GASTROENTEROLOGY 1998;114:456–461

Gastric Pacing Improves Emptying and Symptoms in Patients With Gastroparesis

RICHARD W. McCALLUM,* JIAN DE Z. CHEN,† ZHIYUE LIN,* BRUCE D. SCHIRMER,§ RONALD D. WILLIAMS,‖ and ROBERT A. ROSS‖

*Department of Medicine, University of Kansas Medical Center, Kansas City, Kansas; Departments of §Medicine and ‖Electrical Engineering, University of Virginia, Charlottesville, Virginia; and †Institute for Healthcare Research, Baptist Medical Center, Oklahoma City, Oklahoma

See editorial on page 598.

Background & Aims: No effective treatment is available for patients with gastroparesis refractory to standard medical therapy. The aim of this study was to investigate the effects of gastric pacing on gastric electrical activity, gastric emptying, and symptoms in patients with gastroparesis. *Methods:* Nine patients with gastroparesis participated in this study. Four pairs of cardiac pacing wires were implanted on the serosa of the stomach. The protocol consisted of two portions: a temporary inpatient study period and an outpatient study for a period of 1 month or more. *Results:* Gastric pacing entrained the gastric slow wave in all subjects and converted tachygastria in 2 patients into regular 3-cpm slow waves. Gastric emptying was significantly improved after the outpatient treatment with gastric pacing. The gastric retention at 2 hours was reduced from 77.0% ± 3.3% to 56.6% ± 8.6% ($P < 0.05$). Symptoms of gastroparesis were substantially reduced at the end of the outpatient treatment (1.51 ± 0.46 vs. 2.84 ± 0.61; $P < 0.04$). Eight of 9 patients no longer relied on jejunostomy tube feeding, and no adverse events were noted related to the pacing unit. *Conclusions:* Gastric pacing seems to be able to improve symptoms of gastroparesis and to accelerate gastric emptying in patients with gastroparesis. More controlled studies are necessary to further investigate the role of gastric pacing in clinical practice.

Gastroparesis is a chronic disorder of gastric motility, defined as delayed gastric emptying of a solid meal. Symptoms of gastroparesis range from early satiety and nausea in mild cases to chronic vomiting, dehydration, and nutritional compromise in severe cases.[1] Gastroparesis may be attributed to impaired motor activity and/or impaired myoelectrical activity.[2–5] It is known that gastric motility is regulated by gastric myoelectrical activity. Therefore, it is conceivable that abnormalities in gastric myoelectrical activity may lead to impaired gastric motility, and in turn to delayed gastric emptying. Gastric myoelectrical abnormalities include abnormal frequency of the gastric slow wave (gastric dysrhythmia), abnormally low amplitude of the gastric slow wave, which may be below the necessary threshold for contractions, and uncoupling of the gastric slow wave, which may result in a lack of propagated gastric contractions.

The commonly used medical therapy for gastroparesis is prokinetic agents.[6] These agents were developed to stimulate gastric motility, i.e., contractility of smooth muscles of the stomach, and coordinate gastric-duodenal motor activity.[1] Those who are refractory to the prokinetic agents may require placement of a feeding jejunostomy tube (J-tube) to sustain nutritional support, although that procedure will not effect gastroparesis.

Recently, gastric pacing (electrical stimulation of gastric smooth muscles) has received increasing attention among researchers and clinicians. A number of studies have been performed to investigate the acute effects of gastric pacing on gastric myoelectrical activity, gastric motility, and gastric emptying. The majority of studies in both humans and dogs have indicated that gastric pacing with appropriate parameters is able to entrain gastric slow waves and normalize gastric dysrhythmias.[7–13] This has led some to propose the use of gastric pacing to treat patients with gastroparesis. However, studies to this point have produced conflicting results. Although gastric pacing improved gastric emptying in the dog model with gastroparesis,[12] it did not accelerate gastric emptying in patients after surgery.[10] Very few studies have assessed the effect of gastric pacing in patients with gastroparesis.[14–16] The aim of this study was to investigate systematically the effect of gastric pacing on gastric myoelectrical activity, gastric emptying, and gastrointestinal symptoms in patients with gastroparesis.

