*Digestive Diseases and Sciences, Vol. 47, No. 8 (August 2002), pp. 1737–1745 (© 2002)*

# Changes in Gastric Myoelectric Activity During Space Flight

DEBORAH L. HARM, PhD,* GWENN R. SANDOZ, BA,† and ROBERT M. STERN, PhD‡

The purpose of the present study was to examine postprandial myoelectric activity of the stomach and gastric activity associated with space motion sickness using electrogastrography. Three crewmembers participated in this investigation. Preflight, subjects exhibited normal postprandial responses to the ingestion of a meal. Inflight, crewmembers exhibited an abnormal decrease in the power of the normal gastric slow wave after eating on flight day 1, but had a normal postprandial response by flight day 3. Prior to and during episodes of nausea and vomiting, the electrical activity of the stomach became dysrhythmic with 60–80% of the spectral power in the bradygastric and tachygastric frequency ranges. These findings indicate that gastric motility may be decreased during the first few days of space flight. In addition, changes in the frequency of the gastric slow wave associated with space motion sickness symptoms are consistent with those reported for laboratory-induced motion sickness.

**KEY WORDS:** electrogastrogram; space motion sickness; postprandial responses.

Space motion sickness (SMS) is the most clinically significant phenomenon occurring during the first few days of space flight. Symptoms of SMS have been reported by 48% of the cosmonauts in the Soviet space program (1). In 44 flights of the US space shuttle (through January 1992), the incidence of SMS was 73% in first-time flyers (2). Gastrointestinal symptoms have their onset from minutes to hours after orbital insertion. Symptom resolution usually occurs between 30 and 48 hr, with a reported range of 12–72 hr (3), although a very small percentage of crewmembers do not recover or become symptom-free for the duration of the flight. Russian researchers report that 54% of the cosmonauts have symptoms lasting 1–3 days, 25% have symptoms lasting 14 days or longer, and 17% (8 of 46 cosmonauts) on long-duration missions (85–365 days) periodically develop vertigo and queasiness, especially during the last 10–14 days of the mission when their activity level typically increases (4).

Even in the absence of stomach discomfort, nausea, and/or vomiting, crewmembers almost universally experience loss of appetite. Similar symptoms often accompany a variety of gastrointestinal disorders. Reduced gastric motility has long been recognized as a characteristic of acute motion sickness (5). In terrestrial studies of motion sickness, gastric emptying decreases (6) and mouth-to-cecal transit time increases (7) profoundly in motion-sick subjects. In addition, gastric dysrhythmias have been observed during a wide variety of laboratory-induced motion sickness stimulus conditions (8–14). In patients, a decreased postprandial power of the normal gastric slow wave has been associated with delayed gastric emptying (measured by scintigraphy) (15–19). Gastric dysrhythmias have also been observed in patients with delayed gastric emptying (20), gastroparesis (21–23), nausea and vomiting (18, 21, 24), and dyspepsia (18, 25–27).

Manuscript received August 29, 2001; revised manuscript received February 25, 2002; accepted February 28, 2002.

From the *Neurosciences Laboratory, NASA Johnson Space Center, Houston, Texas 77058; †Krug Life Sciences, Houston, Texas 77058; and ‡Department of Psychology, Pennsylvania State University, University Park, Pennsylvania 16802.

This work was supported by the NASA-Mir Phase I project.

Address for reprint requests: Deborah L. Harm, Mail Code SK3, NASA Johnson Space Center, 2101 Nasa Rd. 1, Houston, Texas 77058.

To date, gastrointestinal symptoms that occur during space flight have been measured almost exclusively by self-report, with only three exceptions. Decreased bowel sounds during the first few days of space flight, as measured by auscultation, have been reported (14, 28). In those subjects, bowel sounds returned after symptom resolution. Russian researchers have observed a decrease in electrogastrogram (EGG) amplitude and a decrease in the EGG response to ingestion of solids and liquids in monkeys during space flight (29). Most techniques used in clinical evaluation and diagnosis of gastrointestinal disorders are impractical for use in space flight (scintigraphy, serosal electrodes, and intraluminal transducers). Electrogastrography, however, is a noninvasive technique for recording gastric myoelectric activity using cutaneous electrodes. EGG may be a useful objective indicator of potential changes in postprandial gastric myoelectric activity and the development of space motion sickness symptoms.

In man, gastric smooth muscle pacesetter potentials oscillate around a frequency of 3 cycles per minute (cpm), or 0.05 Hz, with amplitudes of 100–500 $\mu$V; pacesetter potentials are propagated across the stomach from the antrum toward the pylorus. Gastric myoelectric activity is responsible for coordinating stomach contractions and regulating gastric emptying and is mediated by the autonomic nervous system. Increased vagal (parasympathetic) activity augments gastric motility (30), whereas increased sympathetic activity or vagal withdrawal decreases gastric motility (31, 32).

