

Patterns of Abnormal Gastric Pacemaking After Sleeve Gastrectomy Defined by Laparoscopic High-Resolution Electrical Mapping

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Abstract

Background Laparoscopic sleeve gastrectomy (LSG) is increasingly being applied to treat obesity. LSG includes excision of the normal gastric pacemaker, which could induce electrical dysrhythmias impacting on post-operative symptoms and recovery, but these implications have not been adequately investigated. This study aimed to define the effects of LSG on gastric slow-wave pacemaking using laparoscopic high-resolution (HR) electrical mapping.

Methods Laparoscopic HR mapping was performed before and after LSG using flexible printed circuit arrays (64–96 electrodes; 8–12 cm²; $n = 8$ patients) deployed through a 12 mm trocar and positioned on the gastric serosa. An additional patient with chronic reflux, nausea, and dysmotility 6 months after LSG also underwent gastric mapping while undergoing conversion to gastric bypass. Slow-wave activity was quantified by propagation pattern, frequency, velocity, and amplitude.

Results Baseline activity showed exclusively normal propagation. Acutely after LSG, all patients developed either a

distal unifocal ectopic pacemaker with retrograde propagation (50%) or bioelectrical quiescence (50%). Propagation velocity was abnormally rapid after LSG (12.5 ± 0.8 vs baseline 3.8 ± 0.8 mm s⁻¹; $p = 0.01$), whereas frequency and amplitude were unchanged (2.7 ± 0.3 vs 2.8 ± 0.3 cpm, $p = 0.7$; 1.7 ± 0.2 vs 1.6 ± 0.6 mV, $p = 0.7$). In the patient with chronic dysmotility after LSG, mapping also revealed a stable antral ectopic pacemaker with retrograde rapid propagation (12.6 ± 4.8 mm s⁻¹).

Conclusion Resection of the gastric pacemaker during LSG acutely resulted in aberrant distal ectopic pacemaking or bioelectrical quiescence. Ectopic pacemaking can persist long after LSG, inducing chronic dysmotility. The clinical and therapeutic significance of these findings now require further investigation.

Keywords Obesity surgery · Gastric motility · Gastric dysrhythmia · Gastroesophageal reflux · Interstitial cells of Cajal

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Introduction

Laparoscopic sleeve gastrectomy (LSG) is an increasingly popular surgical treatment for morbid obesity and its associated comorbidities. Major complications such as leaks and the development of fistulae are relatively infrequent [1]; however, patients may also suffer symptomatic sequelae including gastroesophageal reflux disease (GERD), nausea, and post-operative food intolerance [2, 3]. These complications are incompletely understood and may be difficult to treat.

Gastric motility is coordinated by an omnipresent bioelectrical activity termed slow waves. Slow waves originate from the pacemaker region located at the upper corpus region of the greater curvature and propagate aborally in bands at a

frequency of approximately 3 cycles per minute (cpm) [4, 5]. The interstitial cells of Cajal (ICCs) initiate and propagate slow waves, and abnormalities of ICC and disruption of their networks have been implicated in several gastrointestinal (GI) motility disorders, including chronic unexplained nausea and vomiting, gastroparesis, and reflux with regurgitation [6–8]. During LSG, the dominant sites of ICC pacemaking along the greater curvature are resected, including the normal gastric pacemaker region. However, there have been very few studies investigating the acute or chronic implications of resection of these regions on gastric pacemaking after LSG [9, 10], and it is unknown if slow-wave organization always re-models correctly or may be compromised.

Previous clinical methods for investigating slow-wave abnormalities, i.e., body surface electrogastragraphy (EGG) and sparse-electrode serosal recordings, were limited by their lack of spatiotemporal detail [8, 11]. To improve the accuracy of slow-wave analysis, high-resolution (HR) electrical mapping was recently translated to the GI field from cardiology, involving the use of dense arrays of electrodes to map slow-wave propagation sequences in fine spatiotemporal detail [12]. HR mapping allows a more comprehensive and reliable description of slow-wave initiation and conduction disorders [6, 13, 14], now allowing novel applications in problems related to gastrointestinal surgery.

In this study, we hypothesized that abnormalities of slow-wave initiation could arise after LSG, and particularly ectopic pacemakers, due to resection of normal pacemaker regions. The primary aim of this study was therefore to define the characteristics of human gastric slow-wave activity before and after LSG, using HR mapping techniques. Slow-wave frequency, propagation direction, amplitude, and velocity were compared pre- and post-surgery to analyze the acute effects of pacemaker resection. This work was then extended to evaluate intra-operative HR mapping as a diagnostic procedure in a patient suffering persistent gastric dysmotility, nausea, and food intolerance 6 months after LSG.

