Impaired Postprandial Gastric Myoelectrical Activity in Chinese Patients with Nonulcer Dyspepsia

CHING-LIANG LU, MD, CHIH-YEN CHEN, MD, FULL-YOUNG CHANG, MD, LIH-JIUN KANG, BS, SHOU-DONG LEE, MD, HAN-CHANG WU, BS, and TE-SON KUO, PhD

Using a homemade electrogastrography (EGG) system, we studied the characteristics of the myoelectrical rhythm in Chinese patients with nonulcer dyspepsia (NUD). Based on shortterm Fourier transformation, recorded slow waves could be automatically analyzed to obtain the following parameters: dominant frequency/power, percent of normal frequency (2-4 cpm), power ratio, etc. EGG parameters, Helicobacter pylori status, histological examination of gastric mucosa, and dyspeptic symptoms were recorded in 27 NUD patients. Compared to 32 healthy controls, the Chinese NUD patients had abnormal postprandial EGG parameters including a lower percentage of regular 2-4 cpm slow waves (70.10 \pm 2.97% vs 79.08 \pm 2.95%, P < 0.05), a lower level of increment of dominant power (0.62, ± 0.91 vs 3.76 ± 0.58 dB, P < 0.05), lower power ratio (1.42 ± 0.28 vs 2.79 ± 0.39 , P < 0.05) and a higher instability coefficient (0.36 ± 0.03 vs 0.26 ± 0.03 , P < 0.05). However, Helicobacter pylori infection and its associated gastritis did not influence any EGG parameters in NUD patients. Six main dyspeptic symptoms and total symptom score had no correlation with any EGG parameters. In conclusion, Chinese NUD patients may have abnormal postprandial stomach myoelectrical activity, but these EGG abnormalities are not a direct result of Helicobacter pylori infection and its related gastritis and do not contribution to the dyspeptic symptoms.

KEY WORDS: electrogastrography; gastrointestinal motility; nonulcer dyspepsia; gastritis; Helicobacter pylori.

Nonulcer dyspepsia (NUD), or functional dyspepsia, is a heterogeneous disorder characterized by the presence of chronic intermittent symptoms of epigastric pain and fullness, early satiety, nausea, vomiting, etc (1). At present, no relevant abnormalities can be directly measured by standard modalities, including laboratory tests and image studies for NUD patients (2). However, some studies indicate that NUD subjects may have gastrointestinal motor abnormalities. For example, decreased antral and duodenal contractility to an ingested meal, uncoordinated and non-propagated duodenojejunal peristalsis, and prolonged gastric emptying have been reported in NUD patients (3–7).

The stomach muscle itself has a myogenic mechanism in mediating motility, namely slow wave or electrical control activity. This electrophysiologic activity originating from the proximal stomach consists of continuous rhythmic change in the membrane potential and will propagate to the distal antrum with a

Manuscript received February 26, 2000; revised manuscript received September 13, 2000; accepted October 2, 2000.

From the Division of Gastroenterology, Department of Medicine and Surgery, Taipei Veterans General Hospital and School of Medicine, National Yang-Ming University; and the Department of Electrical Engineering, National Taiwan University, Taipei, Taiwan.

Address for reprint requests: Dr. Full-Young Chang, Division of Gastroenterology Veterans General Hospital-Taipei No. 201, Sec 2, Shih-Pai Rd. Taipei, Taiwan 11217.

regular frequency of about 3 cpm (8, 9). Physiologically, this slow wave triggers the onset of the action potential of the stomach, in turn eliciting muscle contraction (10, 11). Hence this slow wave is an essential trigger for stomach movement including gastric emptying (12, 13). Electrogastrography (EGG) is a noninvasive method that can be used to record this rhythmic myoelectrical signal on the stomach smooth muscle by employing the electrodes positioned on the abdominal skin (14, 15).

Slow wave dysrhythmia is encountered in patients with diabetic gastroparesis, motion sickness, and pregnant women (15). The role of gastric myoelectrical dysrhythmia in the pathogenesis of NUD remains debatable since diverse results, including inotropic dysfunction (decreased postprandial power) (21), chronotrophic dysfunction (gastric dysrhythmias) (21, 22) or even a normal EGG (23), have been found. It has been estimated that 39-87% of NUD patients have chronic Helicobacter pylori (H. pylori) infection (24, 25). An interaction between gastric mucosal inflammation and H. pylori colonization is known to exist (26). Whether H. pylori colonization or its associated gastritis will have any impact on EGG parameters is controversial (23, 27). In addition, previous EGG studies on NUD patients were performed in Western countries. The environmental, socioeconomic, and cultural status are quite different between Occidentals and Orientals. For instance, it is known that Chinese have a higher rate of H. pylori infection than Caucasians (28). Hence, we are interested to know: (1) the probable EGG changes in Chinese NUD patients; and (2) the influence of H. pylori status, histological gastritis, and dyspeptic symptoms on the EGG parameters among Chinese NUD patients.

