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Characterization of Gastric Myoelectrical Rhythms in Patients with Systemic Sclerosis Using Multichannel Surface Electrogastrography

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The aim of this study was to characterize multichannel surface electrogastrography (EGG) recordings in patients with systemic sclerosis (scleroderma, SSc) compared to normal controls. Ten SSc patients and 13 healthy age-matched controls were enrolled in this study. Gastric myoelectrical activity was recorded using a four-channel electrogastrograph with abdominal surface electrodes. The EGG was recorded in the morning for 1 hr in the fasting state, and 1 hr after a standardized (500 kcal) test meal. It was found that: (1) The regularity of the gastric slow wave was significantly lower in the SSc patients when compared with the normal controls in both fasting and fed states. (2) There was a significantly higher incidence of bradygastria in the SSc patients. (3) The SSc patients showed a significantly lower percentage of slow wave coupling among the four-channel EGGs than the controls. (4) In comparison with the controls, the patients showed an impaired spatial distribution of gastric slow wave power in both fasting and fed states and an impaired spatial distribution of slow wave frequency in the fasting state. It was concluded that SSc patients have an abnormal gastric slow wave as shown in the multichannel EGG as a decreased percentage of normal slow waves and impaired spatial coordination of gastric slow waves. The multichannel EGG may serve as a simple, noninvasive, and cost-effective method to assess gastric motility disorders and their relevance in patients with SSc.

KEY WORDS: gastric motility; scleroderma; gastric slow waves; electrogastrography.

Nearly 90% of patients with systemic sclerosis (scleroderma, SSc) have at least one upper or lower gastrointestinal symptom (1–3). Most are related to esophageal dysmotility and gastroesophageal reflux disease (4, 5). Delayed gastric emptying may contribute to esophageal symptoms. Although infrequently involved as the major gastrointestinal pathology in SSc, gastric symptoms of nausea, vomiting, abdominal bloating, and early satiety may contribute to significant weight loss and morbidity.

The stomach, like the heart, has an intrinsic pacemaker that generates gastric myoelectrical activity that is composed of slow waves and spikes (6–8). The gastric slow wave originates in the corpus and propagates distally, with an increase in wave amplitude to

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assure normal contractility and mobility. It determines the maximum frequency and propagation of gastric contractions. Abnormalities in the gastric slow wave include tachygastria (slow wave frequency higher than 4 cycles/min, cpm), bradygastria (slow wave frequency lower than 2 cpm), and arrhythmia (no dominant rhythmicity) and have been reported in patients with gastric motility disorders and/or symptoms of gastroparesis (9, 10). Impaired gastric myoelectrical activity has also been reported in patients with SSc (11–13).

Gastric myoelectrical activity regulates gastric motility. It determines the maximal frequency, propagation direction, and coordination of gastric contractions. It is known that coordinated gastric contractions (peristalsis) are necessary for emptying ingested solid food. Accordingly, not only the rhythmicity but also the spatial coordination of gastric slow waves is crucial in maintaining normal gastric motility. *In vitro* muscle strip studies have revealed a decreased frequency (about 3 cpm in the corpus and about 1.5 cpm in the distal antrum) but an increased amplitude of the gastric slow wave from the corpus to the distal antrum. *In vivo* studies using internally implanted serosal electrodes, however, have indicated that the gastric slow wave in the intact and normal stomach originates in the corpus and propagates distally with a single frequency of about 3 cpm. This is because the muscle in the proximal stomach generates a slow wave of 3 cpm and paces the rest of the stomach to this same frequency. The amplitude of the slow wave in the stomach increases distally, similar to the *in vitro* studies (14).

Gastric myoelectrical activity can be recorded using a noninvasive method called electrogastrography (15). In this method, gastric myoelectrical activity is measured using abdominal surface electrodes. The recorded gastric myoelectrical activity is called the electrogastrogram (EGG). Numerous studies have shown that the EGG is a reliable measurement of gastric slow waves (8, 14). When appropriately recorded, the dominant frequency of the EGG accurately reflects the frequency of the gastric slow wave and the relative amplitude of the EGG dominant power is shown to be associated with gastric contractility (8, 15, 16). Most EGG studies have been performed using a single-channel EGG device from which information on the spatial distribution of gastric slow waves is not available. Recently, a multichannel EGG has been proposed (17) and its application in the identification of spatial abnormalities of gastric slow waves in patients with functional dyspepsia has been reported (18).

