

# Quantification of Velocity Anisotropy During Gastric Electrical Arrhythmia

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**Abstract**—In this study, an automated algorithm was developed to identify the arrhythmic gastric slow wave activity that was recorded using high-resolution mapping technique. The raw signals were processed with a Savitzky-Golay filter, and the slow wave activation times were identified using a threshold-varying method and grouped using a region-growing method. Slow wave amplitudes and velocities were calculated for all cycles. Arrhythmic events were identified when the orientation of a slow wave at an electrode exceeded the 95% confidence interval of the averaged orientation of several normal cycles. A second selection criterion was further developed to identify the arrhythmic events by an anisotropy ratio. In both pig and human studies, arrhythmias were associated with the emergence of circumferential velocity components and higher amplitudes.

## I. INTRODUCTION

There is an omnipresent electrophysiological activity in the stomach, termed gastric slow waves. The periodic occurrence and propagation of gastric slow waves coordinate the motility of the stomach during digestion. The onset of gastric slow wave arrhythmia has been highlighted as a potential contributor to a number of gastric disorders, including gastroparesis, dyspepsia, and gastroesophageal reflux disease [1].

Recent high-resolution (HR) mapping studies of gastric slow waves in canines have revealed complex arrhythmic activation patterns such as premature slow waves, single aberrant slow waves, bursts, regular tachygastric, and irregular tachygastric [2]. HR mapping studies using printed-circuit-board (PCB) electrode arrays have also recently been conducted in pig and human subjects [3], [4], [5]. To date, the location and propagation of arrhythmic episodes from the published studies have generally been visually assessed and entire cycles treated as either normal or arrhythmic [2]. However, visual assessments of arrhythmic activities are time consuming and subjective, and may be inaccurate because a cycle (the slow wave activities belonging to the wave

grouping) with arrhythmia may also contain areas of normal slow wave activity. Therefore, a method of automatically identifying the arrhythmic slow wave activities is required.

## II. RECORDING METHOD

The analysis was performed on previously recorded gastric slow waves from a normal pig, and a human patient with severe gastroparesis who underwent upper gastrointestinal (GI) surgery for a gastric stimulator implantation. The methods of recording were as described previously [4], [5]. Ethical approval for the animal study was granted by the University of Auckland Animal Ethics Committee, and ethical approval for the human study was granted by the University of Mississippi Review Committee. The International Guiding Principles for Biomedical Research Involving Animals and Human Subjects were followed. Briefly, the recording sessions followed a mid-line laparotomy on an anesthetized and intubated subject, exposing the anterior gastric serosal (outside) surface with minimal gastric handling. PCB electrodes (up to 256 electrodes; inter-electrode spacing: 7.62 mm for pig and 4 mm for human) were gently placed on the serosal surface of the stomach and held in place with gauze-packs soaked with warm saline solution, as previously described [3]. No suture was required to hold the electrodes in place. The slow wave signals were acquired using a passive, unipolar Biosemi ActiveTwo system. The acquisition system was connected to a Dell M1450 notebook computer via a fiber-optic cable. The sampling rate was set to 512 Hz.

## III. ARRHYTHMIA ANALYSIS

### A. Activation Times Identification

The slow wave signals were filtered using a Savitzky-Golay filter (Polynomial-order: 9; Window-size: 1.6 s). Slow wave activation times were identified using a Falling-Edge, Variable-Threshold (FEVT) detection method, which calculated the convolution of the signal and a falling-edge kernel, and used a time-varying threshold to detect the times when the convolved signals exceeded the threshold [6]. The identified activation times were partitioned into clusters of individual cycles of slow wave events using an established Region Growing Using Polynomial surface-estimate Stabilization (REGROUPS) method [7]. All of the identified and partitioned activation times were also screened visually to correct any automated errors.

