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Increased Masticatory Muscle Stiffness during Limb Muscle Flaccidity Associated with Succinylcholine Administration

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The resistance of mouth opening to a constant force of 1.7 N was measured in 44 pediatric subjects anesthetized with enflurane and paralyzed with succinylcholine or vecuronium. Measurements were made during a deep level of anesthesia before relaxant administration, immediately after the loss of the adductor pollicis muscle twitch and 45 s later. In 22 patients receiving succinylcholine, there was a significant reduction in mean mouth opening (from 16.9 ± 2.8 to 12.6 ± 4.3 to 13.0 ± 4.3 mm; $P < 0.0005$) and an increase in jaw stiffness (from 102.3 ± 21.9 to 154.5 ± 77.4 to 150.5 ± 77.0 Nm/degree; $P < 0.02$) immediately after disappearance of the evoked thenar muscle twitch, as well as 45 s later. In six patients receiving succinylcholine, measurements were continued at 1 min intervals; mouth opening reduction and jaw stiffness increase lasted up to 10 min and extended beyond the return of visible twitch. One patient had a reduction of mouth opening from 20 to less than 1 mm; his corresponding jaw stiffness changed from 83.4 to 3335.4 Nm/degree. This patient, considered by us to have masseter spasm, required several attempts at tracheal intubation due to an increased resistance to mouth opening, as did one other patient. Patients receiving vecuronium showed a significant ($P < 0.02$) increase of mouth opening 45 s following loss of twitch (from 19.8 ± 3.6 to 20.9 ± 4.1 mm; jaw stiffness changed from 87.0 ± 15.3 to 83.0 ± 17.2 Nm/degree). Anesthesia and surgery proceeded normally; in most patients, in excess of 1 h. None of the patients developed a hypermetabolic state or hyperthermia. The changes in jaw muscle tone following succinylcholine administration are not consistent with agonist effects of succinylcholine on striated twitch muscle fibers. (Key words: Anesthetics, volatile: enflurane. Complications: masseter spasm. Malignant hyperthermia. Muscles: masticatory. Neuromuscular relaxants: succinylcholine; vecuronium.)

IN A PREVIOUS STUDY in humans, an unexpected reduction of mouth opening and increase in jaw stiffness during halothane anesthesia was associated with administration of succinylcholine; mouth opening was either unchanged or increased following pancuronium or vecuronium administration, while jaw stiffness stayed the same or was reduced.[1] This unexpected response following succinylcholine occurred at a time when limb musculature was clinically flaccid and peripheral nerve stimulation demonstrated loss of thenar muscle twitch. Clinical reports of masseter spasm usually concern pediatric patients receiving succinylcholine during halothane anesthesia. Given the different neuromuscular effects of halothane and enflurane, as well as the paucity of clinical reports of masseter spasm during enflurane anesthesia, the objective of this study was to examine the effects of succinylcholine on mouth opening and jaw stiffness during enflurane anesthesia in a pediatric population. The onset and duration of fasciculations and the loss and return of the adductor pollicis muscle twitch were examined with respect to the changes in the jaw musculature.

Materials and Methods

The protocol was approved by the University of Michigan's review committee for human experimentation. Forty-four subjects, 2–13 yr of age and ASA physical status I or II, who required general anesthesia and endotracheal intubation for an elective surgical procedure, were selected for this study. Patients with known temporomandibular joint (TMJ) disorders, muscle disease, or craniofacial disproportions were excluded from this study. General anesthesia was not administered by the investigators, but by a separate anesthesia team responsible for the patient. The methods of measurement in the current project were similar to those described in a previous study, and are here given in a condensed form.[1]

Patients were not premedicated, and anesthesia was induced with an oxygen/nitrous oxide mixture with increasing concentrations of enflurane. The concentrations were maintained at 3–5% inhaled, depending on the individual patient, until a level of anesthesia was reached at which the patient's trachea could, in the judgement of the anesthesia team, be intubated. This level was determined from clinical parameters, including heart rate, blood pressure, respiratory rate, tidal volume, pupil size, and time, as well as from the patient's response to stimuli, such as the percutaneous insertion of an iv catheter and manipulation of the jaw (direct laryngoscopy) without evoking movements or changes in respiratory rate and depth. This level of anesthesia was usually reached after 8–14 min. The con-

