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Gastric Myoelectrical Activity in Patients with Type I Diabetes Mellitus and Autonomic Neuropathy

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In patients with diabetes mellitus and gastroparesis, dysrhythmias of gastric myoelectrical activity, especially tachygastrias, are thought to be involved in the pathogenesis of dyspeptic symptoms. Using surface electrogastrography we studied the prevalence of these abnormalities, and their relationships to dyspeptic symptoms and the extent of cardiac autonomic neuropathy in 30 euglycemic patients with type I diabetes mellitus and 12 controls. Neither in the fasting nor in the postprandial state were differences in mean frequency of gastric electrical control activity and its variability found between patients and controls. In the fasting state, the power content of the 3 cpm component in the power spectrum of the electrogastrogram was even higher in patients than in controls ($P = 0.049$). In the fasting state, second harmonics of the 3 cpm fundamental gastric signal were seen more often in patients than in controls ($P = 0.03$). In patients with symptoms during the study, no second harmonics were found after the meal. The postprandial/fasting power ratio was decreased in patients with symptoms during the study as compared to patients without symptoms and controls ($P < 0.05$). The incidence of dysrhythmias, such as tachygastrias and bradygastrias, was not higher in patients than in controls (17% and 8%, respectively). No correlation was found between electrogastrographic parameters and the severity of autonomic neuropathy or dyspeptic symptoms scored before the study. In conclusion, this study has shown that patients with type I diabetes mellitus and autonomic neuropathy studied under euglycemic conditions do not have grossly disturbed myoelectrical activity, except when symptomatic during the study.

KEY WORDS: electrogastrography; diabetes mellitus; autonomic neuropathy; gastric myoelectrical activity; dyspeptic symptoms.

Dyspeptic symptoms occur frequently in diabetes mellitus. Disordered gastrointestinal motility is thought to play an important role in the genesis of these symptoms (1–4). Gastrointestinal motility is controlled by rhythmically recurring electrical potentials, referred to as electrical control activity (ECA). In the human stomach these potentials originate in the gastric pacemaker in the orad corpus at a frequency of 3 cycles per minute (cpm) (5–8) and propagate aborally to the pylorus. In man, the frequency of the ECA is 11–12 cpm in the duodenum and decreases to 8–10 cpm in the terminal ileum (9–10). When phasic contractions occur, the ECA is accompanied by a second slow component with or

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without superimposed spike potentials, which are referred to as electrical response activity (ERA) (6). Normal gastric myoelectrical activity and dysrhythmias can be recorded in a noninvasive way by cutaneous electrodes. This method is known as electrogastrography or EGG (6, 11–12).

Several types of abnormal gastric myoelectrical activity were found in a variety of clinical syndromes, in which gastric motor disorders and dyspeptic symptoms are present: (1) instability of the ECA; (2) absence of the postprandial amplitude increase of the EGG signal; (3) waveform changes in the EGG signal; and (4) dysrhythmias, such as regular and irregular bradygastrias and tachygastrias (3, 13–22). Dysrhythmias, especially tachygastrias (4–9 cpm), are thought to be involved in the pathogenesis of dyspeptic symptoms in patients with diabetes mellitus. These dysrhythmias can be associated with altered antral motility or impaired gastric emptying (3). The pathogenesis of these dysrhythmias is obscure. Hyperglycemia, which is associated with impaired gastric emptying, might contribute to the induction of dysrhythmias (23). To our knowledge, a study of the gastric myoelectrical activity under standardized glycemic conditions had never been undertaken.

The aim of the present study was to examine: (1) the prevalence of abnormalities in gastric myoelectrical activity in a well-defined group of patients with type I diabetes mellitus and autonomic neuropathy studied under euglycemic conditions, and (2) the relationship of these abnormalities with the extent of autonomic neuropathy and the severity of dyspeptic symptoms.

