ELSEVIER

NeuroImage

www.elsevier.com/locate/ynimg
NeuroImage 32 (2006) 790 – 798

Sensory gating of auditory evoked and induced gamma band activity in intracranial recordings

P. Trautner,[a,*] T. Rosburg,[a] T. Dietl,[a] J. Fell,[a] O.A. Korzyukov,[b] M. Kurthen,[a] C. Schaller,[c] C.E. Elger,[a] and N.N. Boutros[b]

[a] *Department of Epileptology, University of Bonn, Sigmund-Freud-Str. 25, D-53115 Bonn, Germany*
[b] *Wayne State University, School of Medicine, Department of Psychiatry and Behavioral Neurosciences, UPC-Jefferson, 2751 E. Jefferson, Detroit MI 48207, USA*
[c] *Department of Neurosurgery, University of Bonn, Sigmund-Freud-Str. 25, D-53115 Bonn, Germany*

Received 15 July 2005; revised 10 March 2006; accepted 3 April 2006
Available online 30 June 2006

Oscillatory activity in the gamma band range (30–50 Hz) and its functional relation to auditory evoked potentials (AEPs) is yet poorly understood. In the current study, we capitalized on the advantage of intracranial recordings and studied gamma band activity (GBA) in an auditory sensory gating experiment. Recordings were obtained from the lateral surface of the temporal lobe in 34 epileptic patients undergoing presurgical evaluation. Two kinds of activity were differentiated: evoked (phase locked) and induced (not phase locked) GBA. In 18 patients, an intracranial P50 was observed. At electrodes with maximal P50, evoked GBA occurred with a similar peak latency as the P50. However, the intensities of P50 and evoked GBA were only modestly correlated, suggesting that the intracranial P50 does not represent a subset of evoked GBA. The peak frequency of the intracranial evoked GBA was on average relatively low (~25 Hz) and is, therefore, probably not equivalent to extracranially recorded GBA which has normally a peak frequency of ~40 Hz. Induced GBA was detected in 10 subjects, nearly exclusively in the region of the superior temporal lobe. The induced GBA was increased after stimulation for several hundred milliseconds and encompassed frequencies up to 200 Hz. Single-trial analysis revealed that induced GBA occurred in relatively short bursts (mostly ⩽100 ms), indicating that the duration of the induced GBA in the averages originates from summation effects. Both types of gamma band activity showed a clear attenuation with stimulus repetition.

Keywords: Gamma band activity; Sensory gating; Auditory evoked potentials; ERP; Electrocorticography

Introduction

Sensory gating refers to the basic ability of the brain to suppress the response to repeated (and possibly irrelevant) environmental stimuli (Venables, 1964). The typical experimental setup for electrophysiological studies of sensory gating in the auditory domain applies pairs of clicks, separated by a short interstimulus interval (ISI, 0.5 s) and a long interpair interval (8–12 s) (Adler et al., 1982). Healthy control subjects have on average reduced P50 amplitudes as response to the second stimulus, as compared to the P50 elicited by the first stimulus. This amplitude reduction or suppression is regarded as an indicator of sensory gating (Nagamoto et al., 1991).

Most studies on sensory gating are restricted to the analysis of the P50, although there were some studies investigating also the suppression of later auditory evoked potential (AEP) components (Boutros et al., 2004) and the suppression of the evoked gamma band activity (GBA) (Clementz et al., 1997). In neuromagnetic recordings, such an evoked (or phase-synchronized) GBA with a maximum at 40 Hz was reported to occur in a latency range of 30–100 ms (Pantev et al., 1991). GBA is of particular interest in neuroscience research as it has been linked to a variety of perceptual and cognitive functions such as feature binding, object representation or selective attention (Bertrand and Tallon-Baudry, 2000; Engel and Singer, 2001; Fell et al., 2003; Keil et al., 2001; Tiitinen et al., 1993; Varela et al., 2001). Alterations in GBA have also been related to pathological processes (Lee et al., 2003; Llinas et al., 1999).

The P50 and the evoked GBA are difficult to separate because they overlap in the time and frequency domain. It has even been proposed that P50 and evoked GBA represent the same phenomenon (Basar et al., 1987; Clementz et al., 1997). The AEP is typically filtered from 10 to 50 Hz for the study of the P50 and from 30 to 50 Hz for the study of the evoked GBA. Furthermore, the study of GBA and P50 in surface recordings is handicapped by

* Corresponding author.
E-mail address: p.trautner@web.de (P. Trautner).
Available online on ScienceDirect (www.sciencedirect.com).

1053-8119/$ - see front matter © 2006 Elsevier Inc. All rights reserved.
doi:10.1016/j.neuroimage.2006.04.203

small signal amplitudes and possible contamination with high-frequency muscle artefacts. Intracranial recordings avoid an attenuation of the EEG signal by intervening tissues between the cortical generators and surface electrodes particularly in the higher frequency range (Pfurtscheller and Cooper, 1975) and might, therefore, be regarded as ideally suited for the investigation of GBA. However, the opportunity for intracranial recordings in humans is rare and restricted to presurgical evaluation of epilepsy and tumor patients.