MATERIALS AND METHODS

Subjects. This study was conducted with 32 healthy controls (18 women, 14 men, age range 24–50 years) without any dyspeptic symptoms and 27 NUD patients (17 women, 10 men, age range 27–66 years). Those patients who had a history of mechanical gastrointestinal obstruction, biliary tract or liver disease, abdominal surgery, diabetes, serious systemic disorders, or using medications known to alter gastrointestinal motility, including prokinetic agents, anticholinergic agents, and calcium channel blockers, were excluded from the study. NUD was defined according to Talley et al (29). Briefly, patients had had at least moderate pain or discomfort (or both) centered in the upper abdomen as their predominant symptoms for a minimum of three days in the week before entering the study; had had

dyspepsia for at least three months and had normal endoscopic findings in the esophagus, stomach, and duodenum. The dyspeptic symptoms were assessed based on a standard protocol. The symptoms, including nausea, vomiting, anorexia, epigastric pain, epigastric fullness, and early satiety were graded according to the following scale: 0 = not present; 1 = mild, occasional, slightly influencing daily activities; 2 = moderate, often influencing daily activities; 3 = severe, very often, with strong impact on concentration and daily activities (30). The study was approved by the Institutional Review Board at Taipei Veterans General Hospital. Informed consent was obtained in all cases prior to the study.

Endoscopy, H. pylori Determination, and Histological Examination. All the NUD patients received an upper GI endoscopy to exclude gastroesophageal reflux, peptic ulcers, erosions, and gastric tumors. Four forceps-biopsied specimens were obtained from the mucosa of the antrum and body to evaluate H. pylori status by a rapid urease test (CLO-test, Delta West Limited, Canning Vale, Australia), culture, and histology. The tissue was mined and then put under 99% humidity and 10% CO2 at 37°C on a chocolate agar plate for four days. The colony was then examined under a microscope, and H. pylori colonization was diagnosed when the typical curved-shaped bacteria were found and the urease test was found to be positive.^{[13}C] Urea breath test (UBT) was also performed in all NUD patients. Briefly, breathing samples from baseline and samples 15 min after drinking 100 mg[¹³C] urea (UBIT, Otsuka Pharmaceutical Co., Osaka, Japan) were collected. The ${}^{13}CO_2$ to ${}^{12}CO_2$ isotope ratio (${\delta}^{13}CO_2$) in the collection bag was measured by an infrared spectrometer (UBiT-IR200, Otsuka Electronics Co., Osaka, Japan). H. pylori was acknowledged to be positive when the increment of $\delta^{13}CO_2$ exceeded 3.50/00(31). The H. pylori colonization in NUD was defined when at least two of four tests were positive. In the control group, the H. pylori status was only determined by an enzyme-linked immunosorbent assay kit (HEL-p TESTII; Amrad, Kew, Australia). Histological assessment for the severity of gastritis was scaled according to a modified Whitehead scoring system as follows: 0 = no polymorphonuclear (PMN) leukocytes in the antral mucosa; 1 =few PMN, plasma cells (PC), and lymphocytes (L); 2 = moderate infiltration with PMN, PC and L; and 3 = extensive invasion with PMN, PC and L) (32). The presence of chronic atrophic gastritis and intestinal metaplasia were also recorded.

Electrogastrography (EGG) System. Our EGG system included a signal acquisition device, a notebook PC (Pentium 166 MHz), the power supply, and electrodes (Figure 1). Recorded signals were initially preamplified by an instrumental amplifier to fit the high impedance of the human body. A fourth order active high-pass filter with 0.01-Hz cutoff frequency and another fourth order active low-pass filter with 0.5 Hz cutoff frequency were installed to filter out unnecessary noises such as heartbeat, respiration, and body movement artifacts. Then a high-gain amplifier was used to achieve a total gain with range of 4000–40,000. A 2.0-Hz precision clock source was designed to provide a stable 1 Hz sampling rate. An At89C52 (Atmel, San Jose, California, USA) microprocessor chip was installed and regulated by the precision clock. The analog multiplexer processed the

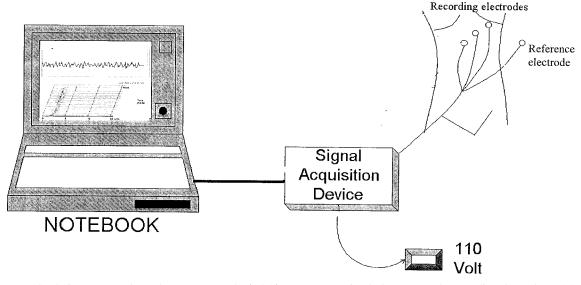


Fig 1. The design concept of our electrogastrography (EGG) system. It consisted of power supply, recording electrodes, EGG component, and a notebook PC. This EGG component was powered by 12 V direct current and recorded electrophysiological signals from studied subjects. These signals were then processed, amplified and digitized in the component and finally transmitted into a notebook PC for real-time automatic analysis and display on the monitor.

