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Experimental study

Impact of putamen stroke on task context updating: Evidence from P300 brain waves

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ABSTRACT

According to the context updating theory, the oddball P300 component indexes brain activities underlying revision of the mental representation induced by incoming stimuli. It involves an attention-driven comparison process that evaluates the representation of the previous event in working memory. Delayed latencies have been reported for various types of stroke such as unilateral thalamic stroke. We investigated memory updating effects in patients with putamen stroke. Two groups of patients with putamen and thalamic stroke were recruited along with two control groups of young and old healthy participants. Auditory and visual P300 were elicited respectively in a two-stimulus oddball paradigm. The auditory P300 peak latencies were significantly longer in patients with a putamen lesion than in the aged and young control groups and the same pattern was found in the thalamus-lesioned patient. The delayed auditory P300 component in both patient groups but neither control group suggests impairment of memory updating in patients with putamen stroke comparable with thalamic stroke. Our study illustrates the important role of subcortical structures subserved in context updating.

1. Introduction

Discrimination or categorization of new and old events is a highly evolved human ability that develops in early infancy [12]. Updating the current state of the environmental context is critical to survival. The oddball P300 wave is widely used to assess stroke patients' cognitive functions. It is thought to depend on the processing of the stimulus context and levels of attention and arousal [37]. The P300 recorded using the classic oddball paradigm is concerned with volitional target detection. After initial sensory processing, an attention-driven comparison process evaluates the representation of the previous event in working memory [24]. Stimulus item that differs from representation maintained in memory from previous exposures, such as in a working memory, tend to produce larger P300 components than items previously encountered [9,29]. Previous studies demonstrated that the P300 indexes update activity in corticolimbic circuits during attention and working memory [8,40].

P300 is known to be abnormal in various diseases and in patients with focal brain lesions [36]. For example, its latency is prolonged in demented patients, regardless of the etiology of dementia [32,13]. Severe endogenous depression seems to reduce P300 amplitude and slightly increase its latency [35]. In demented or elderly persons, abnormalities of P300 have been shown to be related in particular to cognitive deficits such as defective short-term memory and perceptual processing speed, as well as decreased alertness and attention [32,48]. Changes in P300 latency and amplitude might reflect the integrity of various cortical/subcortical systems linked to the psychological processes responsible for P300 generation [21,15].

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The locus of P300 generation has been explored in many studies [43,44,39], but we still need more evidence to clarify the complete picture of the locus. It is fed from multiple cortical and subcortical areas, including particularly the auditory cortex, hippocampus, and amygdala, as well as the brainstem and thalamic structures [43,22,47]. Intracranial recordings from the thalamus in patients with intractable pain, Parkinson's disease, or refractory partial epilepsy have led to the identification of two possible generators: one located between the dorsal thalamic and orbitofrontal regions, and the other between the subthalamic and striatal regions [49]. These two sites are very close to the thalamus and putamen, the structures we were interested in for the present study.

Delayed P300 components have been found in patients with unilateral thalamic infarction [33,47]. A clinical study revealed a negative correlation between P300 latency and regional cerebral blood flow in the thalamus [31]. The prevailing opinion based on previous studies [7] is that the thalamus serves as a kind of "gate," choosing by filtering which information from various parts of the body it relays to the cerebral cortex, and it is from this relaying that the P300 arises. Several studies have emphasized the functions of the thalamus in aspects of cognition beyond sensory processing. The thalamus contributes to a range of basic cognitive processes that include learning and memory, inhibition control, decision-making, and the control of visual orienting responses [41,17].

On the other hand, we hypothesize that the cognitive functions of the putamen involve in various types of categorization. Knowlton, Squire and Gluck [23] found that patients with a lesion of the right neostriatum (i.e., caudate and putamen) were impaired on a classification task. Kéri, Szlobodnyik, Benedek, Janka & Gádoros [19] found that a focal basal ganglia lesion affected category learning. Researchers found that patients with a basal ganglia lesion showed a deficit in rule-based category learning [10,11]. These studies are compatible with those findings regarding the locus of P300 generation [49] suggesting the importance of putamen. We therefore predict that deficits of the P300 component will occur in patients with a putamen lesion.

