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Succinylcholine-induced Fasciculations and Intragastric Pressure during Induction of Anesthesia

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Since Snow and Nunn[1] stated their concern more than 20 years ago that "depolarization of the abdominal muscles might raise the intragastric pressure," regurgitation during anesthetic induction has been considered a potential hazard when succinylcholine is used for tracheal intubation. Recommendations for avoiding this catastrophe include rapid intravenous induction of anesthesia,[1,2] avoidance of depolarizing muscle relaxants,[1] prior use of atropine,[2] small doses of nondepolarizing relaxants or local anesthetics prior to succinylcholine,[3,4] and various mechanical maneuvers.[1,2,5] Although one early investigation reported that "fasciculations were found to have little effect on intragastric pressure . . .",[6] a direct relationship between subjectively scored succinylcholine-induced fasciculations and gastric pressure increases was subsequently demonstrated.[4] In the present study, we utilized electromyography to examine quantitatively measured total abdominal muscle activity and intragastric pressure changes in surgical patients receiving succinylcholine in three different dosages and thiopental as part of their anesthetic induction sequence.

METHODS

Forty-eight men scheduled for elective abdominal surgery consented to participate in this study approved by the Human Studies Subcommittee. Age ranged from 25 to 88 years, and all were ASA physical status I or II. After im premedication with a tranquilizer or narcotic and an antisialogogue (atropine, scopolamine, or glycopyrrolate) and application of solutions of tetracaine 0.5 per cent and lidocaine 4 per cent to the naso- and oropharynx, a ballon-tipped gastric catheter was inserted into the stomach through the nose. The air-filled latex balloon and 4-mm (OD) polyethylene catheter was connected to a pressure transducer, and intragastric pressure (IGP) was simultaneously displayed and recorded. Intragastric positioning was determined by advancement of the catheter to the point of abrupt transition from negative to positive pressure deflections with inspiration during quiet, spontaneous breathing. IGP was measured in units of centimeters of water zeroed to atmosphere with the transducer placed at the midchest level.

Abdominal wall muscle activity was quantified with an integrated electromyogram (IEMG) system consisting of three gel-type surface electrodes: two electrodes were placed 5 cm apart, about 10 cm lateral to the midline at the level of umbilicus, the third electrode serving as reference ground placed over the left lower abdominal quadrant. Electrodes were connected to an isolated ECG amplifier set at 200 μV/cm with the output electronically integrated and activity expressed as mean integrated EMG activity, in microvolts. Peak-to-peak microvolt activity was observed but not recorded. Electronic filtering circuits excluded signals with frequencies below 10 cps or above 2500 cps. In 33 patients, a technician-observer not participating in the anesthetic management of the patient was present to score visible muscle fasciculations on a scale of 0 (none) to +3 (severe).

After measurement of awake control values for IGP and IEMG, anesthesia was induced with thiopental, 4 mg/kg, given in 15–20 s. After an interval of 20 to 40 s, succinylcholine 1.0, 1.5, or 2.0 mg/kg was given in a 5- to 10-s period. This nonpretreated control group consisted of 34 patients. The remaining 14 patients, chosen at random, underwent the identical sequence except for the administration of *d*-tubocurarine, 4 mg, iv, immediately prior to oxygen

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TABLE 1. Integrated Electromyogram after Injection of Thiopental and Succinylcholine

	Awake	After Thiopental			After Succinylcholine		
	$\overline{IEMG}$*	$\overline{IEMG}$	↑†	↓†	$\overline{IEMG}$	↑†	↓†
Pretreated group							
1.5 mg/kg, SC‡ (n = 14)	11.3 ± 2.8	11.1 ± 2.6	1	1	10.8 ± 2.5	0	1
Control group							
1.0 mg/kg SC (n = 11)	13.6 ± 2.4	13.8 ± 2.2	1	0	15.5 ± 4.0	2	3
1.5 mg/kg SC (n = 12)	10.2 ± 1.2	10.6 ± 1.5	1	1	18.2 ± 4.6	6	1
2.0 mg/kg (n = 11)	14.1 ± 3.4	14.2 ± 3.4	0	1	16.2 ± 2.8	3	1
All patients							
(n = 48)	12.2 ± 1.3	12.3 ± 1.2	3	3	15.0 ± 3.0	11	6

* $\overline{IEMG}$ = mean integrated electromyogram (μV) ± SE.
† ↑ or ↓ = number of patients showing significant increase (↑) or decrease (↓) from previous value.
‡ SC = succinylcholine.

breathing and comprised the pretreated group. Pretreated patients received a standardized intravenous dose of succinylcholine, 1.5 mg/kg, 5.5 to 6 min after receiving *d*-tubocurarine. IGP and IEMG were measured and recorded continuously in all patients from oxygen breathing through tracheal intubation which occurred 1.5 to 2 min after succinylcholine injection. Patients remained apneic and undisturbed until tracheal intubation. After intubation, the gastric catheter and electrode systems were removed, and surgery was begun.

