Experimental gastric dysrhythmias and its correlation with \textit{in vivo} gastric muscle contractions

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\textbf{INTRODUCTION}

Gastric motility is under the control of gastric myoelectric activity (GMA). Normally GMA originates from the junctional area of the fundus and proximal stomach, propagates aborally, controlling the frequency and direction of gastric contractions. Disturbances in GMA, so-called gastric dysrhythmias, have been frequently observed in patients with various diseases including gastroparesis\cite{1}, dyspepsia\cite{2}, anorexia nervosa\cite{3}, gastroesophageal reflux diseases (GERD)\cite{4}, motion sickness\cite{5} and pregnancy\cite{6}, etc. Gastric dysrhythmias have also been linked to gastrointestinal symptoms such as nausea and vomiting\cite{7}, and improvement of gastric dysrhythmias seems to be associated with relief of such symptoms\cite{8,9}.

Gastric dysrhythmias are classified into tachygastria (frequency higher than normal), bradygastria (frequency lower than normal) and arrhythmia (no rhythmic activity), based on the dominant frequency of GMA. Although it is believed that gastric motility could be affected inevitably, the relationship between gastric dysrhythmias and gastric motility has not well been established. Various techniques including electrogastrography (EGG), gastric emptying and simultaneous recording of EGG and intraluminal pressure have been applied, and available evidence suggests that tachygastria may be associated with hypogastric motility\cite{10,11}, while the effect of bradygastria on gastric motility remains controversial\cite{12,13}. The association between specific gastric dysrhythmia and gastric muscle contractions has never been carefully investigated.

To address these issues, in this study we specifically evaluated: (1) the effect of intravenous vasopressin on GMA; (2) the correlation between gastric muscle tone and GMA; (3) the effect of experimentally-induced gastric dysrhythmias on gastric muscle contractions, in a canine model.

\textbf{MATERIALS AND METHODS}

\textbf{Animal preparations}

Seven healthy female hound dogs (15-22 kg) were anesthetized with intravenous infusion of thiopental sodium (20 mg/kg) and maintained with inhalation of isoflurane (1%-2%). A midline laparotomy was performed, and four pairs of temporary cardiac pacing wires (A&E Medical, Farmingdale, NJ) were implanted under the serosal surface along the greater curvature of the stomach. The most distal pair was 2 cm above the pylorus, and the distance between adjacent pairs of electrodes was...
slow waves is a quantitative assessment of the regularity of GMA:

muscle tone

Analysis of gastric myoelectrical activity and gastric muscle tone

Regularity of GMA: The percentage of 4- to 6-cpm slow waves is a quantitative assessment of the regularity of the GMA measured from the EGG. It was defined as the percentage of time during which normal 4- to 6-cpm gastric slow waves were observed in a specific EGG recording. The percentage of normal 4- to 6-cpm slow waves was computed from the running power spectra of the GMA using an adaptive spectral analysis method\(^1\). One power spectrum was generated for every 2 min of EGG data, and the spectral peaks in each spectrum were examined visually. A spectrum was defined as normal if it had a clear peak in the 4- to 6-cpm range. The percentage of regular 4- to 6-cpm slow waves was determined by computing the ratio between the numbers of normal and total spectra.

Gastric dysrhythmias: The percentage of time of gastric dysrhythmias was also computed using running spectral analysis. Bradygastria was defined as a frequency of 0.5-4.0 cpm. Tachygastria was defined as a frequency of 6.0-15.0 cpm. Arrhythmia was defined as any irregular rhythm.

Gastric muscle tone: Reflected by the energy of the strain gauge signals, which were derived by calculating the area under the curve.

Statistical analysis

GMA regularity within each session was compared with One-Way ANOVA. The correlation between gastric muscle tone and gastric dysrhythmias was analyzed with Pearson’s correlation test. All data were presented as mean ± SE. \( P < 0.05 \) was taken as significance.

RESULTS

Gastric myoelectrical activity and gastric muscle tone at baseline

Intrinsic, distally propagating gastric slow waves were observed in all animals at a mean frequency of 5.6 ± 0.3 cpm, with a range of 4.8-6.2 cpm. Rhythmic variations in gastric muscle tone were consistently present, and coupled with each gastric slow wave at an identical frequency (Figure 2).
Effect of vasopressin on GMA

The regularity of GMA was correlated with the decrease of GMA regularity (r = 0.96, P < 0.05, vs baseline, P < 0.05) and tachygastria (r = -0.95, P > 0.05).

