Electrogastrography in Patients with Functional Dyspepsia, Joint Hypermobility, and Diabetic Gastroparesis

Abdullah Al Kafee[1], Talar Cilacı[2], Yusuf Kayar[3], Aydın Akan[4]

[1]Department of Biomedical Engineering, İstanbul University, İstanbul, Turkey
[2]Department of Physiotherapy and Rehabilitation, İstanbul Bilim University, İstanbul, Turkey
[3]Division of Gastroenterology, Department of Internal Medicine, Van Education and Research Hospital, Van, Turkey
[4]Department of Electrical and Electronics Engineering, İzmir University of Economics, İzmir, Turkey

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ABSTRACT

Background: *Transcutaneous electrogastrography is a novel modality to assess the human stomach's gastric myoelectrical activity. The purpose of this study was to compare functional dyspepsia, joint hypermobility, and diabetic gastroparesis patients with healthy control subjects in terms of gastric motility abnormalities through electrogastrography evaluations, and to then evaluate the correlation among variations in their blood parameters.*

Methods: *This study analyzed 120 subjects with functional dyspepsia (n = 30), joint hypermobility (n = 30), diabetic gastroparesis (n = 30), and control subjects (n = 30). The electrogastrography parameters included the dominant frequency, dominant power, power ratio, and instability coefficient, which were analyzed preprandially and postprandially. Although there are similar studies in the literature, there is no other study in which all groups have been studied together, as in our study.*

Results: *The electrogastrography results showed that preprandial dominant frequency (P = .031*), dominant power (P = .047*), and instability coefficient (P = .043*), and postprandial dominant frequency (P = .041*) and dominant power (P = .035*) results were statistically significant among the functional dyspepsia, joint hypermobility, diabetic gastroparesis, and control groups.*

There was no significant difference found in terms of power ratio (P = .114) values. However, only glucose (P = .04) and calcium (P = .04*) levels showed statistical significance. Several blood tests including hemoglobin (P = .032*), creatinine (P= .045*), calcium (P = .037*), potassium (P= .041*), white blood cells (P = .038*), and alanine aminotransferase (P = .031*) also showed correlation with the dominant frequency, power ratio, and instability coefficient parameters.*

Conclusions: *This joint methodology demonstrated that it is possible to differentiate between functional dyspepsia, joint hypermobility, and diabetic gastroparesis patients from healthy subjects by using electrogastrography. Moreover, the majority of patients showed adequate gastric motility in response to food.*

Keywords: *Blood test, diabetic gastroparesis, electrogastrography, functional dyspepsia, joint hypermobility*

INTRODUCTION

The stomach's smooth muscles generate spontaneous rhythmic electrical activity that controls the gastric motility, and simultaneously aids the digestion processes.[1] This rhythmic electrical activity of the stomach is called "gastric myoelectric activity," and consists of 2 rhythmic activities: slow-wave activity and spike activity.[2,3] In a healthy individual, the frequency of slow-wave activity is estimated to be 3 cycles/minute (cpm) or 0.05 Hz, which also controls the rhythm and propagation of gastric contractions. Spike potentials are directly associated with gastric contractions.[3,4] Therefore, gastric muscle contractions occur while the slow wave is accompanied by spike potentials.[5] Although with the cutaneous electrogastrography (EGG), it is impossible to learn the spikes' waveform, it can detect the power increase caused by the spikes or slow waves in the stomach.[5]

The pathogenesis of functional dyspepsia (FD), joint hypermobility (JH), and diabetic gastroparesis (GP) remains unclear, but it has been proposed that they might have common pathophysiological findings.[4,6-8] (EGG) is a rather new noninvasive technique that does not diagnose a specific syndrome. However, it delivers the information in terms of rhythmic activity, gastric slow waves in terms of dysfunction in amplitude, power, and frequency ranges. Different electrophysiological abnormalities play significant roles in gastrointestinal (GI) motility disorders such as FD, JH, and GP.[2,7,9] At present, the EGG method is utilized in GI motility disorders for

