Serum gastrin and blood glucose levels during halothane-nitrous oxide anaesthesia and strabismus surgery in children

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The purpose of this study was to determine whether serum gastrin levels are increased by reflexogenic stimuli applied to the extrinsic muscles of the eye. Serum gastrin and blood glucose concentrations were measured in ten normal children aged between 5 and 12 yr during general anaesthesia with halothane and nitrous oxide and during strabismus surgery. Fasting basal concentrations of gastrin ($33.6 \pm 14.8 \text{ pg} \cdot \text{ml}^{-1}$) and of glucose ($4.43 \pm 0.72 \text{ mmol} \cdot \text{L}^{-1}$) were in the normal range of values for children. Intravenous atropine ($0.01 \text{ mg} \cdot \text{kg}^{-1}$), general anaesthesia with halothane in nitrous oxide and oxygen by mask for three minutes, tracheal intubation, extraocular muscle stimulation and surgical stress did not cause any variation in the mean serum gastrin concentration. On the contrary, tracheal intubation and surgical stress increased blood glucose concentrations ($P < 0.05$). There was no difference in the serum gastrin levels after extraocular muscle stimulation between children with positive or negative oculocardiac reflexes ($44.5 \pm 16.7 \text{ pg} \cdot \text{ml}^{-1}$ vs $38 \pm 14.7 \text{ pg} \cdot \text{ml}^{-1}$, respectively). The incidence of vomiting pre-discharge was 60 per cent. Serum gastrin levels did not differ between children who vomited and children who did not ($44.3 \pm 18.5 \text{ pg} \cdot \text{ml}^{-1}$ vs $47.1 \pm 16.9 \text{ pg} \cdot \text{ml}^{-1}$, respectively). Vomiting after strabismus

surgery cannot be attributed to high gastrin serum levels. Consequently, it is unlikely that vomiting after strabismus surgery is linked to an "oculogastric reflex" with the vagus nerve as the efferent pathway.

Cette étude a été conçue dans le but de vérifier si les concentrations sériques de gastrine se trouvent augmentées après la stimulation des muscles extrinsèques de l'œil. Les concentrations sériques de gastrine, de même que les concentrations sanguines de glucose, furent mesurées, chez dix enfants normaux, âgés de 5 à 12 ans, durant l'anesthésie générale à l'halothane et au protoxyde d'azote et durant la chirurgie pour correction de strabisme. La gastrinémie basale et à jeun ($33,6 \pm 14,8 \text{ pg} \cdot \text{ml}^{-1}$) et la glycémie basale et à jeun ($4,43 \pm 0,72 \text{ mmol} \cdot \text{L}^{-1}$) étaient dans les limites des valeurs normales pour ce groupe d'âge. L'administration d'atropine IV ($0,01 \text{ mg} \cdot \text{kg}^{-1}$), l'anesthésie générale à l'halothane et au protoxyde d'azote par masque pendant trois minutes, l'intubation trachéale, la stimulation des muscles extraoculaires et le stress chirurgical n'ont pas causé de variations significatives dans les concentrations sériques de gastrine. Par contre, l'intubation trachéale et le stress chirurgical ont causé des augmentations significatives des concentrations de glucose dans le sang ($P < 0,05$). Il n'y a pas eu de différence significative, après la stimulation des muscles extrinsèques de l'œil, entre la gastrinémie moyenne des enfants qui ont présenté un réflexe oculocardiaque positif et la gastrinémie moyenne des enfants dont le même réflexe était négatif ($44,5 \pm 16,7 \text{ pg} \cdot \text{ml}^{-1}$ et $38 \pm 14,7 \text{ pg} \cdot \text{ml}^{-1}$, respectivement). L'incidence de vomissements post-opératoires, avant le congédiement de l'hôpital, était égale à 60 pour cent. La gastrinémie n'était pas significativement différente entre les enfants qui ont vomi et ceux qui n'ont pas vomi ($44,3 \pm 18,5 \text{ pg} \cdot \text{ml}^{-1}$ et $47,1 \pm 16,9 \text{ pg} \cdot \text{ml}^{-1}$, respectivement). Les vomissements post-correction de strabisme ne peuvent pas être attribués aux variations de la gastrine sérique. Par conséquent, il est fort improbable que les vomissements post-correction de strabisme soient dus à un réflexe « oculo-gastrique » dont le vague serait la voie éfferente.

Key words

ANAESTHESIA: paediatric;
BLOOD: glucose;
HORMONES: gastrin;
SURGERY: ophthalmological;
VOMITING.

