

The Effects of Succinylcholine on Mouth Opening

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Mouth opening and the resistance to opening developed by the muscles of mastication were measured in 63 children anesthetized with halothane and relaxed with succinylcholine, pancuronium, or vecuronium. Measurement of mouth opening, induced by a constant test force, was made when each patient was deeply anesthetized, as judged by clinical parameters. Succinylcholine, vecuronium, or pancuronium was then administered. The mouth opening measurement was repeated immediately after the loss of limb muscle twitch response and 45 s following the loss of twitch response. For the 24 patients receiving succinylcholine, there was a significant reduction in mean mouth opening ($P < 0.0001$) and a significant increase in jaw stiffness ($P < 0.0001$) immediately after limb relaxation. Forty-five seconds after full limb relaxation was attained, the mean mouth opening was still reduced ($P < 0.0001$) and the mean jaw stiffness was still increased ($P < 0.0003$) in the succinylcholine group. Patients receiving either vecuronium or pancuronium did not show a significant change of mouth opening or jaw stiffness following limb relaxation. Three patients, who received succinylcholine, required several attempts at tracheal intubation due to increased resistance to mouth opening. Anesthesia and surgery proceeded in all patients. None of the patients developed malignant hyperthermia. In view of the fact that a reduction in mouth opening was a constant finding when succinylcholine was administered during halothane anesthesia, the assumption that isolated "masseter spasm" or jaw stiffness heralds malignant hyperthermia should be reconsidered. (Key words: Anesthetics, volatile: halothane. Complications: masseter spasm. Malignant hyperthermia. Muscles: masticatory. Neuromuscular relaxants: pancuronium; succinylcholine; vecuronium.)

INABILITY TO ADEQUATELY open the patient's mouth when succinylcholine is used after induction of general anesthesia has been noted clinically; the increased resis-

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tance to mouth opening has been called "masseter spasm."¹ It has been postulated that "masseter spasm" is associated with malignant hyperthermia (MH) and that it heralds MH.^{1,2} "Masseter spasm" occurs in patients of all ages, but most commonly in children, in whom general anesthesia is often induced with halothane followed by succinylcholine to facilitate tracheal intubation. Succinylcholine, a depolarizing neuromuscular relaxant, induces flaccid paralysis in human limb muscles, but also a persistent increase in tone of extraocular and other mammalian muscles.³ The response of the human muscles of mastication to succinylcholine is unknown. This study was undertaken to assess the effects of succinylcholine administration on the stiffness of the muscles of mastication during deep halothane anesthesia.

Materials and Methods

The following protocol was approved by the institutional review committee for human experimentation of the University of Michigan. Patients with ASA physical status I or II, who were admitted to the University of Michigan Children's Hospital for an elective surgical procedure requiring general anesthesia, were selected for this study. Patients with abnormal cranio-facial morphology, temporomandibular joint (TMJ), or muscle disease were excluded. General anesthesia was administered by a separate anesthesia team caring for that patient. No premedications were administered. The choice of muscle relaxant, either succinylcholine, vecuronium, or pancuronium, was at the discretion of the attending anesthesiologist. Age, height, weight, and sex were recorded.

General anesthesia was induced by inhalation of 3-5% halothane in a two-to-one nitrous oxide-oxygen mixture. The same concentrations of the anesthetics were maintained throughout the study period, unless hemodynamic compromise occurred. Electrocardiogram leads, precordial stethoscope, blood pressure cuff, and temperature probe (in the lower esophagus, rectum, and/or axilla) were applied. An intravenous infusion (iv) was started and neuromuscular blockade was monitored by transcutaneous stimulation of the ulnar nerve with supramaximal, square wave stimuli (frequency 1 Hz, 0.2 s duration). When a clinically deep level of anesthesia with 3-5% of halothane was obtained, as determined by clinical parameters including heart rate, blood pressure, respiratory rate, tidal vol-

