Electrical stimulation of gastric electrical control activity

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SARNA, S. K., AND E. E. DANIEL. Electrical stimulation of gastric electrical control activity. Am. J. Physiol. 225(1): 125-131. 1973.—Gastric electrical control activity in anesthetized dogs was stimulated by voltage pulses of 100 msec duration and 1- to 25-v amplitude. The gastric control waves could be entrained from any site in the electrically active region on the anterior and posterior sides. The maximum frequency at which the gastric control waves could be entrained by the electronic pacemaker varied from dog to dog. Mean maximum driven frequency (MDF) was 6.98 cycles/min. The strength of stimulus required to entrain gastric control waves increased for larger differences between the driven frequency and the natural frequency of gastric control waves. Total phase lag between control waves in the corpus and in the antrum increased when the control waves were driven at a frequency higher than their natural frequency. The stomach was divided into three segments by circumferential cuts in muscle layers. The proximal segment that contained the highest intrinsic frequency oscillator had the least MDF (mean 6.92 cycles/min). The mean MDF increased distally (middle segment mean MDF, 7.55 cycles/min; distal segment mean MDF, 7.74 cycles/min). Intravenous injection of physostigmine (20-60 µg/kg) decreased the MDF of the distal segment, whereas intravenous injection of atropine (5-20 µg/kg) increased its MDF. These drugs, in particular physostigmine, had a small effect on the MDF of the middle segment and very little or no effect on the MDF of the proximal segment. This study confirms that a system of bidirectionally coupled relaxation oscillators can account for more characteristics of the gastrointestinal electrical control activity than a relaxation oscillator model with only forward coupling or a cable model. The study also shows that nerves could have an important role to play in determining some of the characteristics of gastric relaxation oscillators, such as their ability to be driven by electronic pacemakers.

THE ELECTRICAL CONTROL ACTIVITY (ECA) of the stomach and the small intestine has been shown to result from a system of bidirectionally coupled relaxation oscillators (16, 17). In such a system of coupled relaxation oscillators, the oscillator with the highest intrinsic frequency pulls the frequency of other lower intrinsic frequency oscillators toward or even higher than its own (17). Furthermore, the control wave of the highest intrinsic frequency oscillator has phase lead over the control waves of all other oscillators. In the stomach, the highest intrinsic frequency oscillator is located in the corpus near the greater curvature (7, 16, 20) and the direction of phase lag is aboral.

During gastric peristalsis a ring of contraction originates in the corpus of the stomach and moves aborally. The response activity (spikes) associated with a contraction always occurs just after a control potential (3-5). Our hypothesis is that this aboral movement of the ring of contraction propels gastric contents, and this propulsion is controlled in space and time by the electrical control activity.

This study was undertaken with the following objectives in mind: 1) to further establish that the gastric electrical control activity is due to an array of bidirectionally coupled relaxation oscillators and to determine the properties of this system by driving the gastric control waves from different sites and at different frequencies. 2) To determine if gastric relaxation oscillators in different regions of the stomach have different abilities to follow the electronic pacemaker and if so, are these differences related to differences in intrinsic frequencies and coupling factors among different regions of the stomach (19)? 3) To determine if drugs that influence the nervous mechanism can affect the ability of gastric relaxation oscillators to follow an electronic pacemaker.

METHODS

Healthy female dogs weighing 10-17 kg were used in all experiments. Dogs were fasted for 24 hr before experiments. Chloralose (2%) and urethan (10%) (3 ml/kg) solution was used as the anesthetizing agent. Subsequent doses of sodium pentobarbital (60 mg) were given intravenously when required.

Access to the abdominal cavity was obtained by a midline incision from the xiphoid process to about 4-5 cm below the umbilicus. An additional cut was made below the rib cage on the left side. Abdominal temperature was monitored with a thermometer and maintained between 38.5 and 40 C by heat radiation and control of room temperature.

Monopolar silver wire electrodes (0.15 mm diam) were implanted in the seromuscular layer of gastric wall to record electrical activity. The ground electrode was placed subcutaneously in the left thigh. Bipolar silver wire electrodes (0.15 mm diam, 1 cm long and 0.5 cm apart) were implanted in the seromuscular layer for electrical stimulation. The stimulating electrodes were implanted on both the anterior and the posterior sides of the first three dogs, but in subsequent experiments these were implanted on the anterior side and near the greater curvature (Fig. 1).
The voltage applied across the stimulating electrodes was measured by an oscilloscope. The rise time (10–90%) of stimulus across the stimulating electrodes was of the order of 30 msec for voltages up to 10 v and 70 msec for higher voltages. No appreciable change in rise time occurred following varying degrees of stimulation. All recordings were made on a 6-channel Beckman Dynograph model R with a curvilinear ink writer. Lower and upper cutoff frequencies were set at 0.35 and 22 Hz, respectively.

