Failure of Lower Esophageal Contractility to Predict Patient Movement in Children Anesthetized with Halothane and Nitrous Oxide

Mehernoor F. Watcha, M.D.,* Paul F. White, Ph.D., M.D.†

The clinical usefulness of monitoring the frequency of spontaneous lower esophageal contractions (SLEC) to assess the depth of anesthesia was evaluated in 38 children. The hypothesis that SLEC can predict movement in response to skin incision during nitrous oxide and halothane anesthesia was tested. Although movement in response to skin incision was correlated with an increase in heart rate, blood pressure, and an increased SLEC frequency, there was no correlation between the SLEC prior to skin incision and movement in response to this stimulus. Moreover, cardiovascular responses preceded the detection of SLEC changes. The relationship between changes in SLEC activity and times to awakening during emergence from anesthesia was also examined. During emergence from anesthesia there was an increase in SLEC, but the time to awakening could not be predicted from the SLEC. Cardiovascular changes again preceded SLEC changes during emergence. Monitoring of the lower esophageal contractility rate would appear to be of limited usefulness in evaluating the depth of anesthesia in unparalyzed children during nitrous oxide and halothane anesthesia. (Key words: Anesthesia: pediatrics. Anesthetic potency: minimum alveolar concentration (MAC). Anesthetics, gases: nitrous oxide. Anesthetics, volatile: halothane. Esophagus: contractility. Monitoring: lower esophageal contractility (LEC).)

AN IDEAL DEPTH of anesthesia monitor would be simple, noninvasive, with a consistent dose-response relationship to anesthetic drugs, and should predict patient response to surgical stimuli regardless of the anesthetic agent used. This would permit the anesthesiologist to adjust the anesthetic dose more precisely, and might decrease recovery times and reduce the incidence of anesthetic-related side effects. The spontaneous rate of contractility in the lower esophagus (SLEC) has been reported to correlate with autonomic responses during surgery in adults. In addition, an inverse relationship has been demonstrated between SLEC activity and inspired halothane or isoflurane concentrations. 1-3 Recently, Sessler et al. 4 reported that the SLEC could predict patient movement with skin incision in patients anesthetized with halothane, but not in those receiving alfentanil and nitrous oxide. No data are available regarding SLEC activity in anesthetized children. This study was designed to test the hypothesis that patient movement in response to surgical stimuli can be predicted by the rate of spontaneous contraction of the lower esophagus during halothane-nitrous oxide anesthesia in children. We evaluated the relationship between clinical signs of anesthetic depth, hemodynamic responses, and SLEC activity.

Materials and Methods

After obtaining institutional approval and written informed consent from the parents and, where applicable, from the child, we studied 38 patients, ranging in age from 7 to 17 yr (mean \pm SD = 12.1 \pm 2.4 yr). Patients were selected from a group scheduled to undergo surgical procedures where the anesthetic plan included nitrous oxide, halothane, tracheal intubation, and insertion of an esophageal stethoscope. We excluded children younger than 7 yr of age, children with a history of esophageal disease, or those receiving drugs thought to affect lower esophageal contractility (e.g., theophylline, atropine, beta-adrenergic, or calcium channel blockers). No preoperative sedative drugs were used.

At the discretion of the attending anesthesiologist, anesthesia was induced by iv thiopental, 3–5 mg/kg, followed by the inhalation of nitrous oxide and halothane, or using nitrous oxide and halothane alone. Following induction the trachea was intubated without the use of neuromuscular blocking agents. Ventilation was controlled to achieve a plateau end-tidal carbon dioxide tension of 32–35 mmHg. A specially designed 18-Fr esophageal probe with a distal water-filled pressure sensing balloon (Cat. No. 43412-017, American Antec, Valencia, California) was passed into the esophagus to a predetermined depth as obtained from the following formula⁵:

Depth of insertion (cm) = 12 + [(height in cm - 50)/5]

The pressure sensing balloon was connected to the transducer of a LECTRON 302 esophageal monitor (American Antec, Valencia, California, distributed by the Pharmaseal Division, Baxter Health Care Corp, Woodstock, Illinois), with a threshold sensitivity set at 10 mmHg to minimize artifacts from cardiac and respiratory motion. The LECTRON® monitor uses the average esophageal contractility over a 3-min period as the SLEC rate per minute. The SLEC rate was recorded at 1-min intervals, in addition to the heart rate derived from the R-R interval

^{*} Assistant Professor of Anesthesiology.

[†] Professor of Anesthesiology, Director of Clinical Research.

