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Gastric Myoelectrical Activity in Patients With Cervical Spinal Cord Injury

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Objective: **Dyspeptic symptoms are common in patients with cervical spinal cord injury (SCI). The supraspinal control of sympathetic innervation to the stomach is interrupted in these patients. Gastric emptying has been reported to be delayed in some patients with cervical SCI. Gastric myoelectrical activity is known to regulate gastric motility and is correlated with gastric emptying. The change in gastric myoelectrical activity after cervical SCI is unknown; our aim was to investigate it.** ***Methods:*** **The study was performed in 12 cervical SCI patients and 14 healthy controls. Gastric myoelectrical activity was recorded using surface electrogastrography for 30 min in the fasting state and 1 h after a standard test meal. Spectral analysis was performed to compute the following parameters from the electrogastrogram; investigated were the percentage of 2–4 cycles/min (cpm) slow waves, the instability coefficient (IC) of the dominant frequency, the postprandial increment of dominant frequency (δF), and its power (δP).** ***Results:*** **In both fasting and fed states, regular and stable gastric slow waves were observed in both the control group and patients with cervical SCI. The percentage of normal 2–4 cpm slow waves (preprandial, 80.7 ± 3.6% *vs* 91.5 ± 3.7%, $p > 0.05$; postprandial, 82.0 ± 4.4% *vs* 87.2 ± 4.2%, $p > 0.05$) and IC (preprandial, 0.19 ± 0.04% *vs* 0.28 ± 0.05%; postprandial, 0.24 ± 0.04% *vs* 0.27 ± 0.02%, $p > 0.05$) were not significantly different between the two groups. The dominant frequency and its power were also similar between the two groups, no matter whether in the fast (frequency, 2.92 ± 0.3 *vs* 2.93 ± 0.06 cpm; power, 30.05 ± 1.29 *vs* 29.08 ± 1.23 dB, $p > 0.05$) or fed (frequency, 3.17 ± 0.07 *vs* 3.02 ± 0.06 cpm; power, 32.55 ± 0.90 *vs* 32.07 ± 1.18 dB, $p > 0.05$) state. The postprandial response measured by δF (0.25 ± 0.09 *vs* 0.09 ± 0.07 cpm, $p > 0.05$) and δP (2.52 ± 1.10 *vs* 2.24 ± 1.20 dB, $p > 0.05$) were also similar between the two groups.** ***Conclusion:*** **Gastric myoelectrical activity was not altered after cervical SCI. (Am J Gastroenterol 1998;93:2391–2396. © 1998 by Am. Coll. of Gastroenterology)**

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INTRODUCTION

Chronic gastrointestinal (GI) problems are common in patients with spinal cord injury (SCI) (1–3). These chronic symptoms may be significant enough to alter lifestyle and require chronic treatment or even hospitalization (1). Upper GI symptoms, such as abdominal bloating, acid regurgitation, and vague abdominal pain, are the common symptoms in these SCI patients. These symptoms may be secondary to the chronic constipation that is the most common complication in these patients. However, the possibility of altered upper GI motility may also contribute to the symptoms.

Gastric myoelectrical activity is mainly controlled by myogenic properties. However, evidence has also accumulated showing that extrinsic nervous control can influence gastric slow waves (4, 5). In patients with cervical SCI, the connection between the thoracolumbar sympathetic outflow and the central control is disrupted, while the vagus pathway is preserved. This could lead to an imbalance between parasympathetic and sympathetic outflow from the spinal cord to the GI tract, which might contribute to dysmotility of the upper GI tract in these patients (6). Few studies have been done to evaluate gastric motility in SCI patients. In patients with the cord injury below the T10 level, gastric emptying seems not to be affected (6–10). On the contrary, for patients with high or cervical cord injury (above T1), some reported a delayed gastric emptying (6–8), whereas others showed an unaltered gastric emptying for either liquid or solid meals (9, 10).

Gastric motility is known to be modulated by gastric myoelectrical activity, which is composed of gastric slow waves (or electrical control activity) and spike/second potentials (or electrical response activity). The gastric slow wave is omnipresent, and its frequency in humans is ~3 cycles/minute (cpm). It determines the propagation and maximal frequency of gastric contractions. The occurrence of antral contractions is directly associated with spike/second potentials that are superimposed on the slow waves (11–13).