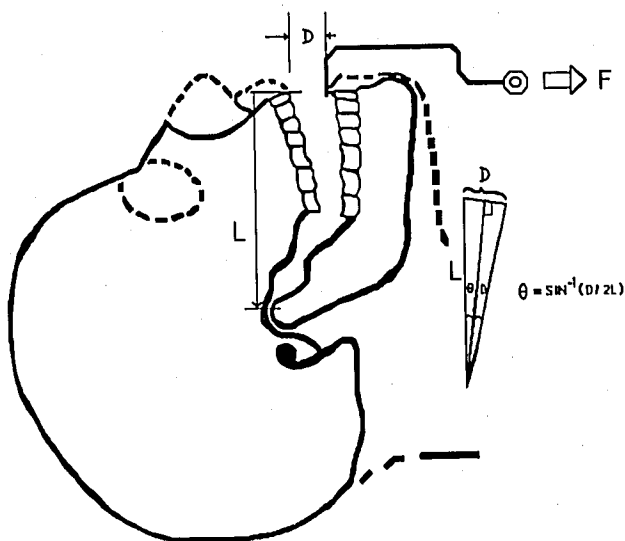


FIG. 1. A schematic of the face, lateral view, indicating the TMJ to incisor distance (L), the incisor distance (D), and the direction of the constant force F .

ume, pupil size, and time, the patient's head was placed in the sniffing position. Care was taken to maintain the same head position for all measurements. Then, at time T_1 , the mask was removed and, while the patient continued to breathe spontaneously, mouth opening was accomplished by means of a constant force (1.67 Newton) spring (Negator Motor[®]) which was connected to a traction device positioned over the lower central incisors (fig. 1). The opening force was directed in the sagittal plane at a 90° angle to a line from the maxillary incisors to the TMJ (fig. 1). The resulting mouth opening distance (D_i), expressed as the separation of the upper and lower incisors, was measured in mm in the sagittal plane using a machinist's scale, whose end was placed against the upper central incisor and whose markings lay adjacent to the lower central incisor. Parallax was avoided by sighting across two markings, separated by 10 mm. The measurement took approximately 10 s and the mask was reapplied. Subsequently, a muscle relaxant was infused over 15 s through a rubber injection port at the hub of the iv catheter. The iv continued to drip slowly into the patient, and no attempt was made to control its rate of flow. The dose of relaxant was 1, 1.5, or 2 mg/kg succinylcholine, or 0.1 mg/kg pancuronium, or 0.1 mg/kg vecuronium. Ventilation was controlled with the concentrations of inhalation agents unchanged unless hemodynamic compromise occurred. The presence of fasciculations was noted. When the visible limb muscle response to nerve stimulation was lost completely at time, T_2 , the mouth opening, D_2 , was measured as described for D_1 . Ventilation was resumed, and a third measurement, D_3 , was

taken at T_3 , 45 s after T_2 . Following the third measurement, the concentration of halothane was reduced and the trachea was intubated, typically 90 s after T_2 . The return of the first visible twitch was recorded in patients receiving succinylcholine; the end tidal CO_2 was monitored after tracheal intubation in most patients. Anesthesia with halothane was further conducted as indicated by the operation.

The resistance to mouth opening arises from the muscles of mastication and connective tissues. In order to assess this resistance, jaw stiffness K_i , the rotational resistance to mouth opening under an applied test moment M , was calculated for each mouth opening D_i .⁴ Jaw stiffness (K_i) was calculated from the formula:

$$K_i = M \cdot (2 \cdot \theta)^{-1} = F \cdot L \cdot (2 \cdot \sin^{-1}(D_i \cdot (2 \cdot L)^{-1}))^{-1},$$

where M equals the applied test moment ($F \cdot L$) about the TMJ, θ is the half angle of mouth opening from the fully closed position, F is the constant test force exerted on the traction device, L is the distance in the sagittal plane from the anterior surface of the condyle of the TMJ to the edge of the central maxillary incisors measured using a ruler to the nearest mm, D_i equals the distance between the upper and lower central incisors, and i represents the index of the corresponding mouth opening (fig. 1).

STATISTICAL ANALYSIS

The differences of mouth opening and jaw stiffness, before and after relaxation, were compared in the same subject within each relaxant group using the two-tailed, paired Student's t test. In these comparisons, Bonferroni's correction for multiple statistical tests on the same sample was used and is reported as p_b in the text as well as in tables 1-4. Multi-factorial repeated measures analysis of variance (rm-ANOVA) was used to assess the overall effects of the three relaxant groups (the between-group factor), of the repeated measures, D_i and K_i (the within-group factors) and of their interaction on the mouth opening, and jaw stiffness. Analysis of variance (ANOVA) was used to compare the patient population of the three relaxant groups (with respect to age, height, weight, sex, L , D_1 , and K_1), as well as to compare the between-relaxant-group differences in mouth opening and jaw stiffness for each D_i and K_i and their respective changes over time (D_2-D_1 , D_3-D_1 , and K_2-K_1 , K_3-K_1). The Scheffe multiple comparison approach (F -test) is applied for each treatment comparison for the ANOVAs, and is supplied in tables 2 and 3.