EGG signals while this microprocessor obtained the signals via a 12-bit analog/digital converter card. Finally the digitized signals were transmitted to the PC via RS-232 interface for on-line operation and further real-time analysis (Figure 2). Using the short-term Fourier transformation, the software in the PC could track the time variations of the signal frequency. The EGG power spectral density was computed from the fast Fourier transformation of the data sequence with appropriate statistical averaging, such as Hanning windowing being introduced to analyze the signals with limited data in our system. Then, both dominant frequency (DF) and power (DP) were obtained from the short-term Fourier transformation. Other myoelectrical parameters, including percent of normal slow wave frequency (2-4 cpm) and instability coefficient (IC) of slow wave as well as power ratio (PR), were also simultaneously exhibited and renewed on the PC monitor every 64 sec.

EGG Recording. Studied subjects were placed in a supine position and asked not to move, fall asleep or talk throughout the measurement. The abdominal skin was cleaned and gently scrubbed until the appearance of redness to decrease the impedance. Four silver-silver chloride electrodes filled with electrode jelly (Red Dot-2237, 3M, St. Paul, Minnesota, USA) were placed on the examined area. Three electrodes were placed along the long axis of the stomach while the reference electrode was placed on the left forearm (Figure 1). After a basal recording in fasting status for 30 min, the studied individual was asked to consume a standard test meal that included 250 ml milk and cake (347 kcal; carbohydrate 51 g; protein 13.2 g; and fat 10 g). Another 30-min postprandial recording was then undertaken. Finally, the channel with the fewest artifacts was chosen to analyze the gastric myoelectrical parameters of each studied subject

Statistical Analysis. All EGG parameters were presented as mean \pm SEM. Student's *t* test, Mann-Whitney U test, or

Pearson correlation test were used for statistical analysis where appropriate. A finding of P < 0.05 was considered to be significant.

RESULTS

During the fasting recording, there was no difference in EGG parameters between the NUD patients and controls (Table 1). However, postprandial recording did find some abnormalities in EGG frequency and power among NUD patients when compared to the controls. For instance, NUD patients had less chance of normal rhythm (2–4 cpm) after a meal (P < 0.05), and in particular had an increased chance of tachygastria (P < 0.05). This effect on EGG frequency in NUD patients was confirmed by the increased chance of postprandial IC of DF ($36 \pm 3\%$ vs $26 \pm 3\%$, P < 0.05). NUD patients also manifested less DP after the meal as compared to the controls, while the PR again showed a lower value in the NUD patients (1.42 ± 0.28 vs 2.79 ± 0.39 , P < 0.05).

Among 32 controls, 15 (46.9%) had *H. pylori* colonization as confirmed by serology. Table 2 illustrates that the *H. pylori* colonization did not influence EGG parameters in these subjects. *H. pylori* colonization was diagnosed in 18 NUD patients according to our criteria. Although the prevalence of *H. pylori* looked higher in the NUD patients, the difference did not reach a significant level (66.7% vs 46.9%, P > 0.05). Similarly, *H. pylori* infection had no impact on any of the EGG parameters in the NUD patients (Table 2).

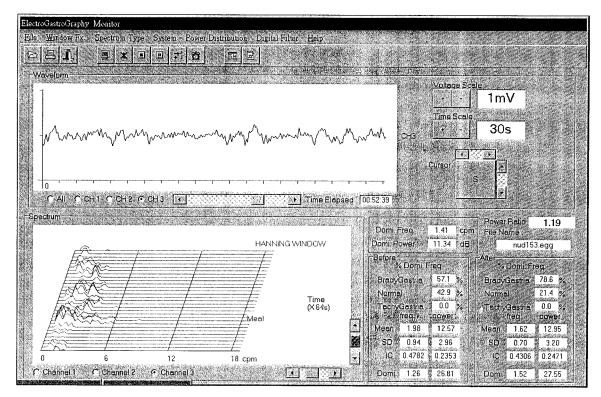


Fig 2. The electrogastrographic recording of a nonulcer dyspepsia patient. Upper panel: postprandial slow-wave signal recorded in one of three channels. Lower panel: pseudo three-dimensional graph of power spectra in the Hanning window illustrates the relationship of frequency domain and power. The order of processing was ranked from top to bottom. Right lower panel provided various slow-wave parameters, which were automatically renewed every 64 sec.

Of the NUD patients with *H. pylori* colonization, 17 (94.4%) had histological evidence of gastritis, which was located at body (N = 14, grade 1 = 8, 2 = 5, 3 = 1) or antrum (N = 17, grade 1 = 3, 2 =9, 3 = 5). The presence of gastritis grades 1–3 did not significantly affect any EGG parameters (Table 3). In addition, five (18.5%) NUD patients had atrophic gastritis, whereas three (11.1%) had intestinal metaplasia. Neither atrophic gastritis nor intestinal metaplasia had an influence on the

