

## Electrogastrographic (EGG) and Gastric Emptying Changes in Patients with Duodenal Ulcer .The Effect of *H. Pylori* Eradication

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**Abstract:** *Helicobacter pylori* infection and the use of non-steroidal anti-inflammatory drugs (NSAIDs) are now generally accepted as critical factors in the pathogenesis of peptic ulcer (PU). *Helicobacter pylori* (*H. Pylori*) infection is associated with over 90% of duodenal ulcer. *Helicobacter Pylori* infection may result in several alternation of gastro-duodenal physiology. **Aim of the work:** was to study the possible gastric myo-electrical activity changes in patients with *H. Pylori* associated duodenal ulcer and their relation to gastric emptying, as well as the effect of *H. Pylori* eradication on such changes. **Patients and methods:** 60 subjects were enrolled in this study and classified into three groups: Group I: Twenty patients with *H. Pylori* infection associated with duodenal ulcer Group II: Twenty patients with *H. Pylori* negative duodenal ulcer. Group III: Twenty age and sex matched "healthy" asymptomatic volunteers; ten positive and (IIIa) ten negative (IIIb) for *H. Pylori*. For all patients; complete history and physical examination, upper endoscopy and pyloric antral biopsy for *H. Pylori* detection, abdominal ultrasound, and electro-gastrogram were performed. **Results:** in group I patients mean of power ratio (PR) was lower than that of group IIIa (1.42±0.74 versus 2.94±1.9 ( $P<0.01$ ), while, gastric emptying parameters showed insignificant difference. No significant difference was found between group II and group IIIb as regards EGG parameters, while full time and ½ emptying time in-group II was significant by lower than group IIIb. (13±2.8min, 15.7±2.8min. respectively  $P<0.05$ , and 4.89±2.2min, versus 6.7±2.48min for ½ emptying time  $P<0.05$ ). After eradication therapy 87% of our patients became negative for *H. Pylori*, EGG repeated for group I patients after eradication. The mean of % power at rest, The mean of %CPM at meal, and The mean of PR was significantly lower before than after treatment ( $P<0.05$ ,  $P<0.05$ , &  $P<0.01$ ). The mean of power at rest of DF was statistically significant higher before than after treatment  $P<0.01$ . Mean of 1/2 in-group I patients was higher before than after eradication therapy). **Conclusion:** *Helicobacter pylori* infection can affect gastric motility, especially in duodenal ulcer infected patients. Eradication therapy improved not only EGG abnormalities but also aids in restoration of abnormal water gastric emptying.

[Samia A. Abdo, HishamS. Abdel Fattah and Uomna Kamel. **Electrogastrographic (EGG) and Gastric Emptying Changes in Patients with Duodenal Ulcer. The Effect of *H. Pylori* Eradication.** *Life Sci. J* 2013;10(1):3081-3088]. (ISSN: 1097-8135). <http://www.lifesciencesite.com>. 381

**Keywords:** Electrogastrography (EGG), gastric motility, *Helicobacter Pylori*, duodenal ulcer.

### 1. Introduction

Duodenal ulcer is one of the most common gastrointestinal disorders, with a lifetime prevalence of 4–15%. *Helicobacter pylori* (*H. pylori*) is a spiral bacterium that infects the lining of the stomach and predisposes the patient to gastritis and ulcer. *H. pylori* infection is observed in 70–90% of individuals with duodenal ulcer (1). *H. Pylori* infection may result in several alternation of gastro duodenal physiology (2) and have been proposed as the main mechanisms leading to duodenal ulcer.

Various pathogenic factors originating from *H. pylori* have been implicated in the effect of this bacterium on the gastric mucosa (3). *H. pylori* infected patients had decreased in the antral somatostatin concentrations as well as in the somatostatin levels of gastric fluid. *H. pylori* infection changes the concentration of regulatory peptides such as

somatostatin, gastrin and cholecystokinin in the gastric mucosa. Somatostatin is the major inhibitory hormone of gastrointestinal secretion and motility (4) Konturek *et al.*, found that *H. pylori* infection in DU patients is accompanied by enhanced gastric emptying that can be reversed by the eradication of *H. pylori*. They concluded that both CCK and somatostatin might contribute to normalization of gastric emptying following *H. pylori* eradication in DU patients (2).

