Electrogastrography in non-ulcer dyspepsia

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Abstract

Fasting and fed gastric electrical activity was recorded by cutaneous electrodes (electrogastrography) in 14 children with unexplained recurrent symptoms of upper intestinal dysfunction, and in 10 controls. The unexplained symptoms included vomiting, epigastric pain, fullness, and early satiety. Mean (SD) age was 7.0 (3) and 7.5 (2) years, respectively. Gastric emptying time of a solid-liquid meal was also measured by real time ultrasonography in all subjects (patients and controls). In all patients radiography and endoscopy excluded structural and focal abnormalities of the gastrointestinal tract. Gastric emptying time was significantly more prolonged in patients than in controls. It was also found that there were appreciable irregularities of gastric electrical rhythm (tachygastria, bradygastria, flat line pattern, and mixed arrhythmia) in 12 fasting and 10 fed patients, whereas controls showed short and rare episodes of arrhythmia during both fasting and fed recording periods. The percentage distribution of the total electrogastrographic energy power across three frequency bands of electrical activity (low, normal, and high) showed that patients were different from controls both for reduced activity of normal frequency and for increased incidence of high and low abnormal frequencies. It is concluded that gastric electrical abnormalities are found in a high proportion of children with recurrent unexplained upper gastrointestinal symptoms. Electrogastrography can be a valuable tool in the assessment of these patients.

Chronic or recurrent symptoms suggesting functional upper gastrointestinal disorders, such as postprandial vomiting, regurgitation, belching, bloating, fullness, epigastric pain, early satiety, and pyrosis, are a frequent cause for referring children to paediatricians. This constellation of symptoms, in the absence of structural abnormalities and focal mucosal lesions of the gut, is known as functional or non-ulcer dyspepsia.¹⁻³

Recently, both evidence of abnormal gastrointestinal motor patterns and the beneficial effects of prokinetic drugs on some patients with non-ulcer dyspepsia suggest that the syndrome might be due to disordered gastroduodenal motility.⁴⁻⁶ It is also worth noting that a number of reports have described abnormalities in gastric electrical activity in patients with unexplained dyspeptic symptoms and delayed gastric emptying.^{7,8} This is not surpris-

ing as gastrointestinal phasic mechanical contractions are preceded by changes in the electrical activity of smooth muscle cells.

The aim of this study was to measure both fasting and fed gastric myoelectrical activity and gastric emptying time in children with unexplained dyspeptic symptoms. Gastric myoelectrical activity was recorded with electrogastrography, a non-invasive method, by means of cutaneous electrodes attached to the epigastric skin.

Subjects and methods

SUBJECTS

A total of 24 subjects participated in the study: 14 children had symptoms of non-ulcer dyspepsia and 10 were clinically asymptomatic. Mean (SD) age was 7.0 (3) and 7.5 (2) years, respectively. Postprandial vomiting (n=12) was the most common symptom and it occurred both within (early) and after (late) 30 minutes from feeding and was often preceded by epigastric pain (n=8) or distension (n=6). Other presenting complaints were regurgitation (n=5), pain located in the upper or central region of the abdomen unrelated to vomiting (n=10), fullness (n=6), anorexia or early satiety (n=9), and belching (n=6). In five children a weight:height ratio below the 5th centile was detected.

In all patients upper gastrointestinal radiography excluded structural abnormalities of the gut, and furthermore endoscopy did not reveal focal mucosal lesions (that is, oesophagitis, peptic ulcer disease). Infectious, metabolic, and neurological diseases were also excluded. In five children 24 hour intraoesophageal pH monitoring revealed an abnormal oesophageal acid exposure; normal degrees of this variable have been previously reported.⁹

GASTRIC EMPTYING TIME MEASUREMENT

All subjects (patients and controls) underwent measurement of gastric emptying time by real time ultrasonography performed with a high resolution real time scanner having a 3.5 MHz linear array transducer, applied with minimal abdominal compression. Ultrasonographic determinations were carried out by an operator who was unaware of both clinical condition and diagnosis of the subjects. After an overnight fast children were examined sitting at 45° to the horizontal plane of the examination couch in the fasting state and after eating a 1.25 MJ (300 kcal) solid-liquid meal (bread, butter, ham, fruit juice). The emptying time was determined by measuring every 15 minutes changes in the

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cross sectional area of the gastric antrum, calculated by measuring the longitudinal and antero-posterior diameters at the level of a sagittal plane passing through the superior mesenteric vein.¹⁰ Final gastric emptying time was considered when the section area had the same value as during the fasting state and no food particles were detected in the antral region; furthermore, it was related to the start of the meal.

