In Reply—Dr. Warner questions our recommendation of using a rapid sequence induction technique with atracurium facilitated muscle relaxation due to suboptimal intubating conditions in five of our seven groups studied. He also suggests that we may have missed potentially harmful swings in IOP due to the intermittent nature of our measurements.

First, we agree that all combinations of iv anesthesia and atracurium (0.6–0.8 mg/kg) in our study did not consistently provide intubating conditions that would be considered optimum in patients with open eye injuries during rapid sequence induction. However, we feel that our study did demonstrate that atracurium, 0.6–0.8 mg/kg, had little to no effect on IOP. Also, the purpose of our study was to evaluate the effect of atracurium with various induction regimens and doses. It was not intended to imply that, because we found no effect of atracurium on IOP, all of the combinations would be considered ideal. As we stated in the results, intubating scores were improved with increasing doses of atracurium at the same dose of thiopental and when atracurium was administered prior to thiopental. We also noted that the group in which iv lidocaine and a large dose of thiopental (7 mg/kg) was given had the best intubating scores overall.

Secondly, we agree with Dr. Warner’s statement that intubating stress could be responsible for undesirable dramatic transient increases in IOP for patients with open eye injuries. We feel that our recommendations for the use of atracurium (0.6–0.8 mg) with the proper selection of both an induction agent and dose is justified. It currently represents the best selection for such patients. The use of succinylcholine in these patients is controversial, and the longer-acting non-depolarizing agents have their limitations. Therefore, we feel that the use of larger doses of thiopental (7 mg/kg or greater) and prevention of laryngeal-tracheal induced reflexes, in conjunction with atracurium or vecuronium, provides excellent intubating conditions and is associated with minimal changes in IOP.

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“Stiff-Baby” Syndrome: An Expression of the Same Neural Circuitry Responsible for Opiate-induced Muscle Rigidity?

To the Editor—Cook and Kaplan, in a recent case report, discuss the management of neuromuscular blockade in a patient with the so-called “Stiff-Baby” syndrome (hyperekplexia). It is interesting to speculate whether the etiology of this genetic disorder is due to an alteration in the same neural circuitry which is responsible for opiate-induced muscle rigidity in normal patients.

The “Stiff-Baby” syndrome is characterized by generalized muscle rigidity, continuous EMG activity, and an exaggerated startle. Opiate-induced catatonia, which has been extensively studied in animals and humans, has very similar characteristics. For example, rats which have received a high dose of opiates exhibit akinesia and a profound extensor muscle rigidity. In addition, these animals display a markedly enhanced startle response, called “explosive motor behavior” (EMB). It is postulated that opiate-induced catatonia is a manifestation of the immobility or “death-feign” reflex, an innate reflex seen in many prey species which is believed to be mediated by endogenous opiates. For example, if a prey animal is suddenly trapped by a predator, it will acutely become stationary and profoundly stiff. This terminal defense mechanism may reduce the risk of sustained attack. The reflex is often followed by explosive escape behaviors.

In rodents, it is known that muscle rigidity occurs if there is an inhibition or interruption of the inhibitory GABAergic pathway which runs from the basal ganglia via the ventromedial thalamus and the superior colliculus to the brainstem. Dopaminergic neurons within the basal ganglia may also be involved. Several midline nuclei within the medullary and pontine reticular formation appear to play a role in opiate-induced rigidity (unpublished). Nuclei in this region are the primary source of serotonergic innervation in the brain, and serotonin has been implicated in both opiate and non-opiate rigidity. There are also data to suggest that this brain region is responsible for integrating the motor components of EMB.

With regard to “Stiff-Baby,” or the apparently similar “Stiff-Man,” syndrome, it seems reasonable to speculate...
that these patients have some kind of genetic defect which affects one of these brain pathways. It would be interesting to know if agents which alter endogenous opiate (naloxone), GABA (baclofen), or serotonin (ketanserin) activity would be clinically effective in reducing their muscle rigidity. For the future anesthetic management of these patients, one might anticipate that drugs which increase brain opiate activity (narcotics or nitrous oxide) would make their rigidity worse, while drugs which have been shown to have central muscle relaxant properties (benzodiazepines, barbiturates, ketanserin, inhalational agents) would attenuate the rigidity.

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REFERENCES
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Yet Another Reason to Use a Fiberoptic Bronchoscope to Properly Site a Double Lumen Tube

To the Editor:—Among the various methods for one-lung ventilation, a recently introduced endotracheal tube with a movable blocker (Univent tube,® Fuji Systems Co. Ltd., Tokyo, Japan) has the advantages of ease of suctioning through its large main lumen, and for one-lung high-frequency ventilation through its small tube.1 We describe an unusual complication with the use of this tube.

A 77-yr-old male patient (weight 37.5 kg, height 163 cm) with esophageal carcinoma was scheduled for esophagectomy. Following induction of anesthesia and paralysis, the trachea was intubated with a Univent® tube (size 37 F). After intubation, a fiberoptic bronchoscope (FOB; Olympus BF type 4B2, outer diameter 4.8 mm) was introduced into the main tube through a suction adaptor used for ventilation. Under direct vision, the tube was twisted approximately 90° toward the right side to be occluded, and the small tube was advanced. At that time, the silicon cap covering the tip of the small tube was dislodged and remained on the wall near the carina (fig. 1). The patient was placed in a head-down position to prevent the cap from moving distally, and the cap was successfully removed with a biopsy forceps via the suction channel of the FOB. The tube was removed, and another Univent tube® was placed without incident. There were no long-term sequelae.

FIG. 1. The tip of the small tube of the Univent® tube and the dislodged cap.