Impairment of Gastric Myoelectricity After Catheter Ablation of Atrial Fibrillation

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Background: Dyspepsia is not uncommon in patients undergoing ablation of atrial fibrillation (AF). The purpose of this study was to investigate the change in gastric myoelectrical activity and its relationship to the outcome after ablation by electrogastrography (EGG).

Methods and Results: The study prospectively enrolled 32 patients: 16 patients (Group 1) had AF and 16 (Group 2) had paroxysmal supraventricular tachycardia. The myoelectricity-like signals of the gastrointestinal tract were recorded using EGG before, immediately, and 3 months after ablation. Both fasting and postprandial abnormal gastric wave percentages increased immediately after ablation in Group 1 when compared with before ablation. Increased fasting (12.8±15.2% vs. 22.3±21.0%, P=0.04) and postprandial (10.4±6.2% vs. 21.2±14.5%, P=0.01) bradygastria percentages were noted immediately after ablation in Group 1 when compared with before ablation. These findings were not observed in Group 2. The normal gastric wave percentage returned to baseline 3 months after procedure. In total, 4 (25%) patients in Group 1 had a recurrence of AF after a follow-up of 18±3 months; those without recurrence had a significantly higher percentage of postprandial abnormal gastric waves immediately after ablation when compared with recurrence.

Conclusions: Transient gastric dysrythmias with bradygastria does occur in AF patients after ablation, which may indicate periesophageal vagal plexus damage after ablation. Careful application of radiofrequency energy to prevent esophageal injury is important. (Circ J 2013; 77: 2014–2023)

Key Words: Ablation; Atrial fibrillation; Electrogastrography; Myoelectricity; Outcome

A
dominal bloating and dyspepsia have been observed in some patients after catheter ablation of atrial fibrillation (AF). 1–3 It may develop within a few hours to 2 days after the procedure and an incidence of 1% has been reported. 1,4,5 Gastric hypomobility with a markedly prolonged gastric half-emptying time have been noted after ablation. 1,2 Symptoms and tools such as esophagogastroduodenoscopy or an upper gastrointestinal series are currently used for the diagnosis of periesophageal vagal injury, 3 but it is difficult to fully discover and evaluate a motility disorder with these tools. Additionally, the incidence seems to be higher in clinical practice than that reported in the literature because some symptoms are vague and difficult to be measured.

Gastric motility is modulated by gastric myoelectrical activity, which is composed of gastric slow waves and spiked potentials. The slow waves are normally 3 cycles/min (cpm) in humans and determine the propagation and gastric contraction frequency. They can be measured noninvasively by placing electrodes on the abdominal skin, a technique known as electrogastrography (EGG). EGG can measure the gastric slow waves and detect any underlying physiological or pathological gastric myoelectrical activity. 6–11 The purpose of this study was to investigate gastric myoelectrical activity by noninvasive EGG and evaluate the relationship of changes to long-term

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outcome after catheter ablation of AF.

**Methods**

We prospectively enrolled 32 consecutive patients: Group 1 comprised 16 patients who underwent an electrophysiological study/ablation therapy for AF (paroxysmal/non-paroxysmal: 12/4) and Group 2 consisted of 16 patients who underwent an electrophysiological study/ablation therapy for a non-AF condition (including 10 atrioventricular nodal reentrant tachycardias, 2 orthodromic atrioventricular reciprocating tachycardias, 1 typical atrial flutter, and 3 sick sinus syndrome) as the control group. All of them underwent EGG before the electrophysiological study/radiofrequency catheter ablation (RFCA) procedure. The EGG was performed 1 day before, and immediately (within 24 h) and 3 months after the ablation procedure. The Group 1 patients also underwent Holter monitoring, transesophageal echocardiography and computed tomography before and after RFCA.

