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Vomiting and Gastroesophageal Motor Activity in Children with Disorders of the Central Nervous System

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ABSTRACT

Background: Vomiting is common in children with disorders of the central nervous system(CNS) and is usually ascribed to gastroesophageal reflux (GER). However, recent acquisitions on the pathophysiology of vomiting suggest that the dysmotility of the foregut may be more widespread.

Methods: Fifty-five children with CNS disorders, 50 of whom suffered from retching and/or vomiting (18 following fundoplication) were studied. We assessed GER by 24 hour pH monitoring and endoscopy, gastric electrical activity by electrogastrography, and gastric half-emptying time (T½) of a milk meal be electrical impedance tomography.

Results: Of the 50 vomiting patients, 29 had GER (reflux index of 5.7%-87.4%; controls: <5%), and 31 had gastric dysrhythmias (12 tachyarrhythmia at 5.5-11.2 cpm, 4 bradyarrhythmia at 1.7-1.9 cpm, 15 unstable electrical activity; controls: 2.2-4.0 cpm). Sixteen patients had GER and gastric dysrhythmias. Eleven of 18 patients with fundoplication had gastric dysrhythmias. Gastric T½ was delayed in 12 of 13 patients with gastric dysrhythmia (6 with GER), versus 2 of 5 with GER alone. No abnormalities were detected in the 5 patients who did not suffer from vomiting.

Conclusions: Children with CNS disorders who vomit have abnormal gastric motility as often as GER. Following fundoplication, many patients continue to have symptoms possibly related to gastric dysrhythmias, the effects of which may be unmasked by fundoplication. Foregut dysmotility may be related to abnormal modulation of the enteric nervous system by the CNS or to involvement of the enteric nervous system by the same process affecting the brain.

Recurrent vomiting with aspiration and failure to thrive occurs in 10% to 15% of institutionalized children with psychomotor retardation related to disorders of the central nervous system (CNS) (1). It is commonly ascribed to gastroesophageal reflux, which is found, with or without hiatus hernia, in approximately 75% of such patients (2-5). The response to conventional medical treatment is usually poor (6), and antireflux surgery, although usually very successful in controlling gastroesophageal reflux, has a higher than average rate of morbidity (7). Therefore, vomiting and its consequences are a considerable burden for the patients and their families and a challenge to those involved in their management.

Vomiting may be effortless and result from gastroesophageal reflux, or it may be caused by a reflex mechanism associated with nausea and other autonomic manifestations (8). Knowing that vomiting may continue after antireflux surgery and that some patients with cerebral palsy and developmental delay retch and experience nausea, we investigated gastroesophageal reflux and gastric antral electrical control activity as a means of detecting activation of the emetic reflex as well as a more wide-spread foregut dysmotility in vomiting patients with psychomotor retardation.

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MATERIALS AND METHODS

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Patients and Controls

Fifty-five children-27 boys and 28 girls of mean age 4.5 years (range, 4 months-15.5 years)-were studied. Forty (6 with spastic quadriplegia) had moderate to severe global neurodevelopmental delay, and 15 had severe spastic cerebral palsy (12 quadriplegia and 3 diplegia), from a variety of primary causes (birth asphyxia 22, chromosomal defects 14, metabolic disease 2, kernicterus 1, herpes virus embryopathy 1, idiopathic 15). Twenty-eight (51%) had evidence of gross anatomic abnormalities of the CNS (17 brain atrophy, 9 ventricular dilation, 2 disordered neuronal migration) seen on computed tomographic or nuclear magnetic resonance scan. Five patients (2 with cerebral palsy) were not vomiting and had not undergone previous major surgery. Fifty patients had recurrent retching or vomiting severe enough to require repeated hospital admissions. Most of them were also troubled by a number of clinical problems other than vomiting (especially recurrent respiratory symptoms and failure to thrive), which complicated their care; these are summarized in Table 1. Thirty-seven (74%) of these 50 patients required tube feeding. Thirty-six were fed by nasogastric tube and 11 by gastrostomy, which had been carried out at the time of fundoplication. Eighteen still had symptoms (mainly recurrent retching and severe postprandial discomfort, with occasional vomiting) after a Nissen fundoplication had been performed(together with a feeding gastrostomy in 11 patients).

