To the Editor—Mphanza et al.1 described a problem encountered during percutaneous dilational tracheostomy. The problem was thought to occur because the wire guide had been threaded through the Murphy eye of the endotracheal tube, preventing successful passage of the 8-French catheter guide.

Since Ciaglia et al.2 first described the technique of percutaneous tracheostomy in 1985, it has been noted that many of the possible complications were primarily caused by the blind nature of the procedure.3 This has led to the common practice of passing a fiberoptic scope down the endotracheal tube before passing the needle into the trachea. This not only allows visualization of the needle and subsequent guide wire passage, but it also serves as a safeguard as the tube is pulled back during the procedure. Carrillo et al.4 described their experiences with a series of 35 patients in which 33 of the procedures were accomplished with bronchoscopic guidance. They observed no significant complications and documented a significant savings with the bedside procedure. Although Berrouschot et al.5 reported comparable rates of complications between “blind” versus bronchoscopic-aided percutaneous tracheostomies, the complications were more severe in the blind group.

Thus far, the only disadvantage of the bronchoscopic portion has been the potential for increased intracranial pressure. Carrillo et al.4 noted increased intracranial pressure in one of their patients. Reilly et al.6 compared three methods of tracheostomy: percutaneous endoscopic, percutaneous Doppler, and standard surgical technique. In some patients the endoscopic technique resulted in significant hypercapnia and an increase of intracranial pressure to unacceptable levels.6

The addition of fiberoptic bronchoscopy to the percutaneous dilatational tracheostomy procedure does not guarantee 100% success of elimination of all complications but certainly could have prevented the problem encountered by Mphanza et al.1 Percutaneous dilatational tracheostomy can be accomplished with relatively low risk in a blind technique. However, the procedure has been shown to be safer with the assistance of bronchoscopic guidance and should be undertaken in that manner whenever it is not otherwise contraindicated.

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Vascular Effects of Isoflurane: No Inconsistency between Data

To the Editor.—We read with interest the article by Zhou et al.1 entitled 'Isoflurane-induced dilation of porcine coronary arterioles is mediated by adenosine 5'-triphosphate-sensitive potassium channels' recently published in Anesthesiology. The authors used a unique in vitro microvessel imaging system that allows the investigation of isolated microvessels apart from confounding variables related to the surrounding myocardium and in the absence of shear forces, blood flow, and circulating vasoactive substances. The authors observed that microvessels averaging 172 ± 51 μm (SD) in diameter that were precontracted with either acetylcholine or the thromboxane analog U46619 relaxed by a mean of 25% of the vessel diameter. This relaxation was partly inhibited in the presence of the ATP-sensitive potassium channel blocker glibenclamide. The authors concluded appropriately that isoflurane induces isolated precontracted, porcine coronary arterioles in vitro in a manner similar to that observed in studies in vivo. However, the authors claim that the findings are in conflict with those of Park et al.2-4. Park et al. reported that isolated coronary microvessels from the rat,5 rabbit,6 and, to a lesser extent, the pig,7 contract slightly in response to isoflurane. However, there are several differences between the study by Zhou et al.1 and those of Park et al. First, vessels in the studies by Park et al.2-4 only contracted when the vessels were studied in a noncontracted or a predilated state.2-4 Contraction was never observed when vessels were precontracted. Secondly, a markedly heterogeneous sensitivity of isolated microvessels to the effects of isoflurane was observed in vessels from the rat and rabbit. Park et al.2-5 found that the smaller the coronary vessel (e.g., <100 μm), the greater the observed contractile response. Microvessels greater than 260 μm dilated potently in response to isoflurane, even when precontracted.5 Because the vessels in the study by Zhou et al.1 averaged 172 μm in diameter, these differences could be explained in part by the differences in vessel size. Zhou et al.1 commented in the discussion that one explanation for the perceived discrepancy between the studies may be caused by the rate of administration of isoflurane. Acute administration of isoflurane causes greater relaxation than if isoflurane is given slowly and long-term. This is a very good point and may in part explain the differences between the findings of the two laboratories. However, the other factors need to be addressed. We believe that the study recently published by Zhou et al.1 is very informative, well executed, and complimentary with those of Park et al.2-4. There is no inconsistency between the data obtained by the two groups of investigators. Park et al.2-4 never stated that isoflurane is not a potent vasodilator of coronary arteries. They only claimed that isoflurane causes a heterogeneous response of coronary microvessels, with larger microvessels dilating more potently than smaller vessels, and that the response is largely dependent on the preexisting tone of the vessels, as it is with most other vasodilators. We appreciate your attention to this matter.

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