Correspondence

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Modified Endobronchial Tube

To the Editor:—The well-known difficulty of ventilation of the lungs in the presence of a large bronchial fistula and the anesthetic problems presented by constrictive pericarditis led us to devise a modification of the White* double-lumen endobronchial tube for use in a patient who had both diseases. The patient, a 64-year-old man, had severe constrictive tuberculous pericarditis (with a central venous pressure of 30 cm H_2O), bilateral pleural effusions, severe bilateral pneumonia, and a bronchoesophageal fistula approximately 8 mm in diameter near the origin of the right upper lobe bronchus. Administration of oxygen, 50 per cent, by face mask was necessary to maintain an arterial blood oxygen tension above 50 torr with an arterial carbon dioxide tension of 34 torr. The patient was alert and responsive although tachypneic and weak. He was scheduled for ligation of the gastroesophageal junction, gastrostomy, and diverting cervical esophagostomy, as it was thought that these procedures would stress the patient as little as possible and offer a chance for his pulmonary condition to improve, permitting correction of the fistula and pericarditis in the future. Our experience with the unmodified White tube has been that it often fails to form a good seal in the right bronchus, and we believed that it would be unlikely to occlude the fistula. We thought that the benefit to respiration of fistula occlusion would outweigh the loss of ventilation of the right upper lobe. Therefore, we modified a White tube by affixing a soft cuff with a pilot balloon over the right bronchial cuff of the tube, securing the inflation tubing of the soft cuff to the proximal end of the tube by means of a 2-0 silk ligature.

After placement of a radial-artery catheter to monitor blood pressure, anesthesia was slowly induced with diazepam, 10 mg, and morphine sulfate, 3 mg, while having the patient breathe oxygen with enflurane, 0.5 per cent. In his debilitated condition, this technique permitted laryngoscopy, laryngotracheal anesthesia with 4 per cent lidocaine, and insertion of the modified White tube without abolishing spontaneous ventilation. The tracheal and bronchial cuffs were inflated and the tube was checked for correct position by briefly occluding the ventilation to each lung. The patient continued spontaneous ventilation, and as cardiac status permitted, respiration was assisted without clinical evidence of a leak through the bronchial fistula. Arterial blood–gas values were satisfactory throughout the surgical procedure. Anesthesia was maintained with nitrous oxide–oxygen, 50 per cent each. At the termination of operation, with the patient still breathing spontaneously, the trachea was extubated. His ventilatory pattern and arterial blood–gas values returned to their preoperative condition. We found this modified endobronchial tube extremely helpful in the anesthetic management of a patient with an unusual combination of diseases.

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* Rusch, Inc.

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"Fail Safe"? Unsafe!

To the Editor:—The so-called low-pressure "fail-safe" oxygen system has in the past decade become standard equipment for anesthesia machines. Even the Joint Commission on Accreditation of Hospitals' surveys include checking this item. It is our contention, however, that this system as presently utilized in most institutions does not constitute a significant safety factor. In a majority of hospitals, the oxygen source to the anesthesia apparatus comes from piped oxygen. The "fail-safe" apparatus does not function so long as the line pressure is maintained even in the absence of any flow through the flowmeter. This is also true in situations where cylinders are used.

In a personal communication, Dr. L. Rendell-Baker

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informed us that to his knowledge only one type of gas machine* from a single manufacturer employs a truly fail-safe device, whereby closing the flowmeter valve on the oxygen line will automatically shut off all other gas flow. It becomes obvious that if nitrous oxide or other gases continue to be delivered in the absence of oxygen, as is readily possible with the usual apparatus, "fail-safe" is a misnomer. We contend that no-one administering anesthesia should ever rely on this type of equipment, and accordingly it becomes an expensive and dangerous addition to the machine. The one brand of equipment containing an actual fail-safe mechanism could well be emulated by other manufacturers and by this manufacturer in its other models.

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* Foregger Company—Model 710.

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Cardiovascular *vs.* Renal Effects of Dopamine

To the Editor:—The communication by Birch and Boyce purports to demonstrate that the butyrophenone drug droperidol does not prevent "dopamine-induced renal vasodilatation" when given in a dose of 0.1 mg/kg.[1] Their evidence for this is a measured increase in renal blood flow when dopamine was infused into patients that was not significantly attenuated by droperidol administration. However, the dosage of dopamine used (20 μg/kg/min) would not be expected selectively to stimulate only the vascular dopaminergic receptors that would be blocked by droperidol.[2] This selective stimulation typically occurs only at very low doses of dopamine where cardiac output and systemic blood pressure are unaffected. Robie and Goldberg found that at a dose of dopamine of 1.25 μg/kg/min in dogs, cardiac output, total peripheral resistance, and systemic blood pressure did not change significantly, but renal vascular resistance decreased and renal blood flow increased significantly.[3] At the high dose ranges used by Birch and Boyce, cardiac output should be markedly increased by beta-receptor stimulation.[2,3] Cardiac output *per se* was not measured, but the authors did state that [dopamine] "increased systolic blood pressure, diastolic blood pressure, and renal blood flow." These changes would be the expected results of a large increase in cardiac output. Drs. Birch and Boyce have demonstrated that if you increase cardiac output with a potent inotropic agent, renal blood flow will increase. This is hardly surprising. The study should be repeated using more appropriate doses of dopamine.

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REFERENCES

1. Birch AA, Boyce WH: Effects of droperidol–dopamine interaction on renal blood flow in man. ANESTHESIOLOGY 47:70–71, 1977
2. Goldberg LI: Cardiovascular and renal actions of dopamine: Potential clinical applications. Pharmacol Rev 24:1–29, 1972
3. Robie NW, Goldberg LI: Comparative systemic and regional hemodynamic effects of dopamine and dobutamine. Am Heart J 90:340–345, 1975

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In reply:—It was not the purpose of our study to show whether droperidol prevented dopamine-induced renal vasodilation. This was done by Yeh and others with a similar drug, haloperidol.[1] Ours was a study of droperidol–dopamine interaction. We agree that the increased blood flow following dopamine in patients pretreated with droperidol could be entirely on the basis of increased cardiac output or some combination of increased cardiac output and increased renal blood flow. The important point is that the renal blood flow increased following the usual clinical doses of dopamine and droperidol, 0.1 mg/kg.

To determine how much of the effect resulted from increased cardiac output and how much from direct vasodilation would require more sensitive equipment than we presently have available. For example, using low-dose dopamine, 1.25 μg/kg/min, the renal blood flow changes in Robie's dogs were only about 50 ml/min.[2] This is within the error of our flow probes used in man. Another problem we had with our patients