Received from the Department of Anesthesiology, Washington University School of Medicine, St. Louis, Missouri. Accepted for publication June 21, 1989.

Address reprint requests to Dr. Watcha: Department of Anesthesiology, Washington University School of Medicine, Box 8054, 660 South Euclid Avenue, St. Louis, Missouri 63110.

on lead II of the ECG, blood pressure by automatic oscillotonometry (Dinamap® Critikon, Tampa, Florida), and oxygen saturation by a Nellcor® N100 pulse oximeter (Nellcor, Hayward, California). The inspired and endtidal concentrations of nitrous oxide, halothane, nitrogen, oxygen, and carbon dioxide were measured by a mass spectrometer (Perkin-Elmer MGA 1100, Perkin Elmer, Pomona, California) that had been calibrated with gases analyzed to ±0.02% accuracy (Scott Medical Products, Plumsteadville, Pennsylvania).

To minimize errors from alveolar-to-arterial pressure gradients, we maintained a minimal gradient between the inspired and the end-tidal anesthetic partial pressures.⁶ The end-tidal nitrous oxide and halothane concentrations were kept constant for at least 15 min before skin incision.⁶ The end-tidal nitrous oxide concentration was kept constant throughout the study at 66.1 \pm 1.7% (mean \pm SD). The end-tidal halothane concentration for an individual patient was predetermined using Dixon's up-and-down method.7 The halothane concentration was increased or decreased by $0.1 \pm 0.05\%$ depending on whether the previous study patient moved or did not move in response to skin incision. Movement was defined as an all-or-none response of purposeful withdrawal to skin incision. Nonpurposeful motor activity, grimacing, or motion at sites other than the skin incision were not considered as movement.6

Some patients had more than one skin incision performed at different times during the same anesthetic episode. In these patients the end-tidal halothane concentration was different for each incision but was kept constant for a minimum of 15 min before each incision. If a patient moved in response to skin incision or at other times during surgery, the anesthesiologist administered thiopental, additional halothane, and/or opioids. At the end of the surgical procedure nitrous oxide and halothane were discontinued and the oxygen inflow increased to 12 l/min. Heart rate, mean arterial blood pressure, and endtidal gas concentrations were measured at 1-min intervals until the patient opened his/her eyes. Because the LEC-TRON® monitor cannot distinguish between coughing and true tertiary contractions, we stopped recording the SLEC when the patient began coughing during emergence from anesthesia. The time from discontinuation of anesthesia to patient coughing and to eye opening were also recorded.

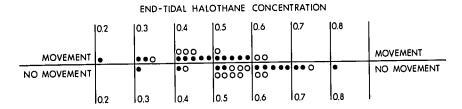
Continuous variables were analyzed by a one-way analysis of variance (ANOVA). Discrete variables were analyzed by chi-square test with Yates' correction for continuity. Student's t test was used to compare variables in two groups. The relationship between movement/no movement response versus halothane or SLEC frequency was examined using a biserial correlation analysis. P values < 0.05 were considered statistically significant.

Results

Twenty patients underwent an inhalation induction (group 1). Five of these 20 patients had two skin incisions during the same anesthetic episode, but no anesthetic agents other than nitrous oxide and halothane were administered prior to either incision. A total of 25 responses to skin incision after inhalation induction are therefore available for analysis in this group. Another 18 patients (group 2) received thiopental, 3-5 mg/kg, for induction of anesthesia. Because each subject in this group had only one skin incision during the surgical procedure, a total of 18 responses to skin incision were analyzed in the thiopental induction group. There were no statistically significant differences between the two induction groups in age or sex. The mean time (±SD) between induction and skin incision was 28.1 ± 6.6 min for group 1, 29.5 ± 6.8 min for group 2, and 29.2 ± 6.8 for the combined groups. The minimum alveolar concentration of halothane required to prevent movement with skin incision in 50% of subjects (MAC) was 0.52% for group 1, 0.51% for group 2, and 0.52% for pooled data. Because the two induction groups were similar, we pooled these data for the subsequent analysis.

In figure 1 movement following skin incision was plotted against the end-tidal halothane concentration. The relationship between movement and LEC activity immediately prior to skin incision is presented in figure 2. There was a statistically significant relationship in both induction groups between the probability of movement and the end-tidal halothane concentration ($r^2 = 0.81$ for group 1, 0.80 for group 2, and 0.91 for pooled data, P < 0.01). The biserial correlation coefficient for move-

FIG. 1. Patients moving or not moving in response to skin incision at different end-tidal halothane concentrations (each circle represents one patient). Closed circles represent patients with halothane induction and open circles represent thiopental induction patients. The position of the circle on the horizontal axis represents the end-tidal halothane concentration at which skin incision occurred.