Corresponding author: ***Abdullah Al Kafee****, e-mail:* ***a_alkafee@yahoo.com***
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many conditions such aspostural tachycardia syndrome, stomach cancer, abnormal gastric motility observed in systemic sclerosis, pregnancy, recurrent vomiting, and diabetes mellitus.[1,3,8-12]

The purpose of our study was to examine functional dyspepsia (FD), joint hypermobility (JH), and diabetic gastroparesis (GP) patients, along with healthy control subjects (CT) with regard to preprandial and postprandial EGG abnormalities. Further, we investigated the possible correlation between EGG abnormalities and their blood parameter variations.

MATERIALS AND METHODS

Subjects

This study's medical procedures were performed by a gastroenterologist, a nuclear radiologist, and an expert physician approved by the university ethics committee. A total of 120 subjects, 30 patients with FD, 30 patients with JH, 30 patients with GP, and 30 healthy subjects (CT) were included in the study. None of the participants were taking medicines that might affect GI motility. All of the participants underwent fasting blood tests, their blood parameters were measured, including glucose, blood urea nitrogen (BUN), creatinine (Cr), hemoglobin (Hb), calcium (Ca), sodium (Na+), potassium (K+), aspartate aminotransferase (AST) platelets (PLT), alanine aminotransferase (ALT), white blood cells (WBC), hemoglobin A1c (HbA1c), and thyroid-stimulating hormone (TSH). Asymptomatic participants who had all of their abdominal ultrasounds, esophagogastroduodenoscopy (EGD), Beighton score, and fasting blood test reports in the normal range according to the reference chart were included in the CT group. Abdominal ultrasonography (USG) was performed for all patients. Video esophagogastroduodenoscopy (EGD) was performed by an experienced gastroenterologist (AD) under conscious sedation following an 8-hour fasting period and intravenous injection of midazolam. Focal lesions, esophagitis, gastric or duodenal ulcers, erosions, and malignancy were investigated. Biopsies were taken from the stomach antrum and corpus to test for *Helicobacter pylori*. Patients with pathology detected by the laboratory, ultrasonography, and endoscopic tests, those with cardiovascular, gastrointestinal (GI), pulmonary, hepatic, renal, metabolic, neurological, and psychiatric diseases, malignancy, gastroesophageal reflux disease, irritable bowel disease, pregnancy, major abdominal surgery history, and gastric movement, and those undergoing the treatments that affect the disease were excluded from the study.

Functional Dyspepsia Group

Patients fulfilling the Rome III criteria for diagnosed functional dyspepsia were included in the FD group.[9,13] To exclude any organic diseases, abdominal USG, scintigraphy test, and EGD were performed. Following an 8-hour fasting period, all the patients were injected with midazolam intravenously, and an experienced gastroenterologist performed video EGD under conscious sedation. Thirty subjects were included in the final analysis.

Joint Hypermobility (JH) Group

An expert physician diagnosed joint hypermobility with a 9-point Beighton score based on joint flexibility. Only subjects with a Beighton score of 6/9 were included.[14-18] If the subjects had a BMI of $\geq$ 30, metabolic or organic diseases, vomiting, nausea, diarrhea, or other symptoms, had undergone abdominal surgery, or were taking drugs affecting GI motility, they were excluded. Thirty subjects were included in the final analysis.

Gastroparesis Group

A standardized questionnaire was used to assess symptoms indicating gastroparesis.[19] Patients with anorexia, nausea, vomiting, and feeling of early satiety, stomach fullness, and abdominal discomfort were evaluated. The gastric emptying scintigraphy (GES) test, which is a noninvasive procedure in which the gastric emptying rate is measured in patients with existing complaints, was performed by a nuclear medicine specialist. This test requires the ingestion of an international-standard test meal (usually scrambled egg with slices of bread and 330 mL juice) driven by radionuclides. The test is done in the morning after fasting overnight. Agents that can accelerate or delay gastric emptying should be discontinued 48 $\pm$ 72 hours before the procedure. Diabetic patients are instructed to take half their normal morning dose of insulin to avoid hyperglycemia that can delay gastric emptying. Gastric emptying was reported as the percentage of the meal emptied (or the percentage of the food held after a certain time) or the time to empty 50% of the meal.