Abbreviation used in this paper: J-tube, jejunostomy tube.

0016-5085/98/$3.00

Materials and Methods

Subjects

Nine patients (3 men and 6 women; mean age, 37 years; range, 19–48 years) with severe gastroparesis participated in the study. All patients had documented delayed gastric emptying of an isotope-labeled solid meal before enrollment. Five patients were diagnosed as having diabetic gastroparesis, 3 were idiopathic, and 1 was postsurgical. The common symptoms of these patients included nausea, vomiting, bloating, abdominal pain, weight loss, and anorexia. All patients had been receiving a full regimen of standard medical therapy but still required an abdominal surgery (laparoscopic approach[17]) for the placement of a J-tube for nutritional support. The study protocol was approved by the Human Investigation Committee at University of Virginia Health Science Center, and written consent forms were obtained from all subjects before the study.

Placement of Serosal Electrodes

During the scheduled surgery for the placement of the feeding J-tube, four pairs of temporary 28-gauge cardiac pacing wires (A & E Medical, Farmingdale, NJ) were implanted on the serosal surface of the stomach. They were arranged in an arching line along the greater curvature from the corpus to pylorus (Figure 1). The distance between two electrodes in the pair was 1 cm, and the distance between two adjacent pairs of electrodes was 4 cm. The most distal pair of electrodes were 2–4 cm above the pylorus. The pacing electrodes were affixed to the gastric serosa by partially embedding the wire in the seromuscular layer of the stomach. The electrode wires were brought out through the abdominal wall percutaneously and placed under a sterile dressing. The most proximal electrodes were used to deliver electrical stimulation, and the remaining pairs were used for recording gastric myoelectrical activity.

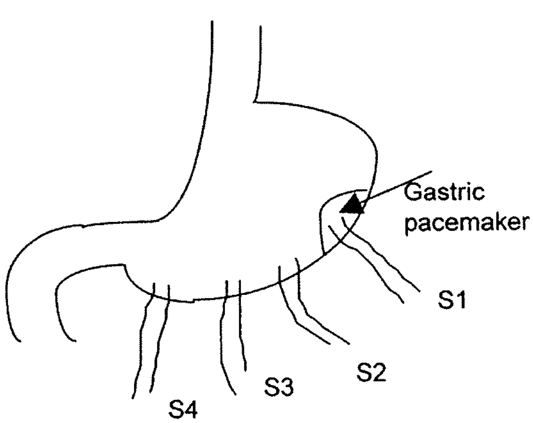

Figure 1. Location of serosal electrodes. The most proximal pair (S1) was used for pacing, whereas the rest were used for recording gastric myoelectrical activity.

Experimental Protocol

The study protocol consisted of (1) temporary inpatient gastric pacing to determine its effect on gastric myoelectrical activity, (2) baseline gastric emptying study without gastric pacing, (3) outpatient electrical stimulation for a period of 1 month or more, and (4) gastric emptying study with gastric pacing at the end of the study protocol. Patients remained on their individual prokinetic drug regimens during the baseline and throughout the outpatient protocol. All patients were taking cisapride (20 mg, four times daily) before and during the study. Metoclopramide was used intermittently in 4 patients with a dose of up to 60 mg/day. The study was initiated in November 1994 and completed in September 1996.