Simultaneous recordings of serosal or mucosal and cutaneous myoelectric activity have established that the EGG accurately records the gastric slow wave that controls the frequency and propagation of gastric contractions (22, 33–36). Gastric dysrhythmias, including tachygastria (fast frequency waves) and bradygastrias (slow frequency waves), also can be detected by EGG (37). EGG is becoming a useful clinical tool in evaluating gastric myoelectric abnormalities (rhythm and/or amplitude) associated with gastrointestinal functional disorders such as gastroparesis and functional dyspepsia and, therefore, may be useful in evaluating these same abnormalities in SMS.

The purpose of this investigation was to: (1) assess changes in gastric myoelectric responses to ingestion of food over the first few days of space flight, and (2) examine gastric myoelectric activity during space motion sickness symptoms, as well as the temporal relations between gastric activity and subjective reports of symptoms.

## MATERIALS AND METHODS

### Subjects

Three male crewmembers participated in this investigation. All subjects met the rigorous physical and health requirements for maintaining flight status and provided written informed consent to participate in the study. It was required that all crewmembers refrain from the consumption of alcohol or drugs for the 24 hr preceding any test session. Use of anti-motion sickness drugs, either prophylactically or at the time of motion sickness, was acceptable, but not desirable. Since some medications can affect gastric rhythms, gastric amplitude, and/or autonomic balance, the use of any medication, regardless of purpose, was recorded. This information included the name of the drug, the dosage and route of administration, the time at which it was taken, and any symptoms at that time.

### Procedures

**Apparatus.** Electrogastrographic data were recorded using field effect transistor (FET) type electrodes and a Digital Biolog (UFI, Morro Bay, California, USA), an ambulatory recording instrument developed for use during space flight. This unit used FETrodes to amplify the EGG signal at the source, thus greatly increasing the signal-to-noise ratio. The main portion of the unit provided additional signal amplification (with a frequency range of the lower 3 dB = 0.01 Hz and the upper 3 dB = 0.24 Hz), sampled the signal at 10 Hz, and stored the data on a static RAM card. Twenty-four hours of data could be stored on a 1 Mb RAM card. The lightweight unit (0.6 kg) was worn on a fabric belt and powered by a standard 9V battery. Event markers on the recording instrumentation (coded 1–5) were used to mark the occurrence of an event (defined in this experiment as eating, drinking, stomach awareness/discomfort, nausea, and vomiting) by inserting the code number of the event pressed into the data stream along with the associated mission elapsed time (MET). The insertion of event markers was accomplished without a suspension in data logging.

Three electrodes were placed on the crewmember's abdomen: one along the left midclavicular line 1–2 in. below the costochondral margin, one at the midpoint between the xiphoid process and the umbilicus, and the third (ground) on the rib cage of the crewmember's right side. If abdominal hair was present on the electrode sites, those areas were shaved. Prior to electrode placement, the skin was lightly abraded using Omniprep and a protective barrier (Mentor Shield Skin) was applied. Electrode impedance was checked after application. An impedance greater than 10 k$\Omega$ necessitated replacement of the electrode. For sessions conducted on the ground, signal output was verified on an oscilloscope.

**Preflight Training and Baseline Data Collection.** Two preflight training and two baseline data collection (BDC) sessions were required of each subject for this experiment. The first training session, held on or about L-12 months (12 months prior to launch), included a science orientation and introduction of the inflight procedures. Subjects were instructed on where and how to place the electrodes, proper skin preparation, electrode impedance testing, and use of the event markers. Practice of the procedures was also

included. The second training session (L-10 months) included review and practice of the inflight procedures and training in malfunction procedures.

The two BDC sessions were used to establish subject responses to a gastric fluid load (session 1) and ingestion of a small test meal (session 2). Only the responses to the small test meal are presented here. These data were used for comparison with inflight data corresponding to eating. For data consistency, subjects were asked to fast (no food or drink) for 6.5 hr prior to the data collection session. Additionally, the subjects were instructed to refrain from consuming alcohol and taking any medications during the 24 hr preceding the session.

The test meal BDC began with a fasted baseline recording of 20 min, after which the subject ingested a typical inflight breakfast. The subjects were queried prior to the session as to the appropriate contents of this meal. The meal was eaten within a 5 to 10-min period. Data recording continued for 3 hr after the meal.

Within 8 days prior to launch, a 10-min session to mark proper electrode placement was required. The subjects were instructed to arrive with a full stomach to allow accurate localization of the stomach. The locations for the electrodes were marked on the abdomen of the subject with a Sharpie indelible marker (Sanford, Bellwood, Illinois, USA). This was done to ensure proper and consistent placement of the electrodes inflight.

**Inflight Data Collection.** The Biolog equipment was donned as early in the inflight timeline as possible. Data were collected continuously for at least the first three flight days. The electrodes, RAM card, and battery were replaced every 24 hr during this time. For these change-outs, alcohol swabs were used in place of Omniprep to avoid overabrasion of the skin, and the protective barrier was reapplied prior to the placement of fresh electrodes. Use of the barrier reduced the possibility of skin irritation from the electrodes. The crewmembers were instructed to press the event markers whenever appropriate to record the occurrence of eating, drinking, stomach awareness/discomfort, nausea, or vomiting.