| EGG recordings | Controls ($N = 32$) | NUD (N = 27) | Р | |
|---------------------------------|-----------------------|------------------|--------|--|
| Preprandial | | | | |
| Dominant frequency (cpm) | 3.08 ± 0.03 | 3.00 ± 0.06 | NS | |
| Dominant power (dB) | 24.46 ± 0.62 | 25.49 ± 0.82 | NS | |
| Normogastria (%) | 66.62 ± 3.42 | 61.62 ± 3.77 | NS | |
| Bradygastria (%) | 30.24 ± 3.30 | 34.47 ± 3.62 | NS | |
| Tachygastria (%) | 3.09 ± 0.89 | 3.81 ± 1.60 | NS | |
| IC of dominant frequency | 0.40 ± 0.03 | 0.42 ± 0.03 | NS | |
| Postprandial | | | | |
| Dominant frequency (cpm) | 3.25 ± 0.04 | 3.18 ± 0.05 | NS | |
| Dominant power (dB) | 28.22 ± 0.69 | 26.12 ± 0.56 | < 0.05 | |
| Normogastria (%) | 79.08 ± 2.95 | 70.10 ± 2.97 | < 0.05 | |
| Bradygastria (%) | 20.58 ± 2.95 | 28.04 ± 2.96 | NS | |
| Tachygastria (%) | 0.22 ± 0.15 | 1.58 ± 0.72 | < 0.05 | |
| IC of dominant frequency | 0.26 ± 0.03 | 0.36 ± 0.03 | < 0.05 | |
| Increase in dominant power (dB) | 3.76 ± 0.58 | 0.62 ± 0.91 | < 0.05 | |
| Power ratio | 2.79 ± 0.39 | 1.42 ± 0.28 | < 0.05 | |

Table 1. Fasting and Postprandial Electrogastrography (EGG) Parameters in Controls and Chinese Patients with Nonulcer Dyspepsia (NUD)*

*Values are mean ± SEM; normogastria = 2-4 cpm; IC = instability coefficient; NS = not significant.

| | Controls H. pylori | | | NUD H. pylori | | | |
|---------------------------------|--------------------|--------------------|----|-------------------|-------------------|----|--|
| EGG recording | (Pos.) $(N = 15)$ | (Neg.) (N = 17) | Р | (Pos.) $(N = 18)$ | (Neg.) (N = 9) | Р | |
| Preprandial | | | | | | | |
| Dominant frequency (cpm) | 3.10 ± 0.05 | 3.07 ± 0.04 | NS | 3.03 ± 0.06 | 2.94 ± 0.13 | NS | |
| Dominant power (dB) | 24.54 ± 1.15 | 24.04 ± 0.68 | NS | 24.92 ± 0.95 | 26.65 ± 1.56 | NS | |
| Normogastria (%) | 64.59 ± 5.63 | 68.29 ± 4.32 | NS | 58.96 ± 5.13 | 66.93 ± 4.60 | NS | |
| Bradygastria (%) | 31.90 ± 5.25 | 28.95 ± 4.33 | NS | 36.71 ± 95.00 | 29.98 ± 4.25 | NS | |
| Tachygastria (%) | 3.51 ± 1.03 | 2.75 ± 1.40 | NS | 5.32 ± 2.30 | 2.09 ± 0.89 | NS | |
| IC of dominant frequency | 0.43 ± 0.07 | 0.36 ± 0.04 | NS | 0.49 ± 0.01 | 0.37 ± 0.01 | NS | |
| Postprandial | | | | | | | |
| Dominant frequency (cpm) | 3.29 ± 0.06 | 3.23 ± 0.06 | NS | 3.22 ± 0.01 | 3.08 ± 0.01 | NS | |
| Dominant power (dB) | 28.54 ± 1.05 | 27.97 ± 0.94 | NS | 26.42 ± 0.50 | 25.52 ± 1.39 | NS | |
| Normogastria (%) | 77.96 ± 5.27 | 79.96 ± 3.40 | NS | 70.94 ± 3.42 | 68.43 ± 5.98 | NS | |
| Bradygastria (%) | 21.54 ± 5.24 | 18.95 ± 4.32 | NS | 26.68 ± 3.42 | 30.77 ± 5.84 | NS | |
| Tachygastria (%) | 0.21 ± 0.26 | 0.23 ± 0.20 | NS | 2.37 ± 1.05 | 1.02 ± 0.15 | NS | |
| IC of dominant frequency | 0.28 ± 0.04 | 0.25 ± 0.02 | NS | 037 ± 0.03 | 0.35 ± 0.06 | NS | |
| Increase in dominant power (dB) | 4.00 ± 0.96 | 3.56 ± 0.73 | NS | 1.50 ± 1.00 | -1.12 ± 1.79 | NS | |
| Power ratio | 3.50 ± 0.79 | 2.24 ± 0.28 | NS | 1.72 ± 1.58 | 0.74 ± 0.51 | NS | |

 TABLE 2. IMPACT OF H. PYLORI COLONIZATION ON ELECTROGASTROGRAPHY (EGG) PARAMETERS AMONG CONTROLS AND PATIENTS WITH NONULCER DYSPEPSIA (NUD)*

*Values are mean \pm SEM; normogastria = 2–4 cpm; IC = instability coefficient; NS = not significant.

