is anatomic in origin, the evidence they present indicates it is produced by the sheath, which is inserted to the depth of the inferior wall of the SCV, kinking the catheter between its tip and the vessel wall.

We have observed three instances of intraoperative PAC malfunction manifested by dampening of the pulmonary artery tracing and marked resistance to injection that occurred upon sternal retraction during cardiac surgery. As in the authors' case, the catheters were inserted without difficulty through the right EJV with the use of a modified Seldinger technique and flushed continuously. In both instances proper catheter function was restored by withdrawing the sheath 2–3 cm, leaving the PAC in its original position and allowing it to resume a less acute course through the EJV–SCV junction.

Pulmonary artery catheterization via the EJV is a safe and effective technique, avoiding the complications associated with internal jugular cannulation.[2–4] The risk of carotid artery puncture, cervical hematoma, phrenic nerve injury, and pneumothorax can be avoided in 75% of patients by preferential use of the EJV.[3,4] It would be unfortunate if Bromley and Moorthy's report were to discourage use of the EJV for pulmonary artery catheterization.

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(Accepted for publication November 1, 1983.)

Anesthesiology
60:514, 1984

Reported Nerve Conduction Velocities Clarified and Confirmed

To the Editor:—There is conflict between direct and indirect indications of conduction distance in the article by Fink and Cairns.[1] Length of nerve in compartment b of their nerve chamber is stated to be 20 mm. However, conduction distance calculated from reported latencies and conduction speeds equals about 50 mm. Inspection of the diagram of the incubation chamber rules out an additional 30-mm conduction distance in compartments a and c of the nerve chamber, assuming proportional diagraming of the chamber.

The conflict leaves open the question of whether the conduction velocities actually were half of what are stated (*i.e.*, 0.27 to 18.7 m/s, instead of 0.55 to 37.5 m/s). Regardless of which conduction velocities are correct, Fink and Cairns have confirmed elegantly the observation de Jong and I made years ago using compound action potential recordings[2]—namely, that myelinated axons having conduction velocities in the 3–15-m/s range are more sensitive to lidocaine than is a population of small unmyelinated fibers.

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(Accepted for publication November 1, 1983.)

Anesthesiology
60:515, 1984

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,[1] 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,[2] 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[1] and fully documented subsequently,[3] 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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(Accepted for publication November 1, 1983.)

Anesthesiology
60:515–516, 1984

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[1] 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,

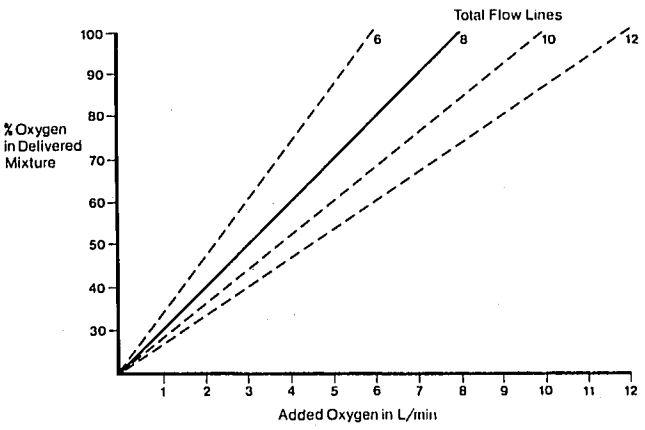

FIG. 1.