Correlations Among Electrogastrogram, Gastric Dysmotility, and Duodenal Dysmotility in Patients With Functional Dyspepsia

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Aim: The aim of this study was to assess gastric slow waves, antral and duodenal motility simultaneously, and determine the correlation among all these measures in patients with functional dyspepsia.

Methods: Thirty-one patients with functional dyspepsia were assessed for severity of upper gastrointestinal symptoms with the electrogastrography (EGG) and antroduodenal manometry. The EGG and manometry were recorded for 3 to 4 hours in the fasting state and for 2 hours after a solid meal. Computerized spectral analysis methods were used to compute various EGG parameters.

Results: The EGG was abnormal in 71.0% of patients. The abnormalities included normal slow waves lower than 70% in the fasting state (51.6% of patients) and in the fed state (48.4% of patients), a decrease in dominant power in 28.9% of patients. Antral motility was abnormal in 80.6% of patients and duodenal motility was abnormal in 74.2% of patients. For the EGG and antral motility, 19 of 31 patients had both abnormal EGG and abnormal antral motility; 2 of 31 patients had both normal EGG and normal antral motility. For the EGG and duodenal motility, these values were 16/31 and 2/31, respectively. By both EGG and antroduodenal manometry, abnormal gastric motor function was found in 93.5% of patients. However, quantitative one-to-one correlation between any of the EGG parameters and the antroduodenal dysmotility was not noted. The patients showed high symptom scores particularly to upper abdominal pain, nausea, and belch. No one-to-one correlation was noted between the symptom scores and any of the EGG or motility parameters.

Conclusions: More than two-thirds of patients with functional dyspepsia have abnormalities in the EGG and antral/duodenal motility. The sensitivity of these 2 different methods is essentially the same. EGG and antroduodenal manometry can complement each other in demonstrating gastric motor dysfunction in patients with functional dyspepsia.

Key Words: functional dyspepsia, gastric myoelectrical activity, electrogastrography, manometry, motility, postprandial symptom

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Functional dyspepsia is a common clinical syndrome defined as pain or discomfort centered in the upper abdomen, which is not explained by any identifiable structural or biochemical abnormality.[1,2] It occurs in 14% to 20% of the population.[3,4] The afflicted often complains of symptoms related to meals, including fullness, early satiety, bloating, regurgitation, nausea, and/or vomiting.[5,6] In this syndrome, no cause can be identified using conventional diagnostic testing.

Although the pathogenesis of functional dyspepsia remains unclear, abnormal gastric, and/or small bowel motility has been found in up to 50% of patients with unexplained dyspepsia, such as gastric hypomotility and uncoordinated antral duodenal contractions.[7–12] A number of studies have shown that patients with functional dyspepsia have disordered gastric motility.[8–10,13] The most common abnormality is antral hypomotility, as defined by antroduodenal manometry.[14–16] The motor function of the gastrointestinal tract is characterized by contractions that are coordinated in time and space by the rhythmic myoelectrical activity known as electric control activity or slow waves. Excitatory neural input or hormones may induce spikes, which can potentially be superimposed on the slow waves, leading to contractions. Adjacent slow waves are coupled to each other through low resistive areas, known as gap junctions, enabling the gastrointestinal smooth muscles to generate coordinated contractions. Thus, slow waves, in essence, control the underlying motility. This has led to the study of slow waves as a tool for the investigation of normal and altered motility.

It has been implied that gastric motor disorders in patients with functional dyspepsia may be at least partially attributed to gastric myoelectrical dysrhythmias.[17,18] As gastric myoelectrical activity modulates gastric motor activity, abnormalities in gastric myoelectrical activity may lead to gastric motor abnormalities. Electrogastrography (EGG) measures gastric myoelectrical activity by means of electrodes attached to the abdominal surface, similar to electrocardiogram recording. It provides information on the frequency and relative amplitude of antral contractions.[17,19] EGG abnormalities have been found in patients with gastroparesis and nonulcer dyspepsia,[20,21] but the clinical utility of EGG in evaluating patients with symptoms of upper gastrointestinal motor dysfunction, or dyspepsia, has not been well established. Nor is the relationship between abnormalities in the EGG and abnormalities in gastric and duodenal motility in patients with functional dyspepsia.[22]

Abnormalities in gastric slow waves and motility are frequently reported in patients with functional dyspepsia. However, simultaneous assessment of the slow wave and motility abnormalities has rarely been performed. The aims of this study were to assess gastric slow waves and antroduodenal motility simultaneously and to determine

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the correlation among these measures in patients with functional dyspepsia.