Gastric myoelectrical activity can be recorded by using electrogastrography, a non-invasive way by means of electrodes attached to the abdominal skin. Electrogastrography (EGG) is a reliable and accurate technique for the measurement of gastric myoelectrical activity. It provides information about the frequency and regularity of the gastric slow wave (5). Unlike the electrocardiogram with which a physician can perform diagnosis by examining the tracing, the EGG must be

subjected to computerized spectral analysis. This is because the EGG signal is weighted summation of all gastric slow waves presented in the stomach.

EGG recordings were analyzed to derive the following parameters: (1) percentage of normal gastric waves (2.0-4.0 cpm), bradygastric waves (1.0-2.0 cpm), and tachygastric waves (4.0-10.0 cpm); (2) dominant frequency (DF); (3) dominant power (DP); (4) The early postprandial frequency dip and (6) the ratio of post-prandial to fasting in DP. A percentage of normal slow wave frequency of more than 70% was defined as normal (5). The EGG power ratio is believed to be associated with gastric contractility; the increase in the EGG power ratio reflected an increase in gastric contractions.

*H. pylori* eradication was highly efficient using eradication therapy. Eradication treatment using various regimens is a standard measure for *H. pylori* infected duodenal ulcer patients (6) Eradication of infection provides a long term cure of duodenal ulcers in more than 80% of patients whose *H. pylori* was positive. If *H. pylori* colonization indeed disturbs gastric emptying, the restored gastric emptying will be obtained after effective *H. pylori* eradication (7).

**Aim of the work** was to assess the possible gastric myoelectrical activity changes in patients with *H. pylori* positive duodenal ulcer and their relation to gastric emptying, as well as the effect of *H. pylori* eradication on such changes.

## 2. Patients and Methods

Sixty subjects attending Ain Shams University Hospital and Misr University for Science and Technology Hospital were enrolled in this study, and classified into three groups:

Group I: Twenty patients with *H. pylori*- positive ulcer.

Group II: Twenty patients with *H. pylori*- negative duodenal ulcer.

Group III: Twenty age and sex matched "healthy" asymptomatic volunteers; divided into two subgroups; group IIIa, ten *H. pylori* positive and group IIIb, ten *H. pylori* negative subjects. *H. pylori* status in the control group III was assessed by absence of *H. pylori* antigen in the stool by using: The Femto Lab *H. pylori* Cnx enzyme immunoassay (EIA) The detection of the antigen is based on the principle of enzyme linked immunosorbent assay (ELISA).

### Exclusion criteria:

1. Patients with diabetes mellitus.
2. Patients with end-organ failure
3. Patients with calculargall bladder.
4. Pregnant females.
5. Patients taking drugs affecting gastric motility (e.g. prokinetics, anticholinergics, antibiotics like

macrolides, dopaminergic agonists, adrenergic agents, morphine, contraceptive pills).

6. Patients with previous abdominal surgery.
7. Patients with systemic illness.

### All participants were subjected to the following:

Patients and volunteers gave informed consent prior to the investigations

#### 1. Upper gastrointestinal endoscopy:

The endoscopy was done in Ain Shams University Hospital and Misr University For Science And Technology Hospital in the endoscopy theater by Olympus GIF Type 2 T 200 and GIF 180 video scope gastroduodenoscope.

The endoscopy was cleaned mechanically and by detergent, disinfectant glutaraldehyde and rinse in tap water after each examination for elimination of *H. pylori* and other bacterial transmission through the endoscope.

The patients were fasting for at least 6 hours before the endoscopic examination. Two biopsies from the antrum and corpus were taken and were prepared for histopathological examination after being kept in formaldehyde 10%.

#### 2. Histopathological examination:

Gastric biopsies were routinely processed to paraffin block and 3mm sections were cut. One of the two biopsies kept in formaldehyde 10% stained with haematoxylin and eosin stain and was subjected to histopathological examination. Grading of gastritis was carried out, the mucosa designated as being normal unless inflammatory cells were present in greater than usual numbers. Identification of *H. pylori* in all specimens was made by viewing the haematoxylin and eosin (8).