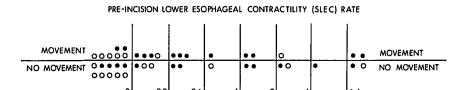


FIG. 2. Movement during skin incision plotted against the SLEC prior to skin incision. Each circle represents one patient. Closed circles represent halothane induction patients and open circles represent thiopental induction patients. The position of the circle on the horizontal axis represents the SLEC at 1 min prior to incision. The position of the circle above or below the line indicates movement or no movement with skin incision.

ment/no movement response *versus* end-tidal halothane concentration was 0.82 (P < 0.05).

There was no obvious relationship between movement and LEC activity in either group or in pooled data $(r^2 = 0.12$ for group 1, 0.35 for group 2, 0.19 for pooled data P > 0.05). The biserial correlation coefficient for movement/no movement response versus pre-incision SLEC was 0.01 (P > 0.05). We conclude that the SLEC prior to skin incision did not predict patient movement.

There was no significant correlation between the preincision SLEC and end-tidal halothane concentration ($r^2 = 0.006$, P = 0.62). However, there was a positive correlation between movement during skin incision and the SLEC 1 min after skin incision (fig. 3). Only 5 of 25 with a postincision SLEC value < 0.5/min moved with surgical stimulus, representing a significant difference in LEC activity between those who moved *versus* nonmovers, P < 0.05. Movement with skin incision had a statistically significant relationship to hemodynamic responses (*i.e.*, >15% increase in heart rate and/or mean arterial pressure measured 1 min after skin incision). Thus, purposeful movement with skin incision was associated with cardiovascular responses and with an increased esophageal motility (table 1).

During emergence from anesthesia there was no obvious relationship between the SLEC at the time when nitrous oxide was discontinued and the time to awakening. Finally, increases in heart rate and mean arterial pressure preceded SLEC changes during emergence from anesthesia (table 2).

Discussion

The lower third of the esophagus in humans contains smooth muscle innervated by the myenteric plexus.⁸

Esophageal contractions are of three general types: 1) primary peristaltic contractions to propel food to the stomach; 2) secondary contractions following mechanical dilation of the esophagus; and 3) tertiary nonpropulsive contractions limited to the smooth muscle. These tertiary contractions are usually spontaneous but can also be triggered by emotional stress or sounds. 9,10 Additionally, tertiary contractions are absent in brain dead patients. 11

Potent inhalation agents cause a dose-dependent suppression of spontaneous tertiary contractions in the lower esophagus.¹⁻⁴ Hence, Evans *et al.*³ suggested that monitoring esophageal contractility may be a useful method to judge the depth of anesthesia. Our study also indicated that there is a correlation between the postincision SLEC, patient movement following skin incision, and cardiovascular responses to surgical stimuli. These data are consistent with the concept that tertiary esophageal contractions represent a visceral autonomic response to noxious surgical stimuli. Other manifestations of this response include sudomotor (sweating), hemodynamic, and stress hormone changes.

Ideally, a depth of anesthesia monitor would provide information before the appearance of commonly accepted clinical signs of light anesthesia (patient movement, tearing, sweating, hypertension, tachycardia, and tachypnea). In the current study movement and cardiovascular changes with skin incision preceded changes in the SLEC by 15–30 s.

The time course of SLEC responses to surgical stimuli limits the clinical usefulness of the LECTRON 302 monitor in children undergoing nitrous oxide—halothane anesthesia. The delayed response of esophageal motility to surgical stimuli noted in our study is similar to the response of electromyographic (EMG) and electroen-



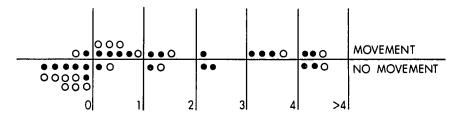


FIG. 3. Movement during skin incision plotted against the SLEC rate 1 min after skin incision. The position of the circle on the horizontal axis and above or below the horizontal line are the same as in figure 2. Each circle represents one patient. Open circles represent thiopental induction patients and closed circles represent halothane induction patients.