**EGG System**

**EGG Recording** The gastrointestinal myoelectricity activity was recorded using an EGG recorder (EGG, MMS, Inc, The Netherlands). In brief, the skin at the recording sites was cleaned with an abrasive skin-preparation (OmniPrep, Weaver & Co, Aurora, CO, USA) to reduce the impedance; hair was shaved if present. Three silver-silver chloride electrodes were placed on the abdominal surface. In brief, 1 electrode (electrode 1) was placed at the midpoint between the xiphoid and navel; another (electrode 2) was placed 5 cm to the left and 3 cm above that point; and a reference electrode (electrode 0) was placed in the lower quadrant close to the left costal margin. The bipolar EGG signal was derived from electrodes 1 and 2 and amplified through the EGG recorder with low and high cutoff frequencies of 1 and 18 cpm, respectively.

All patients fasted overnight before the EGG study. A 30-min baseline EGG recording in the fasting state was made after the placement of the electrodes on the abdomen. Next, a solid meal consisting of a 450 Kcal sandwich was consumed within 10 min and a postprandial EGG was recorded for 1 h. All recordings were made in a quiet room with the patient supine and asked not to talk during the procedure.

**EGG Dominant Frequency (DF)** The stored signals on the computer were analyzed using a commercialized system to obtain the dominant frequency. Briefly, a smoothed power spectral analysis was used to produce the overall power spectrum of the entire recording period that displayed the peak power and this was defined as the DF. The DF of the EGG has been shown to be equal to the frequency of the gastric slow waves measured from implanted serosal electrodes. The smoothed power spectral analysis was used to produce the overall power spectrum of the EGG during each recording period.

**Definition of Normal and Abnormal Gastric Slow Waves**

The percent of regular gastric slow waves, which reflected the regularity of the gastric myoelectrical activity, was defined as the percentage of time during which normal 2.4–3.5 cpm slow waves were present over the entire observation period. This was computed by the adaptive running spectral analysis method. Each EGG recording was divided into blocks of 1 min without overlap. The power spectrum of each 1-min EGG was calculated and examined to see if the peak power was within the range of 2.4–3.5 cpm. The 1-min EGG was defined as normal if the DF was within the 2.4–3.5 cpm range, otherwise it was defined as a dysrhythmia. Bradygastria was defined as a DF within 1.0–2.4-cpm, and tachygastria as a DF within 3.5–9.8 cpm. Therefore, the percentage of abnormal gastric waves (gastric dysrhythmia) was defined as the sum of the percentage of bradygastria and tachygastria.

**Questionnaire**

A Chinese version of the validated questionnaire suggested by the Rome Committee for the Investigation of Functional Gastrointestinal Disorders was evaluated before and immediately after RFCA on the day after the EGG recording. The questions assessed symptoms of chest pain, heart burn or acid regurgitation; postprandial abdominal fullness, bloating or swelling; early satiety; abdominal pain or fullness, relieved after a bowel movement; hard or lumpy stools, loss of mushy stools; straining during a bowel movement; and feeling of incomplete emptying after a bowel movement.

**Electrophysiological Study (EPS) and RFCA**

After providing written informed consent, each patient underwent an EPS and RFCA while in the fasting non-sedated state. All antiarrhythmic drugs except for amiodarone were ceased for at least 5 half-lives beforehand. Local anesthesia was used in the procedure and no analgesic agent was used before or after RFCA in either group.

**RFCA of AF**

The details have been described in our previous publications. In brief, for the patients with paroxysmal AF, continuous circumferential lesions were created encircling the right and left pulmonary vein (PV) ostia guided by 3D electroanatomical mapping (EnSite NavX, St. Jude Medical, St. Paul, MN, USA) using an irrigated-tip 4.0-mm (Chilli II, EPT, Boston Scientific Corporation, Natick, MA, USA or CoolPath, St. Jude Medical) tipped ablation catheter. RF energy was applied continuously while repositioning the catheter tip every 40 s (temperature 35–40°C, power 25–30 W). Supplementary ablation applications were applied along the circumferential lines close to the earliest ipsilateral PV potentials. Furthermore, ablation of the residual PV potentials was performed from the atrial side of the PV antrum using the electrogram-guided approach to obtain an entrance block. Successful circumferential PV isolation was demonstrated by the absence of any electrical activity inside the PV or dissociated PV activity during AF.

