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Magnetoenterography for the Detection of Partial Mesenteric Ischemia

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Abstract

BACKGROUND—Acute mesenteric ischemia represents a life-threatening gastrointestinal condition. A noninvasive diagnostic modality that identifies mesenteric ischemia patients early in the disease process will enable early surgical intervention. Previous studies have identified significant changes in the small bowel electrical slow wave (SW) parameters during intestinal ischemia caused by total occlusion of the superior mesenteric artery (SMA). The purpose of this study was to employ noninvasive biomagnetic techniques to assess functional physiological changes in intestinal slow waves in response to partial mesenteric ischemia.

METHODS—We induced progressive intestinal ischemia in normal porcine subjects (N=10) by slowly increasing the occlusion of the superior mesenteric artery at the following percentages of baseline flow: 50%, 75%, 90% and 100% while simultaneous transabdominal magnetoenterogram (MENG) and serosal electromyogram (EMG) recordings were being obtained.

RESULTS—A statistically significant serosal EMG amplitude decrease was observed at 100% occlusion compared to baseline while no significant change was observed for MENG amplitude at any progressive occlusion levels. MENG recordings showed significant changes in the frequency and percentage of power distributed (PPD) in brady- and normoenteric frequency ranges at 50%, 75%, 90% and 100% vessel occlusions. In serosal EMG recordings, a similar PPD effect was

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AUTHOR CONTRIBUTIONS

Somarajan S: analyzed the data and drafted the manuscript

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observed at 75%, 90% and 100% occlusion levels. Serosal EMG showed a statistically significant increase in tachyenteric PPD at 90% and 100% occlusion. We observed significant increase in tachyenteric PPD only at 100% occlusion level in MENG recordings.

CONCLUSIONS—Ischemic changes in the intestinal slow wave can be detected early and noninvasively even with partial vascular occlusion. Our results suggest that noninvasive MENG may be useful for clinical diagnosis of partial mesenteric ischemia.

Keywords

magnetoenterogram; slow wave; SQUID magnetometer; electromyogram

1. INTRODUCTION

Despite recent technological advances in imaging, acute mesenteric ischemia remains complicated to diagnose. Vascular compromise results in ischemic segments of bowel which can progress to complete necrosis with sepsis and death if not diagnosed early [1-3]. Therefore, early detection and intervention are critical to positive outcomes. Previous studies by Chen et al. reported a low signal-to-noise-ratio in the cutaneous electroenterogram (EENG) recording of the small bowel [4, 5]. Alternating high- and low-conductivity abdominal layers attenuate and distort the extracellular potentials from the GI syncytium, reducing the signal-to-noise ratio of EENG to nearly unrecordable levels in normal subjects and rendering its utility questionable for early detection of intestinal ischemia [6].

Intestinal slow wave magnetic fields are not as affected by abdominal volume conduction as electric potentials [6, 7]. The magnetoenterogram (MENG) enables the characterization of spatial and temporal changes in the intestinal magnetic fields [8-10]. In addition to the signal frequency, MENG can detect signal amplitude at multiple abdominal locations and determine the percentage of slow wave coupling. All of these parameters are known to be altered in disease states such as mesenteric ischemia and motility disorders of the small bowel [11, 12]. Several researchers have investigated the effect of intestinal ischemia on small bowel electrical activity using non-invasive biomagnetic techniques [10, 13-15]. Total occlusion of the superior mesenteric artery (SMA) resulted in a significant decrease in slow wave MENG frequency during ischemia. The effects of ischemia were potentially reversible upon reperfusion, depending on the duration of occlusion [15]. To date, however, no studies have examined the effect of partial occlusion of the SMA lumen on enteric slow waves. We hypothesized that noninvasive MENG could distinguish partially ischemic bowel from normal bowel.

II. PROCEDURE

A. Materials and Methods

All animal studies were reviewed and approved by the Vanderbilt Institutional Animal Care and Use Committee. Serosal EMG and MENG were measured simultaneously in adult pigs (N=10) with a mean body weight of 40.9 ± 1.9 kg (mean $\pm$ standard deviation). We measured serosal EMG using nonmagnetic silver electrode platforms [16] and MENG using the Tristan Model 637 Superconducting QUantum Inteferece Device (SQUID)

magnetometer [8]. The test subjects were anesthetized and laparotomy was performed to place serosal EMG electrodes. Electrode platform containing sixteen silver/silver-chloride electrodes were sutured onto the serosal surface of the small intestine on the proximal jejunum closest to the artery occlusion. Manual occlusion cuff and flow probes were also placed around the proximal superior mesenteric artery to regulate and monitor intestinal blood flow. We then closed the abdominal wound in layers.