TABLE 1. Relationship Between Movement, SLEC, and Hemodynamic Responses to Skin Incision

Movement Responses	SLEC Responses	Hemodynamic Responses		
	Increase in SLEC Rate (contractions/min)	% Increase in Heart Rate	% Increase in Mean Arterial Blood Pressure	
Movement No movement	0.85 ± 0.3* 0.30 ± 0.3	28.8 ± 5.1* 7.9 ± 4.8	23.1 ± 2.9* 9.1 ± 2.7	

Values are mean ± SEM. All hemodynamic and SLEC responses represent the change in value of the measurements at 1 min after skin incision compared with pre-incision values.

cephalographic (EEG) activity during nitrous oxidemethohexital anesthesia. When a Datex Anesthesia Brain Monitor (Puritan Bennett Corp., Kansas City, Missouri) was used to record intraoperative EMG and EEG activity, there was a significant increase in the EMG values in patients who moved with surgical stimuli. However, the EMG changes occurred 5–10 s after patient movement. In a more recent study, there was no correlation between the EEG spectral edge frequency and hemodynamic responses to surgical stimuli during propofol–nitrous oxide anesthesia. Is

A depth of anesthesia monitor should also predict patient response to surgical stimuli. Although Sessler et al. reported that SLEC activity predicted movement in response to skin incision in adults undergoing halothane anesthesia,4 we were unable to confirm this finding in the children we studied. Our study differs from that of Sessler et al.4 in several important respects. First, our study was limited to children, and they may differ from adults in their esophageal contractile responses to anesthesia. Second, we administered nitrous oxide with halothane. Third, we used a lower contraction threshold of 10 mmHg compared with 15 mmHg in the previous adult study⁴ because the peak pressures caused by esophageal contractions are lower in children than in adults.14 Fourth, we did not use any muscle relaxants to facilitate tracheal intubation.

The recorded SLEC activity will be increased when a lower contraction threshold is used and also by movement artifact in the absence of neuromuscular blockade. Interestingly, some patients moved despite the fact that they had a SLEC of zero before skin incision. Yet, all of these patients had an increase in the SLEC immediately after the skin incision, suggesting that the pressure sensing esophageal balloon had not passed into the stomach, where esophageal contractions could not be detected.

Sessler et al.⁴ and Erickson et al.¹⁵ have shown that the predictability of SLEC monitoring varies with the type of anesthetic agent used. Although Sessler et al.⁴ used nitrous oxide with halothane for induction, the nitrous oxide was discontinued after only 3 min. Our patients received nitrous oxide with halothane throughout the operation. The inability of SLEC to predict patient response in our study may be a reflection of an increased suppression of esophageal contractility by low (sub-MAC) doses of halothane in children compared with adults, or it may represent the effects of nitrous oxide on esophageal contractility. There are no data on the effects of nitrous oxide on SLEC in adults. Nevertheless, SLEC activity has not been shown to predict patient movement with surgical stimuli in patients undergoing nitrous oxide and alfentanil anesthesia.⁴

In our study, I 1 of 18 patients receiving thiopental for induction had an SLEC of zero prior to skin incision. The larger number of cases with a pre-incision SLEC of zero in the thiopental induction group might represent a greater suppression of esophageal contractility by thiopental in children compared with adults. ^{1-4,15} However, there was no correlation between patient movement and the SLEC activity before skin incision in patients who received or did not receive thiopental. The mean MAC values for both induction groups were similar, although the slopes differed. In addition, the skin incision occurred at least 20 min after the induction dose of thiopental. The time interval between induction and skin incision may explain the relative lack of effect of thiopental on the mean MAC values.

Some of our patients had two separate skin incisions during the same operation. Gregory *et al.*¹⁶ have shown that this does not alter MAC. The variation in our value

TABLE 2. Changes in SLEC, Heart Rate, and Mean Arterial Blood Pressure during Emergence from Anesthesia

Minutes after N ₂ O discontinued No. of cases*	0 38	1 38	2 33	3 22
Mean SLEC (±SEM) (contractions/min)	1.2 ± 0.5	1.3 ± 0.4	1.9 ± 0.5†	2.3 ± 0.6†
Mean heart rate (±SEM) (beats/min)	83 ± 4	101 ± 4†	111 ± 4†	116 ± 2†
Mean arterial blood pressure (±SEM) (mmHg)	81 ± 2	97 ± 2†	99 ± 3†	99 ± 3†

^{*} Because SLEC was recorded only until patient coughed, the number of cases for which SLEC data are available decreases as time increases.

^{*} P < 0.05 for subjects with purposeful movement *versus* no movement on skin incision.