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centration of enflurane was continued at the same percentage in each patient until the end of the measurement period, unless hemodynamic compromise occurred. During the time of induction, electrocardiogram leads, precordial stethoscope, blood pressure cuff, and temperature probe were applied. An intravenous infusion (iv) was started and neuromuscular blockade was monitored visually by the evoked twitch of the adductor pollicis muscle during transcutaneous stimulation of the ulnar nerve with supramaximal, square wave stimuli (frequency 1 Hz, 0.2 ms duration). The visual twitch was monitored by an observer (not an investigator) who was blinded to the type of anesthesia and muscle relaxant. Once the deep level of anesthesia was attained, the patient's head was placed in the sniffing position. Care was taken to place the head consistently in the same 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 (Neg'ator Motor™) that was connected to a traction device positioned over the mandibular central incisors. The opening force was applied in the midsagittal plane perpendicular to the line from the maxillary incisors to the TMJ. The resulting mouth opening (D_1), defined as the separation of the maxillary and mandibular incisors, was measured in mm in the midsagittal plane using a machinist's scale. Parallax was minimized by sighting across two sets of scale markings separated by 10 mm. The measurement took approximately 10–15 s and the mask was reapplied. Subsequently, the muscle relaxant was infused over 15 s. The choice of muscle relaxant, either succinylcholine (1.5 mg/kg iv) or vecuronium (0.1 mg/kg iv), was at the discretion of the anesthesia team. The investigators were not blinded with respect to the type of relaxant administered, since signs and symptoms associated with succinylcholine (fasciculations, extremity movement, dysrhythmias, and the rapidity of the loss of twitch), as opposed to vecuronium administration, would reveal the relaxant's identity in the majority of cases.[1] Ventilation was controlled. The presence of fasciculations was noted. When the visible adductor pollicis muscle response to nerve stimulation was lost completely at time, T_2, the second mouth opening, D_2, was measured as described for D_1. Ventilation was resumed and a third measurement, D_3, was made at T_3, 45 s after T_2. Following the third measurement, the tracheas of most patients were intubated, typically 90 s after T_2. However, in those patients whose mouth opening was reduced by 50% or more at either T_2 or T_3 compared to baseline at T_1, intubation was not attempted and measurements were continued at 1-min intervals until either the mouth opening had returned to within 2 mm from the baseline or 10 min had elapsed. The time to return of the first visible twitch was recorded in patients receiving succinylcholine. End-tidal CO_2 was monitored after tracheal intubation in most patients, except for those patients who had a pronounced mouth opening reduction. In these subjects, an end-tidal CO_2 monitor was used during the continued controlled ventilation by mask. Anesthesia with enflurane was further conducted as indicated by the operation.

For the i^{th} ($i = 1,2,3$) mouth opening D_i, the jaw stiffness K_i, the rotational resistance to mouth opening under an applied test moment M, was calculated and is expressed in 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 refers to the applied test moment and θ to the half angle of mouth opening from the fully closed position; F represents the constant test force exerted by 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; and D_i refers to the mouth opening in mm.

