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Mismatch negativity is abnormal but not lateralizing in temporal lobe epilepsy

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ABSTRACT

We investigated the changes of mismatch negativity (MMN) in patients with temporal lobe epilepsy (TLE) and explored the possible role of MMN in lateralizing their seizure focus. Thirty patients with TLE and thirty healthy controls were included. MMN was elicited in each subject. Patients with TLE were divided into three subgroups: unilateral left TLE; unilateral right TLE, and bilateral TLE. MMN amplitudes and latencies were compared between the patients with TLE and the control group, and also among the three subgroups of TLE, using repeated measures analyses of variance (ANOVA). To assess the lateralizing value of MMN, MMN latencies and amplitudes at the mastoid sites between the ipsilateral and contralateral sides of epileptic focus in patients with unilateral TLE were compared using *t*-test. Compared with controls, each subgroup of patients with TLE had longer latencies of MMN at both fronto-central and mastoid sites, but the amplitudes of MMN were not significantly different. The amplitudes and latencies of MMN were not significantly different between the ipsilateral and contralateral sides of seizure focus at mastoid sites. The present findings of prolonged latencies of MMN are suggestive of cognitive impairment in TLE. Both the mastoid sites and the fronto-central sites are involved, which likely reflect widespread cortical abnormalities in TLE. However, the changes of MMN during the interictal phase are not useful for lateralizing the seizure focus in patients with TLE.

1. Introduction

Cognitive dysfunction is one of the most common comorbidities of epilepsy, which may even exist prior to the seizure onset [1–3]. As the most common focal epileptic syndrome in adults, temporal lobe epilepsy (TLE) is associated with a wide range of cognitive impairments of various severity, depending on the specific syndrome and its associated underlying characteristics [4,5]. Due to the involvement of mesial temporal structures or the limbic system, memory decline is considered as the most significant cognitive deficit in TLE [2,6].

Mismatch negativity (MMN) is an early auditory event-related potential (ERP) that is the result of an automatic memory-based comparison process that detects a discrepancy between the neural representation of the regularity of recent stimulation and the representation of the deviant sound. MMN is believed to reflect the earliest cortical event in the cognitive process of auditory information and to be a part of auditory pre-attentive memory [7].

The major MMN generators are considered to be located in the superior temporal cortex near the primary auditory cortex [8], and the frontal cortex is the additional generator [9]. Therefore, MMN has been extensively applied in neurological disorders affecting the temporal lobe, including TLE [10–12]. In TLE studies, MMN is often recorded both at fronto-central sites and mastoid sites. Many studies have indicated that the frontally-recorded MMN is composed of contributions from both the auditory and frontal cortices, whereas the mastoid 'MMN' receives a contribution from the auditory-cortex MMN generator only [13]. However, we find that the existing literature evaluating MMN response in patients with TLE is limited and discrepant. Delayed latencies of MMN [14,15] and its magnetic equivalent [16] were observed in patients with intractable epilepsy compared to healthy controls. However, studies reported differences in the amplitude changes at the fronto-central and the mastoid sites.

Miyajima et al. reported an increase of MMN amplitude at fronto-central sites in patients with TLE, but no significant changes at mastoid sites [14]. On the other hand, Hara et al. found that the MMN was diminished at mastoid sites but not at the frontal-central sites [15]. In Lin's study, the amplitudes had no significant changes at all sites [16]. Given that the source of MMN is closer to the presumed epileptogenic zone in TLE, we hypothesized that MMN at the mastoid sites would be more likely to be abnormal. TLE has been well acknowledged as a network disease involving different structures in the temporal lobe and also in regions remote from the epileptogenic zone [17]. Therefore,

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this study aimed to investigate the MMN in patients with TLE, and also to explore whether the MMN at the fronto-central sites is affected.

Mismatch negativity is often elicited at bilateral mastoid sites, which are considered to reflect temporal lobe activity only. Previous research analyzed both patients with left and right temporal lobe epilepsy together [14,16]. This might preclude the possibility of assessing the lateralizing value of MMN. It is therefore of interest to explore whether the alteration of MMN, especially the mastoid mismatch component, has a possible role of lateralizing the seizure focus in TLE.

Therefore, the current study investigated the changes of MMN at both fronto-central sites and bilateral mastoid sites and explored whether the alteration of MMN at the mastoid sites has a possible role of lateralizing the seizure focus in TLE patients.

2. Methods

2.1. Subjects

Consecutive patients with TLE were recruited from the epilepsy monitoring unit, West China Hospital, Sichuan University from 2014 to 2016. Age- and sex-matched healthy controls were also recruited during the same period.