The temporary inpatient gastric electrical stimulation was performed in the laboratory 1 week or more after the patient had recovered from surgery and, particularly, was off all narcotic medications. On the day of the study, the patient fasted for 6 hours or more and was given no medications with known effects on gastrointestinal motility. A 30-minute baseline serosal recording was made in the fasting state via all the pacing wires to assess the intrinsic frequency of the gastric slow wave. Then a 30-minute recording was made from the three distal pairs of electrodes while the most proximal pair of electrodes was sham stimulated (the patient was told that electrical stimulation was performed, but no electrical current was actually delivered to the electrodes). This was followed with another 30-minute recording with real electrical stimulation. The stimulator used was a commercial product from World Precision Instruments (Sarasota, FL). It delivered a series of constant electrical current with an amplitude of 4 mA, width of 300 milliseconds, and frequency of 10% higher than the intrinsic frequency of the gastric slow wave measured in the baseline recording. These parameters had been determined in our laboratory by previous experiments on these patients and others with gastroparesis to be the appropriate combination for achieving entrainment of the gastric slow wave.[11]

The baseline gastric emptying was performed when the patient was completely recovered from the surgery before being discharged from the hospital. A portable pacemaker unit was developed in collaboration with the Department of Electrical Engineering at the University of Virginia.[18] This system delivered constant electrical current with the same parameters as used in the initial laboratory setting. It was operated by batteries and was the size of a Walkman. Before being sent home with the pacemaker, the patient was trained in its use, and written instructions were given. During the following month or more when the patient was at home, electrical stimulation was performed for up to 1 hour before the meal and for up to 3 hours after the meal. The patient was asked to turn on the device for at least two meals every day during the entire outpatient study period. Patients used the J-tube at night from 8PM to 7AM initially and ate at 9AM, 1PM, and 5PM if possible. Meals began as liquids and were slowly advanced to soft and semisolid depending on symptom tolerance. Patients were instructed to never use the J-tube during the day and to decrease the rate of J-tube feeding at night as the oral intake increased with improved symptom control. Their goal was to have no J-tube feeding at night and rely only on their ingested calories. The patient was also given a daily symptom score form and was asked to complete it daily for the entire study period. The duration of this outpatient study in individual patients ranged from 35 to 90 days with a mean value of 49 days. At the end of the outpatient treatment, the patient returned for another gastric emptying test. The procedure for this second gastric emptying was the same as for the baseline test, except that electrical stimulation was performed via the most proximal pair of electrodes using the portable pacemaker during the entire gastric emptying testing period. Also at that time, the patient's clinical status was assessed by reviewing the symptom grading in the diary, current oral nutritional intake, and J-tube needs. The patient was also asked to comment on the need for medical encounters including emergency room visits and, in the case of diabetics, their day-to-day glucose control and an overall global view of the change in quality of life.

Recording and Analysis of Gastric Myoelectrical Activity

The recording of gastric myoelectrical activity from the implanted serosal electrodes was made using a Hewlett Packard model 7758B Stripchart Recorder (Palo Alto, CA) chart recorder. The signals were displayed on the chart and simultaneously recorded on a tape recorder. The low and high cutoff frequencies of the recording device were 0.02 and 30 Hz, respectively. At the end of the study, all signals were played back from the tape recorder, digitized by a 12-bit analog-digital convertor (DATAQ Instruments Inc., Akron, OH), and stored in ASCII files on a 486 personal computer. The sampling frequency was 60 Hz. To reduce the volume of data, the digitized recording was filtered by a digital low-pass filter with a cutoff frequency of 0.5 Hz and sampled again at 2 Hz. The smoothed power spectral analysis, called periodogram method,[19] was applied to compute the power spectrum of the recording. The frequency and amplitude of the gastric slow wave were assessed from the power spectrum. Figure 2 shows a portion of the serosal recording of the gastric slow wave from a patient and its smoothed power spectrum. Slow waves (3 cpm) can be appreciated from both the tracing and the spectrum.

