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Effect of Liquid and Solid Test Meals on Symptoms and Gastric Myoelectrical Activity in Patients with Gastroparesis and Functional Dyspepsia

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Abstract

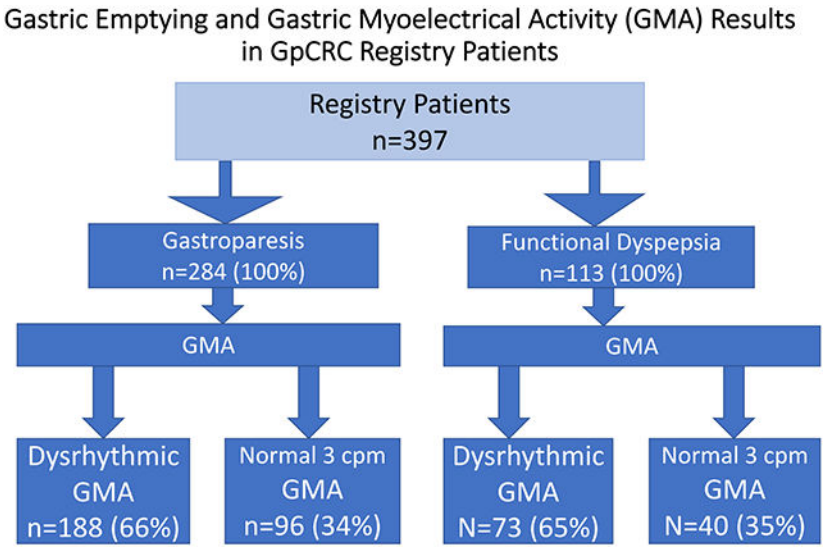
Patients with gastroparesis (GP) and functional dyspepsia (FD) have similar symptoms, but the pathophysiology of postprandial symptoms remains uncertain. Aims: To compare symptoms and gastric myoelectrical activity (GMA) after liquid and solid test meals in patients with GP and FD.

Methods: Patients enrolled in the Gastroparesis Clinical Research Consortium Registry were studied. Clinical characteristics were measured with standard questionnaires. GP was determined by 4 hour solid-phase gastric solid-phase scintigraphy. GMA was measured using electrogastrography before and after ingestion of a water load or nutrient bar on separate days. Symptoms were measured on visual analog scales. GMA responses to the water load for individual patients were also determined.

Results: 284 patients with GP and 113 with FD were identified who ingested both test meals. Patients with GP and FD had similar maximal tolerated volumes of water [Mean (SD) 378(218) mL vs 402(226) mL, p=0.23] and reported similar intensity of fullness, nausea, bloating, and abdominal discomfort after the test meals. Twenty-six percent and 19% of the patients with GP and FD, respectively, ingested subthreshold (<238 ml) volumes of water (p=0.15). Gastric dysrhythmias were recorded in 66% of the GP and 65% of the FD patients after the water load. Symptoms and GMA were similar in both groups after ingestion of the nutrient bar.

Conclusion: The similarity in GMA responses and symptoms after ingestion of solid or liquid test meals suggests GP and FD are closely related gastric neuromuscular disorders.

Graphical Abstract



Patients with gastroparesis (GP) and functional dyspepsia (FD) have similar symptoms, but the pathophysiology of postprandial symptoms remains uncertain. Results show symptoms and gastric myoelectrical (GMA) responses after ingestion of solid or liquid test meals are similar and suggests GP and FD are closely related gastric neuromuscular disorders.

Keywords

Gastroparesis; functional dyspepsia; postprandial distress syndrome; gastric dysrhythmias; water load satiety test; nutrient bar meal

Introduction

Symptoms associated with gastroparesis (GP) include early nausea, vomiting, early satiety, prolonged fullness, bloating, and abdominal discomfort or pain in the absence of mechanical obstruction (1). Symptoms associated with functional dyspepsia (FD) include bothersome early satiation, fullness after meals with supporting symptoms of epigastric burning (not pain), bloating, belching, and nausea and are termed postprandial distress syndrome (PDS); and patients with unexplained epigastric pain are termed epigastric pain symptoms (EPS) (2). These symptoms are present in the absence of other diagnoses after routine investigations including upper endoscopy (2,3). Most patients with GP also have symptoms that meet the definition of FD and the majority have PDS (3-8).

The pathophysiological mechanisms of symptoms associated with GP and FD remain uncertain. The symptoms associated with GP do not always correlate with the delayed rate of gastric emptying and the relevance of delayed gastric emptying as the primary pathophysiological mechanism of postprandial symptoms has been questioned (3,5-8). Transition from delayed gastric emptying status to normal emptying status at 48 weeks follow up did not affect GCSI scores (9). Thus, other gastric neuromuscular abnormalities in patients with GP and FD include abnormal gastric accommodation, gastric hypersensitivity,

and gastric dysrhythmias that may be more relevant to the postprandial symptomatology (10-13).

Gastric dysrhythmias are present in patients with GP and FD when the numbers of interstitial cells of Cajal (ICCs) per high power field (hpf) in the corpus-antrum and normal 3 cycle per min (cpm) gastric myoelectrical activity (GMA) are diminished (14-18). The ICCs, the pacemaker cells of the stomach, are severely depleted in patients with GP (18) and are modestly depleted in patients with chronic unexplained nausea and vomiting and normal gastric emptying (9,19). With loss of ICCs normal 3 cpm GMA is diminished and gastric dysrhythmias appear (9,19).

Postprandial symptoms, gastric dysrhythmias, gastric capacity and sensitivity to gastric distention after ingestion of test meals may be detected in real time during satiety tests (10,11,13,20). The aim of this study was to compare GMA and symptoms in response to liquid and solid test meals in patients with GP and FD. We hypothesized that patients with GP would have more intense symptoms and poorer gastric capacity, less 3 cpm GMA, and more gastric dysrhythmias after the water load satiety test (WLST) and after ingestion of a solid nutrient meal compared with FD patients.

Methods

Patients

Patients in the Registry were 18 years or older, had four-hour, solid-phase gastric emptying by scintigraphy, and had no structural abnormalities on upper endoscopy. Patients with delayed gastric emptying or with normal gastric emptying were enrolled at nine clinical centers from September 2012 to March 2018. In this report the term FD is used to describe symptomatic patients with normal gastric emptying. Written informed consent was obtained prior to enrollment in the Registry.

Four-Hour Gastric Emptying Study

Gastric emptying scintigraphy was performed using a low-fat egg white meal (EggBeaters®) with imaging at 0, 1, 2, and 4 hours after meal ingestion (21). Medications affecting gastrointestinal motility were stopped 3 days prior to the study. Tests were performed after an overnight fast. In patients with diabetes, low blood sugar (hypoglycemia <70 mg/dl) or high blood sugar (hyperglycemia >270 mg/dl) was corrected or the study was rescheduled for another day under better glucose control. Gastric retention of Tc-99m >60% at 2 hours and/or >10% at 4 hours was considered delayed gastric emptying of solids (21).