## Data Reduction and Analysis

Data from the Biolog RAM cards was downloaded to a PC via a Databook TMD-500 Thin Card Drive (Databook, Inc., Ithaca, New York, USA), converted from binary to ASCII format using software developed by UFI, and transferred to a Macintosh computer running MATLAB (The MathWorks, Inc., Natick, Massachusetts, USA), a numeric computation program with signal processing capabilities. The data were then calibrated and divided into smaller segments on the basis of event markers.

For EGG analysis, a fourth-order high-pass digital elliptical filter with a 0.015-Hz cutoff was first applied to the data followed by a fourth-order low-pass elliptical filter with a 0.5-Hz cutoff. Segments of data corresponding to periods of interest (eg, reports of SMS symptoms and eating) and without movement artifact were identified for running spectral analysis. Each segment was subdivided into smaller, overlapping segments 2048 points (3.4 min) in length. Each of these segments overlapped the previous one by 75%, resulting in 512 points (51.2 secs) of new data analyzed with each segment. Each segment was linearly detrended and Hamming-windowed prior to computation of the fast Fourier transform (FFT). FFT is based on the Fourier series and assumes that all complex wave forms are composed of a series of sinusoidal waves. FFT analyzes the complex wave form and provides a power spectrum of the signal indicating the relative strengths of various frequency components. The power data obtained provides information on both frequency and amplitude of the EGG signal. There are a number of EGG parameters that can be computed. In this paper we will report on two parameters that are of particular clinical significance. The change in postprandial power of the normal gastric slow wave frequency (3 cpm) was used to examine and compare gastric responses to eating pre- and inflight. The percent of the spectral power in the normal, bradygastric, and tachygastric frequency ranges was used to examine gastric dysrhythmias associated with the nausea and vomiting that accompanies SMS.

## Definition and Computation of EGG Parameters

**Postprandial Change in Normal 3-cpm Power.** The relative change in the power of the normal gastric slow wave frequency (3 cpm) is the difference between the powers (in dB) before and after eating. An increase in the postprandial normal power primarily reflects an increase in gastric contractile activity. A decrease in the normal power, however, is definitely considered an abnormal response to eating and is indicative of postprandial gastric motility disorders (38). The power in the normal slow-wave frequency range was averaged across the segments of the running spectrum for the 15 min prior to eating and for each of the first two 15-min periods following a meal. The preprandial spectral power was then subtracted from each of the two 15-min postprandial powers to compute the change in normal 3-cpm power.

**Percent Distribution of Spectral Power.** The percent spectral power (PSP) assesses the regularity of the gastric slow waves. It is the percent of time that normal gastric slow waves are observed in the EGG, and the percent of time that bradygastric and tachygastric rhythms are observed. First, the power in each of the normal (2.34–3.78 cpm), bradygastria (0.9–2.04 cpm) and tachygastria (4.08–9.6 cpm) ranges was computed for each segment by summing the powers of the frequencies within each band. Then, the average power of the segments within 15 min periods before and after reports of nausea or vomiting was calculated for each frequency range. The total power was then calculated by summing the powers across all three frequency ranges. Percent distribution of spectral power was determined by calculating the ratio of power in each frequency range to the total power. The PSP of normal gastric slow waves is particularly useful in studies designed to examine the effects of exogenous stimulation on the regularity of gastric slow waves (38).

# RESULTS

## Postprandial Responses

Figure 1 shows the average change in postprandial power of normal 3 cpm activity for the three astro-

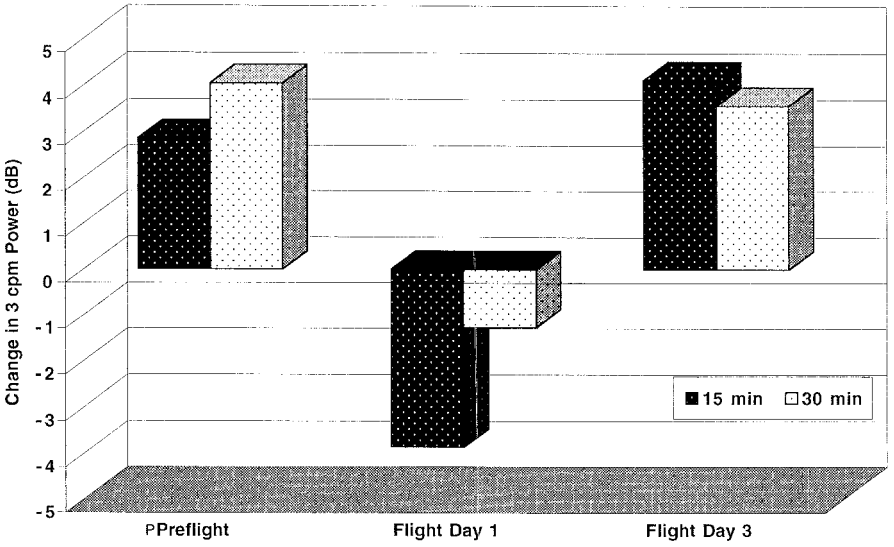


**Fig 1.** The average change in postprandial spectral power of the normal (3 cpm) gastric slow wave for the three astronauts (the difference in power before and after eating). This shows a normal increase in postprandial power preflight, an abnormal decrease on flight day 1 with a return to a normal response by flight day 3.