MATERIALS AND METHODS

The study was performed in 30 patients with insulin-dependent (type I) diabetes mellitus (fasting C-peptide < 0.1 nmol/liter) with cardiovascular signs of autonomic neuropathy (AN). These patients had been recruited from the outpatient endocrinology clinic of our hospital. In all patients, a scintigraphic gastric emptying test was performed within one month before the study, using a solid meal (pancake with 276 kcal, 14% protein, 15% fat, and 71% carbohydrate). Autonomic nerve function was assessed by standard cardiovascular reflex tests as described by Ewing and Classche (24), and as summarized below. On the basis of these tests, patients were classified as suffering from early, definite, and severe involvement of cardiovascular AN. For each group, 10 patients were selected. The patients were 11 women and 19 men, age (mean ± SEM): 45.5 ± 2.5 years, weight: 69.9 ± 2.4 kg and duration of diabetes 25.6 ± 8.6 years. Mechanical obstruction or other diseases responsible for the symptoms were excluded by routine laboratory tests, proximal endoscopy, and ultrasound examination. The blood glucose level was kept in the range of 6–12 mmol/liter during all studies. To achieve this, the blood glucose was measured every 15 min with a blood glucose meter (Reflolux S, Boehringer, Mannheim, Germany) and if necessary insulin (Actrapid Human, Novo Industri BV, Amsterdam, The Netherlands) or glucose was given intravenously.

Twelve healthy (sex-, age-, and weight-matched) volunteers (five women and seven men; age: 46.3 ± 3.7 years and weight: 74.8 ± 2.7 kg) served as control subjects. Informed consent was obtained in each case and the study protocol was approved by the Ethical Committee of the University Hospital Utrecht.

Assessment of Cardiovascular Autonomic Neuropathy. Autonomic nerve function was assessed by standardized cardiovascular reflex tests (24): (1) reduction in systolic blood pressure in response to standing up, (2) heart rate in response to upright position with calculation of the ratio between the heart rate response that occurs at the 15th and 30th beat after standing up, (3) beat-to-beat rate variation during deep breathing, (4) quotient of the heart rate during and after Valsalve maneuver, and (5) blood pressure response to sustained handgrip. All tests were scored as normal (0 points), borderline (0.5 points) or abnormal (1 point). From the total score (0–5 points) the degree of involvement was obtained (normal: 0–0.5 points; early involvement: 1–2 points; definite involvement: 2.5–3.5 points; severe involvement: 4–5 points).

Assessment of Gastrointestinal Symptoms. All subjects (patients and control subjects) were interviewed at the beginning of the study to assess the severity of symptoms, experienced during the week before the study. The symptoms experienced during the study were also assessed in patients and control subjects. This was done before the start of the meal and at the end of the study. A symptom score was assigned to the symptoms nausea, vomiting, early satiety, fullness, bloating, and epigastric pain using a scale from 0 to 3 (0 = absence of symptoms; 1 = mild symptoms noticed only when paid attention to; 2 = moderate, symptoms clearly noticed without interfering with normal daily activities; 3 = severe, symptoms interfering with normal daily activities). From these scores the mean symptom score was calculated.

Electrogastrography. Gastric electrical activity was recorded from five disposable pregelled silver–silver chloride surface electrodes (Red Dot 3M, Canada Inc., Ontario, Canada), placed on the upper abdomen (Figure 1). Four EGG signals were recorded bipolarly from these five electrodes as potential difference between each of the four electrodes (M1–4) and the central electrode (MC). A reference electrode was placed on the right ankle. EGG signals were band-pass filtered (0.01–0.50 Hz). After analogue-to-digital conversion the signals were stored online (sample frequency 1 Hz) on the hard disk of a personal computer. A respiratory signal, monitored by a temperature-sensitive probe positioned at a nostril, was also recorded. Recordings were made during 2 hr fasting and 3 hr after a solid meal. The meal consisted of a pancake containing 14% protein, 15% fat, and 71% carbohydrate (276 kcal).

For data acquisition, data analysis and graphical pre-

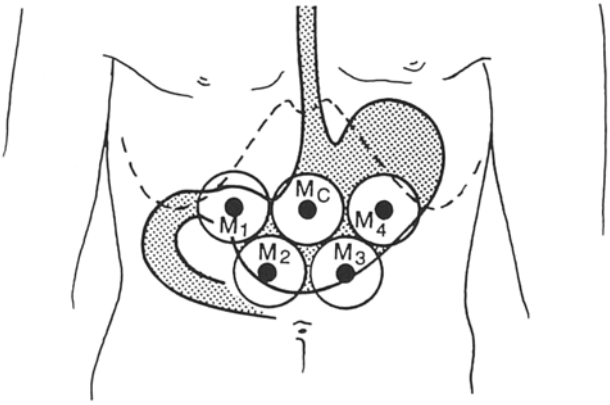

Fig 1. Positions of the electrodes used in EGG recording. EGG signals were recorded as the potential difference between MC, the central electrode, and each of the four surrounding electrodes (M1–4).