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## B. Velocity Calculation

The velocity field was calculated from the activation times field using a finite-difference scheme with a Gaussian filter to eliminate any high frequency noise that may be amplified through the differentiation process. The velocity vector of each slow wave event was calculated using Eqn. 1.

$$\begin{bmatrix} V_x \\ V_y \end{bmatrix} = \begin{bmatrix} \frac{t_x}{t_x^2 - t_y^2} \\ \frac{t_y}{t_x^2 - t_y^2} \end{bmatrix} \quad (1)$$

where  $V_x$  and  $V_y$  denote the velocity components in the  $x$  and  $y$  coordinates of the electrode array, respectively;  $t_x$  and  $t_y$  denote  $\frac{\partial t}{\partial x}$  and  $\frac{\partial t}{\partial y}$  respectively. The orientation of each velocity vector was calculated using the following expression,

$$\theta = \tan^{-1}\left(\frac{V_x}{V_y}\right) \quad (2)$$

where  $\theta$  is the angle of a normal velocity vector (Fig. 1). The variation of angles of the normal slow waves was quantified by calculating the standard deviation ( $\delta$ ) of the angles of all normal velocity vectors at each electrode for all normal slow wave cycles, which were used as the baseline activity for each study. Here, two key assumptions were made. First, the averaged angle of the normal slow wave velocity vectors ( $\bar{\theta}$ ) at each electrode was assumed to be the orientation of the longitudinal axes, *i.e.*, no circumferential component occurred during normal propagation away from the normal pacemaker region [4], [5]; Second, the circumferential direction was assumed to be perpendicular to the longitudinal direction at each electrode in the recording field.

Each subsequent cycle of slow wave activity was compared to the baseline activity. The difference in angle of each velocity vector in the cycle ( $\theta'$ ), and  $\bar{\theta}$  was compared to a threshold, which was calculated to be  $10^\circ + 95\%$  confidence interval of the standard-error-mean (SEM) of the velocity vectors from the normal cycles (Fig. 1). An activity was deemed arrhythmic if  $|\theta' - \bar{\theta}|$  was greater than or equal to the threshold. The components of the arrhythmic velocity ( $V'_x, V'_y$ ), were then decomposed into the longitudinal component ( $V'_l$ ) and the circumferential component ( $V'_c$ ) of the arrhythmic activity as follows,

$$V'_l = V' \cos(|\theta' - \bar{\theta}|), \quad (3)$$

$$V'_c = V' \sin(|\theta' - \bar{\theta}|) \quad (4)$$

An additional anisotropy ratio (AR)-threshold, *i.e.*,  $\frac{V'_c}{V'_l}$ , could also be used to identify arrhythmic velocity vectors with a relatively large circumferential component. An AR-threshold of 2.1 was used to define a slow wave activity with a dominant  $V'_c$  (as per a previous cardiac study [8]).

## C. Statistical Analysis

Because each arrhythmic activity could be paired with a normal activity, a paired Student's t-test was used to compare the averages of amplitudes and velocities of normal and