Methods

Study Population

Ethical approval for this work was granted by the Health and Disability Ethics Committee, New Zealand. Nine adult patients of either sex were invited to participate and provided informed consent. Eight patients were scheduled to undergo laparoscopic sleeve gastrectomy (LSG) and one patient a revision LSG to roux en-Y gastric bypass (RYGB).

All experiments were performed in vivo on patients in the operating room following general anesthesia and laparoscopic port placement. No restrictions were placed on the anesthetic protocol; routine anesthesia does not induce changes in slow-wave propagation [15], and existing descriptions of human gastric slow-wave propagation are derived from studies performed under anesthesia [4, 5]. Anesthetic agents included propofol, a short-acting intravenous opiate, muscle relaxant, and sevoflurane.

Patients undergoing LSG had no known history of gastric dysmotility, such as functional dyspepsia or gastroparesis, were not pregnant, and were not on any medications known to affect gastric slow-wave activity.

HR Mapping

Extracellular potentials were recorded using sterile HR flexible printed circuit (FPC) arrays (FlexiMap, New Zealand), previously validated for in vivo slow-wave mapping. [16, 17]. Each array had 32 electrode contacts of 0.3 mm diameter, spaced 4 mm apart, arranged in a 16×2 configuration, and up to three arrays were used simultaneously in this study (64–96 electrodes total; 8–12 cm²) (Fig. 1a).

HR mapping was performed immediately following insertion of laparoscopic ports and abdominal insufflation, but prior to tissue dissection or mobilization. The arrays were deployed into the abdominal cavity via a 12-mm trocar in the

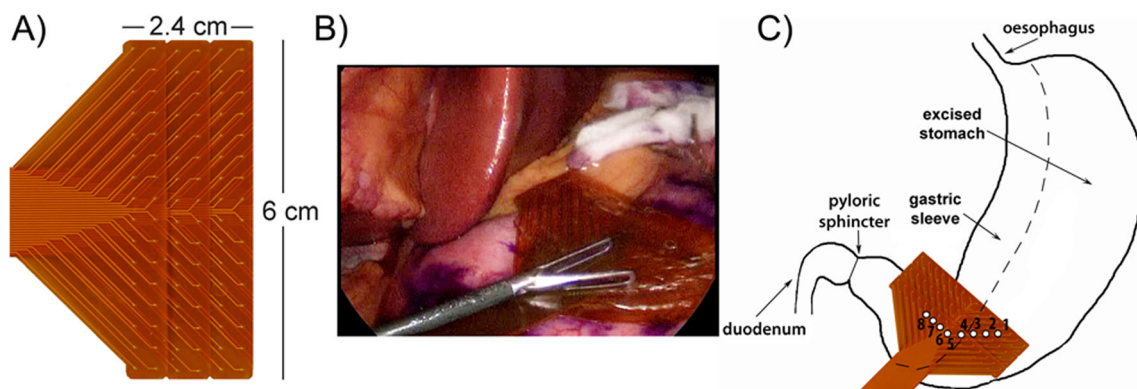


Fig. 1 Methods of data collection and electrode positioning. **a** FPC arrays with gold contacts arrayed for 96 recording channels 6×16 grid with 4 mm spacing. **b** Representative photograph of the FPC arrays being placed on the stomach. Positioning was established using the angularis

incisura marked with surgical ink as a reference point. **c** A schematic of the stomach indicating the location of the three FPC arrays in **b**. Eight recording channels are highlighted as per Fig. 2

epigastrium and were positioned on the anterior serosal surface of the stomach in the region of the distal corpus or antrum. The angularis incisura (defined by the nerves of Latarjet) was used for anatomical reference, and the procedure was recorded. Warm, moist saline-soaked gauze packs were placed on top of the arrays to maintain contact, and a 5–10-min recording period was commenced. Reference electrodes were placed on the shoulders. Following the gastric sleeve and prior to omentopexy, another 5–10 min of HR mapping was performed in the same marked region of the distal corpus or antrum. Recordings from the revision LSG patient were collected using the same techniques, stabilization and recording periods, immediately after trocar placement, and prior to RYGB. Slow-wave dysrhythmias were classified as either abnormalities of initiation (e.g., stable or unstable ectopic gastric pacemaking, quiescence) or abnormalities of conduction (e.g., conduction block and re-entry) according to previously described criteria [6, 8].