Analysis of variance (ANOVA) was used to compare the age, height, weight, L, D_1, and K_1 of the two relaxant groups. Two-factor repeated measures analysis of variance (rm-ANOVA) was used to assess the overall effects on mouth opening and jaw stiffness of the two muscle relaxants (the between-group factor), the repeated measures, D_i and K_i (the within-group factor), and their interaction. When interactions were observed, further analysis of the repeated measures (the within-group factor) and of the relaxant groups (the between-group factor) was undertaken within levels of the other factor. Changes in mouth opening and jaw stiffness following muscle relaxant administration were compared within each relaxant group separately, using the one-population repeated measures ANOVA (one-population rm-ANOVA). For each analysis, the overall test statistic is followed by the attained significance levels, corrected for multiple pairwise comparisons. ANOVA was used to compare the differences in mouth opening and jaw stiffness among the succinylcholine and vecuronium groups for each D_i and K_i, as well as for their respective absolute and fractional changes [D_2-D_1, D_3-D_1, (D_1-D_2)/D_1, (D_1-D_3)/D_1 and K_2-K_1, K_3-K_1, (K_1-K_2)/K_1, (K_1-K_3)/K_1]. Analyses of covariance (ANCOVA) using baseline values (D_1 or K_1, respectively), were completed for analyses of D_2, D_3, K_2, and K_3, thereby adjusting for differences in baseline values among the two groups. Because of uncertainty regarding the distribution of mouth opening distances, nonparametric analysis using the Mann-Whitney U test was also performed. Furthermore, because of the differ-

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

	N	Age	Height	Weight	L	D_1	K_1	F:M
Suc	23	5.7 ± 3.4	116.9 ± 22.7	23.6 ± 12.2	72.1 ± 7.5	17.0 ± 2.8*	101.5 ± 21.8†	9:14
Vec	21	7.0 ± 3.5	123.5 ± 22.3	25.8 ± 12.9	74.5 ± 8.4	19.8 ± 3.6	87.0 ± 15.3	6:14

The two relaxant groups with their age in years, height in cm, weight in kg, the temporomandibular joint to maxillary incisal distance L in mm, the baseline mouth opening D_1 in mm, the baseline jaw stiffness K_1 in Nm/degree and the female to male ratio. All values are expressed as the mean ± standard deviation. ANOVA indicated no significant differences between the relaxant groups for these variables except for D_1 and K_1, which differed significantly (*$F = 8.0$, $P < 0.007$ for D_1; †$F = 6.4$, $P < 0.015$ for K_1).

ences in magnitude of the standard deviations of jaw stiffness between the two relaxant groups, the data were logarithmically transformed and the above analyses were again executed.

Results

The group of patients receiving succinylcholine (N = 23) did not differ significantly from the group receiving vecuronium (N = 21) with respect to age, weight, and height (ANOVA). However, the two groups differed significantly with respect to the baseline mouth opening (D_1) and jaw stiffness (K_1) (table 1). Two-factor rm-ANOVA for mouth opening demonstrated a significant ($P < 0.0001$) interaction between the relaxants and the repeated measures (D_i) factors, while this analysis for jaw stiffness demonstrated a non-significant ($P < 0.18$) interaction between the relaxants and the repeated measures (K_i) factors. This discrepancy between mouth opening and jaw stiffness could be accounted for by one outlying case in which the mouth opening was reduced to less than 1 mm and jaw stiffness was increased over 35-fold following succinylcholine administration. The standard deviation for jaw stiffness of the succinylcholine group was thus 100 times that of the vecuronium group. The data analysis is, therefore, given without this outlier. Thus, two-factor rm-ANOVA for mouth opening, as well as for jaw stiffness, demonstrated a significant interaction ($P < 0.0001$) between the two factors.

Using each patient as her/his own control (one-population rm-ANOVA), mean mouth opening in the succinylcholine group was significantly reduced after the loss of twitch ($P < 0.0001$), as well as 45 s later ($P < 0.0005$) with comparisons made with respect to the initial mouth openings, D_1 (table 2). Mean jaw stiffness increased concomitantly (table 3). In contrast, patients who received vecuronium developed a significant ($P < 0.05$) increase in mean mouth opening at T_3; jaw stiffness did not change significantly (tables 2, 3). When the patients receiving succinylcholine were compared to those receiving vecuronium, their mean mouth opening and jaw stiffness, as well as their absolute and relative changes, were significantly different (ANOVA) (tables 2, 3). Because ANOVA and Mann-Whitney U analysis on the original, as well as the transformed, data were uniformly consistent with the ANOVA on the original data, the latter are presented.

