An Electrogastrographic Study of Gastric Myoelectrical Activity in Acute Pancreatitis

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ABSTRACT
Objectives: Oral feeding is traditionally prohibited in acute pancreatitis patients. This is partly due to an assumption that acute pancreatitis is associated with some degree of gastrointestinal atony, and that oral feeding would thus not be tolerated. Gastric motility has never been studied in acute pancreatitis. The aim of this study was to determine whether gastric myoelectrical activity as measured by electrogastrography (EGG) was altered in a group of acute pancreatitis patients compared to a group of healthy controls. **Methods:** Nine acute pancreatitis patients and thirteen healthy volunteers were assessed. Three electrodes were used to record EGG for 30 minutes before the subject drank 500 ml of water, and then for 60 minutes afterwards. EGG parameters measured included dominant frequency, power ratio and percent normogastria. **Results:** In the control group, the dominant frequency and percent normogastria were found to decrease significantly after water ingestion, as expected. The mean power ratio in the control group was greater than 1, showing an increase in power after ingestion of water. The acute pancreatitis patient group (n = 9) studied on the day following admission showed no significant difference in any of the EGG parameters measured when compared to the control group. **Conclusion:** The results of this study show that there is no abnormality in gastric myoelectrical activity in acute pancreatitis. This indicates, due to the close relationship between myoelectrical activity and motility, that gastric motility is normal in acute pancreatitis.

INTRODUCTION

Acute pancreatitis is an acute inflammatory process of the pancreas, a mixed gland that secretes both digestive enzymes and endocrine hormones such as insulin and glucagon. The effects of acute pancreatitis on gastric motility have never been studied. It has been shown that gastric motility is regulated by gastric myoelectrical activity, which can be measured cutaneously by EGG.[1]

Gastric myoelectrical activity consists of slow waves of depolarisation originating from a pacemaker area along the greater curvature of the stomach and propagating with increasing velocity towards the pylorus at a rate of approximately 3 cycles per minute (cpm).[2] These slow waves are always present and determine the propagation and maximum frequency of contractions of the gastric smooth muscle. Electrogastrography measures gastric myoelectrical activity by cutaneous electrodes placed on the abdominal skin over the stomach, recording a sinusoidal wave reflecting the overall electrical activity of the stomach.[3] Gastric myoelectrical activity, as measured by EGG, has been shown to modulate gastric motility.[4] Abnormalities in myoelectrical activity are associated with motility disturbances, such as those seen in gastroparesis, motion sickness, post-gastric surgery and other clinical disorders.[5]

The parameters most commonly measured by an EGG are dominant frequency, percent normogastria and power ratio. The dominant frequency of this EGG wave has been shown to be the same as that of the gastric slow wave measured from both serosal and mucosal internal electrodes.[6] Frequencies between 2 and 4 cpm are considered normal. Frequencies above and below this range are considered tachygastria and bradygastria respectively.[5] The dominant frequency and percent normogastria are indicators of normal gastric function and the rhythmic stability of the motor pattern.[4] It is generally agreed that the absolute value of EGG amplitude is not useful since it could be related to the variable distance between the stomach and the recording electrodes.[7] Therefore, a ratio of the pre: post- stimulus power is used to cancel out these variables. The stimulus used in clinical practice is usually a standardised meal. The dominant power ratio may be reliably used as a measure of spike activity and contractile strength of the stomach wall.[6] In clinical practice, there are three criteria used to determine whether an electrogastrogram is normal. The dominant frequency must be within the range of 2-4 cpm, the percent normogastria post-stimulus must be greater than 70% and the power ratio must be greater than 1.[5]

The non-invasive nature of the EGG procedure means it can feasibly be used in this critically ill group of acute pancreatitis patients. The stimulus in this case had to be water, because oral feeding of acute pancreatitis patients is traditionally prohibited. This prohibition is based on two assumptions. The first is that feeding the gut will stimulate pancreatic exocrine secretion, thus aggravating the clinical condition. There have been studies contradicting this view, showing that while oral feeding may indeed induce exocrine

secretions, it does not exacerbate the disease.[8,9] Secondly, there exists a belief that acute pancreatitis is associated with gastric and intestinal atony, and that oral feeding would thus not be tolerated.[10] In this study, it is proposed to investigate the assumption that there is a gastric motility abnormality associated with acute pancreatitis. Electrogastrography will be used in order to determine whether gastric myoelectrical activity is altered in a group of acute pancreatitis patients compared to a group of healthy controls.