ELECTROGASTROGRAPHY

Electrogastrography was recorded by three pairs of cutaneous electrodes (Red Dot 225 3M Co) positioned on the epigastric skin along the axis of the distal stomach that was previously localised by ultrasound. The electrodes were connected to a rectilinear polygraph (R 611, SensorMedics) through nystagmus couplers (9859, SensorMedics). The low and high frequency cut offs were set at 0.016 Hz (time constant 10 seconds) and 0.30 Hz, respectively. Because of inter-individual anatomical variations, various recording leads were used and the lead with the best signal to noise ratio was selected for analysis. The electrogastrography signal was simultaneously recorded on the polygraph and on a FM tape recorder. This was later digitised

(real time sampling frequency 1 Hz) and fed into a computer for spectral analysis, that is, a method by which the wave form data is decomposed into various frequency components and the relative contribution of a given frequency band to the entire signal is called the 'power' of that frequency band. For example, fig 1 shows a frequency analysis of a signal in a fasting control subject. This analysis has the greatest contribution of its frequencies in the range of 2.5–3.5 cycles per minute (cpm) (that is the normal frequency of gastric electrical signal in humans) and thus has highest 'power' at that frequency range. The percentage power distribution was defined as the ratio between power within one of the three major frequency ranges (1–2, 2.5–3.5, and 4–9 cpm) and total power in all three bands. Spectral analyses were represented by pseudo three dimensional plots, where electrical frequency (0–14 cpm) were plotted on the horizontal axis and time (in 64 second intervals) on the vertical axis. Spectra were obtained as follows: every 64 seconds a power spectrum was computed from the preceding 256 seconds of the electrogastrographic time signal.¹¹ This procedure generates a series of overlapping spectra and makes both frequency and time analysis possible. The electrogastrography recordings were performed for one hour during a fasting period and for one hour after feeding the same meal that was given during ultrasound measurement of gastric emptying time. Children were examined in a supine position after an overnight fast.

A frequency included between 2.5 and 3.5 cpm was considered normal. Tachygastria was defined as a frequency between 5 and 9 cpm. Frequencies slower than 2.5 cpm were considered as bradygastria, periods of absent electrical signal were defined as 'flat line pattern', and an irregular rhythm resulting from a combination of both tachygastria and bradygastria was defined as 'mixed arrhythmia'. Arrhythmic periods were taken into account if they lasted ≥2 minutes. An arrhythmic index, that is the percentage of arrhythmic time during the time of evaluation, was calculated (60 minutes fasting and 60 minutes postprandially).

Comparison of data between groups was made using non-parametric tests and the level of significance taken was 0.05. Values were given as median and ranges. The study was approved by the ethical committee of our faculty and parents gave informed written consent.

Table 1 Electrogastrographic data for children with non-ulcer dyspepsia and controls. Number (and mean duration in minutes) of arrhythmic episodes

	Tachygastria		Bradygastria		Flat line		Mixed arrhythmias	
	Fasting	Fed	Fasting	Fed	Fasting	Fed	Fasting	Fed
Patients								
1			3 (4.6)				1 (5)	
2				6 (5)		2 (5)		
3	2 (5.5)						2 (4.5)	
4	1 (6)				1 (5)			
5		2 (4.5)	2 (4.5)	1 (3)	2 (6)			1 (4)
6			3 (4.6)	5 (4.6)		2 (6.5)		1 (2)
7	3 (5.3)	1 (3)					1 (3)	1 (14)
8								
9		1 (3)	4 (5)	1 (3)	1 (4)	1 (3)	1 (5)	
10			4 (5.3)	5 (6)	1 (3)	2 (4.5)		
11		1 (4)	3 (5.3)		1 (7)			1 (5)
12		1 (8)	3 (7)	1 (4)			1 (3)	1 (3)
13	3 (6.6)	1 (3)		1 (5)	1 (4)		1 (6)	
14	1 (6)	1 (4)		1 (5)				
Controls								
1				1 (1.3)	1 (1)			
2				1 (1)				
3			1 (2)				1 (2)	
4			1 (1)					
5							1 (1)	
6		1 (1.3)						1 (2)
7		1 (2)						
8								
9						1 (1)	1 (1)	1 (2)
10				1 (1)				

Table 2 Distribution of electrogastrography power (%) (median values and ranges) for three frequency bands in children with non-ulcer dyspepsia and in controls

	Frequency band (cpm)		
	1–2	2.5–3.5	4–9
Fasting:			
Non-ulcer dyspepsia	30.8** (13.3–67.1)	33.4** (13.0–64.1)	27.4* (14.0–47.1)
Controls	17.0 (15.2–31.8)	65.7 (39.3–72.6)	18.7 (13.0–35.4)
Fed:			
Non-ulcer dyspepsia	28.0* (3.8–72.1)	31.5* (15.8–82.5)	29.0 (5.5–50.2)
Controls	15.0 (3.5–25.3)	70.4 (53.7–90.9)	16.1 (4.5–27.3)

*p<0.05 and **p<0.01 patients v controls.