Rectangular pulses of duration 100 msec and amplitude 1.0–25.0 v were used for stimulation. With smaller pulse durations, the amplitude of stimulus required to drive gastric ECA increased somewhat, whereas larger pulse durations increased the durations of stimulus artifact and thus obscured the recordings. The 100-msec pulse duration was judged to be optimal. Details of strength duration curves will be reported in a later communication. Although the output of the stimulator was floating, leakage of stimulus current in the extracellular fluid caused appreciable artifact in the recordings. To avoid excessive deflections of the pens due to this artifact, all recordings were made in the limiting position of the recorder. Doses of atropine and physostigmine were in terms of sulfate salts.

The significance of the difference between the means was investigated by analysis of variance (21).

RESULTS

Electrical stimulation from different sites. Six stimulating electrodes were implanted in the electrically active region (16) of each of the anterior and posterior sides of three dogs. Four of these electrodes were near the greater curvature (2.5, 5.5, 7.5, and 11.5 cm from pylorus) and two were near the lesser curvature (5.0 and 8.0 cm from the pylorus). Gastric control waves of the entire electrically active region in each dog could be entrained from each of these sites on either side of the stomach.

In an undriven stomach, the control wave of the highest intrinsic frequency oscillator (located in the corpus near the greater curvature (16)) has phase lead over all other control waves. When driven electrically, the control wave of the oscillator beneath the stimulating electrodes had phase lead over all other control waves. When the electronic pacemaker was implanted near the lesser curvature (three dogs), the direction of phase lag was from the lesser curvature to the greater curvature and vice versa when the pacemaker was implanted near the greater curvature (see below).

For a given pulse duration, the minimum pulse amplitude for the entrainment of gastric control waves depended on the difference between natural gastric frequency and the driven frequency. Minimum pulse amplitude required for entrainment increased with an increase in the difference between driven and natural frequencies as shown in Fig. 2A for one dog. A similar relationship was found in three other dogs.

The gastric control waves were driven from three sites (Fig. 1) in eight dogs to study the process of entrainment, the process of control waves resuming their natural patterns on switching off the electronic pacemaker, and the maximum driven frequencies. The entrainment of gastric control waves by the electronic pacemaker was gradual. In all cases,

![Diagram showing placement of stimulating electrodes and recording electrodes](http://ajplegacy.physiology.org/fig1.jpg)

**FIG. 1.** Diagram showing placement of stimulating electrodes (SE 1, SE 2, and SE 3) and recording electrodes (1–6). This arrangement of stimulating electrodes was used in all dogs except first 3 in which 6 stimulating electrodes were implanted on each of anterior and posterior sides. Broken lines show cuts made to divide stomach into 3 segments.

![Graph showing stimulus amplitude vs driven frequency](http://ajplegacy.physiology.org/fig2a.jpg)

**FIG. 2A:** Minimum stimulus amplitude required to entrain gastric control waves at different driven frequencies. Electronic pacemaker was implanted between electrodes 1 and 2 (Fig. 1). Natural gastric frequency was 4.85 cycles/min. **B:** Phase lag as a function of driven frequency. Phase lag was measured between electrodes 2 and 6 (Fig. 1) implanted at 7.8 and 1.0 cm from pylorus, respectively. Natural gastric frequency was 5.2 cycles/min.
control waves distal to the electronic pacemaker were entrained first and then the proximal ones were entrained. Figure 3 shows the gradual entrainment of gastric control waves when the pacemaker was located near the greater curvature 6.3 cm from the pylorus.

Before stimulation, the control wave at electrode 1 had phase lead over all other control waves, and the direction of phase lag was aboral. When entrained by the electronic pacemaker, the control wave at electrode 4 had phase lead over all other control waves, and the direction of phase lag proximal to the pacemaker was oral, while distal to it the direction of phase lag was aboral as before. This is further evident by tracing the last driven control potential of each control wave when stimulation was stopped as shown in Fig. 4.

