Protein meals reduce nausea and gastric slow wave dysrhythmic activity in first trimester pregnancy

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Jednak, Michelle A., Elizabeth M. Shadigian, Michael S. Kim, Michelle L. Woods, Forrest G. Hooper, Chung Owyang, and William L. Hasler. Protein meals reduce nausea and gastric slow wave dysrhythmic activity in first trimester pregnancy. *Am. J. Physiol.* 277 (*Gastrointest. Liver Physiol.* 40): G855–G861, 1999.—First trimester nausea is associated with gastric slow wave dysrhythmias (tachygastria, bradygastria). We tested the roles of meal composition and caloric content on nausea and slow wave rhythm in 14 nauseated pregnant women. Electrogastrography quantified dysrhythmic activity and signal power responses to meals. Symptomatic women reported mild to moderate nausea and exhibited increased dysrhythmias during fasting ($P < 0.05$). Protein-predominant meals reduced nausea and dysrhythmic activity to greater degrees than equicaloric carbohydrate and fat meals and noncaloric meals ($P < 0.05$). Meal consistency did not affect symptom responses, although liquid meals decreased dysrhythmias more than solids ($P < 0.05$). Carbohydrates and fats increased electrogastrographic power to similar degrees as proteins, whereas responses to noncaloric meals were less. In conclusion, protein meals selectively reduce nausea and gastric slow wave dysrhythmias in first trimester pregnancy. Meal consistency is a limited factor in the favorable effects of protein. Electrogastrographic power changes do not explain the symptom response to protein. Thus dietary modulation of gastric myoelectric rhythm with protein supplementation may provide symptomatic benefit in nausea of pregnancy.

electrophysiology; gastrointestinal motility; electrogastrography; gastric emptying

NAUSEA AND VOMITING affect 50–80% of women during the first trimester of pregnancy and produce significant loss of productivity as well as impaired health status (8–10, 16, 18). Medications and cutaneous stimulation techniques (acupressure, acupuncture) are advocated for treatment by some clinicians, but their efficacy is unproven in many instances (1, 5, 12, 20, 24). Because of concerns about adverse medication reactions for the fetus, management of first trimester nausea has relied on modification of dietary intake to provide foods that reduce symptoms (21). Obstetricians commonly recommend ingestion of solid carbohydrate meals, such as soda crackers, based primarily on anecdotal reports (2, 7, 13, 20). However, more recent investigations indicate that protein meals may be more beneficial for symptom control and that carbohydrates and fats either have no effect or worsen nausea (6, 15, 23, 29).

The pathophysiology of nausea in pregnancy is poorly understood, in part because most technologies for evaluating gastrointestinal motility are invasive or expose the fetus to ionizing radiation. The technique of electrogastrography noninvasively measures gastric myoelectric activity (27). In healthy individuals, the stomach exhibits rhythmic electrical depolarizations (slow waves) that control gastric motor function. In some diseases, nausea may be associated with altered gastric motility and disturbances in slow wave rhythm in which cycling is too rapid (tachygastria) or too slow (bradygastria) (11, 17, 26, 28). Furthermore, in normal individuals, the electrogastrographic amplitude or power increases after eating; an absence of this increase correlates with delays in solid phase gastric emptying (3, 27). A pathogenic role for slow wave disturbances in generating nausea is suggested by studies that show that dysrhythmias begin before nausea is experienced, are correctable by treatments that reduce symptoms, and can be normalized by gastric electrical pacing (4, 14, 22, 26).

Previous investigations have shown that women with first trimester nausea exhibit slow wave dysrhythmias that resolve after delivery and that can be mimicked by exogenous progesterone and estrogen administration to nonpregnant women (19, 30). In contrast, asymptomatic pregnant women show no rhythm disruptions (25). The acute effects of different dietary components and their liquid vs. solid consistency on nausea and slow wave rhythm in first trimester pregnancy have not been rigorously explored.

The aim of this controlled investigation was to determine which meal characteristics offer the greatest reduction in symptoms in women with first trimester nausea and whether these meals have specific effects on electrogastrographic parameters. We examined whether meals consisting predominantly of protein, carbohydrate, or fat selectively reduce nausea and dysrhythmic activity or modify the power responses. We then compared the effects of solid and liquid nutrient meals to determine whether meal consistency affects symptom and slow wave responses to eating. Finally, to assess whether the caloric content is important, nausea and slow wave activity were measured after ingestion of noncaloric liquid and solid meals. Through these studies we hoped to gain insight into dietary modulation of symptoms and gastric myoelectric activity in patients with nausea of first trimester pregnancy.