Results

Mean (SD) gastric emptying time (minutes) was significantly prolonged in dyspeptic patients compared with controls (182.14 (35.3) v 102.1 (11.2), p<0.01). In all controls, a gastric electrical activity of about 3 cpm was clearly evident both in the fasting and postprandial states (fig 1 A, B). The difference between patients and controls for arrhythmic index was appreciably significant both in the fasting state (patients: 33.3 (range 10.0–48.3) and controls: 1.6 (range 1.6–33.3), p<0.01) and in the fed period (patients: 15.0 (range 11.6–66.6) and controls: 2.7 (range 1.6–5.5), p<0.01). Twelve

Figure 1 (A) Example of electrogastrography tracing (a) and running spectral analysis (b) from a fasting control subject showing a normal 3 cpm activity. In this and the following figures the running spectral analysis represents consecutive segments of electrogastrography data, of which each is overlapped with the preceding segments. Thus, each line represents 64 seconds of new data added to the previous 192 seconds for a total of 256 seconds of electrogastrography data per line. (B) Postprandial recording from a control subject. The electrogastrography tracing (a) and running spectral analysis (b) show a normal 3 cpm activity.

and 10 patients exhibited gastric arrhythmias during fasting and the postprandial period, respectively (figs 2 and 3). The number of fasting and fed arrhythmic episodes for each patient and for controls are reported in table 1. Table 2 shows the electrogastrography power percentage for three major frequency ranges (1–2, 2.5–3.5, and 4–9 cpm); the fasting and fed normal frequency range was significantly less well represented in the patients compared with

controls. Low frequency electrogastrography activity was significantly more represented in patients than in controls during both fasting and fed periods, whereas patients were different from controls for an increased high frequency activity only during fasting periods. A comparison of the total electrogastrography power across the three frequency bands is represented in fig 4. Among four patients with normal fed electrogastrography tracings only one had also a normal tracing in the fasting state, whereas three showed appreciable fasting arrhythmias; one patient with bradycardia after feeding had a regular tracing at baseline. Arrhythmic episodes were coincident with dyspeptic symptoms such as nausea, feeling of fullness, bloating, and belching in seven patients (four in fasting and fed states, two only after feeding, one only in the fasting state).

Figure 2 Spectral analysis (b) from a fasting patient with non-ulcer dyspepsia showing an initial regular 3 cpm activity which is followed by a period characterised by absence of 3 cpm activity and a tachygastria pattern at approximately 5–6 cpm peaks. The last part of the spectral analysis shows return of 3 cpm activity. The electrogastrography tracing (a) shows a tachygastria pattern with waves at 5–6 cpm.

Figure 3 Postprandial spectral analysis (b) from a patient with non-ulcer dyspepsia showing a flat line pattern and periods of low frequency peaks (1 to 2 cpm) (bradycardia). The electrogastrography tracing (a) shows 2 cpm waves.

Discussion

Gastric motor contractions are controlled by electrical activity of smooth muscle cells of the distal two thirds of the stomach; these generate cyclicly recurring depolarisations of cell membrane and are known as slow waves or electrical control activity.¹² In the human stomach, the interval between two consecutive depolarisations is about 20 seconds (frequency 2.5–3.5 cpm). Slow waves originate from a pacemaker area, located in the proximal part of the corpus at the greater curvature, and propagate in the aboral direction. The slow waves, which are constantly present whether contractions occur or not, control frequency and spatial organisation of mechanical contractions of the gut.¹³ The latter are usually associated with fast oscillating potential changes, called 'spike potentials' or electrical response activity.