nauts preflight, on flight day one (FD1), and flight day three (FD3). Preflight, subjects exhibited normal postprandial responses to ingestion of a meal, that is, an increase in the amplitude and power of the normal 3 cpm slow wave. Inflight, there was an abnormal decrease in the dominant power on FD1 with a return to a normal response on FD3. There were, however, individual differences in postprandial responses, as shown in Figure 2. Preflight, one subject (A2) exhibited an abnormal decrease in postprandial dominant power 15 min after the meal, which was still evident 30 min after eating. Although there is no clear explanation for this response, the crewmember may have been experiencing unreported gastric symptoms on the day of testing. On FD1, all three astronauts showed an abnormal decrease in postprandial power 15 min after the meal, and two of the three astronauts continued to show this response 30 min after the meal. By FD3, all three crewmembers exhibited a normal increase in postprandial dominant power at both 15 and 30 min after the meal. All three crewmembers reported moderate to severe loss of appetite on FD1 and two of the crewmembers experienced mild loss of appetite on FD3. The fact that A2 showed a normal postprandial response on FD3 suggests that his abnormal preflight response was probably due to an acute, temporary problem rather than a chronic gastric motility disorder.

**Nausea and Vomiting Responses**

One crewmember reported two episodes of nausea, and another crewmember reported one episode of vomiting. The top two panels of Figure 3 (A and B) show the percent distribution of spectral power (PSP) in each of the EGG frequency ranges before and after nausea was reported. In the first episode of nausea (panel A), approximately 60% of the spectral power was associated with dysrhythmic activity before nausea was reported, 40% bradygastria and 20% tachygastria. The PSP associated with dysrythmic activity increased to approximately 75% during the 15 min following the report of nausea, then gradually decreased to about 45% at 45 min following the initial report. Preflight, the subjects' fasted and postprandial PSP in the normogastria range was 15% and 67%, respectively. In the second report of nausea (panel B), the PSP associated with dysrhythmic activity remains high at 75–80% throughout the recording period. Since we do not know the duration of the nausea, we can only speculate that the data may reflect a more rapid recovery of nausea in the first episode compared to the second episode. A second point of comparison between the two nausea episodes is the difference in PSP associated with bradygastria versus tachygastria. In the first episode, the PSP associated with bradygastria is two to three times greater than that of tachygastria throughout the re-

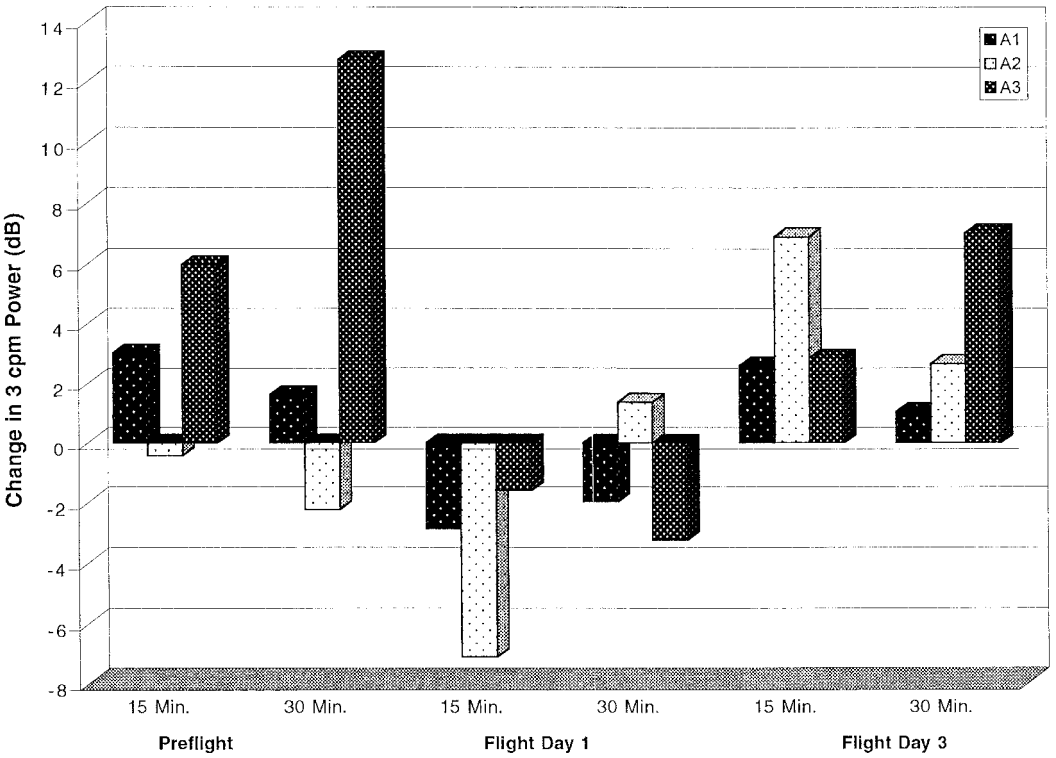


**Fig 2.** The change in postprandial spectral power of the normal (3 cpm) gastric slow wave for individual astronauts. Astronaut 2 showed an abnormal decrease in postprandial power preflight.

cording period. However, in the second episode, generally a higher PSP is associated with tachygastria during the periods following the report of nausea. In other studies, tachygastria is more commonly observed during motion sickness than bradygastria (10–12).