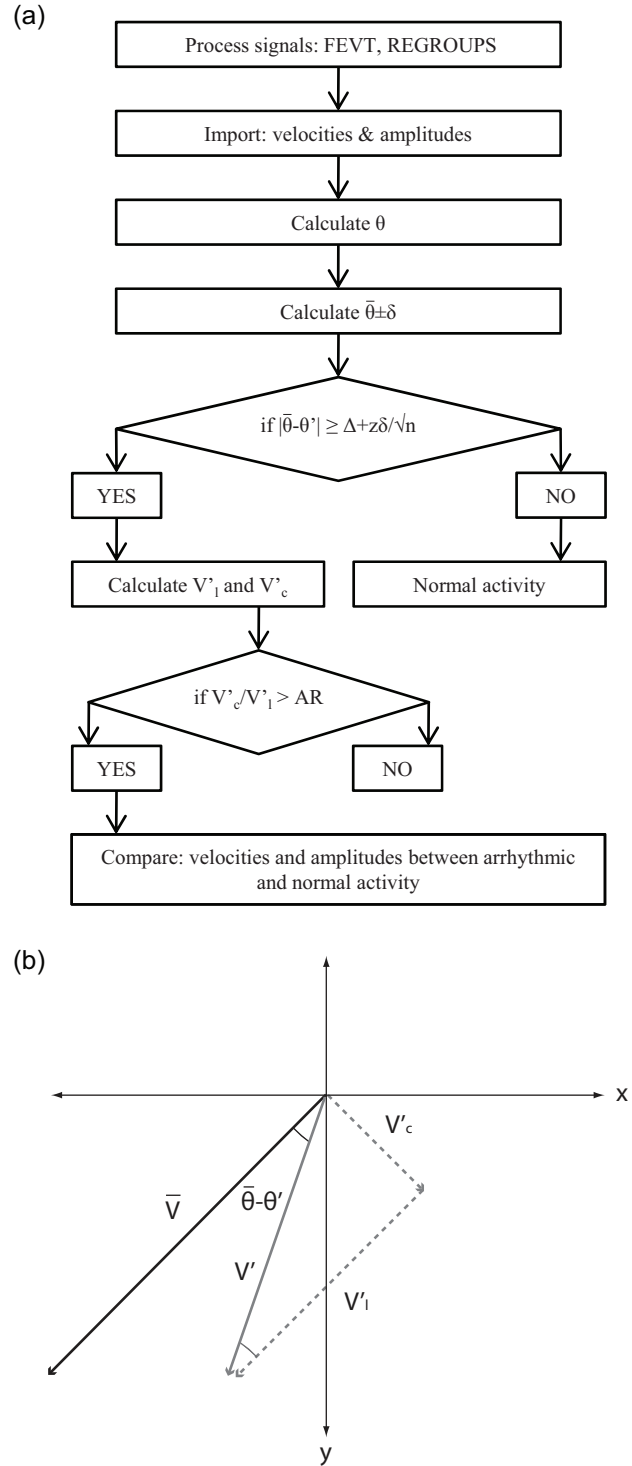


Fig. 1. Arrhythmic slow wave identification. (a) Flowchart of the identification algorithm. The orientation of the velocity was used as the indicator of arrhythmia. The angle of each velocity ( $\theta$ ) and the average angle of the normal activities ( $\bar{\theta}$ ) was calculated. The standard-error-mean was calculated from the standard deviation ( $\delta$ ), number of events ( $n$ ), and the  $z$ -value at 95% confidence interval. The threshold value was calculated by adding an offset value ( $\Delta$ ) to the standard-error-mean. (b) An identified arrhythmic velocity ( $V'$ ) was decomposed into a longitudinal component ( $V'_l$ ) and a circumferential component ( $V'_c$ ), with the longitudinal direction defined by the direction of the normal velocity ( $V$ ).

arrhythmic activity at each electrode for every cycle that contained arrhythmic slow waves. A p-value < 0.05 was deemed statistically significant.

#### IV. RESULTS AND DISCUSSION

##### A. Pig Study

Gastric slow waves were recorded from the distal fundus and proximal corpus of the pig (Fig. 2a). Five cycles of regular antegrade gastric slow waves were used as baseline activity, *i.e.*, all the slow waves in these cycles were deemed as normal slow wave activity. Arrhythmic activities in the form of ectopic slow waves in the corpus were identified in two further cycles. The onset of arrhythmia was spontaneous and followed the five cycles of normal activity. The average of activation times and velocities of the five normal cycles were computed (Fig. 2b) and compared to a cycle with arrhythmic slow waves without the AR-threshold (Fig. 2c) and with the AR-threshold (Fig. 2d).

