

Clinical Workshop

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Inhibition of Succinylcholine-induced Increased Intra-gastric Pressure by Nondepolarizing Muscle Relaxants and Lidocaine

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Rapid induction of anesthesia with thiopental and succinylcholine (SCh) followed immediately by tracheal intubation is a technique recommended for inducing anesthesia in patients with a full stomach. However, SCh may provoke regurgitation of gastric contents by producing fasciculations of abdominal musculature, which in turn may increase intragastric pressure.¹⁻⁴ We determined the increase in intragastric pressure produced by a rapid injection of SCh intravenously into man and whether this increase could be prevented by eliminating the SCh-induced fasciculations by prior administration of a nondepolarizing relaxant⁵ or a local anesthetic.

METHODS

Intra-gastric pressures were determined before and during anesthesia in 66 unpremedicated † men ranging in age from 18 to 30 years who had sustained acute war wounds. Measurements were made with a thin-walled rubber balloon attached to a water manometer

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† Six patients received atropine, 0.6 mg intravenously, 15 to 30 minutes prior to the induction of anesthesia.

calibrated in cm H₂O, using a technique described by Roe.² The maximum quantity of air that the balloon would hold while still registering a pressure reading of zero was first determined. The balloon was then deflated and inserted into the stomach and the predetermined volume of air injected. The volume of air injected was usually 45 ml (range 42 to 50 ml). The location of the balloon was confirmed by fluctuations in pressure with respiration and by roentgenographic examination.

Anesthesia was induced with the patient supine by intravenous administration of thiopental, 4 mg/kg, followed by SCh, 1 mg/kg. Intra-gastric pressure was monitored continuously. The maximum changes in intra-gastric pressure were recorded: 1) two minutes after the administration of thiopental; and 2) during SCh-induced muscle fasciculations or within two minutes after the administration of SCh if fasciculations did not occur. These intra-gastric pressures were compared with the awake values. The intensity of fasciculations was graded by an independent observer using the following scale: maximal (+++); moderate (++) ; or minimal (+). The independent observer was an anesthesia corpsman who did not know the measured intra-gastric pressures during his grading of fasciculation intensity.

Thirty patients were studied in the above manner. Another 30 patients were studied in an identical manner except that 15 were given gallamine triethiodide, 20 mg, and the other 15 were given *d*-tubocurarine chloride, 3 mg, intravenously three minutes prior to injection of SCh. Finally, an additional six patients were given lidocaine hydrochloride, 6 mg/kg intravenously, over a two- to three-minute period prior to injection of SCh.

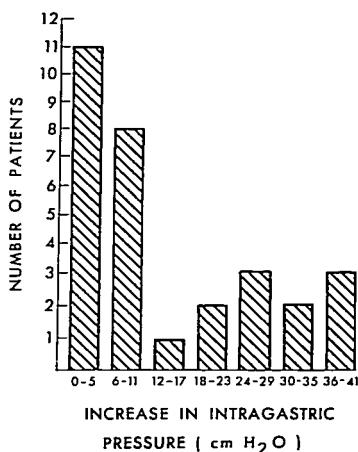


FIG. 1. This figure relates the number of patients studied with increases in intragastric pressure (cm H₂O) in response to an intravenous injection of succinylcholine, 1 mg/kg. The range of intragastric pressure readings was subdivided into 5-cm H₂O intervals. The number of patients in each interval is illustrated.

RESULTS

Changes in intragastric pressure after thiopental were not significant, with a mean increase of 2.3 cm H₂O (range -2.1 to +3.2), but after administration of SCh alone, intragastric pressure increased more than 11 cm H₂O in 11 of the 30 patients studied (fig. 1). Of the 11 patients in the 0 to 5 cm H₂O range of intragastric pressure illustrated in figure 1, seven had no change in intragastric pressure with administration of SCh alone. Of the 30 patients receiving SCh alone, six were premedicated with atropine, 0.6 mg iv; they developed a mean increase in intragastric pressure of 9.5 cm H₂O (range 0 to 23 cm H₂O). The increases in intragastric pressure were directly related to the intensity of SCh-induced skeletal muscle fasciculations, with increases in intragastric pressure associated with (++++) fasciculations greater than those associated with (+) fasciculations ($P < 0.01$) (fig. 2). Prior administration of gallamine, *d*-tubocurarine, or lidocaine prevented significant in-

creases in intragastric pressure associated with SCh administration (table 1). Although gallamine was more effective than *d*-tubocurarine in attenuating SCh-induced increased intragastric pressure (table 1), the difference was not statistically significant.