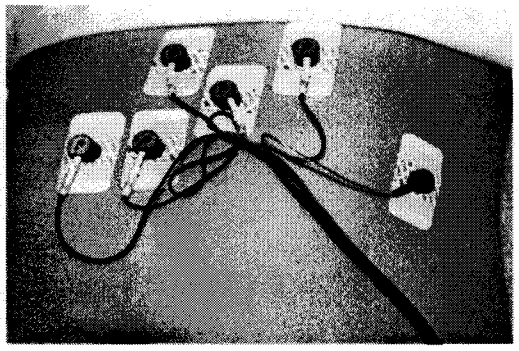

Fig 1. Cutaneous placement of multiple electrodes.

The aim of this study was to comprehensively characterize gastric slow waves in patients with SSc using the newly proposed multichannel EGG.

MATERIALS AND METHODS

Human Subjects

The research protocol was approved by the UTMB Institutional Review Board, and written informed consent was obtained from all subjects before study entry. SSc patients (8 women, 2 men) were recruited for this study. All SSc patients satisfied the ACR criteria for scleroderma (19). Four patients had diffuse and six had limited cutaneous scleroderma. SSc patients were excluded if they were: (1) unable to give informed consent; (2) currently taking prokinetic, anticholinergic, or dopaminergic agents that could potentially modify gastric motility; (3) unable to recline with a head elevation of 30 degrees for 1 hr; or (4) had a history of abdominal surgery. Thirteen healthy, age-matched, asymptomatic volunteers were recruited as normal controls. Age and gender do not affect any of the EGG parameters in normal subjects (20). The test subjects completed a questionnaire to identify regularly experienced gastrointestinal symptoms.

Multichannel Surface Electrogastrogram

Gastric myoelectrical activity of the subjects was measured using surface electrogastrography with a specially designed multichannel device (Medtronic-Synectics, Shoreview, Minnesota, USA). The electrogastrograph consisted of four identical amplifiers with cutoff frequency ranges of 1.0 to 12.0 cpm. A 12-bit 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 abdominal surface was shaved, if hairy, and cleaned with sandy skin preparation paste to reduce electrical impedance before electrode attachment. The patient was placed in a reclining position with a head and chest elevation of 30 degrees. Six silver chloride EGG electrodes were secured to the abdominal skin (Figure 1). Four active surface electrodes (E1–E4) were positioned over the stom-

ach, reflecting approximately the corpus (channel 1), proximal antrum (channel 2), distal antrum (channel 3), and pylorus region (channel 4). One reference electrode and a ground electrode were also placed. Placement of the electrodes has been standardized to assure accurate readings. Electrode 3 was placed first, 2 cm above the middle point between the xiphoid process and the umbilicus; electrode 4 was placed 4 cm horizontally to the right of electrode 3. Electrodes 2 and 1 were placed 45 degrees superior and to the left of electrode 3, respectively, with a 4- to 6-cm interval, depending on subject size. The common reference electrode was placed at the cross point of two lines, one horizontal, connecting electrode 1, and one vertical, connecting electrode 3. The ground electrode was placed on the left costal margin horizontal to electrode 3. Connection of the four active electrodes to the common reference electrode generated the four-channel EGG signals (17).

Experimental Protocol

The patients were admitted to the UTMB General Clinical Research Center (GCRC). Surface EGG was initiated after at least 6 hr of fasting, with discontinuation of drugs that are known to alter the gastric myoelectrical activity for at least 48 hr before testing. The subjects were placed in a comfortable reclining position with the head and chest elevated to 30 degrees, in a quiet room, and asked to remain awake and as still as possible during the whole recording period to avoid motion artifacts. The fasting state was recorded for 60 min. The patient was allowed to sit for 15 min to ingest a standardized test meal of 500 kcal (30% fat, 30% protein, 40% carbohydrate), with 250 ml of a noncaffeinated, noncarbonated beverage. The test meal was immediately followed by a 1-hr postprandial recording in the preprandial reclining position. This standardized protocol for conducting four-channel EGG has been previously described (17).