METHODS

Nine acute pancreatitis patients (3 men, 6 women) were assessed on the day following admission to St. James's Hospital, Dublin. The average age was 51.44 ± 6.38, ranging from 23 to 90. Diagnosis of acute pancreatitis in all patients was based on a suggestive clinical picture and consistent morphological findings on computed tomography (CT) scan. Clinical and biochemical data from these patients are seen in table 1. Only intravenous electrolyte solutions, antibiotics and analgesics were administered before the study. No patients developed any major complications of acute pancreatitis and there were no mortalities. Thirteen healthy volunteers (6 men, 7 women) with no gastrointestinal symptoms were assessed on one occasion only. The average age in this control group was 41.46 ± 4.88, ranging from 22 to 85. The St. James's Hospital Ethics Committee approved the study protocol and written consent was obtained from each subject prior to the study.

EGG Measurement

Gastric myoelectrical activity was measured non-invasively by EGG. Each subject was fasting for at least six hours (usually overnight) prior to the investigation. The skin over the abdomen at the recording sites was shaved, if necessary, to remove excess hair and carefully abraded with sandy skin-prep jelly to reduce the impedance between electrodes. Three silver/silver-chloride electrodes were placed on the skin over the stomach after application of electrode gel, as can be seen in figure 1. The first recording electrode was placed halfway between the xiphoid process and the umbilicus in the midline. The second recording electrode was placed 45^{o} to the left and 5 cm above the first. The reference electrode was placed on the left flank underneath the ribcage. After placement, the resistance between the electrodes was checked using an ohmmeter and found to be below 5kΩ in each subject. The electrodes were connected to a portable EGG recording device (Digitrapper EGG, Synectics Medical Inc, Irving, Texas). Subjects were lying in a semi-recumbent position and were asked not to move during the recording. Data was recorded for 30 minutes before the subject drank approximately 500ml of water, and then for 60 minutes afterwards. The EGG data stored on the recorder were downloaded to an IBM personal computer and underwent spectral analysis at 2Hz. A Fast Fourier Transform (FFT) was applied to consecutive 256 second signal stretches that have an overlap of approximately 75%. This resulted in a series of minute-by-minute frequency spectra. The dominant frequency of the EGG was measured in contractions per minute and the dominant power, defined as the amplitude of the EGG at the dominant frequency, was measured in dB. The power ratio, which is defined as the ratio of the amplitude of the dominant frequency pre-water and post-water, was also assessed. Other parameters recorded were percent bradygastria (percent time that the dominant frequency lay in the range 0-2cpm), percent normogastria (percent time that the dominant frequency lay in the range 2-4cpm) and percent tachygastria (percent time that the dominant frequency lay in the range 4-10cpm).

Blood samples were taken from each acute pancreatitis patient on the day of admission. Serum amylase and Glasgow score were recorded as shown in table 1. The sex, age and cause of acute pancreatitis were also recorded for each patient. All data were expressed as mean ±

Table 1. Clinical characteristics and biochemical data of patients with acute pancreatitis.

Case No.	Age	Sex	Aetiology	Amylase (IU/L)	Glasgow Score
P1	23	F	Gallstone	316	0
P2	52	F	Gallstone	652	2
P3	90	F	Gallstone	1076	3
P4	37	F	Alcohol	382	1
P5	59	M	Alcohol	779	3
P6	37	F	Alcohol	35	2
P7	59	F	Gallstone	530	4
P8	60	M	Alcohol	89	5
P9	46	M	Alcohol	1447	1

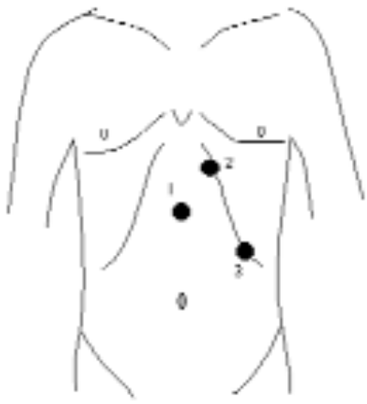

Figure 1. Placement of EGG electrodes is as shown. The electrodes are then connected to a portable EGG device and data recorded for 30 minutes before and 60 minutes after ingestion of approximately 500 ml of water.

standard error and $p<0.05$ was considered statistically significant. Because the data were not normally distributed due to the small sample size, non-parametric statistical tests were used to determine significance. Wilcoxon's signed rank sum test was used to compare EGG parameters within groups and Wilcoxon's unpaired test was used to compare between groups.