Electrogastrography and the Water Load Satiety Test (WLST) and the Nutrient Bar Meal

Standard electrogastrography methods were used to record GMA in response to the WLST (11,22). EKG-type electrodes were placed in standard position on the upper abdominal surface after the skin was cleaned with alcohol wipes. Electrodes were connected to the electrogastrogram (EGG) recording device to record GMA (3CPM Company, Towson, MD). The EGG signal was digitized for computer analysis (11,22).

Patients stopped proton pump inhibitors, histamine2- receptor antagonists, prokinetics drugs, opiates, anticholinergics, cannabinoids, over-the-counter laxatives, isotonic polyethylene glycol electrolyte preparations, and prescription laxatives for 3 days before the studies. Patients fasted oernight before the test meal. On the morning of the studies, insulin-requiring patients with diabetes injected half of their usual long-acting insulin dose.

Water Load Satiety Test—On the day of the WLST (or the nutrient bar test meal), glucose levels over 270 mg/dl in patients with diabetes were treated or the test was rescheduled. Patients were seated in a comfortable chair in a quiet area. A baseline fasting EGG recording was performed for 15 minutes. For the WLST, patients ingested water until they achieved the sensation of “completely full” during the timed and continuos five-minute period for water ingestion (11,13, 22). The volume of water ingested was recorded. The volume of water ingested reflects gastric capacity and gastric accommodation to that volume ingested. Ingestion of <238 ml of water in the five-minute period is 2 SD below the mean volume ingested by healthy controls and was considered abnormal (11). The patients indicated the intensity of fullness, hunger, abdominal discomfort, bloating, and nausea on a 100 mm visual analog scale (VAS) before and 10, 20, and 30 minutes after the water was ingested. GMA was recorded for 30 minutes after the water load was ingested.

Nutrient Bar Test Meal—The nutrient bar was consumed with the capsule on a separate day with preparations and placement of electrodes for EGG recordings on the abdomen as described above for the WLST. Each patient ingested one nutrient bar (244 Cal, 66% carbohydrate, 17% protein, 2% fat, 3% fiber) over a 10-minute period with 50 ml water. GMA was recorded during the 15-minute baseline and for 90 minutes after the meal. Patients reported intensity of symptoms on the VAS as described above before and at 15, 30, 45, 60, and 90 minutes after the bar was ingested.

Analyses of GMA in response to WLST and Nutrient Bar Meal

The raw GMA signal is digitized and subjected to fast Fourier transform and running spectral analysis. The power calculation in the running spectral analysis reflects the amplitude of GMA in four frequency ranges: 1 - 2.5 cpm (bradygastria), 2.5-3.7 cpm (normal range), 3.7-10.0 cpm (tachygastria) and 10-15.0 cpm (duodenal/respiration range) before and after the WLST or nutrient bar test. The power in each frequency range is divided by the total power in the 1-15 cpm range. This calculation provides the percentage distribution of power for each of the four frequency ranges listed above (11,13,22). The percentage distribution of power in the four frequency ranges is plotted over time and compared with controls as shown in Results. The average percentage distributions of GMA from the patients with GP and FD were compared at baseline and for each time from 1-10, 11-20, and 21-30 minutes after the WLST (11,13, 22). Similarly, for the nutrient bar meal, the GMA percentages in each frequency range were calculated for baseline and for the 1-15, 16-30, 31-45, 46-60, and 75-90-minute periods after ingestion of the bar and results from GP and FD cohorts were compared.

In addition, the GMA in response to the WLST was determined for each patient. The patient’s results were compared with controls and individual EGG diagnoses in response

to the WLST were determined. The definitions for EGG diagnoses are based on the GMA response to the WLST. The dysrhythmias include tachygastria, bradygastria, mixed gastric dysrhythmia, duodenal–respiration and hyponormal 3 cpm. The normal 3 cpm diagnoses include normal 3 cpm GMA, hypernormal 3 cpm GMA and normal 3 cpm GMA with dysrhythmias (Supplement Table 1) (11,13, 22). The EGG diagnosis for each patient was determined by one of the authors (KK) who was blinded to the normal or delayed emptying status of the patient and to the clinical site.

Patient Assessment of Upper Gastrointestinal Disorders Symptoms (PAGI-SYM)

Gastroparesis symptom severity was determined by the Gastroparesis Cardinal Symptom Index (GCSI) score, which includes nine questions from the PAGI-SYM. PAGI-SYM subscale and individual scores for upper GI symptoms were calculated (24).

The Nausea Profile

Nausea is one of the most common and debilitating symptoms reported by patients with GP and in subtypes of FD, but nausea and the extent of symptoms associated with nausea can be difficult to describe. The Nausea Profile questionnaire consists of 17 questions that describe the sensation of nausea in words other than the word nausea (25). Factor analysis of 416 such words yielded three dimensions of nausea that correlated with overall nausea intensity. The three dimensions are GI Distress (sick, stomach awareness/discomfort, as if he/she might vomit, ill, queasy), Somatic Distress (fatigue, weak, hot, sweaty, lightheaded, shakiness), and Emotional Distress (nervous, scared, afraid, worry, upset, panic, hopeless).

Statistical Methods

Characteristics at enrollment were compared between patients with GP and FD-PDS using t-test for unequal variance for continuous variables and Fisher’s exact test for categorical variables. Symptoms and EGG results from the water load test and nutrient bar test were compared between the FD and GP patients using robust regression which downweights the effect of outliers. The average of the post-test time points was used as the outcome for change. The baseline value was regressed on FD vs GP group status, and change was regressed on FD vs GP group status and the baseline value of change. Random effects linear regression models were used for analyses of change from pre-test levels averaged across all post-test time points. P-values were two-sided and not corrected for multiple comparisons. Analyses were conducted using SAS and Stata (26,27)

Results

Clinical Characteristics of Patients with Functional Dyspepsia and Gastroparesis

Table 1 summarizes demographic and clinical characteristics of the 284 patients with GP and the 113 patients with FD who completed the WLST and ingested the nutrient bar. Eighty-four percent of the GP patients and 90% of the FD patients were women. The average age of patients in both groups was 44 years and 90% of the patients with GP vs 93% of the patients with FD were white. Diabetes was present in 33% of the GP patients and 25% of the FD patients (P=0.08). The onset of symptoms was acute in 40% of GP patients and

34% of FD patients and duration of symptoms at the time of evaluation were similar in both groups.

The overall severity of symptoms as assessed qualitatively by the investigators was similar in both groups. Laboratory values were similar in the two groups. The general classes of medications that the GP and FP patients received were similar. Fibromyalgia and bipolar disorders were more frequent in FD vs GP (25% vs 15%, P=0.03) and (12% vs 6%, P=0.03), respectively. The use of tobacco, alcohol, opioids and marijuana were similar in the two groups. The Rome III questionnaire was used to determine the PDS or EPS designation in the patients with normal gastric emptying. Eighty-five percent of the patients with FD (n=95) had symptoms associated with PDS, EPS or both and fifteen percent had neither PDS or EPS (n=18). Details are shown in Supplement Table 3.

