that there is a routine increase of masseter muscle tone in normal patients anesthetized with halothane or enflurane and given succinylcholine at the same time there was complete relaxation of limb muscle clinically, as well as loss of thenar muscle twitch.[1,2] There is certainly good evidence from *in vitro* studies to suggest that partial depolarization (produced by succinylcholine) combined with prior exposure to halothane will result in tension development in normal muscle. This is due to a halothane-induced shift in the voltage dependence of calcium release in the muscle.[5] It now becomes evident why the clinical determination of trismus is so difficult. It is very difficult to determine the difference between an exaggerated normal response to succinylcholine and a pathological response in which the increase in masseter tone may herald malignant hyperthermia susceptibility. Ideally, the use of the word "trismus," or "masseter spasm," should be reserved for the abnormal response of the masseter muscle to succinylcholine, but, practically, this may be very difficult. Fortunately, the incidence of malignant hyperthermia is very rare, occurring anywhere from 1 in 15,000 children up to 1 in 50,000 adults. The incidence of abnormal masseter muscle response (masseter spasm) with a combination of halothane and succinylcholine occurs in 1% of the patients in Boston (1 in 100) and 0.01% of the patients in Charlottesville (1 in 10,000).[6] The reason for the great differences in occurrence is not apparent, although it may represent subtle differences in clinical practice.

The first problem faced by the clinician is what to do if a patient does develop masseter spasm after succinylcholine. The recommendations cover a broad spectrum. Gronert recommends that the anesthesiologist continue the anesthetic with nontriggering agents, while monitoring end-expired CO_2, venous and/or arterial blood gases, blood pressure, pulse, temperature, and muscle tone.[3] If there is any aberration, then the case is cancelled and treatment begun with dantrolene. On the other hand, if the situation is stable, the anesthetic can proceed with nontriggering agents. We agree with this management. At the other end of the spectrum is the opinion as expressed by Rosenberg with whom we strongly disagree.[4] He would cancel all surgery where there is "trismus" following succinylcholine. We disagree because of the aforementioned variability in the definition of trismus, and because we have monitoring techniques which permit us to detect developing malignant hyperthermia and dantrolene to treat a malignant hyperthermic episode.

A second problem that must be addressed when masseter spasm occurs is whether or not to obtain a muscle biopsy for an MH-susceptibility contracture study. Rosenberg and Gronert recommend it in all patients who develop trismus with succinylcholine.[3,4] If truly 1% of pediatric patients develop trismus with succinylcholine and halothane, then muscle biopsy would be the most frequently performed operation in the United States. Another problem with muscle biopsy is that there are a limited number of centers doing it,[15] and it would require great time and expense for families to travel to these centers.

There is an ongoing debate about the role of succinylcholine in the armamentarium of the anesthesiologist. There is no question that every drug has a risk/benefit ratio. There is also no question that there are certain risks from succinylcholine that are infrequent, recognizable, and treatable. On the other hand, there are certain benefits from succinylcholine that no other muscle relaxant possesses. These benefits are 1) rapid onset, 2) rapid offset, and 3) effectiveness following intramuscular injection. It is evident that the "ideal" muscle relaxant is not yet available for clinical practice. Until such time, the anesthesiologist must weigh the risk/benefits of all of the muscle relaxants, just as is done with every other drug that is used. This is the basis for the "art" of anesthesia.

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In Reply: The letter from Drs. Barry and Lynch succinctly raises many questions plaguing clinicians concerning the management of trismus following succinylcholine. Based on the findings that approximately 50% of patients with masseter spasm are truly MH susceptible on contracture testing,[1] and also Van Der Spek's work[2] showing that succinylcholine may increase muscle tone of the masseter muscle in normal patients, it is certainly clear that increased muscle tone after succinylcholine may or may not be related to MH. Whether MH can be differentiated from this hypertonic response found in normals by quantitatively measuring the extent of muscle tone increase has not yet been explored. Clearly, therefore, there is a subjective element to diagnosing trismus.

Again, because of the subjective nature of trismus, the incidence of trismus has been difficult to establish with accuracy. Unfortunately, studies of the incidence of trismus are retrospective. The published studies indicate a range of incidence of 1% in children following halothane and succinylcholine[5] to 1 in 12,000 in the Danish population.[4] I suspect that the much lower incidence of trismus in Charlottesville is in part related to the mode of administration of succinylcholine. I understand the im route of administration of succinylcholine is more

common in that institution, whereas the published studies focused on intravenous administration.