For each patient, three mean values (control, after thiopental, and after succinylcholine) for IGP and IEMG were compared for significant differences by analysis of variance. To minimize sampling errors produced by minor fluctuation of baseline values associated with respiratory, cardiac, and minor movement artifacts, each of these IGP and IEMG values represented the mean of 20 values seen on the original continuous recording at 1-s intervals during the 20-s period immediately prior to or after completion of injection of thiopental or succinylcholine. Other mean values which represented the average IGP or IEMG of a group of patients at a given point in the induction sequence were compared by *t* test. Chi-square analysis with Fisher correction was used to determine the statistical significance of differences in the incidence of fasciculations and gastric pressure changes in the respective groups. A P value ≤0.05 was the criterion of statistical significance.

TABLE 2. Correlation between Subjective Scoring of Succinylcholine-induced Fasciculations and Change in Mean Integrated Electromyogram, $\Delta\overline{IEMG}$*

Score	$\Delta\overline{IEMG}$† (μV)	n
0 (none)	−1.4 ± 0.4	17
+1 (mild)	+3.9 ± 1.8	7
+2 (moderate)	+8.6 ± 0.9	3
+3 (severe)	+17.9 ± 6.7	6

* Values are means ± SE.
† Mean ΔIEMG values significantly different by analysis of variance, F = 10.76.

RESULTS

The mean awake control IEMG for all pretreated patients was statistically indistinguishable from the corresponding value for the nonpretreated control group (table 1). Following injection of thiopental, mean IEMG values did not change significantly for either group but analysis of variance indicated that thiopental produced significant IEMG changes in six patients, three increases and three decreases. The distribution of those changes was not significantly related to patient group. Subsequent injection of succinylcholine increased abdominal muscle activity as measured by IEMG in 11 patients in the control group, but no patient pretreated with *d*-tubocurarine demonstrated a significant increase in IEMG, a difference in incidence that was statistically significant. Using *t* test comparison of the mean IEMG values after succinylcholine for the control and pretreated groups failed, however, to demonstrate significant differences for the respective groups. In the 33 patients observed for gross abdominal fasciculation activity, individual changes in mean IEMG during the period from 20–40 s after succinylcholine injection correlated directly with the subjective scoring of gross abdominal fasciculations (table 2). Neither mean IEMG values for the respective patient groups nor the incidence of individual significant IEMG increase varied in a manner that correlated with the dosage of succinylcholine.

There was no significant difference between the awake IGP value for the pretreated and control patient groups (table 3). Following injection of thiopental, no statistically significant change in mean IGP for either group could be demonstrated, but analysis of variance indicated that IGP had changed significantly from the awake value in 30 of the 48 patients studied, 24 patients showing a significant individual reduc-

TABLE 3. Intragastric Pressure after Injection of Thiopental and Succinylcholine

	Awake	After Thiopental			After Succinylcholine		
	$\overline{IGP}$	$\overline{IGP}$	↑	↓	$\overline{IGP}$	↑	↓
Pretreated group							
1.5 mg/kg SC (n = 14)	6.7 ± 1.4	5.8 ± 1.2	4	8	4.6 ± 1.2	1	6
Control group							
1.0 mg/kg SC (n = 11)	10.0 ± 1.1	8.6 ± 1.5	1	6	7.3 ± 1.2	1	4
1.5 mg/kg SC (n = 12)	5.6 ± 1.1	4.1 ± 1.3	1	5	5.2 ± 1.4	6	3
2.0 mg/kg SC (n = 11)	9.0 ± 1.2	7.6 ± 1.4	0	5	7.4 ± 1.3	3	3
All patients							
(n = 48)	7.7 ± 0.6	6.4 ± 0.7	6	24	6.1 ± 0.6	11	16

$\overline{IGP}$ = mean intragastric pressure, cm H_2O, ± SE.
↑ or ↓ = number of patients showing significant increase (↑) or decrease (↓) from previous value.
SC = succinylcholine.

tion in IGP following thiopental. Similarly, mean IGP for the pretreated and control patient groups as a whole were not changed significantly from their respective awake or post-thiopental values by the subsequent injection of succinylcholine. However, analysis of variance indicated that compared to the post-thiopental values, 10 of the 33 control patients had significant increases in IGP after succinylcholine injection, but only 1 of the 14 pretreated patients experienced a significant rise in IGP.

In those patients in whom both IGP and IEMG increased significantly, the magnitude of the changes were linearly and directly correlated (fig. 1). For all patients studied, the range of observed awake IGP was 0.4–16.2 cm H_2O, for observed IGP after thiopental, 0.3–20.4 cm H_2O, and after succinylcholine, −0.7–16.3 cm H_2O. There was no significant difference in the incidence of IGP elevation in patients receiving different dosages of succinylcholine.

