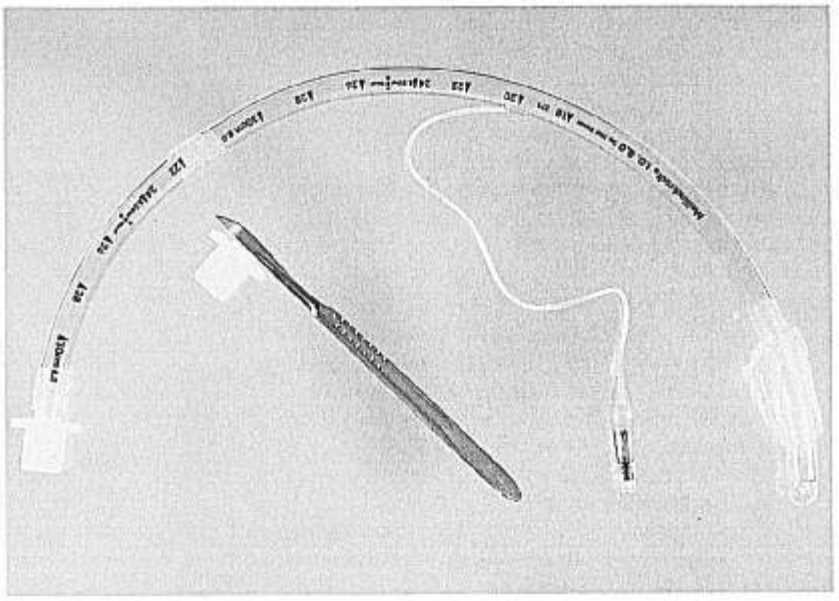

FIG. 1. Extended tracheal tube illustrating site of incision at the 15-mm connector, and location of the cut segment within the modified tube. This part is used as a bridge from the proximal original tracheal tube to the extension segment of a second tracheal tube.

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The Relationship Between Malignant Hyperthemia and Neuroleptic Malignant Syndrome

To the Editor:—During the past year, two somewhat contradictory articles,[1,2] addressing the possible relationship between malignant hyperthemia (MH) and neuroleptic malignant syndrome (NMS) have been published. These two iatrogenic highly fatal hyperthemic syndromes are clinically very similar and a common pathophysiology has been suggested.[1,3] Therefore, some have warned that patients with a history of NMS might be at a greater risk for developing MH when exposed to volatile anesthetics.[1,3] The risk of developing MH has also been suggested in susceptible NMS patients undergoing electroconvulsive therapy (ECT), a procedure involving succinylcholine administration just prior to every repeated electrical stimulation.[3,4] Based on their findings of enhanced *in vitro* response of skeletal muscle to halothane in NMS patients, Carrof *et al.*[1] suggest an association between the two syndromes, and imply that a conservative approach, avoiding triggering agents (*e.g.*, halothane, succinylcholine), should be taken during anesthesia of known NMS patients. Since NMS is not very rare (0.5–1.4% of neuroleptic exposures),[5] and as major psychiatric disorders are quite common, annual incidence of new NMS cases might exceed thousands each year. Denying these many patients life-saving surgery or ECT is a very crucial decision and should be based on a convincing body of evidence. In order to elucidate the actual risk for MH in patients who suffered from NMS, we undertook a retrospective study of their surgical and ECT histories.[6] We recruited 20 patients who fulfilled Levenson's criteria for definite diagnosis of NMS and inquired about their surgical and ECT history. Nine of the NMS patients had 12 uneventful operations involving the administration of succinylcholine and halothane. Twelve of the patients had received ECT, including five patients who were safely treated with ECT for their NMS episodes. In no case did complications ensue, despite 147 iv administrations of succinylcholine in a dose range of 15–30 mg. This outcome is corroborated by published reviews of this issue.[2,6] In summary, our retrospective study, as well as another recent *in vitro* study,[7] did not show significant cross vulnerability for MH in post-NMS patients, and leads us to conclude, in contrast to Carrof *et al.*,[1] that NMS patients are not at a considerable risk for fatal consequences of MH.

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In Reply:—We appreciate the interest of Dr. Hermesh and associates in our article on the relationship between neuroleptic malignant syndrome (NMS) and malignant hyperthermia (MH).[1] In our study, we found *in vitro* contracture responses consistent with MH-susceptibility in five of seven NMS patients. These results resemble those of Araki *et al.*[2] who reported MH-like caffeine contractures in skeletal muscle from six of eight NMS patients. In contrast, Krivosic-Horber *et al.*[3] reported one MH-equivocal and five MH-negative contracture responses in muscle from six NMS patients. As discussed in our article, this disparity of findings may reflect the lack of standardization of methodology or a lack of specificity of *in vitro* contracture testing in muscle taken from patients with neuromuscular disorders. Other studies have shown similar abnormalities in the contracture response to halothane and caffeine among patients with myopathic or neurogenic disorders.[4] Further controlled investigations may clarify whether these responses imply clinical MH-susceptibility or represent non-specific effects of diseased muscle.

As reviewed in our article, several lines of evidence suggest that NMS and MH are triggered by different mechanisms that culminate in a similar dantrolene-responsive hypermetabolic state. This is supported by the attenuation of MH in swine by neuroleptics, the ameliorating effect of depolarizing muscle relaxants and centrally active dopamine agonists in NMS, the absence of familial susceptibility to MH in NMS patients, and reports of the safe use of triggering anesthetics in NMS patients.[1] Based on a retrospective review of the uneventful use of succinylcholine with and without halothane in 20 NMS patients, Hermesh *et al.* provide additional evidence against MH-susceptibility in NMS patients. However, it would be important to know more about these procedures, *e.g.*, the duration and dosages of all drugs administered, monitoring techniques, perioperative complications, etc. Furthermore, in retrospective studies, up to 70% of MH-susceptible patients did not develop hyperthermia during general anesthesia administered on previous occasions.[5]

In terms of anesthetic management, there is certainly no reason to withhold life-saving surgery from either NMS- or MH-susceptible patients. In addition, the risk of MH in a recovered NMS patient during ECT seems negligible, since MH has never been reported as a complication of this procedure in any patient. This may be due to the brief exposure to succinylcholine, the absence of inhalational agents, or the suppressive effect of barbiturates used for anesthesia during ECT.[6] Nevertheless, a careful approach to anesthesia may be warranted, especially for patients in the midst of NMS episodes, since these metabolically unstable patients may be at risk for anesthetic-related complications apart from MH.[7,8]

Additional studies that correlate *in vitro* testing and use of anesthesia in NMS patients may be worthwhile in determining anesthetic risk associated with NMS and the potential for cross-reactivity between MH and NMS.

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