To obtain MENG recordings, the animals were placed underneath the Tristan 637 (Tristan Technologies, San Diego, CA, USA) biomagnetometer in a magnetically shielded room (see Figure1). The biomagnetometer consists of a collection of SQUID gradiometers that record data at 29 sensor locations using an array of detection coils, with 10 of the sensors oriented to record magnetic field components tangential to the body surface and 19 orthogonal sensors. The detection coils are located in a plane at the bottom of a dewar filled with liquid helium and are inductively coupled to SQUID sensors that convert magnetic flux threading the detection coils to voltage signals. The voltage data were then amplified (Model 5000, Quantum Design), digitized, and stored on a personal computer. For this study, we utilized only the data from the 19 sensors oriented orthogonal to the patient's abdomen. The detection coils were positioned directly above the sixteen-electrode platform during the experiment. SQUID and electrode signals were time-synchronized during data collection. Simultaneous recordings of electrical potentials and magnetic fields were obtained from the SQUID and the serosal electrodes for a 30 min baseline. At several times during data collection, we suspended respiration by completely stopping mechanical ventilation to allow the comparison of noise reduction techniques [9].

After baseline recording, we examined the effects of occluding blood flow at the following percentages of baseline flow: 50%, 75%, 90% and 100%. Simultaneous MENG and serosal EMG recordings were continued for a period of 30 minutes following 50% occlusion of the artery. This process was repeated at 75%, 90%, and 100% occlusion levels.

B. Data acquisition

Electrode signals were acquired at 256 Hz with an optically-isolated electrode amplifier (ActiveTwo, BioSEMI) and resampled to 30 Hz for analysis. SQUID data were collected with a sampling rate of 3 kHz (imposed by hardware filters on the SQUID amplifiers), and down-sampled to 30 Hz for comparable analysis with biopotential recordings. For all studies, the data acquired from 17 normal-component SQUID sensors were used; two sensors were not in service due to sensor malfunction.

C. Data Analysis

All simultaneously recorded MENG and serosal EMG signals were subjected to spectral analysis. Recorded signals were analyzed using MATLAB (Mathworks, Natick, MA, USA) and filtered using a continuous wavelet transform (CWT) filter with a bandpass of 6 - 120 cycles per minute (cpm). We computed frequency spectra using the Fast Fourier Transform (FFT). For MENG signals, in addition to filtering, we also applied a Second-Order Blind Identification (SOBI) signal processing algorithm to reduce the interfering noise signals [8, 17, 18]. SOBI decomposes the multichannel SQUID data into separate signal and noise

components. We used an automated component selection algorithm to isolate enteric signals (9-18 cpm) using the following criteria: An enteric source component must have a correlation coefficient of at least 0.5 with the best-fit sinusoid at a frequency equal to the dominant frequency of the SOBI source. SOBI components not classified as enteric- corresponding to respiratory, cardiac, motion artifact and other noise sources- were eliminated and the remaining dominant enteric signal components were reconstructed onto the sensor array, giving us a spatial distribution of SOBI-MENG enteric signals. In our notation, SOBI-MENG refers to the filtered MENG signals processed using the SOBI algorithm to identify enteric signal components. We used the central channel of the reconstructed SOBI-MENG array for computations of enteric slow wave parameters. The central channel is chosen because of its presumed proximity to serosal electrodes closest to the artery occlusion, though it contains enteric signal contribution from the entire recording array[10]. The mean frequencies of both serosal EMG and SOBI-MENG components were calculated during baseline and at different occlusion levels for each animal. The results were expressed as mean $\pm$ standard error of the mean (SEM). The unpaired Student's t test was used to compare baseline and ischemic frequencies. Statistical significance was set at $p < 0.05$. We also calculated the percentage of power distributed in the FFT by defining the normal enteric frequency range as ± 2 standard deviations of the mean baseline enteric frequency determined by serosal EMG [9]. Based on this, we defined bradyenteric frequencies as 9-11 cpm, normoenteric between 11 and 15 cpm, and tachyenteric as 15 - 18 cpm.