Though still a topic for debate (14, 15), some previous studies have shown that the cutaneous electrogastrogram (EGG) is an accurate measure of the gastric slow wave

TABLE 1
Characteristics of Patients With Cervical Spinal Cord Injury

Patient No.	Level of Injury	Duration of Injury (yr)	Age (yr)	Gender	Symptoms
1	C2	1	4	F	Y
2	C6,7	25	41	M	N
3	C5,6	9	31	M	Y
4	C7	3	22	M	N
5	C5,6	28	46	M	N
6	C4,5	5	29	M	Y
7	C5	15	43	M	N
8	C5,6	18	45	M	Y
9	C4,5	11	40	M	N
10	C4,5	15	43	M	N
11	C5,6	1	36	M	N
12	C6,7	1	45	M	N

(16–19). By measuring the EGG, we can detect underlying gastric myoelectrical activity in various physiological and pathological situations. Growing evidence has suggested an association between abnormalities in gastric myoelectrical activity and gastric motor dysfunction (20, 21). Therefore, one of the possible mechanisms of the action of SCI on gastric emptying is through the disturbance of gastric myoelectrical activity. To date, there is no report investigating gastric myoelectrical activity after cervical SCI. The aim of this study was to determine whether transection of the cervical spinal cord affects the regularity, stability, and normal postprandial response of gastric myoelectrical activity using electrogastrography.

MATERIALS AND METHODS

Subjects

The study was performed in 12 patients with cervical SCI (11 men; one woman; mean age, 35.4 ± 3.6 yr) and 14 healthy and ambulatory controls (seven men, seven women; mean age, 30.7 ± 1.8 yr). The quadriplegic patients had sustained a complete cervical spinal cord injury for not less than 1 yr. The mean duration after the injury was 11.0 ± 2.7 yr, with a range of 1–28 yr (Table 1). None of the subjects had a history of previous abdominal surgery or took medications known to alter gastrointestinal transit during the 3 days before the study. The protocol was approved by the Institutional Review Board of the Baptist Medical Center of Oklahoma, and the consent form was signed by all subjects before the study.

EGG

Surface electrogastrography was applied to record gastric myoelectrical activity. Before the placement of electrodes, the abdominal skin at the recording sites was cleaned with sandy skin-prep (Omni Prep, Weaver & Co., Aurora, CO) to reduce impedance. The skin was rubbed until pinkish. The hair, if present, was shaved. Three silver-silver chloride ECG electrodes (Snap, Lombard, IL) were placed on the abdomen. One electrode (electrode 1) was placed at the midpoint between the xiphoid and the navel; one (electrode 2) was placed 5 cm to the left and 3 cm above this point; and a reference electrode (electrode 0) was placed in the lower quadrant close to the left costal margin. The bipolar EGG signal was derived from electrodes 1 and 2 and was amplified using a portable EGG recorder (Digitrapper EGG, Synectics Medical, Inc., Minneapolis, MN) with low and high cutoff frequencies of 1 and 18 cpm, respectively. Online digitization with a sampling frequency of 1 Hz was performed using an analog-digital converter installed on the recorder, and digitized samples was stored in the recorder.

Study protocol

All SCI patients and healthy subjects fasted overnight before the study. Before the EGG study, every subject was evaluated by a symptom questionnaire composed of six major dyspeptic symptoms, *i.e.*, nausea, vomiting, anorexia, early satiety, postprandial bloating, and belching. A 30-min baseline EGG recording in the fasting state was made after the placement of the electrodes on the abdomen. Then a solid meal of 450 Kcal turkey sandwich (32.4% fat, 17.5% protein, and 50.1% carbohydrate) was given and consumed within 10 min. Postprandial EGG was recorded for 1 h. All recordings were made in a quiet room with subjects in a supine position. They were asked not to talk during the procedure.

Data analysis

At the end of the study, the EGG data stored in the recorder was downloaded to an IBM 486 personal computer. All data were subjected to computerized spectral analysis using programs previously developed in our laboratory (22). The following parameters were computed from the EGG using spectral analyses.

EGG dominant frequency and power. The frequency at which the overall power spectrum of an entire EGG recording had a peak power in the range of 0.5–9.0 cpm was defined as the EGG dominant frequency. The dominant frequency of the EGG has been shown to be equal to the frequency of the gastric slow wave measured from implanted serosal electrodes (16). It was computed using the smoothed power spectral analysis method (22). The smoothed power spectral analysis was used to produce the overall power spectrum of the EGG during each recording period, *i.e.*, 30 min in the fasting state and the 60 min after the test meal. The power at the dominant frequency in the power spectrum of the EGG was defined as the EGG dominant power. Though still under discussion (14, 15), some previous studies (16, 18, 23) have shown that the relative change of the EGG dominant power reflects gastric contractility.