In patients with non-paroxysmal AF, if AF persisted after PV isolation, an additional CFE (complex fractionated atrial electrogram) ablation was performed sequentially based on the results of the CFE maps after circumferential PV isolation. The CFE ablation was confined to the continuous CFEs in the left atrium (LA), right atrium (RA), superior vena cava (SVC), and proximal coronary sinus. The endpoint of the CFE ablation was prolongation of the cycle length, elimination of the CFEs (thus, fractionation interval >120 ms) or abolition of the local fractionated potentials (voltage <0.05 mV). The CFE sites were reassessed by an automatic algorithm immediately after ablation. If AF terminated during linear ablation through the CFE sites, complete linear ablation to an anatomic obstacle or the nearest line was performed to prevent proarrhythmias. Pain or the vagal response during catheter ablation was recorded while targeting the LA posterior wall (during PV isolation or CFE ablation).

RA cavotricuspid isthmus ablation was performed with an 8-mm-tip EPT ablation catheter (Boston Scientific Corporation: max. power 70 W, temperature 70°C, 120 s). Bidirectional conduction block of the cavotricuspid isthmus was confirmed after restoration of sinus rhythm. If non-PV ectopic beats initiating
AF were identified after cardioversion, isolation of the arrhythmogenic SVC or coronary sinus ostium was guided by the circular catheter recordings from the SVC-RA junction. In patients with other non-PV AF ectopic beats, RFCA was performed in the area with the earliest electrical activity or a local unipolar QS pattern of the ectopic beats preceding the AF. A 4-mm conventional tip temperature control model (50–55°C, power 45–50 W, 20–40 s) was used. The endpoint of the ablation was disconnection between the SVC and RA, and coronary sinus and RA, and elimination of other non-PV ectopic beats with negative provocation of AF. The endpoint of AF ablation was disconnection between the PVs and LA, and noninducibility of atrial arrhythmias (<1 min). If induced AF or LA flutter was sustained for more than 1 min, additional LA substrate linear ablation at the mitral isthmus and anterior roof was performed at the end of the procedure. In all the AF patients, high-frequency stimulation was not performed for ganglionated plexi ablation.

RFCA of Paroxysmal Supraventricular Tachycardia (PSVT)
In patients with PSVT, each patient underwent an EPS in the fasting, unsedated state after discontinuation of all antiarrhythmic drugs for at least 5 half-lives. The methods used in this laboratory for the mapping and ablation of the slow pathway and accessory pathways have been described previously. For the antegrade slow pathway, the presumed ablation site was the 1 that exhibited a putative slow pathway potential or bipolar electrogram recorded from the distal ablation electrode, with an A/V ratio of 0.1–0.5. The presumed ablation site for the accessory pathways exhibited a typical AV fusion or possible accessory pathway potential. A catheter with a deflectable and large-tip electrode (4-mm, Boston Scientific, Inc, Watertown, MA, USA) was used for mapping and ablation. RF energy was applied with an EPT-1000 generator (EP Technology, Inc, San Jose, CA, USA) and a temperature control model [temperature 50°C (for slow AV node pathway) or 60°C (for accessory pathway)] was used. Procedural success for the AV nodal reentrant tachycardia ablation was defined as elimination of the slow pathway or modification of the slow pathway with noninducibility of AV nodal reentrant tachycardia. The endpoint of ablation of the atrioventricular reciprocating tachycardia was complete elimination of the accessory pathway conduction without an inducible tachycardia.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group 1 (n=16)</th>
<th>Group 2 (n=16)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51±19</td>
<td>52±17</td>
<td>0.82</td>
</tr>
<tr>
<td>Sex (male, %)</td>
<td>14 (88)</td>
<td>7 (44)</td>
<td>0.02</td>
</tr>
<tr>
<td>Hypertension (n, %)</td>
<td>7 (44)</td>
<td>5 (31)</td>
<td>0.72</td>
</tr>
<tr>
<td>DM (n, %)</td>
<td>2 (13)</td>
<td>1 (6)</td>
<td>1.00</td>
</tr>
<tr>
<td>CAD (n, %)</td>
<td>2 (13)</td>
<td>3 (21)</td>
<td>1.00</td>
</tr>
<tr>
<td>CHF (n, %)</td>
<td>2 (13)</td>
<td>1 (6)</td>
<td>1.00</td>
</tr>
<tr>
<td>Stroke history (n, %)</td>
<td>1 (6)</td>
<td>0 (0)</td>
<td>1.00</td>
</tr>
<tr>
<td>PU history (n, %)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>N/A</td>
</tr>
</tbody>
</table>