Gastric Emptying Test

The meal used in the gastric emptying test consisted of 7.5 oz of commercial beef stew mixed with 30 g of chicken livers. The chicken livers were microwaved to a firm consistency and cut into 1-cm cubes. The cubes were then evenly injected with 18.5 MBq of ^{99m}Tc–sulfur colloid. The liver cubes were mixed into beef stew, which was heated in a microwave oven. On the day of the gastric emptying test, the patient fasted over 6 hours. After the intake of this isotope-labeled solid meal, the patient was asked to lie supine under a gamma camera for 2 hours. Continuous anterior images were obtained at 1-minute intervals for 120 minutes. The gastric region of interest was outlined by hand on the initial images by a nuclear medicine technologist who had no interest in the outcome of the study. The counts were decay corrected and normalized to give percent of solid food retained in the stomach over time. Greater than 70% retention at 2 hours represents two standard deviations above the mean for the normal subjects established in our laboratory.

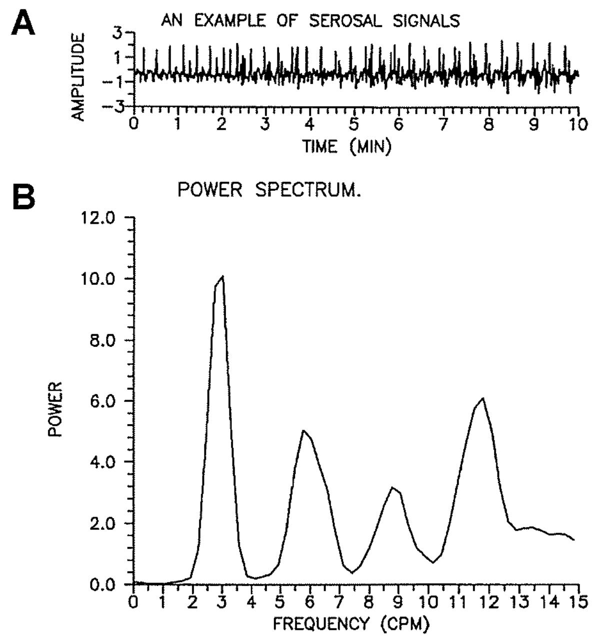

Figure 2. (*A*) A portion of serosal recording of the gastric slow wave in a patient and (*B*) its smoothed power spectrum. The peak at 3 cpm in the spectrum represents the frequency of the slow wave. The peaks at the multiples of 3 cpm are harmonics of the 3-cpm slow wave.

Assessment of Symptoms

A daily symptom score form was developed. Each subject was asked to report symptoms of nausea, vomiting, bloating, and abdominal pain. Each symptom was rated as 0 (none), 1 (mild), 2 (moderate), or 3 (severe). The mean total symptom score was calculated based on the completed form, and a comparison was made between the mean total symptom scores from the first week to the last week of the study. Patients were also asked to assess their overall quality of life, J-tube feeding requirement, and emergency room visits and hospitalizations and, in the diabetics, their impression of glucose control.

Statistical Analysis

Analysis of variance (ANOVA) and paired Student's *t* test were used to investigate the effect of electrical stimulation on gastric myoelectrical activity, gastric emptying, and gastrointestinal symptoms. All data were represented as mean ± SEM. Results were considered significant when P values were <0.05.

Results

Effect on Gastric Myoelectrical Activity

Gastric slow waves (2–4 cpm) were observed in 7 of the 9 patients in the baseline recordings. The mean frequency of the gastric slow wave in these patients was 2.9 ± 0.1 cpm (range, 2.6–3.2 cpm). Gastric dysrhythmias were identified in the other 2 patients. Electrical stimulation with a frequency of 10% higher than the intrinsic slow-wave frequency completely entrained the gastric slow wave in all 9 patients. The time required for the entrainment ranged from 1 to 10 minutes in individual subjects with a mean value of 5 minutes. A typical example illustrating the entrainment of the gastric slow wave is presented in Figure 3. Gastric dysrhythmias observed in the 2 patients were converted into 3-cpm slow waves with electrical stimulation within 10 minutes of the onset of stimulation. In comparison with sham stimulation, the amplitude of the gastric slow wave during electrical stimulation was significantly increased in the distal antrum (13.00 ± 1.83 vs. 10.71 ± 1.43 mV; $P < 0.05$), and in the proximal antrum (5.64 ± 0.73 vs. 4.79 ± 0.60 mV; $P = 0.07$).