Data Analysis

Before the computerized analysis of the four-channel EGG, segments of the recording with motion artifact were deleted. The motion artifact was featured with abnormally high amplitude simultaneously in all four-channels. Previously validated quantitative analysis software was used to derive the following parameters (18):

Percentage of Slow Wave Coupling in Four-Channel EGG. Cross-spectral analysis was developed to compute the percentage of slow wave coupling. The percentage of slow wave coupling was defined as the percent of time during which the slow wave was determined to be coupled. The computation was carried out on a minute-by-minute basis. Each channel of the EGG recording was divided into blocks of 1 min without overlapping. The power spectrum of each 1-min EGG was calculated and the slow waves in two channels were defined as coupled if the difference in their dominant frequencies was <0.5 cpm. The percentage of coupling between every possible pair among the four channels was computed, and the values were then averaged.

EGG Dominant Frequency/Power. The frequency at which the EGG power spectrum had a peak power in the range of 0.5–9.0 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 two parameters were calculated using the smooth power spectral analysis method (21).

Percentage of Normal Gastric Slow Waves. The percentage of normal gastric slow waves was defined as the percentage of time during which regular 2–4 cpm slow waves were present over the entire analyzed period. It was computed using the adaptive spectral analysis method (21). In this method, each EGG recording was divided into blocks of 1 min without overlapping. The power spectrum of each 1-min EGG was calculated and examined to see if the peak power was within the range of 2–4 cpm. The 1-min EGG was called normal if the peak power was within the 2–4 cpm range. Otherwise it was defined as dysrhythmia.

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 1-min EGG was called tachygastria if the peak power was in the range of 4–9 cpm, bradygastria if in the range of 0.5–4 cpm, and arrhythmia if there was no dominant peak.

Statistical Analysis

All data are presented as means ± standard error (SE). Analysis of variance (ANOVA) was used to identify any spatial difference among the four-channel EGGs. Paired Student's t test was applied to investigate the difference between the patients and the controls. $P < 0.05$ was considered significant.

RESULTS

Gastrointestinal Symptoms in SSc Patients. Nine patients had the SSc diagnosis for less than five years. The age range was 36–63 years and eight of the SSc patients were female. All SSc patients reported experiencing at least one gastrointestinal symptom on their questionnaire, including retrosternal or epigastric pain (100%), nausea/vomiting (20%), early satiety (30%), bloating or postprandial epigastric fullness (30%). The normal controls (6 women, 7 men) had no history of gastrointestinal diseases or abdominal surgery and were free of gastrointestinal symptoms. None of the SSc patients or normal controls had lower gastrointestinal complaints of abdominal pain, diarrhea, or constipation.

Disorganized Gastric Slow Waves in SSc Patients. The EGG tracings of the SSc patients were grossly abnormal compared to the normal controls. Figure 2 demonstrates the EGG recordings obtained from channels 1–4 in one normal subject (a) and one representative SSc patient (b). The predominant rhythm in the normal control is the regular slow wave with a frequency of 3 cpm. The four-channel EGG recordings from the normal control show consistent amplitude and periodicity of the gastric rhythms,

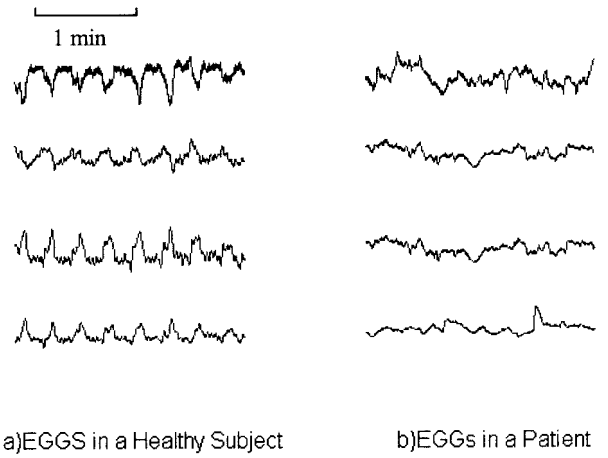

Fig 2. Typical four-channel EGG tracings in a control (left panel) and a patient with SSc (right panel).

whereas the recordings from the SSc patient demonstrate a stark contrast, with bradygastria (<2 cpm), irregular rhythms of diminished amplitude, and no interchannel periodicity. The propagation of the gastric slow wave in the four-channel EGG was less obvious in the patients than in the controls. No slow wave is generated or appropriately propagated to the distal regions of the stomach by the SSc patient, as shown in Figure 2b. The gastric rhythms from each region in the SSc patient seem to be generated independently from one another and, therefore, are less likely to generate an effective, coordinated propulsive wave.