sentation-dedicated locally developed software was used as described by Bruijs et al (25). After the recording session, the EGG signals were subjected to fast Fourier transform (spectral analysis). Power spectra were obtained using signal stretches of 256 sec that overlapped for 192 sec (running spectrum analysis). The mean of the power spectra for the entire recording period was calculated. The EGG signal with the highest power in the 3 cpm band was then selected for further analysis (the preferential EGG signal). The mean frequency of the normal 3 cpm component, its standard deviation (SD), and its power content (μV^2) was calculated for the fasting and postprandial period (the entire postprandial period and each hour separately). Higher harmonics were identified in the spectrum using the criteria that they occur at frequencies that are exact multiples of the fundamental frequency and that their power should be at least 5% of the power of the fundamental component. The more a time signal deviates from a sinusoid, the more harmonics are present in the frequency spectrum (18). To express the frequency variability of the 3 cpm component, the frequency instability factor was calculated. This parameter is defined as the ratio of the standard deviation (SD) of the frequency of a series of spectra (eg, the entire fasting or postprandial period) to the frequency SD of one representative spectrum (containing a clearly present 3 cpm component) of this series (18). The early postprandial frequency dip of the normal 3 cpm gastric component was identified. The frequency minimum and the subsequent frequency maximum of the dip were calculated by means of line-to-line analysis of the first 10 running spectra after the meal. The power ratio (the ratio of the power of the mean spectrum of the postprandial state to the power of the mean spectrum of the fasting state), indicative of the postprandial increase in gastric motor activity, was calculated for the total postprandial period and for each postprandial hour separately.

Dysrhythmias were defined as follows. A tachygastria was considered to be present when the power spectrum contained a sharp-peaked component with a frequency >3.7 cpm (0.06 Hz) and <10.8 cpm (0.18 Hz), which was not of respiratory origin. These definitions are based on observations made in studies in healthy control subjects (18, 26, 27). For a definite diagnosis of tachygastria, it was required that at the same time the normal gastric signal (2.6–3.7 cpm) be absent in all four EGG signals and that the abnormal rhythm be present for at least 2 min. When a tachygastric frequency was found in the presence of a normal gastric signal in one of the four EGG signals or the frequency was ≥10.8, the diagnosis of tachygastria was considered probable, but not definite. A so-called bradygastria was defined as presence of a sharp peak at a frequency less than 2.6 cpm, in the absence of a normal 3 cpm component in all four EGG leads. Duration and mean frequency of the dysrhythmias were measured.

Statistical Analysis. All values are expressed as mean ± SD, unless stated otherwise. Statistical analysis of differences between groups was performed by the Mann-Whitney rank-sum test for unpaired data. Differences between patients with symptoms, patients without symptoms during the study, and control subjects were assessed by the chi-square test. $P < 0.05$ was considered significant. Correlations between variables were assessed by calculation of Spearman's rank correlation coefficients.

RESULTS

General Characteristics. All patients (mean duration of diabetes mellitus: 25.6 ± 8.6 years) had one or more other complications, such as retinopathy, nephropathy, or peripheral neuropathy (90%, 57%, and 93% of the patients, respectively). Eighteen patients (60%) had a delayed gastric emptying. Twenty-six patients (87%) had upper abdominal symptoms varying in frequency and severity. The symptom score before the study (mean ± SD) was 1.2 ± 1.0 (range 0–2.8). One patient did not complete the postprandial part of the study because of nausea and vomiting, starting during fasting. Eight other patients but none of the control subjects reported fullness, early satiety, and bloating postprandially. Five of these patients had also nausea and vomiting. The symptom score of these patients during the study was 1.9 ± 0.8. No correlation was found between the severity of these symptoms and the symptom score before the study. Metabolic control was considered poor in 12 patients (HbA_1 at the time of the study >10%), as moderate in 16 patients (HbA_1 8–10%), and as good in four patients (HbA_1 <8%). In patients, the mean blood glucose level was 9.2 ± 2.2 mmol/liter in the fasting state and 9.5 ± 2.7 mmol/liter in the postprandial state, compared 5.0 ± 0.6 and 5.2 ± 0.8 mmol/liter, respectively, in the control subjects.