Effect on Gastric Emptying

A significant acceleration in gastric emptying was observed at the end of the study. Before the study, the mean gastric retention at 2 hours was 77.0% ± 3.3% with the upper limit of normal being <70% at 2 hours for our test meal. All patients had a gastric retention >60% at 2 hours. After the outpatient treatment with electrical stimulation, a significant improvement in gastric retention was observed at 90 minutes (68.6% ± 7.1% vs. 86.1% ± 3.1%; $P < 0.05$, paired *t* test) and at 2 hours (56.6% ± 8.6% vs. 77.0% ± 3.3%; $P < 0.04$, paired *t* test) (Figure 4). Gastric retention at 2 hours was <70% in 6 of the 9 patients (67%) at the end of the study.

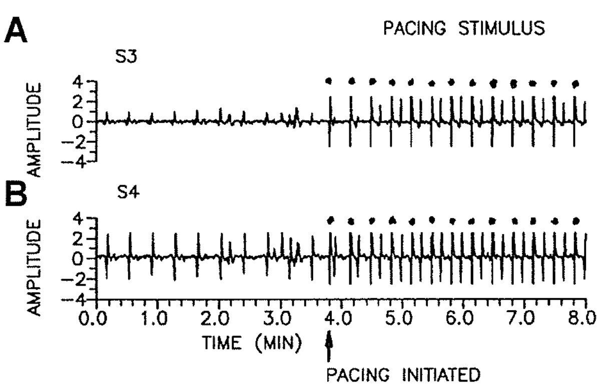

Figure 3. Gastric slow waves (*A*) before and (*B*) during gastric pacing. The dots indicate pacing stimulus. Entrainment can be appreciated from the fact that the slow waves are phase-locked with the pacing stimulus about 2 minutes after the initiation of pacing.

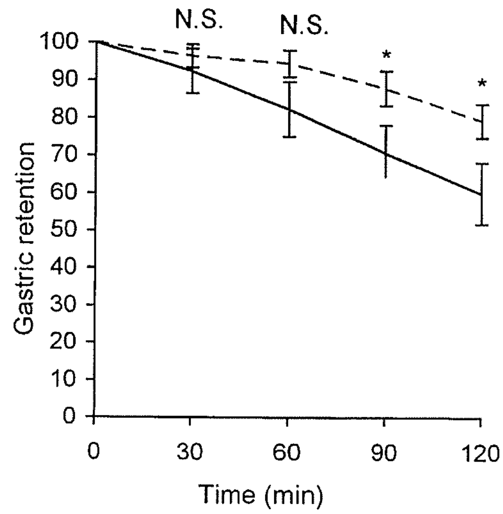

Figure 4. Effect of gastric pacing on gastric retention of a radionuclide solid meal. In comparison with the baseline gastric emptying test without pacing (------), gastric retention at 90 minutes and 2 hours was significantly reduced (*$P < 0.05$) in the gastric emptying test performed at the conclusion of long-term treatment with gastric pacing (——). N.S., not significant.

Effect on Gastroparesis Symptoms

Figure 5 presents serial symptom scores over the outpatient study period. A significant reduction in the symptoms was observed ($P < 0.005$, ANOVA). The average total symptom scores during weeks 4 (1.3 ± 0.4) and 5 (1.6 ± 0.4) were significantly lower than that during week 1 (2.8 ± 0.4) ($P < 0.05$, ANOVA). No patient required admission for symptom control during the study. The most consistent observation was not only the reduction in symptom severity but also the stability, lack of extreme symptom fluctuation, and no symptom

