Pediatr Surg Int (1997) 12: 340–343

ORIGINAL ARTICLE

M. Yagi · S. Homma · M. Iwafuchi · M. Uchiyama
Y. Matsuda · T. Maruta

Electrogastrography after operative repair of esophageal atresia

Accepted: 28 June 1996

Abstract Esophageal atresia (EA) is a life-threatening disorder associated with operative complications. Postoperative gastric electrical control activity detected by a non-invasive electrogastrography (EGG) technique was investigated in 13 children aged 1–17 years to clarify whether gastric motility disorders were present. EGG abnormalities were present in 5 patients; persistent dysrhythmias were found in 3. Roentgenographic examinations showed mild gastroesophageal reflux in 3 (60%) of the dysrhythmic patients; 2 others had postprandial dysrhythmias. The mean spectral frequency (MSF) of EA cases with dysrhythmia was significantly higher than that of patients without dysrhythmia in both fasting and postprandial states ($P < 0.05$). The variability of the peak spectral frequencies (PSFV) in patients with dysrhythmia was significantly higher than in those without dysrhythmia in both fasting and postprandial states ($P < 0.05$). There were no significant differences in MSF and PSFV between EA patients without dysrhythmia and controls. These results suggest that gastric motor activity may be disordered in patients following operative repair of EA, although they remain asymptomatic. EGG may be a useful screening examination for postoperative gastric functional disorders.

Key words Esophageal atresia · Electrogastrography

M. Yagi (✉) · M. Iwafuchi · M. Uchiyama · Y. Matsuda
Department of Pediatric Surgery,
Niigata University School of Medicine,
1-757 Asahimachi-dori, Niigata-City,
Niigata 951, Japan

S. Homma · T. Maruta
Department of Physiology II,
Niigata University School of Medicine,
1-757 Asahimachi-dori, Niigata-City, Niigata 951, Japan

Introduction

The most severely life-threatening congenital anomalies of the esophagus are esophageal atresia (EA) and its variants.

Esophagoesophagostomy using a single-layer technique has been commonly used for many years for the repair of EA.

Even in long-gap cases, this anastomosis is carried out after bougienage for elongation. Colonic interposition is sometimes used in such cases. However, feeding difficulties and many kinds of gastrointestinal (GI) complaints are common in the postoperative period. It is essential to perform long-term follow-up in growing children as well as appropriate postoperative management.

Although many kinds of postoperative symptoms are probably related to disordered gastric motility and abnormal gastric emptying, few studies have been carried out to determine the motility following radical operations. The aim of this study was to evaluate postoperative gastric function in patients after operative repair of EA with the use of electrogastrography (EGG).

Patients and methods

Thirteen patients (9 boys and 4 girls) who had undergone repair of EA and 5 control subjects (1 boy and 4 girls) were evaluated. Their ages ranged from 1 to 17 years (mean 7.6 years) in the EA group and 5 to 11 years (mean 8.2 years) in the controls. All the EA patients were nearly asymptomatic. According to Gross's classification, there were 3 type A and 10 type C cases. There was no evidence of any GI disorders at the time of the investigation in the controls.

Bipolar recording was performed with a pair of electrodes attached to the skin of the epigastric region. A high cut-off filter and time constant were set at 0.5 Hz and 5 s, respectively. The EGG signals were saved on a data recorder for later computer analysis. All patients were fasted after the evening meal on the day before recording. The EGG recordings were obtained about 30 min before and after a low-residue test meal (Clinimeal, Eizai, Japan) with a calorie density of 1 kcal/ml and 10 kcal/kg body weight. Patients were supine during EGG recording.

The EGG signals were analyzed spectrally using the maximum entropy method (MEM), which employed a personal computer for identifying the periodicity of the recorded data. It has been reported that MEM is superior to the fast Fourier transform method for analyzing short samples to obtain smooth spectra [6]. Running spectra, or pseudo-three-dimensional displays, were made by piling up each analyzed spectrum. One spectrum was derived from one file of original waves for 128 s (256 points); this indicated the time course of the peak spectral frequencies (PSFs) and their amplitudes or power. Ensemble means of spectral power representing the fasting and postprandial periods were calculated to derive the mean spectral peaks and their powers. The power ratio (PR) was defined as the ratio of postprandial/fasting signal power at around 3 cycles/min (cpm) of the ensemble means of spectral power.