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Strabismus surgery implies the manipulation of one or several extraocular muscles. These muscles are richly endowed with sensory receptors.¹ Consequently, strabismus correction is associated with a high incidence of oculocardiac and oculo-respiratory reflexes, particularly when highly reflexogenic stimuli (type A or square wave tractions) are applied to the extrinsic muscles of the eye.²⁻³ Moreover, 62.5 to 85 per cent of the children not receiving any antiemetic vomit after strabismus surgery.⁴⁻⁶ It has been suggested that this high incidence of vomiting is related to an "oculogastric reflex"⁷ or to an "oculo-emetic reflex".⁸ The aetiology of vomiting following strabismus surgery remains unknown.

The present study was designed to determine whether serum gastrin levels were increased by reflexogenic stimuli applied to the extraocular muscles. The rationale for this study was as follows. If stimulation of an extrinsic muscle of the eye causes vagal stimulation of the stomach, as it causes stimulation of the cardiac depressor nerve, then it may cause vagal stimulation of gastrin release and an increase of the serum gastrin concentration. It is known that gastrin as well as synthetic pentapeptide (pentagastrin) inhibit gastric emptying, in dogs, at physiological doses.⁹⁻¹¹ Gastrin, cholecystokinin and opiates relax the proximal third of the stomach, increase the contractility of the antrum, and increase pyloric and duodenal contractility and pressure resulting in inhibition of the gastric emptying with nausea and vomiting.¹²⁻¹³ A dopaminergic mechanism for gastrin release was suggested in dogs when secretion of gastrin by apomorphine was suppressed by prior treatment with haloperidol.¹⁴ Central inhibition of gastric motility has been suggested by using carotid infusions of pentagastrin in sheep.¹⁵

As gastrin release may be affected by blood glucose levels⁹ we decided to minimize the blood glucose variations by using a glucose-free fluid regimen (lactated Ringer's solution) and measured the blood glucose levels occurring during general anaesthesia and strabismus correction.

There are no previous reports of serum gastrin levels during general anaesthesia and surgery.

Methods

With approval of the hospital Committee on Medical Ethics, informed consent was obtained from the parents of ten children (six girls and four boys), ASA physical status I, between 5-12 yr, and requiring correction of strabismus under general anaesthesia.

Children were fasted 10-15 hr. No premedication was given. The following monitors were used in all children: precordial stethoscope, continuous ECG (CM-5), blood pressure measurements with an automatic cuff (Dinamap®), pulse oximeter (Ohmeda®), capnograph (Siemens-

TABLE I Numbers and times for the blood samples

Sample number	Time of the blood sample
1	Control or basal levels - before any medication
2	3 min after atropine IV (0.01 mg · kg ⁻¹)
3	3 min after induction of anaesthesia
4	3 min after tracheal intubation
5	15 min after tracheal intubation
6	3 min after extraocular muscle stimulation
7	Immediately before tracheal extubation (15-41 min after extraocular muscle stimulation)

Sirecust 322®) and rectal thermometer. After the insertion of an IV cannula a first blood sample (2 ml) was collected from a peripheral vein in order to obtain the control (basal) values for serum gastrin and blood glucose. This IV cannula was kept patent by a heparin in saline solution (5 units · ml⁻¹). Intravenous atropine, 0.01 mg · kg⁻¹, was then given. After discarding the first 2 ml, six other blood samples for measurements of serum gastrin and blood glucose concentrations were collected at the times indicated in Table I. Induction of anaesthesia was achieved with halothane, nitrous oxide and oxygen (40 per cent) by mask. Anaesthesia was then maintained with halothane (1.5 per cent inspired), nitrous oxide and oxygen (40 per cent) by mask, with manually assisted ventilation, for three minutes. A second IV cannula was then inserted in the contralateral limb and connected to an infusion of lactated Ringer's solution (8 ml · kg⁻¹ · hour⁻¹). Tracheal intubation was performed, without the use of muscle relaxants, under deep halothane-oxygen anaesthesia. After intubation, anaesthesia was maintained with halothane (1.5 per cent inspired), nitrous oxide and oxygen (33-40 per cent), using an Ohio circle system with CO₂ absorption, and mechanically controlled ventilation was adjusted to maintain an expired carbon dioxide (PETCO₂) of 32-38 mmHg (4.26-5 kPa) and a peripheral oxygen saturation ≥98 per cent.