When the electronic pacemaker was located near the highest intrinsic frequency oscillator (SE 1, Fig. 1), the gastric control waves could be driven at higher frequencies without any change in the direction of phase lag. If the pacemaker was located near the pylorus (SE 3, Fig. 1), the direction of phase lag was reversed all the way up to the highest intrinsic frequency oscillator.

After the entrainment of gastric control waves, if the pacemaker was switched off, the highest intrinsic frequency oscillator did not resume its normal frequency and phase lead over other control waves for periods lasting up to 2 min (Fig. 4). Immediately after switching off the pacemaker, the control waves had longer periods than their normal period, and there was no definite pattern of phase lag among them during this period. This phenomenon occurred only if the control waves were being driven at a frequency higher than their natural frequency at the time of switching off of the electronic pacemaker.

The maximum driven frequency (MDF) of the intact stomach varied in different dogs. Maximum driven frequency was taken to be the highest frequency at which the
control waves at all the recording electrodes and presumably of the entire electrically active region in the stomach could be entrained by the electronic pacemaker with no systematic variation in phase lag for periods greater than 10 min. This definition was necessary because, as shown below, the distal stomach could be driven at a higher frequency than the proximal stomach. If the pacemaker was implanted in the antrum and the stomach was driven at a frequency higher than the MDF of isolated corpus, only the antral control waves were entrained. The frequency of control waves in the corpus was merely being pulled, i.e., the proximal control waves had a higher frequency than before stimulation but were not phase-locked with the antral control waves and the electronic pacemaker.

The range of values for MDF was 6.5–7.7 cycles/min in eight dogs and the mean value was $6.98 \pm 0.542$ SD cycles/min. The mean undriven frequency in these dogs was 5.07 $\pm 0.373$ SD cycles/min.

**Effect of electrical stimulation on phase lags.** Driving the gastric control waves at a frequency higher than their natural frequency increased the phase lag among them. Table 1 shows phase lags (in seconds) between control waves at electrodes 2 and 6 (Fig. 1) in the driven and undriven stomach of six dogs. Mean increase in phase lag per centimeter when gastric control waves were driven at frequencies higher than their natural frequencies was 0.260 $\pm 0.13$ SD sec (5 dogs). No increase in phase lag occurred when the control waves were just entrained by the pacemaker at their natural frequency.

Figure 2B shows the total phase lag between control waves in the corpus and in the antrum as a function of the driven frequency. Phase lag increased at higher driven frequencies. At each driven frequency the amplitude of stimulus was kept at threshold.

Total phase lag between control waves in the corpus and in the antrum did not depend on the direction of phase lag (aboral or oral), provided the control waves in both cases were driven at the same frequency from either a proximal or a distal electronic pacemaker. Phase lags between control waves at electrodes 2 and 5 when driven by a pacemaker in the corpus (see Fig. 1) or in the antrum (see Fig. 1) of four dogs were 10.8, 10.6; 11.1, 11.0; and 11.3, 11.6 sec. The corresponding driven frequencies were 5.4, 5.8, and 6.3 cycles/min. When the gastric control waves were driven at a frequency higher than their natural frequency by an electronic pacemaker in the antrum (see Fig. 1), the phase lag in the oral direction between recording electrodes 5 and 1 was more than the phase lag between the same electrode during normal control activity.

Increasing the amplitude of stimulus beyond threshold had very little effect on the total phase lag between corpus and antrum at any driven frequency. In one dog when the electronic pacemaker was implanted between electrodes 1 and 2, phase lag between control waves at electrodes 2 and 5 were 11.3, 11.3, 11.1, 11.0, 11.0, and 10.9 sec when the amplitude of stimulus was 2.2 v (threshold), 4.5 v, 6.0 v, 8.5 v, 9.5 v, 15.0 v, and 21.0 v, respectively. Phase lag between the stimulus and the nearest control wave at electrode 2 (0.75 cm from cathode), however, depended on the amplitude of superthreshold stimulus. The phase lag reduced from 1.6 sec at 2.2 v (threshold stimulus) to 0.083 sec at 21.0 v. Amplitudes of the control potentials of this wave also depended on the stimulus amplitude. The amplitudes of control potentials at electrode 2 increased from 0.3 mv at 2.2 v to 2.7 mv at 21.0 v. No detectable difference in amplitude was observed in the control records at other electrodes. Similar observations were made in three other dogs.