The power or amplitude around 3 cpm usually increases in the postprandial state in adults [1, 5]. The absolute power values may be influenced by various factors, including electrode-skin resistance, tissue conductivity or fat content, and the distance from the electrode to the gastric wall. Therefore, comparison of inter-individual power data was not practical, so that fasting and postprandial power changes in each individual were used to determine the PR [3, 6].

To compare rhythm irregularities of the gastric electrical control activity (ECA), the variability of the PSF was defined as follows: the means and standard deviations (SD) of PSFs ranged from 1 to 9 cpm with maximum power in each file. These SDs were defined as variability of PSF (PSFV). Furthermore, we defined PSFVs of fasting or/and postprandial spectral frequencies larger than 1.3 as dysrhythmias.

Results

The fasting and postprandial PSFs in the controls were 3.14 ± 0.331 cpm and 2.99 ± 0.303 cpm, respectively. Those of the EA group were 3.31 ± 0.778 and 3.43 ± 0.504, respectively. There was no significant difference between the groups. Dysrhythmias occurred in 5 of the EA cases, all of them arrhythmias [1]. Only 2 patients had postprandial dysrhythmias; the others showed dysrhythmias in both the fasting and postprandial states (Fig. 1). Although roentgenographic examinations showed mild gastroesophageal reflux (GER) in 3 (60%) of the 5 dysrhythmic patients, they did not have any symptoms. Two (67%) of these GER patients were type A in the Gross classification. Another 2 dysrhythmic patients did not have any GI symptoms.

The mean spectral frequencies (MSFs) of the EA patients with fasting and postprandial dysrhythmias were 3.97 ± 0.896 and 3.79 ± 0.588 cpm, respectively, those of EA patients without dysrhythmias 2.90 ± 0.266 and 3.20 ± 0.288 cpm, respectively. The MSFs of the EA patients with dysrhythmias were significantly higher than those of the patients without dysrhythmias in both the fasting and postprandial states ($P < 0.05$) (Table 1). There was no significant difference in MSF between the EA patients without dysrhythmias and the controls.

The PSFVs of the EA patients with dysrhythmias in the fasting and postprandial states were 1.35 ± 0.809 and 1.97 ± 0.425 cpm, respectively, those of the patients without dysrhythmias 0.539 ± 0.338 and 0.629 ± 0.263 cpm, respectively. The PSFVs in patients with dysrhythmias were significantly higher than those of the patients without dysrhythmias in both the fasting ($P < 0.05$) and postprandial states ($P < 0.001$) (Table 2). There was no significant difference in PSFV between EA patients without dysrhythmias and controls.

The PR values were above 1 in 10 (77%) of the EA cases and 4 (80%) controls. The PR of the EA group was 2.62 ± 1.73 and that of the controls 7.56 ± 8.98. The PR of the controls was significantly higher than that of the EA group ($P < 0.05$). The PRs of the EA patients with and without dysrhythmias were 2.45 ± 1.51 (n=5) and 2.74 ± 1.95 (n=8), respectively. The presence of dysrhythmias had no relationship with PR value (Table 3).