Symptoms in Patients with GP and FD

The PAGY-QOL, SF-36, PAGY-SYM and GCSI total scores were similar in the GP and FD patients and the subscores for vomiting, postprandial fullness, and bloating were also similar in each group as shown in Table 1. Regarding dimensions of nausea from the Nausea Profile, the GI distress scores for GP was 74% and FD was 73%. Somatic distress trended higher in GP (49%) vs FD (44%) (P=0.07). Emotional distress symptoms were the lowest scores and were similar at 29% and 27% in the GP and FD patients, respectively.

Symptoms and GMA elicited by the WLST and Nutrient Bar Meals in GP and FD

Figure 1 shows the maximal tolerated volumes of water ingested in the five-minute time limit of the WLST. The mean +/− SD for volume ingested by patient with GP and FD was 378 mL +/− 218 and 402 mL +/− 226, respectively (P=0.23) (Table 2). Abnormally low volumes (<2 SD was 238 mL) were ingested by 26% of the patients with GP and by 19% of the patients with FD (P=0.15).

The intensity of symptoms and the percentage distribution of power in the four GMA frequency ranges before and after the WLST are compared for the GP and FD groups in Table 2. Baseline and change in symptoms of fullness, bloating, nausea, and abdominal discomfort for the total 1-30-minute post WLST period were similar in the GP and FD groups. At baseline the average percentage distribution in the tachygastria range was significantly greater in the GP group compared with FD (23% vs 19%, P=0.01) and average percentage bradygastria was significantly lower (49% vs 55%, P=0.02). After the water load was ingested, changes in the mean percentage distribution of GMA activity in the four frequency ranges in the combined 1-30-minute time period were similar in both groups.

Table 3 shows symptoms and GMA before and for 90 minutes after ingestion of the nutrient bar. These data are shown in a format similar to Table 2. Patients with GP ingested approximately 83% of the nutrient bar compared with 90% in the patients with FD (P=0.006), a difference of approximately 18 calories. The average intensity of symptoms before and after ingestion of the nutrient bar was similar in both groups. The GMA percentages in the four frequency ranges, the averaged sum of the five time points after ingestion of the nutrient bar, were similar in the patients with GP and FD.

Figure 2 shows the average symptom scores reported before and during each of the three 10-minute periods after the WLST and before and during the five periods after ingestion of the nutrient bar in the GP and FD patients. The symptoms reported are similar in each group before and after ingestion of the water load and the nutrient bar.

Figure 3 shows the average GMA percentages in the four frequency ranges before and 10, 20, and 30 minutes after the WLST. At baseline the average percentage tachygastria was significantly higher in the GP patients compared with FD patients and average percentage bradygastria was higher at baseline in the FD patients as noted above in Table 1. After the water load was ingested, there were no differences in average GMA in the two groups. Figure 3 also shows the GMA results at baseline and 15, 30, 45, 60, and 90 minutes after the nutrient bar meal was ingested. Before and after ingestion of the nutrient bar, mean percentage distributions of GMA in the four frequency ranges were similar in the GP and FD cohorts.

Individual GMA responses to the WLST in patients with GP and FD

The individual EGG diagnoses based on the GMA response to the WLST are shown in Table 4. Of the 284 patients with GP, 184 (66%) had gastric dysrhythmias and 96 (34%) had normal 3 cpm GMA. Of the patients with GP, 31% had tachygastria, 17% had bradygastria, 9% had mixed dysrhythmias, and 9% had hyponormal 3 cpm GMA only; and, of the patients termed normal 3 cpm GMA there was variably patterns: 15% had normal 3 cpm GMA, 5 % had hypernormal 3 cpm and 14% had normal 3 cpm with dysrhythmias.

In regards to the 113 patients with FD, 31% had tachygastria, 20% had bradygastria, 1 % had mixed dysrhythmia, and 13 % had hyponormal 3 cpm only; and of the patients with overall normal 3 cpm diagnoses 19% had normal 3 cpm GMA, 9 % had hypernormal 3 cpm GMA, and 7% had 3 cpm GMA with dysrhythmia. The distribution of individual diagnoses in the patients with GP and FD are generally similar; comparison of the diagnostic categories in the two groups by logistic regression showed that there were significantly fewer patients with mixed gastric dysrhythmias in the FD group compared with the GP group (P=0.05).

Comparison of characteristics in patents with GP and normal 3 cpm GMA versus patients with GP and dysrhythmic GMA showed that the group with dysrhythmic GMA had significantly higher BMI (29 vs 26 kg/m^2), were prescribed more steroids and anxiolytics, had higher GCSI retching scores and consumed more water during the WLST. These comparisons for patients with FD showed that patients with dysrhythmic GMA had significantly higher BMI (29 vs 26 kg/m^2, P=0.05), lower incidence of acute onset of symptoms, longer duration of GP symptoms and were prescribed more neuropathic pain medications compared with those with normal 3 cpm GMA (Supplement Table 3).

Examples of normal 3 cpm GMA and tachygastria in the EGG recordings, running spectral analyses, and percentage distribution of power in the four frequency ranges in individual patients with GP and FD are shown in Supplement Figures 4-7.

Discussion

The key findings of this study are 1) patients with GP and FD had similar symptoms in response to liquid and solid test meals and 2) the GMA response to the WLST revealed gastric dysrhythmias in two-thirds of the patients and normal 3 cpm GMA in one third. In addition, almost 25% of the patients with GP and FD ingested less than 238 ml of water during the WLST, indicating poor gastric accommodation. These findings indicate the similarity of gastric pathophysiological abnormalities in patients with GP and FD.

Dysrhythmic GMA was present in 66% of the patients with GP and may be relevant to the selection and efficacy of drug or device therapies. Gastric electrical stimulation therapy was significantly more effective in reducing symptoms in patients with GP who had 3 cpm GMA, less tachygastria and greater numbers of ICCs compared with GP patients with tachygastria and fewer ICCs (28). Domperidone treatment improved symptoms and increased 3 cpm GMA in patients with diabetic GP and gastric dysrhythmias (29) and cisapride improved symptoms and 3 cpm GMA in 71% of patients with idiopathic GP and gastric dysrhythmias (30). On the other hand, almost 1/3 of the patients with GP had normal 3 cpm GMA. Normal 3 cpm GMA is present when there are 5 or more ICCs/ hpf in the gastric antrum and corpus (18,19). Normal 3 cpm GMA in patients with GP would seem to be discordant, but 20% and 50% of patients with diabetic and idiopathic GP, respectively, had normal numbers of ICCs by immunohistological studies (15,16). The patients with GP and normal numbers of gastric ICCs had milder delay in gastric emptying at four hours compared with patients with GP and low ICC numbers (22 +/− 9.4% vs 47.6 +/− 25.6%, respectively, $P<0.05$) (16). Ultrastructural abnormalities in ICCs and enteric neurons on the other hand have been detected by electron microscopy in all patients with GP (15). Thus, it is possible ICCs and enteric neurons are normal in number but are dysfunctional in some patients with GP and normal 3 cpm GMA.