Figure 3C shows the PSP for the vomiting episode. Like the nausea episodes, 75–85% of the spectral power is associated with dysrhythmic activity before and up to 30 min following emesis. Forty-five minutes after vomiting the gastric myoelectric activity is essentially normal, with approximately 70% of the power in the normal gastric slow wave frequency range. The crewmembers' preflight fasted and postprandial PSP in the normogastria range was 16% and 39%, respectively.

One final observation is that for both the nausea episodes and the vomiting episode, the crewmembers exhibited clear dysrhythmic activity at least 30 min prior to reporting the symptom. It should also be noted that the crewmember who vomited specifically stated that he had no symptoms prior to vomiting. Sudden vomiting without prodromal symptoms is a fairly common characteristic of SMS (39).

Figures 4 and 5 depict the running spectra of one of the nausea episodes and the vomiting episode, respectively. Both figures show the running spectra of the EGG for 30 min before and after the nausea/vomiting episode. The darker gray spectra represent the EGG before the event and the lighter gray spectra represent the EGG following the report of nausea or vomiting. Figure 4 shows dysrhythmic activity prior to nausea is dominated by bradygastria, whereas tachygastria dominates the period following the report of nausea. The increased tachygastria observed during nausea is consistent with EGG responses observed during terrestrial motion sickness.

The vomiting episode (Figure 5) is marked by high-amplitude, severe dysrhythmic activity prior to emesis, where the spectral power is fairly equally distributed across bradygastria and tachygastria. Immediately following emesis, there is a rather dramatic decrease in the EGG amplitude across all frequencies. Finally, at about 20 min after vomiting we begin to see a recovery of the normal (3 cpm) gastric slow wave.

## DISCUSSION AND CONCLUSIONS

The present study represents the first attempt to obtain objective measures of gastric myoelectric ac-

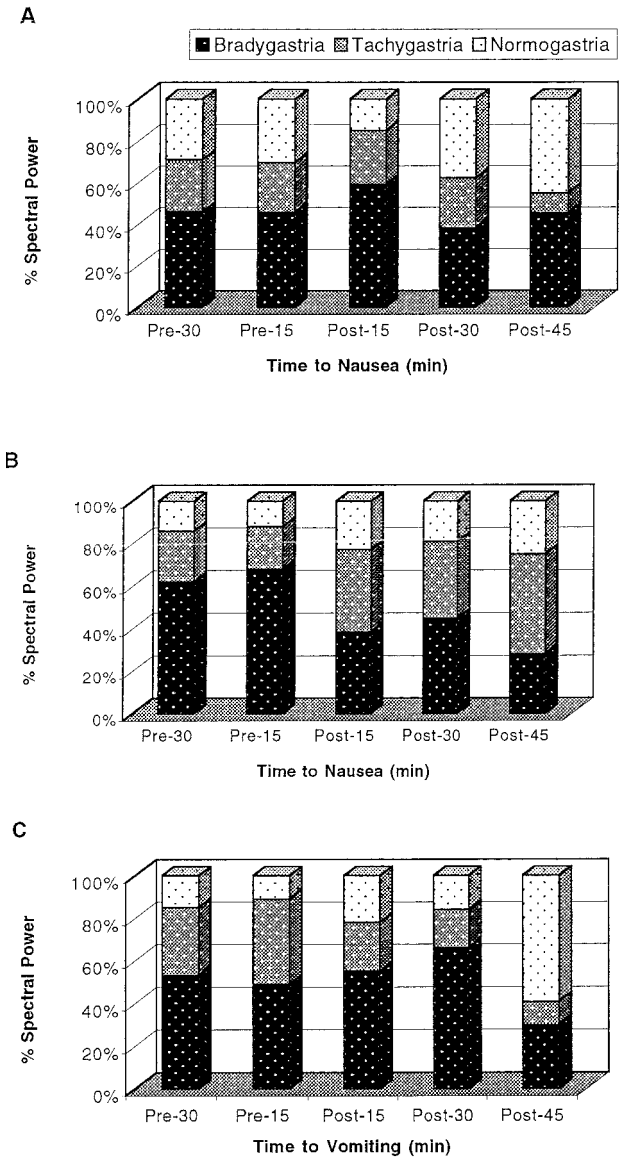


**Fig 3.** The percent distribution of spectral power for the three frequency ranges of the gastric myoelectric activity: bradygastria (0.9–2.04 cpm), tachygastria (4.08–9.6 cpm), and normogastria (2.34–3.78 cpm). (A and B) The percent distribution of spectral power before and after two episodes of nausea in the same crewmember. (C) The percent spectral power before and after a vomiting episode experienced by a different crewmember.