For the evaluation of gastric emptying by scintigraphy, scanning was started in a static acquisition mode immediately after ingestion for each patient (time 0). Then, the study was repeated at post-ingestion times of 30 minutes, 60 minutes, 90 minutes, 2 hours, and also 4 hours. Delayed gastric emptying or gastric retention was defined as 90% retention at the 1-hour, more than 60% at the 2-hour, and more than 10% at the 4-hour scans. Patients

whose food was emptied by less than half during this period were evaluated as having gastroparesis.[20,21] Even though patients with clinical complaints suggestive of gastroparesis had a minimum of 6 months of chronic history, the scintigraphy test reports that did not show delayed gastric emptying were not included in the study. Thirty subjects were included in the final analysis.

EGG

Thirty minutes of EGG recording was performed over the fasting stage (preprandial), and a 10-minute break was provided for eating the test meal. After 60 minutes (postprandial), the recording was done. The 500 kcal standard test meal for all subjects consisted of one fried egg, two pieces of toast, and a cherry juice (330 mL) (protein, fat, and carbohydrate). EGG was assessed with three-channel electrogastrography by using surface electrodes placed over the stomach, on the abdominal skin. The first active electrode was placed on the midline, half-way between the xiphoid process and umbilicus. In contrast, the second active electrode was attached between the first electrode and the lower rib. The reference electrode (Ref) was placed on the left lower quadrant of the left costal edge.[2,9] In addition, the respiration rate was recorded using an attached belt placed on the upper chest under the armpits. All these channels were connected to the 3 CPM EGG device (Towson, MD, USA) with the United States Food and Drug Administration (FDA) approval. All the recorded EGG signals were filtered through the EGG software analysis system (EGGSAS) program. These EGG signals were digitally stored with a maximum sampling frequency of 4 Hz.

A running spectral analysis (RSA) based on the fast Fourier transform (FFT) method was applied to extract the features of EGG signals. In the RSA method, EGG signals were divided into predefined segments; then, FFT was applied for each segment. The power spectrum of the total recording that had the highest peak power on this frequency spectrum, in the range between 0.5 cpm and 9.0 cpm, was represented as the EGG dominant frequency (DF). The gastric dominant frequency is nearly 3 cpm in healthy humans; thus, a 2.5-3.5 cpm range was defined as normal. The DF of EGG signal less than 2.5 cpm was called bradygastria, where the number of antral contractions was decreased because of the bradygastric activity. Dominant frequency greater than 3.5 cpm was called tachygastria.[3,5,7,22] Dominant power (DP) is the power value observed at DF.[1,23,24] The instability of the myoelectrical frequency was also analyzed by the instability coefficient (IC), which was identified as the standard deviation divided by the mean value of frequency. The power ratio (PR) was computed as the postprandial value of power divided by the preprandial value of power.[2,24] The DF, DP, PR, and IC features were computed to evaluate each subject in both preprandial and postprandial periods.

Statistical Analysis

All EGG signal processing and statistical analyses were carried out using MATLAB version R2018b software (Math Works Inc., Natick, Mass, USA). The one-way ANOVA was applied for comparison among the parameters of the 4 groups. Moreover, the Student's *t*-test was performed to analyze the differences between 2 groups. Correlation between blood analysis and EGG parameters were studied by linear regression analysis. Power analysis was done before the experimental design. Although the calculated sample size was 28, we recruited 30 patients for each group. In the analyses, the *P* value of less than .05* was accepted as statistically significant.

RESULTS

This study was performed on a total of 120 subjects. Their demographic features and initial biochemical values are shown in Table 1.