MATERIALS AND METHODS

Study Subjects

The study was performed on 31 patients with functional dyspepsia (23 women, 8 men, mean age 49 y) based on clinical and laboratory diagnoses. All patients had a minimum 3-month history of chronic, persistent, or recurrent upper abdominal discomfort or pain after meals, and had endoscopy that was negative for any focal lesions, including esophagitis, gastric or duodenal ulcers or erosions, and esophageal or gastric malignancy within 3 months before the study. None of the patients had been diagnosed for gastroesophageal reflux disease, irritable bowel syndrome, chronic intestinal pseudo-obstruction, or diabetes. None of these patients had any clinical, biochemical, or ultrasonographical evidence of any organic diseases. Patients with a history of gastrointestinal surgery or taking any drugs that might alter gastric motility were excluded from the study. The selection of these patients was based on the Rome II criteria and a symptom score with a history of the symptoms of $\geq$6-month duration (continuous and intermittent). Common gastrointestinal symptoms of epigastric pain: postprandial bloating, nausea, vomiting, early satiety, anorexia, belch, diarrhea, heart burn, chest pain, regurgitation, acid reflux, and dyspepsia were assessed for symptom severity and graded by the patient as none (0), mild (1), moderate (2), severe (3), or very severe (4). None of the subjects took any medications known to affect gastrointestinal motility during the 3 days before the study. The study protocol was approved by the Human Subjects Committee at the University of Texas Medical Branch at Galveston, and written consent forms were obtained from all subjects before the study.

Study Protocol

After an overnight fast of $\geq$8 hours, the subject was examined at the Division of Gastroenterology Motility Laboratory in the morning. A water-perfused manometric catheter was placed into the duodenum under fluoroscopy with 3 sensors in the stomach and 3 sensors in the duodenum. Antral-duodenal manometry was performed for 3 hours in a supine position in the fasting state. The subject was then allowed to sit for 15 minutes and consumed a standard test meal of 500 kcal (30% fat, 30% protein, 40% carbohydrate) with 250 mL of noncaffeinated, noncarbonated juice. After the meal, the recording was resumed for another 2 hours in the same supine position The recording of the electrogastrogram was made simultaneously. At the end of the study, the recording devices were disconnected and the catheter and the electrodes were removed.

Recording of Gastric Myoelectrical Activity

Gastric myoelectrical activity of the subjects was measured using surface EGG with a specially designed multichannel device (Medtronic-Synectics, Shoreview, MN). The EGG consisted of 4 identical amplifiers with cutoff frequency ranges of 1.0 to 12.0 cycles/min (cpm). An analog to a digital converter was installed in the recording device for the online digitization of the EGG. The sample frequency was 4 Hz. The placement of electrodes and the preparation of the epigastric skin were the same as those reported by Lin and Chen.[11] All recordings were made in a quiet room and the subject was asked not to talk and to remain as still as possible during the recording to avoid motion artifacts.

Recording of Gastric and Duodenal Motility

The manometric catheter consisted of 6 side holes with an interval of 3 cm for the 3 proximal side holes and an interval of 10 cm for the 3 distal side holes. The catheter was placed fluoroscopically and the location of the catheter was again verified fluoroscopically at the completion of the recording. The catheter was connected to pressure transducers and the manometric recording was amplified using a PC-Polygraph (Synectics, Stockholm, Sweden).