Percentage of normal 2–4 cpm slow waves. The percentage of normal 2–4 cpm gastric slow waves, which reflects the regularity of gastric myoelectrical activity, was defined as the percent of time during which normal 2–4 cpm slow waves were present over the entire observation period. It

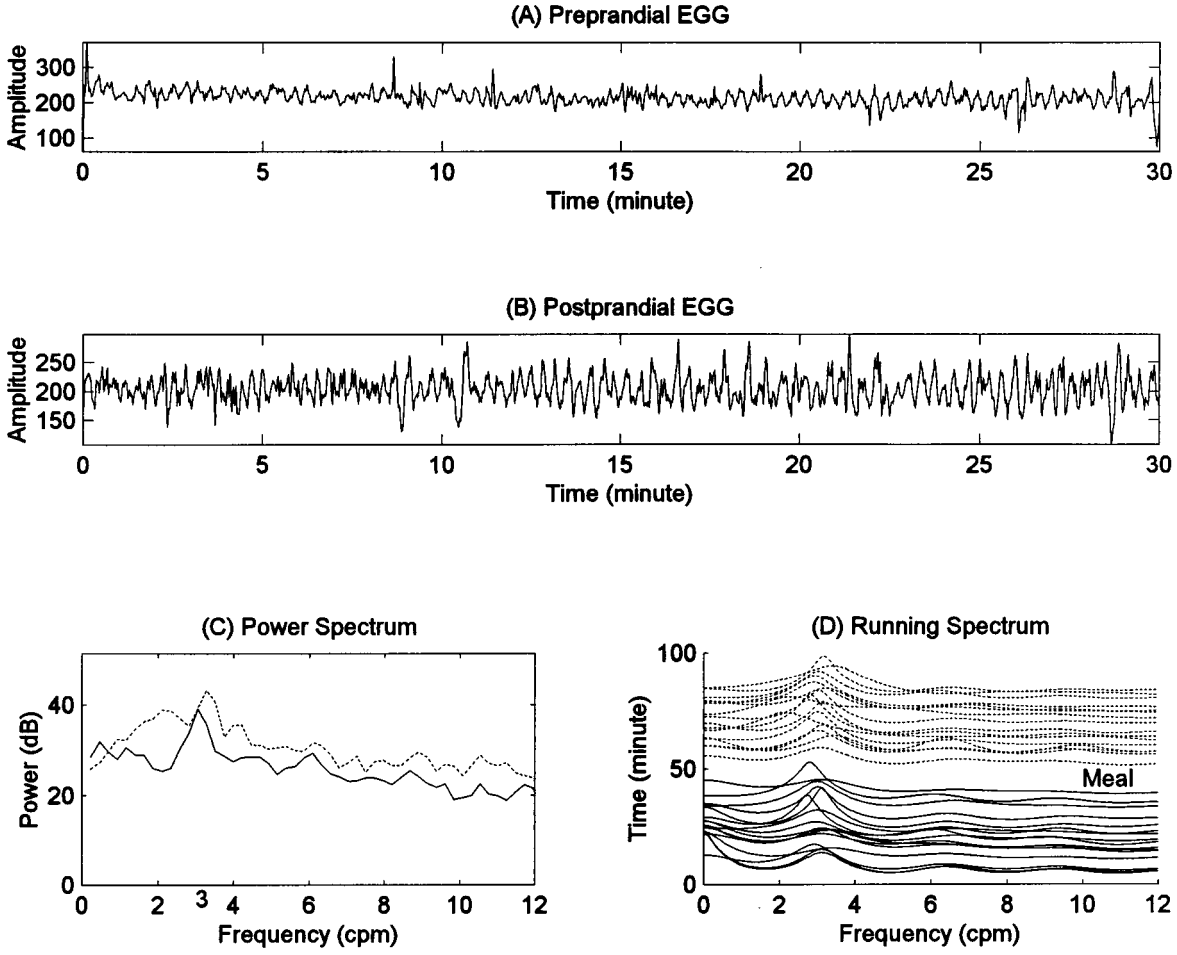

FIG. 1. *A*, Typical pre- and postprandial electrogastrographic recording in one patient with cervical cord injury; *B*, a 30-min EGG recording; *C*, Pre- (solid line) and postprandial (dotted line) power spectrum of the EGG, *D*, Pre- (solid line) and postprandial (dotted line) running power spectra. It was computed according to the adaptive spectral analysis method. Each line represents the power spectrum of 1-min of EGG data.

was computed using the adaptive running spectral analysis method (24). 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 dominant power was within the 2–4 cpm range.