Graphic Table 1

Overall, 59 subjects aged 3 months to 15 years acted as controls. Forty-seven had been referred for a suspected gastrointestinal motility disorder that was excluded after appropriate investigation (including 24-hour intra-oesophageal pH monitoring in 26 and electrogastrography [EGG] in 21), and a variety of diagnoses were finally made (23 eating disorders, 8 recurrent respiratory infections, 5 cyclical vomiting, 4 nonulcer dyspepsia, 3 psychogenic vomiting, 2 postenteritis enteropathy, 2 Crohn's disease). Twelve healthy asymptomatic children were used as controls for the gastric emptying studies.

Permission to carry out the study was granted by the Ethical Committee of the Hospital for Sick Children, London.

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Methods

Any antireflux treatment (milk-thickening agents, antacids, H2-blockers, prokinetics) was stopped at least 48 hours before the investigation. A barium contrast study and follow-through examination was performed in all patients to detect anatomic abnormalities of the foregut.

We used intra-oesophageal pH monitoring and upper gastrointestinal endoscopy to determine the presence and severity of gastroesophageal reflux, EGG to record gastric antral electrical control activity and thus detect antral dysrhythmias, and electrical impedance tomography to measure the gastric emptying time. A well-validated ambulatory pH monitoring system(Synectics Medical, Enfield, UK) was used. Under fluoroscopic control, an antimony pH electrode was placed at the level of the T9 to T10 vertebral bodies (or above, if a hernia sac had been demonstrated at this level by the barium study), and a silver-silver chloride reference electrode was attached to the skin. pH values were recorded every 6 seconds, and every shift in intra-oesophageal pH that caused the pH level to fall below 4 lasting more than 20 seconds was defined as an acid reflux episode. Data were collected and stored on a portable Digitrapper for 24 hours for off-line analysis on a personal computer, using dedicated software (EsopHogram, Gastrosoft Synectics Medical, Enfield, UK). Pathologic acid gastroesophageal reflux was present whenever the percentage of time spent with intra-oesophageal pH <4 (reflux index) was more than 5%. Reflux was graded as mild (reflux index 5-10%), moderate (reflux index 10-20%) and severe (reflux index >20%). Other parameters measured were: number of reflux episodes, number of reflux

episodes lasting longer than 5 minutes, and duration of the longest reflux episode. Upper gastrointestinal endoscopy was carried out with a 5.3-mm diameter paediatric fiber endoscope (Olympus GIF N30), with the patient under conscious sedation. Macroscopic and histologic oesophagitis was graded according to well-validated and widely accepted criteria (9).

Gastric antral electrical control activity was measured non-invasively, using the technique of surface EGG for 1 hour after an overnight fast. Milk(20 ml/kg) was given halfway through to induce postprandial activity. After detorsion with 70% ethyl alcohol and gentle abrasion of the skin to reduce impedance (2.5 ± 2.0 k[OMEGA]), four bipolar silver-silver chloride electrodes (Medicotest, Ölstykke, Denmark) were attached to the epi- and mesogastric region. The electrical signal was amplified and passed through an analog low-pass filter (Gould Electronics, UK; time constant 3.2 seconds, cut-off slope 6 dB/octave), before it was displayed on a Gould eight-channel polygraph and simultaneously digitized at 1 Hz by an analog-to-digital converter (Data Translation 2801A) and stored on the hard disk of an IBM-compatible desktop computer for later off-line analysis. The digital signal was subjected to running spectral analysis, using a sequence of computerized algorithms (PC-DATS, Prosig Computer Consultants, Fareham, UK) adapted by the authors. Each 1-hour recording was subdivided into 53 segments of 128 seconds with a 75% overlap, which were band-pass filtered (Butterworth digital filter, 0.008-0.25 Hz; slope, 24 dB/octave) and the frequency spectra were determined by autoregressive modelling (10). The peak frequency of each segment was extracted, and the dominant frequency (that present in the majority of segments) was calculated in cycles per minute (cpm) from the channel with the best signal-to-noise ratio. The resulting analysis was displayed as a pseudo three-dimensional plot. Patients were considered to have an antral dysrhythmia whenever their electrical control activity had a dominant frequency slower or faster than 2 standard deviations (SD) removed from the mean dominant frequency of the control subjects, and in such cases the respective terms “bradyarrhythmia” and“tachyarrhythmia” were used. When the abnormally slower or faster dominant frequency was stable (i.e., present for >75% of the recording) the terms “bradygastria” and “tachygastria” were also used. When the electrical activity was so variable that no dominant frequency could be detected, we used the term “unstable electrical control activity,” if the frequencies remained within the control range, or“mixed dysrhythmia,” if the frequencies included slower and faster peaks.