Data Analysis

Before the computerized analysis of the EGG, segments of the recording with motion artifact were deleted. The motion artifact was distinguished by abnormally high amplitude occurring simultaneously in all 4 channels. Previously validated quantitative analysis software was used to derive the following parameters[23,24]:

EGG Dominant Frequency/Power

The frequency at which the EGG power spectrum had a peak power in the range of 0.5 to 9.0 cpm was defined as the EGG dominant frequency. The power at the dominant frequency in the power spectrum was defined as the EGG dominant power. These 2 parameters were calculated by using the smooth power spectral analysis methods. The EGG dominant frequent was defined as abnormal if it was out of the range of 2 to 4 cpm. The EGG dominant power was defined as abnormal if there was a decreased dominant power postprandial.

Percentage of Normal Gastric Slow Waves

The percentage of normal gastric slow waves was defined as the percentage of time during which regular 2 to 4 cpm slow waves were present over the entire recording period and was computed by using the adaptive spectral analysis method. That the percentage of normal slow wave frequency was more than 70% was defined as normal.

Percentage of Gastric Dysrhythmia

Gastric dysrhythmia includes tachygastria, bradygastria, and arrhythmia. The calculation of the percentage of gastric dysrhythmia was performed in the same way as the percentage of normal gastric slow waves. The EGG was called tachygastria if the peak power was in the range of 4 to 9 cpm, bradygastria if in the range of 0.5 to 2 cpm, and arrhythmia if there was no dominant peak.

Analysis of Manometric Data

The manometric recordings were analyzed visually for the assessment of any abnormal motility patterns including abnormal propagation or configuration of phase 3, and sustained (> 20 min) periods of uncoordinated phase activity, inability of the test meal to induce conversion of fasting motor activity to a fed pattern (neuropathy) and low amplitude (< 20 mm Hg) contractions (myopathy). Automated numerical analysis used a validated computer program, which was set to detect contractions with the amplitude greater than 10 mm Hg. The following parameters were measured: mean frequency and amplitude of contractions during 3 hours of fasting and during the hour

immediately after the intake of the test meal at the most distal sensor, the duration, frequency, amplitude, and propagation velocity of spontaneous migrating motor complex phase. Whenever more than one phase 3 complex appeared in fasting, the measurements were made on the first.

Statistical Analysis

All data are presented as means ± standard error. Paired Student t test was applied to investigate the difference before and after the test meal. Correlation analysis was performed using bivariate correlation analysis and χ^2 analysis. A P value less than 0.05 was considered statistically significant.

RESULTS

Disorganized Gastric Slow Wave in Patients With Functional Dyspepsia

The EGG was abnormal in 71.0% (22/31) of patients (Fig. 1). The abnormalities included (a) percentage of normal slow waves lower than 70% in 51.6% (16/31) of patients in the fasting state and 48.4% (15/31) in the fed state; (b) 28.9% (9/31) of patients had abnormal responses to test meal (Fig. 2). Among the slow wave abnormalities, arrhythmia had the highest prevalence (38.7% of the patients in the fasting state and 31.9% in the fed state), followed by tachygastria (25.8% of the patients in both fasting and fed states), and bradygastria (3.2% of the patients in the fasting and 6.4% in the fed state). Typical running spectra showing normal slow waves and arrhythmia are presented in Figure 3. Absence of spectral peaks (arrhythmia) was noted in Figure 3b (see spectra at the bottom of the figure).

When comparing preprandial and postprandial states, the dominant frequency and dominant power were significantly increased in the fed state. Whereas, the percentage of normal slow waves, bradygastria, tachygastria, and arrhythmia, showed no significant changes after the test meal (Table 1).