Results

The groups of patients ($N = 24$) receiving succinylcholine did not differ significantly from the groups re-

TABLE 1. Comparison of Patient Groups Receiving Succinylcholine (Suc), Pancuronium (Pan), or Vecuronium (Vec)

	N	Age	Height	Weight	L	D ₁	K ₁	FM
Suc	24	6.1 ± 4.2	116 ± 29	24.6 ± 17.7	77 ± 9.1	19.6 ± 3.5	88.3 ± 16.7	14:10
Pan	16	7.7 ± 4.9	128 ± 27	29.4 ± 14.9	75 ± 7.5	17.8 ± 3.9	98.1 ± 21.6	10:6
Vec	23	6.9 ± 5.2	118 ± 31	25.8 ± 15.7	75 ± 9.5	19.1 ± 4.7	93.2 ± 26.5	8:15

The three relaxant groups with their age in years, height in cm, weight in kg, sex in ratio female to male, D₁ in mm, L in mm, and K₁ in Nm/degree. All values are expressed as the mean ± standard deviation.

ANOVA indicated no statistically significant differences between the relaxant groups for these variables.

ceiving pancuronium (N = 16) or vecuronium (N = 23) with respect to age, weight, height, sex, L, D₁, and K₁ (ANOVA) (table 1).

After limb relaxation with succinylcholine at T₂, the mean mouth opening was significantly reduced by 24% from 19 to 15 mm, and mean jaw stiffness was significantly increased by 38% from 88.1 to 123.8 Nm/degree (p_b < 0.0002) (tables 2-4). At T₃, the mean mouth opening and jaw stiffness of the succinylcholine group were reduced to 14 mm and increased to 137.1 Nm/degree, respectively; a significant difference from values at T₁ (p_b < 0.001). When the values at T₃ of the succinylcholine group were compared to those at T₂, ten patients demonstrated a further decrease in mouth opening and an increase in jaw stiffness at T₃, and ten patients showed no change in mouth opening and jaw stiffness from those at T₂. In three patients, the mouth opening increased and jaw stiffness reduced but did not return to the levels obtained at T₁, while, in one patient, mouth opening and jaw stiffness had returned to its base line values at T₁. No significant differences in mean mouth opening (D₂, D₃) and jaw stiffness (K₂, K₃) at T₂ or T₃ occurred in the patients receiving pancuronium or vecuronium when compared to their base line values (D₁, K₁) at T₁, (P < 0.05) (tables 2, 3).

The results of the comparisons of D_i and K_i between subjects, before and after relaxation, between the relaxant groups are as follows. The two-factor rm-ANOVA for mouth opening demonstrated that the interaction between the two factors (relaxants, repeated measures of D_i) was significant (F = 35.5, P < 0.0001), while that analysis for jaw stiffness demonstrated that the interaction between the two factors (relaxants, repeated measures of K_i) was also significant (F = 14.9, P < 0.0001). Because these interactions were observed, further analysis of the repeated measures (the within-group factor) and of the relaxant groups (the between-group factor) was required. The within-group, intra-subject comparisons have already been addressed with the paired Student's *t* test above. The ANOVA was used to assess differences in means for mouth opening (D_i) and jaw stiffness (K_i) and their respective changes over time (D₂-D₁, D₃-D₁, and K₂-K₁, K₃-K₁) among the three relaxant groups; the Scheffe F-tests for each treatment are also reported in the tables where applicable, (tables 2, 3). The mean D₁ and K₁ among the three relaxant groups did not differ significantly. The mean D₂ and D₃ among the three relaxant groups differed. Pairwise comparison among relaxant groups using Scheffe confidence intervals showed significance for D₂