RESULTS

In the control group of 13 subjects, the dominant frequency was found to decrease after water ingestion from 2.97 ± 0.087 cpm to 2.58 ± 0.12 cpm ($p = 0.05$, $n = 13$), as seen in figure 2. The frequency of the slow waves remained within the normal 2 to 4 cpm range for over 80% of the recording time, and this percentage decreased significantly after water (88.56 ± 3.074 versus 82.31 ± 2.393 cpm, $p = 0.0403$, $n = 13$). There was a significant increase in the percent bradygastria after water (9.35 ± 2.684 versus 12.94 ± 2.447 cpm, $p = 0.0421$, $n = 13$). The mean power ratio in the control group (figure 3) was greater than 1,

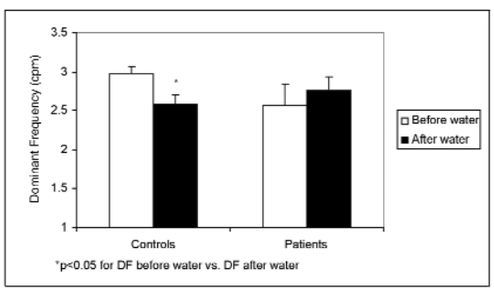

Figure 2. Changes in dominant frequency (cpm) with ingestion of water in control and patient group. There is a significant decrease in the mean dominant frequency after water in the control group, (n=13) but no significant change in the patient group (n=9). Data are expressed as means ± SE.

showing an increase in power after ingestion of water (mean power ratio=4.18±1.482).

The acute pancreatitis patient group (n = 9) studied on the day following admission showed no significant difference in any of the EGG parameters measured when compared to the control group. The dominant frequency before water was 2.58 ± 0.263 cpm in the acute pancreatitis group and 2.97 ± 0.087 cpm in the control group ($p = 0.13$). Post-water dominant frequency also failed to reach significance between these groups (2.76 ± 0.178 cpm for patients versus 2.58 ± 0.12 cpm for controls, $p = 0.17$). The mean dominant frequency in the patient group showed an increase after water (as opposed to the decrease seen in the control group), but this increase was not significant (2.58 ± 0.263 versus 2.76 ± 0.178 cpm, $n = 9$, $p = 0.29$).

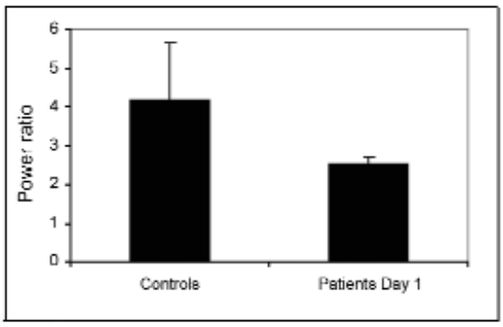

Figure 3. Power ratios (PR) in each group. The power ratio is the ratio of the amplitude of the dominant frequency pre: post water. Both groups showed an increase in power after water ingestion. There was no significant difference in PR between controls and patients.

No significant differences were observed when comparing the pre- and post- water percent bradygastria, normogastria and tachygastria between the patients on day of admission and the control group. The mean percentage of slow waves with a frequency between 2 to 4 cpm (normogastria) was 81.31 ± 4.668% before water and 85.64 ± 5.059% after water. The mean power ratio in the patients group was 2.53 ± 0.584, which shows that the amplitude of the EGG signal increased after water, as it did in the control group. There was no significant difference between power ratios in the Patients Day 1 group when compared to the control group (figure 3).

DISCUSSION

The aim of this study was to determine whether or not gastric myoelectrical activity was altered in acute pancreatitis patients compared to a group of normal controls. Electrogastrography was used to measure gastric myoelectrical activity, due to its non-invasive nature and proven accuracy in recording the gastric slow wave.[6]

Controls

EGG parameters obtained from the control group remained within the normal ranges used in clinical practice.[5] As expected, ingestion of a water stimulus caused a significant change in the dominant frequency as measured by electrogastrography (see figure 2). The decrease in dominant frequency seen in this group of normal healthy subjects is in agreement with results obtained in other studies. A study by Chen & McCallum investigated the effects of water ingestion on the postprandial electrogastrogram.[11] During the first ten minutes after drinking water, the dominant frequency was lower than or equal to the preprandial value in nine out of ten subjects. In a separate study of ten subjects, a decrease in the dominant frequency was found in all ten subjects after water ingestion.[6] In the present study, water also induced a significant reduction in the percent time that the frequency remained within the normal 2 to 4 cpm range (normogastria). Since the two groups mentioned previously did not measure percent normogastria, this reduction was not previously documented. The mean power ratio in the control group was greater than 1, as expected (figure 3), and therefore indicated increased contractility of the stomach following ingestion of water.

Acute Pancreatitis Patients

No significant differences were observed in any of the EGG parameters measured for the patient group assessed on day after admission when compared to the control group. In fact, water did not induce any significant changes in EGG parameters in the patient group assessed on the day of admission. The reason for this may be the smaller sample size in the Patient Day 1 group (n = 9) compared to the control group (n = 13). However, all parameters measured in acute pancreatitis patients were within the normal ranges set out by Lawlor *et al*.[5] In this study, no abnormality in gastric myoelectrical activity assessed by EGG was observed in acute pancreatitis patients.