Decreased Percentage of Normal Slow Waves in SSc Patients. Figure 3 demonstrates the mean percentage of slow waves from channels 1–4 in the normal controls and the SSc patients in the fasting and fed states. The mean percentages of slow waves were significantly lower in the SSc patients than the controls in all channels in both fasting and fed states. In the fasting state (upper panel), the mean percentage of regular 2–4 cpm slow waves in the patients among the four channels was 44.0 ± 5.7%, which is significantly lower than that in the controls (95.6 ± 1.7%, $P < 0.01$). In the patients, none of the channels showed a percentage of regular 2–4 cpm slow waves higher than 55%, whereas in the controls the percentage of regular 2–4 cpm slow waves was higher than 90% in every single channel. The difference between the patients and the controls was statistically significant in every channel. In addition, there was a spatial difference in the percentage of regular 2–4 cpm slow waves in the patients (channels 1–4: 53.4 ± 6.4%, 35.5 ± 5.6%, 33.1 ± 5.3%, 54.1 ± 5.5%,

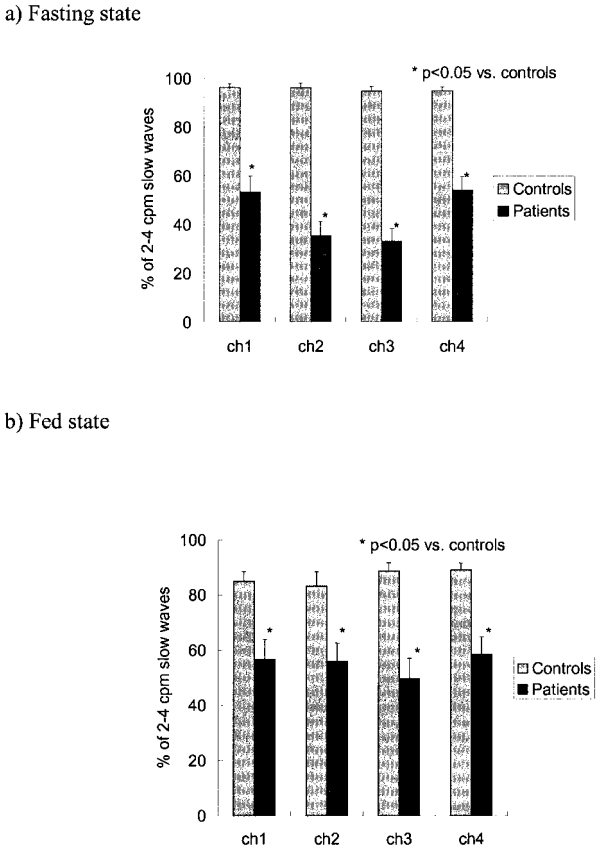

Fig 3. Percentage of regular 2–4 cpm slow waves in controls and patients with SSc in the fasting (upper panel) and fed (lower panel) states.

$P < 0.01$, ANOVA). The most impaired regions in the patients were from channels 2 and 3, approximately reflecting the proximal and distal antrum of the stomach, respectively.

Similar differences in the percentage of regular 2–4 cpm slow waves between the patients and controls were also noted in the fed state (Figure 3, lower panel). The mean percentage of the regular 2–4 slow waves among the four channels was 55.3 ± 6.9% in the patients and 86.6 ± 3.6% in the controls ($P < 0.01$). In the SSc patients, an increase in the mean percentages of regular 2–4 cpm slow waves was demonstrated in the fed state in all channels. The increase was statistically significant in channel 2 ($P < 0.02$) and channel 3 ($P < 0.04$). A spatial difference in the percentage of regular 2–4 cpm slow waves was also present in the fed state (channels 1–4: 56.7 ± 7.2%, 55.9 ± 6.7, 49.8 ± 7.3, 58.6 ± 6.3%, respectively, $P < 0.05$, ANOVA) and the value from channel 3 was the lowest ($P < 0.03$, vs channel 1 or 4).