Patients with GP secondary to pyloric stenosis have normal 3 cpm GMA (31). In the vast majority of patients with GP and 3 cpm GMA the pylorus appears normal at endoscopy (32), but pyloric neuromuscular dysfunction has been appreciated in GP. Mearin et al reported pylorospasm in patients with diabetic GP (33). Fisher et al described premature closure of the terminal antrum before peristaltic waves reached the pylorus in patients with GP (34). Poor distensibility of the pylorus was found in almost 30% of patients with moderate to severe GP (35). Furthermore, ICCs were decreased and fibrosis was increased in the pylorus in 83% of patients with GP (36). Improvement in symptoms was reported after botulinum toxin injection or balloon dilation of the pylorus in 78% of the patients with GP and normal 3 cpm GMA (32) and in selected patients who subsequently underwent pyloroplasty, symptoms decreased and gastric emptying was normal or improved six months after the operation (37).

Of the 113 patients with FD 65% had gastric dysrhythmias and 35% had normal 3 cpm GMA after the WLST, incidences similar to the patients with GP. Gastric dysrhythmias are abnormalities found in patients with FD and GP and link the two entities on the spectrum of gastric neuromuscular disorders. Gastric dysrhythmias may also have a role in the pathophysiology of postprandial symptoms in FD (9,19). Symptoms significantly decreased

and normal 3 cpm GMA increased after treatment with cisapride in children and adults with FD and gastric dysrhythmias (38)(39). Patients with chronic nausea (with normal or with delayed gastric emptying emptying) had significantly decreased nausea and decreased tachygastria activity after apprepitant compared with patients who received placebo (12). Normal 3 cpm GMA was recorded in 35% of the patients with FD. The normal 3 cpm GMA in these patients suggests normal numbers of gastric ICCs were present. Patients with FD and normal 3 cpm GMA may have poor gastric accommodation. These patients may also have non-gastric diseases that mimic FD such as atypical gastroesophageal reflux disease (40), rapid gastric emptying (dumping syndrome) (41), gallbladder diseases (42), small intestinal bacterial overgrowth, and irritable bowel syndrome that contribute to their symptoms (2).

In summary, symptoms and GMA responses after liquid and solid test meals were similar in patients with GP and FD. Poor gastric distension and gastric dysrhythmias are pathophysiological abnormalities that link the similar postprandial symptoms and gastric neuromuscular dysfunction in patients with GP and FD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Kim BJ, Kuo B. Gastroparesis and functional dyspepsia: A blurring distinction of pathophysiology and treatment. J Neurogastroenterol Motil 2019;25:27–35. [PubMed: 30509017]
2. Talley NJ, Stanghellini V, Chan FKL, et al. Gastroduodenal Disorders. In Rome IV. Functional Gastrointestinal Disorders. Eds. Drossman DA, et al., Rome Foundation, pp. 203–965, 2016.
3. Carbone F, Vanuytsel T, Tack J. Analysis of postprandial symptom patterns in subgroups of patients with Rome III or IV functional dyspepsia. Clin Gastroenterol Hepatol 2020;18:838–846. [PubMed: 31394286]
4. Jehangir A, Parkman P. Rome IV diagnostic questionnaire complements patient assessment of gastrointestinal symptoms for patients with gastroparesis symptoms. Dig Dis Sci 2018:63:2231–2243. [PubMed: 29808246]
5. Pasricha PJ, Colvin R, Yate, et al. Characteristics of patients with chronic unexplained nausea and vomiting and normal gastric emptying. Clin Gastroenterol Hepatol 2011;9:567–76. [PubMed: 21397732]
6. Lacy BE. Functional dyspepsia and gastroparesis: One disease or two? Am J Gastroenterol 2012;107:1615–20. [PubMed: 23160285]
7. Stanghellini V, Tack J. Gastroparesis: Separate entity or just a part of dyspepsia? Gut 2014;63:1972–8. [PubMed: 25260920]
8. Tack J, Carbone F. Functional dyspepsia and gastroparesis. Curr Opin Gastroenterol 2017;33:446–54. [PubMed: 28832359]
9. Pasricha PJ, Grover M, Yates KP and GpCRC. Functional dyspepsia and gastroparesis are interchangeable syndromes with common clinical and pathological features. Pasricha PJ, Grover

M, Yates KP, and GpCRC. Functional dyspepsia and gastroparesis interchangeable syndromes with common clinical and pathophysiological features. Gastroenterol 2021. Feb 3. SOO16-5085 (21)00337_1doi:10.1053/Jgastro.2021.230

