Selective Bronchial Intubation with the Univent System in Patients with a Tracheostomy

To the Editor.—In routine anesthesia and in certain operations, selective bronchial intubation is warranted; however, in some cases, the procedure may be limited by particular patient characteristics. We report two patients in whom selective bronchial intubation through a tracheostomy was performed using the Univent system.¹

Case 1

A 50-yr-old man was scheduled for a right upper lobectomy to remove a cavitary nodule preoperatively diagnosed as metastatic epidermoid carcinoma. History included epidermoid carcinoma of the larynx treated with a total laryngectomy 5 yr before. To improve the surgical field and because of the unavailability of a Robertsaw tube, selective right bronchial intubation was performed using a no. 8 Univent tube. The tube was introduced through the tracheostomy. After rotation of the tube to the right, the bronchial blocker was introduced to obstruct the right main bronchus (fig. 1).

Case 2

A 65-yr-old man was scheduled for a left pneumonectomy. A total laryngectomy for epidermoid carcinoma of the larynx had been performed 2 months before. We decided to perform selective left bronchial intubation through the tracheostomy and a No. 8 Univent tube was used. Correct position of the blocker was achieved on the first attempt and was confirmed by pulmonary auscultation. The bronchial blocker was withdrawn slightly when the surgeon cut and sutured the left main bronchus.

In both cases the procedure was completed in a few minutes and without complications. After thoracotomy, the efficacy of the selective intubation was confirmed, although the surgeon had to evacuate air from the lungs because of the small lumen of the blocker pilot tube. Although use of the Univent tube is less ideal than a double-lumen tube²—³, i.e., the two lungs cannot be ventilated independently, and aspiration of secretions from the collapsed lung is difficult⁴—it represents an easy-to-use option for selective bronchial intubation of a patient in whom a tracheostomy is present. Confirmation of correct intubation by fiberoptic bronchoscopy is advised, as it would considerably shorten the time required to perform the technique, while limiting the number of possible errors.

Fig. 1. Cross-section of the tracheobronchial tree showing the final position of the Univent tube.
Hypotension after Indigo Carmine

To the Editor—Recently Shir and Raja described four patients who became unexpectedly hypotensive after the administration of indigo carmine intravenously during otherwise stable surgical and anesthesia conditions. Their anesthetic technique included a high epidural block produced by bupivacaine in three patients and lidocaine and tetracaine in one patient. The indigo carmine was injected 80 min after induction of anesthesia in one patient and 2 h after induction in the other three. In discussing the possible causes of hypotension induced by a drug that usually causes hypertension in intact awake humans, they list an idiosyncratic reaction as most plausible, and I would like to suggest another possibility.

Various agents cause both stimulant and depressant effects simultaneously, the predominance depending on the prevailing circumstances. For example, carbon dioxide is a central stimulator of the vasomotor center and a peripheral local depressant. In an anesthetized animal model, carbon dioxide causes hypotension, but when sympathetic blockade is induced, subsequent administration of carbon dioxide causes further hypotension. Various agents have shown that ketamine, which normally results in sympathetic stimulation but also has depressant properties, causes hypotension comparable to an equipotent but small induction dose of thiopental in a hypovolemic model, despite a measured increase in circulating catecholamines. Presumably, in this instance, the stimulant effect is offset by the lack of volume, and the depressant component prevails. Nitrous oxide behaves similarly.

I suggest that the authors consider an effect similar to the above as one possible cause of the phenomenon that they have documented. Possibly, in this instance, the usually observed stimulant effect was prevented by the sympathetic block caused by epidural blockade by long-acting agents, leaving a depressant component of the drug unmasked. The pharmacology of indigo carmine is not described clearly, so one can only conjecture. I am unaware of the exact site of action at which the usually observed stimulant effect is initiated. Could it be at the vasomotor center? Could there always be a 5-HT-like effect such as the authors describe, which is unmasked only when sympathetic blockade prevents the usually more dominant stimulant effect? If my hypothesis is correct, this is hardly an idiosyncratic reaction. Should the title of the report have been "Indigo Carmine-induced Severe Hypotension in Patients with High Epidural Blockade"? Should the Food and Drug Administration indicate that the drug sometimes causes hypotension but define the circumstances? As is so often the case, we need to know more!

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References

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