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Time Delay Mapping of High-Resolution Gastric Slow Wave Activity

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Abstract

Goal—Analytic monitoring of electrophysiological data has become an essential component of efficient and accurate clinical care. In the gastrointestinal (GI) field, recent advances in high-resolution (HR) mapping are now providing critical information about spatiotemporal profiles of slow wave activity in normal and disease (dysrhythmic) states. The current approach to analyzing GI HR electrophysiology data involves the identification of individual slow wave events in the electrode array, followed by tracking and clustering of events to create a spatiotemporal map. This method is labor and computationally intensive and is not well suited for real-time clinical use or chronic monitoring.

Methods—In this study, an automated novel technique to assess propagation patterns was developed. The method utilized time-delays of the slow wave signals which was computed through cross correlations, to calculate velocity. Validation was performed with both synthetic and human and porcine experimental data.

Results—The slow wave profiles computed via the time delay method compared closely with those computed using the traditional method (speed difference $7.2 \pm 2.6\%$; amplitude difference $8.6 \pm 3.5\%$, and negligible angle difference).

Conclusion—This novel method provides rapid and intuitive analysis and visualization of slow wave activity.

Significance—This techniques will find major applications in the clinical translation of acute and chronic HR electrical mapping for motility disorders, and act as a screening tool for detailed detection and tracking of individual propagating wavefronts, without the need for comprehensive standard event-detection analysis.

Index Terms

cross correlation; gastrointestinal; dysrhythmia; stomach; gastric electrical activity; velocity; multi-electrode

I. Introduction

Recording accurate and timely interpretation of bioelectrical events from the muscles of the human body has become an integral facet of clinical care, including in outpatient, ambulatory, ward, peri-operative and intensive care settings. In recent years, real-time analysis of electrophysiological data in high spatiotemporal resolution has achieved striking clinical success, guiding diagnosis and interventional therapies in cardiac and neural fields [1–4]. Similar advances of high-resolution (HR) bio-electrical monitoring have also emerged in the gastrointestinal (GI) field, to advance the basic understanding of slow waves and to develop diagnostic and therapeutics for functional motility disorders [5–7].

Gastric motility is regulated by an omnipresent bioelectrical activity known as slow waves, which are generated and propagated through the network of interstitial cells of Cajal (ICC) in the smooth musculature [8]. In a healthy human stomach, gastric slow waves originate from a pacemaker region along the greater curvature in the proximal stomach, at a frequency of 3 cycles per minute and propagate towards the antrum [9]. It has been shown that gastric slow wave dysrhythmias are associated with major functional motility disorders such as functional dyspepsia, gastroparesis and chronic unexplained nausea and vomiting [10–13], which may be a consequence of ICC depletion [14]. HR GI electrical mapping provides a detailed description of the patterns of slow wave propagation during normal and abnormal activity, enabling comprehensive descriptions of electrophysiological propagation patterns and accurate classifications of abnormal slow wave activity [15]. Using HR techniques, it has been shown in humans that gastric dysrhythmias often occur at a frequency similar to normal activity [10], however, characteristic velocity and amplitude profiles act as reliable signatures of the occurrence of dysrhythmic events [16].

Spatial velocity and amplitude slow wave profiles therefore offer a key indicator for the classification of normal vs abnormal gastric activity [16, 17]. The current standard method for analyzing gastric HR data first requires identification of all distinct slow wave activation times, by choosing the point of maximal deflection from individual electrodes [18]. This is followed by grouping the events into their associated wavefronts to create a spatiotemporal view of propagation [19]. Such methods were initially manually applied in an offline environment, but have since been combined in an automated pipeline [20]. In addition, near real-time techniques have been developed and implemented using similar methodology [21]. However, the effort involved with finding every slow wave event and assigning it to an associated cycle is computationally-demanding when applied to a large number of electrodes over long recording periods.

In this paper we present a new technique to analyze HR gastric slow waves by calculating the time delay of gastric slow wave propagation from one electrode to neighboring electrodes. This technique was inspired by, and originated from, the cardiac field [22–25]. It

was anticipated that these techniques would be reliable and be substantially more efficient when applied to large data sets, enabling rapid and effective analytics of slow wave propagation characteristics. This improvement is anticipated to facilitate the clinical translation of GI HR mapping techniques and will allow for greater research efficiency in clinicopathological correlation studies and the investigation and development of novel therapies.

II. Materials and Methods

A. Experimental Methods

Ethical approval for the porcine studies was obtained from the University of Auckland Animal Ethics committee. Existing human data was sourced from studies that had been approved by the New Zealand Northern Ethics Committee.