The distribution of mouth opening reduction is displayed in figure 1. One patient's mouth opening, referred to above as the outlier, was in maximal occlusion (less than 1 mm) at the loss of twitch, as well as 45 s later. Intubation was not initially attempted in this and five other patients who had a reduction of mouth opening of

TABLE 2. Mean Mouth Opening D_i in mm Before and After Relaxant Administration, Without the Outlier

Muscle Relaxant (N)	D_1	D_2	D_3	D_2-D_1	D_3-D_1	$(D_1$-$D_2)/D_1$	$(D_1$-$D_3)/D_1$
Succinylcholine (22)	16.9 ± 2.8	12.6 ± 4.3*	13.0 ± 4.3*	−4.2 ± 3.4	−3.9 ± 3.6	+0.26 ± 0.21	+0.23 ± 0.23
Vecuronium (21)	19.8 ± 3.6	20.4 ± 4.0	20.9 ± 4.1†	+0.62 ± 1.3	+1.1 ± 1.5	−0.03 ± 0.07	−0.06 ± 0.08
ANOVA	F = 8.6	F = 37.1	F = 37.5	F = 37.4	F = 34.7	F = 37.0	F = 28.9
	$P < 0.006$	$P < 0.0001$	$P < 0.0001$	$P < 0.0001$	$P < 0.0001$	$P < 0.0001$	$P < 0.0001$

The mean distance of mouth opening in patients receiving succinylcholine (1.5 mg/kg) or vecuronium (0.1 mg/kg) during deep enflurane anesthesia prior to the administration of muscle relaxant at T_1 (D_1), immediately after the loss of twitch at T_2 (D_2), and 45 s after T_2 at T_3 (D_3). Also described are the mean absolute [D_2-D_1, D_3-D_1] and fractional [$(D_1$-$D_2)/D_1$, $(D_1$-$D_3)/D_1$] changes. Two-factor rm-ANOVA demonstrated a significant interaction between the repeated measures (D_i) and the relaxant groups ($F = 32.1$, $P < 0.0001$). Values are given as mean ± standard deviation. ANCOVA of D_2 and D_3 with D_1 as their covariate values confirmed the significant differences between the relaxant groups as indicated by the ANOVA. Mann-Whitney U analysis reproduced the ANOVA's results. These differences were confirmed after logarithmic transformation.

* Indicates intra-subject, within-group differences, when compared to D_1, significant (T square = 27.0) at the $P < 0.0001$ (for D_2) and at the $P < 0.0005$ (for D_3) level by one-population repeated measures ANOVA, corrected for pairwise comparison, in the succinylcholine group.

† Indicates intra-subject, within-group differences, compared to D_1, significant (T square = 11.9; $P < 0.02$) by one-population repeated measures ANOVA, corrected for pairwise comparison, in the vecuronium group.

TABLE 3. Mean Jaw Stiffness K_i in Nm/degree Before and After Relaxant Administration, With Exclusion of the Outlier

Muscle Relaxant (N)	K_1	K_2	K_3	K_2-K_1	K_3-K_1	$(K_1$-$K_2)/K_1$	$(K_1$-$K_3)/K_1$
Succinylcholine (22)	102.3 ± 21.9	154.5 ± 77.4*	150.5 ± 77.0*	52.2 ± 64.7	48.2 ± 67.7	−0.48 ± 0.52	−0.45 ± 0.54
Vecuronium (21)	87.0 ± 15.3	84.7 ± 16.3	83.0 ± 17.2	−2.3 ± 6.0	−4.0 ± 8.0	0.03 ± 0.07	0.05 ± 0.08
ANOVA	F = 7.0	F = 16.4	F = 15.4	F = 14.7	F = 12.3	F = 19.6	F = 17.3
	$P < 0.012$	$P < 0.0002$	$P < 0.0003$	$P < 0.0004$	$P < 0.001$	$P < 0.0001$	$P < 0.0002$

The mean Jaw stiffness in patients receiving succinylcholine (1.5 mg/kg) or vecuronium (0.1 mg/kg) during deep enflurane anesthesia prior to the administration of muscle relaxant at T_1 (K_1), immediately after the loss of twitch at T_2 (K_2), and 45 s after T_2 at T_3 (K_3). The mean absolute and fractional changes in jaw stiffness are also given. Values are expressed as mean ± standard deviation. Two-factor rm-ANOVA demonstrated a significant interaction between the repeated measures (K_i) and the relaxant groups ($F = 12.5$, $P < 0.0001$).