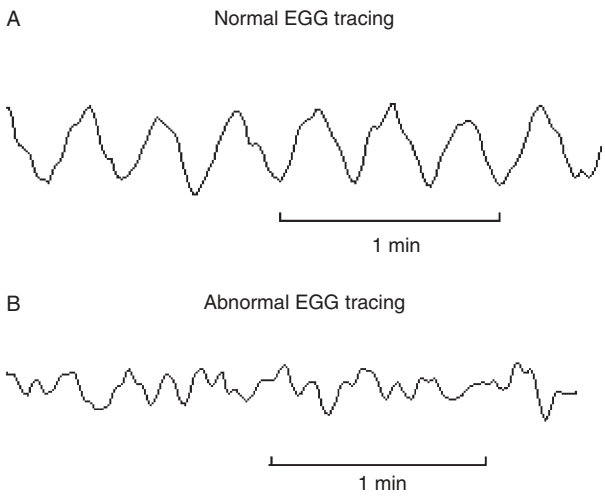

FIGURE 1. Typical normal EGG tracings (upper panel) and arrhythmia (lower panel) in FD patients. A, Normal EGG tracing. B, Abnormal EGG tracing. EGG indicates electrogastrography; FD, functional dyspepsia.

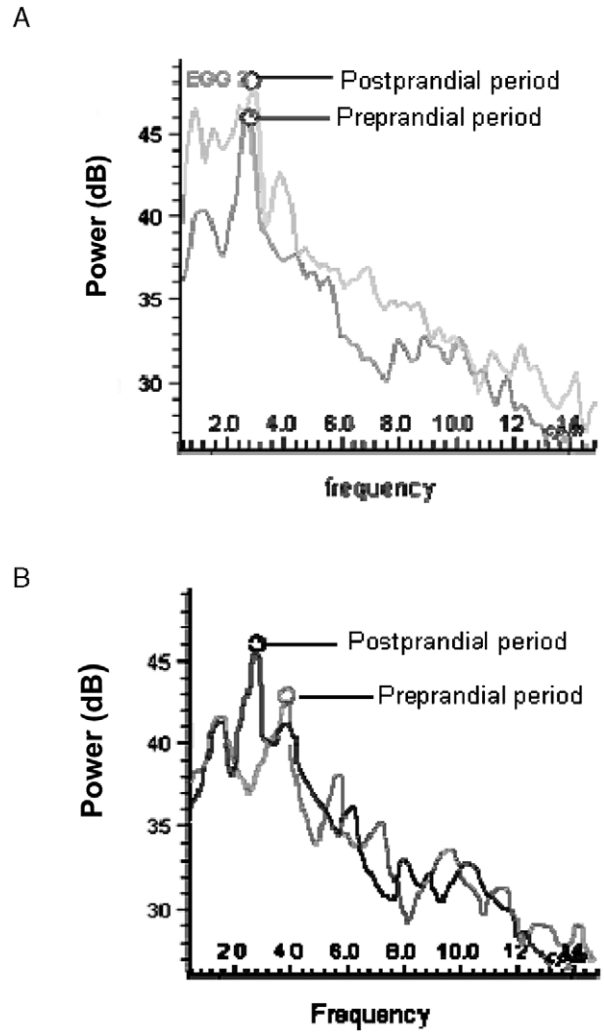

FIGURE 2. The power spectrum of the slow wave frequency showing the normal (upper panel) and abnormal (lower panel) response to the test meal. A, Increase of dominant power in the postprandial period. B, Decrease of dominant power in the postprandial period.

Abnormalities of Antral and Small Bowel Manometry

Visual analysis of manometric charts revealed that antral motility was abnormal in 25 of 31 patients (80.6%) and duodenal motility was abnormal in 23 of 31 patients (74.2%) with functional dyspepsia. Dysmotility of both the stomach and duodenum was seen in 22 of 31 patients. Only 5 patients were judged to have both normal small bowel and gastric motility patterns. There was a significant correlation between gastric and duodenal abnormalities in motility ($P < 0.01$, χ^2 analysis).

Twenty-four patients showed absence of phase 3 of the migrating motor complex in the fasting state. Abnormal antral motility with absent phase 3 and hypotensive postprandial activity was seen in 22 patients. Hypotensive duodenal contractions in the fed state were seen in 20 patients (Fig. 4). Association between abdominal discomfort and duodenal clustered nonpropagating contractions was observed postprandially in 8 patients.