CAD, coronary artery disease; CHF, congestive heart failure; DM, diabetes mellitus; PU, peptic ulcer.
Changes in Gastric Myoelectricity Post AF Ablation

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Statistical Analysis
All continuous variables are reported as the mean ± standard deviation (SD), and categorical variables as proportions. Student’s t-test was used to analyze continuous data and the chi-square test with a Yates correction or Fisher’s exact test was used to compare nonparametric data for the gastric slow wave percentages between groups. Statistical significance was established as P<0.05.

Results
General Characteristics
The baseline characteristics of the study population are shown in Table 1. There was a higher incidence of a males in Group 1.
response during targeting of the left PV posterior wall than in those without pain (56±13 vs. 32±22%, *P*<0.04). There was no percentage difference in postprandial gastric waves between patients with and without pain/vagal response during targeting of the right PV posterior wall (39±20% vs. 48±19%, *P*<0.44). The impairment of gastric slow waves was restored to the baseline level at the 3-month follow-up after the ablation. A detailed analysis showed an increased percentage of bradygastria immediately after the ablation procedure in both the fasting and postprandial states (Figure 3). There was no difference in the tachygastria percentage after the ablation in Group 1. There were no differences in the normal and abnormal gastric waves in Group 2 before, immediately after and 3 months after the ablation in either the fasting or postprandial state (Figure 2B).

### Ablation Results
All patients in Group 1 underwent PV isolation, 1 patient underwent additional anterior roof linear ablation, and 2 patients underwent additional mitral linear ablations. Two patients had CFE ablations and in them, the CFE ablation was performed at the LA mid-posterior wall. The total procedure time was 119±72 min and 26±19 min in Groups 1 and 2, respectively. The RFCA time was 66±35 min in Group 1. The ablation time over the LA posterior wall was 33±12 min. There were 10 patients (63%; 2 while targeting right PV posterior wall, 6 while targeting left PV posterior wall, 2 while targeting both right and left PV posterior walls) in Group 1 who experienced pain/vagal response during targeting of the LA posterior wall. Ablation in the posterior wall was not performed in Group 2. There were no complications after the ablation procedures in either group.

### Results From EGG Study
All patients in both groups underwent EGG studies before, immediately after and 3 months after the ablation. The fasting and postprandial DFs were similar between groups before, immediately after and 3 months after the ablation. There were no significant differences among the DFs before, immediately after and 3 months after the ablation in either the fasting or postprandial state in Groups 1 and 2 (Figure 1).