10. Tack J, Caenepeel P, Piessevaux H, et al. Assessment of meal induced gastric accommodation by a satiety drinking test in health and in severe functional dyspepsia. Gut 2003;52:1271–7. [PubMed: 12912857]
11. Koch KL, Hong S-P, Xu L. Reproducibility of gastric myoelectrical activity and the water load test in patients with dysmotility-like dyspepsia symptoms and in control subjects. J Clin Gastroenterol 2000;31:125–129. [PubMed: 10993427]
12. Pasricha PJ, Yates KP, Sarosiek I, et al. and GpCRC. Aprepitant has mixed effects on nausea and reduces other symptoms in patients with gastroparesis and related disorders. Gastroenterol 2018;154:65–76.
13. Koch KL, Hasler WL, Van Natta M, et al. and GpCRC. Satiety testing in diabetic gastroparesis: Effects of insulin pump therapy and continuous glucose monitoring on upper gastrointestinal symptoms and gastric myoelectrical activity. Neurogastroenterol Motil 2019;00:e13720.
14. Koch KL. Gastric Neuromuscular Function and Neuromuscular Disorders. In Sleisenger and Fordtran's Gastrointestinal and Liver Disease: Pathophysiology/Diagnosis/Management. Eds Feldman M, Friedman LS, Brandt LJ. Elsevier, Inc., Philadelphia, 2015, pp. 811–838.
15. Faussone-Pellegrini MS, Pasricha PJ, Bernard CE, et al. Ultrastructural differences between diabetic and idiopathic gastroparesis. J Cell Mol Med 2012;16:1573–1581. [PubMed: 21914127]
16. Grover M, Bernard CE, Pasricha PJ, et al. and GpCRC. Clinical-histological associations in gastroparesis: Results from the Gastroparesis Clinical Research Consortium. Neurogastroenterol Motil 2012;24:531–e249. [PubMed: 22339929]
17. Farrugia G, Lurken MS, Bernard CE, et al. Cellular changes in diabetic and idiopathic gastroparesis. Gastroenterol 2011;140:1575–1585.
18. O'Grady G, Angeli TR, Du P, et al. Abnormal initiation and conduction of slow-wave activity in gastroparesis, defined by high-resolution electrical mapping. Gastroenterol 2012;143:589–598.
19. Angeli TR, Cheng LK, Du P, et al. Loss of interstitial cells of Cajal and patterns of gastric dysrhythmia in patients with chronic unexplained nausea and vomiting. Gastroenterol 2015;149:56–66.
20. Jones MP, Hoffman S, Shah D, et al. The water load test: Observations from healthy controls and patients with functional dyspepsia. Am J Physiol Gastrointest Liver Physiol 2003;284:G896–904. [PubMed: 12529263]
21. Tougas G, Eaker EY, Abell TL, et al. Assessment of gastric emptying using a low-fat meal: Establishment of international control values. Am J Gastroenterol 2000;95:1456–1462. [PubMed: 10894578]
22. Koch KL. Electrogastrography for Suspected Gastroparesis. In Gastroparesis: Pathophysiology, Clinical Presentation, Diagnosis and Treatment. Eds. McCallum R, Parkman H. Elsevier, 2020.
23. Lee AA, Rao S, Nguyen LA, et al. Validation of diagnostic and performance characteristics of the wireless motility capsule in patients with suspected gastroparesis. Clin Gastroenterol Hepatol 2019;17:1770–1779. [PubMed: 30557741]
24. Revicki DA, Rentz AM, Dubois D, et al. Development and validation of a patient-assessed gastroparesis symptom severity measure: The Gastroparesis Cardinal Symptom Index. Aliment Pharmacol Ther 2003;18:141–50. [PubMed: 12848636]
25. Muth ER, Stern RM, Thayer JF, Koch KL. Assessment of the multiple dimensions of nausea: The nausea profile (NP). J Psychosomatic Research 1996;5:511–520.
26. SAS Institute, Inc. SAS software, version 9.3 of the SAS system for Windows. Cary, NC.
27. StataCorp LP. Stata 15.1. Stata statistical software: release 12. College Station, TX.
28. Lin Z, Sarosiek I, Forster J, et al. Association of the status of interstitial cells of Cajal and electrogastrogram parameters, gastric emptying, and symptoms in patients with gastroparesis. Neurogastroenterol Motil 2010;22:56–61. [PubMed: 19614868]
29. Koch KL, Stern RM, Stewart WR, Vasey MW. Gastric emptying and gastric myoelectrical activity in patients with symptomatic diabetic gastroparesis: Effects of long-term domperidone treatment. Am J Gastroenterol 1989;84:1069–1075. [PubMed: 2773901]

30. Rothstein R, Alavi A, Reynolds JC. Electrogastrography in patients with gastroparesis and effect of long-term cisapride. Dig Dis Sci 1993;38:1518–1524. [PubMed: 8344110]
31. Brzana RJ, Bingaman S, Koch KL. Gastric myoelectrical activity in patients with gastric outlet obstruction and idiopathic gastroparesis. Am J Gastroenterol 1998;93:1083–1089.
32. Wellington J, Scott B, Kundu S, Stuart P, Koch KL. Effect of endoscopic pyloric therapies for patients with nausea and vomiting and functional obstructive gastroparesis. Auton Neurosci 2017;202:56–61. [PubMed: 27460691]
33. Mearin F, Camilleri M, Malagelada JR. Pyloric dysfunction in diabetics with recurrent nausea and vomiting. Gastroenterol 1986;90:1919–25.
34. Fisher RS. Gastroduodenal motility disturbances in man. Scand J Gastroenterol 1985;109:59–68.
35. Snape WJ, Lin MS, Agarwal N, Shaw RE. Evaluation of the pylorus with concurrent intraluminal pressure and EndoFLIP in patients with nausea and vomiting. Neurogastroenterol Motil 2016;28:758–764. [PubMed: 26813266]
36. Bashashati B, Moraveji S, Torabi A, et al. Pathological findings of the antrum and pylori smooth muscle in patients with gastroparesis and gastroparesis-like syndrome compared to gastroparesis: Similarities and differences. Dig Dis Sci 2017;62:2828–2833. [PubMed: 28577248]
37. Wellington J, Stuart P, Westcott C, Koch KL. Obstructive gastroparesis: Patient selection and effect of laparoscopic pyloroplasty. J Gastrointest Surg 2020;24:1778–1784. [PubMed: 31270719]
38. Cucchiara S, Minella R, Riezzo G, et al. Reversal of gastric electrical dysrhythmias by cisapride in children with functional dyspepsia: Report of three cases. Dig Dis Sci 1992;37:1136–40. [PubMed: 1618063]
39. Besherdas K, Leahy A, Mason I, et al. The effect of cisapride on dyspepsia symptoms and the electrogastrogram in patients with non-ulcer dyspepsia. Aliment Pharmacol Ther 1998;12:755–9. [PubMed: 9726389]
40. Brzana RJ, Koch KL. Intractable nausea presenting as gastroesophageal reflux disease. Ann Intern Med 1997;126:704–707. [PubMed: 9139556]
41. Wang P, Wellington J, Koch KL. Clinical features and gastric myoelectrical activity in patients with idiopathic and post-surgical rapid gastric emptying who present with unexplained chronic nausea. Neurogastroenterol Motil. 2021 March;33(3): e13988 E pub 2020 Sep 18. doi.org/10.1111/nmos13988. 2021(Mar);e13988. [PubMed: 32945602]
42. Whitaker LF, Powell MS, Refugia J, Bosley ME, Koch KL, Bennett P, Fernandez A. Outcomes after laparoscopic cholecystectomy in hyperkinetic biliary dyskinesia. (Am Surg. 2021 May 28:31348211023390. doi: 10.1177/00031348211023390. Online ahead of print.

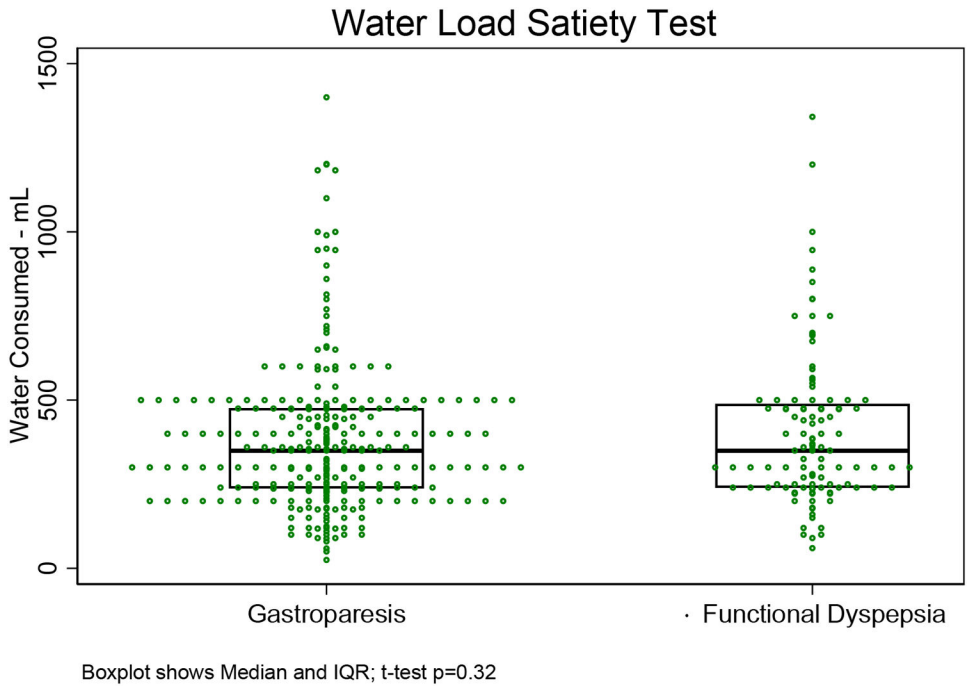

Figure 1.