Electrogastrographic Parameters. As shown in Table 1, mean frequency of the gastric ECA and its variability (SD) in patients with diabetes mellitus

TABLE 1. ELECTROGASTROGRAPHIC VARIABLES IN PATIENTS WITH DIABETES MELLITUS AND CONTROL SUBJECTS IN THE FASTING AND POSTPRANDIAL STATE*

	Patients (N = 30)	*Control Subjects (N = 12)*
Fasting		
Gastric ECA frequency (Hz)	0.0489 ± 0.0006	0.0478 ± 0.0001
Instability factor	1.33 ± 0.05	1.57 ± 0.13
Power in 3-cpm peak (μV^2)	8.01 ± 3.80a	2.13 ± 1.25
Postprandial		
Gastric ECA frequency (Hz)	0.0488 ± 0.0006	0.0494 ± 0.0009
Postprandial frequency dip		
minimum	0.0444 ± 0.0007	0.0457 ± 0.0009
maximum	0.0515 ± 0.0008	0.0522 ± 0.0009
Instability factor	1.43 ± 0.08	1.48 ± 0.27
Power in 3-cpm peak (μV^2)	10.13 ± 3.96	3.70 ± 1.22
Power ratio (postprandial/fasting)	4.51 ± 1.66	4.07 ± 1.54

*Values are expressed as mean ± SEM; a, $P = 0.049$.

were not different from those found in control subjects, both in the fasting and the postprandial state. The frequency instability factor was not different between patients and control subjects. The postprandial frequency decrease was also similar in patients and control subjects. However, the power content of the 3 cpm component was higher in the patients than in the control subjects ($P = 0.049$). In the postprandial state this power content tended to be higher in patients compared to control subjects ($P = 0.1$).

As shown in Figures 2 and 3, in the fasting state, second harmonics were observed more frequently in patients compared to control subjects ($P = 0.03$). In the postprandial state, no differences were found. However, in patients with postprandial symptoms during the study, no second harmonics were found after the meal, which was a significant difference compared to patients without symptoms during the study and control subjects (50% and 54.5%, respectively; $P = 0.03$).

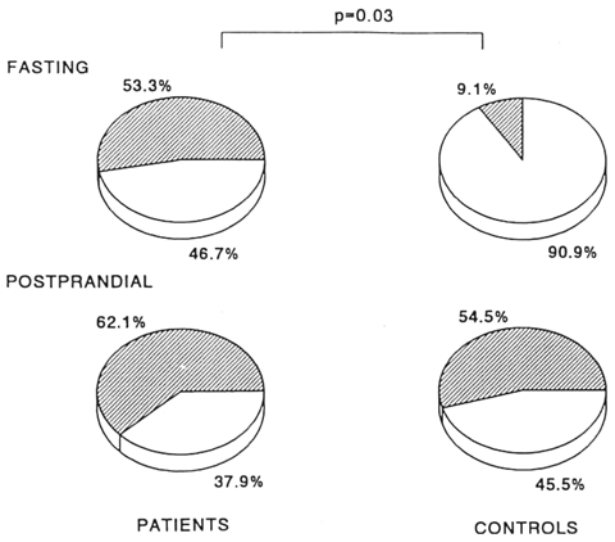

Fig 2. Prevalence of second harmonics in EGG power spectrum of patients with diabetes mellitus (striated pie portions) compared to controls, fasting ($P = 0.03$) and postprandially (NS).

The postprandial/fasting power ratio was not different between patients and control subjects, not only for the total postprandial period, but also for each hour separately (Table 1). As shown in Figures 3 and 4, patients with symptoms during the study had a lower power ratio than patients without symptoms during the study and control subjects ($P < 0.05$). No differences were found in the other EGG parameters between patients with and without symptoms during the study. No correlation was found between the EGG parameters of the gastric signal and the extent of cardiovascular autonomic neuropathy.

