Failure of Double-Lumen Tube Cuff Deflation on the Cuff Leak Test

To the Editor:

Difficulty in removing air from a cuff is usually caused by valve failure or damage to or kinking of the tubing connecting the pilot balloon to the cuff. We experienced another mechanism of cuff deflation failure while performing the cuff leak test.

When intubating with a double-lumen tube, we routinely use a stylet supplied by manufacturers to alter the curvature of the tube to facilitate intubation. We found that if the tube is bent before checking for a cuff leak, it is possible that complete cuff deflation might fail. Difficulty in removing air from a cuff can be caused by the structural characteristics of a double-lumen tube. In some textbooks, it is stated that the stylet may obstruct the cuff-side port. This may result in the cuff on the outer curve of the tube deflating faster than that on the inner curve, resulting in occlusion of the cuff inflation lumen before complete deflation of the cuff (Fig 1A). Recently, most commercially available tracheal tubes are free of this risk because the newer tubes have the cuff inflation lumen on the inner curve of the tracheal tube. With endobronchial double-lumen tubes, the tracheal cuff lumen has to be located on the outer curve because the endobronchial tube joins the tracheal tube at its inner curvature.

When carefully examined, tubes by different manufacturers have the position of the tracheal cuff lumen and its openings positioned slightly differently. They are very close to the midline with the BronchoCath (Mallinckrodt, St Louis, MO), whereas they are slightly away from the midline in Blue Line tubes (Portex Ltd, Hythe, Kent, UK) (Fig 1B). If the cuff lumen or its orifice is away from the midline, it will not be obstructed during cuff deflation.

Although this phenomenon may not be of significance during extubation, while checking the cuff of a double-lumen endobronchial tube, one should be cautious that the tube is never overly bent and also that the shape of an inflated cuff is not deformed.

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REFERENCES

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Postoperative Pain After Cardiac Surgery

To the Editor:

Various studies suggest that coronary artery bypass graft (CABG) surgery may lead to persistent chest wall pain known as post-CABG pain (PCP). In regards to this recognized complication, through similar investigation within the mainland Chinese population, the authors observed a difference in post-CABG pain incidence and performance compared with Caucasian published data.1-3

A total of 717 CABG patients who entered Peking University People’s Hospital Department of Cardiac Surgery between January 2001 and December 2003 were enrolled in the investigation through telephone questionnaire interviews. According to Mailis et al, PCP was defined as pain within the surgical location, different from pain experienced preoperatively, and localized pain occurring postoperatively persisting over 3 months.4 Of the 455 patients who responded, 149 (32.7%) (107 male and 42 female) reported PCP. Pain commenced 5.1 ± 6.8 months after surgery. The mean pain intensity numerical rating scale was 3.9 ± 1.7. Fifty-one (34.2%) patients complained that their pain interfered with their daily activities, and 59 (39.6%) patients complained that their pain had negative effects on their mood. Incidences of PCP observed in the left side of
patients was 41.6%, in the midline region was 72.5%, and in the right side was 12.1% (Fig 1). Hypoesthesia (defined as a reduced sensitivity to cutaneous stimulation) and mechanical allodynia-like pain (defined as extreme pain evoked by a slight friction of cotton underwear) were found in 10 patients (6.7%) and 11 patients (7.4%), respectively. Logistic regression analysis showed that sex and body mass index were independent predictors of pain. The authors defined PCP according to the definition presented by Eisenberg et al; however, the Chinese patients’ pain presented differently possibly because of ethnic differences. PCP mechanisms may involve injury of left intercostal nerves during chest opening and, more specifically, during the process of left internal mammary artery (IMA) harvesting, which was initially suggested by Mailis et al and was further supported by Eng and Wells. The data displayed a higher incidence of left chest pain in patients who had the left IMA harvested than in those who did not (43.1% v 33.3%), but this difference was not statistically significant (p = 0.314).

As of today, there are limited studies regarding PCP influential factors. Rowe and King reported a follow-up study of 51 women who underwent CABG surgery, revealing women with IMA grafts experienced significant (p = 0.003) discomfort. Patients with preoperative angina and those who were overweight or obese (body mass index \( \geq 25 \)) at the time of surgery were more likely to report PCP. This study indicated sex (female) and obesity (body mass index \( \geq 25 \)) to be independent risk factors for PCP. This result suggests female patients may be more sensitive toward pain than male patients, as seen in some other chronic pain syndromes. Overweight patients usually have a longer hospital stay and more postoperative events, suggesting that hospital length of stay and postoperative events are more relevant specific predictors than body mass index. More than half of the patients came from other provinces outside Beijing, causing difficulty in regular full examinations at this hospital. Hence, data could only be collected by telephone interviews without objective neurologic examination. This study was a retrospective study with data from only one institution, which is the main limitation. Trials aimed at reducing PCP prevalence, perhaps through improved surgical techniques, are indicated in the near future, especially for patients with high-risk factors.

### Argatroban “Reversal” Is Caused by Nonphysiologic Stimulation of Coagulation, Not Activated Factor VII

To the Editor:

We read with interest the report on argatroban anticoagulation monitored by thromboelastography (TEG). The authors suggested that activated factor VII (FVIIa) might overcome argatroban anticoagulation based on the “normal” TEG tracing with dual activators (rapid TEG) kaolin and tissue factor (TF). However, there are several issues with this interpretation of which the readers should become aware. First, contact-activated tests (eg, partial thromboplastin time and activated coagulation time) are more sensitive to argatroban inhibition. It is important to mention that platelets and fibrinogen can be rapidly activated when a relatively small amount of thrombin (10-20 nmol/L, peak thrombin >150 nmol/L) is available. A seemingly “normalized” TEG tracing does not represent the recovery of endogenous thrombin generation (Fig 1B). Coagulopathy in the presence of argatroban is typically multifactorial because of different underlying conditions such as heparin-induced thrombocy-