carbon dioxide concentration (EtCO₂) levels vary during the critical moments of the procedure, which begins with the maneuver of dilatation and ends with the tracheostomy tube placed? It is not clear whether the scope is disturbed during these two important phases. Also, a few minutes after the tracheostomy tube has been positioned, the registration of important and/or dangerous variations of the EtCO₂ could be missing.

Figure 1. End-tidal (EtCO₂) values in five consecutive patients during percutaneous dilational bronchoscopy. The separate phases of the procedure are indicated by arrows. Data are presented as mean ± SD.
3. How much were the pressure limits adjusted?
4. How were the tidal volumes maintained at a constant flow during tracheal obstruction by the dilator?

In our study of 40 patients, the technique used ensured airway control with continuous pressure control ventilation, thanks to the gas supply at the carina level, so that it did not interfere with the surgical field and when the operating time was unexpectedly prolonged. Also, it allows the use of a normal flexible fiberoptic bronchoscope (Ø, 6 mm) for the whole procedure, without interfering with ventilation. The ventilation technique that we proposed seems to be secure independently from the percutaneous procedure used in stabilized critically ill patients (mean [± SD] APACHE [acute physiology and chronic health evaluation] III score, 56.80 ± 24.03; PaO2/fraction of inspired oxygen ratio, 253.41 ± 94.83), in whom even a short apnea time can be dangerous.

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Association Between Epidermal Growth Factor Receptor Mutation and Improved Survival in Never-Smokers With Primary Adenocarcinoma of the Lung

To the Editor:

In the August 2004 issue of CHEST, Nordquist and colleagues1 show that never-smokers with primary adenocarcinoma of lung are predominantly female, present at a higher mean age, and have improved survival when compared to current smokers. These findings may indicate that adenocarcinomas occurring in never-smokers may display a distinct natural history. Epidermal growth factor receptor (EGFR) protein overexpression was observed in 32 to 79% of cases of non-small cell lung cancer, and occurred more frequently in small cell lung cancer and bronchoalveolar carcinoma than adenocarcinoma or large cell carcinomas.2

Retrospective analysis of patients receiving single-agent gefitinib, a tyrosine kinase inhibitor that targets EGFR, showed that responses were more frequent among patients who had never smoked, women, and patients with bronchoalveolar carcinoma or adenocarcinoma with bronchoalveolar features as in the present study.3 It has been shown that these dramatic clinical responses in these patients are induced by activating mutations within the EGFR kinase domain,4,5 which stimulates antiapoptotic pathways.6,7 In the light of above information and given the fact that chromosomal abnormalities are infrequent in never-smokers, mutations in the EGFR kinase domain may be one of the most important pathogenetic mechanism in this specific group of patients.

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Sputum Eosinophils and Bronchodilator Reversibility

COPD or Asthma?

To the Editor:

The study by Perng et al1 evaluated the inflammatory cell constituent and bronchodilator reversibility of a group of patients