The baseline fasting and postprandial percentages of the normal and abnormal gastric waves were similar between the groups. In the Group 1 patients, the percentage of normal gastric waves decreased, and the percentage of abnormal gastric waves increased immediately after the ablation (both fasting and postprandial), when compared with before ablation. The finding was more significant after meals (Figure 2A). The percentage of postprandial abnormal gastric waves after ablation was similar in those with and without pain/vagal response during targeting of the LA posterior wall (49±18% vs. 38±22%, *P*=0.35). However, the percentage of postprandial abnormal gastric waves was higher in the patients with pain/vagal response during targeting of the left PV posterior wall than in those without pain (56±13 vs. 32±22%, *P*=0.04). There was no percentage difference in postprandial gastric waves between patients with and without pain/vagal response during targeting of the right PV posterior wall (39±20% vs. 48±19%, *P*<0.44). The impairment of gastric slow waves was restored to the baseline level at the 3-month follow-up after the ablation. A detailed analysis showed an increased percentage of bradygastria immediately after the ablation procedure in both the fasting and postprandial states (Figure 3). There was no difference in the tachygastria percentage after the ablation in Group 1. There were no differences in the normal and abnormal gastric waves in Group 2 before, immediately after and 3 months after the ablation in either the fasting or postprandial state (Figure 2B).

Figure 4 shows an example of a Fourier transformation result from the Group 1 and Group 2 patients. A significant DF spike at 3 Hz was observed before the ablation in both groups during the fasting and postprandial states. Disorganization of the gastric slow waves with a loss of the normal DF spike occurred only in the Group 1 patient after the ablation, especially postprandial. The normal gastric slow wave pattern remained after the ablation in the Group 2 patients.

### Questionnaire Survey
The functional gastrointestinal disorder questionnaire was evaluated before and immediately after the ablation procedure on the day of the EGG evaluation in 10 and 11 patients in Group 1 and Group 2, respectively (Table 2). The patients in Group 1 had a higher percentage of chest pain, heart burn or acid regurgitation, postprandial abdominal fullness, and bloating or swelling symptoms after the ablation procedure when compared with Group 2 patients. There were no differences between the groups for the other gastrointestinal symptoms. shows a detailed comparison between the groups.

According to the questionnaire results, 8 of the 10 patients in Group 1 developed gastrointestinal symptoms after the ablation. In these symptomatic patients, there were significant increases in the postprandial percentages of abnormal gastric slow waves compared with Group 2. There were no significant differences between the 2 groups for the other parameters.
Changes in Gastric Myoelectricity Post AF Ablation

With paroxysmal AF had a better outcome after AF ablation. Regarding the procedural parameters, there was a trend to longer procedure time in Group 1 patients who had recurrence compared with those without recurrence (174±55 min vs. 95±67 min, P=0.06). There were no differences in total ablation time (73±39 min vs. 63±26 min, P=0.35) or total posterior wall ablation time (38±7 min vs. 29±7 min, P=0.13). But the left PV posterior wall ablation time (21±4 min vs. 16±3 min, P=0.04) was longer in those with than in those without recurrence. There were no differences in the pre-ablation fasting, (24±18% vs. 42±18%, P=0.03) and bradygastria (11±6% vs. 24±14%, P=0.04). These finding was not obvious in the asymptomatic Group 1 patients.

Clinical Outcome and EGG Results
During a follow-up of 18±3 months, 4 (25%) patients in Group 1 had a recurrence of AF (AF/AF+atrial tachycardia/AF+atrial flutter: 2/1/1). None of the Group 2 patient had any recurrence of arrhythmias. Table 3 shows the baseline and clinical parameters of those without and with recurrence in Group 1. Patients with paroxysmal AF had a better outcome after AF ablation. Regarding the procedural parameters, there was a trend to longer procedure time in Group 1 patients who had recurrence compared with those without recurrence (174±55 min vs. 95±67 min, P=0.06). There were no differences in total ablation time (73±39 min vs. 63±26 min, P=0.35) or total posterior wall ablation time (38±7 min vs. 29±7 min, P=0.13). But the left PV posterior wall ablation time (21±4 min vs. 16±3 min, P=0.04) was longer in those with than in those without recurrence. There were no differences in the pre-ablation fasting,