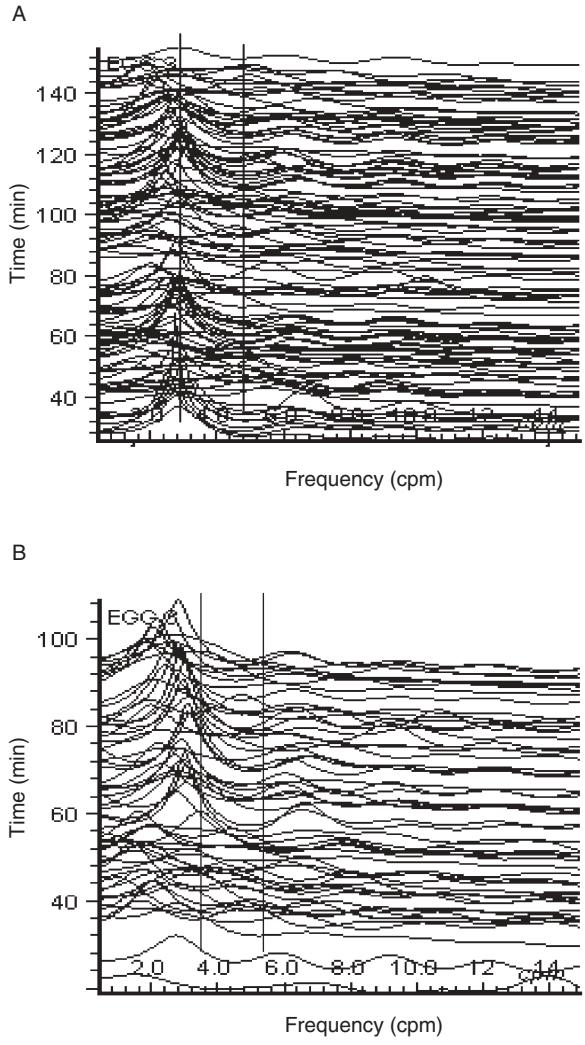

FIGURE 3. The running spectrum of the slow wave frequency showing a regular 2 to 4 cpm frequency (upper panel) and arrhythmia (lower panel) in the preprandial period. A, Normal slow wave frequency. B, Arrhythmia.

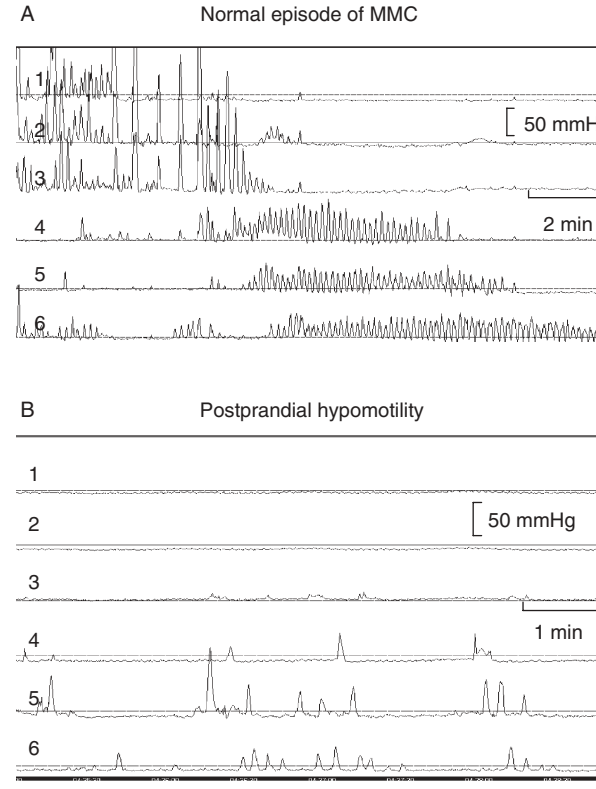

FIGURE 4. Normal and abnormal gastric motility. A, One episode of clustered phasic antral and duodenal contractions propagating distally met the criteria for the phase 3 episode of the MMC. The antral contraction amplitude ranged between 50 and 230 mm Hg (mainly 100 and 140 mm Hg) at a rate of 3/min. Duodenal contractions were with amplitude ranging between 15 and 60 mm Hg (mainly 30 and 40 mm Hg) at a rate of 12/min. B, Postprandial recording with fed pattern showing clusters of propagating intermittent contractions in the distal duodenum. Note the lack of antral contractions (upper 3 leads). MMC indicates migrating motor complex.