Signal Acquisition and Analysis

Signals were acquired using an ActiveTwo system (Biosemi, Amsterdam, The Netherlands), modified for passive recordings. Each array was connected to the ActiveTwo system via a sterilized 1.5-m ribbon cable, which was connected to a laptop via a fiber-optic cable. Signal processing was performed offline using the GEMS v1.6 software (FlexiMap, New Zealand) [18]. Data were filtered using a Gaussian moving median filter (20 s moving window) and a Savitzky-Golay filter (window 1.7 s, polynomial order 9) [19]. Slow-wave activation times were calculated and frequency was established by averaging the cycle-to-cycle intervals across all electrodes [20]. Activation, amplitude, and velocity maps were generated using validated algorithms followed by manual review and correction [21, 22].

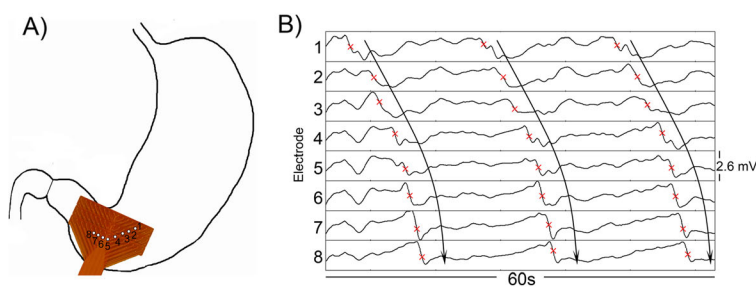


Fig. 2 Example of normal baseline slow-wave propagation (pre-LSG). See also accompanying animation Figure2.mp4. **a** Diagram showing the position of the recording array on the gastric serosa. **b** Representative electrograms showing three successive slow-wave events from a single patient during a 60-s period in the eight electrodes as highlighted in **a**. **c** Consecutive isochronal activation maps illustrate consistent aboral propagation of activity towards the pylorus. Each dot represents an

Statistical Methods

All statistical analyses were performed in IBM SPSS v22.0 (Armonk, NY). Slow-wave frequencies, velocities, and amplitudes are presented as means \pm standard deviation (SD) unless otherwise specified. Paired Student's *t* test was applied to assess statistical differences before and after LSG, with $p < 0.05$ regarded as significant.

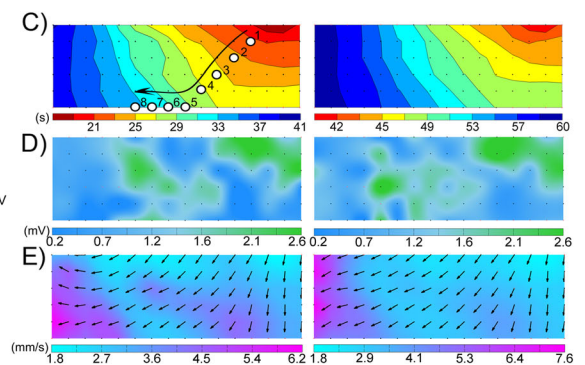
Results

Acute LSG Results

Pre-LSG Data

Intra-operative HR gastric electrical mapping was performed in a total of eight patients undergoing LSG. All of these patients were female, of median age 48 years (range 29–63), and with a median BMI of 41 (range 36–51). Figure 1b displays a representative example of the FPC electrode arrays being placed on the stomach for intra-operative data collection, and Fig. 1c shows the approximate position of the arrays relative to the angularis incisura. The mean pre-sleeve recording period was 6.0 ± 2.3 min per patient, and the mean post-sleeve recording was 5.4 ± 0.8 min.

Pre-sleeve gastric slow-wave activity was recorded laparoscopically with sufficient spatial coverage to enable detailed spatiotemporal mapping of propagation patterns in five patients and was highly concordant. A representative example of baseline gastric slow-wave propagation, captured prior to LSG, is displayed in Fig. 2 (and accompanying animation Figure2.mp4), including activation, velocity, and amplitude maps. Signal morphology was consistent with typical human extracellular slow-wave characteristics, showing a biphasic



electrode, and each color band displays the area propagation per 2-s time-lapse from red (early) to blue (late). **d** Amplitude maps, displayed by color gradient from blue (low) to green (high), and **e** velocity maps show antegrade propagation of activity (as indicated by directional arrows) towards a region of higher velocity (refer color spectrum) located in the distal antrum

morphology with a sharp downstroke and gradual recovery to baseline [17]. The mean frequency of pre-sleeve slow-wave activity was 2.7 ± 0.3 cycles per minute (cpm). Electrogram analysis and activation time mapping demonstrated consistently normal aboral propagation of slow-wave activity in all pre-sleeve recordings (e.g., Fig. 2 and Figure2.mp4), with a mean amplitude of 1.7 ± 0.2 mV and a mean velocity of 3.8 ± 0.8 mm s⁻¹. These baseline data on propagation direction, frequency, velocity, and amplitude are consistent with known normal slow-wave data in the human stomach [5].