Excessive Bradygastria in SSc Patients. The de-

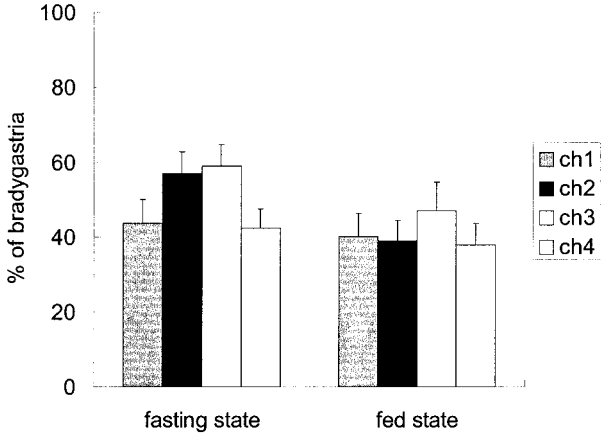

Fig 4. Percentage of bradygastria in patients with SSc.

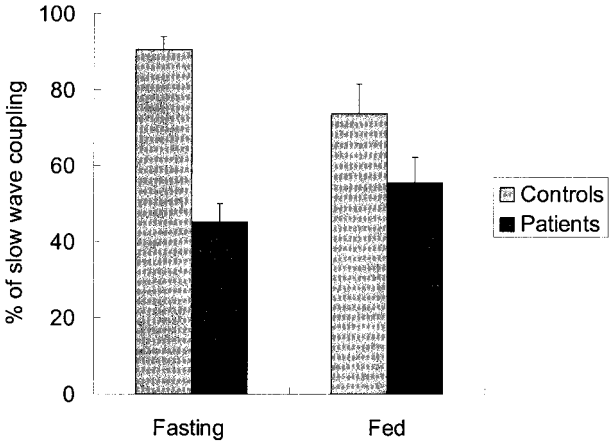

Fig 5. Percentage of slow wave coupling in controls and patients with SSc.

crease in the mean percentages of slow waves demonstrated in the SSc patients was reflected in the increase in the mean percentages of bradygastria seen in all four channels (Figure 4). The mean percentages of bradygastria demonstrated in the fasting state of SSc patients in channels 1–4 were 43.8 ± 6.3%, 56.9 ± 5.9%, 59.0 ± 5.8%, and 42.4 ± 5.2%, respectively (Figure 4) and the spatial difference in this value was statistically significant between channels 2/3 compared to channels 1/4 ($P < 0.002$). In the fed state, the mean percentages of bradygastria were slightly less for all channels: 40.2 ± 6.2%, 39.1 ± 5.5% ($P = 0.06$, vs fasting), 47.1 ± 7.6% ($P < 0.04$, vs fasting), and 37.9 ± 5.8%, respectively, suggesting that the meal stimulated an increase of the normal gastric slow wave and converted some bradygastria to normal slow waves. The highest percentage of bradygastria was seen in channel 3 ($P < 0.04$, vs channel 4).

Decreased Slow Wave Coupling in SSc Patients. The mean percentage of slow wave coupling among the four-channel EGGs in the fasting and fed states in the SSc patients compared to the normal controls was also recorded (Figure 5). In the fasting state, the mean percentage of slow wave coupling of the SSc patients was about 50% of that obtained from the normal controls, (45.3 ± 4.7 vs 90.7 ± 7.3%, $P < 0.01$). Postprandially, it showed a marginal increase (55.6 ± 6.7% vs 45.3 ± 4.7%, $P = 0.07$) but was still significantly lower that that in the normal controls.

Abnormal Spatial Distribution of Slow Wave Power in SSc Patients. Figure 6 shows the spatial distribution of dominant slow wave power (or dominant power of the EGG) in the fasting and fed states of the normal controls and the SSc patients. In the fasting and fed states, the normal controls (upper panel) demonstrate a fairly uniform generation of slow wave power. As shown in Figure 6 (upper panel), the mean dominant power for channels 1 and 2 was