EGG parameters in the NUD patients (data not shown).

early satiety, and total symptom score in NUD patients were 0.32 ± 0.07 , 0.20 ± 0.08 , 0.45 ± 0.10 ,

 1.05 ± 0.14 , 1.98 ± 0.03 , 0.20 ± 0.04 , and 4.12 ± 0.31 ,

respectively, while the total symptom scores divided

according to H. pylori positive or negative status were

 4.22 ± 0.60 vs 3.97 ± 0.36 , respectively (P > 0.05).

Neither the total symptom score nor the individual

In general, the symptomatic scores for nausea, vomiting, anorexia, epigastric pain, epigastric fullness,

symptoms showed a significant correlation against the EGG parameters (Table 4).

DISCUSSION

Our study results mainly indicated that Chinese patients with NUD obviously had abnormal postprandial EGG parameters, such as a lower chance of normal rhythm (normogastragia), obvious irregularity of the slow wave rhythm, and a poor power response after a meal. However, we are still unsure whether

Table 3. EGG Parameters in Non ulcer Dyspepsia Patients With or Without Histological Gastritis*

| | Gastritis | | | |
|---------------------------------|------------------|--------------------|----|--|
| EGG recording | With $(N = 17)$ | Without $(N = 10)$ | Р | |
| Preprandial | | | | |
| Dominant frequency (cpm) | 3.02 ± 0.06 | 2.96 ± 0.12 | NS | |
| Dominant power (dB) | 25.01 ± 1.01 | $26,32 \pm 1.43$ | NS | |
| Normogastria (%) | 58.64 ± 5.44 | 66.67 ± 4.12 | NS | |
| Bradygastria $(\%)$ | 36.78 ± 5.30 | 30.55 ± 3.84 | NS | |
| Tachygastria (%) | 5.63 ± 2.42 | 0.71 ± 0.71 | NS | |
| IC of dominant frequency | 0.49 ± 0.08 | 0.38 ± 0.06 | NS | |
| Postprandial | | | | |
| Dominant frequency (cpm) | 3.23 ± 0.08 | 3.08 ± 0.04 | NS | |
| Dominant power (dB) | 26.40 ± 0.54 | 25.64 ± 1.25 | NS | |
| Normogastria (%) | 70.28 ± 3.56 | 69.80 ± 5.52 | NS | |
| Bradygastria $(\%)$ | 27.20 ± 3.59 | 29.48 ± 5.38 | NS | |
| Tachygastria (%) | 2.61 ± 1.11 | 0.72 ± 0.52 | NS | |
| IC of dominant frequency | 0.37 ± 0.03 | 0.35 ± 0.03 | NS | |
| Increase in dominant power (dB) | 1.39 ± 1.06 | -0.69 ± 1.66 | NS | |
| Power ratio | 1.72 ± 0.38 | 0.74 ± 0.18 | NS | |

*Values are mean \pm SEM; normogastria = 2–4 cpm; IC = instability coefficient; NS = not significant; The gastritis was classified according to Whitehead scoring system grade 1 to 3.

EGG CHARACTERISTICS IN NUD PATIENTS

| | Nausea | Vomiting | Anorexia | Epigastralgia | Fullness | Early satiety | TSS |
|---------------------------------|--------|----------|----------|---------------|----------|---------------|-------|
| Preprandial | | | | | | | |
| Dominant frequency (cpm) | 0.20 | 0.24 | 0.18 | 0.10 | -0.21 | 0.19 | 0.39 |
| Dominant power (dB) | -0.10 | 0.15 | -0.14 | 0.2 | 0.10 | 0.20 | -0.18 |
| Normogastria (%) | 0.15 | -0.18 | 0.21 | 0.27 | 0.16 | -0.18 | 0.23 |
| Bradygastria (%) | 0.13 | 0.23 | -0.13 | 0.15 | 0.15 | 0.12 | 0.22 |
| Tachygastria (%) | 0.18 | 0.28 | 0.16 | -0.18 | -0.17 | 0.18 | 0.15 |
| IC of the dominant frequency | 0.22 | 0.26 | 0.25 | 0.1 | 0.22 | 0.20 | 0.13 |
| Postprandial | | | | | | | |
| Dominant frequency (cpm) | 0.32 | 0.12 | -0.22 | 0.25 | 0.10 | 0.22 | -0.21 |
| Dominant power (dB) | -0.22 | 0.17 | 0.18 | -0.10 | -0.20 | 0.18 | 0.32 |
| Normogastria (%) | 0.12 | 0.18 | -0.20 | -0.12 | 0.24 | 0.16 | 0.20 |
| Bradygastria $(\%)$ | 0.23 | -0.13 | 0.21 | 0.20 | -0.17 | 0.17 | 0.12 |
| Tachygastria (%) | 0.15 | 0.17 | 0.09 | 0.15 | 0.15 | 0.26 | 0.09 |
| IC of dominant frequency | 0.10 | 0.20 | 0.10 | 0.10 | 0.13 | 0.13 | 0.06 |
| Increase in dominant power (dB) | 0.11 | 0.15 | 0.13 | 0.11 | 0.2 | 0.25 | 0.30 |
| Power ratio | 0.15 | 0.16 | 0.18 | 0.15 | 0.14 | 0.2 | 0.32 |

TABLE 4. CORRELATION COEFFICIENT OF INDIVIDUAL SYMPTOMS AND TOTAL SYMPTOM SCORE AGAINST ELECTROGASTROGRAPHY (EGG) PARAMETERS

*All items are statistically insignificant; normogastria = 2-4 cpm; IC = instability coefficient; TSS = total symptoms score.

these characteristic postprandial EGG parameters will be useful in differentiating Chinese NUD patients from healthy controls.