Gastric emptying of a standard milk meal was measured noninvasively by electrical impedance tomography (11). An alternating current of 5 mA at 50 kHz “peak to peak” was passed between each pair of 16 electrodes placed in a circumferential array around the abdomen, and the potential differences among the remaining electrodes were measured during each cycle, which lasted 80 msec. One hundred fifty cycles were added together to form a data set or “frame.” A reference frame was collected just before the milk was given, and the frames collected during and after the meal were back-

projected against it to produce sequential, cross-sectional images related to changes in calculated intragastric resistivity. Time to half-emptying (T½) was calculated as the time required to achieve a 50% decrease of the maximum conductivity recorded in the stomach area at the end of the meal. Gastric emptying was considered to be delayed whenever T½ was more than two SDs removed from the mean control value for gastric emptying.

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RESULTS

Barium contrast study and follow-through examination revealed a hiatus hernia in 4 patients, malrotation in 2 patients, and free gastroesophageal reflux in 17 patients (2 after fundoplication). All of these patients had symptoms of retching, vomiting, or both.

A 24-hour intra-oesophageal pH study, the results of which are illustrated in detail in Table 2, showed a mean ± SD reflux index of 2.2 ± 1.2% (range, 0.4-4.2%) in 26 control subjects and a similar mean reflux index of 2.1 ± 1.5% (range, 0.4-3.7%) in the 5 patients who did not vomit. Twenty-nine of 50 (58%) vomiting patients had significant gastroesophageal reflux (reflux index, 5.7-87.4%; mean, 17.9%). According to the percentage of time spent with an intra-oesophageal pH <4, gastroesophageal reflux was mild in 9 patients, moderate in 10, and severe in 10 (Fig. 1). Eleven of 22 patients with gastroesophageal reflux had endoscopic and histologic evidence of oesophagitis. Oesophagitis was mild (grade 1) in 4, moderate (grade 2) in 2, and severe (grade 3) in 2 patients; and 2 patients had Barrett's oesophagus with intestinal metaplasia of the oesophageal mucosa.

Graphic Table 2 Graphic Fig. 1

The distribution of the frequencies of gastric electrical control activity measured by the EGG in control subjects and in patients is illustrated in Figure 2. In 21 control subjects gastric electrical control activity had a mean ± SD dominant frequency of 3.1 ± 0.5 cpm (range 2.2-4 cpm) and showed a consistent increase in amplitude after the ingestion of food. The 5 patients without vomiting had a similar mean ± SD dominant frequency of 2.9 ± 0.4 cpm (range, 2.4-3.3 cpm). Thirty-one of 50 (62%) patients with vomiting had antral dysrhythmia. Four had bradyarrhythmia (2 bradygastria; dominant frequency 1.7-

1.9 cpm, mean 1.8 cpm), 12 had tachyarrhythmia (4 tachygastria; dominant frequency 5.5-11.2 cpm, mean 8.5 cpm), 12 had unstable electrical activity and 3 had mixed dysrhythmia in which no dominant frequency could be detected (Figs. 3A to 3E). In some cases, the dysrhythmic pattern changed slightly after the ingestion of milk, but food ingestion was not associated with induction or suppression of dysrhythmias. In two patients (twin brother and sister), low-amplitude, slightly unstable gastric electrical control activity became rapidly normal, with a regular, high-amplitude 3 cpm signal during sleep (Fig. 3F).

Graphic Fig. 2 Graphic Fig. 3

In the 12 normal control subjects, gastric T½ of the milk meal was 37 to 88 minutes (mean 60 ± 13.7 minutes). The gastric emptying study was carried out on 23 patients (Fig. 4). Fourteen of 18 patients with vomiting had delayed gastric emptying, with a T½ of 95 to 250 minutes (mean 158 ± 63.1 minutes). Gastric T½ was delayed in 12 of 13 patients with gastric dysrhythmia (6 of whom also had gastroesophageal reflux), and in 2 of 5 with gastroesophageal reflux alone. Five of 7 patients with delayed gastric emptying (4 of whom had gastric dysrhythmia) also had oesophagitis: 3 mild, 1 moderate, and 1 Barrett's oesophagus.