tivity in humans during spaceflight. Crewmembers exhibited abnormal postprandial decreases in the spectral power of the normal gastric slow wave. This observation is consistent with patients who exhibit delayed gastric emptying (15–19). Following recovery of appetite and resolution of SMS symptoms by FD3, postprandial gastric myoelectric responses returned to normal. Dysrhythmic gastric myoelectric activity concomitant with episodes of nausea and vomiting was also clearly observed. Decreased gastric emptying (6) and increased mouth-to-cecal transit time (7) occur in motion-sick subjects on earth. Moreover, gastric hypomotility and the absence of gastric contractions have been observed in association with tachygastria (34). The relationship between gastric motility and bradygastria is somewhat less clear. However, some investigators have observed antral hypomotility (40) and the absence of antral contractions (41) in association with bradygastria. These findings strongly suggest that gastric motility may be decreased during the first few days of space flight, both when symptoms are as minor as loss of appetite and during more significant symptoms of nausea and vomiting.

Disruption of gastric motility has implications for nutrient absorption and for administration of oral medications, both in clinical settings and in the space flight environment. The physically and mentally demanding conditions of space flight make adequate nutrition particularly important for work performance. A variety of physiologic changes occur in microgravity that often require pharmacologic intervention (eg, nasal congestion, back pain, and SMS). Decreased gastrointestinal motility could adversely affect drug absorption and bioavailability of orally ingested medications, potentially reducing their effectiveness. It is, therefore, important to understand changes in gastrointestinal function during space flight.

This investigation demonstrated the feasibility and potential value of using a noninvasive technique for recording gastric myoelectric activity in active astronauts over long periods of time. Clearly, additional space flight research is needed to confirm these preliminary findings. Further work is needed to characterize the temporal relations between the onset of gastric dysrhythmic activity and subjective reports of nausea and/or vomiting (particularly without prodromal symptoms). The current technology for ambulatory recording of the EGG is adequate for research aimed at improving our understanding of changes in gastric function during space flight, but it is not adequate for use as a medical monitoring tool. In order for this technology to be a useful monitoring tool that can aid flight surgeons in pharmacologic treatment decisions (concerning route of drug administration) or provide an early warning of SMS symptom development, it needs to be capable of real or near real-time data analysis.

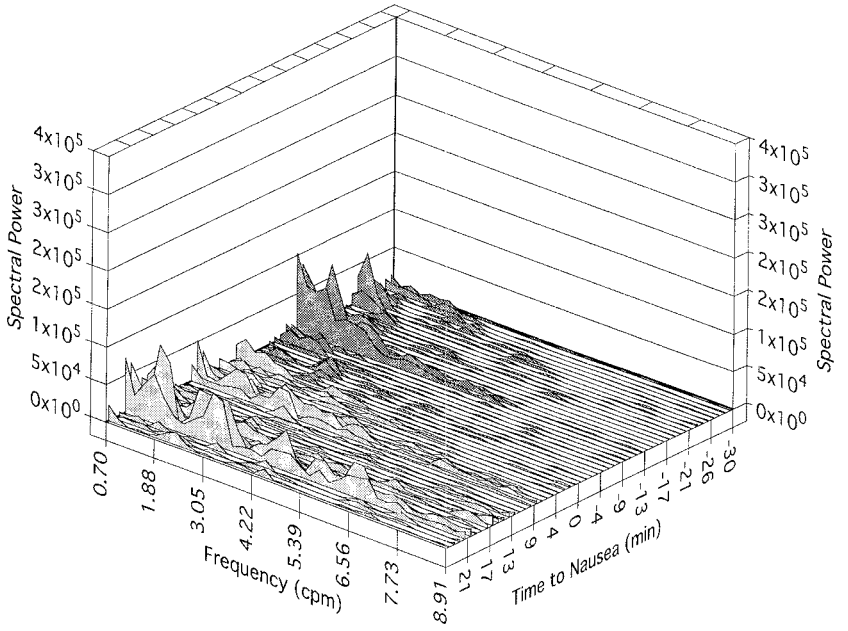


**Fig 4.** The running spectra of the EGG for one of the nausea episodes for 30 min before and 30 min following the report of nausea; negative values on the *x* axis (time to nausea) are the minutes prior to nausea and the positive values are the minutes following nausea. Each segment of the spectra overlaps the previous one by 75%, resulting in 51.2 sec of new data in each segment.