Post-LSG Data

All patients showed major abnormalities of slow-wave initiation acutely after LSG. In half of the patients, the principle abnormality was stable ectopic gastric pacemaking whereas in

the remaining half, the principle abnormality was quiescence (absence of slow-wave activity).

Figure 3 shows examples of ectopic gastric pacemaking from two patients after LSG (see also accompany animations Figure3F.mp4 and Figure 3I.mp4), and summary data is presented in Table 1. In all cases of ectopic pacemaking, the ectopic pacemaker site was located in the distal stomach, inducing slow waves that propagated in all directions and prominently retrograde (e.g., Fig. 3d–i). Post-LSG slow-wave frequency was comparable with the pre-sleeve recordings ($p = 0.7$), and slow-wave amplitude was also similar ($p = 0.7$) (Table 1). However, the propagation velocity of wavefronts emerging from sites of ectopic pacemaking was more than 3× faster after LSG compared to baseline activity (12.5 ± 0.8 vs 3.8 ± 0.8 mm s⁻¹; $p = 0.01$).

All patients who did not develop ectopic pacemaking after the sleeve procedure instead displayed gastric quiescence.

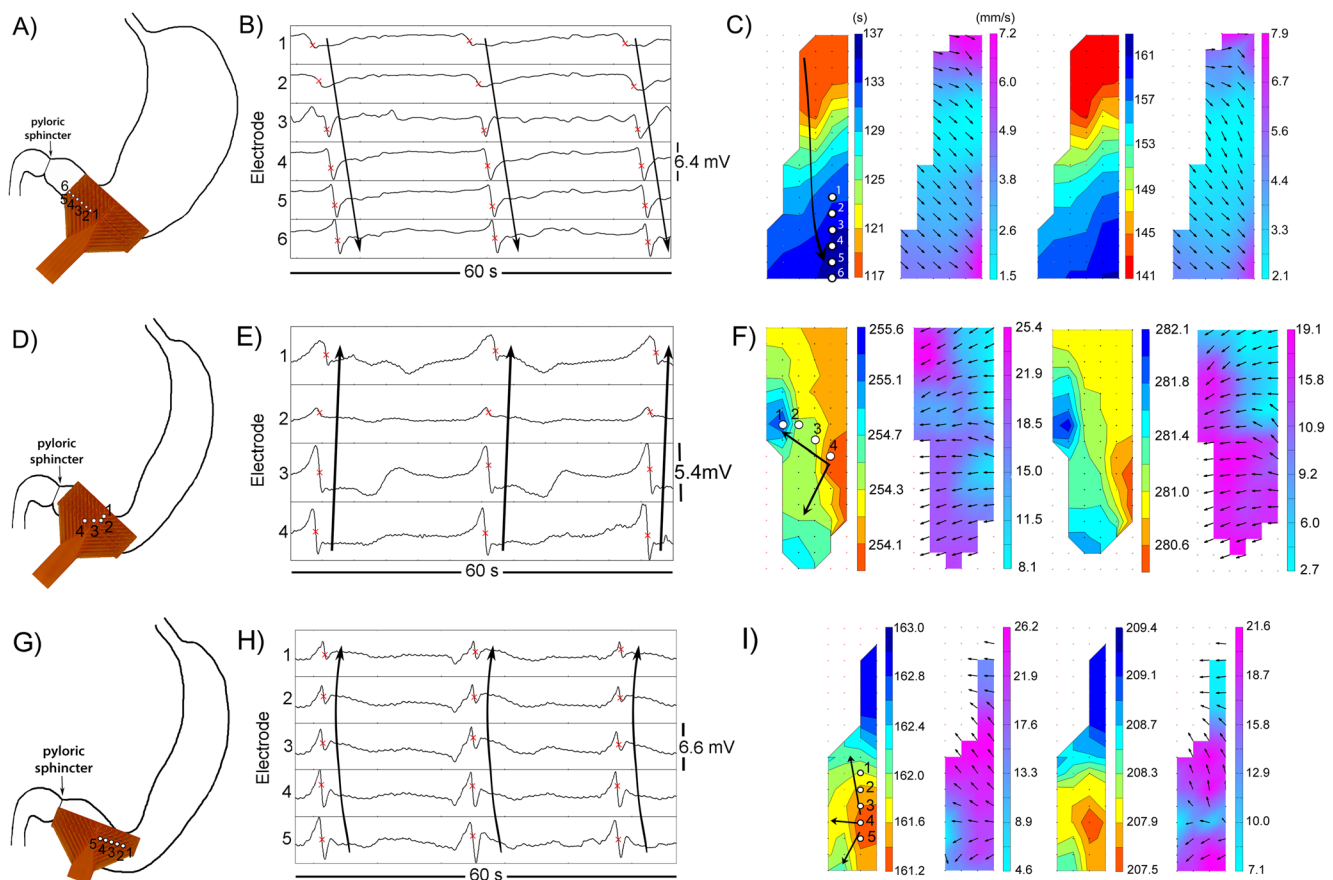


Fig. 3 Examples of ectopic gastric pacemaking after LSG. **a** Diagram showing FPC array position during pre-LSG mapping in one patient. **b** Example electrograms of three normal slow-wave events pre-LSG from a period of 60 s in six electrodes (positions indicated in **c**). **c** Activation time (2 s isochronal interval) and velocity maps (*color gradients* explained in Fig. 2) from two consecutive pre-sleeve events. Both activation and velocity maps display consistent normal antegrade propagation of slow-wave activity pre-LSG. See also animation Figure3C.mp4. **d** Diagram showing electrode array position during post-LSG mapping of the same patient represented in **a–c**. **e** Electrogram examples of three slow-wave

cycles recorded post-LSG over a 60-s period (positions indicated in **f**). **f** Consecutive activation (isochronal interval = 0.2 s) and velocity maps demonstrate rapid slow-wave propagation outward and retrograde from a stable ectopic pacemaker located in the distal antrum. See also animation Figure3F.mp4. **g, h** Position diagram and example electrograms recorded post-LSG from a second patient. **i** Activation (isochronal interval = 0.2 s) and velocity maps from two of these events show a further example of ectopic gastric pacemaking in the distal antrum with high-velocity retrograde propagation post-LSG. See also animation Figure3I.mp4

Table 1 Comparison of pre- and post-sleeve slow-wave characteristics

	Pre-sleeve	Post-sleeve	<i>p</i> value
Frequency (cpm)	2.7 ± 0.3	2.8 ± 0.3	0.7