DISCUSSION

We demonstrated a direct correlation between the magnitude of abdominal IEMG change and acute IGP elevation after injection of succinylcholine. Previously, Miller and Way[4] observed a similar relationship between subjectively scored fasciculations and IGP, but their experimental design monitored only abdominal muscle activity which was grossly visible. In our study, IEMG changes and observed fasciculations were equivalent, suggesting that invisible, coordinated contractions of the abdominal muscles are not a significant component of succinylcholine-induced depolarization and could not play a role in acute elevation of IGP after injection of succinylcholine. The use of the electromyogram to quantify both fasciculations and resting abdominal muscle tone has been verified previously.[7,8]

Our values for resting awake IGP agree closely with previous studies using air-filled balloon catheters and manometer systems,[4,6,9] and we confirmed prior estimates[4,10,11] that about 30 per cent of nonpretreated patients have acute elevation of IGP after injection of succinylcholine, with a wide range of individual variation in resting and post-succinylcholine IGP

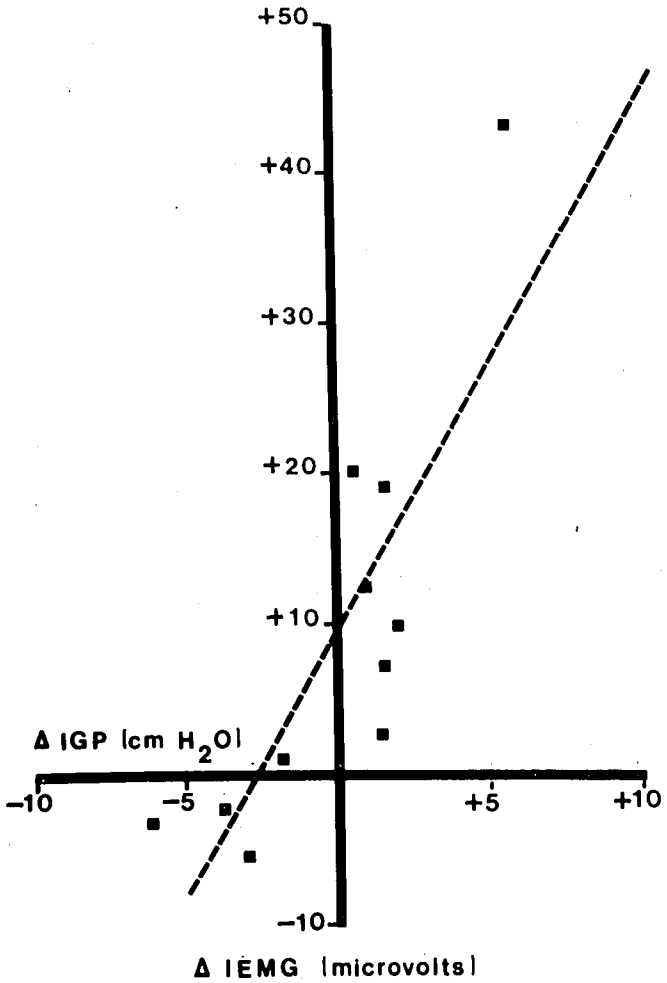

FIG. 1. Correlation between significant change in integrated electromyographic activity (ΔIEMG) and change in intragastric pressure (ΔIGP) in individual patients after intravenous injection of succinylcholine (n = 11, r = 0.83, $P < 0.01$).

values. We were unable to find patients receiving succinylcholine without pretreatment who demonstrated sustained IGP greater than 20–30 cm H_2O, the value cited[1,6,12] as the upper limit of IGP compatible with competence of the normal gastric cardia. In patients with conditions such as pregnancy or hiatal hernia which make the esophago-fundal angle less acute,[12] however, some IGP values measured in our study could have produced regurgitation of gastric contents into the esophagus. Our study also did not include patients with gross pathologic gastric distension which may itself reduce the competence of the cardia.[13]

Both narcotics[13] and antisialogogues[14] appear to reduce intrinsic gastric muscle tone and, presumably, IGP. Since all patients in our study received premedication with drugs from at least one of these categories, we could not evaluate their role in modifying IGP values or the response to succinylcholine injection. We did find, however, that rapid injection of 4 mg/kg thiopental was an important influence on resting IGP, producing a significant drop in IGP in one-half of the patients studied. The consistency and magnitude of these thiopental-induced IGP changes were sufficient to suggest that the large increases in IGP after succinylcholine which have been reported[9,10] may be due, at least in part, to the use of inadequate dosages of thiopental or long intervals between injection of the two drugs.

Coughing during intubation in patients with full stomachs has been shown by LaCour[3] and others[11,15] to produce extreme elevation of IGP as well as subsequent aspiration of gastric contents.[1] Pretreatment with nondepolarizing agents prior to anesthetic induction antagonizes the paralyzing effect of succinylcholine, delays the onset of paralysis,[16] and may be associated with a significant incidence of coughing and difficulty with tracheal intubation[17] unless the dosage of succinylcholine is increased substantially. Our data indicate that the increased succinylcholine dosages required to maintain suitable conditions for intubation following pretreatment do not themselves result in a higher incidence or greater severity of acute IGP elevation.

We conclude that resting abdominal muscle tone, coordinated contraction during coughing, and succinylcholine-induced fasciculations all influence IGP and introduce a risk of regurgitation of gastric contents. If an awake endotracheal intubation cannot be performed, our data support the old concept that an anesthetic induction sequence for patients with full stomachs should include pretreatment with a nondepolarizing muscle relaxant and generous dosages of thiopental and succinylcholine.

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