Correlation Between EGG and Antral, Small Bowel Manometry

For the EGG and antral motility, 19 of the 31 patients had both abnormal EGG and abnormal antral motility whereas only 2 of the 31 patients had both normal EGG and normal antral motility. For the EGG and duodenal motility, 16 of the 31 patients had both abnormal EGG and abnormal duodenal motility.

A significant correlation was noted in the prevalence of abnormal EGG with that of abnormal gastric motility but not abnormal duodenal motility. As shown in Table 2, 15 of the 22 patients with abnormal EGG showed abnormal gastric motility ($P < 0.05$, χ^2 analysis), whereas only 10 of these 22 patients had abnormal duodenal motility. When each of the abnormal EGG patterns was examined, it was found that the prevalence of arrhythmia was correlated with the abnormal gastric slow waves ($P < 0.04$, χ^2 analysis) but other patterns of abnormal EGG were not correlated with gastric or duodenal motility.

TABLE 1. Preprandial and Postprandial Dominant Frequency (DF) and Dominant Power (DP), Percentage of Normal Slow Wave, Bradygastria, Tachygastria, and Arrhythmia in Patients With FD

	Preprandial	Postprandial	Paired *t* Test
DF (cpm)	2.9 ± 0.1	3.1 ± 0.1	$P < 0.001$
DP	40.1 ± 1.3	43.7 ± 1.5	$P < 0.05$
Percentage of normal slow wave	68.4 ± 3.2	71.3 ± 3.0	NS
Bradygastria (%)	6.0 ± 0.8	6.1 ± 0.9	NS
Tachygastria (%)	10.2 ± 1.8	11.0 ± 2.4	NS
Arrhythmia (%)	14.8 ± 2.0	11.8 ± 1.7	NS

FD indicates functional dyspepsia.

Gastrointestinal Symptoms in Patients With Functional Dyspepsia

The patients showed high symptom scores particularly to upper abdominal pain, nausea, and belch, with an average symptom score of 2.5/4 for pain; 2.5/4 for nausea; and 2.4/4 for belch, respectively. The next symptom scores ranked from high to low were 2.2/4 for bloating, 2.1/4 for

TABLE 2. Relationship Between Abnormal EGG and Abnormal Gastric/Duodenal Motility

Slow Wave Abnormalities	Abnormal EGG (No. Patients)	Abnormal Gastric Motility (No. Patients)	Abnormal Duodenal Motility (No. Patients)
Total	22	15	10
Arrhythmia	12	10	6
Tachygastria	8	4	4
Bradygastria	2	1	0

The number in each cell indicates the number of patients with certain abnormalities.
EGG indicates electrogastrogram.

vomiting, 1.7/4 for regurgitation and early satiety, 1.5/4 for diarrhea, 1.4/4 for acid regurgitation and heart burn, 0.9/4 for anorexia, and 0.8/4 for chest pain (Fig. 5). The mean total score was 20/48 for all 12 epigastric symptoms. No significant correlation was found between symptom scores and percentage of normal slow waves ($R = 0.05$, $P = 0.83$, Fig. 6), percentage of bradygastria ($R = 0.06$, $P = 0.81$), percentage of tachygastria ($R = 0.12$, $P = 0.75$), or percentage of arrhythmia ($R = 0.17$, $P = 0.63$). This indicated that the total symptom score was not correlated with any of the EGG parameters.