Amplitude (mV)	1.7 ± 0.2	1.6 ± 0.6	0.7
Velocity (mm s ⁻¹)	3.8 ± 0.8	12.5 ± 0.8	0.01

Recordings were collected from 2 to 3 mapping locations across the stomach in each case, but slow-wave events were not identifiable in any channels. Sufficient contact with the gastric serosa was confirmed visually and by the identification of transmitted cardiac signals in the unfiltered signals. A representative example of gastric quiescence from one patient is shown in Fig. 4, demonstrating the presence of normal antegrade propagation of slow waves prior to LSG (Fig. 4b, c), followed by the absence of activity after the procedure (Fig. 4d).

Mapping in Chronic Dysmotility Post-LSG

The chronic effects of LSG on gastric slow-wave activity were able to be studied in one patient undergoing revision of LSG to RYGB. This 43-year-old male was referred to our unit after undergoing LSG elsewhere 6 months earlier, complicated by chronic post-operative medically refractory nausea, vomiting after every meal, gastroesophageal reflux, and epigastric pain. Body weight following the LSG changed from 110 to 70 kg over the 6-month period. Attempted therapeutic interventions included balloon dilatations of the sleeve, laparoscopy with adhesiolysis and gastropexy to prevent possible sleeve twisting, and prescription of proton pump inhibitors (omeprazole 40 mg b.d.), which were all ineffective. Barium swallow demonstrated holdup of contrast at the level of the distal antrum

and pylorus, with dyscoordinated peristaltic contractions associated with reflux from the residual stomach into the esophagus. The patient was brought forward for revision to RYGB.

Gastric mapping was performed over the anterior serosa of the corpus and antrum in four different locations for a total of 15 min, and the findings are demonstrated in Fig. 5 and accompanying animation Figure5.mp4. Electrical quiescence was observed throughout the upper and middle corpus, whereas in the antrum and lower corpus, a stable ectopic pacemaker was located at the antral greater curvature region of the sleeve, operating at a regular rhythm and frequency of 2.2 ± 0.1 cpm throughout the recording period (Fig. 5a–c). Activity propagated outward and retrograde from the ectopic pacemaker at a rapid propagation velocity of mean 12.6 ± 4.8 mm s⁻¹, with extracellular waveforms showing a mean amplitude of 2.3 ± 1.9 mV. Velocity and amplitude were highest in the vicinity of the ectopic pacemaker location (Fig. 5d, e). The location, propagation characteristics, velocity, and amplitude of this aberrant pacemaking pattern, occurring 6 months after sleeve gastrectomy, were similar to those of the acute ectopic pacemakers mapped immediately after sleeve gastrectomy above.