Instability coefficient (IC) of the dominant frequency. The IC of the dominant frequency was defined as the ratio of the standard deviation (SD) and the mean value of the minute-by-minute EGG dominant frequency (IC = SD/mean) (25).

Postprandial increase of EGG dominant frequency (δF) and power (δP). The postprandial increase of EGG frequency or power was defined as the difference between the EGG dominant frequencies or powers after and before the test meal.

Statistical analysis

Statistical analysis was performed to compare the differences of EGG parameters between the normal controls and patients with cervical SCI using an unpaired *t* test. Statistical significance was assigned for p values of < 0.05. All data were presented as mean ± SEM.

RESULTS

Four of the 12 (33.3%) cervical SCI patients had dyspeptic symptoms, predominantly mild postprandial bloating. None of the healthy controls had dyspeptic symptoms.

Pre- and postprandial gastric slow waves were recorded in both SCI patients and normal controls. A typical example in a patient with cervical SCI is shown in Figure 1.

Regular and stable slow waves could be observed in both the control and SCI patient group, in both fasting and fed states (Table 2). The percentage of 2–4 cpm slow waves before or after the test meal was similar between the control and SCI patients (preprandial; 80.7 ± 3.6% *vs* 91.5 ± 3.7%, $p > 0.05$; postprandial; 82.0 ± 4.4% *vs* 87.2% ± 4.2%, $p > 0.05$). The pre- and postprandial IC of the dominant frequency was also not significantly different between the control and SCI patients (preprandial; 0.19 ± 0.04 *vs* 0.28 ± 0.05, $p > 0.05$; postprandial; 0.24 ± 0.04 *vs* 0.27 ± 0.02, $p > 0.05$).

TABLE 2
Pre- and Postprandial EGG Parameters in Controls and Patients With Cervical Spinal Cord Injury (SCI)

	Controls	SCI Patients	*p* Value
Preprandial			
Percentage of normal 2–4 cpm gastric slow wave (%)	80.7 ± 3.6	91.5 ± 3.7	N.S.
IC of the dominant frequency	0.19 ± 0.04	0.28 ± 0.05	N.S.
Dominant frequency (cpm)	2.92 ± 0.03	2.93 ± 0.06	N.S.
Dominant power (dB)	30.05 ± 1.29	29.08 ± 1.23	N.S.
Postprandial			
Percentage of normal 2–4 cpm gastric slow wave (%)	82.0 ± 4.4	87.2 ± 4.2	N.S.
IC of the dominant frequency	0.24 ± 0.04	0.27 ± 0.02	N.S.
Dominant frequency (cpm)	3.17 ± 0.07	3.02 ± 0.06	N.S.
Dominant power (dB)	32.55 ± 0.90	32.07 ± 1.18	N.S.
Increase in the dominant frequency (δF) (cpm)	0.25 ± 0.09	0.10 ± 0.07	N.S.
Increase in the dominant power (δP) (dB)	2.25 ± 1.10	2.24 ± 1.20	N.S.

IC = instability coefficient; N.S. = not significant.

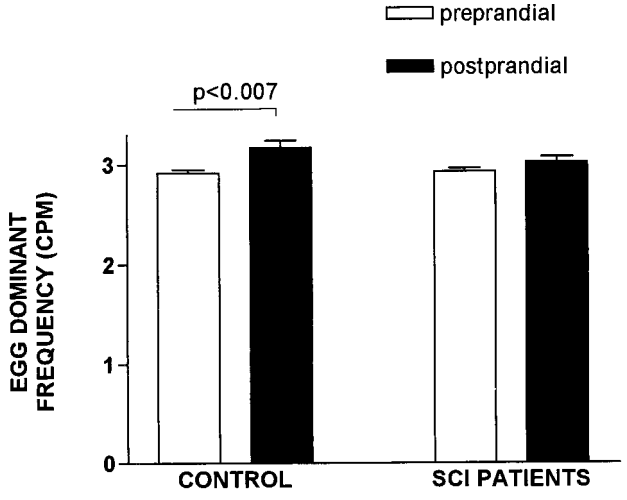

FIG. 2. Pre- and postprandial change of EGG dominant frequency in controls and patients with cervical spinal cord injury (SCI). The dominant frequency in the patient group was significantly increased after the test meal.