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MATERIALS AND METHODS

Study Population

Fourteen pregnant women (20–35 yr old) with first trimester nausea were referred from the Obstetrics and Gynecology Outpatient Clinic at the University of Michigan Medical Center. All women were enrolled in the 7th–12th wk of gestation and had experienced nausea either intermittently or continuously for at least 2 wk before investigation and within 48 h of study enrollment. None of the women had symptoms severe enough to require intravenous fluid administration or inpatient hospitalization. None had a prior history of chronic gastrointestinal illness or gastrointestinal surgery, and none were on medications known to alter gastric motor or myoelectric activity or produce nausea. All studies were approved by the University of Michigan Institutional Review Board. Informed consent was obtained from all subjects before their participation in the study.

Study Design

All studies were performed after overnight fasting. Electrogastrography was performed according to the method of Stern and colleagues (27). Subjects were placed supine in a quiet warm room without visual or auditory distractions. After gentle skin abrasion to enhance electrical conduction, Ag-AgCl electrodes (Accutac diaphoretic electrocardiogram electrodes, NDM, Dayton, OH) were affixed to the abdomen. The first electrode was placed in the mid-clavicular line below the left costal margin. The third electrode was placed midway between the xiphoid and the umbilicus. The second electrode was placed equidistant between the first and third electrodes. A fourth reference electrode was affixed in the right upper abdominal quadrant. Electrodes were connected via direct nystagmus couplers (model 9859, SensorMedic, Anaheim, CA) to a chart recorder for continuous display of slow wave activity. Time constants were set at 10 s and high frequency cutoffs at 0.3 Hz to minimize interference from nongastric signals.

Before initiation of electrogastrographic recording, a 15-min equilibration period was provided to ensure a stable fasting tracing. Fasting recording was performed for 15 min, after which each pregnant woman ate a standardized test meal prepared by the University of Michigan Clinical Research Center. Five minutes were allotted for ingestion of each meal. After completion of the meal, postprandial electrogastrographic recording was performed for 45 min. Each subject recorded her level of nausea with an integral scale from 0 (no nausea) to 10 (severe nausea with vomiting) every 10 min throughout the study (twice during fasting and five times postprandially).

Subjects presented on separate days for each test meal and completed all meal ingestions within 2 wk of study entry. Meals were ingested in random order, and all studies were completed by the 14th gestational week. The order of meal ingestion was provided by the dietician service in the Clinical Research Center and was different for each subject. The 400-kcal caloric meals were formulated to be single-nutrient-predominant for protein, fat, or carbohydrate and to be palatable (Table 1). Liquid and solid meals that were protein-, carbohydrate-, or fat-predominant were given to determine if the physical consistency of the caloric meal affected symptom and electrogastrographic responses to nutrient ingestion. Noncaloric solid and liquid meals of similar volumes assessed the importance of caloric supplementation in reducing nausea and slow wave dysrhythmias in first trimester pregnancy.

Table 1. *Test meal composition for pregnant women with nausea*

Meal	Composition	Caloric Breakdown
Liquid protein	Protein powder,* whipping cream, chocolate syrup, water	400 kcal (53% protein, 13% carbohydrate, 34% fat)
Solid protein	Egg white, turkey white meat, and ham omelet with white bread	400 kcal (53% protein, 12% carbohydrate, 35% fat)
Liquid carbohydrate	Lemonade drink powder, sugar, water	400 kcal (100% carbohydrate)
Solid carbohydrate	Gum drops candy	400 kcal (98% carbohydrate, 2% fat)
Liquid fat	Heavy whipping cream, vanilla ice cream, whole milk	400 kcal (7% protein, 20% carbohydrate, 73% fat)
Solid fat	Cream cheese on saltine crackers	400 kcal (7% protein, 20% carbohydrate, 73% fat)
Liquid noncaloric	Water	0 kcal
Solid noncaloric	Sugar-free gelatin	0 kcal

*ProMod protein supplement, Ross Products Division, Abbott Laboratories, Columbus, OH.

Data Analysis

Interpretation of symptom and electrogastrographic data was performed only from those studies in which fasting or postprandial nausea was reported by the pregnant volunteers. Electrogastrographic recordings from the excluded asymptomatic studies showed no increase in dysrhythmic activity and were not different from those in studies of healthy nonpregnant women in the same laboratory that used identical data analysis protocols, similar to findings previously reported (25). Because of this exclusion, data presented for each test meal do not include results from all 14 pregnant volunteers. However, each nutrient test meal is represented by at least 8 recordings in which nausea was reported. Specifically, eight women ingested the liquid protein, liquid carbohydrate, solid carbohydrate, solid fat, and inert meals, whereas nine women ingested the solid protein and liquid fat meals.

