Abstract / Poster: 3-1 MCG: Basic MCG (1)

activation sequence for timing and location. Activation was first seen in visual cortex - propagating to the temporal lobe region then to inferior frontal gyrus. We have show that the beamformer can provide reproducible results for repeated runs within subjects. Research supported by NIH/NINDS Grant No. R01-NS30914.

2-9-8: MEG Applications for Detecting Dyslexia with Real & Nonsense Word Reading

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The words "dyseidetic" and "dysphonetic" are used to characterize individuals with difficulties in reading via a lexical "whole word" route versus those with a primary difficulty in reading via a sublexical route of grapheme to phoneme translation, respectively. It is unclear to what extent phonological (sublexical) processing may influence lexical reading. We examined the timing and sources of brain activation underlying clinical symptoms of these subtypes of dyslexia. MEG data were acquired from 14 subjects (7 female; 10-40 years of age), 7 with dyslexia (4 dyseidetic and 1 dysphonetic), and 7 normal readers. Visual presentations of four printed letters representing a real word (i.e. "heat") or a nonsense word (i.e. "ateb") were presented. MEG data were collected while each subject read the real and nonsense words aloud. Data were collected at 508 Hz and bandpass filtered 1-30 Hz. Data were analyzed with MR-FOCUSS and Coherence mapping. Normal readers read both real and nonsense words without difficulties. Subjects with dyslexia read real words fluently but had difficulties decoding and synthesizing nonsense words. Inferior frontal areas were found to be more active in both subjects during nonsense words compared to real words consistent with an anterior system involved in word analysis (decoding). Normal readers activate areas in the left angular and supramarginal gyri in both real and nonsense words, whereas subjects with dyslexia rely more on cortical areas in the visual cortex during both tasks suggesting greater reliance on automatic word recognition, but poor assembly of phonology. This study suggests similar, rather than distinct, aberrant systems underlie dysphonetic and dyseidetic subtypes. Accurate models of these neuronal networks may prove useful for the development of future drugs or other treatments. This research was supported by NIH/NINDS Grant R01 NS30914.

2-9-9: MEG study of spectral power in expressive language: potential indicator of lateralisation

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In recent years functional imaging studies investigating multiple aspects of language processing have sparked an interest in applying some of the paradigms to various clinically relevant questions, such as the identification of the cortical regions mediating language function in surgical candidates for refractory epilepsy. Previously we have presented data from a group of adult control participants in order to investigate the potential of using frequency specific spectral power changes in MEG activation patterns to establish lateralisation of language function using expressive language tasks (see Fisher et al 2008). Here we report on a group of adult presurgical patient with refractory epilepsy whose language function was assessed with the same verb generation task. The control group consistently produced left hemisphere decreases in beta-band power accompanied by right hemisphere increases in low beta-band power. However, the majority of the patient group only displayed lateralised decreases in beta-band without the accompanying increase in power in the opposite hemisphere. Further investigation is required to establish concordance with invasive measures but our data suggest that the methods described may serve as a reliable lateralisation marker for clinical assessment. Our findings highlight the potential insight that can be gained from MEG investigations of differential neural mechanisms underlying language function between healthy and pathological brain.

Poster: 3-1 MCG: Basic MCG

3-1-1: Magnetocardiography in non-human primate model of cardiovascular diseases

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Cardiovascular diseases (CVDs) such as ischemic heart disease and heart failure represent the leading cause of death and a major health problem worldwide. Large animal models of CVD that reliably mimic human disease are thus required so that the causative mechanisms can be investigated and novel diagnoses established. The close phylogenetic relationship of cynomolgus monkeys to humans has resulted in their widespread use as a preclinical model of CVD. We
have developed various non-human primate models of CVD, such as heart failure, arrhythmia, valvular disease and congenital disease. Magnetocardiography (MCG) can be used for rapid, non-invasive detection of CVD. The purpose of this study was to use MCG in non-human primate models of CVD. MCG was recorded in non-human primate models of dilated cardiomyopathy and myocardial infarction using a 64-channel MCG system in a shielded room with an animal monitoring system. Cardiac function and structure in this model was evaluated with electrocardiography, echocardiography, radiography, magnetic resonance imaging, blood testing and histological analysis. MCG detected depolarization abnormalities in dilated cardiomyopathy and bigeminy and repolarization abnormalities in myocardial infarction. MCG images reflecting symptoms of dilated cardiomyopathy and myocardial ischemia can thus be successfully obtained in non-human primate models. In this study, systems based on MCG that can evaluate non-human primate models of CVD will lead to novel diagnoses of human conditions and elucidate the electrophysiological mechanisms in CVD.