Association between abdominal discomfort and duodenal clustered nonpropagating contractions was observed postprandially in 8 patients. Although overall, the patients had significant symptoms and also significant abnormalities in manometry, a one-to-one quantitative correlation was not found. In other words, the patients had abnormalities in motility and also symptoms of dyspepsia, but the severity in impaired motility was not found to be correlated with the severity in symptoms.

DISCUSSION

Motility abnormalities of the stomach and upper small bowel have been described in many patients with unexplained dyspeptic symptoms.[7–13,16,25] In this study, however, EGG, gastric, and duodenal motility were simultaneously measured in patients with functional dyspepsia. The results showed that 71.0% of the patients with functional dyspepsia had abnormal gastric myoelectrical activity, 80.6% of the patients had abnormal gastric motility, and 74.2% of the patients had abnormal duodenal motility. The major abnormalities in gastric myoelectrical

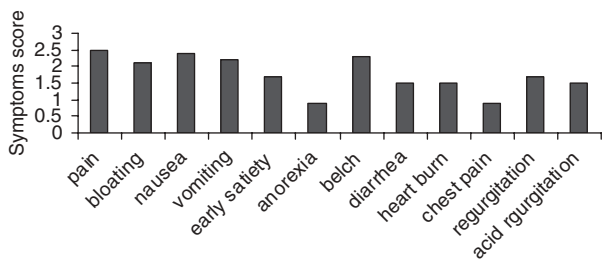

FIGURE 5. Symptom distribution of patients with functional dyspepsia.

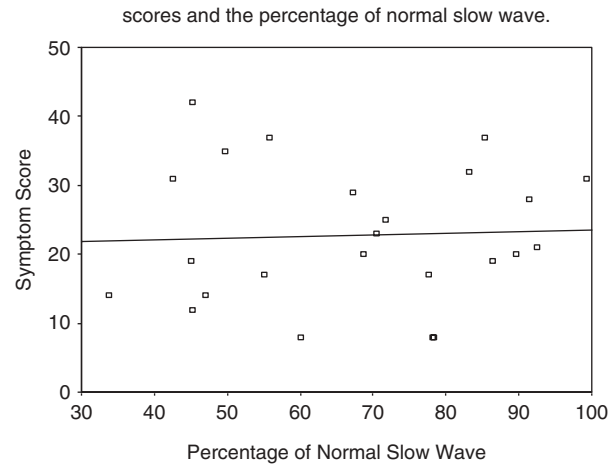

FIGURE 6. Correlation between the percentage of normal slow wave and symptom scores in FD patients with EGG assessment showing no correlation between the symptom scores and the percentage of normal slow wave. EGG indicates electrogastrography; FD, functional dyspepsia.

activity observed in patients with functional dyspepsia were the abnormal rhythmicity of the gastric slow wave and an absence of postprandial increase in the dominant power.

The prevalence of abnormal slow waves revealed from the spectral analysis of the EGG was high in this study. However, these findings were in agreement in many respects with previous studies in patients with various gastric motor disorders. In studies using mucosal electrodes, dysrhythmias were found in 50% of the patients with unexplained dyspepsia symptoms, whereas no dysrhythmias were found in healthy volunteers.[26] In EGG studies, 50% to 66% of patients with functional dyspepsia showed abnormal myoelectrical activity of the stomach characterized by instability of slow wave frequency, tachygastria in both fasting and postprandial EGGs, and absence of the normal amplitude increase in the postprandial EGG.[13,22]

Similarly, the prevalence of abnormal gastric/duodenal motility was also higher than expected. This could be attributed to special referral to the tertiary medical center; patients with more server symptoms are typically referred to major medical centers with well-established motility programs. The patients in this study were referred by gastroenterologists from other hospitals in different regions. According to the literature, similar high prevalence of abnormal EGG and antral/duodenal motility was also reported by other groups from tertiary medical centers. Malagelada and Stanghellini[27] reported a prevalence of 72% of manometric abnormalities in a cohort of 104 patients with functional upper gut symptoms with stationary antroduodenojejunal manometry. Another study using 24-hour antrojejunal ambulatory manometry showed a high prevalence of antral and jejunal dysmotility in both the interdigestive period (71% of patients) and the postprandial period (78%) in patients with severe motility-like dyspepsia.[28]