DYSRHYTHMIAS

Definite dysrhythmias were seen in five of the patients (17%) and one of the control subjects (8%), the difference not being significant. Definite tachygastrias were seen in two patients, with frequencies of 4.2 cpm and 10.2 cpm (duration: 5 and 15 min, respectively) and in one of the control subjects, with a frequency of 4.2 cpm (duration: 4 min). In the patients, all tachygastrias were associated with vomiting, fasting in one patient and postprandially in the other patient (Figure 5). The only tachygastria observed in the control subjects occurred during fasting and was not associated with symptoms. Probable tachygastrias (high frequency simultaneously present with a normal 3 cpm gastric signal)

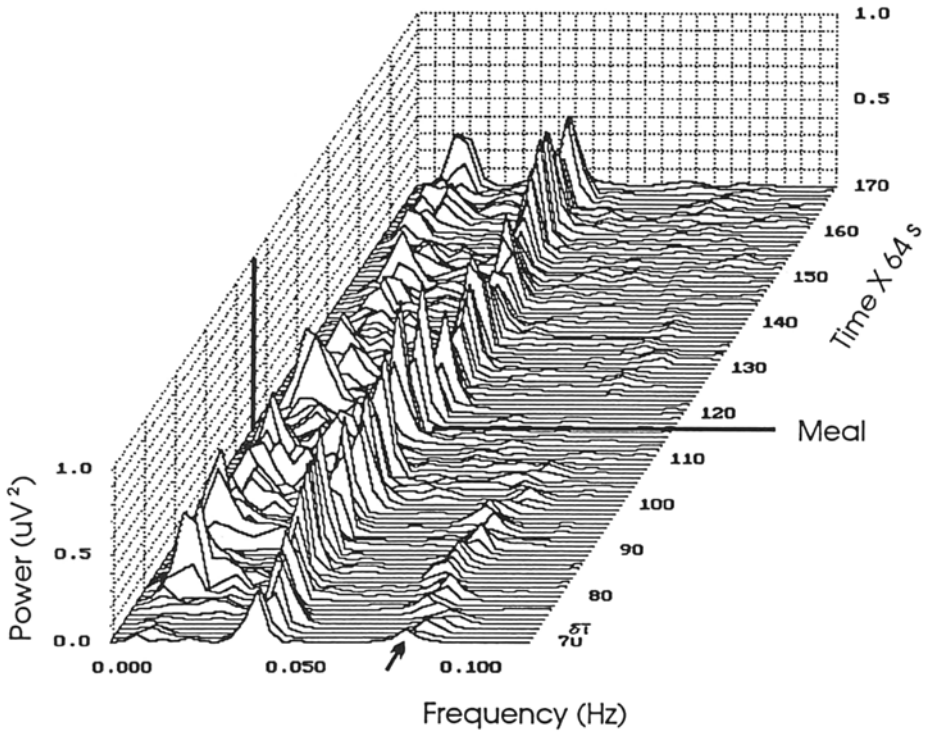

Fig 3. Pseudo-3-dimensional display of running spectra of EGG signals from a patient with diabetes mellitus, in whom no postprandial power increase occurred (postprandial/fasting power ratio 0.5). A second harmonic is recognized in the spectra of the fasting state (↑).

were seen in the same two patients with definite tachygastrias, in six other patients, and in two (other) control subjects. In the patients these probable tachygastrias were in the range of 8.4–10.8 cpm, except one with a frequency of 4.2 cpm (mean duration: 61 ± 14.1 min). In the two control subjects, the frequencies were 4.2 cpm and 9.6 cpm (duration 5 and 45 min, respectively).

Bradygastrias with a duration of 3–10 min were seen in five patients (in one patient during fasting and in four patients postprandially) and in one of the control subjects (during fasting) showing frequen-

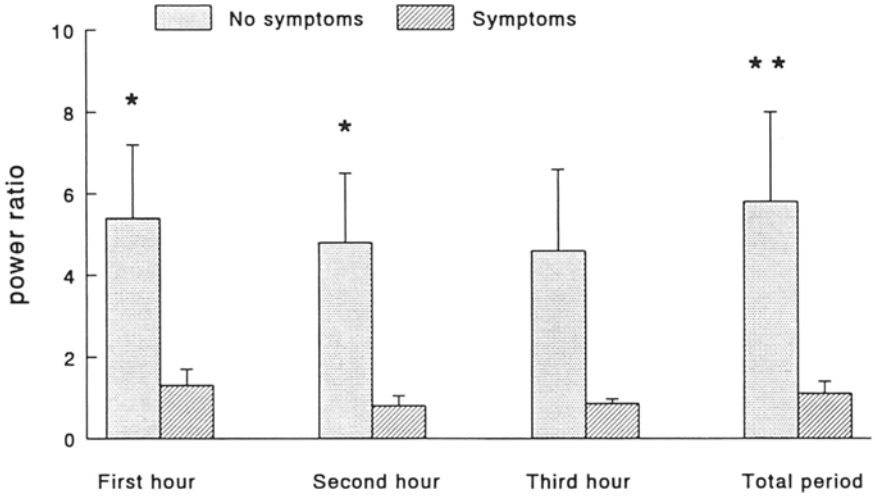

Fig 4. Postprandial/fasting power ratio in patients with symptoms during the study (striated bars) compared to patients without symptoms (dotted bars), for each hour separately (*P = 0.03) and for the total postprandial period (**P = 0.048).