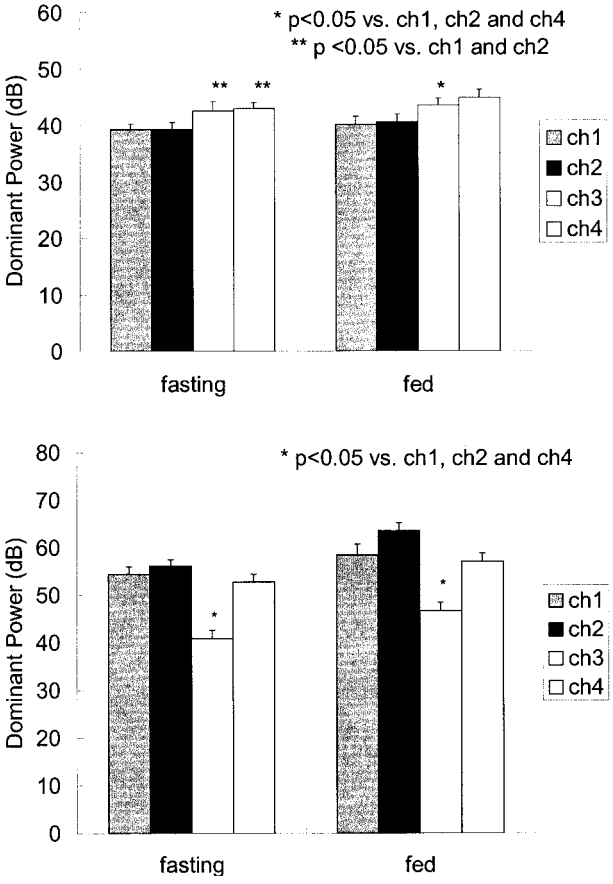

Fig 6. Spatial distribution of dominant power in the four-channel EGGs.

about 38 dB, whereas the dominant power in channels 3 and 4 was about 45 dB, significantly higher than that in channels 1 and 2. The normal controls essentially maintained the same values in spatial distribution of slow wave power in the fed state.

In the SSc patients, there was a spatial abnormality noted in the dominant slow wave power (Figure 6, lower panel). The dominant slow wave power in channel 3 was lower compared to the other channels in both fasting and fed states. In the fasting state, the dominant power in channel 3 was significantly lower compared to channel 1, 2, or 4 (40.9 ± 1.7 dB vs 54.5 ± 1.5, 56.1 ± 1.4, or 52.8 ± 1.7 dB, $P < 0.01$). Postprandially, there was a significant increase in the dominant slow wave power in all channels (channel 1–4: 58.5 ± 2.3, 63.6 ± 1.7, 46.7 ± 1.8, and 57.1 ± 1.8 dB, respectively, $P < 0.05$ vs fasting). However, the spatial abnormality was still present in the fed state, ie, the dominant slow wave power in channel 3 was significantly lower than the other channels ($P < 0.001$ vs channel 1, 2, or 4).

Abnormal Spatial Distribution of Dominant Slow Wave Frequency in SSc Patients. The dominant slow wave frequency in the normal controls measured from the EGG was the same among four channels in both fasting and fed states, suggesting a single pacemaker in the stomach. In the SSc patients, however, there was a significant decrease in the dominant slow wave frequency in channel 3 compared with other channels (Figure 7, lower panel, 2.81 ± 0.17 cpm (channel 3) vs 3.01 ± 0.14 (channel 1), 3.08 ± 0.21 (channel 2); or 3.03 ± 0.14 cpm (channel 4), $P < 0.05$). This abnormal distribution among the four channels was corrected in the fed state (Figure 7, upper panel).