III. RESULTS

Because of the stepwise slow-wave frequency gradient in the small intestine, the dominant frequency from serosal electrodes closest to the artery occlusion (where we expected maximal ischemia from SMA occlusion) was used as a standard to validate the SOBI-MENG components. A typical set of progressive ischemia data at different occlusion levels (baseline, 50%, 75%, 90%, and 100%) obtained from one of the studies is shown in Figure 2. Figure 2 (a-b) shows data from the reconstructed SOBI-MENG central channel and the corresponding power spectra at the five occlusion levels. CWT-filtered serosal EMG signals with their corresponding FFTs at the five occlusion levels are shown in Figure 2 (c-d). During baseline, the regular enteric slow wave activity is clearly evident in both modalities. The frequency of the slow-wave signals decreased with increasing occlusion levels in both serosal EMG and SOBI-MENG.

Figure 3 shows the mean value of enteric slow-wave frequency computed using SOBI-MENG and filtered serosal EMG components in all ten studies. The SOBI-MENG enteric slow-wave frequency decreased from 12.4 ± 0.3 cpm at baseline to 11.5 ± 0.3 cpm ($p < 0.05$) at 50% occlusion, 10.9 ± 0.2 cpm ($p < 0.001$) at 75% occlusion, 10.5 ± 0.2 cpm ($p < 0.0001$) at 90% occlusion, and 10.3 ± 0.3 cpm ($p < 0.0001$) at 100 % occlusion. The slow wave recorded in serosal electrodes decreased in frequency from 13.0 ± 0.1 cpm at baseline to 12.8 ± 0.2 cpm ($p = 0.54$) at 50% occlusion, 11.9 ± 0.2 cpm ($p < 0.001$) at 75% occlusion, 11.2 ± 0.3 cpm ($p < 0.0001$) at 90 % occlusion, and 11.1 ± 0.3 cpm ($p < 0.0001$) at 100% occlusion.

The percent power distribution in frequency ranges associated with normal slow waves, bradyenteria, and tachyenteria is normally indicative of functional changes in enteric slow waves [19, 20]. MENG PPDs showed increased activity in bradyenteric frequencies at 50% ($p < 0.05$), 75% ($p < 0.01$), 90% ($p < 0.0001$), and 100% ($p < 0.0001$) occlusion levels relative to baseline, as seen in figure 4(a). The normal frequency range contained correspondingly decreased power at 50% ($p < 0.05$), 75% ($p < 0.01$), 90% ($p < 0.0001$), and 100% ($p < 0.0001$) occlusion levels. Statistically significant tachyenteric MENG PPDs were evident only at 100% occlusion level ($p < 0.05$).

In serosal EMG, the PPD in bradyenteric frequencies was significantly increased at 75% ($p < 0.001$), 90% ($p < 0.0001$), and 100% ($p < 0.0001$) occlusion levels relative to the baseline, as illustrated in figure 4b. The normoenteric PPD showed a concomitant decrease at 75% ($p < 0.0001$), 90% ($p < 0.0001$), and 100% ($p < 0.0001$) occlusion levels. We also noted significant change in power in serosal EMG tachyenteric frequencies at 90% ($p < 0.05$) and 100% ($p < 0.01$) occlusion levels.

Neither MENG nor serosal EMG showed significant differences in amplitude at any occlusion level compared to baseline, with the exception of a decrease in serosal EMG amplitude at 100% occlusion ($p < 0.001$), as shown in figure 5b.

IV. DISCUSSIONS

Several researchers have investigated the effect of ischemia on intestinal slow wave activity using invasive and non-invasive methods [12, 13, 21]. Golzarian et al. first reported the feasibility of early detection of intestinal smooth muscle abnormalities resulting from arterial ischemia using non-invasive MENG recordings [22]. These studies showed a decrease in enteric slow-wave frequency associated with intestinal ischemia in the exteriorized small intestine of anesthetized rabbits. Subsequently, Richards et al. demonstrated the clinical usefulness of the MENG for the diagnosis of acute total artery occlusion by detecting a significant drop in the enteric slow wave frequency in rabbit models, noninvasively through the intact abdominal wall [13]. A recent study showed that noninvasive multichannel MENG identified intestinal slow wave dysrhythmias in patients with chronic mesenteric ischemia (CMI) and also tracked the normalization of dysrhythmic activity after revascularization in patients with CMI [10]. Although intensive research has focused on the non-invasive detection of mesenteric ischemia [14, 15], the identification of partial occlusion mesenteric ischemia has not been previously reported. The present study demonstrates the first noninvasive detection of partial mesenteric ischemia that occurs with progressive occlusion of SMA in a swine model.

