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Occipital gamma-oscillations modulated during eye movement tasks: Simultaneous eye tracking and electrocorticography recording in epileptic patients

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ABSTRACT

We determined the spatio-temporal dynamics of cortical gamma-oscillations modulated during eye movement tasks, using simultaneous eye tracking and intracranial electrocorticography (ECoG) recording. Patients with focal epilepsy were instructed to follow a target moving intermittently and unpredictably from one place to another either in an instantaneous or smooth fashion during extraoperative ECoG recording. Target motion elicited augmentation of gamma-oscillations in the lateral, inferior and polar occipital regions in addition to portions of parietal and frontal regions; subsequent voluntary eye movements elicited gamma-augmentation in the medial occipital region. Such occipital gamma-augmentations could not be explained by contaminations of ocular or myogenic artifacts. The degree of gamma-augmentation was generally larger during saccade compared to pursuit trials, while a portion of the polar occipital region showed pursuit-preferential gamma-augmentations. In addition to the aforementioned eye movement task, patients were asked to read a single word popping up on the screen. Gamma-augmentation was elicited in widespread occipital regions following word presentation, while gamma-augmentation in the anterior portion of the medial occipital region was elicited by an involuntary saccade following word presentation rather than word presentation itself. Gamma-augmentation in the lateral, inferior and polar occipital regions can be explained by increased attention to a moving target, whereas gamma-augmentation in the anterior–medial occipital region may be elicited by images in the peripheral field realigned following saccades. In functional studies comparing brain activation between two tasks, eye movement patterns during tasks may need to be considered as confounding factors.

1. Introduction

Intracranial electrocorticography (ECoG) recording is utilized in epilepsy centers, to localize brain areas activated in sensorimotor and cognitive tasks. In general, augmentation of task-related gamma-oscillations on ECoG (50 to 200 Hz) is considered to represent cortical activation elicited by a given task (Crone et al., 2006). A human study with ECoG sampled mostly from the frontal and temporal regions previously demonstrated that a task requiring voluntary saccades elicited sustained gamma-augmentation in the frontal eye field around the onset of eye movement (Lachaux et al., 2006). Studies using scalp EEG and ECoG recordings also demonstrated that ocular and myogenic artifacts derived from eye movements resulted in a transient gamma-augmentation of non-cerebral origin in the anterior temporal regions (Yuval-Greenberg et al., 2008; Kovach et al., 2011). Such artifactual gamma-augmentation was temporarily locked to eye movements occurring 200 to 500 ms following presentation of visual stimulus; the degree of such artifactual gamma-augmentation was most severe in the anterior temporal regions and least severe in the occipital regions (Jerbi et al., 2009; Keren et al., 2010).

It is still uncertain how occipital gamma-oscillations are modulated during an eye movement task. Due to the lack of sampling from the occipital lobe, none of the previous human ECoG studies could determine the spatio-temporal dynamics of eye movement-related gamma-oscillations involving the occipital regions. A recent study using simultaneous eye tracking and scalp EEG recording suggested the presence of event-related potentials originating from the occipital region 100 to 400 ms following the onset of saccade (Dimigen et al., 2009). Previous studies of monkeys using implanted microelectrodes showed that saccadic eye movements were accompanied by increased spiking rates in portions of V1, V2 and V4 neurons and a decrease in

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other portions of V1 neurons (Leopold and Logothetis, 1998; Martinez-Conde et al., 2000). Previous fMRI studies have suggested the presence of eye movement-related brain responses in the occipital lobe in addition to the frontal and parietal eye fields as well as Rolandic cortex (Bodis-Wollner et al., 1999). In these regions, therefore, gamma-augmentations are expected to be elicited by a voluntary eye movement task.

In this study, participants were instructed to follow a target circle intermittently and unpredictably moving from one place to another either in an instantaneous or smooth fashion during extraoperative ECoG recording. We tested the following specific hypotheses: (i) Presentation of target motion would elicit augmentation of cortical gamma-oscillations in lateral occipital sites as well as the parietal and frontal eye fields; (ii) Subsequent eye movement would elicit gamma-augmentation in other occipital sites; (iii) The degree of gamma-augmentation in occipital regions would differ between saccade and pursuit trials. If a difference in gamma-modulations existed between these trials, it would further support the notion that eye movement patterns should be taken into account as confounding factors in brain mapping studies comparing cortical activation between two tasks.

As a secondary analysis, three patients were asked to read a single word popping up on the screen. We tested the following hypothesis: (iv) involuntary saccades following word presentation (rather than word presentation itself) would elicit gamma-augmentation in the anterior portion of the medial occipital region (i.e., in the presumed primary visual cortex for the peripheral vision; Wong and Sharpe, 1999; Yoshor et al., 2007). A behavioral study found that involuntary saccades during attempted fixation are linked to enhanced visibility of peripheral visual targets (Martinez-Conde et al., 2006).