Until now, few studies have investigated gastric myoelectrical activity in adult NUD patients, and the results obtained are subject to debate. For instance, normal or little disturbed EGG parameters have been found in NUD patients in some studies (21, 23). In contrast, Pfaffenbach B *et al* (22) reported an increase in fasting tachygastria and a lower chance of normogastragia in NUD patients. Interestingly, Lin *et al* (20) pointed out that the changes in some EGG parameters in American NUD patients could only be identified after the test meal. Likewise, the power increase or PR after the test meal was lower in American NUD patients. This result was very comparable with ours. In addition, our study further found a lower postprandial DP.

Since stomach motility is determined primarily by the myoelectrical activity, abnormal slow waves may lead to stomach motor disturbance or delayed gastric emptying (33). Although the exact pathogenesis of NUD remains unclear, motility disturbance in the stomach has been identified as the leading cause (10). Although we did not measure gastric emptying in NUD patients, our observations of impaired postprandial EGG parameters, either in frequency or power, suggest that disturbed stomach motility likely exists in NUD patients.

The Chinese may have an apparent dysrhythmic myoelectrical activity in the stomach. Such an impression is evidenced by the lower chance of normogastria (75% vs 80-90%) and obvious tachygastria (20% vs 6%) in the Chinese population when compared to

Occidentals (28, 34, 35). Our study also confirmed such an apparent EGG dysrhythmia based on the similar percentage of normogastria recorded in the controls, which is comparable to Lin's results (28). However, bradygastria rather than tachygastria was the main component of dysrhythmia in our control subjects. It has been suggested that the high prevalence rate of *H. pylori* in the Chinese population is responsible for this EGG dysrhythmia (28), whereas, we found similar EGG parameters in the studied subject irrespective of *H. pylori* infection. We suggest that *H. pylori* infection is not the main factor responsible for the apparent dysrhythmia in the Chinese, either in controls or NUD patients.

It has been indicated that a positive rapid urease test alone is not sufficient to diagnose *H. pylori* infection (36). Therefore, *H. pylori* colonization in our NUD patients was defined as present when two or more tests, ie, rapid urease test, culture, histology, and [¹³C] UBT, were positive since all these tests except culture, have a sensitivity or specificity of over 95% (37–39). In fact, the 17 NUD patients had a positive reaction to all four *H. pylori* tests.

The prevalence of *H. pylori* colonization in NUD patients usually ranges from 39% to 87% according to the defined criteria (24, 25). It remains highly debatable whether *H. pylori* colonization is the only factor responsible for NUD symptoms. Some studies have indicated that NUD patients with chronic *H. pylori* infection have disturbed gastrointestinal motor function. Likewise, shortening of phase I of the migrating motor complex (MMC), and absence of phase III MMC have been recorded in NUD patients (40–42). Interestingly, only two studies have evaluated the

relationship of EGG parameters in NUD patients, but they reached conflicting results. Thor et al (27) reported a prolonged period of tachygastria and absence of postprandial power response in H. pyloricolonized NUD patients. They suggested that these abnormalities might be in reaction to mucosal inflammation rather than H. pylori colonization. In contrast, Pfaffenbach et al (22) recorded that there were no difference in EGG parameters between H. pylori (positive) and (negative) NUD patients. Our study indicated that the abnormal EGG parameters in NUD patients were not directly related to H. pylori infection and its associated gastritis. Our study results was very similar to the later because the observations of abnormal postprandial EGG parameters in NUD patients were not directly related to H. pylori infection and its associated gastritis.

Tachygastria and dysrhythmias are common in patients with unexplained nausea, vomiting, and bloating (17, 43); pregnant women experiencing nausea (44); diabetic gastroparesis (45); and motion sickness (46). However, our study, as well as that of Lin et al (20) did not find a close relationship between abnormal EGG parameters and main dyspeptic symptoms. Recently, augmented visceral afferent sensation, apart from dysmotility, has drawn attention as a possible mechanism to produce dyspeptic symptoms in NUD patients (47, 48). It is probable that abnormal visceral afferent sensation has little effect on EGG parameters.

In conclusion, Chinese NUD patients may have abnormal postprandial EGG parameters although these abnormalities are not directly related to *H. pylori* infection and its related gastritis.

ACKNOWLEDGMENTS

This study was sponsored by a grant from Taipei Veterans General Hospital (grant 89-214).