* Indicates intra-subject, differences, compared to K_1, significant (T square = 14.5) at the $P < 0.01$ (for K_2), and $P < 0.02$ (for K_3) level by one-population repeated measures ANOVA, corrected for pairwise comparison, in the succinylcholine group. ANOVA as well as Mann-Whitney U analysis demonstrated significant differences between relaxant groups. ANCOVA of K_2 and K_3 with K_1 as their covariate confirmed the results of the ANOVA. Analyses of the transformed data confirmed the between- and within-group differences.

50% or more at either T_2 or T_3. Measurements were continued in these six patients. Their changes in mouth opening are graphed in figure 2; the arrows indicate the time at which the first visible twitch returned in the thenar muscles. Their mouth opening remained reduced beyond the appearance of the first visible twitch. As recovery from peripheral succinylcholine relaxation progressed, mouth opening also returned towards baseline values, while an increase in masticatory muscle stiffness was observed for up to 10 min. The outlier and one of the five subjects required multiple attempts at laryngoscopy to expose the glottis after the measurement period; this was felt to be due to the increased resistance to mouth opening. In clinical terms, the mouth opening reduction of the outlier could be described as a mouth closed shut after succinylcholine administration. It could not be opened, even with considerable effort, because the physiological overbite did not permit the insertion of an instrument. The mouth opening measurement could be made because the mandibular retractor was placed and left *in situ* before the succinylcholine administration. The jaws relaxed with time (fig. 1). Patients in whom the mouth opening measurements were continued did not differ from the other patients. In three of these six patients, fasciculations occurred. The baseline mouth opening and jaw stiffness, and their respective changes, did not correlate significantly with age, weight, or height.

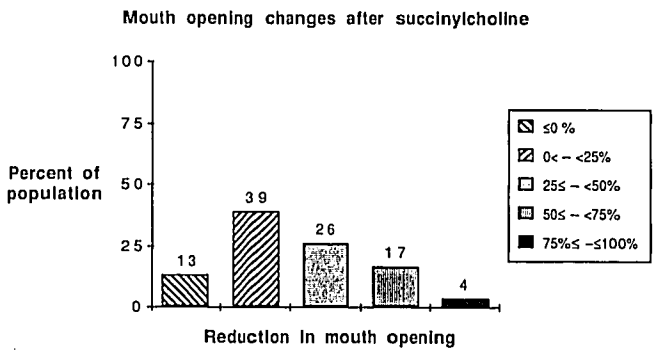

FIG. 1. The distribution of mouth opening reduction in the sample population. The abscissa presents the percent reduction of mouth opening in five categories. The ordinate gives the percent of subjects in the sample population.

All patients receiving succinylcholine had complete loss of their adductor pollicis twitch response. The mean time to onset of loss of twitch in these patients was 36 s (range 16–65 s). The first visible twitch returned after an average of 262 s (range 167–363 s). Fasciculations occurred in 14 of 23 patients receiving succinylcholine; their onset ranged from 6 to 32 s, mean ± SD 16 ± 8 s. Fasciculations ceased before the loss of twitch in the majority of patients, while, in a few (3) patients, fasciculations continued up to or just beyond the loss of twitch. Mouth opening measurements were made after the disappearance of fasciculations. No fasciculations were noted in the patient referred to as the outlier, while they were present in the second patient who was difficult to intubate. Patients who experienced fasciculations did not differ from those who did not fasciculate. Loss of twitch was obtained in all patients in the vecuronium group; the mean time to loss of twitch was 93 s (range 58–180 s). In addition to the loss of twitch, all patients had flaccid limbs and cessation of spontaneous ventilatory efforts. The temperature and end-tidal CO_2 changes were within the range of normal values for clinical anesthesia. General anesthesia was continued with enflurane in oxygen and nitrous oxide in all patients for the duration of the anesthetic procedure (mean ± SD, 123 ± 55 min; range 45–250 min). The mean duration of anesthesia in the six patients of figure 2 was 1 h 40 min (range 45–185 min), with that of the outlier lasting 185 min and 75 min in the other patient who was difficult to intubate. The outlier and another of the six patients had undergone a previous anesthetic procedure without problems. A hypermetabolic state did not develop by clinical criteria (heart rate, minute ventilation, etc.), end-tidal CO_2 criteria (during con-