The inclusion criteria of the TLE patients were: 1. Ictal semiology strongly suggests a temporal origin, including epigastric rising sensation, experiential phenomena (most commonly fear) and gustatory or olfactory sensations, decreased responsiveness, oro-alimentary and/or motor automatisms [18]. 2. All TLE patients underwent long-term

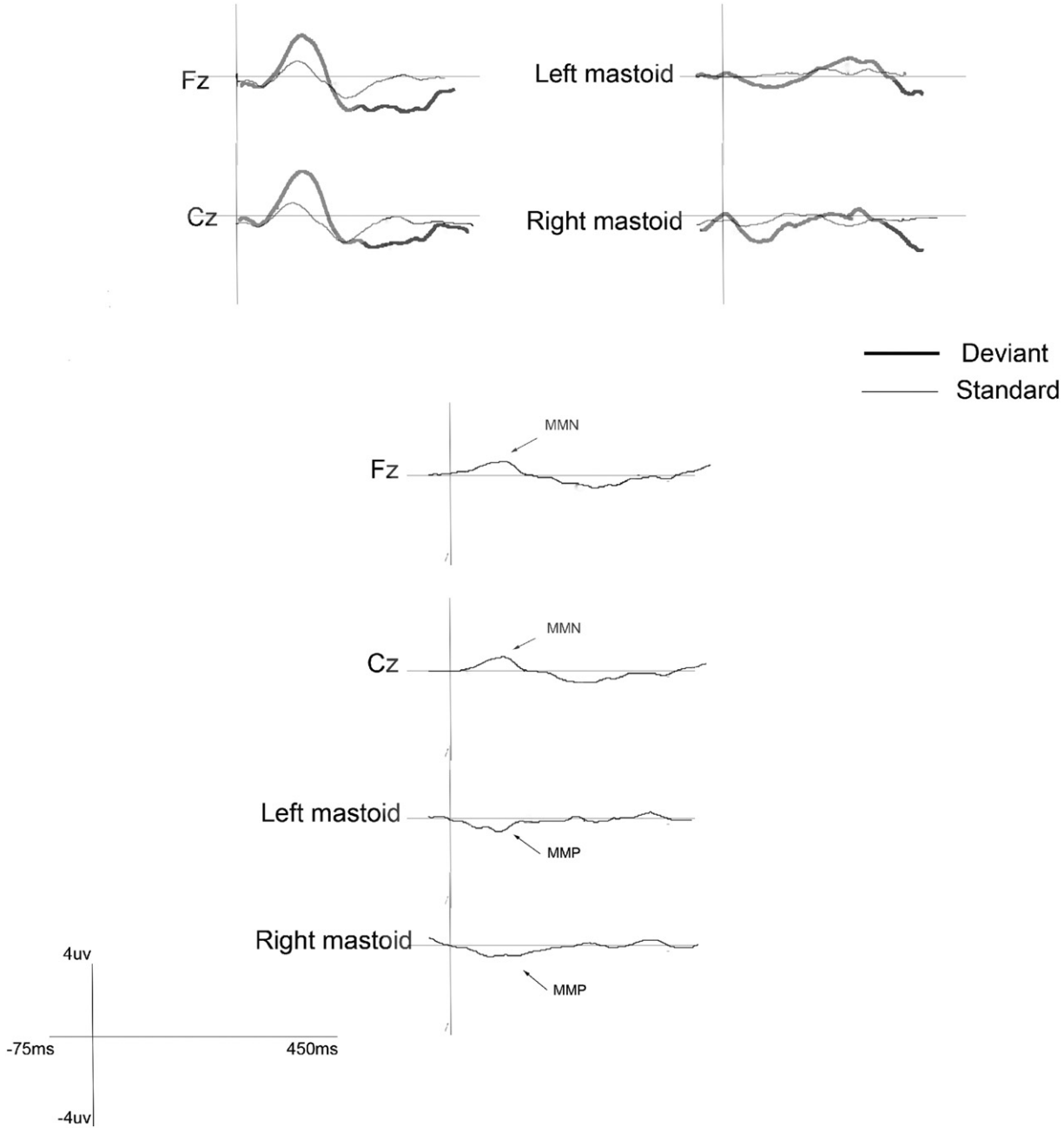

Fig. 1. Grand-averaged MMN/MMP elicited by deviant stimuli (probability = 0.20) and standard stimuli (probability = 0.80) in the auditory oddball paradigm MMN: Mismatch negativity at fronto-central sites MMP: Mismatch negativity at mastoid locations.