Incision of the conjunctiva was done by a fornix approach with minimal mobilization of the globe in order not to elicit the oculocardiac reflex (OCR). The dissection was kept to a minimum to avoid uncontrolled stimulation of the extraocular muscle. Finally, a muscle hook was gently engaged under the muscle tendon and a square wave traction of 200-250 g, lasting 20 sec, was applied as a standardized stimulus to the first extraocular muscle to be operated upon.² Ten extraocular muscles were so stimulated: three medial recti; four lateral recti; and three superior recti. Four children (two boys and two girls) had more than one muscle (two muscles) repaired. In these patients, the second extraocular muscle was very gently manipulated, using a very slow gradual traction (slow slope traction), in order not to elicit the OCR.² Positive

TABLE II Demographic data

	Boys	Girls
<i>n</i>	4	6
Age (yr)	8.5 ± 1	8.3 ± 3
Weight (kg)	30.4 ± 4.8	27.8 ± 6.3
Fasting time (hr)	12 ± 0.8	12.4 ± 0.8
Duration of anaesthesia (min)	57.5 ± 13.7	64.2 ± 6.3

Means ± SD.

OCR was defined as a sudden slowing of the heart rate ≥ 12 beats $\cdot$ min⁻¹ and/or cardiac arrhythmia secondary to extraocular muscle traction.²⁻³

Blood glucose determinations were performed using a reflectance meter technique (Glucometer®, Ames, Elkhart, IN).¹⁶ This is an accurate method for perioperative blood glucose measurements in children, when compared with the glucose-oxidase method, even at low blood glucose concentrations.¹⁷ For the purpose of this study hypoglycaemia was defined as a blood glucose concentration less than 3.3 mmol $\cdot$ L⁻¹ (60 mg $\cdot$ dl⁻¹).¹⁸ For the serum gastrin measurements, samples of blood were collected in vacuum-tubes and were centrifuged at 2000 rpm for 20 min at 8°C. Serum was separated immediately and frozen at -60°C until assayed. Serum gastrin concentration was measured using the gastrin radio-immunoassay kit provided by Becton-Dickinson (BD Kit Catalog No. 255017). This method uses an antibody with a high affinity and reactivity to sulphated and non-sulphated forms of gastrin-17 (G-17) and gastrin-34 (G-34). Measurements of serum gastrin for the same patient were performed in the same assay to avoid inter-assay imprecision. Sensitivity of this assay is 4.8 pg $\cdot$ ml⁻¹.

The tracheal tube was removed in the operating room, before the cough reflex returned. Then the lactated Ringer's solution was replaced by a five per cent dextrose and lactated Ringer's solution (3 ml $\cdot$ kg⁻¹). The incidences of vomiting were recorded by nurses in the recovery room and in the day care unit. Acetaminophen, 10 mg $\cdot$ kg⁻¹, per rectum was administered to any child who experienced pain after surgery. Intramuscular dimenhydrinate, 1 mg $\cdot$ kg⁻¹, was administered to any child with two or more episodes of vomiting $\cdot$ hour⁻¹.

Data were analyzed for statistical significance using one-way ANOVA, ANOVA for repeated measures followed by the Scheffé multiple comparisons test, and Fisher's exact test (two-tail) when appropriate. A *P* value < 0.05 was considered statistically significant.¹⁹⁻²⁰ Except in the figures, data are presented as means ± SD.

Results

The mean age, weight, fasting time and duration of

anaesthesia did not differ significantly between boys and girls (Table II). Also, the number of extraocular muscles repaired and the incidence of positive OCR were not significantly different when boys were compared with girls. Positive OCR (50 per cent) were observed during the first extraocular muscle stimulation and not during the second muscle manipulation. Positive OCR was always linked to sinus bradycardia and not to other types of cardiac arrhythmia. There was no difference in the serum gastrin levels after extraocular muscle stimulation between children with a positive or negative OCR (44.5 ± 16.7 pg $\cdot$ ml⁻¹ and 38 ± 14.7 pg $\cdot$ ml⁻¹ respectively).

Fasting and basal (control) concentrations of serum gastrin were in the normal range (33.6 ± 14.8 pg $\cdot$ ml⁻¹; maximum = 72 pg $\cdot$ ml⁻¹; minimum = 21 pg $\cdot$ ml⁻¹; normal = 20-160 pg $\cdot$ ml⁻¹).²¹ Fasting and basal (control) blood sugar concentrations (4.43 ± 0.72 mmol $\cdot$ L⁻¹) were normal for children five to twelve years old (normal = 3.3-5.5 mmol $\cdot$ L⁻¹).¹⁸

Intravenous atropine (0.01 mg $\cdot$ kg⁻¹), general anaesthesia with halothane in nitrous oxide and oxygen by mask for three minutes, and tracheal intubation did not cause any changes in the mean serum gastrin levels. Also, extraocular muscle stimulation with a standardized highly reflexogenic stimulus as well as surgical stress related to strabismus correction did not cause any variation in the serum gastrin concentrations (Figure 1). On the contrary, tracheal intubation and surgical stress caused significant increases in the blood glucose concentrations (Figure 2).