![Figure 4. Representative Fourier transformation results from Group 1 (A,B) and Group 2 (C,D) patients. The solid arrow indicates the timing sequence. The spectrum below the dotted line represents the DF analysis in the fasting state, while the spectrum above the dotted line represents the postprandial analysis. A 3-Hz DF spike in the fasting state can be seen in all 4 panels. Disorganization of the gastric slow waves with a postprandial loss of the normal DF spike can be seen in (B). AVNRT, atrioventricular nodal reentrant tachycardia; DF, dominant frequency.]
diately after ablation of AF. An attenuation of normal gastric slow waves with an increasing bradygastria component, especially postprandial and in patients who had pain/vagal response during isolation of posterior wall left PVs, accounts for the impairment of gastric myoelectrical activity. The gastric dysrhythmias were transient and restored to baseline 3 months after the ablation. The gastric myoelectrical activity remained normal in the patients who underwent PSVT ablation. This finding indicates the possibility of thermal damage to the periesophageal vagal plexus during RFCA of AF in the posterior wall of the LA. The incidence of periesophageal damage is higher than expected, so careful application of RF energy to prevent esophageal injury and fistula formation is important.

**Discussion**

**Main Findings**

Gastric dysrhythmia and dyspepsia symptoms do occur imme-

postprandial and immediately postablation fasting DFs or the percentage of abnormal gastric waves between those without (n=12) and with recurrence in Group 1. In those without recurrence, there was a significantly higher percentage of postprandial abnormal gastric myoelectrical activity immediately after ablation when compared with that with recurrence (Figure 5). Gastric myoelectrical activity was restored to baseline by 3 months after the ablation.

**Table 2. Questionnaire Survey for Dyspepsia Symptoms Immediately After Ablation**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Group 1 (n=10)</th>
<th>Group 2 (n=11)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain, heart burn or acid regurgitation (%)</td>
<td>70</td>
<td>18</td>
<td>0.03</td>
</tr>
<tr>
<td>Postprandial abdominal fullness, bloating or swelling (%)</td>
<td>30</td>
<td>0</td>
<td>0.04</td>
</tr>
<tr>
<td>Early satiety (%)</td>
<td>20</td>
<td>9</td>
<td>0.59</td>
</tr>
<tr>
<td>Abdominal pain or fullness, relieved after a bowel movement (%)</td>
<td>20</td>
<td>0</td>
<td>0.21</td>
</tr>
<tr>
<td>Hard or lumpy stools, loss of mushy stools (%)</td>
<td>0</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>Straining during a bowel movement (%)</td>
<td>0</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>Feeling of incomplete emptying after a bowel movement (%)</td>
<td>0</td>
<td>0</td>
<td>N/A</td>
</tr>
</tbody>
</table>

