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In Reply:—We thank Fiacchino for pointing out an ambiguous statement in our review article[1] that, following an upper motor neuron (UMN) lesion, "The upper limb muscles, relative to lower muscles, are more sensitive to the effects of nondepolarizing muscle relaxants (NDMR)." The message that was conveyed was that "the proximal muscles compared to distal appear more sensitive to the effects of NDMR." This conclusion was based on the reports that central or UMN denervation causes resistance to NDMR[2–4] and on the electromyographic evidence that central denervation, more frequently, affects the distal rather than proximal muscles.[5,6] The reports of Fiacchino *et al.* substantiate this claim whereby following UMN denervation, the trapezius muscle was more sensitive than the abductor digiti minimi[7] and that the adductor pollicis brevis was more sensitive than the flexor hallucis brevis.[8] Unfortunately, because of the lack of controls in these latter studies,[7,8] it was not possible to determine whether the sensitivity of these muscles was increased or decreased compared to normal muscles. We, however, disagree that syringomyelia is a disease of the lower motor neuron. The syringomyelic cavity dissects into and progressively replaces the gray matter of the posterior and anterior horns of the spinal cord.[9] Depending on the stage and severity of the disease, symptoms and signs of upper and/or lower motor neuron lesion may be present.

The claim that axonal sprouting should be regarded as a predisposing factor for subsequent development of *increased* sensitivity to NDMR is not consistent with other reports. Changes occurring with immobilization of a limb for example include, among others, terminal nerve sprouting,[10] yet resistance to NDMR has been observed.[11] Following reinnervation recovery from injury or remobilization, the response to depolarizing or NDMR will be quite variable,[12–14] and this variability may be related to prejunctional and postjunctional factors, including total receptor number and proportion of mature to immature receptors.[1]

We concur with Fiacchino's views that upper or lower motor denervation is not always accompanied with resistance to NDMR or hyperkalemia to succinylcholine. In our review,[1] we have enumerated reports in which exceptions have occurred (*vide* page 829 of review[1] and references 133–140). We also have listed a number of reports confirming hyperkalemia with succinylcholine following UMN denervation in which sepsis, concomitant chronic treatment of NDMR, or other predisposing factors were not present. We disagree with the notion that extrajunctional proliferation of acetylcholine receptors is not a normal consequence of UMN dysfunction. Increased sensitivity to acetylcholine or succinylcholine due to receptor spread[14,15] and proliferation of extrajunctional acetylcholine receptors, quantified by ^{125}I-α-bungarotoxin, has been observed following cordotomy[16] or other UMN disease of the spinal cord.[17] Electromyograph studies following stroke in humans have confirmed the denervation state by the presence of fibrillation potentials and positive sharp waves.[5] The magnitude and the duration of these changes, however, may not be as prominent as that seen following lower motor neuron denervation.

Jeevendra Martyn, M.D.
Department of Anesthesia
Massachusetts General Hospital
Boston, Massachusetts 02114

Gerald Gronert, M.D.
David White, M.D.
Department of Anesthesia
University of California, Davis
Medical Center
Sacramento, California 95817

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Undetected Leak in Corrugated Circuit Tubing in Compressed Configuration

To the Editor:—We report an incident involving a disposable anesthesia circuit. The original compressed configuration of the circuit's corrugation showed no sign of a leak during a preoperative positive pressure test. However, after intravenous induction of general anesthesia, the tubing corrugation was extended and a previously undetected flaw in the circuit (fig. 1) became the source of a major leak. Ineffective positive pressure ventilation ensued, leading to a decline in the patient's hemoglobin oxygen saturation (Sp_{O_2}). The circuit was abandoned for an ambu bag until the leak was identified and the circuit replaced. We suggest doing a preoperative positive pressure leak test with this type of circuit in the fully extended configuration to detect potential flaws in the circuit that might otherwise be missed.

Douglas J. Reinhart, M.D.
Department of Anesthesia
Clinical Faculty, University of Utah
2571 South 1825 East
Ogden, Utah 84401

Ralph Friz, M.D.
Director of Anesthesia
McKay-Dee Surgical Center
3903 Harrison Boulevard
Ogden, Utah 84409

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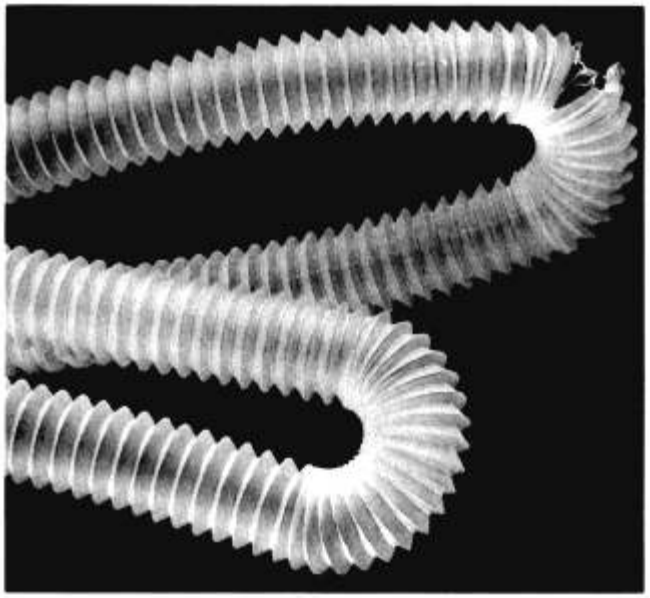

Fig. 1. Corrugated tubing showing defect.