Blood Analysis

The blood test parameters of the 4 groups (glucose, Hb, BUN, Cr, Na, K, Ca, AST, ALT, HbA1c, WBC, TSH, and PLT) were analyzed simultaneously. Only glucose and calcium ($P = .04^{*}$ and $.04^{*}$, respectively) were statistically significant among the 4 groups (Table 1). Moreover, specific blood test parameters such as Hb, Cr, Ca, K, WBC, and ALT were correlated with the EGG parameter. In the CT group, preprandial DF was correlated with Hb, Cr, and Ca (*P* = .032*, .045*, and .037*, respectively). Furthermore, in the FD group, preprandial IC correlated with K (*P* = .041*). Moreover, in the JH group, the power ratio correlated with Hb (*P* = .038*). The GP postprandial IC also showed correlation with WBC (*P* = .002*). Similarly, preprandial IC was correlated with ALT (*P* = .031*).

EGG Signal Parameters

The preprandial DF, DP, and IC parameters were analyzed using one-way ANOVA. The FD, JH, and GP groups demonstrated (*P* = .07) lower incidence of normal slow waves compared to the CT group [FD: 6 (20%), GP: 4 (13.3%), JH: 11 (36.67%), and CT: 25 (83.3%)] and a higher rate

Table 1. Demographic Characteristics of the FD, JH, GP, and CT Groups

	FD Group, Mean ± SD	JH Group, Mean ± SD	CT Group, Mean ± SD	GP Group, Mean ± SD	*P*
Age	35.4 ± 9.3	23.36 ± 3.12	36.58 ± 7.6	38.9 ± 8.3	.82
Gender (F)	24 (80%)	26 (86.67%)	23 (76.67%)	25 (83.3%)	.74
Height	163.4 ± 6.4	166.8 ± 0.05	165.63 ± 7.3	162.3 ± 3.5	.07
Weight	66.6 ± 12.9	56.9 ± 10.2	66.8 ± 14.4	76.5 ± 10.6	.94
BMI	23.8 ± 4.3	20.3 ± 3.08	23.7 ± 2.8	24.6 ± 2.9	.41
WBC	7.62 ± 1.52	7.7 ± 1.6	7.86 ± 1.92	8.06 ± 1.44	.29
Hb	13.29 ± 1.38	13.55 ± 1.3	13.60 ± 1.61	12.48 ± 1.24	.85
PLT	261.16 ± 67.55	264.23 ± 62.16	257.23 ± 60.36	297.10 ± 79.22	.85
Glucose	91.43 ± 10.56	92.63 ± 8.70	91.63 ± 9.0	195.37 ± 104.15	.04*
HBA1c	5.308 ± 0.22	5.1 ± 0.26	4.9 ± 0.90	8.586 ± 1.61	.90
TSH	1.96 ± 1.18	1.84 ± 0.43	1.70 ± 0.66	2.10 ± 1.09	.33
BUN	9.76 ± 2.67	10 ± 1.28	10.9 ± 2.79	15.97 ± 4.48	.05
Cr	0.59 ± 0.12	0.58 ± 0.21	0.59 ± 0.13	0.7 ± 0.19	.62
AST	18.7 ± 6.34	20.3 ± 4.38	19.93 ± 3.98	22.20 ± 9.12	.82
ALT	19.46 ± 15.13	18.3 ± 4.9	17.5 ± 6.6	24.27 ± 12.17	.25
Na	139.6 ± 1.54	139.6 ± 1.8	139.6 ± 1.3	139.82 ± 2.50	.15
K	4.50 ± 0.57	4.1 ± 0.32	4.12 ± 0.82	4.47 ± 0.45	.89
Ca	9.53 ± 0.40	9.1 ± 0.15	9.25 ± 0.37	9.2 ± 0.42	.04*