Discussion

LSG is an increasingly popular bariatric procedure, due to its effective weight loss outcomes, low complication rates, and relatively simplicity [23]. However, the effects of LSG on the underlying slow-wave activity that coordinates gastric motility have rarely been considered [9]. This may be because accurate tools for the clinical measurement of abnormal gastric slow-wave propagation, i.e., HR electrical mapping, have only recently been developed [12, 14].

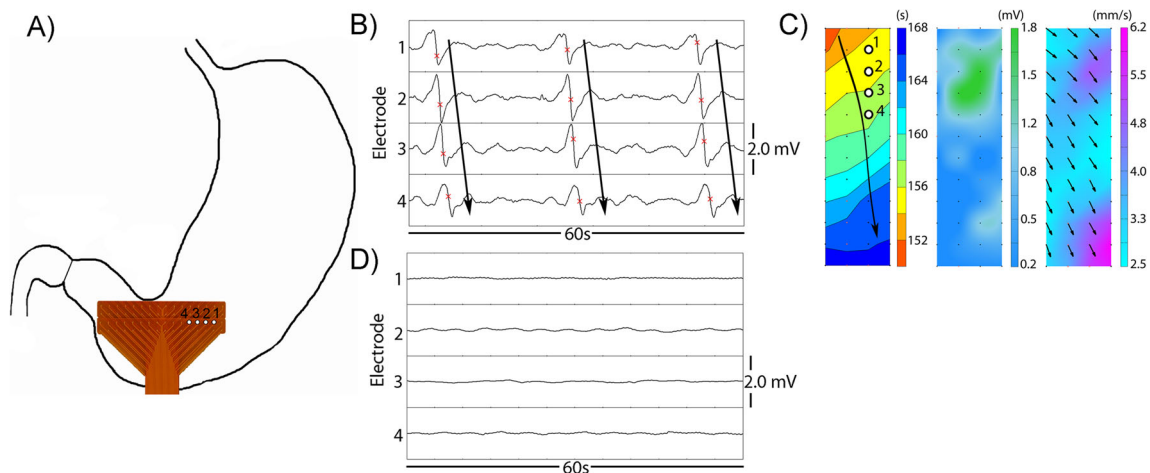


Fig. 4 Representative example of post-surgical quiescence. **a** Schematic showing electrode position on the stomach. **b** Example electrograms of slow-wave activity prior to LSG displaying three slow-wave events over a period of 60 s. **c** The activation map (isochronal interval 2 s) and

directional arrows on the velocity map indicate normal antegrade propagation. **d** Post-LSG electrograms from the same patient, revealing no slow-wave activity (quiescence)