Nausea scoring. Nausea scores under fasting and postprandial conditions were calculated for pregnant women with first trimester nausea. The fasting nausea score was calculated as the mean of two reports at 10-min intervals. Postprandial nausea at 5, 15, 25, 35, and 45 min after meal ingestion was plotted as the net change (increase or decrease) in the recorded nausea score compared with fasting.

Electrogastrographic analysis. Electrogastrographic recordings from the three leads were analyzed visually to determine which provided the signal most free of motion artifact and respiratory interference. This lead selection and all subsequent electrogastrographic analyses were performed in a blinded fashion, so that the investigator did not know the identity of the volunteer or the test meal condition being studied. This signal was digitized at 4 Hz by the analog-to-digital converter and filtered above 15 cycles per minute (cpm) and below 0.5 cpm to remove high- and low-frequency noise. Commercially available software (Fourier Perspectives III, Alligator Technologies, Fountain Valley, CA) was em-

ployed to perform power spectral analysis on 4-min recording segments in the fasting and postprandial periods. A running spectral analysis plot was generated across the frequency range of 0.5–9 cpm at 2-min intervals, so that each successive line in the pseudo-three-dimensional plot represented the mean amplitudes at the different frequencies of the 4-min recording segments acquired every 120 s in overlapping fashion. Data from the power spectral analyses were converted to spreadsheet format (Lotus 1–2–3 2.0, Lotus Development, Cambridge, MA) to quantify gastric slow wave disturbances.

Power spectra were divided into bradygastric (≥0.75 cpm and <2 cpm), normal (≥2 cpm and <4.5 cpm), and tachygastric (≥4.5 cpm and <9 cpm) frequency ranges. Signal powers in each frequency band were summed in 0.25-cpm increments, divided by the sum of the signal powers from 0.5 to 9 cpm, then multiplied by 100 to give a percent value. Bradygastric, normal, and tachygastric activities were thus expressed as a percentage of total signal power. A dysrhythmic index was calculated by summing the bradygastric and tachygastric signal powers. Under normal conditions, >50% of the signal power resides in the normal frequency range (2–4.5 cpm). Because electrogastrographic signals do not exhibit pure sinusoidal morphology and because each tracing exhibits some baseline electrical variation and noise, some power is present in both the bradygastric and tachygastric frequency ranges in health. With development of dysrhythmias, bradygastric or tachygastric signal powers increase, with a corresponding decrease in power in the normal frequency range. This is associated with visible loss of normal 3-cpm activity on the raw tracing with replacement with waveforms of low or high frequency, respectively (27). The fasting period was divided into two equal time blocks to correlate with the times fasting nausea was assessed. A fasting dysrhythmic index was calculated as the mean of the two dysrhythmic indexes calculated for these time blocks. The postprandial period was divided into five equal time blocks to correlate with the times that nausea scores were reported. Dysrhythmic activity for each time block after meal ingestion was plotted as the net change (increase or decrease) in dysrhythmic index compared with the mean fasting level.

A second electrogastrographic parameter, the change in signal power after meal ingestion, also was measured in the pregnant subjects. In health, electrogastrographic power in the normal frequency range increases soon after eating; this increase in power persists for the time that food remains in the stomach (27). Delays in solid phase gastric emptying have been correlated by some investigators with loss of this physiological postprandial power increase (3). The postprandial period was divided into five equal time blocks to correlate with the times that nausea scores were reported. The power value in the normal frequency range from 2 to 4.5 cpm for each postprandial time block was calculated as a function of recording time and divided by the fasting power value, also calculated as a function of recording time. Thus the signal power for each postprandial time block was expressed as a multiple of the fasting level. A value <1 was considered to be abnormal.

Statistical Analysis

All results are expressed as means ± SE. Repeated measures ANOVA was performed on the nausea scores, dysrhythmic indexes, and postprandial power increases to compare responses to meal consistencies and nutrient subclasses and to test if there were significant interactions between meal consistency and time after eating and between nutrient subclass and time after eating. Repeated measures ANOVA determined that there was no effect of the order of meal ingestion on nausea scores, dysrhythmic indexes, or power increases. Repeated measures ANOVA was performed separately within each meal consistency and nutrient subclass to estimate the magnitudes of the nutrient effects. When the main effect of nutrient subclass was significant within an analysis, the Newman-Keuls multiple range test was employed to identify differences between nutrients. Statistical significance was accepted at a P value of <0.05.