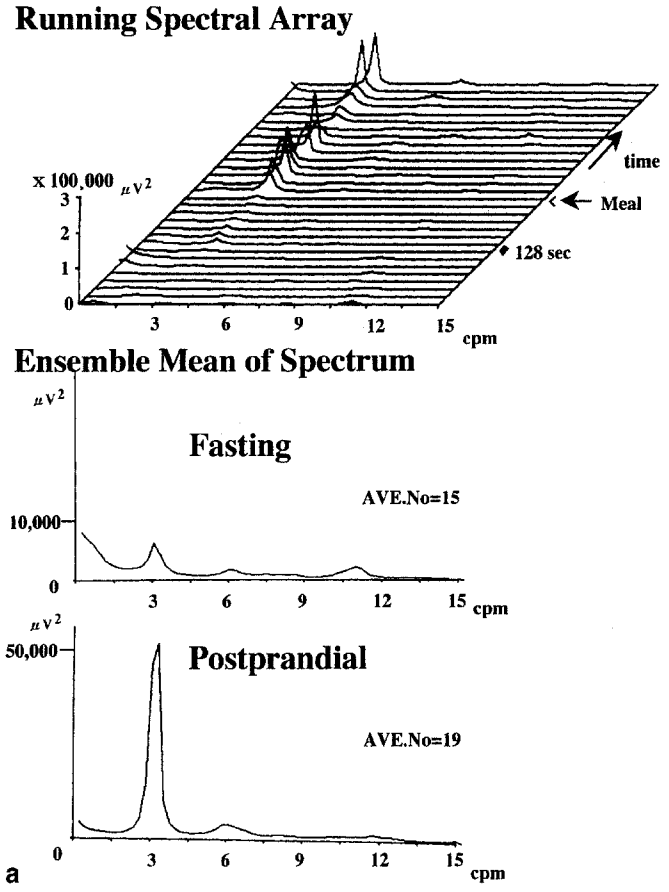
Discussion

EA, which is usually associated with tracheoesophageal fistula, is fatal if not diagnosed and treated surgically. Although steady improvement in the care of such patients has occurred, the management continues to be a challenge to all those caring for these infants. Long-term results for patients who survive associated anomalies and EA repair are generally good. However, uncorrected GER persists in 60% of cases [7]. Although the cause of GER is speculative, it is generally associated with delays in gastric emptying.

We evaluated postoperative gastric function in patients after operative repair of EA by means of EGG. The electrical activity of the stomach consists of cyclic depolarization of the membrane potential of smooth-muscle cells, called ECA or slow-wave activity. ECA emanates from a pacemaker area located along the greater curvature and is propagated in an aboral direction toward the pylorus at a frequency of 3 cpm in humans. It is not necessarily associated with gastric mechanical activity, but is believed to set motor contractions temporally. When ECA shows an increase in plateau depolarization with superimposed spike potentials, the stomach exhibits motor contractions that are involved in mechanical trituration and propulsion of ingested food [10]. There is experimental evidence that smooth-muscle cells of the stomach can exhibit an abnormal slow-wave frequency after inhibition of cholinergic activity [8].

Gastric dysrhythmias are disorders of electrical rhythm. They include tachygastria (frequency $\geq$ 5 cpm), bradygastria (frequency < 2 cpm), and arrhythmias, which are defined as irregular slow waves including tachy- and bradyarrhythmias [1]. Gastric dysrhythmias are generally recorded in a variety of clinical syndromes in which gastric motility disorders and symptoms of nausea are present [11]. It is commonly believed that the gastric threshold to dysrhythmias is increased postprandially due to the effects of mechanical and neurohormonal changes on gastric smooth muscle [4]. Furthermore, there is a suggestion that gastric dysrhythmias can impair motility by either inhibiting the strength of antral contractions or destroying the aboral propagation of electrical potential and peristalsis [2].