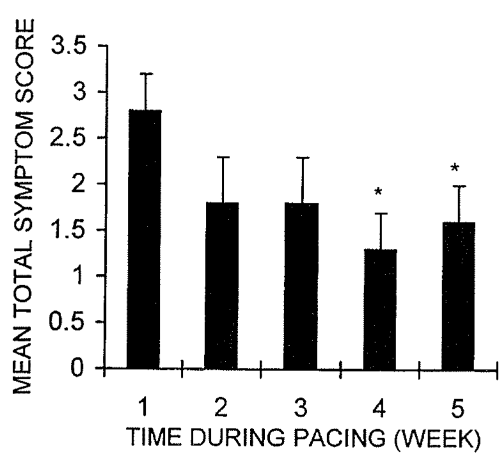

Figure 5. Serial mean total symptom scores over the outpatient study period. A significant reduction was observed during weeks 4 and 5 in comparison with week 1 (*$P < 0.05$, ANOVA).

breakthroughs, which was prevalent with prokinetics alone. The diabetic patients also reported less fluctuation in blood glucose and better day-to-day control.

At the end of the study (between 2 and 3 months of gastric pacing), 8 of the 9 patients no longer required J-tube feeding. These patients had returned to adequate oral intake and were able to take their medications orally. One patient who still required continued nutritional J-tube support was a patient with vagotomy and Billroth II who had initially fully responded to the electrical stimulation, but the electrodes connections broke and gastric pacing was not possible. The J-tube feeding then had to be continued. She subsequently underwent a total gastrectomy as the only remaining option.

The electrodes and pacing stimulus were well tolerated, and there were no infections with pacing wires. Some electrodes did become displaced and fell out during the trial, or the connection tips snapped off. Within 4 months in all cases, all electrodes were finally removed. This was easily achieved by gently tugging and pulling on the external wires, freeing the electrodes from their serosal site.

Discussion

In this study, we have systematically investigated the effect of gastric pacing on gastric myoelectrical activity, gastric emptying, and clinical profiles in patients with gastroparesis. The experimental data showed that gastric electrical stimulation was able to entrain the gastric slow wave and improve gastric emptying and clinical symptoms. Previous studies have associated delayed gastric emptying in patients with gastroparesis with impaired gastric motility and/or abnormal gastric myoelectrical activity. Our data seem to indicate that the improvement of the gastric emptying and clinical symptoms might be attributed to the entrainment and improvement of the gastric slow wave with gastric pacing. It is unknown, however, whether gastric contractility was increased with gastric pacing due to the fact that gastric motility was not simultaneously recorded in this study.

Only a few studies have previously investigated the effect of gastric pacing on gastric emptying, and their results were conflicting. Experiments in dogs with healthy stomachs showed that gastric emptying was not accelerated by gastric pacing.[13] On the other hand, the study of a canine model of gastroparesis indicated an acceleration of gastric emptying with electrical stimulation.[12] This model of gastroparesis was implemented in 5 dogs with truncal vagotomy and combined with an injection of glucagon, which induced gastric myoelectrical dysrhythmias. Studies in human subjects reported similar conflicting findings. A very limited number of patients with gastroparesis were previously studied.[15,20,21] Gastric emptying was improved in a few patients[20,21] but not in others.[15] Hocking et al.[10] investigated the effect of gastric pacing on gastric emptying in patients after gastric surgery. Eighty-eight percent of the patients had delayed gastric emptying at the baseline. Two gastric emptying studies of a solid test meal were performed in a random order on consecutive days with and without gastric pacing, and no difference was observed in the gastric emptying rates. In this current study, however, gastric emptying was significantly accelerated. It should be noted that the current study differed from previous studies reported in the literature in the following three factors: (1) the patients in the present study were all referred to one of the investigators for the treatment of severe gastroparesis, and all were refractory to standard medical therapy; (2) the parameters used for gastric pacing were optimized[11] by careful laboratory trials to establish that the entrainment of the gastric slow wave was achieved; that is, the gastric slow wave was always entrained even with an initial dysrhythmia present; and (3) a gastric emptying test was performed at the end of outpatient gastric pacing with a duration of 1 month or longer.