Graphic Fig. 4

Among the 50 patients with vomiting, 13 (26%) had gastroesophageal reflux only, 15 (30%) had antral dysrhythmia only, and 16 (32%) had both gastroesophageal reflux and antral dysrhythmia. The severity of reflux did not significantly differ between patients with gastroesophageal reflux only (mean reflux index 16.6%, range 6.5-47.5%; 6 of 10 oesophagitis) and patients with gastroesophageal reflux and gastric dysrhythmia (mean reflux index 19.1%, range 5.7-87.4%; 5 of 9 oesophagitis). Patients with gastroesophageal reflux were older (mean age, 6.42 years) than patients with gastric dysrhythmia (mean age, 4.22 years) and patients with reflux and dysrhythmia (mean age, 2.92 years). Percentages of disorders in tube-fed patients were 61.5% with gastroesophageal reflux, 66.7% with gastric dysrhythmia, and 81.2% with gastroesophageal reflux and gastric dysrhythmia.

Fifteen of 18 (83%) patients who had undergone a Nissen fundoplication had abnormal findings: 4 had gastroesophageal reflux (1 with malrotation), 9 had antral dysrhythmia (4 of 5 with delayed gastric emptying), and 2 had gastroesophageal reflux and antral dysrhythmia (1 with malrotation). The

prevalence of gastric dysrhythmia and delayed gastric emptying did not significantly differ between patients with and without gastrostomy, and the patients with gastrostomy did not exhibit any specific EGG pattern. One patient with tachyarrhythmia had known long-segment Hirschprung's disease and had previously undergone subtotal colectomy.

Given the very high prevalence (88%) of foregut dysmotility among patients with vomiting, we looked at the prevalence of the clinical problems other than vomiting listed in Table 1 to see whether a relationship could be established with gastroesophageal reflux and disordered gastric electrical activity. Failure to thrive was significantly more common in patients with gastroesophageal reflux alone than in patients with gastroesophageal reflux and gastric dysrhythmia. Although the difference was not significant, spasticity, seizures, and scoliosis were found more frequently in patients with gastroesophageal reflux alone than in patients with gastric dysrhythmias (with or without gastroesophageal reflux) and in patients without foregut motor abnormalities, whereas constipation was found more frequently in patients with gastric dysrhythmias than in patients with gastroesophageal reflux (Table 3).

Graphic Table 3

In 6 of 50 patients with vomiting (3 with fundoplication), and in all 5 patients without vomiting, no abnormality was found.

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DISCUSSION

Recurrent vomiting is a major problem in children with disorders of the CNS. The results of this study show that several mechanisms are involved and that gastric antral dysrhythmias are at least as common as acid gastroesophageal reflux.