The results of previous ERP studies performed on patients with cerebrovascular disease have been inconsistent. Gummow, Dustman and Keaney [16] reported decreased P300 amplitudes in patients with stroke in the middle cerebral artery, but latencies were unaffected. Unilateral thalamic hemorrhages [33] and multiple lacunar brain infarcts [46] were shown to delay the P300 wave without reducing its amplitude, but no changes were observed in its scalp distribution. Onofrj, Thomas, Paci, Scesi and Tombari [34] demonstrated normal latency, amplitude, and topographic distribution of P300 in 4 patients with basilar artery thromboembolisms, leading to the locked-in syndrome. Trinka et al. [47] found delayed P300 components in patients with unilateral thalamic infarction, suggesting an important role of the thalamus in the generation of the P300 potential.

In this study, we attempted to clarify the role of the putamen in generating the P300 through assessment of stroke patients' ability to update infrequent items in their memory. We also assessed the thalamic stroke patients for comparison because there is much evidence that the thalamus is critical in the generation of P300. We thus predicted that the pattern of brain waves in patients with a putamen lesion will be similar to that in patients with a thalamic lesion.

2. Method

2.1. Participants

The two patient groups consisted of (a) 7 patients, 5 with hemorrhage (2 female, 3 male) and 2 with infarction (1 female, 1 male) in the putamen (PUT) and (b) 7 patients (1 female, 6 male) with hemorrhage in the thalamus (THA). All the patients were recruited from the China Medical University Hospital at last 4 weeks after the stroke onset and were medically stable. Their unilateral lesions were identified by magnetic resonance imaging as located in the putamen or thalamus. Patients with medical complications, psychiatric disturbance, substance abuse, psychoactive drug treatment, and other neurological diseases possibly affecting the study were excluded.

Twenty-six healthy controls with no history of neurological or psychiatric illness were also recruited. The control groups consisted of 8 older healthy adults (OC; 3 female, 5 male) and 18 young adults (YC; 7 female, 11 male). The demographic, clinical characteristics and cognitive functioning for each of the 4 groups are summarized in Table 1. The study was approved by the Ethics Committee of the China Medical University Hospital (IRB DMR99-IRB-135) and written informed consent was obtained from patients and controls.

2.2. Task and procedure

A two-stimulus oddball paradigm (both the auditory and visual aspects) was used to elicit P300. Effortful attention was achieved by instructing participants to count silently the number of rare (target) stimuli, delivered in a pseudorandom manner with a probability of occurrence p = .2. To keep patients from guessing, there were 7 blocks, each with a different number of trials. The numbers of rare stimuli in the 7 blocks were 8, 10, 7, 8, 9, 8, and 10. The experiment included 300 trials in total.

The visual stimuli were pictures of a red disk (rare stimulus) and a green disk (frequent stimulus) presented for 100 ms in the center of a 17-inch. color monitor with a 70 Hz refresh rate. The frequencies of the rare and frequent auditory stimuli were 1200 Hz and 800 Hz respectively. All auditory stimuli were presented from the speaker of the computer. Prior to the first recording session, all participants received 20 training trials in each modality. Before receiving the oddball task, the Mini Mental State Examination (MMSE; [14]) was used to assess participant's general cognitive functioning.

2.3. EEG recordings

EEG was recorded from 36 sintered Ag-AgCl electrodes placed in accordance with the 10–20 International System [20] using a

Table 1
Means (standard error) of demographic, clinical characteristics, and cognitive functioning for each of the 4 groups.

Group	Putamen	Thalamus	OC	YC
n	7	7	8	18
Age	54.86(5.63)	57(4.47)	62.38(3.89)	21.28(1.02)
MMSE	20.86(2.59)	20.6(2.66)	29.38(0.26)	29.72(0.11)
AR	0.85(0.03)	0.87(0.04)	0.89(0.03)	0.87(0.02)
VP3_ACY	0.49(0.15)	0.51(0.16)	0.98(0.02)	0.97(0.01)
AP3_ACY	0.41(0.14)	0.43(0.15)	0.98(0.02)	0.98(0.01)

OC = Older healthy controls; YC = Young healthy controls (the same abbreviations are used in the following figures); MMSE = Mini Mental State Examination; AR = Accepted rate of artifact-free EEG epochs; ACY_V = Accuracy data in visual oddball task; ACY_A = Accuracy data in auditory oddball task.

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