Effect of vasopressin on strain gauge energy

The strain gauge energy was significantly decreased during the 20-min infusion of vasopressin. The total energy was 48.4 ± 1.3 dB at baseline, and decreased to 36.6 ± 4.5 dB, 44.5 ± 4.3 dB, 45.8 ± 1.9 dB during the following consecutive three 20-min periods during and after the infusion (t/t baseline, P < 0.05).

Correlation of GMA and strain gauge energy

The decrease in strain gauge energy was positively correlated with the decrease of GMA regularity (r = 0.96, P < 0.05). Gastric muscle contractions were reduced or disappeared during periods of bradygastria or tachygastria (Figure 5). Strain gauge energy was negatively correlated with bradygastria (r = -0.96, P < 0.05) and tachygastria (r = -0.95, P > 0.05).

DISCUSSION

Through this study we have found: (1) Intravenous vasopressin at proper doses consistently produced gastric dysrhythmias; (2) strain gauge implanted on gastric wall could reliably detect gastric muscle contractions; gastric muscle contractions were coupled with intrinsic gastric slow waves, and each gastric slow wave was capable of inducing a change in gastric muscle tone; (3) gastric muscle contractions was positively correlated with the regularity of the gastric slow waves; and (4) the reduction or disappearance in gastric muscle contractions was associated with both bradygastria and tachygastria.

Considering the fact that all muscle contractions are coupled and superimposed with myoelectric activities, it is strongly believed that gastric dysrhythmias could cause disturbances in gastric motor functions. A larger number of clinical studies have been performed recently, and indeed, gastric dysrhythmias have been observed in various motility disorders like gastroparesis, functional dyspepsia and motion sickness, et al. In patients with gastroparesis, gastric emptying scintigraphy and EGG were performed concurrently, and it was found that postprandial gastric dysrhythmias correlated with delays in solid phase gastric emptying. Gastric dysrhythmias may also be in association with the occurrence of GI symptoms. We have noted on many occasions that degeneration of GMA preceded the occurrence of nausea and vomiting in our previous canine studies. Other investigators also reported similar findings, and all these indicate a possible causative role for the dysrhythmia in the production of nausea and vomiting.
old male infant suffering from severe gastric retention and symptoms of intractable nausea, vomiting and weight loss. The symptoms were attributed to impaired motor function of the stomach. The patient underwent resection of the distal ¼ of the stomach. The excised tissues were studied in vitro by means of intracellular electrodes, and abnormally fast slow waves (> 5 cpm) were detected . The association between gastric dysrhythmia and gastric motor disorders was further substantiated by other studies. You et al observed tachygastria in a 26-year-old woman with persistent nausea, vomiting and abdominal pain who was found to have severe impairment of antral motor functions. In contrast to tachygastria, data on bradygastria are inconsistent. Van Der Schee et al observed bradygastria in dogs and found that it was correlated with strong antral contractions , while Abell et al induced bradygastria in humans with glucagon, and found that it was associated with absence of antral contraction.

In our present study, we attached a stain gauge to the site of recording electrodes, which allows us to study the direct correlation of gastric contractions and GMA. In comparison to the traditional intraluminal manometry, strain gauge is more sensitive and more direct in the detection of subtle contractions of gastric wall than any other methods. In addition, gastric dysrhythmias in this study were assessed quantitatively rather than qualitatively in the previous studies. Our results indicate that both tachygastria and bradygastria are negatively associated with gastric muscle contractile activity, which is consistent with most of the previous reports. The exact mechanisms causing gastric dysrhythmias have not been fully understood, though it is well known that GMA is generated by interstitial cells of Cajal, and is influenced by central and autonomic nerve systems, certain hormones and peptides. Tachygastria and bradygastria are different. Tachygastria usually originates from distal stomach, and can be considered as an ectopic rhythm, while transient or persistent bradygastria usually originates from the region of the normal pacemaker at a reduced frequency. Gastric dysrhythmia can be induced experimentally with various agents including vasopressin . Vasopressin is a peptide released into the peripheral circulation from the pituitary during intravenous administration of vasopressin could impair gastric slow waves and gastric muscle contractions. Experimentally induced tachygastria and bradygastria are negatively correlated with the gastric muscle contractions, suggesting that gastric dysrhythmias, either tachygastria or bradygastria, may be indicative of gastric hypomotility.

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