Chen et al. reported that normal slow-wave frequency in the EGG was related to normal gastric motility and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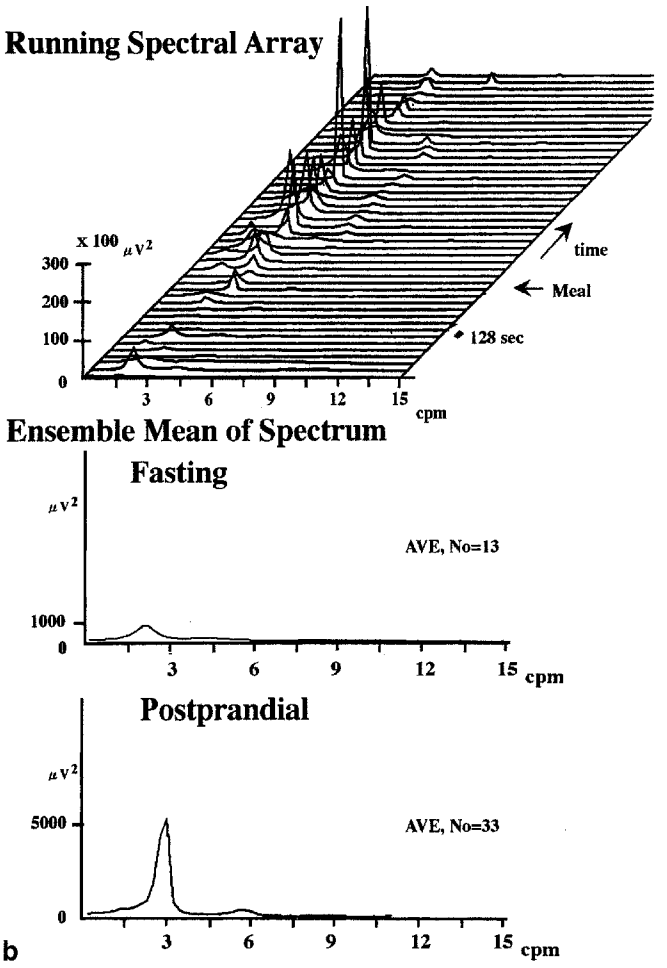
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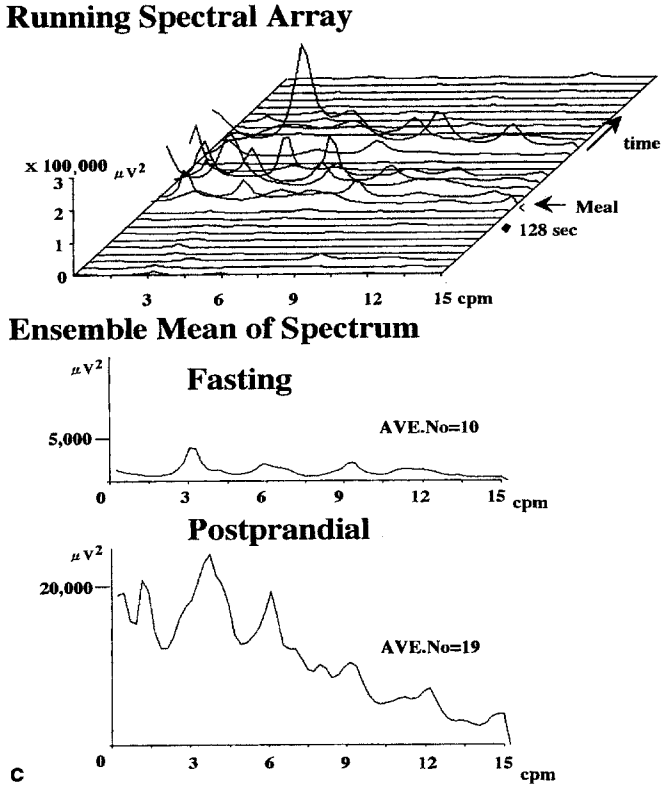
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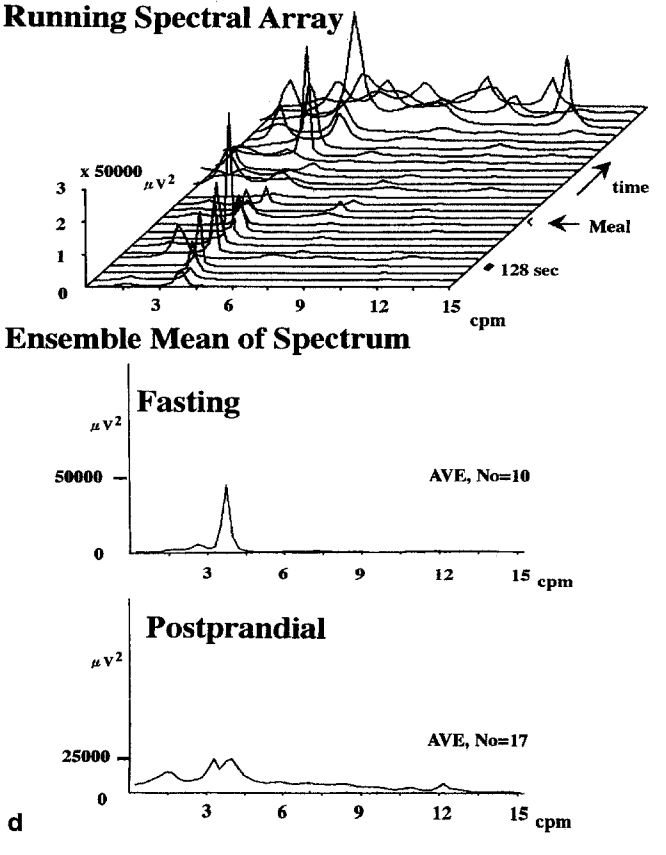
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AVE, No=10
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Postprandial
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AVE, No=17
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3 6 9 12 15 cpm
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Table 1 Relationship between mean peak spectral frequencies and dysrhythmias in patients with esophageal atresia

