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**AES 2013 Annual Meeting – Online Abstract Supplement
December 6 – December 10, 2013, Washington, DC**

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Results: 1192 stimulation sites were probed in 13 patients (average 92 per patient, standard deviation 39.4).

Afterdischarges: we observed ADs upon stimulation of 9 sites in 4 patients (0.8% of all stimulated sites, 31% of all patients). In all 9 cases, the stimulated sites were part of the seizure onset zone. On one occasion, the AD evolved into a clinical seizure. ADs arose upon stimulation of 3/828 subdural electrodes, 2/89 depth electrodes, and 4/275 stereo-electrodes. They were significantly more frequent upon depth or stereo-electrode stimulation than upon subdural electrode stimulation (Fisher's exact test, $p=0.0277$). There were no differences in the frequency of ADs between depth and stereo-electrodes ($p=0.6372$).

Heart rate: during low-frequency stimulation, the beat-to-beat heart rate was below 50/min on stimulation of 64 sites in 4 patients (5.4%), the majority of which were observed in one patient with a slow baseline heart rate; it never decreased below 40/min. The heart rate was above 100/min on stimulation of 114 sites in 5 patients (9.6%), above 110/min on stimulation of 40 sites in one patient (3.4%), and above 120/min on stimulation of 2 sites in the same patient (0.2%), who had a fast baseline heart rate. It never rose above 130/min.

Conclusions: Recording CCEPs through systematic low-frequency bipolar stimulation of intracranial electrodes is a safe procedure. The overall risk of triggering ADs is low. Triggering ADs is more common upon stimulation of depth and stereo-electrodes than subdural electrodes. It may represent a specific, if insensitive, test for localizing the seizure onset zone. Low-frequency stimulation appears to carry no risk of clinically significant bradycardia or tachycardia.

1.099

MAPPING CORTICAL EXCITABILITY USING SINGLE PULSE ELECTRICAL STIMULATION IN STEREOELECTROENCEPHALOGRAPHIC STUDIES

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Rationale: Intracranial single pulse electrical stimulation (SPES) is an increasingly used method for mapping the epileptogenicity of various brain areas during presurgical evaluation of patients with refractory epilepsy. In combination with stereoelectroencephalography (SEEG), SPES represents a powerful tool for exploring the networks within the epileptic brain in the interictal period, providing valuable information about the epileptogenicity of structures involved in seizure onset and the propagation pathways.

Methods: We have designed a stimulation protocol that consists of a sequence of single pulses having variable amplitude in the range 0 to 5 mA, with 0.25 mA step. Compared to standard constant-amplitude protocols used in other investigations, this protocol allowed us to map not only the propagation of the stimulation through the epileptogenic network, but to also the cortical excitability by analyzing the stimulus-response curves. The pulses (biphasic, 3ms pulse width, 15 seconds interval) were applied in a pseudo-random amplitude sequence, in order to allow decoupling of the time and amplitude factors during subsequent data analysis, using a programmable stimulator (Guideline LP+, FHC Inc, Bowdoin, ME) that allows definition of complex waveforms. We recorded fast responses (100 msec window) to variable amplitude SPES in 7 subjects undergoing presurgical evaluation by means of SEEG method. We used the third quartile value of pooled responses recorded for all stimulated sites as a threshold for calculating the

individual contact activation. For responses on each contact, we calculated a 50% activation threshold based on the stimulus-response curve obtained by fitting the data points using LOWESS (locally weighted scatterplot smoothing) non-parametric regression. All patients were operated and rendered seizure free for a period of at least 6 months. Results were retrospectively analyzed and were correlated with the standard method for establishing seizure onset zone (SOZ) and ictal propagation networks.

Results: The activation current threshold was minimal (0.3 to 0.7 mA) when stimulation was performed within SOZ or adjacent epileptogenic regions in 5 out of 7 patients (71%). The number of activated contacts was maximal when stimulation was applied within epileptogenic areas within 5 of the 7 patients (71%). This shows that automated fast responses analysis and stimulus-response threshold calculation have a good specificity for the localization of the SOZ, however additional information, including presence of delayed responses and stimulation-evoked high-frequency oscillations has to be considered for a more accurate localization.

Conclusions: Evaluation of thresholds from stimulus-response curves can provide valuable complementary information to recording spontaneous activity and responses to standard stimulation protocols for better defining the epileptogenic network, reducing the duration of the invasive monitoring phase.

1.100

CORTICAL EXCITABILITY DIFFERENCE IN POSTICTAL STATE OF EPILEPTIC SEIZURE AND SYNCOPE ASSOCIATED WITH LOSS OF CONSCIOUSNESS

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Rationale: To investigate whether there is difference of cortical excitability change in epileptic seizure and syncopal attacks, we measured cortical excitability by use transcranial magnetic stimulation (TMS) after each event in patients with loss of consciousness (LOC) due to epileptic seizure and syncopal attack.

Methods: We recruited 20 patients with drug-naïve first epileptic seizure and 15 patients with syncope and 20 healthy normal controls were enrolled. Resting motor threshold (RMT), motor evoked potential (MEP), cortical silent period (CSP), intracortical inhibition (ICI), intracortical facilitation (ICF) were measured within 24 hour after LOC event in patient with epileptic seizure and syncope, in 7 day later.

Results: In 20 patients with epileptic seizure, 13 had partial to secondarily generalized seizure, 7 had idiopathic generalized seizure. MEP and CSP decreased more significantly in epileptic seizure group than in patients with syncope group. After 7 day of events, the decreased parameters in epileptic seizure group normalized and the parameter in syncope group unchanged.

Conclusions: Our study showed that cortical excitability transiently decreased after epileptic seizure and unchanged after syncopal attack. these finding suggested the TMS study within 24 hour of loss of consciousness could be useful tool for differentiation between the epileptic seizure and non-epileptic seizure.

1.101

INCREASED CORTICAL SILENT PERIOD IN PATIENTS WITH EXTRATEMPORAL EPILEPSY IN COMPARISON TO HEALTHY CONTROLS

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