3-1-2: Hypoxia Magnetocardiogram in Rabbit
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Abstract/BackgroundThe aim of this study was to evaluate how an animal-model magnetocardiogram (MCG) detects a ST-T change during medicine-induced respiratory arrest.Methods and Results MCGs of anesthetized rabbits before and after injection of terfenadine were measured by a 64-channel MCG system and were used to evaluate the ST shift and current distribution. The changes in ST amplitude 2-4 min after terfenadine injection were about twice as large as the changes in the current-arrow angle of a T-wave peak at the same time. Terfenadine did not, however, induce a prolonged QT interval or premature ventricular contractions. ConclusionsOur findings suggest that a ST-T change due to the respiratory-arrest condition of the heart appears early. It could therefore be concluded that the occurrence of an abnormal current distribution in the T wave (instead of the ST segment) reflects ventricular impairment due to hypoxia.
Key words: magnetocardiogram, current-arrow map, hypoxia, respiratory arrest, ST segment

3-1-3: Abnormal MCG(Magnetocardiography) of model mice with a high incidence of myocardial infarction
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Nowadays, varieties of heart-disease-model mice generated by gene modification or by administering drugs are available for studies of disease development or treatment. Many researches on those mice were accomplished using invasive method with anatomy. Since non-invasive method permits continuous observation of the condition of the heart in a particular mouse, the non-invasive method is suitable for studying disease occurrence, progression, and the recovery process with medicine. We have constructed a SQUID system for mice to obtain continuous MCG. In the previous study, we measured the MCG with four mice (NZW/N×BXSB F1 type) with a high incidence of myocardial infarction(MI) and a NZW/N type mouse for control. The magnetic contour map pattern changed abnormally with two of the MI-model mice, one or two weeks before death. These results show the abnormal function of the heart. However, it is necessary to verify whether the change is caused by myocardial infarction. Therefore, we measured the continuous MCG anew on seven NZW/N×BXSB F1 type mice and a NZW/N type mouse for control. And then we investigated the relation between the abnormal contour-map-pattern change and myocardial infarction by the echocardiography, the hemodynamic test and the morbid anatomy on the two of the model mice just after abnormal pattern occurrence in MCG. Abnormality was not found with the result of the echocardiography and the hemodynamic test. Some symptom of the progression of MI, however, was detected. We observed hypertrophy of ventricle walls and infiltration of blood with an MI-model mouse, and hypertrophy of ventricle walls and fibrosis of myocardium with another MI-model mouse. These results suggest the possibility that we can detect an attack of MI at an early stage by using MCG measurement.

3-1-4: Comparison of anesthetic effects on MCG and ECG waveforms of rabbits
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In this work, we used a High-Tc (HTc) YBCO rf SQUID system to record the magnetocardiogram (MCG) signals of conscious and anesthetized rabbits in a magnetically shielding room, and measured the Electrocardiogram (ECG) signals simultaneously. The MCG waveforms changed obviously after the rabbits were anesthetized, while the ECG waveforms did not. We also anesthetized the rabbits with different agents and considerable changes were only observed in MCG waveforms. The results reveal the feasibility for MCG to be used in pharmacological research.
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3-1-5: Quinidine-induced QT prolongation in guinea pig is automatically analyzed by micro-magnetocardiography system: Comparison with ECG

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**Background:** Drug-induced QT prolongation is a critical problem for drug development process. The guideline of International Conference on Harmonization requires to evaluate pre-clinical potential hazards of drug-induced QT prolongation. We have developed a micro-magnetocardiography (MCG) system for small animals comprised of ultrafine magnetometer array consisting of a 3×3 matrix of superconducting quantum interference device (SQUID) on a single silicon chip of 10mm square. We compared QT interval in MCG with that in ECG.