The incidence of vomiting predischarge (first four to six hours of the postoperative period) was 60 per cent. Three of the six children who vomited had a positive OCR; two of the four children who did not vomit had a positive OCR. The mean serum gastrin levels did not differ between children who vomited and children who did not vomit (Table III).

Discussion

This study did not show any changes in serum gastrin levels during general anaesthesia with halothane and nitrous oxide and/or during strabismus surgery. As we obtained negative results, we considered the possibility of having failed to detect significant changes in serum

TABLE III Serum gastrin levels (pg $\cdot$ ml⁻¹) after extraocular muscle stimulation in children who vomited and children who did not vomit during the predischarge postoperative period

	Children who vomited	Children who did not vomit
Blood sample #6	44 ± 19.8	39.4 ± 13.2
Blood sample #7	44.3 ± 18.5	47.1 ± 16.9

Means ± SD.

FIGURE 1 Serum gastrin levels during anaesthesia and strabismus surgery in children. Means (●) $\pm$ 95 per cent Scheffé Confidence Intervals for Means (I).

gastrin concentrations because of the sample size. However, power analysis¹⁹ of the ANOVA with repeated measures for the serum gastrin levels showed that using a power ≥ 0.88 , we found that highly reflexogenic stimuli (type A or square wave tractions of 200–250 g lasting 20 sec) applied to the extrinsic muscles of the eye² did not produce a significant increase of the serum gastrin levels even when a positive OCR was elicited. Consequently, it is unlikely that vomiting after strabismus surgery is related to central inhibition of gastric motility due to high serum levels of gastrin.¹⁵

The effects of vagal and cholinergic stimulation and inhibition on gastrin release are complex.⁹ Vagal stimulation of gastrin release is mediated by antral vagal fibres whereas vagal inhibition requires intact vagal fibres to the proximal stomach.^{9,22–24} Theoretically it is possible that extraocular muscle stimulation causes vagal inhibition of the proximal stomach and vagal stimulation of the antrum. But the gastrin-containing cells, G-cells, are concentrated in the glands of the atral mucosa⁹ so that it is difficult to admit that the stimulation of these cells does not increase the serum gastrin level. Vomiting after strabismus surgery is not related either with the incidence of positive OCR^{4,7} or with the serum gastrin levels. Accordingly, our results do not support the hypothesis

FIGURE 2 Blood glucose levels during anaesthesia and strabismus surgery in children. Means (○) 95 per cent Scheffé Confidence Intervals for Means (I). * $P < 0.05$ vs Sample #1, ● vs Sample #2, + vs Sample #3.

that an "oculogastric reflex" causes gastrin release and subsequent inhibition of gastric emptying.

This study showed that, in healthy children, intravenous atropine ($0.01 \text{ mg} \cdot \text{kg}^{-1}$) caused a slight although not significant increase of serum gastrin concentrations. Given the persistence of small variations in serum gastrin concentration after intravenous atropine, it seems unlikely that atropine blocked the effects of extraocular muscle stimulation (Figure 1). The pharmacological effects of atropine on gastrin release vary with the animal species and, in the same species, with the doses used. It has been reported that low doses of atropine enhance the gastrin response to feeding in dogs and enhance the gastrin release caused by insulin hypoglycaemia, sham feeding, and gastric distension in humans.⁹

We suspect that extraocular muscle stimulation, instead of enhancing gastrin release, directly stimulates dopaminergic receptors in the chemoreceptor trigger zone (CTZ) or in connection with the vomiting centre. Indeed, dopaminergic pathways, found to be inhibitory, are involved in the control of gastric emptying^{12,25} and in the pathophysiology of vomiting.²⁶ Levodopa delays gastric emptying in humans.²⁷ Intravenous droperidol, an anti-dopaminergic antiemetic, seems more effective when

given ten minutes before than after manipulation of the eye.^{6,28-29} Moreover, metoclopramide and promethazine, two dopamine antagonists with several other mechanisms of antiemetic action, reduce the incidence of vomiting following strabismus surgery in children.³⁰⁻³¹

We conclude that, in fasting ASA physical status I children hydrated with IV lactated Ringer's solution, increased blood glucose levels are indicators of the stress response due to tracheal intubation and surgical stress.³²⁻³³ Serum gastrin levels are not changed during general anaesthesia with halothane and nitrous oxide and are not increased by reflexogenic stimuli applied to the extrinsic muscles of the eye. Vomiting after strabismus surgery cannot be explained by high serum levels of gastrin causing or favouring a centrally mediated inhibition of gastric emptying. Also, it is unlikely that vomiting after strabismus surgery is linked to an "oculogastric reflex" with the vagus nerve as the efferent pathway. More controlled studies are needed to elucidate the aetiology of vomiting following strabismus surgery.

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