It is conceivable that the degree of entrainment of the gastric slow wave may be associated with the improvement of gastric emptying. Previous studies have shown that there is a higher prevalence of gastric myoelectrical abnormalities in patients with gastroparesis.[2–5] The abnormalities may include gastric dysrhythmias (the frequency of gastric slow wave is abnormally higher or lower than the normal value or irregular) and low amplitude of the gastric slow wave. Using the optimized parameters obtained from our previous study,[11] full entrainment of the gastric slow wave was achieved in all subjects. Ectopic gastric dysrhythmia were observed in 2 of the patients and normalized to regular 3-cpm slow wave with gastric pacing. This normalization was achieved within 10 minutes of starting gastric pacing. The amplitude of the gastric slow wave present in patients with gastroparesis was significantly increased during gastric pacing. It is not known whether this amplitude increase achieved the same level as could be expected in a normal subject because no data on normal subjects in a gastric pacing setting were available. Our data suggest that the mechanism that led to the improvement of gastric emptying may result from the entrainment of the gastric slow wave, normalization of gastric dysrhythmia, and enhancement of slow wave amplitude. We hypothesize that this in turn would result in the presence of stronger and better coordinated gastric contractions and hence accelerated gastric emptying.

The effect of electrical stimulation on symptoms of gastroparesis has not been clearly known. Our current study has indicated an improvement in the symptoms. This is in agreement with another recent study, in which a dramatic improvement in the symptoms of nausea and vomiting in patients with gastroparesis was revealed after 1–4 weeks of gastric pacing.[16] However, the parameters used in their study were different from our current study (pacing frequency, 12 cpm; pulse width, 300 microseconds). The electrodes used were also different, and they were implanted deeper in the gastric muscles than in our study. The effects of gastric pacing on gastric myoelectrical activity and gastric emptying were not reported in that previous study.

It should also be noted that the patients in our study experienced a greater stability in their quality of life, with no admissions required during the period of the study. The diabetic patients, in particular, reported less fluctuation of blood glucose levels and no "peaks and valleys" in their symptom levels with no subsequent breakthrough vomiting leading to admission. Most importantly, 90% of the patients were able to return to oral intake to sustain all nutritional needs. In these patients, all medications were able to be administered orally, whereas in the past the medication was given via the J-tube.

Although this study provided promising data with gastric pacing, it should be completely pointed out that a possible placebo effect could not be ruled out due to the fact that data with sham stimulation was not available.

In conclusion, gastric pacing seems to be able to improve symptoms of gastroparesis and to accelerate gastric emptying in patients with severe gastroparesis. Gastric pacing may have a future as a therapeutic option in the treatment of gastroparesis. However, further controlled studies are required to investigate the role of gastric pacing in clinical practice.