TABLE 2. Mean Mouth Opening D_i in mm Before and After Relaxant Administration

Muscle Relaxant (N)	D ₁	D ₂	D ₃	D ₂ -D ₁	D ₃ -D ₁
Succinylcholine (24) 1.0-2.0 mg/kg	19 ± 3.5	15 ± 4.4†	14 ± 4.8‡	-4.7 ± 2.5*§	-5.4 ± 3.4*¶
Pancuronium (16) 0.1 mg/kg	18 ± 3.9	18 ± 3.0	18 ± 3.1	+0.0 ± 1.8	+0.3 ± 1.9
Vecuronium (23) 0.1 mg/kg	19 ± 4.7	19 ± 4.9	19 ± 4.9	+0.3 ± 1.5	+0.5 ± 1.3
ANOVA	F = 0.9 P = 0.4	F = 6.5 P < 0.003	F = 9.5 P < 0.0004	F = 43.2 P < 0.0001	F = 41.5 P < 0.0001

The mean distance of mouth opening in patients receiving succinylcholine, pancuronium, and vecuronium during deep halothane anesthesia prior to the administration of muscle relaxant at T₁ (D₁), immediately after relaxation at T₂ (D₂) and 45 s after full relaxation at T₃ (D₃). T₁, T₂, and T₃ are defined in the text. Values are the mean ± standard deviation expressed in mm. N represents the number of subjects. Two-factor rm-ANOVA demonstrated a significant interaction between the repeated measures (D_i) and the relaxant groups (F = 35.5, P < 0.0001).

* Indicates intra-subject difference significant at the P < 0.0001 (pb < 0.0002) level by paired Student's *t* test within the succinylcholine group.

Other superscript symbols indicate significant differences in means between relaxant groups demonstrated by the ANOVA:

† Corrected pairwise comparison (Scheffe F-test), suc versus vec—significant at the 99% level.

‡ Corrected pairwise comparison (Scheffe F-test), suc versus vec—significant at the 99% level; suc versus pan—significant at the 95% level.

§ Corrected pairwise comparison (Scheffe F-test), suc versus vec and pan—significant at the 99% level.

¶ Corrected pairwise comparison (Scheffe F-test), suc versus vec and pan—significant at the 99% level.

TABLE 3. Mean Jaw Stiffness K_i in Nm/degree Before and After Relaxant Administration

Muscle Relaxant (N)	K_1	K_2	K_3	K_2-K_1	K_3-K_1
Succinylcholine (24) 1.0-2.0 mg/Kg	88.1 ± 16.6	123.8 ± 44.8‡	137.1 ± 66.0§	+35.7 ± 32.5*¶	+49.0 ± 56.4†**
Pancuronium (16) 0.1 mg/Kg	98.1 ± 21.3	96.6 ± 16.6	94.9 ± 14.7	-1.5 ± 12.6	-3.1 ± 13.6
Vecuronium (23) 0.1 mg/Kg	93.4 ± 26.9	91.7 ± 25.1	89.9 ± 22.4	-1.7 ± 6.4	-3.4 ± 7.9
f-ANOVA	F = 1.0 P = 0.4	F = 6.4 P < 0.004	F = 7.9 P < 0.001	F = 22.6 P < 0.0001	F = 15.8 P < 0.0001

The mean jaw stiffness in patients receiving succinylcholine, pancuronium and vecuronium during deep halothane anesthesia prior to the administration of the muscle relaxant at T_1 (K_1), immediately after relaxation at T_2 (K_2) and 45 s after full relaxation at T_3 (K_3). The values are the mean ± standard deviation expressed in Nm/degree. Two-factor rm-ANOVA demonstrated a significant interaction between the repeated measures (K_i) and the relaxant groups ($F = 14.8$, $P < 0.0001$).

* Indicates intra-subject difference significant at the $P < 0.0001$ ($pb < 0.0002$) level by paired Student's t test within the succinylcholine group.

† Indicates intra-subject difference significant at the $P < 0.0003$ ($pb < 0.001$) level by paired Student's t test within the succinylcholine

group.

Other superscript symbols indicate significant differences in means between relaxant groups demonstrated by the ANOVA:

‡ Corrected pairwise comparison (Scheffe F-test), suc *versus* vec—significant at the 99% level; suc *versus* pan—significant at the 95% level.

§ Corrected pairwise comparison (Scheffe F-test), suc *versus* vec—significant at the 99% level; suc *versus* pan—significant at the 95% level.

¶ Corrected pairwise comparison (Scheffe F-test), suc *versus* vec and pan—significant at the 99% level.

