35. David MP, Bergman A, Delighdish L: Cervico-isthmic pregnancy carried to term. Obstet Gynecol 56:247–252, 1980
36. Studdiford WE: Cervical pregnancy: A partial review of the literature and a report of two probable cases. Am J Obstet Gynecol 49:169–185, 1945
37. Baptisti A: Cervical pregnancy. Obstet Gynecol 1:353–358, 1953
38. Plumer MH: Bleeding problems, Obstetric Anesthesia: The Complicated Patient. Edited by James FM, Wheeler AS. Philadelphia, FA Davis Co, 1982, pp 185–207
39. Clark SL, Yeh S, Phelan JP, Bruce S, Paul RH: Emergency hysterectomy for obstetric hemorrhage. Obstet Gynecol 64:376–380, 1984
40. Shnider SM, Levinson G: Anesthesia For Obstetrics. Baltimore, Williams & Wilkins, 1987, pp 159–178

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Intravenous Lidocaine Reduces the Incidence of Vomiting in Children after Surgery to Correct Strabismus

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One of the authors (GLR) noticed that the incidence and severity of vomiting after surgery to correct strabismus had decreased in the past few years. The turnabout seemed to coincide with a change in anesthetic technique: the routine administration of iv lidocaine in place of succinylcholine to facilitate endotracheal intubation for short procedures during halothane anesthesia.

We had initially utilized lidocaine to treat—and, subsequently, to prevent—dysrhythmias generated by succinylcholine and airway instrumentation in lightly anesthetized children. Eventually, we realized that tracheal intubating conditions in most patients receiving lidocaine iv without succinylcholine were excellent.

We compared randomly chosen records of 50 patients from 1979 who had had surgery to correct strabismus, and who had received succinylcholine to facilitate intubation of the trachea, to the records of 50 similar patients in 1986 who had received lidocaine instead of succinylcholine. We found that the incidence of postoperative emesis had decreased from 54% to 28%. However, there were enough other variables to warrant a controlled study to substantiate this observation, which is described below.

MATERIALS AND METHODS

The Human Subjects Research Committee approved our project, and each parent/guardian signed a consent form. The subjects were limited to ASA I outpatients undergoing surgery to correct strabismus, aged 18 months through 7 yr. Seventy-five consecutive children who met study criteria were randomized into three groups of 25 by using sealed envelopes: group I received lidocaine, 2 mg/kg, 90 s prior to laryngoscopy; group II received iv succinylcholine, 1 mg/kg, 1 min prior to laryngoscopy; group III received both lidocaine and succinylcholine (same doses and timing). Group III was added to determine if succinylcholine would negate any proposed positive effect from lidocaine.

All received an oral solution containing 0.02 mg/kg of atropine sulfate, 0.75 mg/kg of promethazine, and 25 mg/kg of chloral hydrate 1 h before surgery. Anesthesia was induced with a halothane (2.5%)-nitrous oxide (70%)-oxygen mixture; drugs used to facilitate tracheal intubation were administered when the child reached a light surgical plane. Respirations were assisted in all, and then controlled with the onset of paralysis in the two groups receiving succinylcholine. All children were hyperventilated for several seconds just prior to laryngoscopy: this was necessary in the "lidocaine only" group to abolish inherent respiratory drive during laryngoscopy and tracheal intubation. Anesthesia was maintained with a halothane (1.5–2%)-nitrous

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TABLE 1. Patient Characteristics

Group	Sex (F/M)	Age in Years (Mean ± SD)	Weight in kg (Mean ± SD)
I. Lidocaine	12/13	4.6 ± 1.8*	20.8 ± 6.6*
II. Succinylcholine	13/12	3.4 ± 1.8	15.8 ± 4.9
III. Lidocaine and Succinylcholine	12/13	3.6 ± 1.6	16.1 ± 3.7

* Significantly different from groups II and III.

oxide (50%)-oxygen mixture. Respirations were assisted or controlled during the procedure, and the endotracheal tube removed before the cough reflex returned. The stomach was not routinely aspirated. All received a minimum of 25 ml/kg of intravenous fluids (5% dextrose in Lactated Ringer's solution) in the perioperative period.