Oral Feeding in Acute Pancreatitis

Oral feeding of acute pancreatitis patients is traditionally prohibited. This prohibition is based on two assumptions. The first is that feeding the gut will result in stimulation of enteral hormone secretion and direct activation of pancreatic enzymes, thus aggravating the severity of the inflammation and worsening the pain. A study on orally fed acute pancreatitic rats questions this convention.[8] Acute pancreatitis was induced in all rats by ligation of the main biliopancreatic duct, and rats were then fed either orally or parenterally. While rats fed orally did indeed have higher levels of enteral enzymes such as amylase, histopathological examination of the pancreas revealed more severe inflammatory changes in those rats fed parenterally. The authors suggest that some enteral hormones, such as motilin and cholecystokinin, have a protective effect, thus limiting the inflammation process in the pancreas. Additionally, intestinal stasis due to lack of oral feeding may promote bacterial translocation and further aggravate local and systemic complications of acute pancreatitis.[8]

Further evidence to suggest that oral feeding does not exacerbate acute pancreatitis comes from a clinical trial involving 34 acute pancreatitis patients who were randomised into enteral and parenteral groups.[9] Patients with mild to moderate disease were fed orally on a clear fluid diet with nutrition supplements and those in the parenteral group via a peripheral intravenous line. Patients with severe disease were fed enterally via a nasojejunal feeding tube or parenterally via a central venous catheter. Clinical outcome parameters such as systemic inflammatory response syndrome, sepsis, multiple organ failure and hospital stay were all improved in the enterally fed patients. C-reactive protein and Modified Glasgow score were also significantly reduced in patients receiving oral or nasojejunal feeding. [9]

Gastric Motility in Acute Pancreatitis

The assumption that some degree of gastrointestinal atony exists in acute pancreatitis patients is the second reason for the avoidance of oral feeding in this patient group. In this study, it has been found that gastric myoelectrical activity of acute pancreatitis patients is normal on the day after admission to hospital. No other studies investigating gastric motility in acute pancreatitis were found in the literature. There have been several studies that show that gastric motility is altered in chronic pancreatitis. Accelerated gastric emptying of a low-energy liquid meal and a shorter duration of the interdigestive motor cycle were observed in patients with severe chronic pancreatic insufficiency. Pancreatic enzyme supplementation was associated with a longer fed pattern and decelerated gastric emptying.[12] The fact that enzyme supplementation, and therefore decreased malabsorption, reversed the changes observed in pancreatic insufficiency suggests that the motility changes are caused by malabsorption and increased nutrient delivery to the small intestine. Another study reiterated these findings and further showed that the motility changes observed in chronic pancreatitis were correlated with the degree of exocrine pancreatic insufficiency.[13] In chronic pancreatitis patients, the fed motor pattern was

longer, antral motility indexes were reduced and the interdigestive cycle length was shorter than in controls. These abnormalities were much more pronounced in chronic pancreatitis patients with exocrine pancreatic insufficiency compared to chronic pancreatitis patients without exocrine pancreatic insufficiency, and pancreatic enzyme supplementation reversed the changes towards normal.[13] Therefore, gastric motility is altered in chronic pancreatitis due to exocrine pancreatic insufficiency.

In acute pancreatitis, levels of exocrine pancreatic enzymes measured by a duodenal intubation perfusion technique remain within the normal range. Secretion of pancreatic amylase, trypsin and chymotrypsin continued to be cyclical as normal in acute pancreatitis patients with a serum amylase threefold the upper limit of normal and clinical evidence of the disease. No statistical differences were found in pancreatic enzyme output per hour within a cycle or per secretory peak, as compared to a group of healthy controls.[14] Even though exocrine pancreatic enzyme levels are normal, there is a possibility that other pancreatic hormones and peptides could be involved in the modulation of gastric motility in acute pancreatitis. Amylin, for example, is a peptide synthesised in the endocrine beta cells of the pancreas and has been shown to inhibit gastric emptying in rats.[15] Pancreatic polypeptide stimulates gastric motility under normal conditions by acting indirectly via a vagal cholinergic mechanism.[1] It is interesting that hyperglycaemia has also been shown to inhibit gastric motility and this effect is mediated by impaired vagal activity.[16] Although pancreatic polypeptide concentrations have been found to be normal in acute pancreatitis, the concentrations of amylin have yet to be studied.[14]

The results of this study show that there is no abnormality in gastric myoelectrical activity in acute pancreatitis. This indicates, due to the close relationship between myoelectrical activity and motility, that gastric motility is normal in acute pancreatitis. Further studies are needed in order to fully investigate gastric motility in acute pancreatitis. These would involve the use of more invasive motility tests, such as gastric emptying of an isotope-labelled meal and antroduodenal manometry, which directly measure motility rather than myoelectrical activity. If motility were established to be normal using these techniques, then a review of the nutritional management of acute pancreatitis patients would surely be necessary and the prohibition on oral feeding could feasibly be lifted.

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