**Electrical stimulation of isolated gastric segments.** The stomach was divided into three segments by circumferential cuts through muscle layers only, and each segment was driven by a separate electronic pacemaker. The maximum driven frequencies of the three segments were different. We consistently found that the maximum driven frequency of the most proximal segment was less than that of the other two. The mean maximum driven frequencies of the proximal, middle, and the distal segments in nine dogs were 6.92 $\pm 0.6704$ SD, 7.55 $\pm 0.7561$ SD, and 7.74 $\pm 0.9098$ SD. The difference in means was, however, not significant at 5% level of significance ($F^2 = 2.60, F_{0.05,2,4} = 3.40$). This is probably due to the large variation in the maximum driven frequencies of the three segments, particularly of the distal segment. The maximum driven frequency of the distal segment depended on the level of parasympathetic activity in the dog as shown below. Although the intrinsic frequencies of distal gastric oscillators are lower than those of proximal oscillators (16, 20), their maximum driven frequencies are as high as or higher than those of the proximal gastric oscillators.

### Table 1. Effect of electrical stimulation on phase lags

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Normal Frequency, cycles/min</th>
<th>Driven Frequency, cycles/min</th>
<th>Phase Lag Between Electrodes 2 and 6, cm</th>
<th>Increase of Phase Lag/cm, sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.8</td>
<td>4.8</td>
<td>6.8</td>
<td>0.0</td>
</tr>
<tr>
<td>2</td>
<td>5.0</td>
<td>5.9</td>
<td>13.3</td>
<td>12.6</td>
</tr>
<tr>
<td>3</td>
<td>5.1</td>
<td>6.0</td>
<td>9.0</td>
<td>13.2</td>
</tr>
<tr>
<td>4</td>
<td>5.4</td>
<td>6.6</td>
<td>9.8</td>
<td>11.2</td>
</tr>
<tr>
<td>5</td>
<td>5.2</td>
<td>7.4</td>
<td>6.2</td>
<td>9.2</td>
</tr>
<tr>
<td>6</td>
<td>5.2</td>
<td>7.3</td>
<td>6.8</td>
<td>9.2</td>
</tr>
</tbody>
</table>

1. The driven control waves were observed for a minimum period of 10 min after entrainment. If during this period, phase lag between the stimulus and the control waves did not increase, the control waves were considered to be entrained indefinitely. We observed that if the control waves were not entrained but were merely pulled up in frequency, the phase lag would increase gradually and eventually the control waves would get unphase-locked. We were unable to drive gastric control waves at frequencies lower than their natural frequency for indefinite periods. Gastric control waves were phase-locked temporarily at lower frequencies but became unphase-locked in less than 10 min.

2. To test the hypothesis of no difference in population means, the ratio $F = n S^2 / \frac{S^2}{2}$ called variance ratio was examined, where $S^2$ is the variance of the sample means, $S^2$ is the pooled variance, $n$ is the number of samples (in this case number of dogs). Critical value of $F$ (see $F$ tables) is denoted by $F_1$, $F_2$, $F_3$ and $F_4$ are the degrees of freedom of the numerator and denominator, respectively. The hypothesis of no difference was rejected if the computed value of $F$ was greater than its critical value.
The maximum driven frequency of the distal segment was affected by intravenous injection of physostigmine and atropine. Physostigmine lowered the maximum driven frequency, whereas atropine raised it (Table 2). The maximum effect of physostigmine was achieved with a dose of 20–60 μg/kg and that of atropine was achieved with a dose of 5–20 μg/kg. Higher doses of atropine (up to 100 μg/kg) had no further effect on the maximum driven frequency. The maximum driven frequencies of distal segments in four dogs in the normal state with physostigmine and with atropine are shown in Table 2. The difference in means of the three columns was investigated by analysis of variance. The means were significantly different at 5% level of significance (F-value for middle segment = 76.6, F0.05,2,9 = 4.26).

The highest maximum driven frequency of antrum under the influence of atropine was 9.9 cycles/min. The intrinsic frequency of this segment was 2.8 cycles/min.

The effects of intravenous injections of physostigmine and atropine on the middle and the proximal segments are also summarized in Tables 3. These drugs had very little or no effect on the maximum driven frequencies of these segments. The difference in mean maximum driven frequencies of these segments in the normal state with physostigmine and with atropine was not statistically significant at 5% level of significance (F-value for middle segment = .81, F-value for proximal segment = 0.053, F0.05,2,9 = 4.26).