The methods of surgical intervention, care and physiological monitoring were undertaken as described in O'Grady et. al.[9] and Egbuji et. al.[26]. For the porcine studies, anesthesia was administered using Tiletamine HCl 50 mg mL⁻¹ and Zolazepam HCl 50 mg mL⁻¹, and maintained with Isoflurane (2.5–5% with an oxygen flow of 400 mL), after which a mid-line laparotomy was performed to gain access to the gastric serosa [26]. Unlike in humans, weaner pigs frequently demonstrate spontaneous dysrhythmias under these standard experimental conditions [27].

HR flexible printed circuit arrays with an inter-electrode distance spacing of 4–5 mm were placed on the stomach, and were packed with gauze for optimal contact with the recording surface. Unipolar recordings were obtained using an ActiveTwo (BioSemi, Amsterdam) passive recording system with a sampling frequency of 512 Hz, with an effective bandwidth from DC (0 Hz) to 400 Hz. The reference (common mode sense) electrode and the right-leg drive electrode was placed on the right hind leg of the body for pig studies and on the right and left shoulder for humans [9, 26].

B. Technical Methods

Signal filtering was performed according to previously published studies [28, 29]. In brief, the signals were downsampled to 30 Hz, followed by baseline drift removal via a Gaussian moving median filter and high-frequency noise removal via a Savitzky-Golay filter [28].

Velocity fields were then computed by the new automated technique using the time delay of the slow wave events across the signals in the electrode array. The time delay approach was informed by the observation of slow wave propagation sequences, whereby slow wave events are represented as a reliable delay in time across the electrode array. Within a subset of the array, in a short time window, both amplitude and velocity estimates were computed. The time delays were computed via cross correlation of the derivative signals within the short time window with the center as the reference subset signal (Fig. 1). To determine if the time delay estimation was reliable, two constraints were applied. First, the signals were required to have similar morphology and this was quantitatively defined as having a Pearson Cross Correlation (PCC) greater than 0.5. The second was an allowable range of speed,

based on the time delay and known inter-electrode distance spacing. This range of speed was restricted to be less than 15 mm s^{-1} based on previous findings [10, 26].

The time window for time delay computation was dependent on the lowest speed of the slow wave profile (assumed to be 3 mm s^{-1} , based on normal observations [9]) and the longest distance in the electrode array, along with a safety factor of 2 (1).

$$\text{Time Window} = \frac{\text{Longest distance in array}}{\text{Average speed}} \times \text{Safety Factor} \quad (1)$$

This approach ensures at least one slow wave propagating profile is captured. The subsequent shift in the time window was determined based on average slow wave period (set as 20 s, based on normal slow wave activity in pigs and humans [9, 26]). The subset array was a 3x3 grid which was sequentially moved across the entire array to calculate the time delays for velocity estimation (described below) at the central reference subset electrode (Fig. 1).

1) Velocity Mapping—The velocity was calculated using the following linear model (2) at each of the subset reference electrode grids [25] (Fig. 1A).

$$\Delta\tau_{ij} = \alpha(x_i - x_j) + \beta(y_i - y_j) \quad (2)$$

A system of linear equations were formed from the central electrode to surrounding electrodes in the subset grid, where τ_{ij} is the time delay across two signals, α and β are the unknown coefficients of the linear model, and x and y are the coordinate positions of electrodes i and j , from which the time delay was computed.

The speed and angle of the propagation profile was computed based on the coefficients α and β (3, 4), which was solved via an ordinary least squares fit for each central subset grid. To ensure reliable estimation of the velocity profile, at least three of the eight electrode were required to have passed the constraints of time delay computation. If this criteria was not met, the velocity at that location was not calculated.

$$\text{Speed} = \frac{1}{\sqrt{\alpha^2 + \beta^2}} \quad (3)$$

$$\text{Angle} = \tan^{-1} \frac{\beta}{\alpha} \quad (4)$$

2) Amplitude Mapping—At each central subset grid electrode, the maximum and minimum potential values were determined, with their difference providing an estimate of the amplitude within the time window (Fig. 1B). Values beyond a pre-defined threshold of 0.1 to 3 mV were discarded, as they were attributed to non-GI-physiological events or due to artifacts, based on previous data [9, 26].