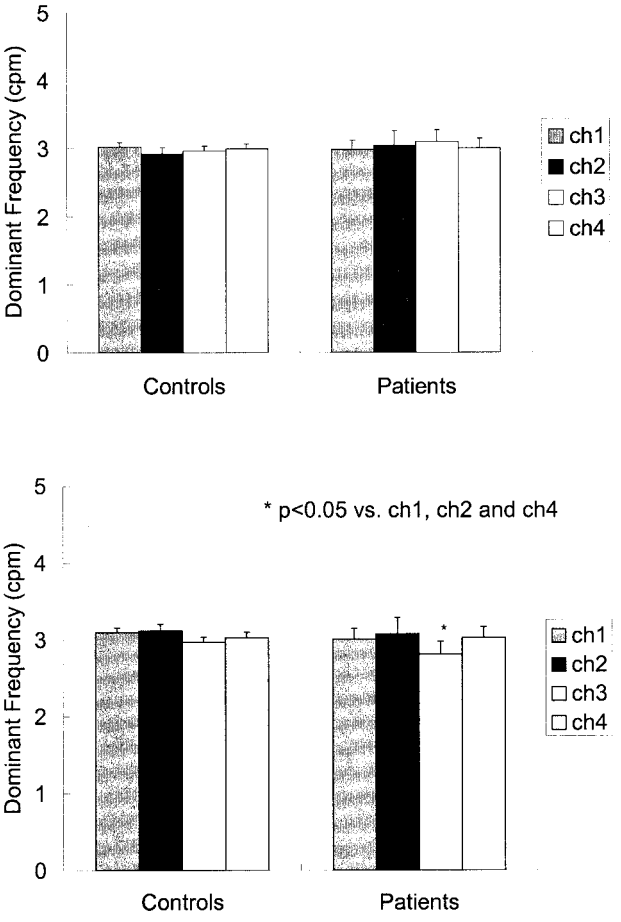

Fig 7. Spatial distribution of dominant frequency in the four-channel EGGs.

DISCUSSION

Gastric myoelectrical activity in patients with SSc was measured using a novel method of multichannel electrogastrography. Abnormalities were observed in the rhythmicity and spatial distribution of the gastric slow wave. These include: (1) a decreased percentage of normal gastric slow waves in both fasting and fed states, attributed to an increased percentage of bradygastria; (2) a reduced percentage of slow wave coupling among the four-channel EGGs; and (3) abnormal spatial distribution of slow wave amplitude and frequency.

Scleroderma or systemic sclerosis (SSc) is a multisystemic autoimmune disorder characterized by widespread small vessel vasculopathy and fibrosis of skin and various internal organs (22). The gastrointestinal tract is affected in nearly 90% of patients, esophageal reflux and dysphagia being the most common complaints. Impairment in the stomach is observed in 10–75% of patients with SSc (1–5). A significant impairment in gastric myoelectrical activity was found in the current study, suggesting a possible role of electrogastrography for the assessment of gastric involvement in patients with SSc. Previous clinical studies have demonstrated that the EGG is an accurate measurement of gastric myoelectrical activity (8, 10). Abnormalities in gastric myoelectrical activity measured from the EGG have been frequently reported in patients with gastrointestinal motility disorders, such as, gastroparesis (23–25), functional dyspepsia (26–28), gastric esophageal reflux (29), chronic intestinal pseudoobstruction (30), etc. Previous validation studies with simultaneous recordings of the EGG and gastric motility have demonstrated that gastric dysrhythmia measured from the EGG is associated with

gastric hypomotility (31). Impaired gastric myoelectrical activity measured from the EGG predicts delayed gastric emptying with an accuracy of about 85%, although normal gastric myoelectrical activity does not guarantee a normal emptying of the stomach (32). Since almost all other methods for the assessment of gastric motility or emptying are either invasive or radioactive, such as gastric manometry and gastric scintigraphy, the noninvasive EGG would be an attractive alternative for the assessment of gastric motility abnormalities in patients with SSc.

Identification of gastric motility disorders in SSc patients using the EGG may lead to better treatment of gastrointestinal symptoms in these patients. Several previous studies have shown that prokinetic therapy is effective for the treatment of gastrointestinal symptoms in patients with abnormalities in gastric myoelectrical activity measured from the EGG (23, 25, 33). In patients with dyspeptic symptoms, it was found that the treatment using cisapride yielded a significant improvement in symptoms over the placebo in those with abnormal gastric myoelectrical activity measured from the EGG, whereas in the patients with a normal EGG, cisapride was not better than the placebo (33).

While there have been a few EGG studies in patients with SSc (11–13), this is the first experiment characterizing gastric myoelectrical activity using multichannel electrogastrography. The conventional single-channel EGG reveals information on the rhythmicity or regularity of gastric slow wave and the response of the slow wave to the test meal. The multichannel EGG, as used in this study, provides additional information on the spatial distribution of the EGG parameters, including spatial distribution of the frequency, amplitude, and percentage of the normal waves and coupling of the gastric slow wave. Apparently, the multichannel EGG is more capable of detecting abnormalities of the gastric slow wave than the single-channel EGG. Previous studies have established the methodology of the multichannel EGG and its application in identifying slow wave abnormalities in patients with functional dyspepsia (17, 18).