RESULTS

Effects of Test Meals on Symptoms

The mean fasting nausea score during all studies of pregnant women was 2.8 ± 0.3. Including only those sessions in which fasting symptoms were recorded, the mean fasting nausea score was 3.5 ± 0.3, representing mild to moderate nausea. Meal ingestion produced time-dependent reductions in nausea ($F = 5.73$, $P = 0.0002$). Both liquid ($F = 3.70$, $P = 0.01$) and solid ($F = 2.82$, $P = 0.03$) meals reduced nausea scores in time-dependent fashion, with maximal reductions in nausea score of 1.0 ± 0.3 for each consistency at 45 min after ingestion (Fig. 1*A*). Comparison of the effects of meal consistency on postprandial changes in nausea scores revealed no differences in responses to liquid and solid meals ($F = 0.01$, $P = 0.91$). In contrast, postprandial changes in nausea scores exhibited a significant dependence on nutrient subclass (protein, carbohydrate, fat, or noncaloric; $F = 3.39$, $P = 0.02$; Fig. 1*B*). Analysis of differences between nutrient subclasses detected a significantly greater reduction in postprandial nausea with protein ingestion compared with meals of other subclasses ($P < 0.05$). Protein ingestion produced significant time-dependent reductions in nausea ($F = 9.21$, $P = 0.0009$) with maximal decreases in nausea score of 2.0 ± 0.2 at 45 min, whereas time-dependent decreases in nausea score did not reach statistical significance for carbohydrate, fat, and noncaloric meals. There were no differences in symptom responses to caloric carbohydrate and fat meals and noncaloric meals ($P > 0.05$).

Gastric Slow Wave Responses to Meals

Meal effects on slow wave rhythm. During fasting, pregnant women with nausea exhibited dysrhythmic indexes of 61 ± 2%. This was significantly greater than fasting dysrhythmic indexes from a group of young, healthy, asymptomatic, nonpregnant women measured with an identical protocol (30). In contrast to healthy volunteers who show 3-cpm waveforms on visual inspection of electrogastrographic tracings, nauseated women in first trimester pregnancy exhibited prolonged periods of abnormal slow wave activity with slow (bradygastric) or rapid (tachygastric) rhythms interspersed with normal 3-cpm activity.

Measures of dysrhythmic activity were calculated in a time-dependent fashion to correlate with nausea score findings. As with nausea scores, meal ingestion produced time-dependent reductions in dysrhythmic indexes ($F = 10.08$, $P = 0.0001$). Both liquid ($F = 6.58$, $P = 0.0004$) and solid ($F = 3.39$, $P = 0.02$) meals produced improvements in dysrhythmic activity, with

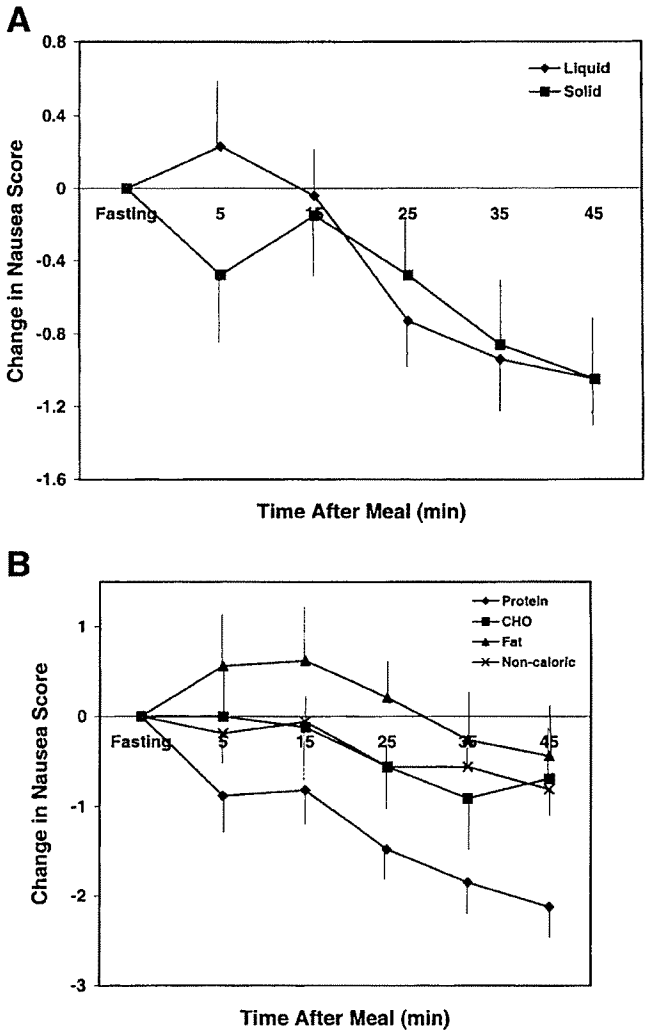

Fig. 1. Effects of test meal ingestion on reports of nausea by women in first trimester pregnancy are shown. *A*: meals of both liquid and solid consistency produced similar reductions in nausea. *B*: protein-predominant meals reduced nausea scores compared with fasting levels and to greater degrees than carbohydrate (CHO), fat, and noncaloric meals ($P < 0.05$). Responses to protein meals became evident within 25 min of meal ingestion. Values are means ± SE.