** Corrected pairwise comparison (Scheffe F-test), suc *versus* vec and pan—significant at the 99% level.

of succinylcholine *versus* that of vecuronium, and for D_3 of succinylcholine *versus* those of pancuronium and vecuronium. The mean K_2 and K_3 among the three groups also differed. Pairwise analysis for the K_2 showed significance for succinylcholine *versus* pancuronium and vecuronium, and for K_3 of succinylcholine *versus* pancuronium and vecuronium. Most importantly, the differences in means for changes in mouth opening (D_2-D_1 , D_3-D_1) and for changes in jaw stiffness (K_2-K_1 , K_3-K_1) over time among the relaxant-groups were significant when compared by ANOVA. The between-group means of D_2-D_1 and of D_3-D_1 differed significantly ($F = 43.2$, $P < 0.0001$, and $F = 41.5$, $P < 0.0001$, respectively). Further analysis of these differences showed that succinylcholine differed from pancuronium and vecuronium. The between-group means of K_2-K_1 and of K_3-K_1 differed significantly ($F = 22.6$, $P < 0.0001$, and $F = 15.8$, $P < 0.0001$, respectively), again showing that succinylcholine differed

from pancuronium and vecuronium by pairwise comparison.

The mean time of onset of complete loss of twitch in the succinylcholine group was 46 s (range 24–105 s). The first visible twitch returned after an average of 292 s (range 150–466 s). In three patients, although a marked reduction of twitch occurred after 1.5 mg/kg of succinylcholine, the twitch was never completely lost. These patients' ages were 2, 2, and 16 yr; their respective incisor distances reduced as follows: 22 to 11 to 7 mm, 20 to 15 to 17 mm, and 20 to 18 to 17 mm, while their jaw stiffness increased from 75.5 to 152.1 to 238.4 Nm/degree, 83.4 to 110.9 to 98.1 Nm/degree, and 83.4 to 92.2 to 98.1 Nm/degree, respectively. In the first of these three patients, it was difficult to intubate the trachea secondary to the reduction of mouth opening. In the other two patients, the tracheas were not difficult to intubate. Of the remaining 21 patients receiving succinylcholine who showed full limb relax-

TABLE 4. Descriptive Data of Mouth Opening and Jaw Stiffness in 24 Patients Before (D_1 , K_1) and After (D_2 , D_3 , K_2 , K_3) Succinylcholine Administration

Mouth Opening in mm	D_1	D_2	D_3	Jaw Stiffness in Nm/deg	K_1	K_2	K_3
Mean ± SD	19 ± 3.5	15 ± 4.4*	14 ± 4.8*	Mean ± SD	88.1 ± 16.6	123.8 ± 44.8*	137.1 ± 66.0†
Range	14–27	7–23	5–22	Range	62–119	73–238	76–334
Reduced by				Increased by			
Range	—	1–11	0–15	Range	—	3–127	0–214
Range %	—	4–53	0–68	Range %	—	5–114	0–214
Mean	—	4.6	5.4	Mean	—	35.7	49.0
Mean %	—	24	28	Mean %	—	38	53

* Indicates intra-subject difference significant at the $P < 0.0001$ ($pb < 0.0002$) level comparing D_1 with D_2 or D_3 and K_1 with K_2 by paired Student's t test within the succinylcholine group.

† Indicates intra-subject difference significant at the $P < 0.0003$ ($pb < 0.001$) level comparing K_1 with K_3 by paired Student's t test within the succinylcholine group.

ation, the tracheas of two were difficult to intubate because of a reduced mouth opening. Their ages were 5 and 10 yr, and their respective mouth opening distances were 15 to 7 to 6 mm and 14 to 8 to 5 mm, with respective jaw stiffnesses of 110.9 to 238.4 to 277.6 Nm/degree and 118.7 to 208 to 332.6 Nm/degree.

The range of jaw stiffness in the 63 patients before relaxation showed three-fold variation, whereas the range of jaw stiffness after succinylcholine increased to a maximum of seven-fold from base line, while little change in the range of jaw stiffness was observed after relaxation with pancuronium or vecuronium. The range of increases in jaw stiffness from T_1 to T_2 or from T_1 to T_3 after succinylcholine was 44-fold.

The reduction of mouth opening and the increase in jaw stiffness did not correlate significantly with factors such as age, weight, height, or sex. Mouth opening (D_1) and jaw stiffness (K_1) did not correlate with age, weight, height, or L.