The volume of water consumed by each of the patients with gastroparesis and functional dyspepsia is shown. The volume is the amount in mL consumed until the patient was completely full within the 5-minte time limit. The average volume was 378 +/− 218 mL in the GP group and 402 +/− 226 mL in the FD group (p=0.32).

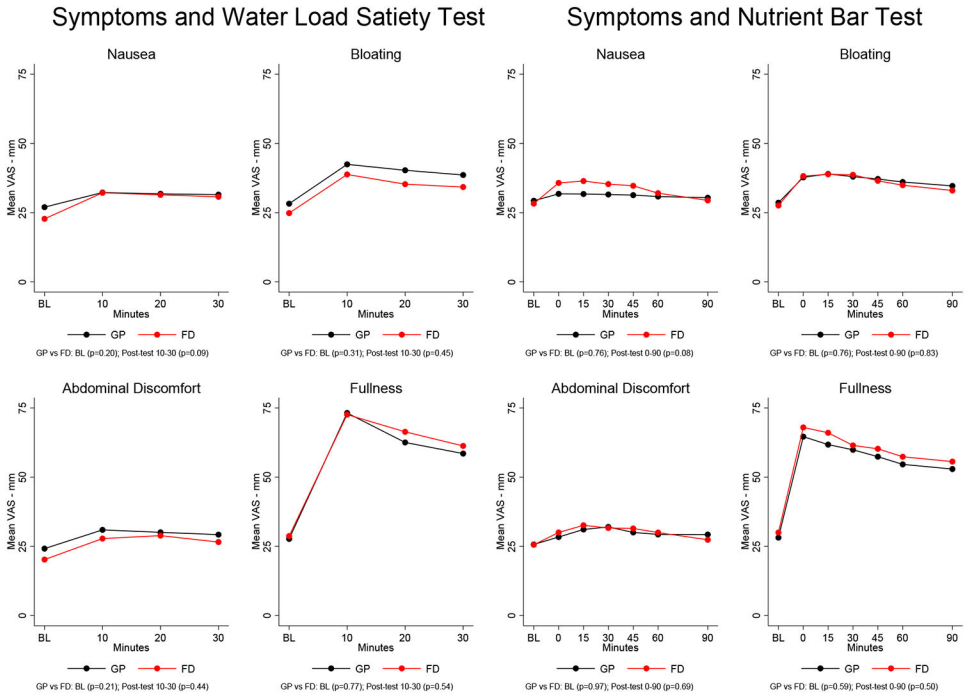

Figure 2.

Symptoms before and after the water load satiety test and nutrient bar test are shown. Symptoms were scored on a visual analog scale from 0-100 for each symptom. BL indicates the time 15 min before ingestion of the water and 0 represents symptoms immediately after ingestion of the nutrient bar. Symptoms increased after the water load in both groups, but there were no significant differences in intensity between the GP and FD groups. Symptoms before and after ingestion of the nutrient bar test meal were similar in the GP and FD groups.

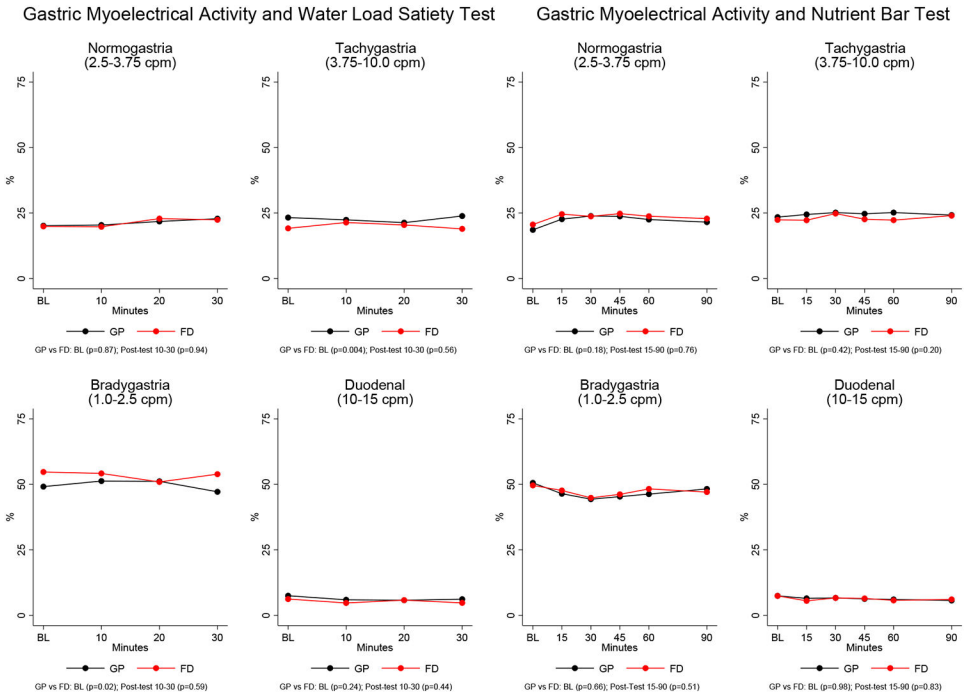

Figure 3.

Percentage (%) distribution of gastric myoelectrical activity (GMA) is shown in the four frequency ranges on the Y axis before and after the water load satiety test and nutrient bar test in the GP and FD groups. On the X-axis BL indicates 15 min before the water load or nutrient bar was ingested and the 10, 20, and 30 minutes after the water load and up to 90 minutes after the nutrient bar was ingested. There were no differences the percentage GMA activity in the patients with GP and FD.

Table 1.