The current study revealed a significantly reduced percentage of regular 2–4 cpm slow waves in both fasting and fed states in comparison with the healthy controls. The difference between the SSc patients and the controls was substantial and significant in every channel. Further, it was found that the reduction in the regular 2–4 cpm waves was attributed to an excessive amount of bradygastria. These findings were similar to a previous study using the conventional single-channel EGG, except that the previous study failed to show a difference between the controls and patients in the percentage of regular 2–4 cpm in the fed state (13). In addition, our current study indicated that the lowest percentage of regular slow waves or highest percentage of bradygastria was measured from channel 3, reflecting the antral region of the stomach. This information was not available in the previous studies reported by other investigators.

The spatial abnormalities of gastric myoelectrical activity reported in this study have never been previously reported, because of the lack of availability of the multichannel EGG. Previously, spatial information on gastric myoelectrical activity was only available in multichannel recordings using surgically implanted serosal electrodes (42). While the serosal method is extremely useful in electrophysiological studies of the stomach, its application in clinical gastroenterology is very limited due to its invasiveness. The spatial distribution of the EGG parameters among the four-channel EGGs reported in this study was normal in the healthy controls but impaired in the SSc patients. In the controls, the dominant frequencies from the four-channel EGGs were the same, suggesting a single pacemaker in the stomach; the dominant power of the EGG was higher in the distal channel than in the proximal channel, implying an increased slow wave amplitude distally. In the SSc patients, however, the EGG measured from channel 3 showed a significantly lower dominant frequency and dominant power (as well as the lowest percentage of regular 2–4 cpm waves, as mentioned above), suggesting an impairment of the gastric slow wave in the antral region of the stomach. In addition, the percentage of gastric slow wave coupling was significantly lower in contrast to the controls. This implies that the gastric slow waves in the stomach were not consistent or appropriately coordinated. Similar findings were reported in a previous study in patients with functional dyspepsia (18).

It was interesting to note that most of the EGG parameters in the patients with SSc showed an improvement after the test meal, suggesting a compensatory response of the stomach to the ingested food. In comparison with the healthy controls, however, most of the parameters did not reach the normal range. This phenomenon of postprandial improvement was in contrast to other groups of patients with functional dyspepsia or gastric esophageal reflux (28, 29). In these groups of patients, abnormalities in

gastric myoelectrical activity are usually more prominent in the postprandial state.

It is known that the major complications of SSc in the gastrointestinal tract is the degeneration of smooth muscle (myopathy). It was expected that there would be a reduction in slow wave amplitude. This was indeed observed: a decreased power at the slow wave frequency in the antrum (channel 3) as shown in Figure 6. In addition, however, abnormalities were also noted in the rhythmicity of slow waves, a phenomenon often observed in patients with neuropathy. These data suggest that either the SSc patients in this study might have neuropathies or the degeneration of smooth muscle may also lead to an impairment in slow wave rhythmicity. It has recently been reported that the interstitial cells of cajal (ICCs) are pacemaker cells generating slow waves. It is, however, unknown whether patients with SSc have impaired or absent ICCs in the stomach.

Currently, no specific antidysrhythmic medications are available in clinical gastroenterology. However, several medications, mainly prokinetic agents, have been shown to be effective for the normalization of gastric dysrhythmia. These include erythromycin (34), cisapride (23, 33), domperidone (25), and indomethacin (35). Acupuncture and acupressure have also been shown to be effective for the treatment of gastric dysrhythmia (36, 37). In one study, acupressure was found to reduce gastric tachyarrhythmia and symptoms of vection-induced motion sickness (36). In the other study, electroacupuncture normalized gastric arrhythmia (37). Mild exercise was reported to enhance postprandial gastric slow waves (38). Recently gastric pacing has also been reported to be able to normalize gastric and intestinal myoelectrical dysrhythmias in patients with gastroparesis (39, 40) or animal models of induced dysrhythmia (41).

In summary, gastric myoelectrical activity is impared in patients with SSc. The impairment is reflected as a decreased percentage of regular 2–4 cpm slow waves and an excessive amount of bradygastria as well as an abnormal spatial distribution of EGG parameters among the four channels, suggesting an impaired coordination of gastric slow waves.

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