Distance of first cut from pylorus = 7.0 cm. Distance of second cut from pylorus = 11.0 cm. Weight of dog = 16.5 kg.

**TABLE 2. Effect of physostigmine and atropine on maximum driven frequencies of isolated gastric segments in one dog**

<table>
<thead>
<tr>
<th>Dose</th>
<th>Maximum Driven Frequency, cycles/min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Distal segment</td>
</tr>
<tr>
<td>Normal</td>
<td>7.4</td>
</tr>
<tr>
<td>200 μg physostigmine</td>
<td>6.9</td>
</tr>
<tr>
<td>50 μg atropine</td>
<td>8.1</td>
</tr>
<tr>
<td>100 μg atropine</td>
<td>8.7</td>
</tr>
<tr>
<td>200 μg atropine</td>
<td>8.8</td>
</tr>
<tr>
<td>1.35 mg atropine</td>
<td>8.7</td>
</tr>
</tbody>
</table>

Distance of first cut from pylorus = 7.0 cm. Distance of second cut from pylorus = 11.0 cm. Weight of dog = 16.5 kg.

**DISCUSSION**

This study shows that the frequency and direction of phase lag of gastric electrical control activity can be influenced by electronic pacemakers. Kelly and La Force (9) have obtained similar results in awake dogs using silver point electrodes and silver wire wrapped around the gastric wall. Bilgutay et al. (2) stimulated the stomach in man but with very different parameters of stimulation. They used a stimulus frequency of 50 Hz applied for 5–10 sec at intervals varying from 1 to 5 min. They claimed that such stimulation when applied for several hours shortened recovery from paralytic ileus. Other investigators (1, 11, 14) in controlled studies of patients with and without electrical stimulation found that the stimulation method of Bilgutay et al. had no effect on gastrointestinal motility.

This study and that of Kelly and La Force (9) suggest a different approach to control of gastric motility, i.e., total number of contractions and their sequence which cause mixing and propulsive movements should be influenced by altering the pattern (frequency, direction of phase lag, etc.) of gastric electrical control activity. It must be emphasized that electrical stimulation to drive gastric electrical control activity did not produce any response activity or contractions. However, if the response activity and the contractions were naturally occurring, increasing the frequency of control activity or altering its direction of phase lag also increased the frequency of occurrence of response activity and altered its direction of phase lag correspondingly.

The entrainment of control waves in the entire electrically active region by an electronic pacemaker implanted at any site in this region can be explained on the basis of the computer model of the gastric electrical control activity proposed by us earlier (16). Comparison of behavior of this model to that of the stomach shows that the gastric control activity is due to an array of bidirectionally coupled relaxation oscillators. When a series of electrical pulses of adequate amplitude and frequency close to the natural gastric frequency are applied at a site, they produce a premature control potential (18) in every cycle of the gastric oscillator at that site. This has the effect of artificially raising the intrinsic frequency of the gastric oscillator at that site. Since in a system of bidirectionally coupled relaxation oscillators the highest intrinsic frequency oscillator dominates, the gastric oscillator beneath the pacemaker pulls the frequencies of other oscillators to that of its own and has the most leading control wave.

These experiments confirm that a model of gastrointestinal electrical control activity must have bidirectional coupling rather than a unidirectional coupling as proposed by Nelsen and Becker (12) and Diamant, Rose, and Davison (6). With forward coupling alone, the entrainment of proximal control waves would not be possible.

Suprathreshold stimuli reduced only the phase lag between the stimulus and the neighboring oscillator, which was being directly driven by the pacemaker. These stimuli had no effect on the phase lags among distal oscillators because they were not being directly driven by the pacemaker. Each distal oscillator was being driven by its nearest proximal oscillator, the amplitude of oscillation of which had not changed. The phase lag between the stimuli and the control potentials at the nearest recording electrode could decrease at suprathreshold stimuli because: 1) an increased amplitude of stimuli is equivalent to a greater coupling factor and the phase lag is reduced as coupling factor becomes larger; 2) with increased stimuli, the current could be spreading over a larger region around the stimulating electrodes, and thus the stimulus would be driving more oscillators directly rather than driving them indirectly through the intervening oscillators, which causes additional phase lag.