Characteristics at Enrollment of Patients with Gastroparesis and Functional Dyspepsia

Characteristics at Enrollment	Gastroparesis (n=284)	Functional Dyspepsia (n=113)	Total (n=397)	P-value
	Mean (SD) / N (%)	Mean (SD) / N (%)	Mean (SD) / N (%)	
Demographics				
Gender (female)	238 (84%)	102 (90%)	340 (86%)	0.11
Age – yr	44 (13)	44 (16)	44 (14)	0.99
Race (white)	257 (90%)	105 (93%)	362 (91%)	0.56
Ethnicity (Hispanic)	44 (15%)	14 (12%)	58 (15%)	0.53
Body mass index (kg/m^2)	28 (7)	28 (10)	28 (8)	0.96
Characteristics of gastroparesis				
Etiology				0.08
Diabetes	94 (33%)	28 (25%)	122 (31%)	
Idiopathic	177 (62%)	83 (73%)	260 (65%)	
Fundoplication	13 (5%)	2 (2%)	15 (4%)	
Acute onset of symptoms	114 (40%)	38 (34%)	152 (38%)	0.25
Severity				0.85
Grade 1, mild	56 (20%)	24 (21%)	80 (20%)	
Grade 2, compensated	188 (67%)	76 (67%)	264 (67%)	
Grade 3, gastric failure	38 (13%)	13 (12%)	51 (13%)	
Duration of symptoms – yr	6.2 (6.8)	6.8 (7.9)	6.3 (7.2)	0.51
Rome III categorization				0.38
Neither PDS nor EPS	53 (19%)	18 (16%)	71 (18%)	
PDS only	56 (20%)	28 (25%)	84 (21%)	
EPS only	27 (10%)	6 (5%)	33 (8%)	
Both PDS and EPS	148 (52%)	61 (54%)	209 (53%)	
Scintigraphy				
2-hr solid retention - %	65 (18)	31 (16)	55 (23)	<0.0001
4-hr solid retention - %	31 (21)	4 (3)	23 (21)	<0.0001
Laboratory results				
HbA1c - %	6.3 (1.8)	6.1 (1.6)	6.3 (1.7)	0.22
Erythrocyte sedimentation rate – mm/hr	19.8 (19.2)	16.3 (15.1)	18.8 (18.2)	0.05
C-reactive protein – mg/dL	3.7 (9.5)	2.2 (7.4)	3.3 (8.9)	0.10
White blood cells – 10^3 cells/μL	7.2 (2.2)	6.7 (2.4)	7.0 (2.3)	0.06
Hemoglobin – g/dL	13.3 (1.5)	13.2 (1.2)	13.2 (1.5)	0.56

Characteristics at Enrollment	Gastroparesis (n=284)	Functional Dyspepsia (n=113)	Total (n=397)	P-value
	Mean (SD) / N (%)	Mean (SD) / N (%)	Mean (SD) / N (%)	
Medication use				
Systemic corticosteroids	61 (21%)	25 (22%)	86 (21%)	0.89
Narcotic pain medications	97 (34%)	35 (31%)	132 (33%)	0.56
Neuropathic pain medications	91 (32%)	34 (30%)	125 (31%)	0.72
Antidepressants	229 (81%)	90 (80%)	319 (80%)	0.89
Mirtazapine (Remeron)	14 (5%)	9 (8%)	23 (6%)	0.24
Prokinetics	95 (33%)	27 (24%)	122 (31%)	0.07
Co-Morbidities				
GERD	187 (66%)	74 (65%)	261 (66%)	1.00
Interstitial cystitis	8 (3%)	3 (3%)	11 (3%)	1.00
Endometriosis	39 (14%)	21 (19%)	60 (15%)	0.28
Migraine headaches	109 (38%)	42 (37%)	151 (38%)	0.91
Chronic fatigue syndrome	35 (12%)	8 (7%)	43 (11%)	0.15
PCOS	27 (10%)	11 (10%)	38 (10%)	1.00
Fibromyalgia	42 (15%)	28 (25%)	70 (18%)	0.03
Eating disorder	9 (3%)	2 (2%)	11 (3%)	0.74
Anxiety requiring treatment	75 (26%)	24 (21%)	99 (25%)	0.31
Major depression requiring treatment	88 (31%)	29 (26%)	117 (29%)	0.33
Bipolar	16 (6%)	14 (12%)	30 (8%)	0.03
Smoking, drinking and marijuana use				
Smoking history				0.71
Never	176 (62%)	75 (66%)	251 (63%)	
Former	67 (24%)	23 (20%)	90 (23%)	
Current	41 (14%)	15 (13%)	56 (14%)	
Alcohol use				0.40
Never	139 (49%)	50 (44%)	189 (48%)	
Monthly or less	96 (34%)	37 (33%)	133 (34%)	
≥ 2 drinks per month	49 (17%)	26 (23%)	75 (19%)	
Marijuana use	34 (12%)	12 (11%)	46 (12%)	0.86
PAGI-QOL				
Total score	2.8 (1.1)	2.8 (1.1)	2.8 (1.1)	0.80
Daily activities subscale	2.6 (1.2)	2.5 (1.2)	2.6 (1.2)	0.49
Clothing subscale	2.9 (1.8)	3.0 (1.8)	2.9 (1.8)	0.54

Characteristics at Enrollment	Gastroparesis (n=284)	Functional Dyspepsia (n=113)	Total (n=397)	P-value
	Mean (SD) / N (%)	Mean (SD) / N (%)	Mean (SD) / N (%)	
Diet subscale	1.8 (1.3)	1.9 (1.4)	1.8 (1.4)	0.49
Relationship subscale	3.3 (1.4)	3.4 (1.3)	3.3 (1.4)	0.86
Psychological wellbeing and distress subscale	3.2 (1.4)	3.2 (1.3)	3.2 (1.3)	1.00
SF-36				
Physical component score	33 (11)	34 (12)	34 (11)	0.83
Mental component score	42 (13)	43 (12)	42 (13)	0.88
PAGI-SYM				
GCSI total score	2.9 (1.1)	2.8 (1.0)	2.8 (1.1)	0.48
Nausea/vomiting subscale	2.1 (1.4)	1.9 (1.2)	2.1 (1.4)	0.15
Nausea	3.2 (1.5)	3.1 (1.5)	3.1 (1.5)	0.50
Retching	1.6 (1.7)	1.5 (1.6)	1.6 (1.7)	0.37
Vomiting	1.6 (1.8)	1.2 (1.6)	1.5 (1.8)	0.06
Post-prandial fullness/early satiety subscale	3.4 (1.2)	3.3 (1.3)	3.4 (1.2)	0.96
Stomach fullness	3.5 (1.3)	3.6 (1.4)	3.5 (1.3)	0.89
Unable to finish meal	3.4 (1.6)	3.4 (1.5)	3.4 (1.6)	0.93
Felt full after meals	3.6 (1.4)	3.7 (1.5)	3.6 (1.4)	0.92
Loss of appetite	2.9 (1.6)	2.8 (1.6)	2.8 (1.6)	0.65
Bloating subscale	3.1 (1.6)	3.1 (1.7)	3.1 (1.6)	0.85
Bloating	3.2 (1.6)	3.2 (1.7)	3.2 (1.7)	0.79
Stomach visibly larger	3.0 (1.8)	2.9 (1.9)	3.0 (1.8)	0.93
Upper abdominal pain subscale	3.0 (1.5)	2.8 (1.5)	2.9 (1.5)	0.37
Lower abdominal pain subscale	2.0 (1.5)	2.0 (1.5)	2.0 (1.5)	0.58
Heartburn/regurgitation subscale	1.9 (1.4)	1.7 (1.3)	1.8 (1.4)	0.22
Constipation	2.5 (1.8)	2.7 (1.7)	2.6 (1.7)	0.40
Diarrhea	1.8 (1.7)	1.5 (1.6)	1.7 (1.7)	0.10
Nausea Profile (0%=none, 100%=severe)				
Overall nausea score	49 (22)	46 (21)	48 (22)	0.25
Somatic distress dimension	49 (27)	44 (26)	48 (27)	0.07
GI distress dimension	74 (23)	73 (23)	73 (23)	0.78
Emotional distress dimension	29 (29)	27 (27)	28 (28)	0.53

*GERD = Gastroesophageal Reflux Disease; PCOS = Polycystic Ovary Syndrome; PDS = Postprandial Distress Syndrome; EPS=Epigastric Pain Syndrome; GI = Gastrointestinal

Table 2.