References

1. Kendall BJ, McCallum RW. Gastroparesis and the current use of prokinetic drugs. Gastroenterologist 1993;1:107–114.
2. Bortolotti M, Sarti P, Barara L, Brunelli F. Gastric myoelectrical activity in patients with chronic idiopathic gastroparesis. J Gastrointest Motil 1990;2:104–108.
3. Abell TL, Camilleri M, Hench VS, Malagelada J-R. Gastric electromechanical function and gastric emptying in diabetic gastroparesis. Eur J Gastroenterol Hepatol 1991;3:163–167.
4. Koch KL, Stern RM, Stewart WR, Vasey MW. Gastric emptying and gastric myoelectrical activity in patients with diabetic gastroparesis: effect of long-term doperidone treatment. Am J Gastroenterol 1989;84:1069–1075.
5. Chen J, McCallum RW. Gastric slow wave abnormality in gastroparetic patients. Am J Gastroenterol 1992;87:477–482.
6. McCallum RW. Clinical pharmacology forum: motility agents and the gastrointestinal tract. Am J Med Sci 1996;312:19–26.
7. Hinder RA, Kelly KA. Human gastric pacesetter potential: site of origin, spread and response to gastric transaction and proximal gastric vagotomy. Am J Surg 1978;133:29–33.
8. Miedema BW, Sarr MG, Kelly KA. Pacing the human stomach. Surg 1992;111:143–150.
9. Waterfall WE, Miller D, Ghista DN. Electrical stimulation of the human stomach (abstr). Dig Dis Sci 1985;30:799.
10. Hocking MP, Vogel SB, Sninsky CA. Human gastric myoelectrical activity and gastric emptying following gastric surgery and with pacing. Gastroenterology 1992;103:1811–1816.
11. Chen JDZ, Lin ZY, Edmunds M, Schirmer BD, Williams RD, Ross R, McCallum RW. Optimization of electrical stimulation and its effects on gastric myoelectrical activities in humans (abstr). Gastroenterology 1995;108:A582.
12. Bellahsene BE, Lind CD, Schirmer BD, Updike OL, McCallum RW. Acceleration of gastric emptying with electrical stimulation in a canine model of gastroparesis. Am J Physiol 1992;262:G826–G834.
13. Eagon JC, Kelly KA. Effect of electrical stimulation on gastric electrical activity, motility and emptying. Neurogastroenterol Motil 1995;7:39–45.
14. Hocking MP. Postoperative gastroparesis and tachygastria-response to electrical stimulation and erythromycin. Surgery 1993;114:538–542.
15. Chen J, Schirmer BD, McCallum RW. Serosal and cutaneous recordings of gastric myoelectrical activity in patients with gastroparesis. Am J Physiol 1994;266:G90–G98.
16. GEMS Study Group. Electrical stimulation for the treatment of gastroparesis: preliminary report of a multicenter international trial (abstr). Gastroenterology 1996;110:A668.
17. Hotokezaka M, Adams RB, Miller AD, McCallum RW, Schirmer BD. Laparoscopic percutaneous jejunostomy for long-term enteral access. Surg Endsosc 1996;10:1008–1011.
18. Chen JDZ, Lin ZY, Schirmer BD, Willliams RD, Ross R, McCallum RW. Long-term gastric pacing with a portable gastric pacemaker to aid gastric emptying in humans. CD-ROM proceedings of the 17th Annual International Conference on IEEE Engineering in Medicine and Biology Society, Montreal, Canada, 1995.
19. Oppenheim AV, Schafer RW. Digital signal processing. Englewood Cliffs, NJ: Prentice Hall, 1975.
20. Bellahsene BE, Schirmer B, Updike O, McCallum RW. Effect of electrical stimulation on gastric emptying (abstr). Dig Dis Sci 1987;32:902.
21. Familoni BO, Abell TL, Nemoto D, Voeller G, Salem A, Gabor O. Electrical stimulation at a frequency higher than basal rate in human stomach. Dig Dis Sci 1997;42:885–891.

Received May 14, 1997. Accepted September 9, 1997.

Address requests for reprints to: Richard W. McCallum, M.D., Department of Medicine-4035D, University of Kansas Medical Center, 3901 Rainbow Boulevard, Kansas City, Kansas 66160-7350. Fax: (913) 588-3975.

Supported partially by a research grant from Medtronic-Bakken Research Center, B.V. Maastricht, The Netherlands.

Presented in part at the annual American Gastroenterological Association meeting in San Francisco, California, May 1996, and in Washington, D.C., May 1997.

The authors thank David Teates, M.D., and the nuclear medicine physicians and assistants for their technical support of this work; gastroenterology fellows who assisted in patient evaluation and care, particularly Dr. Meade Edmunds; the nurses and the staff in the General Clinical Research Center at the University of Virginia Health Science Center, which was supported by National Institutes of Health grant M01-RR00847; and Regina Randall for her assistance in the preparation of the manuscript.