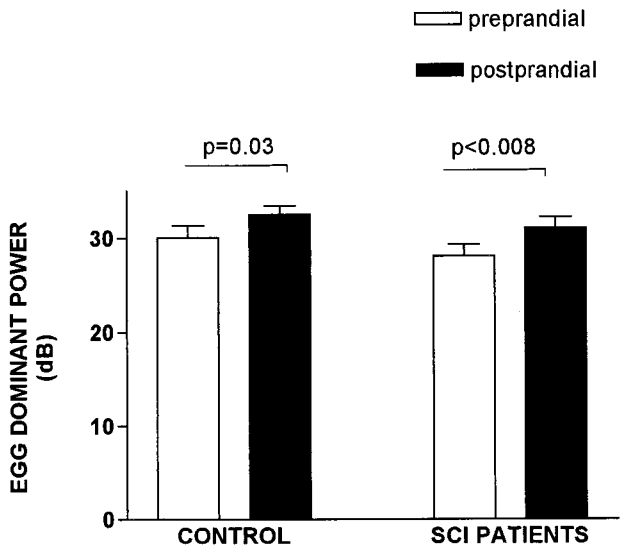

FIG. 3. Pre- and postprandial change of EGG dominant power in controls and patients with cervical spinal cord injury (SCI). The dominant power in both the control and patient groups was significantly increased after the test meal.

In the fasting state, there were no significant differences between the controls and the SCI patients in either the dominant frequency (2.92 ± 0.03 *vs* 2.93 ± 0.06 cpm, $p > 0.05$) or the dominant power (30.05 ± 1.29 *vs* 29.08 ± 1.23 dB, $p > 0.05$) (Table 2). The postprandial dominant frequency (3.17 ± 0.07 *vs* 3.02 ± 0.06 cpm, $p > 0.05$) and power (32.55 ± 0.90 *vs* 32.07 ± 1.18 dB, $p > 0.05$) were also similar between the two groups (Table 2).

The controls and patients showed similar postprandial responses in the EGG dominant frequency and power. In the control group, the dominant frequency significantly increased after the test meal (2.92 ± 0.03 to 3.17 ± 0.07 cpm, $p < 0.007$). In the patients with cervical SCI, the postprandial dominant frequency also showed a strong tendency toward a significant increase when compared with that of the baseline (2.93 ± 0.06 to 3.02 ± 0.06 cpm, $p = 0.07$) (Fig. 2). Both the controls (30.05 ± 1.29 to 32.55 ± 0.90 dB, $p = 0.03$) and the SCI patients (29.08 ± 1.23 to 32.07 ± 1.18 dB, $p < 0.008$) showed significant increases in the postprandial dominant power (Fig. 3). No significant differences were noted in the postprandial increment in the dominant frequency (δF) (0.25 ± 0.09 *vs* 0.09 ± 0.07 cpm, $p > 0.05$) and dominant power (δP) (2.52 ± 1.10 *vs* 2.24 ± 1.20 dB, $p > 0.05$) in both groups (Table 2).

The EGG signals between the symptomatic and asymptomatic SCI patients did not show any significant differences.

DISCUSSION

The present study demonstrated that the regularity, stability, and postprandial response of gastric slow waves were not changed after cervical SCI.

Gastric motility can be affected by both parasympathetic innervation of the vagus nerve (activation) and by sympa-

thetic innervation (deactivation). The cervical SCI usually does not affect the parasympathetic innervation of the stomach because the vagus nerve arises from the brainstem (26, 27). On the contrary, the preganglionic cell body of the sympathetic nervous system is located in the intermediomedial and intermediolateral cell column, arising from the thoracolumbar region of spinal cord (levels T5 through L3) (26, 27). Hence, the sympathetic input from the central control is interrupted after cervical SCI. This will lead to various clinical signs such as orthostatic hypotension, absence of thermoregulatory sweating, and a tendency for developing autonomic hyperreflexia (28, 29). GI function is also affected after cervical SCI. Both upper and lower GI symptoms, such as nausea, diarrhea, constipation, and fecal incontinence, are commonly encountered in patients with SCI (3). We demonstrated that 33.3% of the SCI patients had mild upper GI symptoms. In spite of the well-documented GI symptoms, the underlying mechanism or motility change of the GI tract after cervical SCI is still unclear.