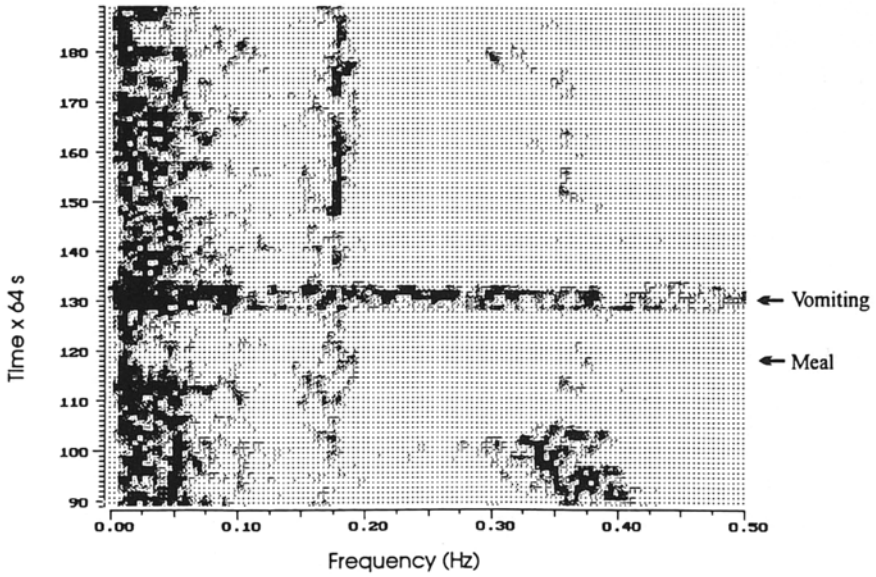

Fig 5. Grey-scale plot in a symptomatic patient, showing a normal 3 cpm gastric signal (0.05 Hz) in the fasting state. After the meal vomiting occurred. Thereafter, a component of 10.2 cpm (0.17 Hz) appeared, and the 3 cpm component became unstable. The spectrum of the respiratory signal can be seen at 0.3–0.4 Hz.

cies of 0.6–2.1 cpm. In four of the patients bradygastrias were temporarily related to symptoms of nausea and vomiting. In two patients and one control subject both tachygastrias and bradygastrias were seen. No correlation was found between dysrhythmias, the severity of symptoms scored before the study, and the extent of cardiovascular AN.

DISCUSSION

Recent studies have suggested that gastric dysrhythmias in patients with diabetes mellitus may contribute to dyspeptic symptoms (3, 16, 18, 20, 22). In the present study, however, the prevalence of dysrhythmias in patients with type I diabetes mellitus and autonomic neuropathy studied under euglycemic conditions was found not to be higher than in healthy control subjects. Definite tachygastrias were observed in two of the 30 patients and in one healthy control subject. This discrepancy in the incidence of dysrhythmias might in part be due to differences in patient selection. Previous studies were performed in selected patient groups with scintigraphically proven gastroparesis, whereas only 60% of our patients had delayed gastric emptying. The discrepancy could also be caused by the blood glucose level during the study (3, 20). In this study a euglycemic level was maintained during the whole study. In the other studies, electrogastrograms were obtained without controlling the serum glucose level. It is therefore possible that hyperglycemia, which also contributes to impaired gastric emptying (23), was a factor in the induction of these dysrhythmias. Another possible explanation for the discrepancy might be found in the definition of dysrhythmia, used in the different studies. For a definite diagnosis of tachygastria, we required the presence of a sharp-peaked component of >3.7 and <10.8 cpm, in the absence of a 3 cpm component. When a 3 cpm component persisted or the abnormal peak had a frequency ≥10.8 cpm, we considered a tachygastria probable, but not definite. Probable tachygastrias, with the simultaneous presence of a normal 3 cpm signal, were seen in eight patients and two control subjects.