	Dysrhythmia (+)(cpm)	Dysrhythmia (−)(cpm)	*P* Value
Fasting	3.97 ± 0.896 (n = 5)	2.90 ± 0.266 (n = 8)	< 0.01
Postprandial	3.97 ± 0.588 (n = 5)	3.20 ± 0.288 (n = 8)	< 0.05

mean ± SD

Table 2 Variability of peak spectral frequencies in patients with esophageal atresia

	Dysrhythmia (+)(cpm)	Dysrhythmia (−) (cpm)	*P* value
Fasting	1.35 ± 0.809 (n = 5)	0.539 ± 0.338 (n = 8)	< 0.05
Postprandial	1.97 ± 0.425 (n = 5)	0.629 ± 0.263 (n = 8)	< 0.001

mean ± SD

Table 3 Comparison of power ratios = ensemble mean of postprandial electrogastrographic (EGG) power/ensemble mean of fasting EGG power; EGG power = power of 3 cpm components of EGG power spectrum; (*EA*; esophageal atresia)

	Power Ratio	
EA overall (n = 13)	2.62 ± 1.73	
EA with Dysrhythmia (n = 5)	2.45 ± 1.51	*
EA without Dysrhythmia (n = 8)	2.74 ± 1.95	* *
Controls (n = 5)	7.56 ± 8.98	*

mean ± SD, *: $P < 0.05$

abnormal slow-wave frequencies were associated with motility disorders [1]. According to the severity of the dysrhythmia, normal mixing and emptying of a meal might be disturbed. Therefore, normal gastric propulsion is impaired, which may lead to gastric stasis.

Although the increased amplitude in the postprandial EGG reflects increased contractile strength, there is no one-to-one correlation between EGG amplitude and the strength of contractions in adults. The relative increase in EGG amplitude provides information about the contractions of the stomach [9]. To compare EGG amplitudes, we defined them as PR. The PR of the controls was significantly higher than that of the EA patients. Therefore, the postoperative contractile activities of the EA patients might be lower than those of the controls.

In this study, there were some dysrhythmic patients without symptoms following operative repair of EA. The dysrhythmias were detected in most of the patients during both fasting and postprandial states. Furthermore, mild GER was present in these cases. None of the EA patients had any serious operative complications. It may be suggested that some EA patients have congenital gastric dysrhythmias. Furthermore, the MSF of the EA patients with dysrhythmias was significantly higher than that in patients without dysrhythmias in both fasting and postprandial states. Although the mechanism is uncertain, the neural abnormalities may be due to loss of intrinsic inhibitory innervation or a lack of extrinsic autonomic inhibition.

It is possible that gastric dysrhythmias can play a pathogenetic role in patients with and without functional GI symptoms after operative repair of EA. Whenever dysrhythmias are detected in the EGG, possible gastric motility disorders should be taken into consideration. Future goals might be to prevent dysrhythmias or restore normal 3 cpm gastric rhythms using drugs and other methods.

In conclusion, gastric dysrhythmias appear to be a marker of gastric neuromuscular dysfunction. EGG is a promising method of studying postoperative gastric function in patients with EA.

Fig. 1a–d Running spectral array and ensemble mean of spectrum in fasting and postprandial states. **a** Control subject: peak spectral frequency (PSF) stable and regular with increase in postprandial power. **b** Patient without gastric dysrhythmia: PSF relatively stable and regular with increase in postprandial power. **c** Patient with gastric dysrhythmia: PSF variable, but increase in postprandial power can still be recognized. **d** Patient with gastric dysrhythmia: PSF variable postprandially with decreased power

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