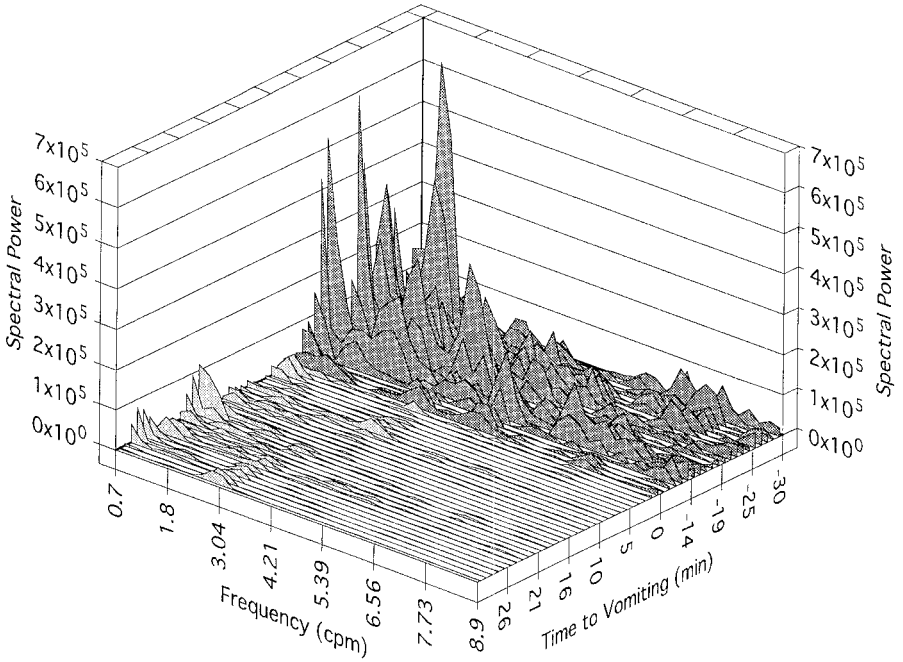


**Fig 5.** The running spectra of the EGG for the vomiting episode for 30 min before and 30 min following the report of nausea; negative values on the *y* axis (time to vomiting) are the minutes prior to vomiting and the positive values are the minutes following nausea. Each segment of the spectra overlaps the previous one by 75%, resulting in 51.2 sec of new data in each segment.

## ACKNOWLEDGMENTS

The authors wish to express their sincere appreciation to the three crewmembers who participated in this study and to thank them for their dedicated efforts. In addition, we would like to thank Dr. Jiande Chen for his very helpful comments and recommendations during the preparation of this manuscript.