Discussion

This study demonstrates that skeletal muscle fasciculations associated with administration of SCh may increase intragastric pressure several-fold over that observed in the resting state. The variability in magnitudes of intragastric pressure elevations was directly related to the variable intensities of fasciculations associated with administration of SCh.

Other investigators have recorded similar intragastric pressures following SCh as high as our readings, but their reported frequencies were lower.^{1,2,4} The higher incidence of markedly elevated intragastric pressure values observed in this study may be related to the

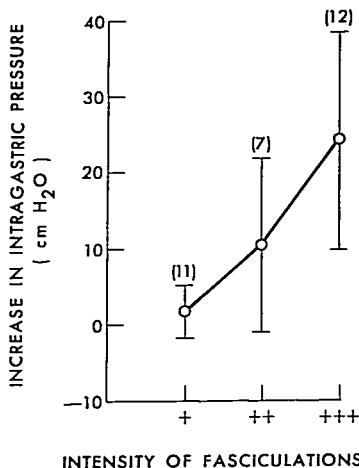


FIG. 2. Relationship between intensity of succinylcholine-induced fasciculations and increased intragastric pressure. The numbers in parentheses represent total numbers of patients. The brackets represent the standard deviation. Increases in intragastric pressure with (++++) fasciculations were significantly different from those observed with (+) fasciculations ($P < 0.01$).

fact that our patients were unpremedicated, muscular, young men, in contrast to the older premedicated patients studied by others. It should not be surprising that intragastric pressure elevations as high as 40 cm H₂O occur as a result of SCh fasciculation, in view of the fact that more coordinated abdominal skeletal muscle activity, such as straight leg raising, may increase intragastric pressure to values as high as 120 cm H₂O.⁷

In addition to skeletal muscle fasciculations, the acetylcholine-like effect of SCh⁸ may be in part responsible for the observed increases in intragastric pressure. Greenan⁹ observed consistent increases in intragastric pressure of 4 to 7 cm H₂O with direct vagal stimulation. Therefore, it may be postulated that prior administration of vagolytic drugs will in part inhibit the increase in intragastric pressure associated with SCh administration. Gallamine, a muscle relaxant with vagolytic actions, was more effective in preventing an increase in intragastric pressure following SCh administration than was *d*-tubocurarine (table 1), although the difference was not significant. Furthermore, those patients premedicated with atropine had lesser increases in intragastric pressure with SCh, but this was not statistically different from the increases in intragastric pressure in unpremedicated patients.

Are the increases in intragastric pressure following SCh enough to cause incompetence of the cardioesophageal junction? Marchand found that intragastric pressures greater than 28 cm H₂O were frequently associated with incompetence of the cardioesophageal junction in cadavers.¹⁰ In 15 anesthetized patients studied by Greenan, six needed less than 28 cm H₂O to cause incompetence of the cardioesophageal junction.⁹ When the normal oblique angle of entry of the esophagus into the stomach is altered, as may occur with pregnancy, an abdomen distended by ascites or bowel obstruction, obesity, or a hiatus hernia, the intragastric pressure required to cause incompetence of the cardioesophageal junction is frequently less than 15 cm H₂O.^{9,10} Under these circumstances, regurgitation of stomach contents following SCh is a distinct possibility, and precautionary measures should be taken to prevent fasciculations.

Objections to administration of a nonde-

TABLE 1

Sequence of Drug Administration	Number of Patients	Increases in Intragastric Pressure (cm H ₂ O)		
		Mean	SD	Range
Succinylcholine plus gallamine	15	1.8	±1.1	0-5.0
Succinylcholine plus <i>d</i> -tubocurarine	15	3.6	±2.5	-1.5-6.5
Succinylcholine plus lidocaine	6	1.5	±2.2	0-6.0

polarizing muscle relaxant prior to SCh are based on the fact that *d*-tubocurarine or gallamine could antagonize the neuromuscular blocking action of SCh.^{11,12} La Cour recommends administration of 4 to 6 mg of *d*-tubocurarine prior to SCh to prevent increases in intragastric pressure.¹³ In La Cour's study, one patient coughed with insertion of the endotracheal tube, which increased intragastric pressure 25 cm H₂O. Inadequate paralysis from SCh in this case nullified the proposed beneficial effect of prior administration of *d*-tubocurarine. Prior administration of a smaller dose of *d*-tubocurarine (3 mg) or gallamine (20 mg) resulted in little clinical antagonism of SCh, 1 mg/kg, in this or previous studies,¹⁴ and still prevented SCh-induced increased intragastric pressure. Since the antagonism between nondepolarizing muscle relaxants and SCh is dose-related, smaller doses of SCh may exhibit greater clinical antagonism to the doses of gallamine and *d*-tubocurarine used in this study.