The temperature and end-tidal CO_2 changes observed were within the range of normal values for clinical anesthesia. General anesthesia was continued with halothane in oxygen and nitrous oxide in all patients who received succinylcholine for the duration of the following surgical procedure, in spite of the reductions in mouth opening and increases in jaw stiffness. Even in the patients in whom these changes led to difficult tracheal intubation, malignant hyperthermia did not develop by clinical criteria (color of skin, limb muscle rigidity, heart rate, minute ventilation, etc.), by end-tidal CO_2 criteria (during constant minute ventilation) and by temperature criteria.

Discussion

The object of this study was to evaluate and compare the mouth opening and jaw stiffness before and after muscle relaxation administration during deep halothane anesthesia. The mouth opening and jaw stiffness responses were compared in each patient within each relaxant group using patients as their own control, so that the effect of possible differences in anesthetic depth between patients and groups was minimized. This analysis demonstrated that a significant reduction in mouth opening occurs following the administration of succinylcholine, at a time when full relaxation of the limb muscles is obtained; this mouth opening reduction represents an increase in stiffness of the muscles of mastication. Such changes were not observed after vecuronium or pancuronium, except in one case following pancuronium. The inspired concentrations of the anesthetics were kept the same within each subject; therefore, the depth of anesthesia was either the same or greater in each patient at the time of the second and third measurement.

The comparisons of mean mouth opening and jaw stiffness at T_2 and T_3 between relaxant groups demonstrated effects of succinylcholine on mouth opening and jaw stiffness different from those of vecuronium and pancuronium. Most importantly, the mean changes in mouth opening (D_2-D_1 and D_3-D_1) and jaw stiffness (K_2-K_1 and K_3-K_1) after relaxant administration were significantly different among the three muscle relaxants. Treatment with succinylcholine, as opposed to vecuronium or pancuronium, was associated with significant changes in mouth opening and jaw stiffness under the conditions of this study. For the validity of such observations, one has to assume that either the anesthetic depth was the same in all patients, or that the variation of anesthetic depth between individuals was randomly distributed; it also assumes that inhalational anesthetics affect mouth opening and jaw stiffness. Since the depth of anesthesia was clinically determined (the 3-5% halothane, clinical parameters, time, appearance of the patient) and not quantified by end-tidal agent monitoring, the deep level of halothane anesthesia may have varied among patients and may have affected the response to succinylcholine or to the other relaxants in a differential manner. If the anesthetic level of the patients in the succinylcholine group before the relaxant administration had been different (for example "lighter") from the other relaxant groups, an initial between-group difference of D_1 and K_1 should have been present; such was not the case. If the anesthesia level in the succinylcholine group had been altered (for example, decreased) after the first base line measurement, either before or during the succinylcholine administration, then the following considerations apply. At any level of anesthesia or, indeed, with no anesthesia at all, when a subject is given sufficient succinylcholine (or other neuromuscular blocking agent) and is fully relaxed, as indicated by a peripheral nerve stimulator, as well as by clinical signs (flaccid limbs, cessation of spontaneous ventilatory effort), then the mouth opening should either be the same or larger at that time. This reasoning assumes that the human masticatory musculature responds in the same fashion to succinylcholine as his limb musculature does under the anesthetic conditions of this study; our data do not support that assumption. Our data suggest that succinylcholine treatment induces a mouth opening reduction and an increase in jaw stiffness during deep halothane anesthesia, even when inter-subject variation in depth of anesthesia is present. The mouth opening after succinylcholine administration was not increased in any patient. Halothane influences neuromuscular blockade; it induces a degree of neuromuscular relaxation and potentiates neuromuscular blocking agents in general. However, the doses of neuromuscular blocking agents used

in this study are capable of producing full neuromuscular blockade of the limbs during an oxygen-nitrous oxide, thiopental-narcotic general anesthetic, *i.e.*, without the use of inhalational agents, such as halothane. Flaccid paralysis of the limbs was accomplished by the doses administered to our patients; limb paralysis would, in all probability, also have been present in the absence of halothane.