This study showed that the EGG parameters were not changed after cervical SCI. Several previous studies have been done to evaluate the change in migrating motor complex (MMC) after SCI. Fealey *et al*. found that the duration of the phases and cycle length of the MMC in the antrum measured by manometry were not different between normal controls and the patients with cervical SCI (6). Bueno *et al*. also reported that the MMC was still present without any changes in recurrence and duration after the transection of spinal cord at the thoracic region in rats (30). Telford *et al*. further demonstrated that although the MMC cycle was immediately lost after the transection between T2 and T3 in spinal cord in dogs, the MMC (fasting or fed) returned at the eleventh day after the transection (31). These data are consistent with our findings, *i.e.*, gastric myoelectrical activity was not altered after cervical SCI. Our data also suggest that the supraspinal sympathetic pathway may not play an important role in controlling gastric myoelectrical activity.

The postprandial dominant frequency was significantly increased in the control group, but not in the patient group. The discrepancy between the two groups might be clinically insignificant because both the fasting and fed EGG dominant frequencies were similar between the two groups.

Our data showed that the EGG parameters were not changed after cervical SCI. This, however, does not necessarily support an unaltered gastric emptying in the patients with cervical SCI. Previous studies concerning gastric emptying in cervical SCI patients or in an animal model with sympathetic denervation showed conflicting results. Segal *et al*. found a delayed gastric emptying with an impairment in the drug bioavailability in patients with cervical SCI (7, 8, 32, 33), whereas Korsten and Brauman showed that gastric emptying was not altered in patients with cervical SCI (9, 10). Furthermore, Foster *et al*. observed that gastric emptying of liquids was not changed in celiac-ganglionectomized rats. This model abolished the sympathetic supply to the rat stomach and might mimic the situation of cervical SCI (34). The reason for the discrepancy among these reports had been reported to be the difference in study species, test meals, body positions, and recording techniques (8). As is well known, gastric emptying is a complicated process involving not only the myoelectrical control but also the coordination between the antrum and the duodenum, as well as various neurohormonal interactions. Fealey *et al*. reported an impairment of antral-duodenal coordination in patients with cervical SCI (6). Therefore, in the presence of normal gastric myoelectrical activity, gastric emptying could still be disturbed through disorganized antral-duodenal motor coordination or electromechanical uncoupling.

In conclusion, gastric myoelectrical activity is not altered in patients with cervical SCI.

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REFERENCES

1. Stone JM, Nino-Murcia M, Wolfe VA, et al. Chronic gastrointestinal problems in spinal cord injury patients: A prospective analysis. Am J Gastroenterol 1990;85:1114–9.
2. Cosman BC, Stone JM, Perkash I. Gastrointestinal complications of chronic spinal cord injury. J Am Paraplegia Soc 1991;14:175–81.
3. Glickman S, Kamm MA. Bowel dysfunction in spinal-cord-injury patients. Lancet 1996;347:1651–3.
4. Chen JD, Pan J, Orr WC. Role of sham feeding in postprandial changes of gastric myoelectrical activity. Dig Dis Sci 1996;41:1706–12.
5. Goldof H, van der Schee EJ, van Blankerstein M, et al. Effects of highly selective vagotomy on gastric myoelectrical activity. An electrogastrographic study. Dig Dis Sci 1990;35:969–75.
6. Fealey RD, Szurszewski JH, Merritt JL, et al. Effect of traumatic spinal cord transection on human upper gastrointestinal motility and gastric emptying. Gastroenterology 1984;87:69–75.
7. Segal JL, Milne N, Brunnemann SR, et al. Metoclopramide-induced normalization of impaired gastric emptying in spinal cord injury. Am J Gastroenterol 1987;82:1143–8.
8. Segal JL, Milne N, Brunnemann SR. Gastric emptying is impaired in patients with spinal cord injury. Am J Gastroenterol 1995;90:466–70.
9. Zang RL, Chayes Z, Korsten MA, et al. Gastric emptying rates to liquid or solid meals appear to be unaffected by spinal cord injury. Am J Gastroenterol 1994;89:1856–8.
10. Rajendran SK, Reiser JR, Bauman W, et al. Gastrointestinal transit after spinal cord injury: Effect of cisapride. Am J Gastroenterol 1992; 87:1614–7.
11. Hinder RA, Kelly KA. Human gastric pacesetter potential. Site of origin, spread and response to gastric transection and proximal gastric vagotomy. Am J Surg 1978;133:29–33.
12. Kelly KA, Code CF, Elveback LR. Patterns of canine gastric electrical activity. Am J Physiol 1969;217:461–70.
13. Sarna SK. Gastrointestinal electrical activity; terminology. Gastroenterology 1975;68:1631–5.
14. Mintchev MP, Kingma YJ, Bowes KL. Accuracy of cutaneous recordings of gastric myoelectrical activity. Gastroenterology 1993;104:1273–80.
15. Brown BH, Smallwood RH, Duthie HC, Stoddard CJ. Intestinal smooth muscle electrical potentials recorded from surface electrodes. Med Biol Eng Comput 1975;13:97–102.
16. Smout AJPM, van der Schee EJ, Grashuis JL. What is measured in electrogastrography? Dig Dis Sci 1980;25:179–87.