FD, functional dyspepsia; JH, joint hypermobility; GP. diabetic gastroparesis; CT, control group; BUN, blood urea nitrogen; Cr, creatinine; Hb, hemoglobin; Ca, calcium; Na, sodium; PLT, platelets; K, potassium; AST, aspartate aminotransferase; ALT, alanine aminotransferase; WBC, white blood cells; HbA1c, hemoglobin A1c; TSH, thyroid-stimulating hormone; BMI, body mass index; F, female; SD, standard deviation. *$P < .05$.

of bradygastria than the CT group [FD: 24 (80%), GP: 20 (66.6%), JH: 19 (63.33%) and CT: 5 (16.6%)]. Additionally, only the GP group had tachygastria [6 (20%)]. A statistically significant difference ($P = .031$*) was found between the preprandial DF values of the FD (2.28 ± 0.41), JH (2.31 ± 0.11), GP (2.24±0.14), and CT (2.4 ± 0.27) groups. In addition, the DP values of the FD (1.3 ± 0.48), JH (1.5 ± 0.21), GP (1.1 ± 0.31), and CT (2.2 ± 0.75) groups were significantly different ($P = .047$*). Moreover, the IC values of the FD, JH, GP, and CT groups (0.6 ± 0.23, 0.5 ± 0.16, 0.6 ± 0.199, and 0.37 ± 0.24 respectively) were significantly different ($P = .043$*) (Table 2).

The postprandial DF, DP, and IC parameters were analyzed by using one-way ANOVA methods. The rate of normal postprandial DF values in the FD, JH, and GP groups was lower compared to the CT group [FD: 4 (13.3%), JH: 10 (33.33%), GP: 5 (16.6%), and CT: 24 (80%)], whereas the rates of bradygastria [FD: 24 (80%), JH: 20 (66.67%), GP: 19 (63.3%), and CT: 6(20%)] and tachygastria [FD: 2 (6.7%) and GP: 6 (20%)] were higher in the GP group. Even though the FD (2.34 ± 0.3), JH (2.35 ± 0.27), and GP (2.29 ± 0.12) groups showed lower rates of DF than the CT (2.43 ± 0.33) group, the difference was statistically significant ($P = .041$*). Furthermore, the DP values of FD (1.4 ± 0.6), JH (1.7 ± 0.13), CT (2.3 ± 1.4), and FD (1.2 ± 0.4) groups were significantly different ($P = .035$*). In addition, the IC values of FD, JH, GP and CT groups (0.59 ± 0.15, 0.52 ± 0.11, 0.58 ± 0.21, 0.44 ± 0.17) were not significantly different ($P = .061$) (Table 2).

Furthermore, the FD, JH, GP, and CT groups were compared with respect to both preprandial and postprandial states on the values of DF, IC, and DP parameters, using Student's *t*-test. In the postprandial state, increased DF values were noticed in all FD [pre (2.28 ± 0.41) post (2.34 ± 0.3), ($P = .181$)]; JH [pre (2.31 ± 0.11) post (2.35 ± 0.27), ($P = .78$)]; GP [pre (2.24±0.14) post (2.29±0.123), ($P = .051$)]; and CT [pre (2.4 ± 0.27), post (2.43 ± 0.33), ($P = .04$*)] groups. However, this increase was significant only for the control group ($P = .04$*). Dominant power in CT subjects showed increase after food ingestion, compared with the preprandial state ($P = .024$*) (Table 3). Similarly, in comparison with the preprandial condition, the patients with

Table 2. Comparison Between the Groups with Respect to Preprandial and Postprandial EGG Parameters