2 We define the antrum electrophysiological as that region in which there is strong transverse and longitudinal coupling (19).
The observation that the entrainment of proximal oscillators by an electronic pacemaker is a gradual phenomenon supports the applicability of a relaxation oscillator model to the gastric electrical control activity as compared to the applicability of a cable model. When the electronic pacemaker is located in the distal stomach, each individual proximal gastric oscillator has to undergo a change in phase relationship with the distal oscillator so that when a control potential is produced in the latter, the former is in a relatively refractory state (18) and hence can be entrained. That the distal control waves are entrained by the electronic pacemaker before the proximal control waves agrees with our earlier findings both in the dog stomach and in the gastric ECA computer model that distal propagation of premature control potentials is easier than proximal propagation. This is because in the stomach the distal control waves are in a relatively less refractory state than the proximal control waves when a premature control potential is produced in the latter part of the control wave cycle (18).

Although it was possible to drive the gastric control waves from any site in the electrically active region, there was a limit to the maximum frequency at which the control waves could be driven. This limit seems to depend on the duration of the absolutely refractory state of a control wave period. Daniel and Irwin (5) and Sarna, Daniel, and Kingma (18) have shown that up to 50-70% (varies from dog to dog) of the control wave period immediately following a control potential is absolutely refractory to the initiation of premature control potentials. In the rest of the period premature control potentials could be produced by external stimulation. These premature control potentials propagate proximally, distally, and sidewise (18).

The mean MDF (6.98 cycles/min) in our experiment was lower than that reported by Kelly and La Force (8.0 cycles/min (7)). This could be due to two reasons: 1) Kelly and La Force did not raise the frequency high enough to study its effects on phase lags or 2) distances between the electrodes at which they measured phase lags were smaller than ours, and hence changes in phase lags might not have been detectable.

In case of phase lag could possibly be explained on the basis of refractory curves of relaxation oscillators. We (13, 18) have shown, both in the computer model and in the dog stomach, that the threshold stimulus required to produce a premature control potential reduces progressively in the relatively refractory state of the control wave cycle. Figure 3 shows the threshold curve of a relaxation oscillator in the computer model. Similar threshold curves exist for gastric relaxation oscillators (15). As the driven frequency is increased, the stimulus arrives earlier and earlier in the cycle and with the strength of stimulus from the neighboring oscillator remaining the same, the excess of stimulus am-
plitude over the threshold amplitude would decrease. Our experiments on electrical stimulation show that as the amplitude of suprathreshold stimulus is decreased, phase lag increases.

No change in phase lag was observed when the control wave propagated proximally or distally at the same frequency. This means that the coupling factors among oscillators and the overall pattern in which oscillators are coupled are not affected by electrical stimulation. Also the coupling factors in both directions are identical.

Increase in the threshold stimulus required to entrain gastric control waves as the driven frequency is increased can also be explained on the basis of threshold curves of gastric relaxation oscillators to produce premature control potentials. Increase in the driven frequency requires that the electrical stimulus be delivered earlier in the control wave cycle, and since the threshold stimulus to produce premature control potentials is higher in the earlier phases of the cycle, the stimulus strength required to produce premature control potentials would also increase.

The maximum driven frequency of the three isolated segments before drug treatment varied from dog to dog. We have shown that parasympathetic activity affects mainly the distal stomach, and most variation occurred in the response of this region. One of the possible reasons for this could be different levels of sympathetic and parasympathetic activities in these dogs. The role of release of norepinephrine due to sympathetic activity in controlling the maximum driven frequency is yet to be studied.

The ability of the distal stomach to be driven at higher frequencies than those of the proximal stomach after withdrawal of the influence of the vagus following atropine could provide a mechanism to explain instances of impaired gastric emptying (from a pathological point of view). If the hypothesis is correct that gastric emptying is caused, at least in part, by sequential contractions controlled by electrical control activity, then the above-mentioned characteristic gives the electrical control activity the ability to reverse the direction of sequential contractions (antiperistalsis) and thus impede emptying. In order to be able to reverse the direction of phase lag, the antrum has to acquire an intrinsic frequency higher than that of the corpus. Though chemical vagotomy with atropine prevents response activity, antiperistalsis can occur in the absence of vagal influences when response activity is induced by acetylcholine-independent mechanisms.

The ability to influence the frequency and direction of phase lag of control waves can prove useful in controlling gastric emptying in the diseased state or in the postoperative state (1, 2, 10, 12, 14).

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REFERENCES