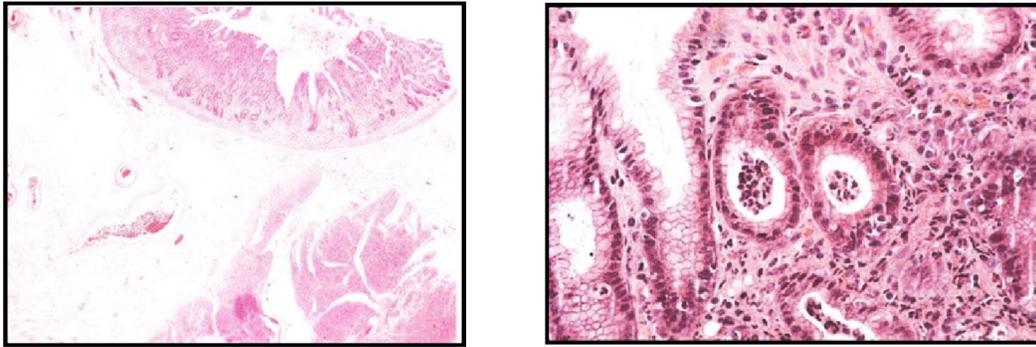
A subject was considered infected with *H. Pylori* only, if typical curved or S shaped bacilli approximately 1 to 3mm long and 0.5mm wide were observed with a rose colored with haematoxylin and eosin stain.

#### 3. Assessment of gastric emptying by ultrasonography:

All subjects were fasting for at least 6 hours before examination; abdominal U/S was performed with 3.5 array transducer.

#### Technique:

Gastric examination was performed by a curvilinear array, low-frequency (3.5-MHz) transducer Toshiba system. Patients were scanned in the supine position. The antrum is usually best visualized in a parasagittal plane just right of the midline, surrounded by the left lobe and caudate lobe of the liver anteriorly and the head or neck of the pancreas posteriorly.



**Fig. (1):** Histopathology of *H. Pylorigastritis*.

The left lobe of the liver, the Inferior vena cava, and the superior mesenteric vein were used as internal landmarks. The two vessels are usually visualized slightly to the right of the abdominal midline. Once these vessels were identified, the transducer was rotated slightly clockwise or counterclockwise to best obtain a true cross-sectional view of the antrum (the smallest possible cross-sectional view). The anteroposterior and craniocaudal diameters were measured in this view (9).

The antrum has a characteristic multilayered wall. The antrum was judged to be empty if it appeared flat, with anterior and posterior walls juxtaposed. The antrum was judged to contain fluid if it appeared to have an endocavitary lumen with hypochoic content and distended walls.

Patient's sitting position and small volume of liquid meal (<300ml water) are safe factors against floating of gastric contents in the corpus and fundus. *The following markers of gastric motility were measured:*

- a) **Basal antral area:** was the mean of two measurements taken 0 and 5 min. before the meal.
- b) **Maximal postprandial antral area:** was measured after maximal widening of the antrum had occurred, usually within 2 min. postprandial.
- c) **Minimal postprandial antral area:** was the smallest area measured at any time postprandially.
- d) **Half emptying time:** was the time in minutes to observe a 50% decrease in maximal antral area ( $t \frac{1}{2}$  time). Calculated by linear regression analysis from the linear part of antral emptying curve. Antral emptying curves were obtained by plotting antral area vs. time(10).