stant minute ventilation), and/or temperature criteria. Limb muscle rigidity did not develop in any patient. None of the patients were treated with dantrolene sodium.

Discussion

The objective of this study was to compare mouth opening and jaw stiffness in relation to limb muscle relaxation following succinylcholine or vecuronium administration. With the effects of differences in anesthetic depth between patients minimized by using each subject as his/her own control, reductions in mouth opening and increases in jaw stiffness were demonstrated after succinylcholine administration at a time when relaxation of the limb muscles was present; in contrast, after vecuronium treatment, mouth opening increased. Jaw muscle stiffness increased after succinylcholine administration, despite an anesthetic depth that was the same or increased during the second and subsequent measurements. The anesthetic depth was not rigidly controlled and, possibly, varied between patients. Such variation can affect comparisons between the two relaxant groups, usually reducing the likelihood of demonstrating differences between the groups. In fact, initial between-group differences of mouth opening (D_1) and jaw stiffness (K_1) were present, raising the question of whether these may have been responsible for differences in mouth opening (D_2, D_3) and jaw stiffness (K_2, K_3) following relaxant administration. Although it is unknown what determines the magnitude of mouth opening during general anesthesia, differences between the two relaxant groups (for example, in age, height, or weight—albeit statistically not significant—or in anesthetic level) may have been a reason for the difference in baseline mouth opening and jaw stiffness. To assess whether the baseline differences in mouth opening and jaw stiffness could account for mouth opening and jaw stiffness differences following relaxant administration, analyses were used to adjust for the differences in baseline (ANOVA of the relative changes and ANCOVA). Through these methods, mouth opening reductions and jaw stiffness increases were demonstrable following succinylcholine administration, but not following vecuronium administration, despite the initial differences in mouth opening and jaw stiffness between the relaxant groups.

Fasciculations represent initial agonist effects of succinylcholine on twitch muscle fibers. During a depolarizing neuromuscular blockade, fasciculations and other contractions are followed by muscle fiber relaxation. Further stimulation of the endplate by either acetylcholine, succinylcholine, or, indirectly, *via* stimulation of the motor nerve does not result in contraction—the muscle is refractory to stimulation and loses tone. In this study, mouth opening was measured in all patients after cessation of fasciculations during complete loss of the adductor pollicis response to ulnar nerve stimulation and during limb muscle flaccidity following 1.5 mg/kg iv succinylcholine administration. Mouth opening reductions and jaw stiffness increases were present in most patients at a time when more than twice the mean time to loss of twitch (36 s) had passed. In six patients, mouth opening reduction and increased masticatory muscle stiffness lasted throughout the period of twitch ablation beyond the time of return of the first visible twitch. The effects of succinylcholine on the masticatory muscles is inconsistent with the known agonist actions of succinylcholine on twitch muscle fibers. The onset of action (complete suppression of the thenar twitch) and the duration of loss of twitch after intravenous succinylcholine in this study are consistent with reports in which succinylcholine-induced muscle relaxation was monitored by other techniques.[2,3] The mouth opening and jaw stiffness responses following succinylcholine administration during anesthesia with enflurane are similar to those observed during halothane anesthesia reported previously.[1] This is noteworthy in view of the differences in muscle relaxant properties of enflurane and halothane.[4,5]