maximal 11 ± 3% and 6 ± 3% decreases in dysrhythmic index at 25 min after eating, respectively (Fig. 2*A*). In contrast to the nausea scoring, decreases in dysrhythmic index were greater with liquid than with solid meals ($F = 5.39$, $P = 0.02$). As with the nausea reports, postprandial changes in dysrhythmic index exhibited a significant dependence on nutrient subclass ($F = 5.06$, $P = 0.003$; Fig. 2*B*). Proteins ($F = 8.94$, $P = 0.001$), fats ($F = 6.26$, $P = 0.004$), and noncaloric meals ($F = 4.02$, $P = 0.03$) produced time-dependent changes in dysrhythmic index, whereas carbohydrates evoked no change in dysrhythmias. Analysis of differences between nutrient subclasses detected a significantly greater reduction in postprandial dysrhythmic indexes with protein ingestion compared with meals of other subclasses ($P < 0.05$), with a maximal 19 ± 3% decrease at 45 min after eating. Analysis between nutrient subclasses detected no differences in dysrhythmic index responses to caloric carbohydrate and fat meals and noncaloric meals ($P > 0.05$). Visual analysis of electrogastrographic tracings before and after protein ingestion showed normalization of the aberrant slow wave activity to 3-cpm waveforms that were most evident in the final 30 min of the postprandial recording periods (Fig. 3). In contrast, visual analysis of electrogastrographic tracings after ingestion of meals of other nutrient subclass predominance showed persistence of abnormal slow wave activity, with periods of bradygastric and tachygastric activity interspersed between periods of normal 3-cpm waveforms (Fig. 4).

Meal effects on electrogastrographic power. Increases in electrogastrographic signal power after test meal ingestion were quantified to correlate with nausea

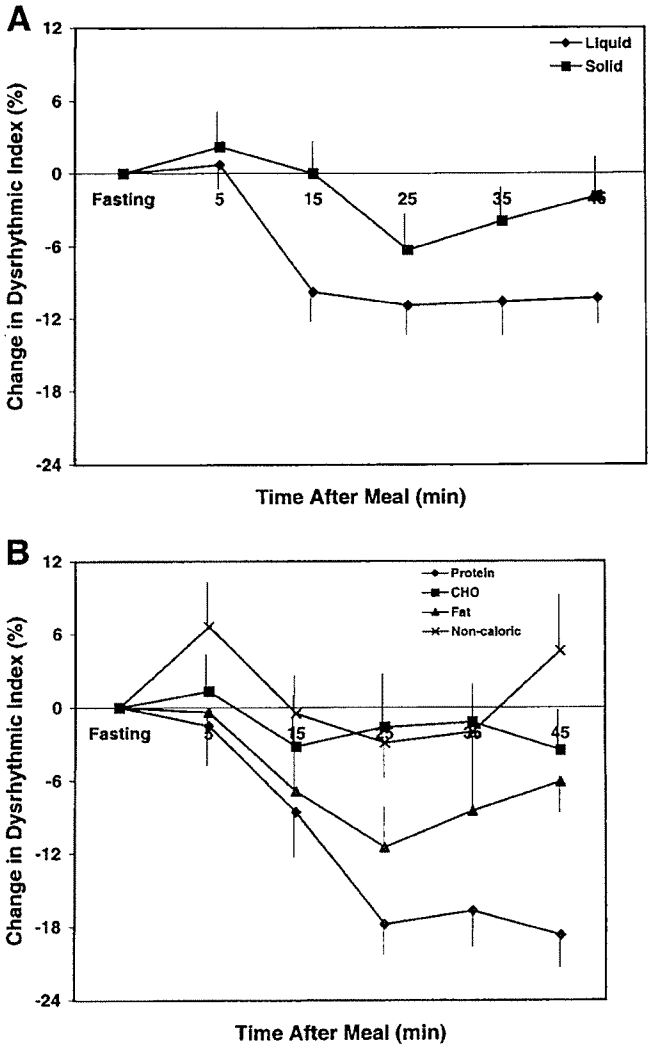

Fig. 2. Effects of meal ingestion on slow wave dysrhythmic activity in women with first trimester nausea are shown. *A*: meals of liquid consistency reduced dysrhythmic indexes to greater degrees than solids ($P < 0.05$). *B*: protein-predominant meals reduced dysrhythmic indexes compared with fasting levels and to greater degrees than carbohydrate, fat, and noncaloric meals ($P < 0.05$). Responses to proteins were evident within 25 min of meal ingestion. Values are means ± SE.