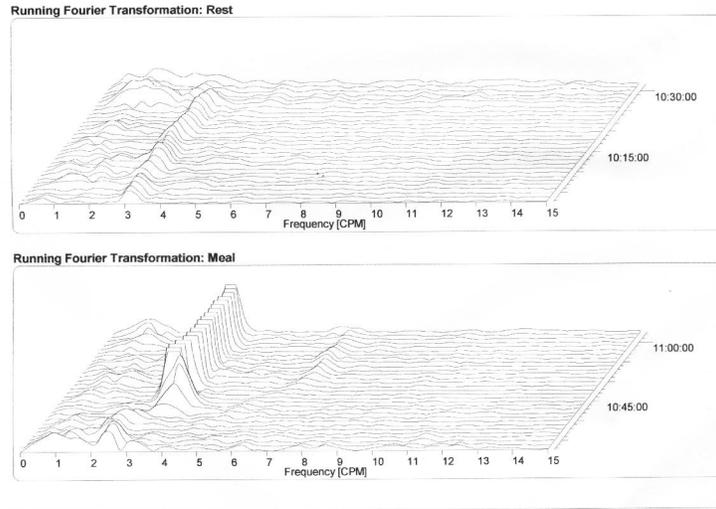
#### 4. *Electrogastrography:*

Gastric electrical activity was recorded from five disposal pregelated silver/silver chloride surface electrodes placed on the upper abdomen. This was

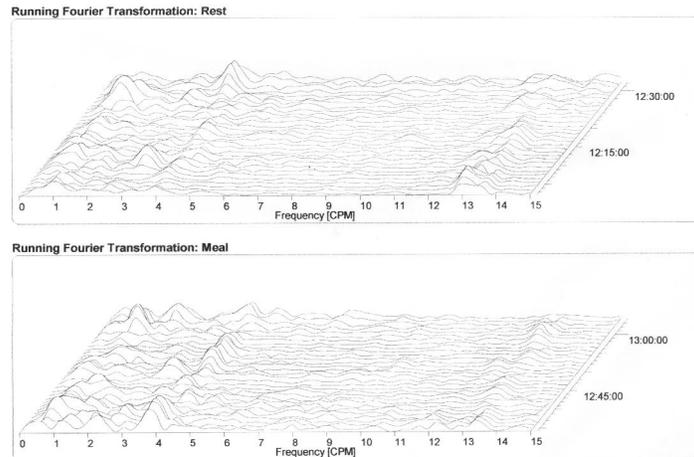
done after the skin has been carefully abraded to decrease resistance to obtain a good signal to noise ratio. One hour recording while the patient is fasting was done, then given a standardized test meal (pastes and 250ml milk) and postprandial recording for one hour was done. After the recording session The EGG signals were subjected to spectral analysis (Fast Fo)

The mean of the power spectra for the entire recording period was calculated. The EGG signal, the highest power in the 3 cpm band, was then selected for further analysis. The mean frequency of the normal 3 cpm component, and its standard deviation and its power content was calculated for the fasting and postprandial period. The early postprandial frequency dip of the normal 3 cpm gastric component was identified. The frequency minimum and the subsequent frequency maximum of the dip were calculated by means of line-to-line analysis of the first 10 running spectra after the meal. The power ratio (the ratio of the power of the mean spectrum of the postprandial state to the power of the mean spectrum of the fasting state), indicative of the postprandial increase in gastric motor activity, was calculated for the first hour of the postprandial period.

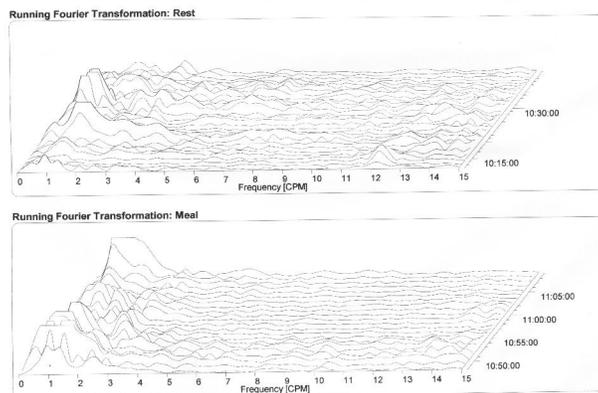
Dysrhythmia was defined as follows: A tachygastric was considered to be present when the power spectrum contained a sharp-peaked component with a frequency >3.7 cpm and <10.8 cpm, which was not of respiratory origin. For a definite diagnosis of tachygastric it was required that at the same time the normal gastric signal (2.6-3.7 cpm) was absent in all four EGG signals and that the abnormal rhythm was present for at least 2min. A so-called bradygastric was defined as presence of a sharp peak at a frequency less than 2.6 cpm, in the absence of a normal 3 cpm component in all four EGG leads(5). Postprandial dip was defined as transient frequency decrease after the food intake.