To minimize experimental noise and unrealistic spatial variations, a Gaussian smoothing filter (G_{filt}) was applied to the velocity and amplitude estimate map (i.e., at each of the short time windows).

$$G_{\text{filt}} = e^{-\frac{Gx^2 + Gy^2}{2\sigma^2}} \quad (5)$$

In (5), Gy and Gx are the size of the filter, with σ the standard deviation. Values $Gx = Gy = 3$ and $\sigma = 0.75$ were chosen as this suppressed noise without eroding the underlying slow wave pattern.

C. Validation

1) Synthetic data—The morphology of synthetic slow wave signals was generated using experimental data via a singular value decomposition, to attain an average waveform (principal component) [29]. These synthetic slow wave morphology events were then used to generate a synthetic high-resolution electrode array signal with three patterns of activation profiles as shown in Fig. 2, using previously used parameters for synthetic data generation [30]. To test the effectiveness of the activation and velocity profile estimation, two sets of simulations were performed with (i) signal noise and (ii) ventilator noise. Signal noise was added to the synthetic signals in terms of normally distributed random Gaussian noise, so that the signal to noise ratio varied from 14 dB to 50 dB in steps of 2 dB. Synthetic ventilator noise was also added to the synthetic signals ranging from 0.1 mV to 1.6 mV (up to ~75% of slow wave event amplitude) in steps of 0.1 mV. Due to the random nature of noise additions, the simulations were performed 20 times at each noise level.

To assess the reliability of the estimating the velocity of propagation profiles, an absolute median difference was computed between the traditional approach and time delay approach. To account for speed variations, a speed percentage error was reported. The speed percentage difference was calculated by dividing the absolute difference of speed between both methods by the maximum value of speed estimate using the traditional method of GI HR analysis. Results are reported as mean $\pm$ standard deviation unless otherwise stated.

2) Experimental data—For validation of the time delay technique, experimental data from three sets of recordings from two patients (duration of 5 to 10 min) and five sets of recordings from four pigs (duration of 2 to 13 min) were used. The experimental data was processed using a standard published approach, via the GEMS software platform [20], to acquire velocity and amplitude estimates to compares to the time delay approach. In brief, for the traditional method, the signals were downsampled and then filtered [29], the slow wave events were detected [18] and grouped into their propagating wavefronts [19].

Previously developed automated techniques were applied, followed by manual correction of event markers as standard practice [16, 17]. Velocity and amplitude fields were then computed from the event markers, via a smoothed finite difference approach and using the derivative of the signal to compute peaks and troughs [29, 30]. The speed, angle and amplitude estimates were compared to the automated estimates at the nearest time and the difference was reported as the mean of the absolute median difference.

D. Results

1) Synthetic data—Fig. 3 shows the speed and angle error with signal and ventilator noise. Overall with signal noise, there was a speed error of $1.0 \pm 1.7\%$ and angle error of $0.05 \pm 0.12^\circ$, while with the ventilator noise there was an overall speed error of $1.2 \pm 0.9\%$ and angle error of $0.04 \pm 0.02^\circ$ (Fig. 3). Ventilator noise had a larger impact on the speed percentage error in comparison to signal noise. In particular, with ventilator noise at 30% of the slow wave amplitude, speed percentage error started to increase (Fig. 3ii). When the slow wave signals had a signal to noise ratio below 30 dB, the accuracy started to reduce for both speed and angle estimates. Velocity angle estimation was not substantially affected by the addition of noise.

An animation was also created to illustrate the estimation of velocity over time, based on the synthetic data shown in Fig. 2 (Supplementary Animation 1). As demonstrated in this animated sequence, the time delay method has the ability to demonstrate changes in velocity variations across the array.

2) Experimental data—Visualization of the vector fields using the time delay estimation resembled accurate representation of the underlying activity (Figs. 4 and 5). In Fig. 4, a normal human gastric slow wave propagation profile is shown, with normal variability of speed shown from the top to the bottom of the array [9].

An abnormal propagation sequence from a porcine experiment is shown in Fig. 5, where regions of both high amplitude and speed are quantified accurately. The smoothing of the amplitude in the short time window provides a more easily interpretable visualization of amplitude difference in comparison to the traditional approach of analysis [20].

Supplementary Animation 2 shows the progression of dysrhythmic slow wave propagation as captured by the time delay approach and the traditional analysis. Due to the variable and dysrhythmic nature of the presented slow wave propagation patterns, automated cycle partitioning of this data set would be difficult, and may require manual segmentation, while the time delay visualization required no manual intervention. However, due to the inherent averaging in the time delay method, individual escape wavefront, would not be captured accurately, as illustrated in Supplementary Animation 2 (video time 7s, and time delay time 88.08s).

