In reply: Dr. Heavner’s comment is much appreciated, but the word “conflict” seems too strong to describe the difference in lengths to which he refers. As stated in our article, the nerve compartment was indeed 20 mm long and the conduction distance typically 50 mm, as illustrated in our figure 2 and noted in the legend. What Dr. Heavner failed to deduce, and what we failed to specify, was that the additional 30 mm of nerve lay in the ganglion compartment. We are glad of the opportunity to present this clarification. The diagram in figure 1 was, of course, not drawn to scale.

It is a pleasure to acknowledge again the priority of Heavner and de Jong, whose article we duly referred to in our discussion, but it is also advisable to emphasize that the fibers they studied were efferent sympathetic, as opposed to the afferent vagal ones studied by us, and that their inferences regarding differential block sensitivity were not conclusive because these were drawn from amplitude changes in compound action potentials, which could have been produced equally well by differential slowing of conduction (differential increase in latency). As noted in our article and fully documented subsequently, our studies of individual units demonstrated that, in our model, lidocaine did indeed produce a significantly larger average increase in latency among the myelinated axons than among the unmyelinated ones.

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REFERENCES


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A Simple, Fool-proof Method to Prevent Hypoxic Accidents

To the Editor.—The prevention of hypoxic accidents during anesthesia is a matter of great concern to anesthesiologists throughout the world. Rendell-Baker and Meyer suggest yet more sophisticated gadgetry by automating the use of oxygen analyzers as a further safeguard against these tragic and, often, expensive accidents. Increasing sophistication, however, also increases the possibility of malfunction as well as cost.

I remain astonished that the problem is still not tackled at source, i.e., if hypoxic gas mixtures were not available, hypoxic gas mixtures could not be administered. Why do we go on tolerating cylinder and pipeline supplies that are capable of delivering 100% nitrous oxide? Surely the medical gas companies could premix pipeline supplies in such a way that the nitrous oxide line delivered, say, 80% nitrous oxide and 20% oxygen. Similarly, where it is necessary for cylinders to be used, surely a premix device could be incorporated with the reducing valve assembly to ensure that the nitrous oxide cylinder could only be used with an attached oxygen cylinder so that the same 80:20 mixture was delivered. Such a system, universally applied, would remove the human error factor from an anesthesiologist delivering hypoxic mixtures and also would remove the hypoxic hazard from accidentally crossed pipeline installations. On anesthetic machines,
apart from the cylinder mixing valve, the only modifications necessary would be relabeling and recalibrating the nitrous oxide flow meter, and these costs should be relatively low.

I accept that anesthesiologists would have to "recalibrate" themselves to some extent in terms of gas flows, but a simple graph, as shown in figure 1, would allow instant calculation of delivered percentages as with air/oxygen mixtures. Thus, at a convenient 8 l/min total flow (solid line), if no added oxygen were given, the nitrous oxide/oxygen mixture alone would be delivered, i.e., 20% oxygen. If one liter of oxygen through the oxygen flow meter were added to seven liters of the nitrous oxide/oxygen mixture (thereby maintaining the eight-liter total flow), the total oxygen concentration would be made up of 20% of seven liters (1.4 l) and the added one liter, giving 2.4 l out of the eight liters total flow, i.e. 30%. Thus, by "exchanging" one liter of the nitrous oxide/oxygen mixture for one liter of pure oxygen, the oxygen concentration of the delivered 8 l/min is increased by 10%. Other "flow-lines" (dotted lines) can be used accordingly. In practice, most anesthesiologists tend to use the same total flow for any given anaesthetic breathing system, so that we would all soon become "preprogrammed."

Such a step, radical though it would be, would permanently protect patients, anesthesiologists, and hospital authorities from the disastrous effects of the delivery of accidental hypoxic mixtures. Of course, there might be an increase in awareness during anesthesia from the delivery of unduly oxygen-rich mixtures, but surely, this is the lesser of two evils!

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Postlaryngospasm Pulmonary Edema in Adults

To the Editor:—The recent report by Lee and Downes states that pulmonary edema following laryngospasm has not been reported in adults.1 I have had the misfortune, however, to be associated with two such cases.

The first was a 48-year-old man undergoing laryngoscopy and bronchoscopy for intermittent upper airway obstruction occurring during sleep or when he was in the supine position while awake. His only other medical problem was hypertension controlled with inderal and hydrochlorothiazide. The patient was induced using enflurane with spontaneous ventilation in a semisitting position. After induction, he was intubated without problem. Anesthesia was maintained with enflurane, nitrous oxide, and oxygen. The endoscopy revealed no pathology other than an irritated upper airway.

On emergence, the patient opened his eyes, became agitated, pulled out his endotracheal tube, and then developed apparent laryngospasm with complete upper airway obstruction. His airway could not be maintained with bag and mask ventilation, and cyanosis rapidly developed. Succinylcholine, 100 mg, was administered, iv resulting in cessation of laryngospasm. The patient was intubated and ventilation was controlled. Within 30 s, copious, frothy pink fluid poured from the endotracheal tube. A chest x-ray showed diffuse pulmonary edema without cardiomegaly. Arterial blood gases on 100% FiO2, showed 

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\text{Pao}_2 \ 116 \text{ mmHg}, \ \text{Paco}_2 \ 41 \text{ mmHg}, \ \text{and pH} \ 7.37.
\]

The patient received lasix and morphine, was placed on mechanical ventilation with 5 cm H2O PEEP, and was transferred to the ICU. The next morning the pulmonary edema had cleared and he was extubated successfully without sequelae.

The second patient was a healthy 21-year-old man who underwent an uncomplicated general anesthetic for septorhinoplasty. At the end, he was extubated and then went into laryngospasm with complete upper airway obstruction. The airway could not be managed with bag and mask ventilation. Succinylcholine, 60 mg, relaxed the laryngospasm, and once spontaneous ventilation resumed, the patient was transferred to the recovery room. In the recovery room, the patient had a persistent cough and dyspnea. A chest x-ray showed bilateral middle lobe infiltrates without cardiomegaly. The patient was given lasix with resolution of dyspnea. Three hours later, the chest x-ray was clear.

These cases show that postlaryngospasm pulmonary edema is not confined solely to children. It can occur in adults with normal or marginally abnormal upper airways. It should be considered as part of the differential diagnosis in all recovery room patients who are coughing or complaining of dyspnea who have had an episode of laryngospasm.