17. Abell TL, Malagelada J-R. Glucagon-evoked gastric dysrhythmias in humans shown by an improved electrogastrographic technique. Gastroenterology 1985;88:1932–40.
18. Hamilton JW, Bellahsene B, Reichelderfer M, et al. Human electrogastrograms: Comparison of surface and mucosal recordings. Dig Dis Sci 1986;31:33–9.
19. Familoni BO, Bowes KL, Kingma YJ, et al. Can transcutaneous recordings detect gastric electrical abnormalities? Gut 1991;32:141–6.
20. Kohagen KR, Kim MS, McDonnel WM, et al. Nicotine effects on prostaglandin-dependent gastric slow wave rhythmicity and antral motility in nonsmokers and smokers. Gastroenterology 1996;110:3–11.
21. Hasler WL, Soudah HC, Dulai G, et al. Mediation of hyperglycemic-evoked gastric slow-wave dysrhythmias by endogenous prostaglandin. Gastroenterology 1995;108:727–36.
22. Chen J. A computerized data analysis system for electrogastrogram. Comp Biol Med 1992;22:45–8.
23. Chen J, Schirmer BD, McCallum RW. Serosal and cutaneous recordings of gastric myoelectrical activity in patients with gastroparesis. Am J Physiol 1994;266:G90–8.
24. Chen J, Stewart WR, McCallum RW. Spectral analysis of episode rhythmic variations in the cutaneous electrogastrogram. IEEE Trans Biomed Eng 1993;40:128–35.
25. Chen JDZ, McCallum RW. Electrogastrographic parameters and their clinical significance. In: Chen JDZ, McCallum RW, eds. Electrogastrography: Principles and applications. New York: Raven Press, 1994: 45–73.
26. Dagradi RM. The gastrointestinal system in patients with spinal cord injury. Proceedings of the Annual Clinical Spinal Cord Injury Conference 1953;2:6–14.
27. Pollock LJ, Boshes B, Chor H, et al. Defects in regulatory mechanisms of autonomic function in injuries to spinal cord. J Neurophysiol 1951; 14:85–93.
28. Corbett JL, Frankel HL, Harris PJ. Cardiovascular reflex responses to cutaneous and visceral stimuli in spinal man. J Physiol (Lond) 1971; 215:395–409.
29. Mathias CJ, Christensen NJ, Corbett JL, et al. Plasma catecolamines during paroxysmal neurogenic hypertension in quadriplegic man. Cir Res 1976;39:204–8.
30. Bueno L, Ferre J-P, Ruckebusch Y. Effects of anesthesia and surgical procedures on intestinal myoelectrical activity in rats. Dig Dis 1978; 23:690–5.
31. Telford GL, Go VL, Szurszewski JH. Effect of central sympathectomy on gastric and small intestinal myoelectric activity and plasma motilin concentrations in the dogs. Gastroenterology 1985;89:989–95.
32. Segal JL, Brunnemann SR, Gordon SK, et al. Decreased theophylline bioavailability and impaired gastric emptying in spinal cord injury. Curr Ther Res 1985;38:831–46.
33. Segal JL, Brunnemann SR. Clinical pharmacokinetics in patients with spinal cord injuries. Clin Pharmacokinet 1989;17:109–29.
34. Foster ER, Green T, Dockray GJ. Efferent pathways in the reflex control of gastric emptying in rats. Am J Physiol 1991;260:G499–504.