In most of these patients the tachygastrias had a frequency of approximately 10 cpm. Simultaneous recording of a respiratory signal excluded that this high frequency was due to respiration. It cannot be completely excluded that this frequency is of small intestinal origin: in the human small bowel the ECA shows a frequency gradient from 12 cpm in the duodenum to 7 cpm in the terminal ileum (11–12, 27). However, the presence of a duodenal frequency in the surface signal is very rare in humans (6). In a study in patients who had undergone a Billroth II resection, a high prevalence of a 10 cpm activity was found. The investigations suggested that this frequency could be caused by retrograde

conduction (into the stomach) of the electrical activity of the jejunum (28). An ectopic focus in the antrum has been unequivocally shown to be the cause of periods in the frequency band of 4–5 cpm (6, 29). This was seen in only one patient and also in one control.

Low-frequency components (bradygastrias) were recorded in patients with and without symptoms during the study. In our opinion, the significance of these low-frequency components is still unclear, and it is far from certain that they originate in the stomach. In our study no correlations were found between dysrhythmias, dyspeptic symptoms scored before the study, and the extent of cardiac AN. This observation is in accordance with findings reported by others (4, 14, 29).

The mean frequency and the frequency instability factor were not different between the groups. The power content of the gastric signal was even higher in the patients than in the control subjects. It is suggested that the power of the signal is related to the amplitude and regularity of the gastric signal and an increase in the power may reflect the presence of contractile activity (6, 10). The absolute value of the power is also influenced by other factors (eg, electrode-skin resistance, tissue conductivity, distance between electrode and gastric wall). Therefore, conclusions about intra- and interindividual power data should be made with caution (30). The postprandial/fasting power ratio, indicative of the postprandial increase in gastric motor activity, was not different between patients and control subjects. However, in patients with symptoms during the study, this power ratio was lower than in patients without symptoms, especially during the first two postprandial hours. In addition, the symptomatic patients showed a decreased incidence of so-called higher harmonics. In healthy subjects, the incidence of higher harmonics increases after a meal. The most likely explanation for this phenomenon is that the occurrence of contraction-related ERA causes a change in the waveform of the EGG, which leads to higher harmonics after a meal (17). Postprandial antral hypomotility and thus absent ERA could be responsible for the absence of higher harmonics in the diabetic patients with symptoms during the study. It is likely, therefore, that both the decreased power ratio and the absence of higher harmonics in the EGG reflected postprandial antral hypomotility in patients.

In conclusion, this study has shown that patients with type I diabetes mellitus and autonomic neuropathy studied under euglycemic conditions do not have grossly disturbed gastric myoelectrical activity, except when symptomatic during the study. In the pathogenesis of disordered electrical activity in patients with diabetes mellitus, the extent of the cardiovascular AN appears to be a factor of limited importance.

ACKNOWLEDGMENTS

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REFERENCES

1. Feldman M, Schiller LR: Disorders of gastrointestinal motility associated with diabetes mellitus. Ann Intern Med 98:378–384, 1983
2. Horowitz M, Harding PE, Maddox AF, Maddern GJ, Collins PH, Chatterton BE, Wishart J, Shearman DJC: Gastric and oesophageal emptying in insulin-dependent diabetes mellitus. J Gastroenterol Hepatol 1:97–113, 1986
3. Abell TL, Camilleri M, Hench VS, Malagelada J-R: Gastric electro-mechanical function and gastric emptying in diabetic gastroparesis. Eur J Gastroenterol Hepatol 3:163–167, 1991
4. Loo FD, Palmer DW, Soergel KH, Kalbflesch JH, Wood CM: Gastric emptying in patients with diabetes mellitus. Gastroenterology 86:485–494, 1984
5. Sarna SK: Gastrointestinal electrical activity: Terminology. Gastroenterology 68:1631–1635, 1975
6. Smout AJPM, van der Schee EJ, Grashuis JL: What is measured in electrogastrography? Dig Dis Sci 25:179–187, 1980
7. Weber J, Kohatsu S: Pacemaker localization and electrical conduction patterns in the canine stomach. Gastroenterology 59:717–726, 1970
8. Hinder RA, Kelly KA: Human gastric pacesetter potential. Site of origin, spread and response to gastric transection and proximal gastric vagotomy. Am J Surg 33:29–33, 1977
9. Hamilton JW, Bellahsene BE, Reichelderfer M, Webster JG, Bass P: Human electrogastrograms. Comparison of surface and mucosal recordings. Dig Dis Sci 31:33–39, 1986
10. Abell TL, Malagelada J-R: Electrogastrography. Current assessment and future perspective. Dig Dis Sci 8:982–992, 1988
11. Diamant NE, Bortoff A: Nature of the intestinal slow-wave frequency gradient. Am J Physiol 16:301–307, 1979
12. Daniel EE, Sarna SK: The generation and conduction of activity in smooth muscle. Annu Rev Pharmacol Toxicol 18:145–166, 1978
13. Abell TL, Malagelada J-R, Lucas AR, Brown ML, Camilleri M, Go WLW, Azpiroz F, Callaway CW, Kao PC, Zinsmeister AR, Huse DM: Gastric electromechanical and neurohormonal function in anorexia nervosa. Gastroenterology 93:958–965, 1987
14. You CH, Chey WY, Lee KY, Menguy R, Bortoff A: Gastric and small intestinal myoelectric dysrhythmia associated with chronic intractable nausea and vomiting. Ann Intern Med 95:449–451, 1981
15. Stern RM, Koch KL, Stewart WR, Lindblad IM: Spectral