Prior administration of lidocaine, 6 mg/kg, was effective in preventing SCh-induced increased intragastric pressure in the six patients studied. Because two of the patients had systolic blood pressure reductions of 22 and 30 torr, respectively, this technique was not continued. Smaller doses of lidocaine (2 to 5 mg/kg) have been shown not to be completely effective in preventing SCh fasciculations.¹⁵

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REFERENCES

- Andersen N: Changes in intragastric pressure following the administration of suxamethonium. *Brit J Anaesth* 34:363, 1962
- Roe RB: The effect of suxamethonium on intragastric pressure. *Anaesthesia* 17:179, 1962
- Wylie WD: The use of muscle relaxant at the induction of anesthesia of patients with a full stomach. *Brit J Anaesth* 35:168, 1963
- La Cour D: Rise in intragastric pressure caused by suxamethonium fasciculations. *Acta Anaesth Scand* 13:225, 1969
- Lamoreaux LR, Urbach KF: Incidence and prevention of muscle pain following the administration of succinylcholine. *ANESTHESIOLOGY* 21:394, 1960
- Usubiaga JE, Wikinski JA, Usubiaga LE, *et al.*: Intravenous lidocaine in the prevention of postoperative muscle pain caused by succinylcholine administration. *Anesth Analg* 46:225, 1967
- Marchand P: A study of the forces productive of gastro-oesophageal regurgitation and herniation through the diaphragmatic hiatus. *Thorax* 12:189, 1957
- Schoenstadt DA, Whitcher CE: Observation on the mechanism of succinylcholine-induced cardiac arrhythmia. *ANESTHESIOLOGY* 24:358, 1963
- Greenan J: The cardio-oesophageal junction. *Brit J Anaesth* 33:432, 1961
- Marchand P: The gastro-oesophageal "sphincter" and mechanism of regurgitation. *Brit J Surg* 42:504, 1955
- Waltz LF, Dillon JB: Clinical studies of the interaction between *d*-tubocurarine and succinylcholine. *ANESTHESIOLOGY* 31:39, 1969
- Katz RL, Katz GJ: Complications associated with the use of muscle relaxants, Muscle Relaxants, *Clinical Anesthesia*. Edited by FF Foldes. Philadelphia, FA Davis, 1966, pp 125
- La Cour D: Prevention of rise in intragastric pressure due to suxamethonium fasciculations by prior dose of *d*-tubocurarine. *Acta Anaesth Scand* 14:5, 1970
- Miller RD, Way WL, Hickey RF: Inhibition of succinylcholine-induced increased intracular pressure by nondepolarizing muscle relaxants. *ANESTHESIOLOGY* 29:123, 1968
- Wikinski R, Usubiaga LJ, Usubiaga JE, *et al.*: Prevention of succinylcholine fasciculations with local anesthetics. *ANESTHESIOLOGY* 26:3, 1965

CASE REPORTS

Malignant Hyperthermia Associated with Hypocalcemia

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Hyperthermia following anesthesia has been a matter of concern for decades. Typically the rise in temperature was slow and progressive, and usually it was attributed to elevated room temperature, heavy draping, or prolonged surgery. Recent reports from Australia, Canada, the United States, Great Britain, and South Africa have described a more alarming type of hyperthermia, which occurs during anesthesia.¹⁻⁵² This condition, which occurs in both man and animals, appears to be biphasic. The initial phase is characterized by an insidious,

progressive rise in temperature, accompanied by appropriately profuse sweating. A prodrome of progressive muscular rigidity may be present but is often unrecognized because of surgical draping.

The early phase, which occurs 30 to 90 minutes after induction, is followed by a more rapid rise in temperature accompanied by inability to sweat and mottling of the skin. Studies made immediately upon recognition of the syndrome reveal severe metabolic and respiratory acidosis and extreme alterations in arterial-to-mixed venous oxygen ratios. Bradycardia progressing to cardiac arrest usually follows; on occasion, this has responded to intravenous calcium chloride. Disseminated intravascular coagulation may be detected fol-

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