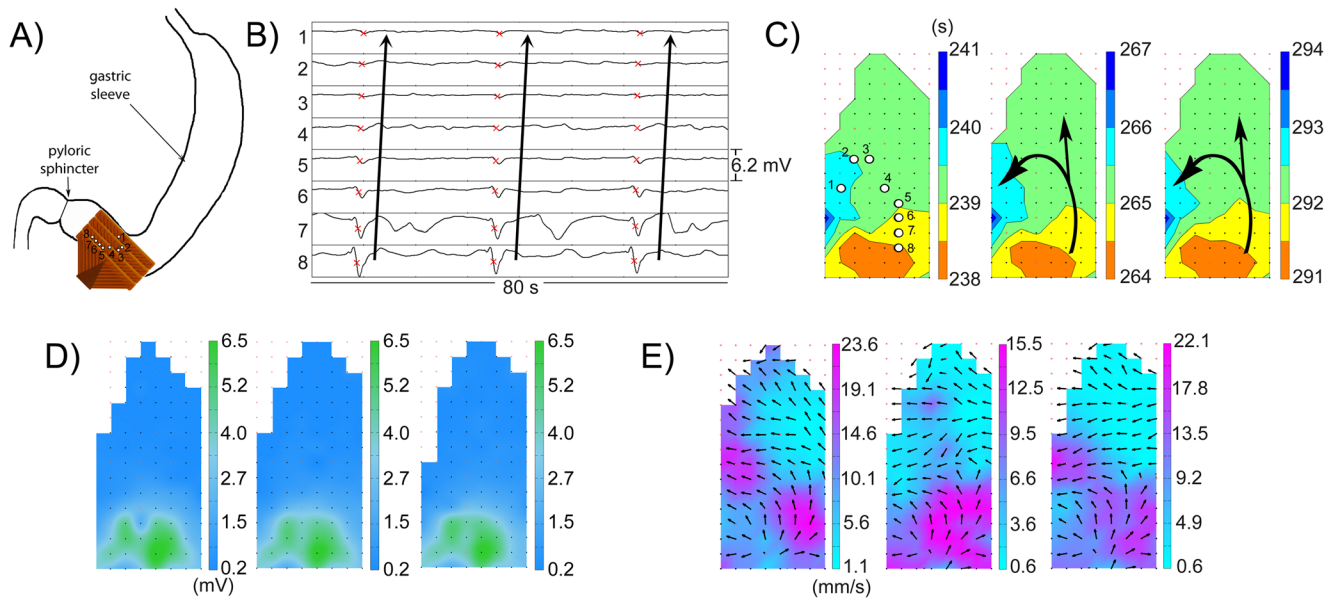


Fig. 5 Example slow-wave signals and associated maps of a patient with chronic dysmotility 6 months after LSG. **a** Electrode position diagram. **b** Electrograms showing three consecutive slow-wave events over an 80-s period. **c** Activation maps (isochronal interval 0.5 s) of the same three successive cycles show gastric dysrhythmia, classified as a stable ectopic pacemaker, with retrograde propagation. *Isochrones* demonstrate the activation sequence from early [8] to late [1], and *black arrows* indicate

the retrograde propagation of slow waves. **d** Consecutive amplitude maps show a consistent area of high amplitude located in the region of the ectopic foci. **e** Successive velocity maps illustrate rapid propagation of slow-wave activity arising in all directions from the ectopic pacemaker source and prominently retrograde (see also accompanying animation Figure5.mp4).

This study applied HR mapping to define the effects of LSG on gastric slow-wave initiation and conduction. The results demonstrate that surgical excision of the primary gastric pacemaker region during LSG induces either the acute gastric slow-wave quiescence or the generation of distal ectopic pacemakers accompanied by a markedly increased propagation velocity. Clinicopathological correlation of ectopic pacemaking was further demonstrated in the mapping of a patient suffering chronic severe nausea and vomiting 6 months after LSG, revealing a persistent unifocal ectopic pacemaker in the distal stomach that generated rapid retrograde-propagating wavefronts, associated with severe gastric dysmotility. Notably, this is also the first HR mapping study to have been performed laparoscopically, apart from validation studies [24], demonstrating a significant step towards minimally invasive translation of this emerging technique.

The findings of this study can be explained by known gastric electrophysiological principles. Gastric ICC networks are arranged as a syncytium with the ability to intrinsically generate slow waves at every point in the network. Coherent propagation is achieved by a process termed “entrainment,” whereby the region of ICC in the network with the highest intrinsic frequencies leads all ICC with lower intrinsic frequencies in a phase-locking manner [25]. The gastric intrinsic frequency gradient ranges from approximately 3 cpm in proximal corpus to 1 cpm in distal antrum in humans [4]. The natural gastric pacemaker site, with highest intrinsic frequency, is normally located at the mid-to-upper corpus on the

greater curvature [5] and is resected during LSG. It might be predicted that another proximal corpus site would automatically resume pacesetter leadership following after LSG at a lower frequency, restoring normal antegrade motility, as occurs after proximal gastric transection [4]. However, such recovery should not be assumed, because the lesser curvature has a lower density of ICC, and moreover, there is likely a second frequency gradient from greater to lesser curvature, with lesser curvature ICC showing lower intrinsic frequencies than at the greater curvature in animal models [26–28]. For example, in classic electrophysiology studies, Kelly and Code performed gastric bisection in the organoaxial direction in six dogs and showed that for several days post-operatively, the lesser curvature half consistently failed to establish a fixed pacemaker site, with irregular and retrograde pacemaking from several temporary ectopic sites and always at a lower rate than the greater curvature half [26]. During this time, the dogs “ate poorly ... and vomited occasionally.”

The current studies were conducted immediately before and after LSG, and the effects of surgical handling, dissection, and stapling may have also contributed to the abnormalities identified in some patients. In particular, surgical dissection or devascularization of tissues can potentially induce loss of slow-wave entrainment, possibly due to prostaglandin release impairing ICC frequency gradients [13, 29]. These observations may explain the acute quiescence observed in half of our patients, and the duration of this quiescent period is currently undefined.