Figs. 6 and 7 show the boxplot of error estimation of the slow wave velocity and amplitude profile using the time delay approach quantified using speed percentage difference, angle difference and amplitude percentage difference. For all experimental datasets, there was a low overall speed percentage difference of $7.2 \pm 2.6\%$, insignificant angle difference of $0.3 \pm 0.2^\circ$, and a low amplitude percentage difference of $8.6 \pm 3.5\%$.

For pig data sets, there was also a low overall speed percentage difference of $6.2 \pm 1.9\%$, insignificant angle difference of $0.2 \pm 0.1^\circ$, and a low amplitude percentage difference of $10.5 \pm 2.5\%$. The human data sets resulted in higher error velocity error compared to pig data sets, with an overall speed percentage difference of $8.7 \pm 2.9\%$ and angle difference of $0.4 \pm 0.2^\circ$. However, the amplitude error was lower in comparison to the pig data sets with an amplitude percentage difference of $5.4 \pm 2.3\%$. The variations in error across datasets are due the type of noise present in the experimental environment.

The time delay mapping method described took on average 70 seconds to process 13 min of data, with 256 electrodes, to generate velocity and amplitude estimates. In comparison the traditional approach takes on average 130 seconds to present automated results with up to 3 hours on manual assessment and correction of slow wave event markers (based on 256 electrodes, 13 min recording). These timing measurements were conducted on an Intel Xeon® (CPU: 2.4GHz, 11GB RAM) desktop computer and the automated algorithms were performed 10 times to obtain average results. The main computation of the time delay method was the cross correlation. Speed gains for the time delay method can be achieved via parallel processing and multiple threaded computation, along with the use of an efficient algorithm for cross correlation.

E. Discussion

In this paper we present an alternative novel approach to analysing GI HR electrophysiology mapping that involves the use of time delays of the slow wave events across the electrode array, within a short time window. This approach is computationally less intensive compared to the traditional approach and is able to quantitatively assess key parameters such as velocity and amplitude profiles of slow wave activity in a reliable manner, without manual intervention. Similar methods have been used in cardiac electrophysiology [22–25], but this is the first time this method has been adapted and applied for GI HR slow wave recording analysis, requiring important modifications to account for multiple wavefronts in the mapped field.

GI HR mapping employs a large number of electrodes (typically > 100) to accurately track slow wave propagation profiles [5, 31]. Prior to HR mapping, it was found that dysrhythmic slow wave activity contributed to functional GI motility disorders, but the specific mechanisms and classification of dysrhythmias could not be elucidated due to the low resolution approaches, such as cutaneous electrogastrograms. Recent HR mapping studies [10] have shown that patients with gastroparesis have reduced ICC counts and dysrhythmic slow wave propagation patterns. It has been shown that dysrhythmic gastric slow wave can occur in a stable and unstable manner and the abnormal site of interest can also vary in distance depending on the propagation pattern present (colliding wavefront, conduction block, ectopic initiation) [9, 16, 26]. With such findings, it is becoming clear that HR mapping techniques are required, along with effective and rapid methods of analysis and visualisation for potential diagnostics and treatment strategies in clinical practice. The novel method presented here will provide a critical step toward the clinical translation of HR GI mapping, by resolving this important analysis problem.

One of the potential drawbacks of the proposed method is that it may not be able to distinguish individual aberrant events, such as an ectopic pacemaker embedded within stable propagations, as the information is averaged over a defined time window. Lowering the time window parameters (through the safety factor in Eq 1. and the time window shift) will improve the ability to detect aberrant events. However lowering the time window too much may result in incomplete coverage of the propagation across the area being mapped. It has been pointed out that single aberrant waves within an otherwise normal sequence may have little physiological consequence [32], and such events may occur spontaneously in healthy adults [33, 34]. Regardless, further physiological studies are required to determine if episodic dysrhythmias are of clinical relevance. In addition, error is introduced when calculating the difference between the two approaches of analysis, as the closest wavefront from the traditional approach was compared to the time delay approach map. Nevertheless, in these investigations, the novel method has shown the ability to reliably represent stable and unstable dysrhythmic patterns of propagation with changes in amplitude patterns and velocity variations as seen in Fig. 5 and Supplementary Material 2.