2. Methods

2.1. Patients

The inclusion criteria consisted of: (i) patients with focal epilepsy undergoing extraoperative subdural ECoG recording as a part of presurgical evaluation at Children's Hospital of Michigan or Harper University Hospital, Detroit; (ii) ECoG sampling involving the occipital region; and (iii) measurement of ECoG amplitude modulations driven by the voluntary eye movement task described below. The exclusion criteria consisted of: (i) presence of massive brain malformations; (ii) visual field deficits detected by confrontation; and (iii) history of previous epilepsy surgery. We studied a consecutive series of five patients satisfying both inclusion and exclusion criteria (age range: 13–21 years; 3 females; Table 1). All patients had normal developmental milestones and normal uncorrected visual acuity. None of the patients had a seizure within 2 h prior to the task. The study was approved by the Institutional Review Board at Wayne State University, and written informed consent was obtained from the adult patient and the guardians of the pediatric patients.

2.2. Subdural electrode placement

For ECoG recording, platinum grid electrodes (10 mm intercontact distance, 4 mm diameter; Ad-tech, Racine, WI) were surgically implanted (Fig. S1 on the website). All electrode plates were stitched to adjacent plates and/or the edge of dura mater, to avoid movement of subdural electrodes after placement. In addition, intraoperative pictures were taken with a digital camera before dural closure, to confirm the spatial accuracy of electrode display on the three-dimensional brain surface reconstructed from MRI (Wu et al., 2011).

2.3. Extraoperative video-ECoG recording

Video-ECoG recordings were obtained during the tasks described below, using a 192-channel Nihon Kohden Neurofax 1100A Digital System (Nihon Kohden America Inc, Foothill Ranch, CA, USA). The sampling frequency was set at 1000 Hz with the amplifier band pass at 0.08–300 Hz. The averaged voltage of ECoG signals derived from the fifth and sixth intracranial electrodes of the ECoG amplifier was used as the original reference. ECoG signals were then re-montaged to a common average reference (Wu et al., 2011). Channels contaminated with large interictal epileptiform discharges or visually-apparent artifacts were excluded from the common average reference. No notch filter was used. All antiepileptic medications were discontinued on the day of subdural electrode placement. Electrodes overlying seizure onset zones or MR lesions were excluded from further analysis. Surface electromyography electrodes were placed on the left and right deltoid muscles, and electrooculography electrodes were placed 2.5 cm below and 2.5 cm lateral to the left and right outer canthi.

2.4. Eye movement recording

Eye movements were recorded with an infrared video-based tracking system with a sampling rate of 50 Hz, a spatial resolution of $<0.1°$, and a gaze position accuracy of $<0.5°$ (iView X RED, SensoMotoric Instruments, GmbH). Horizontal and vertical eye positions were integrated into the ECoG Recording System via the analog output card. This procedure allowed us to review ECoG, electrooculography and eye position measures simultaneously (Figs. S2 and S3 on the website).

Events of saccades and eye blinks were marked with the help of BeGaze 2.2 software (SensoMotoric Instruments, GmbH), which has a built-in saccade, fixation, eye blink detector using a dispersion-based algorithm (Smeets and Hooge, 2003). Uni-directional gradual eye movements of a minimum duration of 250 ms, with a direction corresponding to the direction of a target smooth motion, were visually determined as a pursuit. We recognize the following methodological limitations: (i) our eye tracking system is not designed to detect involuntary saccades with amplitudes ranging less than 0.1° (Martinez-Conde et al., 2009) and (ii) the sampling rate of our eye tracking system indicates the existence of an uncertainty of 20 ms regarding the onsets of eye movements.

2.5. Task 1: Voluntary visually-guided eye movement task

All patients completed this task while being awake, unsedated, and comfortably seated on the bed in a dark room. Yet, complete darkness in the room was not feasible. A target circle with a diameter of 5 mm was binocularly presented on a 19-inch Acer V193 LCD monitor placed 60 cm in front of each patient (Refresh rate:75 Hz; Acer America, San

Table 1
Clinical profiles.

Patient	Gender	Age (years)	Hand dominance	Antiepileptic medications	Histology	VCI	PPVT4
1	Male	13	Right	OXC	Tumor in right parietal region	106	128
2	Female	15	Right	LEV	Tumor in left temporal region	N/A	91
3	Female	16	Right	OXC, LEV	Gliosis in left parietal-occipital region	121	91
4	Male	16	Right	OXC, LEV	Gliosis in right temporal region	87	84
5	Female	21	Right	LEV	Tumor in left occipital region	N/A	94

OXC: Oxcarbazepine. LEV: Levetiracetam. VCI: Verbal Comprehension Index. PPVT4: Peabody Picture Vocabulary Test—Fourth Edition.