analysis of tachygastria recorded during motion sickness. Gastroenterology 92:92–97, 1987
16. Koch KL, Stern RM, Vasey M, Botti JJ, Creasy GW, Dwyer A: Gastric dysrhythmias and nausea of pregnancy. Dig Dis Sci 35:961–968, 1990
17. Geldof H, van der Schee EJ, Smout AJPM, vam de Merwe JP, van Blankenstein M, Grashuis JL: Myoelectrical activity of the stomach in gastric ulcer patients: An electrogastrographic study. J Gastrointest Motil 1:122–130, 1989
18. Geldof H, van der Schee EJ, van Blankenstein M, Grashuis JL: Electrogastrographic study of gastric myoelectrical activity in patients with unexplained nausea and vomiting. Gut 27:799–808, 1986
19. Bortolotti M, Santi P, Barbara L, Burnelli F: Gastric myoelectrical activity in patients with chronic idiopathic gastroparesis. J Gastrointest Motil 2:104–108, 1990
20. Koch KL, Stern RM, Steward WR, Dwyer AE: Gastric emptying and gastric myoelectrical activity in patients with symptomatic diabetic gastroparesis: Effect of long-term domperidone treatment. Am J Gastroenterol 84:1069–1075, 1989
21. Telander RL, Morgan KG, Kreulen DL, Schmatz PF, Kelly KA, Szurszewski JH: Human gastric atony with tachygastria and gastric retention. Gastroenterology 75:497–501, 1978
22. You CH, Lee KY, Chey WY, Menguy R: Electrogastrographic study of patients with unexplained nausea, bloating and vomiting. Gastroenterology 79:311–314, 1980
23. Fraser RJ, Horowitz M, Maddox AF, Harding PE, Chatterton DE, Dent J: Hyperglycemia slows gastric emptying in type I (insulin-dependent) diabetes mellitus. Diabetologia 33:675–680, 1990
24. Ewing DJ, Classche BF: Diagnosis and management of diabetic autonomic neuropathy. Br Med J 285:916–918, 1982
25. Bruijs PPM, van der Schee EJ, Smout AJPM, Akkermans LMA, van Strien HLCJ: Bedside system for cutanous recording and analysis of gastric myoelectrical and impedance signals. Med Biol Eng Comput 29:609–615, 1991
26. Geldof H, Van der Schee EJ: Electrogastrography; clinical applications. Scand J Gastroenterol 24:75–82, 1989
27. Cristensen J, Schedl HP, Clifton JA: The basic electrical rhythm of the duodenum in normal human subjects and in patients with thyroid disease. J Clin Invest 43:1659–1667, 1964
28. Schaap HM, Smout AJPM, Akkermans LMA: Myoelectrical activity of the Billroth II gastric remmant. Gut 31:984–988, 1990
29. Stoddard CJ, Smallwood RH, Duthie HL: Electrical arrhythmias in the human stomach. Gut 22:705–712, 1981
30. Mintchev MP, Kingma YJ, Bowes KL: Accuracy of cutaneous recordings of gastric electrical activity. Gastroenterology 104:1273–1280, 1993