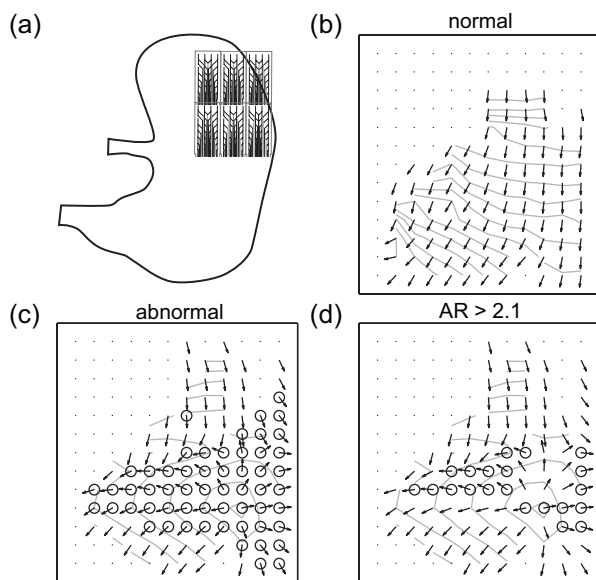


Fig. 2. Pig slow wave recording and identified arrhythmias. The inter-electrode distance is 7.62 mm. (a) The recording arrays were placed on the distal fundus and proximal corpus. (b) Averaged normal slow wave events. The arrows show the velocity field and the lines represent the isochrones at 1 s intervals. (c) Identified arrhythmic events (marked by circle) in a cycle which contained arrhythmia. (d) The arrhythmic events with anisotropy ratio (AR)-threshold  $\geq 2.1$ .

On average, 92 out of 156 electrodes in the normal cycles recorded slow wave events (Fig. 2b). Most of the inactive electrodes were situated over electrically quiescent stomach regions (medial fundus and medial corpus) [4]. For the cycles that contained arrhythmic slow waves, on average, 57 electrodes per cycle were identified as being arrhythmic in terms of the orientation of the velocity vector. A summary of the pig study is presented in Table I.

There was no statistically significant difference between  $\bar{V}_l$  and  $V'_l$ . The average of  $V'_c$  was  $7.40 \text{ mm s}^{-1}$  (no  $V_c$  in the normal activity as stated by the assumption of the normal activity). The average magnitude of  $\bar{V}'$  was higher than  $\bar{V}$ ,

	Wave 1	Wave 2
# of events ID'd (n)	57	57
Amplitude (mV)	0.58 vs 0.81*	0.58 vs 0.78*
$\bar{V}_c$ vs $V'_c$ ( $\text{mm s}^{-1}$ )	0 vs 7.40*	0 vs 7.40*
$\bar{V}_l$ vs $V'_l$ ( $\text{mm s}^{-1}$ )	6.64 vs 5.87	6.64 vs 5.87
$\bar{V}$ vs $\bar{V}'$ ( $\text{mm s}^{-1}$ )	6.64 vs 10.25*	6.64 vs 10.25*

TABLE I

PIG ARRHYTHMIA DATA.\*: P-VALUE < 0.05.

*i.e.*, magnitude of the normal activity, which also equals to  $\bar{V}_l$  ( $10.25$  vs  $6.64 \text{ mm s}^{-1}$ ). The average of the amplitudes of the arrhythmic events was higher than the normal events ( $0.80$  vs  $0.58 \text{ mV}$ , averaged from Table I).

##### B. Human Study

Gastric slow waves were recorded from the human corpus-antrum border (Fig. 3a). Three cycles of gastric slow waves were used as baseline activity. Arrhythmic activities in the form of retrograde propagation in the antrum were identified in three cycles of gastric slow waves. The onset of arrhythmia was spontaneous and followed the three cycles of normal activities. The average activation times and velocities of the three normal cycles were computed (Fig. 3b) and compared to a cycle with arrhythmic slow waves without the AR-threshold (Fig. 3c) and with the AR-threshold (Fig. 3d).