**Table 3. Comparison of Clinical Factors in Patients With and Without Recurrence in Group 1**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No recurrence (n=12)</th>
<th>Recurrence (n=4)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paroxysmal AF (n, %)</td>
<td>11 (92)</td>
<td>1 (25)</td>
<td>0.03</td>
</tr>
<tr>
<td>Sex (Male, %)</td>
<td>1 (8)</td>
<td>1 (25)</td>
<td>0.45</td>
</tr>
<tr>
<td>Age (years)</td>
<td>51±22</td>
<td>45±9</td>
<td>0.77</td>
</tr>
<tr>
<td>Smoking history (n, %)</td>
<td>5 (42)</td>
<td>2 (67)</td>
<td>0.57</td>
</tr>
<tr>
<td>BMI</td>
<td>26±4</td>
<td>28±4</td>
<td>0.56</td>
</tr>
<tr>
<td><strong>Underlying disease</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension (n, %)</td>
<td>6 (50)</td>
<td>1 (25)</td>
<td>0.59</td>
</tr>
<tr>
<td>DM (n, %)</td>
<td>2 (17)</td>
<td>0 (0)</td>
<td>1.00</td>
</tr>
<tr>
<td>CAD (n, %)</td>
<td>1 (8)</td>
<td>1 (25)</td>
<td>0.45</td>
</tr>
<tr>
<td>CHF (n, %)</td>
<td>1 (8)</td>
<td>1 (25)</td>
<td>0.45</td>
</tr>
<tr>
<td><strong>Medications used before RFCA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin (n, %)</td>
<td>7 (58)</td>
<td>3 (75)</td>
<td>1.00</td>
</tr>
<tr>
<td>Warfarin (n, %)</td>
<td>4 (33)</td>
<td>2 (50)</td>
<td>0.60</td>
</tr>
<tr>
<td>β-blocker (n, %)</td>
<td>7 (58)</td>
<td>0 (0)</td>
<td>0.09</td>
</tr>
<tr>
<td>ACEI or ARB (n, %)</td>
<td>3 (25)</td>
<td>2 (50)</td>
<td>0.55</td>
</tr>
<tr>
<td>CCB (n, %)</td>
<td>6 (50)</td>
<td>2 (50)</td>
<td>1.00</td>
</tr>
<tr>
<td>Digoxin (n, %)</td>
<td>0 (0)</td>
<td>1 (25)</td>
<td>0.25</td>
</tr>
<tr>
<td>Propafenone (n, %)</td>
<td>2 (17)</td>
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<td>1.00</td>
</tr>
<tr>
<td>Amiodarone (n, %)</td>
<td>4 (33)</td>
<td>4 (100)</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>Echocardiology parameters</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>LAD (mm)</td>
<td>38±7</td>
<td>43±9</td>
<td>0.26</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>56±10</td>
<td>62±8</td>
<td>0.26</td>
</tr>
<tr>
<td>LVIDd (mm)</td>
<td>48±5</td>
<td>50±3</td>
<td>0.32</td>
</tr>
<tr>
<td>LVIDs (mm)</td>
<td>29±8</td>
<td>32±5</td>
<td>0.38</td>
</tr>
<tr>
<td>RAE (n, %)</td>
<td>4 (33)</td>
<td>2 (50)</td>
<td>0.60</td>
</tr>
</tbody>
</table>

ACEI, angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; ARB, angiotensin-receptor blocker; BMI, body mass index; CAD, coronary artery disease; CCB, calcium-channel blocker; CHF, congestive heart failure; DM, diabetes mellitus; LAD, left atrial diameter; LVEF, left ventricular ejection fraction; LVIDd, left ventricular internal diastolic dimension; LVIDs, left ventricular internal systolic dimension; RAE, right atrial enlargement; RFCA, radiofrequency catheter ablation.
Gastroesophageal Dysrhythmias and AF

A previous publication suggests that the autonomic nervous system is important in the initiation and maintenance of AF. There is complex autonomic innervation of the esophageal mucosa, and the vagal nerve is located at the juxtaposition of the esophagus and atrium. Any irritation of the esophagus (eg, reflux esophagitis) activates the vagal nerve and triggers AF. In fact, dysphagia is not uncommon in patients with atrial tachyarhythmias. A relationship between reflux esophagitis and AF has been reported. According to a self-reported questionnaire survey of more than 5,000 participants in a study by the Mayo Clinic, patients with esophagitis were more likely to develop AF. Based on this rationale, it is obvious that if damage to the periesophageal vagal plexus occurs as a result of RFCA AF, it might also cause motility dysfunction of the esophagus and stomach. Martinek et al reported that a significant number of patients undergoing catheter ablation of AF develop pathologic acid reflux after the procedure.

In previous studies, invasive or high-cost procedures such as esophageal endoscopy, capsule gastrointestinal endoscopy, or computed tomography were used for the diagnosis, but these tests cannot provide any direct evidence of a motility dysfunction. To the best of our knowledge, we are the first to successfully demonstrate, using noninvasive EGG, attenuation of gastric slow waves after undergoing catheter ablation of AF. This phenomenon was not observed in patients who underwent a PSVT ablation, which suggests that LA ablation itself is the most important factor in subsequent gastric dysrhythmias. The normal vagal input to the esophagus and stomach enhances normal peristalsis. The anatomical location of the esophagus is posterior to the LA, especially near the post wall of the left PV antrum. Our study also found that the development of post-ablation gastric dysrhythmia was significantly prominent in the Group 1 patients who had pain/vagal response during encircling of the posterior wall of the left PV or ablating a posterior wall CFE. Detailed evaluation of this study revealed that the dysrhythmias were secondary to an increase in bradygastria. Therefore, the results indicate that damage to the vagal input to the gastroesophageal system is the cause of the dyspepsia that develops after AF ablation. Another possible explanation is local inflammation created by RFCA could cause abnormal gastric slow waves after ablation. Based on the questionnaire, the Group 1 patients also had a higher incidence of dyspepsia symptoms after ablation, compared with Group 2.