**Fig. 1):** Running Fourier Transformation at rest and meal in a patient showing normal 3 cpm gastric signal.



**Fig. 3):** Running fourier transformation at rest and meal of a patient showing tachygastrica with absence of normal 3 cpm gastric signal.



**Fig. 5):** Running Fourier Transformation at rest and meal of a patient showing bradygastrica with absence of normal 3 cpm gastric signal.

**5. *Helicobacter H. pylori* eradication therapy:**

An eradication triple regimen for *H. Pylori* (omeprazole® 20mg bid, amoxicillin 1g bid and

clarithromycin 500mg bid) was given for 10 days for every *H. Pylori* positive patient, followed by 20 days of omeprazole. Omeprazole alone (20mg bid) was given for *H. Pylori* negative duodenal ulcer patient.

**6. Endoscopy, electrogastrography**, and measurement of gastric emptying time using **ultrasonography**, were repeated one month after completion of therapy.

### 3. Results

The number of males in the studied groups was 35, and that of females was 25. The percentage of males in the study was averaged 75%, 70%, 50%, 80% in groups I, II, IIIa and IIIb, respectively. The mean age of the subjects was; 47.7±19.53, 49.65±14.79, 47±16.89, and 43.8±17.3 years in groups I, II, IIIa, and IIIb, respectively

Table 1; was demonstrating the different EGG parameters among group I patients and group IIIa. The mean of power ratio (PR) in group I patients was significantly lower ( $P<0.01$ ) than that of group IIIa (1.42±0.74 versus 2.94±1.9). While the rest of parameters showed insignificant difference.

Table 2; demonstrating high significant %CPM in group II patients than IIIb (86.84 ±17.97, and 68.8 ±34.8 respectively  $P<0.05$ ). No significant difference was found between them as regard other EGG parameters.

The comparison between group-I and group-IIIa regarding gastric emptying parameters, all the parameters showed insignificant difference. (Table 3)

The comparison between groups II and IIIb regarding gastric emptying parameters shows that the

full time and the ½ emptying time in-group II was statistically significant lower than group IIIb. The mean of full time was (13±2.8min) in-group II and (15.7±2.8min.) in-group IIIb ( $P<0.05$ ). The mean of 1/2 emptying time was 4.89±2.2min among group II and 6.7±2.48min among group IIIb ( $P<0.05$ ). The rest of parameters showed no significant difference (Table 4).

After eradication therapy (triple therapy for *H. pylori*), 87% of our patients became negative for *H. pylori*, according to eradication criteria of *H. pylori*. EGG repeated for group I patients before and after eradication therapy, it was found that: The mean of % power at rest in-group I before treatment was statistically significant lower than after treatment (16.9±4.48, and 19.24±5.7 respectively  $P<0.05$ ). The mean of %CPM at meal was significantly lower than after treatment, (74.01±25.2 and 87.65±22.5 respectively  $P<0.05$ ). The mean of PR before treatment was significantly lower than after treatment (1.42±0.73 and 2.06±0.09, respectively  $P<0.01$ ). The mean of power at rest of DF was statistically significant higher before treatment than after treatment (344 1.7±1970.5, 2222.5±1267.5 respectively  $P<0.01$ ). The rest of parameters showed insignificant difference (Table 5).

The difference of various gastric emptying parameters in group I patients before and after eradication therapy, there was increase in the mean of 1/2 emptying time after eradication therapy (5.3±2.6min. before versus 5.8±2.4min. after eradication therapy), but it was non-significant, the rest of parameters showed no significant difference.

**Table(1):** Comparison between Group I, and control (Group IIIa) regarding EGG parameters expressed in mean±SD:

EGG	Group I	Group IIIa	t value	P	Statistical significance
% Power rest	16.9±4.47	15.88±3.6	0.64	>0.05	Non-significant
% Power meal	18.3±7.3	21.67±6.9	1.3	>0.05	Non-significant
DF (cpm)	2.80.73	2.78 ±0.39	0.28	>0.05	Non-significant
%CPM rest	79.9±22.45	76.1±19.1	0.45	>0.05	Non-significant
%CPM meal	74.02±25.15	71.9±18.5	0.23	>0.05	Non-significant
Power rest DF	33441.6±1970.5	2309.8±1543	1.58	>0.05	Non-significant
Power meal DF	4573.8±2637.3	5498.4±2751.3	0.89	>0.05	Non-significant
PR	1.42±0.74	2.94±1.9	3.16	<0.01	Highly significant