Recent advances in the neurophysiology of vomiting have shown that retching and vomiting can occur as a result of a neural reflex-the emetic reflex in which the sensory and motor pathways of the vagus nerve, the area postrema, and the nuclei of the vagus play a major role (8). Afferent inputs of varying origins (peripheral and central) are integrated and processed within the hind-brain to elicit somatic and autonomic outputs involving several target organs, including the foregut. Two major events occur within the foregut: relaxation of the proximal stomach and retroperistalsis. They are caused by the inhibition of the gastric "pacemaker" and the consequent production of antral and duodenal electrical dysrhythmias, and they result in impaired gastroduodenal motility and delayed gastric emptying (8). Experimental evidence suggests that if the antral electrical control activity is slower or faster than normal by more than 30% to 40%, loss of coupling between electrical and mechanical activity takes place, and gastric retention occurs (11-13). Delayed gastric emptying and gastric (especially fundic) distension-either related to a primary disorder of gastric motility or secondary to activation of the emetic reflex-can themselves induce transient relaxation of the lower oesophageal sphincter, which is the single most important pathogenetic factor in gastroesophageal reflux. In this study, however, oesophageal manometry was not carried out, and thus the relationship between gastric dysmotility, lower oesophageal sphincter relaxation, and gastroesophageal reflux could not be established. In findings in a few paediatric studies, the rate of gastric emptying has also been shown to correlate with the severity of gastroesophageal reflux (14) and the presence of oesophagitis (15). In our results, oesophagitis was more common in patients with delayed gastric emptying (5 of 7) than in patients with normal gastric emptying (1 of 3), but we did not find a clear correlation between the grade of oesophagitis and the delay of gastric emptying, perhaps because of the small number of patients (10 of them) who underwent an endoscopic examination and a gastric emptying study. However, that most patients with delayed gastric emptying and oesophagitis also had gastric dysrhythmia suggests that those patients who have more severe reflux may have a more severe underlying motor dysfunction that is not restricted to the oesophagus and that extends throughout the foregut. That a number of children with severe neurodevelopmental handicap with recurrent vomiting are also chronically and severely constipated raises the possibility that some of them may have an even more widespread panenteric dysfunction. The pattern of distribution of motor dysfunction within the gut in children with disorders of the central nervous system probably reflects the density of extrinsic innervation of the gut. There are more foregut symptoms than midgut symptoms, because there is a greater density of vagal afferent and efferent neurones to the foregut than to the midgut. There is a greater incidence of hindgut dysfunction with constipation than midgut problems, but there are fewer foregut problems; again, this is related to the density of extrinsic innervation.

The control of foregut motor function involves muscular, neural (intrinsic and extrinsic) and humoral factors (16). Gastric antral dysrhythmias have been reported in a number of different conditions characterized by nausea and vomiting in which these different control factors are affected (12,13,17-21). In patients with CNS disorders, at the least, the extrinsic innervation is abnormal, with gastric antral dysrhythmias resulting from abnormal activation of the efferent limb of the emetic reflex or from lack of

inhibition of excitatory fibres as a consequence of either disturbed input to the hindbrain from higher centres or anatomic and functional disturbances of the“vomiting centre” and the area postrema (8). Antral dysrhythmia could occur after anoxic-ischaemic insults to the brain, when intraventricular hemorrhage, ventricular dilation, and periventricular atrophy may all interfere with the central regulation of the emetic reflex (8) and the lower oesophageal sphincter (22). However, antral dysrhythmias could also be caused by disordered intrinsic nerves of the enteric nervous system. We have recently described the occurrence of gastric antral tachyarrhythmias in children with intestinal pseudo-obstruction related to alterations of the enteric nervous system (20). In the current study, one patient with idiopathic cerebral palsy and tachyarrhythmia also had Hirschprung's disease. Recently, Hirschprung's disease has been shown to be commonly associated with antroduodenal dysmotility, which has features compatible with a neuropathic disorder (23). It is therefore possible that in some cases a similar neuropathic process might involve the enteric nervous system as well as the CNS. Our patients had various types of gastric antral dysrhythmias, characterized by slower, faster, or disorganized electrical activity. This variability may be the consequence of the variable etiology, pathology, severity, and site of neural lesions at the level of the CNS or the enteric nervous system.

Apart from vomiting, many patients with cerebral palsy and severe neurodevelopmental delay suffer from a number of different problems that may affect the motor function of their foregut. Scoliosis, convulsions, spastic quadriplegia, and prolonged supine positioning are all more prevalent in patients with cerebral palsy and neurodevelopmental delay who have gastroesophageal reflux, with or without hiatus hernia, and may contribute to inducement or aggravation of reflux (2-4,24,25). Our results confirm these findings by showing that scoliosis, seizures, and spasticity are more common in patients with gastroesophageal reflux. The higher prevalence of failure to thrive among our patients with gastroesophageal reflux compared with that in patients with gastroesophageal reflux and gastric dysrhythmia, however, has several explanations. The mean age of patients with gastroesophageal reflux was higher, and they thus had had their disease longer. Patients with gastroesophageal reflux were also more seriously ill, as shown by the higher prevalence of intractable seizures, scoliosis, and spasticity. Furthermore, 81% of patients with gastroesophageal reflux and gastric dysrhythmia were tube fed, compared with 61% of patients with gastroesophageal reflux only, and were therefore more likely to have a higher caloric intake. Some of the clinical problems considered previously could also be pathogenetically related to gastric dysrhythmias. Paroxysmal antral dysrhythmias could be viewed as the gastric equivalent of neuronal discharge from the brain, which causes seizures. Intestinal obstruction in cases of severe, chronic constipation might also affect the motor activity of the stomach and the proximal small bowel; indeed, in our study, constipation occurred more frequently in patients with gastric dysrhythmias. However, our patient population was very heterogeneous; controlled studies on selected patient groups are needed before conclusions can be drawn on the interactions between gastric dysrhythmias, epilepsy, and constipation.