**Methods:** Ten male Hartley guinea pigs (250g) were anesthetized with pentobarbital. Platinum needle electrodes were attached to animals. Excessive quinidine (60mg/kg) was administered intraperitoneally. MCG and ECG were recorded simultaneously at baseline, 3, 5, 7, 10, 15 min after injection. QT interval was automatically analyzed (PowerLab, ADInstruments Pty Ltd) and QTc was calculated by Bazett formula.

**Results:** QTc was significantly prolonged from 286±10 to 299±11 msec in ECG (QTc-ECG) (p<0.001). QTc was prolonged from 283±16msec to 295±11 msec in MCG (QTc-MCG) (p<0.002). QTc-MCG correlated well with QTc-ECG at baseline (r²=0.915, p<0.0001), at 3 min after administration (r²=0.816, p=0.0003), at 5 min (r²=0.772, p=0.0008), at 7 min (r²=0.616, p=0.0072), at 10 min (r²=0.595, p=0.0090) and at 15min after (r²=0.707, p=0.0023). The Bland-Altman plot implied a good intermodality concordance placing 9 or 10 out of 10 guinea pigs between the limits of agreement at each time point of measurement.

**Conclusions:** Micro-MCG successfully measured drug-induced QT prolongation with good correlation with ECG in guinea pig. Non-contact characteristics of MCG may enable high throughput screening test of drug-induced QT prolongation.

3-1-6: Towards a biomagnetic measurement of the human vagus nerve

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The vagus nerve innervates many organs parasympathetically. It is known that heart rate is reduced during vagal stimulation. A decrease in vagal activity can indicate increased mortality risk after acute myocardial infarction. Currently, cardiac vagal activity is measured indirectly through heart rate variability (HRV). A direct measurement of vagal activity seems possible, knowing that evoked fields of peripheral nerves have amplitudes of about 100fT. We hypothesize that the assessment of short-term HRV might lead to the identification of intervals containing vagal nerve signals.

First, the magnetocardiogram (MCG) of two healthy volunteers was measured by the 63-SQUID system of PTB under mild vagal stimulation like hold breath, Valsalva maneuver and controlled breathing. Then, we repeated the experiments with the sensor placed over the neck while the subjects were lying on the left side.

The MCG signals from the neck were sufficient for HRV analysis. The standard HRV parameter pNN60 showed a clear distinction between resting and recovery phases from the activity phase thus indicating vagal changes. Absolute pNN60 values for hold breath and Valsalva maneuver were: 41.18/5.78/34.53 and 45.31/11.34/47.83 (resting/activity/recovery). In controlled breathing, pNN60 values for exhalation were always higher than inhalation values.

From the neck signals, we detected also 10-15Hz components with a distinguished spatial pattern and additional high-frequency components (f > 30Hz) during Valsalva maneuver. While a link of these signals to vagal activity cannot be excluded, we speculate their origin to be brain rhythms and muscle activation due to the stimulation activity. Spectral comparison of muscle reference signals and more elaborate study protocols will help to identify the origin of these components.

3-1-7: Towards an optical multichannel cardio-magnetometer for clinical use

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Over the past years, the Fribourg Atomic Physics Group (FRAP) has developed a laser-pumped room-temperature optical magnetometer capable of detecting cardiac magnetic fields[1]. In a gradiometric array using one active sensor to measure the heart field, about two hours were needed for a full cardiographic map. To shorten the measurement time and increase the sensitivity, FRAP initiated a project to develop an multipole sensor array. While on that road, a compact and scalable Cs magnetometer sensor was designed, based on a 28 mm diameter antirelaxation coated Cs cell