Tabulated data included sex, age, weight, number of muscles for repair, time of day, iv fluid totals, duration of anesthesia, post-anesthesia hospital time, postoperative emesis details, NPO times, history of motion sickness or previous strabismus surgery, and heart rate changes of 10% or more after administering succinylcholine and/or lidocaine, or following extraocular muscle traction.

Nurses and parents were instructed to allow the child to sleep undisturbed postoperatively. As recommended by Berry,[1] oral fluids were not offered until the child expressed hunger. Vomiting/retching episodes occurring in the recovery room were reported by the attending nurse. Following discharge from the recovery room, details of emesis (time, place, color, amount, position, and activity of the child) and the times when liquids and solids were first retained were recorded on a worksheet by the parent/guardian. This information was obtained by a phone call to the home at least 24 h after discharge.

Statistical significance ($P < 0.05$) was determined by one-way analysis of variance (ANOVA) for between-groups analyses of age, weight, iv fluid totals, duration of anesthesia, post-anesthesia hospital time, number of emetic episodes per child, and NPO times. Chi-squares or Fisher's exact tests were performed for between-groups analyses of non-parametric data.

RESULTS

Distribution of the sexes was similar in the three groups. Although patients in groups II and III were similar in ages and weights, patients in group I were significantly older and heavier than those in groups II and III (table 1). There were no significant differences among the three groups in the number of muscles operated on, time of day, amount of iv fluids (average mean of the three groups = 30.3 ml/kg ± 1.9 SD), duration of anesthesia (43.2 min ± 8.9 SD), and post-anesthesia hospital time (3.6 h ± 0.7 SD).

The incidences of vomiting were 16% in group I, 52% in group II, and 20% in group III; groups I and III were significantly different from group II. Approximately one-half of the emetic episodes occurred following discharge (table 2). Prior to discharge, eight patients expressed hunger and all retained fluids. After discharge, only five of the remaining 67 vomited after their initial fluids. The three groups had similar NPO times both prior to and following surgery: the mean time from induction of anesthesia to first successful oral intake averaged 6.5 h ± 2.7 SD.

The incidence of oculocardiac reflex (a sudden change in heart rate of 10% or more secondary to extraocular muscle traction) was comparable in all three groups (25% overall); 32% of emetic patients had an oculocardiac reflex, and 37% of patients with an oculocardiac reflex vomited. Following the administration of lidocaine, one patient exhibited a transient decrease in heart rate which did not require treatment. An unexpected finding was the alteration of a primarily decreased response in heart rate to succinylcholine, to a

TABLE 2. Postoperative Emesis Data

Group	Incidence Number (%)	Number of Episodes per Emetic Child (Mean ± SD)	Total Number of Emetic Episodes	Place of Occurrence		
				Hospital	Car	Home
I. Lidocaine	4 (16%)*	1.75 ± 0.5	7	43%	0%	57%
II. Succinylcholine	13 (52%)	2.23 ± 1.7	29	48%	24%	28%
III. Lidocaine and Succinylcholine	5 (20%)*	2.40 ± 1.5	12	50%	25%	25%

* Significantly different from group II.

TABLE 3. Number (%) of Patients with Heart Rate Changes of 10% or More Following Succinylcholine

Group	Decrease	Increase
II. Succinylcholine	8 (32%)	1 (4%)
III. Lidocaine & Succinylcholine	2 (8%)	6 (24%)

Group II is significantly different from group III.

primarily increased one when preceded by lidocaine (table 3). This difference was statistically significant.

The lowest incidence of emesis (12.5%) occurred in females of groups I and III who received lidocaine (males = 30%), while the highest incidence (66.7%) was in males of group II who received succinylcholine only (females = 38.5%). Although not statistically significant, within all three groups, the incidence of vomiting was greater in males, and slightly greater in older children, afternoon cases, and those with a longer anesthetic. There was no consistent correlation with histories of motion sickness or previous strabismus surgery. Only one patient (group II) required treatment for emesis with rectal promethazine.