In a study on epilepsy patients, GBA elicited by visual stimuli was recorded intracranially while patients performed a Kanizsa task (Lachaux et al., 2000). Interestingly, besides evoked GBA, induced (not phase-synchronized) GBA was observed, occurring in a time range from 200 to 500 ms. Similarly, both kinds of GBA were also recorded from the temporal lobe after auditory stimulation with tones and phonemes (Crone et al., 2001). In this study, power changes in GBA were analyzed in recordings from 4 subjects with tones and phonemes as stimuli. As a major finding, induced GBA was assumed to be confined not only to the well-known GBA at about 40 Hz but also comprised activity of considerably higher frequencies (80–100 Hz). The induced GBA started at about the same time as the invasively recorded N100 but outlasted this component. Topographically, it was found adjacent to electrode sites with a prominent N100 peak, but its topographical distribution was not identical to that of the N100. Induced GBA after acoustic stimulation was also observed in monkeys (Brosch et al., 2002).

The current investigation was conducted within the framework of a larger study on a functional neuroanatomical model of sensory gating. In general, this study targets to describe neuronal correlates of sensory gating by intracranial recordings in humans. Here, we investigated the occurrence of evoked and induced GBA in a typical sensory gating experiment with paired clicks. The first aim of the study was to examine whether evoked GBA can be observed at intracranial leads and compare such an evoked GBA to characteristics of P50. The second aim was to prove whether induced GBA is elicited also by the very short stimuli with a duration of a few milliseconds only, as usually applied in sensory gating experiments. Finally, the effect of stimulus repetition was assessed for evoked and induced GBA as well as for the AEP components P50 and N100. For these purposes, brain signals were analyzed in the time–frequency domain by wavelet transform.

Experimental design and methods

Subjects

Patients with medically refractory epilepsy who were implanted with electrodes for presurgical evaluation participated. The exact placement of electrodes always depended on clinical considerations and considerably varied between patients. Overall 47 patients took part in our recordings between January 2003 and June 2004, with 36 patients having electrodes placed in the region of interest (lateral surface of the temporal lobe). Two data sets had to be excluded from analysis owing to technical reasons, leaving a final study sample of 34 patients (17 male, mean age 36 years, range 19–57 years). Subjects gave written informed consent. The study was approved by the local ethics committee of the University of Bonn.

Data recording

The EEG was recorded with the digital EPAS system (Schwarzer, Munich, Germany) and its implemented Harmonie EEG software (Stellate, Quebec, Canada). The EEG was measured against a reference of left and right mastoid electrodes with a sampling rate of 1000 Hz. Electrode positions were determined by MRI recordings routinely acquired after implantation.

Patients were seated on a comfortable chair in a quiet room illuminated by bright light. During the experiment, the subjects were stimulated with repetitive acoustic stimuli by headphones. The stimuli used were short tone bursts of a single sine wave with 1500-Hz frequency and a duration of 6.6 ms (including rise and fall times of 1.5 ms). A set of 100 pairs of stimuli were administered with an ISI of 0.5 s and an interpair interval of 8 s.

Data analysis

The EEG was segmented into single trials with a total duration of 2000 ms. An interval of 500 ms prior to the first stimulus was included into each trial for baseline correction. Recordings were inspected thoroughly for epileptic activity, and contaminated segments were rejected (on average 10% of the trials). After averaging, all electrode contacts were explored for AEPs. For inspection of the AEPs, band-pass filters were applied (1–20 Hz, 12 dB/oct for the N100 and 10–50 Hz, 12 dB/oct for the P50). The N100 was expected to peak within a latency interval from 70–130 ms and the P50 from 35–75 ms. A possible P50 was only analyzed if an N100 was observed. Both components (P50, N100) were measured with respect to baseline. If a potential was found at more than one electrode, the electrode with maximal potentials was selected. These electrodes were tested for the occurrence of evoked GBA.

As there was no presumption about the exact location of induced GBA, all electrodes were screened by the 'frequency extraction' procedure of the Brain Vision Analyzer software (Brain Products, Munich, Germany). This tool is easy applicable and allows to assess possible contributions of frequency bands in the time domain. For the current purpose, power values were extracted for the frequency band from 60–160 Hz and subsequently averaged over trials. If visual inspection supported the assumption of systematic high frequency activity at an electrode, data were more thoroughly analyzed by means of continuous wavelet transform (Torrence and Compo, 1998). This transform characterizes the brain activity by its dispersion in the time and frequency domain. Within this transform, data were also tested statistically for significance. Morlet wavelets (6 cycles, 102 scales) were used for data transformation. In such a procedure, the frequency content is obtained for each time point, stored in a complex number with real part ($\mathcal{R}(t,f)$, cosine components) and imaginary part ($\mathcal{I}(t,f)$, sine components). From the transformed single trials, two different representations of the data can be obtained by averaging over the trials using either the unchanged wavelet coefficients with preserved phase information or the power values $\left(\mathcal{I}(t,f)^2+\mathcal{R}(t,f)^2\right)$ at each time point without any phase information. If phase information is preserved, signals will increase with their phase locking, while signals which are not phase locked will cancel out each other. Such a transform