## REFERENCES

1. Gorgiladze GI, Bryanov II: Space motion sickness. Kosm Biol Aviakosmicheskaya Med 23:4–14, 1989
2. Jennings RT: Managing space motion sickness. J Vestib Res 8:67–70, 1998
3. Thornton WE, Moore TP, Pool SL, Vanderploeg JM: Clinical characterization and etiology of space motion sickness. Aviat Space Environ Med 58:A1–A8, 1987
4. Kornilova LN, Muller KH, Chernobylskiy LM: Phenomenology of illusory reactions in weightlessness. Fiziol Chel 21:50–62, 1995
5. Reason JT, Brand JJ: Motion Sickness. London, Academic Press, 1975
6. Wood MJ, Wood CD, Manno JE, Manno BR, Redetzki HM: Nuclear medicine evaluation of motion sickness and medications on gastric empting time. Aviat Space Environ Med 58:1112–1114, 1987
7. Muth ER, Stern RM, Koch KL: Effects of vection-induced motion sickness on gastric myoelectric activity and oral-cecal transit time. Dig Dis Sci 41:330–334, 1996
8. Harm DL, Beatty BJ, Reschke MF: Transcutaneous oxygen as a measure of pallor. Aviat Space Environ Med 58:508, 1987 abstract
9. Harm DL, Parker DE: Preflight adaptation training for spatial orientation and space motion sickness. J of Clin Pharmacol 34:618–627, 1994
10. Holmes SR, Griffin MJ: Gastric myoelectric activity during low frequency oscillation. Aviat Space Environ Med 71:420–425, 2000
11. Hu S, McChesney KA, Player KA, Bahl AM, Buchanan JB, Scozzafava JE: Systematic investigation of physiological correlates of motion sickness induced by viewing an optokinetic rotating drum. Aviat Space Environ Med 70:759–765, 1999
12. Stern RM, Koch KL, Leibowitz HW, Lindblad IM, Shupert CL, Stewart WR: Tachygastria and motion sickness. Aviat Space Environ Med 56:1074–1077, 1985
13. Stern RM, Koch KL, Stewart WR, Lindbald IM: Spectral analysis of tachygastria recording during motion sickness. Gastroenterology 92:92–97, 1987
14. Thornton WE, Linder BJ, Moore TP, Pool SL: Gastrointestinal motility in space motion sickness. Aviat Space Environ Med 58:A16–A21, 1987
15. Chen J, Stewart WR, McCallum RW, Richards RD, Davenport KG, Abbitt PL: Gastric slow wave abnormality in gastroparetic patients. Am J Gastroenterol 85:1243, 1990
16. Chen JD, Lin Z, McCallum RW: Noninvasive feature-based detection of delayed gastric emptying in humans using neural networks. IEEE Trans Biomed Eng 47:409–412, 2000
17. 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
18. Soykan I, Lin Z, Jones S, Chen J, McCallum RW: Gastric myoelectrical activity, gastric emptying and correlations with dyspepsia symptoms in patients with gastroesophageal reflux. J Invest Med 45:483–487, 1997
19. Soykan I, Lin Z, Sarosiek I, McCallum RW: Gastric myoelectrical activity, gastric emptying, and correlations with symptoms and fasting blood glucose levels in diabetic patients. Am J Med Sci 317:226–231, 1999
20. Abell TL, Camilleri M, Hench VS, Malagelada JR: Gastric electromechanical function and gastric emptying in diabetic gastroparesis. Eur J Gastroenterol Hepatol 3:163–167, 1991
21. Koch KL, Bingaman S, Sperry N, Stern RM: Electrogastrography differentiates mechanical vs idiopathic gastroparesis in patients with nausea and vomiting. Gastroenterology 100:A99, 1991
22. Lin Z, Chen JD, Schirmer BD, McCallum RW: Postprandial response of gastric slow waves: Correlation of serosal recordings with the electrogastrogram. Dig Dis Sci 45:645–651, 2000
23. Mayaudon H, Bauduceau B, Dupuy O, Ceccaldi B, Farret O, Molinie C: [Diabetic autonomic gastric neuropathy: contribution of the electrogastrogram]. Presse Med 28:577–579, 1999
24. You CH, Lee KY, Chey WY, Nenguy R: Electrogastrographic study of patients with unexplained nausea, bloating and vomiting. Gastroenterology 79:311–314, 1980
25. Jebbink HJ, Bruijs PP, Bravenboer B, Akkermans LM, van-Berge-Henegouwen GP, Smout AJ: Gastric myoelectrical activity in patients with type I diabetes mellitus and autonomic neuropathy. Dig Dis Sci 39:2376–2383, 1994
26. Leahy A, Besherdas K, Clayman C, Mason I, Epstein O: Abnormalities of the electrogastrogram in functional gastrointestinal disorders. Am J Gastroenterol 94:1023–1028, 1999
27. Lin X, Levanon D, Chen JD: Impaired postprandial gastric slow waves in patients with functional dyspepsia. Dig Dis Sci 43:1678–1684, 1998
28. Harris BA, Billica RD, Bishop SL, Blackwell T, Layne CS, Harm DL, Sandoz GR, Rosenow EC: Physical examination during space flight. Mayo Clin Proc 72:301–308, 1997
29. Badakva A: Personal Communication 1994.
30. Fujii K, Mukai M: Neurohumoral mechanisms of excitation of gastric motility in the dog. *In* Gastrointestinal Function, Regulation and Disturbances. MTY Kasuya, F Nagao, T Matsuo (eds). Amsterdam, Excerpta Medica, 1985, pp 15–27
31. Diamant NE, Hall K, Mui H, El-Sharkawy TY: Vagal control of the feeding-motor pattern in the lower esophageal sphincter stomach, and small intestine of dog. *In* Gastrointestinal Motility. J Christensen (ed). New York, Raven Press, 1980, pp 365–370
32. Hall KE, El-Sharkawy TY, Diamant NE: Vagal control of canine postprandial upper-gastrointestinal motility. Am J Physiol 250:G501–G510, 1986
33. Atanassova E, Daskalov I, Dotsinsky I, Christov I, Atanassova A: Non-invasive electrogastrography. Part 1: Correlation between the gastric electrical activity in dogs with implanted and cutaneous electrodes. Arch Physiol Biochem 103:431–435, 1995
34. Chen J, McCallum RW, Richards RD: Frequency components of the electrogastrogram and their correlations with gastrointestinal contractions in humans. Med Biol Eng Comput 31:60–67, 1993

35. Chen JD, Schirmer BD, McCallum RW: Serosal and cutaneous recordings of gastric myoelectrical activity in patients with gastroparesis. Am J Physiol 266:G90–G98, 1994
36. Liang J, Chen JD: What can be measured from surface electrogastrography. Computer simulations. Dig Dis Sci 42:1331–1343, 1997
37. Familoni BO, Abell TL, Bowes KL: A model of gastric electrical activity in health and disease. IEEE Trans Biomed Eng 42:647–657, 1995
38. Chen JZ, McCallum RW: Electrogastrography—Principles and Applications. New York, Raven Press, 1994, p 431
39. Reschke MF, Harm DL, Parker DE, Sandoz GR, Homick JL, Vanderploeg JM:. Neurophysiologic aspects: Space motion sickness. *In* Space Physiology and Medicine, 3rd ed. CLHAE Nicogossian, SL Pool (eds). Philadelphia, Lea and Febiger, 1994, pp 228–260
40. Bortolotti M, Sarti P, Barara L, Brunelli F: Gastric myoelectric activity in patients with chronic idiopathic gastroparesis. J of Gastrointest Motil 2:104–108, 1990
41. Abell TL, Malagelada JR: Glucagon-evoked gastric dysrhythmias in humans shown by an improved electrogastrographic technique. Gastroenterology 88:1932–1940, 1985