CPM; cycle per minute. DF; dominant frequency. PR; power ratio

**Table (2):** Comparison between Group II and control (Group IIIb) regarding EGG parameters are expressed in mean±SD:

EGG	Group II	Group III b	t value	P	Statistical significance
% Power rest	19.07 ±4.48	16.78 ±2.4	1.5	>0.05	Non-significant
% Power meal	24.11 ±8.37	24.2 ±7.08	0.03	>0.05	Non-significant
DF (cpm)	3.33 ±0.9	2.9 ±0.35	1.26	>0.05	Non-significant
%CPM rest	85.18 ±19.27	69.5 ±31.36	1.69	>0.05	Non-significant
%CPM meal	86.84±17.97	68.8 ±34.8	1.88	<0.05	Significant
Power rest DF	2755.4±1764.35	2142.8 ±1763.4	1.04	>0.05	Non-significant
Power meal DF	5091.6±3528.98	4441.5±2124.3	0.53	>0.05	Non-significant
PR	2.14 ±1.5	2.1 ±0.7	0.08	>0.05	Non-significant

CPM; cycle per minute. DF; dominant frequency. PR; power ratio

**Table (3)** :Comparison between Group I and control (group IIIa) regarding gastric emptying parameters are expressed in mean  $\pm$  SD:

Group Emptying	Group I	Group III a	t value	P	Statistical significance
Full emptying time (min.)	13.1 $\pm$ 3.68	15.4 $\pm$ 3.34	1.66	>0.05	Non-significant
1/2 emptying time(min.)	5.24 $\pm$ 2.58	6.39 $\pm$ 2.43	1.68	>0.05	Non-significant
Full antrum (cm2)	6.35 $\pm$ 2.86	8.44 $\pm$ 3.8	1.68	>0.05	Non-significant
Fasting antrum (cm2)	3.86 $\pm$ 1.85	4.3 $\pm$ 1.58	0.67	>0.05	Non-significant

**Table(4):** Comparison between Group II and control (group IIIb) regarding gastric emptying parameters are expressed in mean  $\pm$  SD:

Group G.emptying	Group II	Group IIIb	T value	P	Statistical Significance
Full emptying time (min.)	13 $\pm$ 2.8	15.7 $\pm$ 2.8	2.48	<0.05	Significant
1/2 emptying time (min.)	4.89 $\pm$ 2.2	6.7 $\pm$ 2.48	2.06	<0.05	Significant
Full antrum (cm2)	7.05 $\pm$ 2.34	8.86 $\pm$ 2.44	1.96	>0.05	Non-significant
Fasting antrum (cm2)	4.14 $\pm$ 1.2	3.87 $\pm$ 0.74	0.65	>0.05	Non-significant

**Table (5)** :Comparison between Group I patients before and after eradication therapy as regards EGG parameters are expressed in mean  $\pm$  SD:

Group EGG	Before eradication therapy	After eradication therapy	t value	P	Statistical significance
% Power rest	16.9 $\pm$ 4.48	19.24 $\pm$ 5.72	2.6	<0.05	Significant
% Power meal	18.33 $\pm$ 7.3	22.4 $\pm$ 7.5	1.7	>0.05	Non-significant
DF (cpm)	2.85 $\pm$ 0.73	2.96 $\pm$ 0.32	5.54	>0.05	Non-significant
%%CPM rest	79.87 $\pm$ 22.45	78.08 $\pm$ 27.53	0.25	>0.05	Non-significant
%CPM meal	74.01 $\pm$ 25.15	87.65 $\pm$ 22.46	2.07	<0.05	Significant
Power rest DF	3441.65 $\pm$ 1970.5	2222.45 $\pm$ 1267.47	3.12	<0.01	Highly significant
Power meal DF	4573.85 $\pm$ 2637.33	4354.0 $\pm$ 2355.37	0.52	>0.05	Non-significant
PR	1.42 $\pm$ 0.73	2.0 $\pm$ 0.9	2.86	<0.01	Highly significant