Discussion

Surveys of postoperative emesis in children confirm that patients undergoing surgery to correct strabismus have a high rate: incidences of 50%,[2] 56%,[3] and 76%[4] have been reported. Most controlled studies attempting to diminish vomiting in children have centered on the iv use of droperidol,[5-8] a dopamine antagonist which acts primarily at the chemoreceptor trigger zone. Only one study[5] attained an acceptable incidence of emesis; namely, 16%. We accomplished the same with lidocaine.

Why do patients vomit following surgery to correct strabismus? Possibly extraocular muscle traction stimulates an "oculogastric reflex."[9] Iwamoto and Schwartz[10] suggested that the role of surgical manipulation of the eye needed further attention, and that vomiting might be correlated with the oculocardiac reflex (we found no such correlation). Lerman *et al.*[5] also implicated eye manipulation: they achieved a 16% emesis rate by giving droperidol (75 μg/kg iv) upon induction of anesthesia, and cited another study[8] where the same dose given after surgery commenced was much less effective (43% incidence). Gibbons *et al.*[11] postulated a form of motion sickness resulting from acute eye imbalance.

In our opinion, both eye manipulation and eye imbalance are important factors in the genesis of emesis following surgery to correct strabismus. Human extrinsic eye muscles are richly endowed with somatic sensory receptors;[12] stretching and trauma to the eye muscles incurred by the surgical procedure lead to increased afferent activity. Upon awakening, there is even more sensory input, especially in the child with unpatched eyes who perceives a visual image different from that in his neural "store," and tries to correct it.

Information that coordinates eye movements in response to both labyrinthine and non-labyrinthine proprioceptor stimulation is carried from the vestibular nuclei (VIII) to the nuclei of the extrinsic eye muscles (III, IV, and VI) in the medial longitudinal fasciculi. These same tracts also carry proprioceptive messages from the extrinsic eye muscles to the vestibular nuclei.[13] Thus, there is a powerful convergence of information from the eye to the vestibular structures;[14] unfortunately, the latter are closely involved anatomically with the vomiting center in the brainstem reticular formation.[15] The vestibular and vomiting centers contain abundant muscarinic cholinergic receptors,[16] and increased activity in the vestibular system may radiate through adjacent reticular areas to activate the vomiting center.[17] Other analogous situations probably utilizing the same pathways include vomiting induced by acute nystagmus, or motion sickness caused by visual rather than labyrinthine stimulation (optokinetically induced motion sickness).

We can only speculate how lidocaine might temper the emetic response. Perhaps it simply blocks extraocular muscle sensory input peripherally. Our observation that the administration of lidocaine prior to succinylcholine altered the heart rate response to the latter drug may be significant, and suggests that lidocaine may modify peripheral muscarinic cholinergic activity induced by succinylcholine. It follows that lidocaine might also modify increased central muscarinic cholinergic activity in the reticular formation. In the awake patient, diplopia and disturbed color perception are symptoms of lidocaine toxicity,[18] implying that visual information can be modulated by the drug. And, finally, lidocaine may somehow enhance the central anticholinergic effects of atropine and promethazine given preoperatively; the latter is a very effective antimotion sickness drug,[17] and is frequently used to treat emesis following surgery to correct strabismus. Exactly how, when, and where lidocaine acts requires further investigation.

The fact that patients in group I were significantly older (and, thus, heavier) than those in groups II and III might be construed as a weakness in our study. However, within all three groups, the average age of those children who vomited was greater than those who did not. We might, therefore, expect that, of the two groups (I and III) receiving lidocaine, the older one should have a higher incidence of vomiting. Actually, the opposite was true. For this reason, we feel that the age and weight differences do not invalidate the study.

Our choice of a 2 mg/kg dose of iv lidocaine was initially based on a report advocating lidocaine to facilitate tracheal extubation.[19] We have had no problems with the bolus administration of this dose in the healthy child lightly anesthetized with halothane, but cardiac toxicity in an infant has been reported.[20] Modification of both dose and rate of administration should be considered in the child with a compromised cardiovascular system.