EGG Parameters	FD (n = 30)	JH (n = 30)	CT (n = 30)	GP (n = 30)	*P*
Preprandial					
Normal n (%)	6 (20%)	11(36.67%)	25 (83.3%)	4 (13.3%)	
Bradygastria n (%)	24 (80%)	19 (63.33%)	5 (16.6%)	20 (66.6%)	.47
Tachygastria n (%)	0%	0%	0%	6 (20%)	
DF (cpm)	2.28 ± 0.41	2.31 ± 0.11	2.4 ± 0.27	2.24 ± 0.14	.031*
DP	1.3 ± 0.48	1.5 ± 0.21	2.2 ± 0.75	1.1 ± 0.31	.047*
IC	0.6 ± 0.23	0.5 ± 0.16	0.37 ± 0.24	0.6 ± 0.19	.043*
Postprandial					
Normal n (%)	4 (13.3%)	10 (33.33%)	24 (80%)	5 (16.6%)	
Bradygastria n (%)	24 (80%)	20 (66.67%)	6 (20%)	19 (63.3%)	.082
Tachygastria n (%)	2 (6.7 %)	0%	0%	6 (20)%	
DF (cpm)	2.34 ± 0.3	2.35 ± 0.27	2.43 ± 0.33	2.29 ± 0.12	.041*
DP	1.4 ± 0.2	1.7 ± 0.13	2.3 ± 1.4	1.2 ± 0.4	.035*
IC	0.59 ± 0.15	0.52 ± 0.11	0.44 ± 0.17	0.58 ± 0.21	.061

EGG, electrogastrography; FD, functional dyspepsia; JH, joint hypermobility; GP, diabetic gastroparesis; CT, control group; DF, dominant frequency; DP, dominant power; IC, instability coefficient; cpm, cycles per minute. *$P < .05$.

Table 3. Comparison Between the Groups with Respect to Preprandial and Postprandial DF, IC, and DP

EGG Parameters	Preprandial, Mean ± SD	Postprandial, Mean ± SD	*P*
FD (n = 30)			
DF (cpm)	2.28 ± 0.41	2.34 ± 0.3	.181
IC	0.6 ± 0.23	0.59 ± 0.15	.876
DP	1.3 ± 0.48	1.4 ± 0.6	.075
JH (n = 30)			
DF (cpm)	2.31 ± 0.11	2.35 ± 0.27	.78
IC	0.5 ± 0.16	0.52 ± 0.11	.36
DP	1.5 ± 0.21	1.7 ± 0.13	.06
CT (n = 30)			
DF (cpm)	2.4 ± 0.27	2.43 ± 0.33	.04*
IC	0.37 ± 0.24	0.44 ± 0.17	.401
DP	2.2 ± 0.75	2.3 ± 1.4	.024*
GP (n = 30)			
DF (cpm)	2.24 ± 0.14	2.29 ± 0.12	.051
IC	0.6 ± 0.19	0.58 ± 0.21	.464
DP	1.1 ± 0.31	1.2 ± 0.4	.094

EGG, electrogastrography; FD, functional dyspepsia; JH, joint hypermobility; GP, diabetic gastroparesis; CT, control group; DF, dominant frequency; cpm, cycles per minute; IC, instability coefficient; DP, dominant power; SD, standard deviation. *$P < .05$.

FD [pre (1.3 ± 0.48), post (1.4 ± 0.6), (P = .075)], JH [pre (1.5 ± 0.21), post (1.7±0.13), (P = .06)], GP [pre (1.1 ± 0.31), post (1.2 ± 0.4), (P = .094)] showed an overall increase in the DP after food ingestion. Moreover, statistically significant differences were not detected in the EGG power ratio among the 4 groups (Table 4).

DISCUSSION

This study establishes that EGG is an essential and irreplaceable non-invasive test for analyzing abnormal gastric myoelectric activity in FD, JH, and diabetic GP patients (Figures 1 and 2), and numerous EGG features have a significant correlation with blood analysis parameters. The underlying pathophysiology of FD, JH, and GP patients may have gastric dysmotility or non-coordinated gastric contractions, and complication occurs due to abnormal gastric slow waves, which also increases the amplitude of postprandial gastric slow waves.[8,9,21] Currently, most researchers are focused on the natural pacemaker of the gastric electrical activity originating from the upper corpus to move toward the pylorus.[9,25] Therefore gastric myoelectrical activity is a significant factor in the pathophysiology of FD, JH, and GP.