CPM; cycle per minute.DF; dominant frequency. PR; power ratio

**Table (6):** Comparison between group I patients regarding gastric emptying parameters before and after eradications therapy are expressed in mean  $\pm$  SD:

Group Emptying	Before eradication therapy	After eradication therapy	t value	P	Statistical significant
Full emptying time (min.)	13.1 $\pm$ 3.68	13.1 $\pm$ 1.9	0.0	>0.05	Non-significant
1/2 emptying time (min.)	5.25 $\pm$ 2.58	5.83 $\pm$ 2.36	0.78	>0.05	Non-significant
Full antrum (cm2)	6.35 $\pm$ 2.86	8.35 $\pm$ 4.55	1.53	>0.05	Non-significant
Fasting antrum (cm2)	3.86 $\pm$ 1.85	6.36 $\pm$ 5.27	1.09	>0.05	Non-significant

#### 4.Discussion

Gastrointestinal motility has been attracting attention as an important contributory factor in the pathogenesis of duodenal ulcer. However, the results of previous studies, which assessed gastric motility in duodenal ulcers, are conflicting (11). It has been suggested previously that unusual rapid gastric emptying and resulting duodenal hyperacidity may contribute to the pathogenesis of duodenal ulcer formation (5). In contrast markedly deformed duodenal bulb with coexisting edema, fibrosis and scarring in chronic duodenal ulcer might be involved in mechanical obstruction in gastric emptying (12).

The percentage of normal slow waves is a quantitative assessment of the regularity of the gastric slow wave measured from the EGG. It is defined as the

percentage of time during which normal gastric slow waves are observed in the EGG (5). The percentage of normal slow waves can be computed from the running power spectra of the EGG (Fig. 6).In our method, one spectrum is derived from every 1 minute of EGG data; the minute is considered normal if its EGG spectrum exhibits a dominant power in the range of 2-4 cpm. In human, the normal percentage of gastric slow wave is defined as 70%.

In the present study there was no significant difference between the *H. pylori* positive patients with or without peptic ulcer regarding neither the stomach emptying time, nor the EGG records, except in the power rate, where it was significantly lower in the first group than the second. Power rate was indicative of the postprandial lower gastric motor activity in *H.*

*pylori*positive patients with peptic ulcer disease ( $1.42 \pm 0.74$  in group I and  $2.94 \pm 1.9$  in group IIIa,  $p < 0.01$ ). Power Ratio is a commonly used parameter that is associated with alteration in gastric contractions (13). These results coincide with those of **Chang et al.**, who studied 64 *H. pylori*infected active duodenal ulcer (DU) patients, and liquid gastric emptying was measured. They found that about 35% manifested either enhanced or delayed gastric emptying (7).

Many studies have focused on the effect of duodenal ulcer (especially with *H. pylori*infection) on gastric emptying. In our study, comparing the *H. pylori*negative duodenal ulcer patients with asymptomatic control group, full gastric emptying time and  $\frac{1}{2}$  gastric emptying time were significant shorter in the patients with peptic ulcer than asymptomatic patients. This was coordinated with higher % cpm in group II than group IIIb ( $p < 0.05$ ). These results were the same reported by **Williams et al.**, they concluded that there was enhanced gastric emptying in duodenal ulcer patients (14), whereas similar gastric emptying in comparison with controls was speculated by other studies. Perhaps in duodenal ulcer the elicited distortion of antropyloroduodenal region including ulcer crater, edema and fibrosis is unable to offer a mechanical resistance to gastric emptying (7,13).

Taking together, *H. pylori* infection may affect the gastric motility in infected patients either complicated with peptic ulcer, or infected asymptomatic patients. These results was in accordance with **Konturek et al.**, who concluded that gastric myoelectrical activity of the duodenal ulcer infected with *H. pylori*was impaired (15). The possible effect of *H. pylori*infection on gastric motor function may be mediated by gastric acid hypersecretion and release of cytokines. The role of mucosal cytokines has been suspected because IL-1 $\beta$  was shown to interact with gastric vagal nerve activity in anaesthetized rats (16).