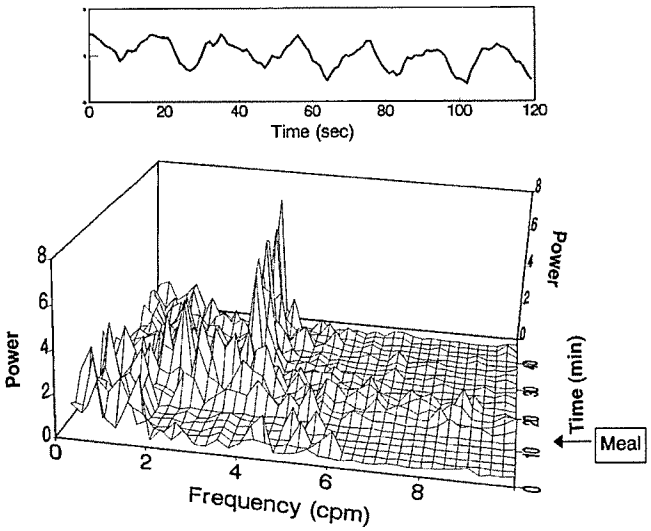

Fig. 3. A representative raw electrogastrographic tracing (*top*) and spectral analysis plot (*bottom*) are shown for a pregnant woman with nausea before and after ingestion of a liquid protein-predominant meal. Postprandial raw signal exhibits a normal-appearing near-sinusoidal oscillation with a period of ~20 s. Spectral analysis reveals extensive dysrhythmic activity during fasting with peaks in both bradygastric and tachygastric frequency ranges. After meal ingestion, signal stabilizes with development of normal 3-cycles-per-minute (cpm) activity.

scoring reports. Time-dependent increases in power were observed after meal ingestion ($F = 11.02$, $P = 0.0001$). Both liquid ($F = 9.35$, $P = 0.0002$) and solid ($F = 7.64$, $P = 0.0006$) meals increased electrogastrographic power (Fig. 5*A*). In contrast to nausea scores and dysrhythmic indexes, maximal increases in power were observed within 5 min of eating. There was a

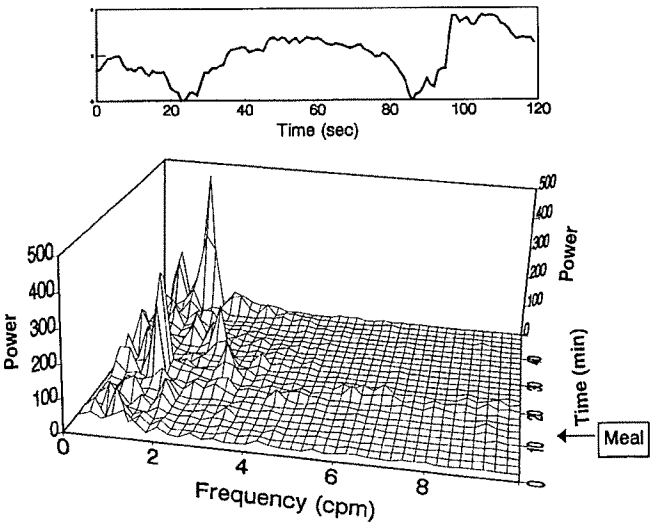

Fig. 4. A representative electrogastrographic tracing (*top*) and spectral analysis plot (*bottom*) are shown for a pregnant woman with nausea before and after ingestion of a liquid carbohydrate-predominant meal. Postprandial raw signal exhibits a low amplitude chaotic signal with no clear single dominant frequency. Spectral analysis shows a lack of a single predominant frequency in fasting and postprandial periods. Rather, there is increased signal power in bradygastric and tachygastric frequency ranges.

