CORRESPONDENCE

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Preventing Complications during Percutaneous Tracheostomy

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. Carillo *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. Carillo *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 dilational 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 dilational 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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In Reply:—Thank you for giving us the opportunity to respond to Drs. Fuhrman and Bouvette. We agree with them in principle that simultaneous bronchoscopic monitoring in our reported case would most likely have prevented the problem.

In comparing groups with and without bronchoscopy, Berrouschot *et al.*[1] found no difference in the rate of perioperative complications; however, there were more severe complications in the group without bronchoscopy. Although invaluable to percutaneous dilational tracheostomy, bronchoscopic guidance is of special value for patients with abnormal or poorly felt surface anatomy. Percutaneous dilational tracheostomy in our intensive care unit is performed as described by Ciaglia[2] and is only performed by experienced consultant anesthesiologists. We do not routinely pass a fiberoptic scope down the endotracheal tube before puncturing the trachea in all our percutaneous dilational tracheostomies. Typically we have a consultant anesthesiologist performing the procedure and a senior resident providing anesthesia and minding the airway. Fiberoptic bronchoscopic proficiency varies among our residents; therefore, to have bronchoscopic monitoring would require the presence of an extra consultant anesthesiologist. We have found that bronchoscopic monitoring makes ventilation more difficult because of the reduced gas flow though

the endotracheal tube because of the presence of the fiberoptic scope. Increasing the fractional inspired oxygen tension ($F_{I_{O_2}}$) to 1 can compensate for oxygenation but hypercapnia remains a problem. Patient selection is vital, we refer obese patients and those with abnormal anatomy to the head and neck surgeons for an open procedure.

Our complication rate is similar to that quoted in the literature,[1,3,4] and so far we have had only minor complications. Bronchoscopic guidance may prevent complications such as the one we reported; we are currently reviewing our practice to incorporate simultaneous bronchoscopic monitoring.

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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 dilates 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,[3] rabbit,[2] and, to a lesser extent, the pig,[4] 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.*[2,3] 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.[3] 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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