With further development of novel HR GI technologies, such as laparoscopic and wireless mapping, which can allow for long term monitoring [7, 35], novel computational approaches are required to handle large data sets. This method can provide useful and reliable information to the clinicians in real-time, and can be data logged for applications in diagnostic and treatment strategies, paralleling current strategies in clinical cardiac and neural electrophysiology. Also, the novel method can be used as an effective pre-screening tool to identify data segments with unique patterns of interest from longer experimental trials, such as rapid propagation with high or low amplitude, which may require further investigation through the traditional analysis approach of HR mapping [5, 29]. In the cardiac field, treatment of arrhythmias via pacing or ablation is aimed at establishing normal propagation profiles, and similar goals are being approached in GI electrophysiology [35, 36]. With the use of divergence and curl measures from the computed velocity field, regions of pacemaking and dysrhythmic activity can also be detected in real-time [37], which can be vital for developing effective interventional treatment protocols in real time. In the cardiac field, the use of Laplacian mapping has proved to be fruitful in signal analysis [38] and similar applications are planned for body surface GI recordings, as well as serosal and mucosal recordings. Signal processing techniques such as the principle of Laplacian (and the method described here) can be designed into a flexible recording electrodes to acquire cutaneous GI slow wave signals [39].

Further developments will include migrating these methods to seamlessly interact across various data acquisition systems and to work in tandem with previous online systems for HR mapping [21]. In particular, a previous online system employed a kurtosis-based measure for bad channel detection, which can be incorporated into the current algorithm, and may alleviate the need to employ additional constraints to assess whether a time delay is considered reliable.

F. Conclusion

In conclusion, we have presented a new approach to analyze GI HR mapping using the time delay of slow wave event across the electrode array owing to propagation. This method is reliable, intuitive and provides the same high level of information and interpretation as traditional HR mapping analysis. Furthermore, the ability to apply these methods in real-time will facilitate clinical translation of GI HR mapping techniques. In particular, it will accelerate efforts to elucidate the mechanisms underlying gastric arrhythmias and offer future diagnostic and interventional treatment strategies to be conducted real-time in a clinical environment.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Illustration of time delay estimation of the slow wave signals to compute the spatiotemporal velocity profile. (A) shows the schematic of the subset array within the electrode array which is used for velocity and amplitude estimation of slow wave propagation. (B) A reference signal is shown along with its gradient and the gradient of a surrounding slow wave signal. The view of the first 20 s is expanded to illustrate the time lag across the slow wave events, which were calculated using the maximum cross correlation between the gradient of the anchor signal and gradient of the electrodes signals.

Figure 2.

Synthetic HR slow wave propagation patterns and associated signals. Three patterns of slow wave propagation (A) linear wavefront, (B) pacemaker activity and (C) colliding wavefronts were created, and the timing of the wavefronts were assigned with a synthetic slow wave signal in the electrode array. (D), (E) and (F) shows the signals from the center column of the electrode array (shown as white circles on the electrode arrays in the maps), from the patterns of (A), (B) and (C) respectively. The amplitude of the synthetic slow waves events were 2.2 mV. Supplementary Animation 1 shows the results of applying the time delay approach to analyzing the displayed synthetic data.

Figure 3.

Effect of signal and ventilator noise on the estimation of velocity (speed and angle).

Ventilator noise affected the speed estimate to a greater degree than signal noise. With signal noise, the overall speed percentage error was $1.0 \pm 1.7\%$, compared to $1.2 \pm 0.9\%$ with ventilator noise. The angle error was not significantly affected with either noise, resulting in estimates less than 0.05° .

Figure 4.

Comparison of normal human slow wave propagation profile detected using the traditional marked approach of analysis (A and B) and time delay approach (C and D). The velocity and amplitude fields are similar, with high speed localized on the right side of the array and a relatively constant amplitude profile across the array.

Figure 5.

Comparison of dysrhythmic gastric slow wave propagation profile in a pig using the traditionally-marked analysis approach (A and B) and the time delay approach (C and D). Velocity was similar across both methods with the regions of high speed detected on the left side of the array, along with vectors in circumferential directions. Regions of high and low amplitude were also analogous across both methods.

Figure 6.

Boxplot of velocity estimation error in experimental datasets using the time delay approach. (A) shows the speed percentage error while (B) shows the angle error. Overall the pig data sets resulted in a speed percentage error of $6.2 \pm 1.9\%$ and angle error of $0.2 \pm 0.1^\circ$, while the human data set resulted in a speed percentage error of $8.7 \pm 2.9\%$ and angle error of $0.4 \pm 0.2^\circ$.

Figure 7.

Boxplot of amplitude estimation error in experimental datasets using the time window approach. Overall the pig datasets had an error of $10.5 \pm 2.5\%$ and the human dataset had an average error of $5.4 \pm 2.3\%$.