Electrical activity of the stomach can be recorded by electrodes implanted on nasogastric (or orogastric) probes and held by suction against antral mucosa.¹⁴ This technique, however, is rather invasive and detachment of the electrodes from gastric mucosa during prolonged

Figure 4 Graphic representation of the total electrogastrography power distribution across three frequency bands of gastric electrical activity. The energy power is reported on the vertical axis as μV^2 . * $p < 0.05$ and ** $p < 0.01$ patients *v* controls.

recordings occurs frequently.¹⁵ Recent reports have shown that gastric electrical activity can be successfully recorded from surface electrodes positioned over the upper abdominal surface.¹⁶ This technique is known as electrogastrography and has been shown to be reliable in detecting abnormalities of electrical rhythm of the stomach.¹⁷

Abnormalities of gastric electrical rhythm detected by electrogastrography have been described in normal subjects with motion sickness,¹⁸ in pregnant women with nausea,¹⁹ and in adults with anorexia nervosa.²⁰ Electro-gastrography has been also used in the diagnostic work up of adult patients with unexplained dyspeptic symptoms and various electrical abnormalities have been detected: decreased or absent postprandial increase in electrogastrography amplitude, instability of gastric frequency, abnormally high (tachygastria) or low (bradygastria) slow wave frequency.^{7,8}

Our study shows that in children with symptoms of non-ulcer dyspepsia abnormal fasting and fed electrogastrography patterns could be discerned, whereas control children without any history of gastrointestinal disorder showed a normal electrogastrographic pattern, with only short periods of arrhythmia. Children with recurrent unexplained symptoms of upper intestinal dysfunction are commonly encountered in paediatric clinical practice and can represent a diagnostic challenge for paediatricians. Recent studies have shown that fasting and/or fed gastrointestinal motor abnormalities can be recorded by manometry in a high proportion of children presenting with symptoms of non-ulcer dyspepsia. These abnormalities included antral and/or duodenojejunal hypomotility, and uncoordinated antroduodenal motility^{2-4,21} and furthermore, delayed gastric emptying has been detected in most of these patients.³

The fact that a large proportion of our patients with dyspepsia and delayed gastric emptying showed gastric arrhythmias is not unexpected as motor events of the stomach are controlled by electrical activity of gastric smooth muscle cells. Abnormal electrical activity of the stomach might disturb motor events either by

destroying the orderly patterns of gastric peristalsis or by inhibiting strength of muscular contractions.²² Whatever the mechanism involved, gastric propulsion and emptying are impaired, and dyspeptic symptoms such as nausea and vomiting can ensue. Recent data in adults indicate that electrogastrography can be used as a screening test in the diagnostic assessment of patients with symptoms suggesting gastric motility disorders.²³ Furthermore, the test seems to be of value in distinguishing functional from mechanical gastroparesis.²⁴

The commonest arrhythmia described in patients with gastric electrical abnormalities is tachygastria; however, in our patients periods of bradygastria were encountered more frequently than tachygastria. If tracings were analysed on the basis of distribution of electrogastrography power, patients with non-ulcer dyspepsia were differentiated from controls for the low frequency range both in the fasting and fed state, and for high frequency ranges only during fasting period. Interestingly, four patients, whose electrogastrography showed a prevailing pattern of bradygastria in the fasting state, developed periods of increased frequency after feeding.

The neurohumoral mechanisms involved in the production of arrhythmias are unknown.²² Gastric arrhythmias can be induced both in dogs and in humans by agents such as prostaglandin E_2 , adrenaline, glucagon, and met-enkephalin.²⁵⁻²⁷ Local prostaglandins seem to mediate the occurrence of arrhythmias by adrenaline and met-enkephalin, whereas glucagon seems to induce gastric arrhythmias by a non-prostaglandin mediated mechanism.²⁶ Whether gastric arrhythmias and dyspeptic symptoms arise from an abnormal release of some of the above mentioned agents in the circular muscle or at level of myenteric plexus of intestinal wall is still unknown. The correlation of gastric arrhythmic patterns with neuroendocrine measures and with dyspeptic symptoms would require more extensive evaluation.

Previous studies have indicated that gastric arrhythmias occur rarely after feeding, most likely because postprandial mechanical and neurohormonal changes might make the stomach less susceptible to arrhythmia.^{28,29} On the other hand, there is experimental evidence that inhibition of cholinergic activity causes shortening of refractory periods of smooth muscle cells and increases slow wave frequency.³⁰ It is tempting to suggest that in patients with non-ulcer dyspepsia defective release of acetylcholine from intramural cholinergic nerves or the effects of arrhythmic agents might give rise to both occurrence of gastric electrical arrhythmias and decreased motility.

In our view, there is reason to believe that electrogastrography is a promising technique in the diagnostic approach to patients complaining of recurrent symptoms suggesting functional gastrointestinal disease and without organic explanation for their symptoms. Both electrogastrography and gastric emptying time measurement can be helpful in identifying altered gastric function. Such information may be quite useful in establishing a correct diagnosis

and in directing a viable management plan. However, the question of how specific and sensitive the findings of gastric arrhythmias are in patients with unexplained gastrointestinal functional symptoms remains to be answered. In addition, it remains to be established whether drugs normalise the arrhythmias and relieve the patient's symptoms.

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