Gastric antral dysrhythmias were found in 11 of 18 patients who had undergone Nissen fundoplication. We were not able to study these patients before and after surgery, and we therefore cannot exclude that the dysrhythmias were secondary to the fundoplication. Vagal damage is not uncommon after fundoplication, and dysrhythmias may occur after vagotomy, although the relationship with subsequent disorders of gastric motility and gastric emptying is unclear (26,27). Alterations of gastric electrical control activity reported after surgery to the foregut and proximal vagotomy in humans and transthoracic vagotomy in dogs are usually mild and are limited to the postoperative period (28-31). Furthermore, in the long term, considerable adaptation of gastric motility appears to take place, and the effects of the vagotomy disappear (32). Eleven patients had also undergone a surgical gastrostomy at the time of fundoplication, but only 5 of them had gastric dysrhythmia; it is thus unlikely that gastrostomy per se may cause dysrhythmia. Surgical gastrostomy, nevertheless, is a well-known pathogenetic factor for secondary gastroesophageal reflux, (33); but this was not the case in our patients, in whom the procedure was carried out at the same time of the antireflux surgery. Finally, we found gastric dysrhythmias associated with gastroesophageal reflux in 14 patients who had not undergone a fundoplication or other type of abdominal surgery. Thus, it is very likely that the gastric dysrhythmias were present before surgery and that the poor response to medical and surgical treatment of many cases of gastroesophageal reflux in children with CNS disorders is related to a more widespread abnormality of foregut motility than has hitherto been suspected. Fundoplication, while controlling the gastroesophageal reflux, may unmask other underlying mechanisms of vomiting-that is, gastric dysmotility and activation of the emetic reflex. In support of this view, patients with disordered gastric emptying are more at risk of the development of gas bloat and persistent retching after fundoplication (34). Such symptoms occur almost exclusively in patients who still have disordered gastric emptying after surgery (35,36). Assessment of gastric emptying has therefore been recommended before antireflux surgery is carried out, to identify those patients who are more likely to develop symptoms after fundoplication (34,35,37); and it has been suggested that a pyloroplasty be done together with fundoplication when gastric emptying is significantly delayed, especially in children with neurologic impairment (37,38).

We have no clear explanation for vomiting and feeding problems in the six patients who had no evidence of gastroesophageal reflux or gastric antral dysrhythmias. The normalization of the EGG pattern during sleep and the hypersensitivity of the mouth found in two of them suggest that behavioral components may play a major role. Oropharyngeal incoordination with impaired swallowing documented in another patient may result in abnormal initiation of oesophageal peristalsis and thereby facilitate regurgitation. Finally, some of these patients were reported to vomit essentially during meals, and it is possible that they had neutral or slightly alkaline gastroesophageal reflux, which pH study would not detect.

In summary, the results of our study show that in children with CNS disease, vomiting is related to gastric dysrhythmias and delayed gastric emptying, possibly due to activation of the emetic reflex, at least as often as to gastroesophageal reflux. A significant proportion of these patients may thus have widespread foregut dysmotility in which gastroesophageal reflux(mainly caused by dysfunction of the lower oesophageal sphincter), gastric antral dysrhythmias (related to dysfunction of the gastric pacemaker), and delayed gastric emptying are associated. Alternatively, because pH monitoring does not distinguish between reflux and reflex vomiting, in a percentage of patients acid gastroesophageal reflux detected by pH study may be simply an epiphenomenon of the underlying gastroduodenal dysmotility (retroperistalsis) related to gastric electrical dysrhythmias and gastroparesis. In patients with postoperative symptoms of retching and vomiting, such dysrhythmias and the resulting disordered gastric emptying may be unmasked by fundoplication.