My reasons for advocating discontinuing the anesthetic after an episode of trismus are stated in the editorial in ANESTHESIOLOGY.[5] I also feel that serial postoperative CKs should be performed in all cases of trismus. We have found that, where the CK is over 20,000, MH is diagnosed with regularity on contracture tests.[6] In some cases, underlying myopathies have been found when the CK does not return to normal within a short period of time.

It is true that a limited number of centers perform muscle biopsies (however, this number is growing each year), and time and expense is involved. However, given the confusion as to whether trismus may imply malignant hyperthermia, failure to biopsy will frequently lead to the patient being labeled as MH susceptible; therefore, he or she, along with other family members, would require "special" care. In some cases, this entails preoperative dantrolene administration and the inconvenience of being referred to only selected hospitals and individuals willing to anesthetize an MH susceptible patient.

Dr. Berry also feels that monitoring techniques are available to detect MH early after its onset. Not all hospitals and not all operating rooms are equipped with end-tidal CO_2 monitoring nor with the availability of venous and arterial blood gas measurement within a reasonable period of time. Indeed, not all facilities have dantrolene on hand![7]

Finally, I certainly agree with Dr. Berry's statement regarding weighing risk/benefit ratios each time we use a drug during anesthesia. It is clear that every "expert" will draw his/her own conclusions regarding the meaning of trismus and the clinical implications of this sign. This problem will continue to plague clinicians until such time as a reliable noninvasive diagnostic test for MH is developed, an understanding of the pathophysiology of trismus and malignant hyperthermia is achieved and/or a satisfactory substitute for succinylcholine is introduced into clinical practice.

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An Infant Model to Facilitate Endotracheal Tube Fixation in the Pediatric ICU Patient

To the Editor:—As pediatric anesthesiologists, we recognize and share the concerns others have expressed regarding inadvertent tracheal extubation in infants and children. These patients are particularly at risk because of their inability to cooperate and because of the short length of the infant trachea. A 13% incidence of "spontaneous" tracheal extubation has been documented in one pediatric ICU setting.[1] Others

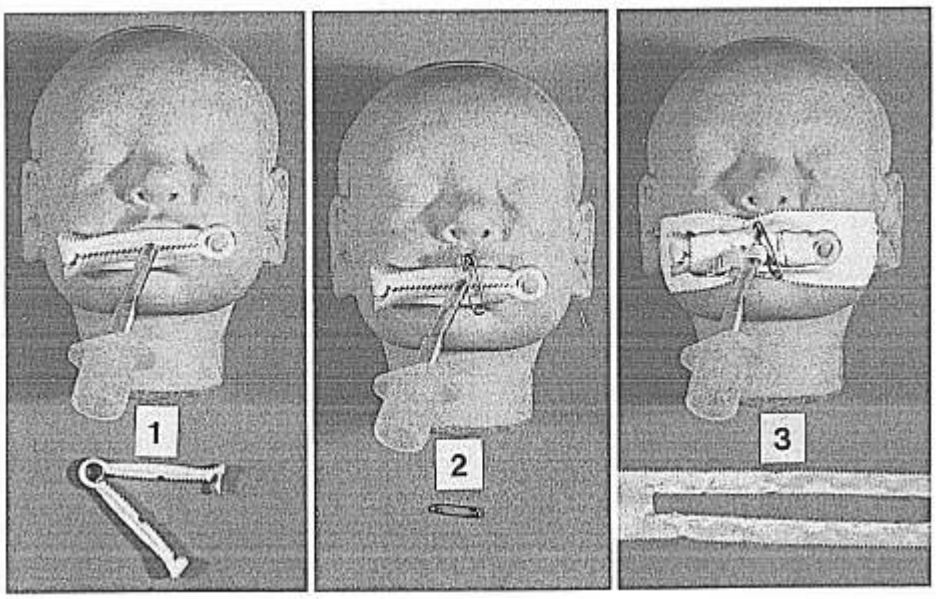

FIG. 1. The infant model for orotracheal tube fixation. The components used to secure the orotracheal tube are numbered in order of their application.