On studying the different electrogastrographic (EGG) parameters in duodenal ulcer patients with *H. pylori*infection (Group I) before and after eradication therapy, there was highly significant increase in the mean of power ratio. EGG dominant power reflects improving gastric contractility after eradication therapy ( $P < 0.05$ ). Also there was increase in % 3cpm at meal after eradication therapy. Also, there was slightly increase in both the dominant frequency (DF), and the distribution of postprandial dip after treatment, while normogastria (%3cpm at rest) was slightly decreased after eradication therapy, but all of them did not reach the statistically significant level.

The results of the present study partially agree with those of **Budzynski et al.**, who studied 15 male patients with active duodenal ulcer with *H. Pylori* infection (17) The electrogastrographic examination in duodenal

ulcer patients was performed before and after one-week *H. pylori*eradication therapy. They found that during fasting the percentage of normogastria, and the postprandial amplitude of EGG in patients with active duodenal ulcer was slightly increased after eradication *H. pylori*. This increase in the postprandial power was significantly small. This difference in EGG amplitude between patients with active ulcer infected with *H. Pylori*and those with eradicated *H. Pylori* or healthy controls suggests disturbed myoelectrical activity by the presence of an ulcer. Also, they observed the ratio of frequency of normogastria increased after eradication, and this was coinciding with those of the present study. They concluded that most changes in gastric myoelectrical activity and its regulation observed in duodenal ulcer patients with active ulcer and *H. Pylori* infection abolished by a successful eradication of *H. Pylori*. After eradication, the EGG recording including both frequency and amplitude of EGG became similar to those in healthy controls (17).

**Konturek et al.**, (15) studied the gastric motility in 10 duodenal ulcer patients before and 8 weeks after eradication of *H. pylori* (with one week triple therapy) demonstrated in their study that *H. Pylori* positive DU patients is accompanied by accelerated gastric emptying and that eradication of *H. Pylori* decreases this emptying rate to the value observed in healthy subjects. Since acid and fat are potent releasers of cholecystinin (CCK) it is possible that this motor defect observed in DU disease results either from reduced release or from impaired action of endogenous CCK. This hormone has been reported before to induce a dose dependent inhibition of gastric emptying in human (4). The major finding of that study was that in *H. Pylori*-positive DU patient's luminal somatostatin was significantly decreased compared to healthy subjects and that eradication of *H. pylori*resulted in a significant increase of luminal somatostatin release. Somatostatin is known to originate from D cells located in the antral and oxyntic mucosa. It is well known phenomenon that *H. Pylori* infection leads to decrease of D cell density and activity in the antral mucosa, therefore it is tempting to speculate that impaired inhibition of gastric emptying in *H. Pylori*-infected DU patients by endogenous CCK might occur due to failure of this CCK to stimulate somatostatin release from D cells (18).

In the present study there was normalization of gastric dysrhythmias after eradication therapy. Yet, there is no previous study discussing this point. It is known that duodenal ulcer patients may have disturbed gastro duodenal motility and, in particular, a more rapid gastric emptying of liquids and an increased acid output than normal subjects. Both these aspects and the impaired interaction between gastric motility and

gastric secretion may have an ulcerogenic role, increasing the exposure of duodenal mucosa to acid. Increased gastric emptying rate has been proposed as one of the factors responsible for duodenal ulcer formation (19). These data suggest that eradication therapy for *H. pylori* infection could modify gastric motility.

The present study speculated that eradication therapy in duodenal ulcer patients with positive *H. Pylori* infection (Group I) was associated with normalized gastric emptying. Although the full emptying time was not changed after eradication therapy, the mean of the 1/2 emptying time was mildly increased after eradication, which means slower emptying after eradication (but still did not reach the significant value  $P>0.05$ ).

### Conclusion

*Helicobacter pylori* infection can affect gastric motility, especially in duodenal ulcer infected patients, presented by disturbed gastric myoelectrical activity; and accelerated liquid gastric emptying. Eradication therapy improved not only EGG abnormalities but also aids in restoration of abnormal water gastric emptying. Multicenter studies are needed to document our results.

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3/15/2013