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REFERENCES

1. Ball TS, Hendrickson H, Clayton J. A special feeding technique for chronic regurgitation. Am J Ment Defic 1974;78:486-93. SFX [Context Link]

2. Sondheimer JM, Morris BA. Gastroesophageal reflux among severely retarded children. J Pediatr 1979;94:710-4. SFX [Context Link]

3. Abrahams P, Burkitt BFE. Hiatus hernia and gastro-oesophageal reflux in children and adolescents with cerebral palsy.Aust Paediatr J 1970;6:41-6. [Context Link]

4. Byrne WJ, Campbell M, Ashcraft E, Seibert JJ, Euler AR. A diagnostic approach to vomiting in severely retarded patients. Am J Dis Child 1983;137:259-62. SFX [Context Link]

5. Staiano A, Cucchiara S, Del Giudice E, Andreotti MR, Minella R. Disorders of oesophageal motility in children with psychomotor retardation and gastro-oesophageal reflux. Eur J Pediatr 1991;150:638-41. SFX [Context Link]

6. Wilkinson JD, Dudgeon DL, Sondheimer JM. A comparison of medical and surgical treatment of gastroesophageal reflux in severely retarded children. J Pediatr 1981;99:202-5. SFX [Context Link]

7. Byrne WJ, Euler AR, Ashcraft E, Nash DG, Seibert JJ, Golladay ES. Gastroesophageal reflux in the severely retarded who vomit: Criteria for and results of surgical intervention in twenty-two patients.Surgery 1982;91:95-9. SFX [Context Link]

8. Andrews PL, Hawthorn J. The neurophysiology of vomiting.Baillieres Clin Gastroenterol 1988;2:141-68. SFX [Context Link

9. Vandenplas Y (Coordinator). Reflux esophagitis in infants and children: A report from the working group on gastro-oesophageal reflux disease of the European Society for Paediatric Gastroenterology and Nutrition.J Pediatr Gastroenterol Nutr 1994;18:413-22. [Context Link

10. Linkens DA, Datardina SP. Estimation of frequencies of gastrointestinal electrical rhythms using autoregressive modelling. Med Biol Eng Comput 1978;16:262-8. SFX [Context Link]

11. Sarna SK, Bowes KL, Daniel EE. Gastric pacemakers.Gastroenterology 1976;70:226-31. SFX [Context Link]

12. Telander RL, Morgan KG, Kreulen DL, Schmalz PF, Kelly KA, Szurszewski JH. Human gastric atony with tachygastria and gastric retention. Gastroenterology 1978;75:497-501. SFX [Context Link]

13. You CH, Lee KY, Chey WY, Menguy R. Electrogastrographic study of patients with unexplained nausea, bloating and vomiting.Gastroenterology 1980;79:311-4. SFX [Context Link]

14. Hillemeier AC, Grill BB, McCallum R, Gryboski J. Esophageal and gastric motor abnormalities in gastroesophageal reflux during infancy. Gastroenterology 1983;84:741-6. SFX [Context Link]

15. Di Lorenzo C, Piepsz A, Ham H, Cadranel S. Gastric emptying with gastro-oesophageal reflux. Arch Dis Child 1987;62:449-53. SFX [Context Link]

16. Johnson LR, ed. Physiology of the gastrointestinal tract. New York: Raven Press, 1987. [Context Link]

17. Abell TL, Camilleri M, Malagelada J-R. High prevalence of gastric electrical dysrhythmias in diabetic gastroparesis.Gastroenterology 1985;88:1299. Abstract. [Context Link]

18. Stern RM, Koch KL, Leibowitz HW, Lindblad IM, Schupert CL, Stewart WR. Tachygastria and motion sickness. Aviat Space Environ Med 1985;56:1074-7. SFX [Context Link]

19. Koch KL, Stein RM, Vasey M, Botti JJ, Creasy GW, Dwyer A. Gastric dysrhythmias and nausea of pregnancy. Dig Dis Sci 1990;35:961-8. SFX [Context Link]

20. Devane SP, Ravelli AM, Bisset WM, Smith VV, Lake BD, Milla PJ. Gastric antral dysrhythmias in children with chronic idiopathic intestinal pseudo-obstruction. Gut 1992;33:1477-81. [Context Link]

21. Ravelli AM, Ledermann SE, Bisset WM, Trompeter RS, Barratt TM, Milla PJ. Foregut motor function in chronic renal failure.Arch Dis Child 1992;67:1343-7. SFX [Context Link]