REFERENCES

- Jebbink HJA, Smout AJPM, van Berge-Henegouwen GP: Pathophysicology and treatment of functional dyspepsia. Scand J Gastroenterol 28(suppl 200):8–14, 1993
- Babara L, Camilleri M, Corinaldesi R, Crean GP, Heading RC, Johnson AG, Malagelada J-R, Stanghellini V, Wienbeck M: The definition and investigation of dyspepsia. Dig Dis Sci 34:1272–1276, 1989
- Hausken T, Berstad A: Effect of glyceryl trinitrate on antral motility and symptoms in patients with functional dyspepsia. Scand J Gastroenterol 29:23–28, 1994
- Wengrower D, Saltzmann S, Karmeli F, Goldin E: Idiopathic gastroparesis in patients with unexplained nausea and vomiting. Dig Dis Sci 36:1255–1258, 1991

- Jian R, Ducrot F, Ruskone A: Symptomatic radionuclide and therapeutic assessment of chronic idiopathic dyspepsia. A double blind placebo controlled evaluation of cisapride. Dig Dis Sci 34:657–664, 1989
- Kerlin P: Postprandial antral hypomotility in patients with idiopathic nausea and vomiting. Gut 30:54–59, 1989
- Stanghellini V, Ghidni C, Ricci Maccarini M, Paparo GF, Corinaldesi R, Barbara L: Fasting and postprandial gastrointestinal motility in ulcer and non-ulcer dyspepsia. Gut 33:184– 190, 1992
- Geldof H, van der Schee EJ, Grashuis JL: Electrogastrographic characteristics of interdigestive migrating complex in humans. Am J Physiol 250:G165–G171, 1986
- You CH, Chey WY: Study of electromechanical activity of the stomach in humans and in dogs with particular attention to tachygastria. Gastroenterology 86:1460–1468, 1984
- Moore JG: Gastroparesis: pathogenesis and evaluation. In Evolving Concepts in Gastrointestinal Motility. MC Champion, WC Orr (eds). Oxford, Blackwell Sciences, 1996, pp 87–108
- Camilleri M, Prather CM: Gastric motor physiology and motor disorders. *In* Sleisenger & Fordtans's Gastrointestinal & Liver Disease. M Feldman, BF Scharschmidt, MH Sleisenger (eds). Philadelphia, WB Saunders, 1998, pp 572–586
- 12. Kelly KA. Pacing the gut. Gastroenterology 103:1967–1969, 1992
- Chen J, McCallum RW: Gastric slow wave abnormalites in patients with gastroparesis. Am Gastroenterol 87:477–482, 1992
- Geldof H, van der Schee J: Electrogastrography, clinical applications. Scand J Gastroenterol 24(suppl):S75–S82, 1989
- Chen JC, McCallum RW: Clinical applications of electrogastrography. Am J Gastroenterol 88:1324–1326, 1993
- Stern RM, Koch KL, Stewart WR, Lindblad IM: Spectral analysis of tachygastria recorded during motion sickness. Gastroenterology 92:92–97, 1987
- You CH, Lee KY, Chey WY, Menguy R: Electrogastrophic study of patients with unexplained nausea, bloating and vomiting. Gastroenterology 79:311–314, 1980
- Chen J, Schirmer BD, McCallum RW: Serosal and cutaneous recordings of gastric myoelectrical activity in patients with gastroparesis. Am J Physiol 266:G90–G98, 1994
- 19. Chen J, McCallum RW. Electrogastrography: Principles and applications. New York, Raven Press, 1994
- Lin X, Levanon D, Chen J: Impaired postprandial gastric slow waves in patients with functional dyspepsia. Dig Dis Sci 43:1678–1684, 1998
- Jebbink HJA, van Berge-Henegouwen GP, Bruijs PPM, Akkermans LMA, Smout AJPM: Gastric myoelectrical activity and gastrointestinal motility in patients with functional dyspepsia. Eur J Clin Invest 25:429–437, 1995
- Pfaffenbach B, Adamek RJ, Bartholomaus C, Wegener M: Gastric dysrhythmias and delayed gastric emptying in patients with functional dyspepsia. Dig Dis Sci 42:2094–2099, 1997
- Geldof H, van Der Schee EJ, Blankenstein MV, Grashuis JL: Electrogastrographic study of gastric myoelectrical activity in patients with unexplained nausea and vomiting. Gut 27:799– 808, 1986
- Rokkas T, Pursey C, Uzoechina E, Dorrington L, Simmons NA, Filipe MI, Sladen GE: Non-ulcer dyspepsia and shortterm De-Nol therapy: A placebo-controlled trial with particu-

Digestive Diseases and Sciences, Vol. 46, No. 2 (February 2001)