The force of 1.67 Newton (approximately 170 g) was chosen to cause a mouth opening of up to 50% (2 cm) of maximum opening capacity (4 cm) in the lowest age range of the anesthetized study population. This is a

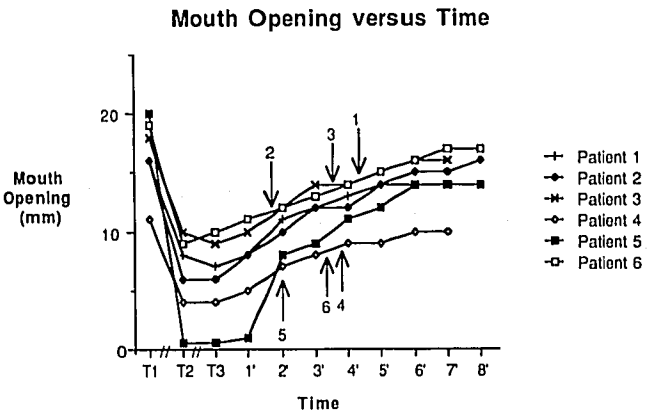

FIG. 2. Diagram illustrating the change of mouth opening over time in the six patients who received 1.5 mg/kg succinylcholine intravenously during enflurane anesthesia and who had a mouth opening reduction of 50% or more at T_2 or T_3. The ordinate represents the inter-incisal distance D_i in mm. The abscissa represents time; T_1 is the time at which the baseline measurement is taken, T_2 is the moment at which the adductor pollicis twitch to ulnar nerve stimulation is lost, and T_3 is 45 s after T_2, followed by intervals of 1 min. Arrows indicate the return of the first visible twitch in each patient; the numeric label refers to the individual patient.

relatively low force compared to those which can be exerted during direct laryngoscopy.[1] Higher forces are likely to accomplish a larger mouth opening in these patients than recorded in this study. The magnitude of mouth opening is rarely assessed objectively after induction of general anesthesia before succinylcholine administration. If, after succinylcholine administration, the mouth opens sufficiently to insert a laryngoscope blade, a reduction of maximal opening capacity may not be recognized as such during intubation. However, when, in a recumbent patient, the jaws are brought into occlusion, little or no horizontal inter-incisal space may be available to open the mouth or to insert a laryngoscope because of the physiological overbite. This may have been referred to as masseter spasm.[6] The mouth of the outlier in this study could not be opened; its reduction lasted several minutes, during which the peripheral twitch was abolished completely. This patient's mouth closing response qualifies, in our opinion, for the description of masseter muscle rigidity.

The paucity of clinical reports on masseter spasm during enflurane anesthesia may stem from the predominant use of halothane for the inhalation induction of anesthesia in children. Mouth opening reductions after succinylcholine have not been reported in adults, except for a rare case of masseter spasm. Perhaps this is because different induction techniques (thiopental-succinylcholine) are commonly used in adults. However, recently jaw closing forces have been recorded after succinylcholine infusions during thiopental anesthesia in adults.†† Changes in maximal mouth opening capacity have been noted by clinicians who regularly intubate the tracheas of children during a deep level of inhalation anesthesia.[7]

Masseter spasm has become associated with malignant hyperthermia susceptibility.[8–11] It has been suggested as pathognomonic for malignant hyperthermia on the basis of the calcium uptake test.[9] In a recent study, this test did not permit the distinction between normal and malignant hyperthermia susceptible muscle, nor did it correlate with the caffeine-halothane contracture tests.[12] Even though 87% of the subjects in this study developed a reduction in mouth opening, including one patient who experienced masseter muscle rigidity, none of these patients developed a hypermetabolic state or hyperthermia.

In conclusion, we have shown that succinylcholine administration during enflurane anesthesia may be followed by a variable degree of mouth opening reduction, that such a reduction is a transient phenomenon (potentially lasting several minutes), and that it occurs despite abolition of neurally evoked responses of limb muscle. The associated increase in masticatory muscle stiffness is not consistent with the known agonist effects of succinylcholine on striated twitch muscle fibers.

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