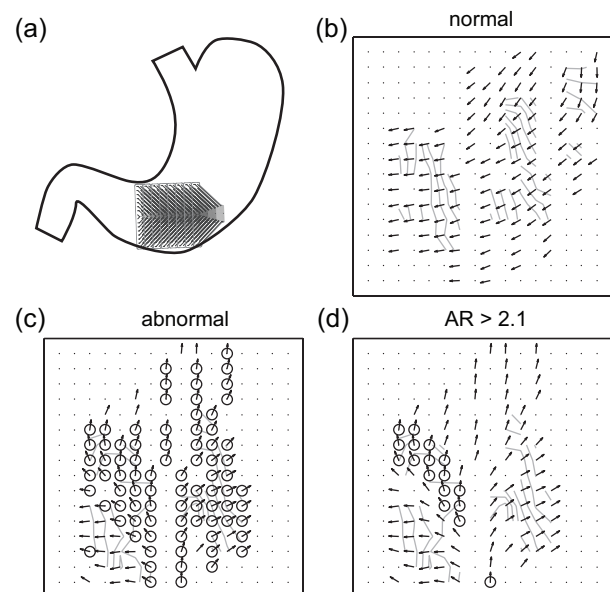


Fig. 3. Human slow wave recording and identified arrhythmias. The inter-electrode distance is 4 mm. (a) The recording arrays were placed on the antrum-corpus border. (b) Averaged normal slow wave events. The arrows represent the velocity field and the lines represent the isochrones at 1 s intervals. (c) Identified arrhythmic events (marked by circle) in a cycle which contained arrhythmia. (d) The arrhythmic events with an anisotropy ratio (AR)-threshold  $\geq 2.1$ .

On average, 121 out of 256 electrodes in the normal cycles recorded slow wave events; the inactive electrodes were mostly situated over the mesentery at the edges of the stomach border (bottom-right corner in Fig. 3a). For the cycles that contained arrhythmic slow waves, on average, 97 electrodes per cycles were identified as being arrhythmic in terms of the orientation of the velocity vector. A summary of the human study is presented in Table II.

	Wave 1	Wave 2	Wave 3
# of events ID'd (n)	102	83	107
Amplitude (mV)	0.25 vs 0.36*	0.27 vs 0.34*	0.24 vs 0.37*
$\bar{V}_c$ vs $\bar{V}'_c$ (mm s <sup>-1</sup> )	0 vs 2.46*	0 vs 2.57*	0 vs 2.59*
$\bar{V}_l$ vs $\bar{V}'_l$ (mm s <sup>-1</sup> )	2.67 vs 3.79*	2.54 vs 2.79*	2.63 vs 3.11*
$\bar{V}$ vs $\bar{V}'$ (mm s <sup>-1</sup> )	2.67 vs 5.11*	2.54 vs 4.21*	2.63 vs 4.57*

TABLE II  
HUMAN ARRHYTHMIA DATA.\*: P-VALUE < 0.05.

All three arrhythmias registered slightly higher  $\bar{V}'_l$  than  $\bar{V}_l$  (3.21 vs 2.63 mm s<sup>-1</sup>, averaged from Table II). The average of  $\bar{V}'_c$  was 2.44 mm s<sup>-1</sup>. The average magnitude of  $\bar{V}'$  was higher than  $\bar{V}$  (4.53 vs 2.63 mm s<sup>-1</sup>). The average of the amplitudes of arrhythmic events was higher than the normal events (0.25 vs 0.35 mV, averaged from Table II).

## V. CONCLUSIONS

An automated method was developed to identify arrhythmic slow wave activity, with validation in a pig and human gastroparetic subject. A set of normal velocity vectors was used as the baseline activity, and the slow wave activity in each subsequent arrhythmic cycle from the same location on the stomach was compared to this baseline activity. The identification method eliminated the need to manually/visually identify arrhythmic activity from the normal activity in the same cycle, increasing the efficiency of identification and allowing an objective analysis. A potential important limitation of this algorithm was that it relied on the accuracy of the velocity calculation method. The physiological significance of the anisotropy ratio threshold requires further validation.

Future studies might also include comparisons of additional waveform characteristics, such as the slope ( $\frac{dV}{dt}$ ) of the slow wave activity, and larger sets of normal and arrhythmic activities should now be evaluated.

The experimental data analyses described here have revealed an interesting and previously unknown finding that circumferential propagation emerges during gastric arrhythmia, and is associated with high amplitude and rapid propagation velocity, which awaits to be validated in a group of animal subjects. The increase in amplitude was also associated with increase in the magnitude of propagation velocity in both animal and human subjects. With the advent of high resolution mapping in patients and the automated identification method introduced here, this initial novel finding can be confirmed and expanded in more studies.

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