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40-Hz steady state response in Alzheimer's disease and mild cognitive impairment

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Abstract

The 40-Hz steady state response (SSR) reflects early sensory processing and can be measured with electroencephalography (EEG). The current study compared the 40-Hz SSR in groups consisting of mild Alzheimer's disease patients (AD) (n=15), subjects with mild cognitive impairment (MCI) (n=20) and healthy elderly control subjects (n=20). All participants were naïve for psychoactive drugs. Auditory click trains at a frequency of 40-Hz evoked the 40-Hz SSR. To evaluate test–retest reliability (TRR), subjects underwent a similar assessment 1 week after the first. The results showed a high TRR and a significant increase of 40-Hz SSR power in the AD group compared to MCI and controls. Furthermore a moderate correlation between 40-Hz SSR power and cognitive performance as measured by ADAS-cog was shown.

The results suggest that 40-Hz SSR might be an interesting candidate marker of disease progression.

Introduction

Cortical neural activity is reflected in the electroencephalogram (EEG). Fast neural oscillations measured with EEG and magnetoencephalography (MEG) have been proposed to be an important mechanism in the integration and binding of neural networks in perceptual and cognitive processes (Herrmann et al., 2004, Joliot et al., 1994, Tallon-Baudry and Bertrand, 1999).

A basic method for measuring these fast oscillations is by evoking a 40-Hz steady state response (SSR). The 40-Hz SSR is elicited by auditory stimulation with “click trains” at 40-Hz.

These very short individual clicks each evoke an event-related response (ERP). The short inter-stimulus intervals in the click trains do not allow the ERP's to return to baseline, resulting in a nearly sinusoidal SSR (Tallon-Baudry and Bertrand, 1999). As a result, the 40-Hz SSR can be considered as a superimposition of middle-latency responses (P50) to each individual click (Galambos et al., 1981). Early gamma band

responses such as the 40-Hz SSR are involved in the sensory processes that precede perceptual or attentional processes (Karakas and Basar, 1998).

Impaired sensory processing is one of the hallmarks of Alzheimer's disease (AD).

A study using magnetoencephalography showed that the 40-Hz SSR in a drug naïve AD population is enhanced compared to that in healthy controls (Osipova et al., 2006). Osipova et al. related their findings to decreased cortical inhibition in AD. This explanation is substantiated by results from studies using a sensory gating or dual-click paradigm. This paradigm involves the presentation of two consecutive clicks, whereby the P50 amplitude to the second click should be lower. This paradigm is considered a measure for cortical inhibition. Several studies have shown that subjects with AD diverged from the paradigm and exhibited increased P50 amplitude to the second click in AD (Cancelli et al., 2006, Jessen et al., 2001). A study that examined healthy volunteers with a family history of AD also showed a similar divergence in the P50 amplitude (Boutros et al., 1995). This suggests that impaired cortical inhibition may be present very early in the course of the disease. With a view to detecting the disease at an early stage, it is important to identify whether the difference in the 40-Hz rhythm is already present in a well-defined MCI population.

The current study aims to examine whether differences, similar to the findings of Osipova et al. (2006) are revealed when EEG is used to study AD patients. Furthermore, it sets out to discover whether these differences are already present in subjects with MCI. The current study reports data from a psychoactive drug naïve population. This is an important methodological issue since several studies showed that pharmacological modulations have a profound effect on the power of the 40-Hz rhythm (Ahveninen et al., 1999, Ahveninen et al., 2002).

To evaluate the robustness and reproducibility of the results, the test–retest reliability (TRR) of the 40-Hz SSR paradigm will be evaluated.

We hypothesized that the 40-Hz SSR is enhanced in patients with AD compared to subjects with MCI and healthy controls. Furthermore, it is to be expected that the SSR in the MCI group will be higher than in the control group, but lower than in the AD group.

Section snippets

Subjects

The study included three different groups of subjects:

(1) Fifteen psychoactive drug naïve patients with a diagnosis of probable AD according to the NINCDS-ADRDA criteria were included (McKhann et al., 1984).…

(2) Twenty psychoactive drug naïve patients with a diagnosis of MCI according to the Petersen criteria were included (Petersen et al., 2001). MCI subjects also received standard blood workup, neuro-imaging and neuropsychological testing. The diagnosis AD or MCI was made at a weekly consensus meeting …

…

Subjects' demographics

Fifteen AD patients were included. They had a mean age of 75.2 (S.D. 6.9) and a mean MMSE of 20.8 (S.D. 2.7, range 17–26). Eleven of the AD patients were male.

Twenty MCI patients were included. They had a mean age of 70.6 (S.D. 7.2) and a mean MMSE of 26.3 (S.D. 1.6, range 23–29). Twelve of the MCI patients were male.

Twenty healthy control subjects were included. They had a mean age of 69.5 (S.D. 6.1) and a mean MMSE of 29.3 (S.D. 0.8, range 28–30). Twelve of the healthy control subjects were…

Discussion

The current study showed almost a universally high TRR and a significant difference in 40-Hz SSR power between the patient groups. The correlations between the sessions ranged from 0.68 to 0.83, which is high. Only the Pz electrode in the control group showed a moderate correlation (i.e. 0.54). These high correlations suggest that the 40-Hz SSR can reliably be assessed in a cognitively impaired population.

40-HzHz SSR power was higher the AD group compared to MCI and control groups.…

Conflict of interest

All authors state that there were no conflicts of interest involved in this study.…

Acknowledgements

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> ...The uniqueness of gamma frequency band activity in that sense could be explained by an inhibitory interneuron impairment in ADD patients with a subsequent increase in gamma activity (Verret et al., 2012; Palop and Mucke, 2016). Decreased GABAergic inhibition was demonstrated in a mice model of ADD (Busche et al., 2008), and suggested as related to increased gamma responses in ADD patients (Stam et al., 2006; Rossini et al., 2006; Osipova et al., 2006; Van Deursen et al., 2008, 2011; Başar et al., 2016a, 2017). During the cognitive tasks, ADD patients respond with a 25% larger gamma response and a delay about 100 ms later in the higher frequency gamma subband (40–48 Hz) (Başar et al., 2016a) without obvious fluctuations (Başar et al., 2016a; Deiber et al., 2010)....

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