Symptoms and GMA before and after Water Load Satiety Test in patients with Gastroparesis and Functional Dyspepsia

	Gastroparesis (n=282)	Functional Dyspepsia (n=112)	P-value¶
	Mean (SD)	Mean (SD)	
Water Load Test			
Amount – mL	378 (218)	402 (226)	0.23
Abnormal (< 238 mL) – n (%)	74 (26%)	21 (19%)	0.15
Test length – min	4.4 (2.5)	4.1 (1.7)	0.86
Symptoms (VAS 0-100)			
Fullness			
Baseline	28 (30)	29 (32)	0.98
Change*	37 (29)	38 (32)	0.44
Hunger			
Baseline	30 (31)	33 (32)	0.43
Change*	−5 (26)	−9 (24)	0.07
Nausea			
Baseline	27 (30)	23 (28)	0.17
Change*	5 (16)	9 (21)	0.17
Bloating			
Baseline	28 (31)	25 (29)	0.34
Change*	12 (23)	11 (20)	0.54
Abdominal discomfort			
Baseline	24 (29)	20 (25)	0.36
Change*	6 (16)	8 (15)	0.52
GMA (Distribution of average - %)			
Bradygastria (1-2.4 cpm)			
Baseline	49 (21)	55 (20)	0.02
Change*	0.7 (19.0)	−1.8 (19.7)	0.63
Normogastria (2.5-3.7 cpm)			
Baseline	20 (14)	20 (14)	0.86
Change*	1.5 (14.3)	1.8 (15.0)	0.93
Tachygastria (3.8-10 cpm)			
Baseline	23 (14)	19 (11)	0.01
Change*	−0.7 (11.8)	1.2 (11.8)	0.34
Duodenal (>10-15 cpm)			
Baseline	7 (9)	6 (10)	0.08

	Gastroparesis (n=282)	Functional Dyspepsia (n=112)	P-value[¶]
	Mean (SD)	Mean (SD)	
Change [*]	−1.5 (7.7)	−1.1 (7.4)	0.40

[*] Mean of 3 values taken at 10, 20 and 30 minutes after start of test – Baseline

[¶] P-value derived from robust regression of outcome on GP type for baseline and robust regression of change in outcome on GP type and baseline value of outcome

GMA=gastric myoelectrical activity; GP=gastroparesis

Table 3.

Symptoms and GMA before and after the Nutrient Bar Test Meal in patients with gastroparesis and functional dyspepsia

	Gastroparesis (n=283)	Functional Dyspepsia (n=112)	P-value¶
	Mean (SD)	Mean (SD)	
Nutrient Meal Test			
Water taken with Smart Bar- mL	53 (45)	60 (60)	0.27††
Smart Bar - % consumed	83 (25)	90 (20)	**0.006**††
Test length – min	8.5 (2.4)	8.5 (2.2)	0.38
Symptoms (VAS 0-100)			
Fullness			
Baseline	28 (30)	30 (32)	0.74
Change*	30 (31)	33 (30)	0.33
Hunger			
Baseline	34 (30)	33 (31)	0.93
Change*	−18 (27)	−20 (26)	0.56
Nausea			
Baseline	29 (31)	28 (31)	0.78
Change*	2 (19)	6 (22)	0.55
Bloating			
Baseline	29 (31)	28 (31)	0.67
Change*	9 (20)	9 (21)	0.66
Abdominal discomfort			
Baseline	26 (30)	26 (30)	0.88
Change*	4 (19)	5 (17)	0.11
GMA (Distribution of average - %)			
Bradygastria (1-2.4 cpm)			
Baseline	51 (20)	50 (20)	0.66
Change†	−4 (18)	−3 (18)	0.50
Normogastria (2.5-3.7 cpm)			
Baseline	19 (13)	21 (14)	0.29
Change†	4 (13)	4 (15)	0.92
Tachygastria (3.8-10 cpm)			
Baseline	23 (12)	22 (11)	0.53
Change†	2 (11)	0 (10)	0.22
Duodenal (>10-15 cpm)			
Baseline	7 (8)	7 (8)	0.98

	Gastroparesis (n=283)	Functional Dyspepsia (n=112)	P-value[¶]
	Mean (SD)	**Mean (SD)**	
Change[†]	−1 (7)	−1 (7)	0.12

[*] Mean of 6 values taken at 0, 15, 30, 45, 60 and 90 minutes after start of test – Baseline

[†] Mean of 5 values taken at 15, 30, 45, 60 and 90 minutes after start of test – Baseline

[¶] P-value derived from robust regression of outcome on GP type for baseline and robust regression of change in outcome on GP type and baseline value of outcome

[**] P-value based on Kruskal-Wallis test due to non-convergence using robust regression;

[††] P-value based on t-test

GMA=gastric myoelectrical activity; GP=gastroparesis

Table 4.

Summary of Gastric Myoelectrical Activity Responses to the Water Load Satiety Test (WLST) in Patients with Gastroparesis and Functional Dyspepsia

	Gastroparesis (n=284)	Functional Dyspepsia (n=113)
Dysrhythmic GMA Response *		
Tachygastria	87 (31%)	35 (31%)
Bradygastria	50 (18%)	23 (20%)
Mixed dysrhythmia	25 (9%)	1 (1%)
Hyponormal 3 cpm GMA †	26 (9%)	14 (12%)
Normal 3 cpm GMA Response		
Normal 3 cpm GMA	44 (15%)	21 (19%)
Hypernormal 3 cpm GMA	14 (5%)	10 (9%)
Normal 3 cpm with dysrhythmia ‡	38 (14%)	9 (7%)

*Dysrhythmic GMA and hyponormal 3 cpm occurs in responses to WLST

†Includes patients with hyponormal 3 cpm GMA with increased activity in duodenal-respiratory range (2 patients with GP and 1 with FD in Dysrhythmic GMA response groups)

‡Includes 36 GP patients with tachygastria and 2 with mixed dysrhythmias, 7 FD patients with tachygastria and 2 with mixed gastric dysrhythmias.