Gastroesophageal Dysrhythmia and Possible Esophageal Injury

Esophageal injury is a potential complication after catheter ablation of the posterior LA. It is rare but fatal if an atrial-esophageal fistula develops, because of the close proximity of the esophagus to the posterior LA ablation points. In this study, the incidence of gastric dysrhythmias after the ablation procedure was higher than expected, which means we have to more gently ablate the posterior wall. Achieving PV isolation without ablating the posterior wall would be an ideal strategy, such as the so-called BOX isolation technique.

Gastroesophageal Dysrhythmia and Long-Term Outcome

It had been reported that abolition of the vagal reflex during PV isolation has a better long-term outcome. A reduction
in heart rate variability would be observed after ablation and those patients have a better long-term outcome. Therefore, PV denervation enhances the long-term benefit of AF ablation. In our study, we also found that the patients who demonstrated increasing postprandial abnormal gastric slow waves immediately after the procedure did not have recurrence of AF during a follow-up of 18 months, which was compatible with previous findings.

In an animal study, the effect of vagal denervation after ablation was transient; re-innervation developed 4 weeks after the ablation. In clinical studies, the alteration in heart rate variability was also restored to baseline 1–6 months after the ablation. In the present study, the gastric slow waves returned to their baseline state at the 3-month follow-up, which suggests that damage to the periesophageal vagal plexus was also transient and did not cause long-term injury to the patient.

Clinical Implications
Dyspeptic symptoms are common after AF ablation, but they are difficult to evaluate. In this study, we identified gastric dysrhythmias after ablation by using non-invasive EGG. Some patients also developed silent dysrhythmias without any subjective dyspepsia symptoms. Therefore, thermal injury to the adjacent vagal innervation occurs more often than expected and so, careful application of the RF energy to prevent esophageal injury and fistula formation is important. The dysrhythmias were secondary to the bradycardia that developed after successful catheter ablation of AF and the use of prokinetic agents may be helpful in patients who develop symptoms. In addition, a successful outcome can be predicted in patients who have gastric dysmotility on EGG.

Study Limitations
In this study, 4 of 16 patients in Group 1 had a recurrence of AF during follow-up. That number of cases of recurrence is limited but because there were consistent and significant results for those patients, we believe that the number of patients was adequate for demonstrating the finding. Those patients had a longer posterior wall ablation times, but lower abnormal gastric slow wave percentages following ablation. The possibility is that most (3 of 4) of them had non-paroxysmal type AF, so disease severity and a trend to a larger LA diameter (43 ± 7 mm vs. 35 ± 6 mm, P=0.11) explain the higher energy application burden.

Conclusions
The current ablative approach targeting the posterior wall of the LA has increased the risk of injury to the adjacent periesophageal vagal plexus. In this study, we found that the incidence of gastric dysrhythmias was higher than expected and some patients even exhibited abnormal gastric slow waves without any symptoms (silent dysrhythmia). An immediate attenuation of the normal gastric slow waves with an increasing bradycardia component, especially postprandial, was noted after AF ablation. Fortunately, the gastric dysrhythmias detected by EGG immediately after ablation were only transient and were restored to baseline during 3 months of follow-up. Nevertheless, the incidence of periesophageal damage is higher than expected, so careful application of RF energy to prevent esophageal injury and fistula formation is very important.

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Disclosures
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