Ischemia of the intestinal tract directly affects spatiotemporal parameters of the intestinal slow wave [23]. SOBI-MENG is able to identify these parameters which allow the characterization of both ischemic and healthy intestine slow waves simultaneously. Previous studies by Erickson et al. reported that Second Order Blind Identification (SOBI), a blind source separation technique, is capable of isolating enteric slow wave activity from noisy, artifact-contaminated SQUID measurements [8]. To identify partially ischemic bowel, our biomagnetic studies utilized SOBI separated MENG signals. Recent studies show that

analyzing signals using SOBI allows the distinction of normal and pathological states [23, 24]. We have previously evaluated various blind source separation (BSS) algorithms to identify signature slow wave oscillation from MENG, including fast ICA [25] and JADE [26], but to date, SOBI has proved superior to these methods for identification of pathologic intestinal magnetic fields. SOBI's measure of statistical independence relies on diagonalizing time-lagged cross-correlation matrices. Therefore, it tends to separate signals which have an underlying periodic timestructure, such as the sinusoid-like slow wave[17].

We observed a decrease in slow wave frequency with progressive ischemia in both serosal EMG and SOBI-MENG signals. Significant PPD changes occurred in bradyenteric and normoenteric MENG frequencies at all occlusion levels. However, similar PPD effect was observed starting only at 75% occlusion level in serosal recordings. A previous study by Seidel et al. reported the detection of intestinal tachyarrhythmias during small bowel ischemia in rabbit models using serosal EMG recordings [27]. In the present study, serosal EMG showed a statistically significant increase in tachyenteric PPD at 90% and 100% occlusion. Interestingly, we observed changes in tachyenteric signals only at 100% occlusion level in MENG recordings. The change in PPD observed in both serosal EMG and MENG recordings likely reflect tissue uncoupling during ischemia[14].

We observed a decrease in EMG amplitude at 100% occlusion but saw no significant change in MENG amplitude at any occlusion level. Previous serosal electrode studies in our lab showed decrease in the amplitude of small bowel slow waves during early episodes of ischemia from acute SMA occlusion[12, 13]. Prolongation of ischemia eventually causes the slow wave amplitude to fall to undetectable levels[15].

This study investigated the effect of partial occlusion of the SMA lumen on enteric slow waves. With a remote inflatable occluder cuff placed around mesenteric vessels, we induced bowel ischemia at progressive levels of occlusion from baseline to 50% to 75% to 90% and finally to 100% occlusion. We observed that SOBI-MENG reflected ischemic changes reliably and noninvasively even with partial occlusion of the mesenteric artery. The results of this study show that intestinal bioelectric and biomagnetic fields reflect underlying pathophysiology in patients with partial mesenteric ischemia and suggest the possible clinical application of MENG as a non-invasive tool to detect mesenteric ischemia. Future studies will exploit recent upgrades in both our SQUID magnetometer system and our bioelectrode amplifiers and allow us to study enteric signals with higher signal fidelity and spatial resolution. We believe that with advancements in SQUID technology, SOBI-MENG has the potential to be a useful non-invasive screening device for detection of the earliest changes in intestinal electrical activity induced by partial occlusion of the blood supply and thus a potentially life-saving device that allows for early intervention and treatment in patients with mesenteric ischemia.

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Figure 1:

Experimental set up to measure MEG using SQUID magnetometer and serosal EMG with serosal electrodes in a porcine model. The bundle of wires emerging from the bottom of the incision is connected to the EMG amplifier. The SQUID sensor array shown in the inset consists of 19 normal-component (z) sensors and five vector channels (marked with x) that also sample x and y magnetic field components[28].

Figure 2:

A characteristic graph showing (a-b) reconstructed SOBI-MENG components with their corresponding FFTs and (c-d) CWT filtered serosal EMG signals with their corresponding FFTs at different occlusion levels ((baseline, 50%, 75%, 90% and 100%) in a normal porcine subject.

Figure 3:
Mean value of the intestinal slow wave frequency at different occlusion levels (baseline, 50%, 75%, 90% and 100%) for MENG and serosal EMG recordings in porcine subjects.

Figure 4:
Percent power distributed in brady-, normo- and tachyenteric frequency ranges at progressive occlusion levels (baseline, 50%, 75%, 90% and 100%) for (a) MENG recordings and (b) Serosal EMG recordings in porcine subjects.

Figure 5: Signal amplitude at progressive occlusion levels for (a) MENG recordings and (b) serosal EMG recordings. Statistically significant changes were denoted by *.