FD is usually diagnosed on the basis of symptom profile according to the Rome III criteria.[14] Therefore, patients fulfilling the Rome III diagnostic criteria were included in

Table 4. Comparison of the Groups with Respect to Power Ratio

	FD (n = 30), Mean ± SD	JH (n = 30), Mean ± SD	CT (n = 30), Mean ± SD	GP (n = 30), Mean ± SD	*P*
Power ratio	2.17 ± 1.76	2.1 ± 1.7	2.08 ± 1.2	2.25 ± 3.4	.114

FD, functional dyspepsia; JH, joint hypermobility; GP, diabetic gastroparesis; CT, control group; SD, standard deviation; cpm, cycles per minute. *$P < .05$.

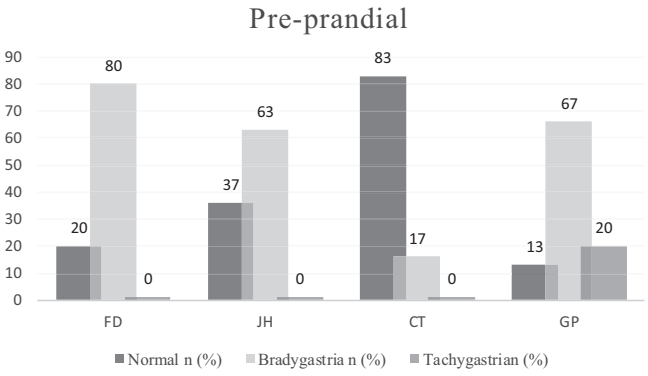

Figure 1. Preprandial EGG analysis in the JH, FD, GP, and CT groups. (JH, joint hypermobility; FD, functional dyspepsia; GP, diabetic gastroparesis; CT, control group).

the FD group. As a non-inflammatory hereditary rheumatologic disease, JH may cause various system abnormalities. For that reason, only JH subjects with a Beighton score 6/9 were included in this study.[8,15,16] Additionally, patients diagnosed with diabetes mellitus I and II were not selected, and only patients with diabetic GP were included in the study.

There are no studies comparing FD, JH, and GP groups with a healthy control group in the literature. Most of the comparison studies had been done between only 2 groups.[3,8,9,17] So far, controversial results have been observed in the studies that were conducted between

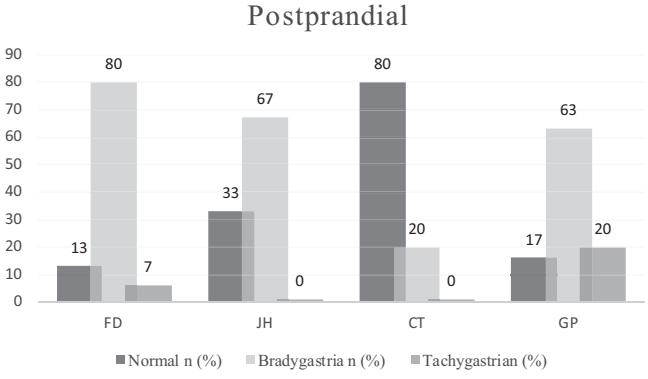

Figure 2. Postprandial EGG analysis in the JH, FD, GP, and CT groups. (JH, joint hypermobility; FD, functional dyspepsia; GP, diabetic gastroparesis; CT, control group).

FD, JH, or GP patients with healthy controls in terms of EGG parameters.[2,8,14] Different methods have been applied in various studies for FD and GP analysis, but they offer only limited comparability.[2,7,26] Several studies have found significant differences between the GP or FD and the healthy group with respect to the gastric rhythm differences.[9,25,27] Studies have also reported that GP patients have a higher incidence of gastric dysrhythmias and delayed gastric emptying, similar to the patients with FD.[9,19] Gastric motility abnormalities are also seen in rheumatologic diseases. McNearney et al.[28] compared 10 systemic sclerosis (SSc) patients with 13 healthy age-matched controls. They found that patients with SSc had significantly lower gastric slow-wave regularity than the control group, both in preprandial and postprandial conditions. The study also revealed significantly higher rates of bradygastria in SSc patients. These findings are consistent with ours, as rheumatologic disease, the JH group had higher rates of bradygastria. The JH group's mean DF value was statistically significantly lower than the CT group in both states.