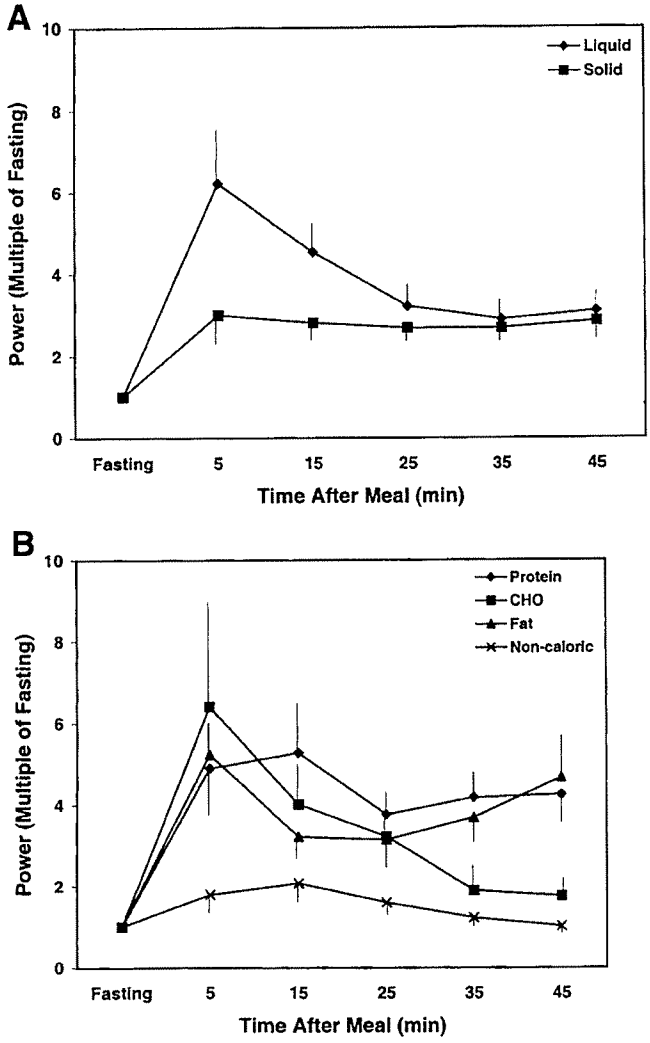

Fig. 5. Effects of meal ingestion on electrogastrographic power in women with first trimester nausea are shown. *A*: meals of both liquid and solid consistency increased signal power ($P < 0.05$). There was a trend to a greater response for liquids compared with solids that did not reach statistical significance. *B*: meals of all nutrient subclasses produced increases in power ($P < 0.05$). Carbohydrates and fats evoked similar increases to proteins, whereas responses to noncaloric meals were lower ($P < 0.05$). Values are means ± SE.

trend toward greater powers with liquid meal ingestion than solids that did not reach statistical significance ($F = 3.57$, $P = 0.06$), with maximal increases to 6.2 ± 1.5- and 3.0 ± 0.7-fold of fasting, respectively. As with nausea scores and dysrhythmic indexes, postprandial increases in power showed significant dependence on nutrient subclass ($F = 4.10$, $P = 0.01$; Fig. 5*B*). Meals in each nutrient subclass produced time-dependent increases in electrogastrographic power compared with fasting levels (protein: $F = 7.63$, $P = 0.003$; carbohydrate: $F = 4.62$, $P = 0.02$; fat: $F = 8.92$, $P = 0.002$; noncaloric: $F = 4.06$, $P = 0.03$). However, in contrast to the nausea and dysrhythmic index findings, analysis of differences between nutrient subclasses did not show a selective effect for protein meals. In this analysis, carbohydrate and fat meals produced similar increases

in power to protein-predominant meals ($P > 0.05$), especially over the first 25 min after meal ingestion, which were greater than those produced by noncaloric meals ($P < 0.05$).

DISCUSSION

The present study provides controlled data on symptom responses to ingestion of meals of different nutrient composition in nauseated women in first trimester pregnancy. As suggested by others, protein-predominant meals produced quantifiable decreases in nausea, whereas carbohydrate- and fat-predominant meals did not produce statistically significant effects (23, 29). Solid and liquid protein meals reduced nausea to similar degrees, indicating that the physical form of the meal has little relevance to its symptomatic effect. The demonstration that noncaloric meals did not decrease nausea is consistent with the hypothesis that the beneficial effects of proteins stem from a positive effect of that nutrient class rather than the alternate explanation that carbohydrates and fats have adverse symptom effects.