 $[\]dagger P < 0.05$ compared with time 0 value.

of MAC and that obtained by Gregory et al. 16 is in keeping with the known effects of nitrous oxide on MAC and the inherent error of the "up-and-down" method, when the end-tidal concentrations are varied by 0.1%. $^{16-18}$

Given the need for a reliable depth of anesthesia monitor during "balanced" anesthesia, it has been suggested that SLEC monitoring might be particularly useful during nitrous oxide-opioid-relaxant anesthesia. Some experts have suggested that although esophageal activity may imply inadequate anesthesia, anesthesiologists cannot be certain that the absence of esophageal motility means the patient is adequately anesthetized and will have no recall of intraoperative events. ¹⁹ The clinical usefulness of SLEC monitoring will have to be determined for each of the commonly used anesthetic techniques. These studies should also be performed in children because their responses may be significantly different from those of adults.

We conclude that SLEC does not predict patient movement in unparalyzed children anesthetized with halothane and nitrous oxide.

The authors thank Dan Kuni and American Antec for providing the LECTRON 302 monitor and probes, the nurses and physicians of St. Louis Children's Hospital for their patience during this study, and Mary Bicknell for secretarial assistance in preparation of the manuscript.

References

- Evans JM, Bithell JF, Vlachnikolis IG: Relationship between lower esophageal contractility, clinical signs and halothane concentration during general anaesthesia and surgery in man. Br J Anaesth 59:1346-1355, 1987
- Maccioli GA, Kuni DR, Silvay G, Evans JM, Calkins JM, Kaplan J: Response of lower esophageal contractility to changing concentrations of halothane or isoflurane: A multicenter study. J Clin Monit 4:247-255, 1988
- 3. Evans JM, Davies WL, Wise CC: Lower oesophageal contractility:
 A new monitor of anaesthesia. Lancet 1:1151-1154, 1984
- Sessler DI, Stoen R, Olofsson CI, Chow F: Lower esophageal contractility predicts movement during skin incision in patients

- anesthetized with halothane, but not with nitrous oxide and alfentanil. ANESTHESIOLOGY 70:42–46, 1989
- Strobel CT, Byrne WJ, Ament ME, Euler AR: Correlation of esophageal length with height: Application to the Tuttle test without prior esophageal manometry. J Pediatr 94:81–84, 1979
- Quasha AL, Eger EI II, Tinker JH: Determination and applications of MAC. ANESTHESIOLOGY 53:315–334, 1980
- Dixon WJ: Quantal response variable experimentation: The upand-down method. Statistics in Endocrinology, Proceedings. Edited by McArthur JW, Colton T. Cambridge, MIT Press, 1970, pp 251-267
- Weisbrodt NW: Neuromuscular organization of esophageal and pharyngeal motility. Arch Intern Med 136:524-531, 1976
- Stacher G, Schmierer G, Landgraf M: Tertiary esophageal contractions evoked by acoustical stimuli. Gastroenterology 77:49– 54, 1979
- Rubin J, Nagler R, Spiro HM, Pilot ML: Measuring the effect of emotions on esophageal motility. Psychosom Med 24:170–176, 1962
- Sinclair ME, Suter PM: Lower oesophageal contractility as an indicator of brain death in paralyzed and mechanically ventilated patients with head injury. Br Med J 294:796–803, 1987
- Chang T, Dworsky WA, White PF: Continuous electromyography for monitoring depth of anesthesia. Anesth Analg 53:315–334, 1980
- White PF, Boyle WA: Relationship between hemodynamic and electroencephalographic changes during general anesthesia. Anesth Analg 68:177-181, 1989
- Boix-Ochoa J, Canals J: Maturation of the lower esophagus. J Pediatr Surg 11:749–756, 1976
- Erickson JP, Foss J, Kuni DR: A controlled trial of the efficacy of lower esophageal contractility as a depth of anesthesia monitor (abstract). ANESTHESIOLOGY 67:A672, 1987
- Gregory GA, Eger EI II, Munson ES: The relationship between age and halothane requirements in man. ANESTHESIOLOGY 30: 488–491, 1969
- Stevens WC, Dolan WM, Gibbons RT, White A, Eger EI II, Miller RD, DeJong RH, Elashoff RM: Minimum alveolar concentrations of isoflurane with and without nitrous oxide in patients of various ages. ANESTHESIOLOGY 42:197–200, 1975
- Eger EI II: Anesthetic Uptake and Action. Baltimore, Williams & Wilkins, 1974, p 3
- Prys-Robert C: Anaesthesia: A practical or impractical construct (editorial). Br J Anaesth 59:1341-1345, 1987