Similarly, this study presented significantly higher values of dysrhythmia in the FD, JH, and GP groups compared to the control group. The dominant power (DP) reflected the amplitude of gastric myoelectrical activity, and the PR reflected the increase in gastric contractions. The EGG and DP increased in the CT subjects and patients with FD, JH, and GP after food ingestion. These data suggest that the gastric contractile activity was increased after food ingestion. Moreover, the physiological EGG signal features and the clinical blood parameters have not always been correlated.[11,12,29-31] However, our observation proved that some blood test results, such as Hb, Cr, Ca, K, WBC, and ALT correlated with EGG parameters. These findings may be helpful for understanding, diagnosing and planning the treatment of the GI symptoms seen in FD, JH and GP patients.

In the studies conducted, contradictory results are reported in comparing FD patients and the control group.[32] In the study conducted on FD patients, the rate of gastric arrhythmia was found to be 36%, and it was reported that it was found to be significantly higher than the control group[32,33] In 60% of FD patients, it was

observed that the postprandial dominant power did not increase sufficiently in most cases, along with gastric slow waves and dysrhythmia.[33] Our study showed that preprandial DF and DP levels were lower, IC levels were higher, and bradygastria was higher in FD patients, similar to GP patients. We also showed that the rate of postprandial bradygastria and tachygastria was higher, DF and DP levels were low, and the IC level was higher. Similar results in FD and GP patients suggest a common pathogenesis. Antral hypomotility, electrical activity changes, and delayed gastric emptying have been the important mechanisms studied. The absence of fundal loosening increases intragastric pressure and causes food to migrate from the proximal end to the distal end. This leads to antral excessive deviation and causes upper abdominal discomfort, bloating, and nausea. In addition, increased sensitivity to gastric distention due to visceral hyperalgesia plays a role in the development of these symptoms.[34]

In GP patients, preprandial and postprandial bradygastria and tachygastria were found to be higher than in other groups. Hasler et al[35] showed that antral motility was significantly decreased in healthy people when blood glucose levels were above 230 mg/dL, and consequently, gastric emptying was delayed.[35] It was observed that antral motility was not affected even in the hyperinsulinemic state created by adding insulin. In this study, it was shown that hyperglycemia caused tachygastria via the prostaglandin-sensitive pathway. When these results were evaluated, it was observed that chronic hyperglycemia in GP patients, and acute hyperglycemia in healthy individuals trigger the mechanisms that cause tachygastria in the stomach. Moreover, gastric emptying scintigraphy (GES) of solid and liquid nutrient test meals utilizes different motor mechanisms and results in different gastric emptying patterns. Many studies have been conducted regarding the different GES of solid and liquid meals, with controversial results.[36,37] Therefore, the Society of Nuclear Medicine (SNM) and the American Neuro Gastroenterology and Motility Society recently agreed on a standard meal and imaging protocol to measure gastric emptying.[38] In this study, the international standard test meal was used during the GES test. Similar to other EGG research, this study may also have limitations in the long-term. EGG signal recording, surface electrode location or position, type of food or calories, computed features, and study populations are not standardized yet, which could be considered the key factors limiting our study's simplification.

In conclusion, these findings and results establish that cutaneous EGG recording produces reliable outcomes to discriminate the healthy subjects from FD, JH, and GP patients regarding gastric motility abnormalities. Moreover, FD, JH, and GP patients show adequate gastric motility in response to food. However, many issues and further research are required to make the EGG an irreplaceable noninvasive test to diagnose and follow up on gastric motility disorders. The mechanisms of gastric myoelectric activity in FD, JH, and GP patients are still not clear, and future modifications in EGG performance are needed.

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