Few previous studies have attempted to investigate the electrophysiological consequences of resecting the normal gastric pacemaker region and/or greater curvature in humans [9, 10, 30]. The studies showed variable results, with some suggesting post-operative impairments but not others, and it is often assumed that slow-wave abnormalities do not significantly contribute to motility abnormalities after gastric surgery [10, 31]. Importantly, these past studies typically applied standard EGG, which has limited sensitivity and specificity and primarily focuses on slow-wave frequency [11]. In our previous work [6, 14], as well as our current study, we show that significant slow-wave abnormalities can occur in humans with stable rhythm at frequencies within the normal range, meaning these abnormalities could be missed by standard EGG. Sparse electrode measurements directly from the organ surface can provide more reliable data [32], but their lack of spatiotemporal resolution also means that major propagation abnormalities could be missed or incorrectly characterized due to signal aliasing [5]. The methods applied here provide more comprehensive and reliable data and could now also be productively used to re-evaluate post-operative gastric dysmotility following other gastric operations including fundoplication, pancreaticoduodenectomy, and gastrojejunostomy.

Additional studies investigating the recovery of slow-wave pacemaking in the days or weeks after LSG are required to further determine the clinical significance our findings. Previous canine studies have suggested that abnormalities in slow-wave initiation may require around 4–14 days to recover [26, 33], although those studies did not specifically involve sleeve gastrectomy. Post-operative monitoring of slow-wave profiles is achievable using implantable wires [34, 35], which could be assisted by wireless technologies to allow patient mobilization [36]. Correlation of electrophysiological data with symptoms will also be necessary to determine if delayed recovery of normal antegrade pacesetting in the sleeve remnant contributes to early post-operative symptoms of food intolerance, nausea, vomiting, and reflux. It is also possible that retrograde motility caused by ectopic pacemaking in some patients could push contents into the fundus post-operatively, inducing distension and increased pressure proximally, which could theoretically contribute to leaks at the proximal staple line junction.

We hypothesize that complete recovery of normal gastric pacesetting may be suboptimal or may never adequately occur in a group of patients after LSG. Proof-of-principle for this idea was established here in a single patient with long-term symptoms of food intolerance, vomiting, and reflux and with uncoordinated peristalsis 6 months after LSG. Further studies are now required to assess the incidence and contribution of aberrant pacemaking to long-term symptoms after sleeve gastrectomy. This case study also serves as an excellent example for the clinicopathological correlation of gastric dysrhythmias, which has been controversial [8], because clinical, imaging,

and electrical pathophysiology findings were in close concordance in this study.

This report indicates the possibility of new therapeutic directions in treating post-LSG symptoms for a patient subgroup, because gastric pacing can be applied to control slow-wave propagation, including post-operatively [35, 37]. Post-operative gastric pacing for dysrhythmias in the context of LSG is likely to be more effective than the pacing attempted for conditions such as gastroparesis, because ICC networks are expected to be intact rather than degraded [38]. Pacing could be theoretically applied either temporarily to enhance recovery or chronically as a rescue therapy via an implant [39]. Another interesting possibility is the recent proof-of-principle demonstration of using ablation therapy, guided by HR mapping, to target and destroy sites of ectopic gastric pacemaking [40]. In the future, the pre-operative use of HR mapping may also help to indicate cases where LSG is unsuitable, due to pre-existing electrical disturbances, as can occur in diabetics [6, 14].

The main limitation of this study was the sample size, which was limited by the technical complexity of the new laparoscopic HR mapping approach. However, the sample size was similar to our other clinical pathophysiological studies investigating gastric dysrhythmia with HR mapping to date [6, 14] and was sufficient to delineate novel aspects of pathophysiology. Promising recent progress in non-invasive methods for accurately mapping gastric dysrhythmias [41, 42], and increasing automation of accurate data analysis [43], offers potential to conduct trials with larger numbers of patients in future. Our patients also did not undergo pre-operative assessment with gastric emptying or manometry, so their baseline motility profiles were unknown.

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Compliance with Ethical Standards Ethical approval for this work was granted by the Health and Disability Ethics Committee, New Zealand.

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Conflict of Interest GOG, PD, NP, TRA, and LKC hold intellectual property in the field of gastric electrophysiology and are shareholders in FlexiMap. RB, TM, and GB declare no conflicts of interest. No commercial or industry financial support was provided for this study.

Ethical Statement All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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