A variety of EGG findings have been reported in dyspeptic patients in prior studies.[11,13,21,29,30] These findings include inotropic dysfunction (decreased postprandial-preprandial power ratio) and chronotropic dysfunction (gastric dysrhythmias: bradygastria and tachygastria), or a

normal EGG. Thus EGG provides information about the potential mechanism underlying abnormal gastric motor function. Orad propagation of slow waves or loss of electromechanical coupling may impair gastric motility either by causing abnormal motor coordination or by decreasing the frequency and strength of antral contractions. Tachygastria has been associated with gastric hypomotility.[31] The correlation between bradygastria and gastric motility is less clear. Bradygastria has been correlated with strong antral contractions and with absent antral contractions.[32–34] As there has been limited data in the literature showing the correlation between gastric myoelectrical activity and antroduodenal motility in patients with functional dyspepsia, we assessed both gastric myoelectrical activity and antroduodenal motility simultaneously. The findings of this study indicated a significant correlation in the prevalence of abnormalities between the EGG and gastric motility but not between the EGG and duodenal motility. Further more, it was found that the prevalence of arrhythmia was highest among different patterns of EGG abnormalities and was significantly correlated with impaired gastric motility. However, other types of abnormal EGG were not correlated with gastric or duodenal motility.

The origin of postprandial symptoms in dysmotility-like functional dyspepsia is unclear. Although a number of physiologic abnormalities have been well demonstrated in this situation, their relationships to symptoms have not been definitely established.[35] Various mechanisms may explain why symptoms after a meal may arise independently of either the appearance of gastric dysrhythmias or their potential consequences, such as antral hypomotility and delayed gastric emptying. Koch et al[36] found that establishment of normal 3-cpm gastric myoelectrical activity and resolution of dysrhythmias, not normalization of emptying rates, was associated with improvement in upper gastrointestinal symptoms in patients with diabetic gastroparesis. Hu et al[37] found that electrical acustimulation reduced the severity of symptoms of motion sickness and seemed to decrease gastric tachyarrhythmia. Upper abdominal discomfort, bloating, and nausea may be caused by impaired fundic relaxation, leading to increased intragastric pressure and antral overdistention produced by displacement of food from the proximal to the distal stomach.[38–41] Furthermore, increased sensitivity to gastric distention due to visceral hyperalgesia may also be involved in symptom production.[42] The elucidation of the roles of each of these physiologic abnormalities in the pathogenesis of functional dyspepsia demands further studies, which will probably require multiple and sophisticated methods to approach several mechanisms. Some researchers suggested that disturbances of gastric myoelectrical activity might unlikely to play a role in the origin of postprandial upper abdominal discomfort and bloating in dysmotility-like functional dyspepsia.[43] Whereas, Parkman et al[22] found that functional dyspepsia patients with both delayed gastric emptying and abnormal EGG had more severe symptoms. Although overall, these patients had significant symptoms and also significant abnormalities in manometry, a one-to-one quantitative correlation was not found. In other words, the patients had abnormalities in motility and also symptoms of dyspepsia, but the severity in impaired motility was not found to be correlated with the severity in symptoms.

In children with functional dyspepsia, Friesen et al[44] found that an abnormal EGG was associated with a higher mean postprandial pain severity. Although conflicting results have been reported in the literature, in general, abnormalities in gastric myoelectrical activity do not seem to be correlated with dyspeptic symptoms, which is also the case for antral motility.

In summary, this study confirms that more than two-thirds of patients with functional dyspepsia have abnormalities in the EGG and antral duodenal motility. The sensitivity of these 2 different methods is essentially the same. EGG and antroduodenal manometry can complement each other in demonstrating gastric motor dysfunction in patients with functional dyspepsia.

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