lar reference to the role of *Campylobacter pylori*. Gut 29:1386–1391, 1988

- Loffield RJ, Stoberingh E, Flendrig JA, Arends JW: Presence of *Helicobacter pylori* in patients with non-ulcer dyspepsia revealing normal antral histological characteristics. Digestion 47:29–34, 1990
- Yoshida N, Granger DN, Evans DJ Jr, Evans DG, Graham DY, Anderson DC, Wolf RE, Kvietys PR. Mechanisms involved in *Helicobacter prlori* induced inflammation. Gastroenterology 105:1431–1440, 1993
- Thor P, Lorens K, Tabor S, Herman R, Konturek JW, Konturek SJ. Dysfunction in gastric myoelectric and motor activity in *Helicobacter pylori* positive gastritis patients with non-ulcer dyspepsia. J Physiol Pharmacol 47:469–476, 1996
- Lin X, Liang J, Ren J, Mu F, Zhang M, Chen JDZ: Electrical stimulation of acupuncture points enhances gastric myoelectrical activity in humans. Am J Gastroenterol 92:1527–1530, 1997
- Talley NJ, Colin-Jones D, Koch KL, Koch M, Nyren O, Strangellini V. Functional dyspepsia: A classification with guidelines for diagnosis and management. Gastroenterol Int 4:145–160, 1991
- Lu CL, Chang FY, Chen TS, Chen CY, Kan LC, Lee SD: Helicobacter pylori colonization is not a major pathogenic factor for patients with non-ulcer dyspepsia. J Gastroenterol Hepatol 13:500–504, 1998
- Miwa H, Murai T, Ohkura R, Nagahara A, Watanabe H, Terai T, Watanabe S, Sato N: Usefulness of the [¹³C]urea breath test for detection of *Helicobacter pylori* infection in fasting patients. J Gastroenterol Hepatol 13:1039–1043, 1998
- Whitehead R: Mucosal biopsies of the gastrointestinal tract. *In* Major Problems in Pathology. JL Benning (ed). Philadelphia, WB Saunders, 1979, p 15
- Chen J, Lin Z, Pan J, McCallum RW: Abnormal gastric myoelectrical activity and delayed gastric emptying in patients with symptoms suggestive of gastroparesis. Dig Dis Sci 41:1538– 1545, 1996
- Parkman HP, Harris AD, Miller MA, Fisher RS: Influence of age, gender, and menstrual cycle on the normal electrogastrogram. Am J Gastroenterol 91:127–133, 1996
- Pfaffenbach B, Adamek RJ, Kuhn K, Wegener M: Electrogastrography in healthy subjects: Evaluation of normal values, influence of age and gender. Dig Dis Sci 40:1445–1460, 1995
- 36. Chan FKL, Sung JJY, Chung SCS, To KF, Yung MY, Leung VKS, Lee YT, Chan CSY, Li EKM, Woo J: Randomised trial of eradication of *Helicobacter pylori* before non-steroidal antiinflammatory drug therapy to prevent peptic ulcers. Lancet 350:975–979, 1997

- Yousfi MM, El-zimaity MT, Cole RA, Genta RM, Graham DY: Detection of *Helicobacter pylori* by rapid urease test: Is biopsy size a critial variable? Gastrointest Endosc 43:222–224, 1996
- Genta RM, Robason GO, Graham GY: Simultaneous visulization of *Helicobacter pylori* and gastric morphology: A new stain. Hum Pathol 25:221–261, 1994
- Goddard AF, Logan RPH. Review article: Urea breath tests for detecting *Helicobacter pylori*. Aliment Pharmacol Ther 11:641–649, 1997
- Qvist N, Rasmussen L, Axelsson CK: *Helicobacter pylori*associated gastritis and dyspepsia. The influence on migrating motor complexes. Scand J Gastroenterol 29:133–137, 1994
- 41. Trestoni PA, Bagnolo F, Bologna P, Colombo E, Bonassi U, Lella F, Buizza M: Higher prevalence of *Helicobacter pylori* infection in dyspepsic patients who do not have gastric phase III of migrating motor complex. Scand J Gastroenterol 31:1063–1068, 1996
- Pieramico O, Ditschuneit H, Malfertheiner P: Gastrointestinal motility in patients with non-ulcer dyspepsia: A role for *Helicobacter* infection? Am J Gastroenterol 88:364–368, 1993
- Parkman HP, Miller MA, Trate D, knight LC, Urbain JL, Maurer AH, Fisher RS: Electrogastrography and gastric emptying scintigraphy are complementary for assessment of dyspepsia. J Clin Gastroenterol 24:214–219, 1997
- Riezzo G, Pezzolla F, Darconza G, Giorgio I: Gastric myoelectrical activity in the first trimester of pregnancy: A cutaneous electrogastrographic study. Am J Gastroenterol 87:702–707, 1992
- 45. Pfister CJ, Hamilton JW, Nagel N, Bass P, Webster JG, Tompkins WJ. Use of spectral analysis in the detection of frequency differences in the electrogastrograms of normal and diabetic subjects. IEEE Trans Biomed Eng 35:935–942, 1988
- Stern RM, Koch KL, Stewart WE, Lindblad IM: Spectral analysis of tacyhygastria recorded during motion sickness. Gastroenterology 92:92–97, 1980
- Mearin F, Cucala M, Azpiroz F, Malagelada J-R: The origin of symptoms on the brain–gut axis in functional dyspepsia. Gastroenterology 101:999–1006, 1991
- Lemann M, Dederding JP, Flourie B, Franchisseur C, Rambaud JC, Jian R: Abnormal perception of visceral pain in response to gastric distensition in chronic idiopathic dyspepsia—the irritable stomach syndrome. Dig Dis Sci 36:1249– 1254, 1991