fibers. The amplitude variations of these potentials, especially in the range chosen by the authors, are not related so much to the number of fibers responding to electrical stimulation, but to temporal dispersion in the conduction velocity of individual fibers, as discussed and demonstrated by Gasser and Erlanger,[3] Franz and Igo,[4] Paintal,[5] and Franz and Perry.[6] Slowing of this velocity (cooling in mammalian nerves, anesthetics) in the fast and usually synchronized A fibers, causes a dispersion of single action potentials with the consequent flattening and widening of the compound action potential representing this population (A wave), and little or no change in the already flat and slow C wave. Thus, "differential sensitivities of mammalian nerve fibers to local anesthetics" should be studied on single nerve fibers since the compound action potentials are limited in their resolution for this type of study.

According to Franz and Perry[6] who worked with single fibers: 1) Small myelinated axons were blocked more quickly than large myelinated axons, but this differential effect could not be accounted for by the differences in anesthetic concentration requirements. 2) The onset of block in non-myelinated axons was slower than or equal to that of small myelinated axons depending on anesthetic concentration. 3) Absolute differential block of non-myelinated (blocked) and small myelinated (not-blocked) axons was obtained by limiting the length of axons exposed to procaine to 2 mm.

Gissen *et al.* did not take into consideration the combined effects of low temperature and local anesthetics on the A fibers, known to be more vulnerable to cooling than the C fibers.[4] They performed their studies at 22°–24° C and at a stimulation frequency of 1/min in mammalian nerves (vagus and sciatic of the rabbit) which normally function at a higher temperature. To be clinically meaningful, studies concerned with the effects of local anesthetics should be related to functional depression of small fibers transmitting at their natural frequencies and temperature of operation.

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(Accepted for publication March 27, 1981.)

Anesthesiology
55:483–484, 1981

In reply: I wish to respond to the comments made by A. Galindo, M.D. concerning my article "Differential sensitivities of nerve fibers."[1]

His objections focus on three points: 1) the use of the combined action potential (CAP) amplitude does not indicate the effect of drugs on single fibers (A or C) because the peak of AP is sensitive to varying changes in conduction velocity of the component fibers. 2) Low temperature does not effect conduction in A and C fiber proportionately. 3) A stimulus frequency of 1 min is unphysiological.

His general comment is one I agree with; when possible, experiments should be performed as close to normal physiological conditions as possible. But, accurate non-traumatizing dissection of C fibers (0.5 micron or less) in lengths sufficient for *in vitro* experiments is not possible; at least not in the same manner as for A fibers (5–10 micron). We chose to use the entire nerve trunk and to "dissect" fiber behavior by conduction velocity of AP peaks.

This, of course, makes the #1 criticism important. His quotes from Franz and Perry[2] imply much more certainty than the original article warrants. I quote from page 200, "non-myelinated and small myelinated axons were blocked (0.2 per cent procaine . . .) at about the same time"; "non-myelinated axons were no more susceptible to block than were the delta and some of the smaller alpha fibers." Or in the article by Gasser and Erlanger,[3] page 588: "reconstructions were made . . . in partially blocked fibers on the assumption that the velocity in fibers of each size would be the same fraction of normal as occurs in the fastest fibers."

His quote from Franz and Iggo[4] is just as selective. Figure 4 from that article presents a graph comparing

nerve temperature (0°–40° C) and conduction velocity (per cent of normal) for myelinated and nonmyelinated nerve. From 17°–40° C both plots are almost congruous (certainly there is no statistical difference). Since our experiments were conducted at 22°–24° C, I don't believe this criticism has any validity.

In addition, Heavner and de Jong[5] reported experiments at normal body temperature with rabbit sympathetic trunk (not desheathed) exposed to lidocaine that duplicated our results (on B and C fibers) with desheathed rabbit vagus at room temperature. They also found that B fibers were more sensitive than C fibers to the action of local anesthetic.

The last point he makes is true but only marginally meaningful. Experiments done at a fixed repetitive tetanic rate are also nonphysiological. Neural traffic during stimulation usually consists of a sequence of APs at varying frequencies, so that neural signals consist of frequency modulation patterns. I have never seen any experiment designed to recognize this fact.

Finally, I note in Galindo's comments (quoting Franz and Perry[2]) that B is compared to A fibers, C is compared to B fibers, but no relationship is established between A and C fibers. Our study very carefully examined, under identical conditions, the relationship between A, B, and C fibers.

I agree with the general tenor of Dr. Galindo's comments. I also wish experimental design was more sophisticated so that results could be directly evaluated in the light of normal physiology. I will gladly accept any criticism that constructively makes this possible. However, I also get the impression that Dr. Galindo is as imprecise in his conclusions as the quoted articles.

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(Accepted for publication April 17, 1981.)

Anesthesiology
55:484–485, 1981

Another Use for the Fiberoptic Bronchoscope

To the Editor:—The flexible fiberoptic bronchoscope is a valuable aid in intubating patients, especially when difficulty with intubation can be predicted. Recently, Rosenbaum *et al.*[1] described use of the instrument in changing endotracheal tubes. We wish to report yet another use for the fiberoptic bronchoscope: as a guide to proper placement of tracheostomy tubes.

Most tracheostomies are performed with an endotracheal tube *in situ*. Once the trachea is exposed surgically, the endotracheal tube is withdrawn and the tracheostomy tube positioned within the trachea. Occasionally, due to loss of exposure or anatomic variation, the tracheostomy tube cannot readily be inserted in the trachea. Unrecognized paratracheal placement of the tube can also occur. The authors have witnessed both complications on more than one occasion. It is therefore suggested that a fiberoptic bronchoscope be inserted into the indwelling endotracheal tube via a Rovenstine or similar adapter prior to withdrawal of the endotracheal tube (fig. 1). Under most circumstances this will be done by the anesthesiologist as the majority of elective tracheostomies are performed in the operating room with an anesthesiologist present to monitor the patient.

In addition to verifying proper placement of the tracheostomy tube, the endoscopist can examine the trachea for secretions, areas of inflammation, and other abnor-