To summarize, in healthy children aged 18 months through 7 yr, premedicated with atropine sulfate, promethazine, and chloral hydrate, and undergoing a halothane, nitrous oxide, and oxygen anesthetic for surgery to correct strabismus, the incidence of postoperative emesis is significantly less with the use of lidocaine (2 mg/kg iv) to facilitate laryngoscopy and tracheal intubation than with the use of succinylcholine (1 mg/kg iv). Lidocaine exerts a beneficial effect whether or not succinylcholine is also administered. Generous iv hydration, minimal visual and vestibular stimulation in the postoperative period, and abstention from oral intake until the child reaches its "hunger point" probably contribute to reducing both the frequency and severity of emesis, even in the absence of lidocaine.

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References

1. Berry FA: The child in the recovery room, Anesthetic Management of Difficult and Routine Pediatric Patients. Edited by Berry FA. New York, Churchill Livingstone, 1986, pp 425–440
2. Weinstock SM, Flynn JT: Brief hospital admissions for pediatric strabismus surgery. Am J Ophthalmol 80:525–529, 1975
3. Hadaway EG, Ingram RM, Traynar MJ: Day case surgery for strabismus in children. Trans Ophthalmol Soc UK 97:23–25, 1977
4. Rowley MP, Brown TCK: Postoperative vomiting in children. Anaesth Intensive Care 10:309–313, 1982
5. Lerman J, Eustis S, Smith DR: Effect of droperidol pretreatment on postanesthetic vomiting in children undergoing strabismus surgery. ANESTHESIOLOGY 65:322–325, 1986
6. Hardy JF, Charest J, Girouard G, Lepage Y: Nausea and vomiting after strabismus surgery in preschool children. Can Anaesth Soc J 33:57–62, 1986
7. Abramowitz MD, Elder PT, Friendly DS, Broughton WL, Epstein BS: Antiemetic effectiveness of intraoperatively administered droperidol in pediatric strabismus outpatient surgery—Preliminary report of a controlled study. Pediatr Ophthalmol Strabismus 18:22–27, 1981
8. Abramowitz MD, Oh TH, Epstein BS, Ruttimann UE, Friendly DS: The antiemetic effect of droperidol following outpatient strabismus surgery in children. ANESTHESIOLOGY 59:579–583, 1983
9. McGoldrick RE: Pediatric anesthesia for ophthalmic surgery, Anesthesia for Ophthalmology. Edited by Bruce RA, McGoldrick RE, Oppenheimer P. Birmingham, Aesculapius Publishing Co., 1982, pp 51–83
10. Iwamoto K, Schwartz H: Antiemetic effect of droperidol after ophthalmic surgery. Arch Ophthalmol 96:1378–1379, 1978
11. Gibbons PA, Nicolson SC, Betts EK, Rosenberry KR, Jobes DR: Scopolomine does not prevent post-operative emesis after pediatric eye surgery (abstract). ANESTHESIOLOGY 61:A435, 1984
12. Cooper S, Daniel PM, Whitteridge D: Muscle spindles and other sensory endings in the extrinsic eye muscles; The physiology and anatomy of these receptors and of their connexions with the brain-stem. Brain 78:564–583, 1955
13. Bach-y-Rita P: Neurophysiology of eye movements, The Control of Eye Movements. Edited by Bach-y-Rita P, Collins CC, Hyde JE. New York, Academic Press, 1971, pp 7–46
14. Brandt T, Dichgans J, Wagner W: Drug effectiveness on experimental optokinetic and vestibular motion sickness. Aerospace Med 45:1291–1297, 1974
15. Barnes JH: The physiology and pharmacology of emesis. Mol Aspects Med 7:397–508, 1984
16. Peroutka SJ, Snyder SH: Antiemetics: Neurotransmitter receptor binding predicts therapeutic actions. Lancet 1:658–659, 1982
17. Wood CD: Antimotion sickness and antiemetic drugs. Drugs 17:471–479, 1979
18. Foldes FF, Molloy R, McNall PG, Koukal LR: Comparison of toxicity of intravenously given local anesthetic agents in man. JAMA 172:1493–1498, 1960
19. Baraka A: Intravenous lidocaine controls extubation laryngospasm in children. Anesth Analg 57:506–507, 1978
20. Garner L, Stirt JA, Finholt DA: Heart block after intravenous lidocaine in an infant. Can Anaesth Soc J 32:425–428, 1985