The usual response of twitch muscle fibers to succinylcholine is an initial depolarization and contraction, immediately followed by relaxation; however, often responses, such as contractures, have been described in slow "tonic" muscle fibers of mammals.^{3,5} The response of the human masticatory muscles to succinylcholine in the present study suggests that the masticatory muscles differ from human limb muscles. The onset time and duration of the twitch depression in the arm in this study is consistent with previous reports.⁶

Despite the reduction in mouth opening after succinylcholine administration, the increase in jaw stiffness by a mean of 38–53% was usually easily overcome during routine intubation. Occasionally, a marked reduction of mouth opening and a marked increase in jaw stiffness may lead to difficulty with insertion of the laryngoscope blade and exposure of the larynx during tracheal intubation. Although the measurements by Chilcoat *et al.*, referred to in the paper by Rocco *et al.*, are not directly comparable, a ten-fold increase from their lowest intubation force could be generated during tracheal intubation.^{7,8} ††

The investigators were aware of the fact that 3–5% halothane was used, that the patient was deeply anesthetized as judged clinically by the anesthesia team, and that the same concentrations of inhalation agents were maintained throughout the study. Blinding of the investigators with respect to the type of relaxant administered was difficult. The signs and symptoms associated with succinylcholine, as opposed to vecuronium or pancuronium, administration during deep halothane anesthesia consisted of the following: fasciculations (60% of the succinylcholine group, none in the other groups), extremity movement (only in the succinylcholine group), rapidity of the loss of twitch (81% of the succinylcholine group lost their twitch in less than 60 s, whereas only 13% [three cases] of the vecuronium group and 12% [two cases] of the pancuronium group had lost their twitch at that time—60 s, and the association of cardiac dysrhythmias especially bradycardia (only in the succinylcholine group).

In the reports of cases noting a reduction in mouth opening and difficulty with intubation, a common element appears to be induction of anesthesia with halothane followed by succinylcholine.⁹ In recent years, several reports have suggested that a reduction of mouth opening and subsequent difficulty with intubation is the result of "spasm" of the masseter muscles. It was further suggested that such "masseter spasm" could be an expression of malignant hyperthermia.^{1,2} In these reports, "masseter spasm" is usually not defined other than the statement that it was difficult to open the mouth and/or difficult to intubate the trachea. The resemblance of jaw rigidity, or "masseter spasm," to generalized rigidity during a full-blown episode of MH may have led to this interpretation. Generalized muscle rigidity during anesthesia in humans is noted in a number of diseases, including myotonias and MH; the latter is accompanied by additional signs and symptoms. "Isolated" increases in muscle tension of normal muscle, that is, increases in tension without other signs or symptoms, have been documented in the extra ocular muscles of humans in response to succinylcholine.¹⁰

There is considerable debate regarding diagnostic criteria and disease heterogeneity of MH.^{11,12} ††† Recent reports associate "masseter spasm" with malignant hyperthermia in 33–100% of cases after muscle biopsy tests were consistent with malignant hyperthermia susceptibility (MHS).^{9,13,14} In one study, this led to a presumptive diagnosis of MHS in one per 100 children undergoing halothane anesthesia followed by succinylcholine administration.⁹ Tests for MHS include contracture testing on living muscle, platelet bioassays, and calcium uptake measurements in thawed specimens from deep frozen muscle. The latter two tests are controversial, and their validity remains in question.^{12,15} ††† The techniques and criteria of contracture testing varies from center to center.^{11,12} ††† An attempt to standardize testing techniques and criteria, as well as a call for more control biopsies, has recently come forth.^{12,16,17}

Although all patients in this study who received halothane and succinylcholine developed a reduction in mouth opening and an increase in jaw stiffness, even leading to difficulty with tracheal intubation, none of these patients developed malignant hyperthermia as judged by increases in CO₂ production, temperature elevation, or other clinical criteria. None of the patients were treated with dantrolene sodium and anesthesia and surgery continued in excess of 1 h in the majority of patients.

†† Chilcoat RT, Senior Biomedical Engineer, B.O.C. Group Technical Center, Healthcare R & D, Murray Hill, NJ, 07974: Personal Communication.

††† The Fourth International Malignant Hyperthermia Workshop, Sep. 1986, York, England.

This study shows that an increase in jaw stiffness and a reduction in mouth opening, without other signs or symptoms, may be a normal pharmacological response to succinylcholine given during deep halothane anesthesia. The marked increase in jaw stiffness observed in some patients may represent one end of the distribution of the response of the normal population to succinylcholine, and should not necessarily be regarded as an abnormal variant. The suggestion that isolated "masseter spasm" or jaw stiffness heralds malignant hyperthermia should be reconsidered.

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