Dysrhythmic activity correlated well with symptom reports in women with first trimester nausea. During fasting, symptomatic pregnant women exhibited slow wave rhythm disruptions that were both bradygastric and tachygastric in morphology. As with nausea scores, protein-predominant meals reduced dysrhythmic activity to significantly greater degrees than did carbohydrates, fats, and noncaloric meals. The time courses for reductions in nausea scores and dysrhythmic indexes after protein ingestion were very similar, with improvements becoming evident ~25 min after eating. Both the nutrient selectivity and the time dependence of the slow wave rhythm responses are consistent with the theory that dysrhythmic gastric myoelectric activity is pathogenic of induction of nausea. The time delay in the dysrhythmic and symptomatic improvements suggests that direct activation of gastric neural pathways does not underlie the beneficial effect of protein ingestion. Rather, it indicates that more slowly activating systems, perhaps involving small intestinal sites or hormonal mediators, are responsible for the slow wave-stabilizing and nausea-reducing effects of the protein test meals. Alternatively, the time dependence of the symptom and electrogastrographic responses may correlate with intragastric meal redistribution from the fundus to the distal stomach before delivery into the intestine.

In contrast to nausea-scoring results, fats and noncaloric meals produced small but significant decreases in dysrhythmic indexes. Furthermore, liquid meals improved dysrhythmic activity to greater degrees than solids. There are several possible explanations for the relatively larger and more broadly based effects of meals on slow wave rhythm disturbances than on symptoms. It is conceivable that there is a threshold reduction in dysrhythmic activity, achieved by protein meals but not by fats or noncaloric meals, that is required to decrease nausea. Alternatively, slow wave rhythm disruption may be one of many cofactors in the production of nausea. With this theory, proteins may have beneficial effects in addition to and separate from their actions on dysrhythmic activity.

The electrogastrographic power responses to meal ingestion did not correlate with reductions in first trimester nausea. In contrast to a previous study that showed blunting of the postprandial power response in asymptomatic pregnant women (25), our investigation demonstrated increases in electrogastrographic power after ingestion of all test meals. Power responses to carbohydrates and fats were not different from responses to proteins. Although lesser in magnitude, power increases also were observed with noncaloric meals, a finding consistent with the accepted criteria for normality (3, 27). Furthermore, the increase in signal power occurred within 5 min with all meals, much sooner than nausea was reported to improve. These nutrient- and time-dependent differences in power response and nausea reports indicate that this electrogastrographic parameter cannot explain the nutrient-specific reductions in symptoms after protein meal ingestion.

There are alternate explanations for our findings. It is possible that meal palatability played a role in symptom reduction and dysrhythmia resolution with protein ingestion. However, the liquid and solid protein meals had very different taste characteristics. Furthermore, the liquid protein, liquid carbohydrate, and solid carbohydrate meals all were sweet, yet only the protein preparation was beneficial. Additionally, patient surveys did not reveal any differences in palatability. Second, it is possible that improvements with protein meals represent responses to a balanced diet, as each meal had carbohydrate and fat components representing 47% of the caloric load. However as part of a prior investigation of women with first trimester nausea, we tested the effects of ingestion of a mixed liquid meal that can be used as a sole enteral nutrition source (30). This supplement, which contains 14% of calories contributed by protein, 64% by carbohydrate, and 22% by fat, did not reduce dysrhythmic activity to any degree, in contrast to the protein-rich meals provided in the current study. Third, this investigation only evaluated single, stereotyped meals given on one occasion. It would be important to administer a diverse range of protein-enriched foodstuffs of differing tastes and seasoning over a period of weeks to confirm the beneficial effects of proteins and to determine that no tolerance to their efficacy developed. Finally, given the relatively small numbers of pregnant women tested, it is conceivable that type II errors may explain the lesser efficacy of carbohydrate, fat, and noncaloric meals in reducing nausea and dysrhythmic activity. In fact, reductions in dysrhythmic indexes and trends to reductions in symptoms were observed with other nutrients. Other studies of gastric myoelectric activity have shown improvement in slow wave stability after meal ingestion, suggesting that these responses may be part of a generalized response to gastric filling (4). Nonetheless, the most striking findings of this investigation relate to

the selectively beneficial effects of protein-predominant meals versus the other nutrient classes.

In conclusion, meals consisting predominantly of proteins improve symptoms and gastric slow wave rhythm in women with first trimester nausea to greater degrees than do carbohydrates, fats, or noncaloric meals. Meal consistency is a limited factor in the beneficial effects of proteins. These findings do not support the prevalent recommendation for carbohydrate meals in first trimester nausea but suggest that dietary modulation of gastric myoelectric activity with proteins may provide benefit to symptomatic pregnant women.

This study was supported in part by National Institute of Diabetes and Digestive and Kidney Diseases Grants RO1 DK-35783 and P30 DK-34933 and General Clinical Research Centers Program Grants MO1 RR-00042 and 3 MO1 RR-00042–32S1.

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Received 25 August 1998; accepted in final form 16 June 1999.

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