22. Vane DW, Shiffler M, Grosfeld JL, Angelides A, Weber TR, Fitzgerald JF. Reduced lower esophageal sphincter (LES) pressure after acute and chronic brain injury. J Pediatr Surg 1982;17:960-3. SFX [Context Link]

23. Faure C, Ategbo S, Ferreira GC, et al. Duodenal and esophageal manometry in total colonic aganglionosis. J Pediatr Gastroenterol Nutr 1994;18:193-9. Ovid Full Text [Context Link]

24. Cadman D, Richards J, Feldman W. Gastro-esophageal reflux in severely retarded children. Dev Med Child Neurol 1978;20:95-8. SFX [Context Link]

25. Kassem NY, Groen JJ, Fraenkel M. Spinal deformities and oesophageal hiatus hernia. Lancet 1965;1:887-9. SFX [Context Link]

26. Minami H, McCallum RW. The physiology and pathophysiology of gastric emptying in humans. Gastroenterology 1984;86:1592-610. SFX [Context Link]

27. Smout AJPM, Akkermans LMA. Postsurgical gastric emptying disorders. In: Motility 1990;11:15-9. [Context Link]

28. Sarna SK, Bowes KL, Daniel EE. Post-operative gastric electrical control activity (ECA) in man. In: Daniel EE, ed.Gastrointestinal motility. Vancouver: Mitchell Press, 1974:73-83. [Context Link]

29. Hinder RA, Kelly KA. Human gastric pacesetter potential. Site of origin, spread, and response to gastric transection and proximal gastric vagotomy. Am J Surg 1977;133:29-33. [Context Link]

30. Geldof H, Van Der Schee EJ, Van Blankenstein M, Smout AJ, Akkermans LM. Effects of highly selective vagotomy on gastric myoelectrical activity. An electrogastrographic study. Dig Dis Sci 1990;35:969-75. SFX [Context Link]

31. Kelly KA, Code CF. Effect of transthoracic vagotomy on canine gastric electrical activity. Gastroenterology 1969;57:51-8. SFX [Context Link]

32. Andrews PL, Bingham S. Adaptation of the mechanisms controlling gastric motility following chronic vagotomy in the ferret.Exp Physiol 1990;75:811-25. SFX [Context Link]

33. Mollitt DL, Golladay ES, Seibert JJ. Symptomatic gastroesophageal reflux following gastrostomy in neurologically impaired patients. Pediatrics 1985;75:1124-6. SFX [Context Link]

34. Papaila JG, Wilmot D, Grosfeld JL, Rescorla FJ, West KW, Vane DW. Increased incidence of delayed gastric emptying in children with gastroesophageal reflux. A prospective evaluation. Arch Surg 1989;124:933-6. SFX [Context Link]

35. Jolley SG, Tunell WP, Leonard JC, Hoelzer DJ, Smith EI. Gastric emptying in children with gastroesophageal reflux. II. The relationship to retching symptoms following antireflux surgery. J Pediatr Surg 1987;22:927-30. SFX [Context Link]

36. Hinder RA, Stein HJ, Bremner CG, DeMeester TR. Relationship of a satisfactory outcome to normalization of delayed gastric emptying after Nissen fundoplication. Ann Surg 1989;210:458-64. Ovid Full Text [Context Link]

37. Fonkalsrud EW, Berquist W, Vargas J, Ament ME, Foglia RP. Surgical treatment of the gastroesophageal reflux syndrome in infants and children. Am J Surg 1987;154:11-8. SFX [Context Link]

38. Fonkalsrud EW, Ellis DG, Shaw A, Mann CM Jr, Black TL, Miller JP, Snyder CL. A combined hospital experience with fundoplication and gastric emptying procedure for gastroesophageal reflux in children. J Am Coll Surg 1995;180:449-55. SFX [Context Link]

Key Words: Cerebral palsy; Electrogastrography; Gastric dysrhythmias; Gastroesophageal reflux; Psychomotor retardation; Vomiting

IMAGE GALLERY

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Table 1 Table 1 Table 2 Table 2 Fig. 1 Fig. 1

Fig. 2 Fig. 2 Fig. 3 Fig. 3 Fig. 4 Fig. 4

Table 3 Table 3

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