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P300 in Posttraumatic Stress Disorder

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Key Words

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Abstract

In the present study, P300 has been recorded in 26 subjects (15 women) 1 month after an aggression without organic complications. Among our sample, 16 subjects fulfilled DSM-III-R criteria for posttraumatic stress disorder (PTSD) and 10 did not. P300 amplitude was significantly lower in the 16 PTSD subjects as compared to the 10 subjects without PTSD. This study supports information processing disturbances in PTSD.

Introduction

Posttraumatic stress disorder (PTSD) usually occurs after a stressor that would evoke significant symptoms of distress in almost everyone. It is characterized by intrusive, avoidance and hyperactivity symptoms [1]. During the past decade, the limited number of studies which have focused on the psychophysiology of PTSD have reported increased psychophysiological responses (heart rate, blood pressure, electromyography) to combat-related stimuli in PTSD patients as compared to control subjects [2].

All those studies only used peripheral physiological markers. More recently, however, event-related potentials (ERP), which provide a psychophysiological index of central processing, were recorded in PTSD patients [3]. This study assessed brain potentials to four intensities of tones according to an augmentation/reduction para-

digm. Among PTSD patients ($n = 12$), 75% were reducers as compared to 17% of the controls, supporting a psychophysiological hyperactivity.

The P300 wave [4] is another ERP with particular relevance to the study of cognitive processes. P300 amplitude has been related to some psychological variables such as expectancy, stimulus significance and attention, and is known to be influenced by various psychopathological conditions [5].

Since PTSD is characterized by important cognitive disturbances, particularly impaired memory and attention [6], P300 could represent an important tool in order to assess the psychological consequences of traumatic stress. Indeed, in a recent study, abnormal information processing assessed by P300 was found in PTSD [7]. Therefore, the purpose of the present study was to replicate specifically the P300 modifications in PTSD subjects.

Methods

Subjects

Twenty-six subjects (15 women) aged 19–53 years (mean age = 35.6 ± 9.9 years) participated in the study. All were victims of hold-up without any physical consequences. Most of them ($n = 20$) worked in post offices, the others were attacked at home. Clinical evaluations were performed by a forensic psychiatrist upon the request of a judge between 4 or 5 weeks after the trauma. Sixteen of the victims fulfilled DSM-III-R criteria for PTSD. The subjects with and without PTSD did not differ regarding their gender distribution (5 men and 11 women vs. 6 men and 4 women, $X = 1.073$, $DF = 1$, $p = 0.3$) and their mean age (35.06 ± 9.3 vs. 36.6 ± 10.3 , $F_{1,25} = 0.14$, $p = 0.71$). All subjects were free of medical and psychiatric illness before the trauma, as assessed by detailed history, reports from general practitioners and using the Schedule for Affective Disorders and Schizophrenia (Lifetime version) [8]. None of the subjects was taking any psychotropic drug at the time of the evaluation.

Electrophysiological Procedure

The ERP procedure was conducted together with the clinical evaluation. Subjects wore earphones and sat in a sound-attenuated room. A series of 300 auditory stimuli were presented with a fixed interstimuli interval of 2.5 s. According to the classic oddball paradigm, 90% of the stimuli (frequent) were tones of 750 Hz, 75 dB and 700 ms duration. The other 10% (rare) were tones of 2,000 Hz, 75 dB and 700 ms duration. The subjects were asked to silently count the rare stimuli.

The EEG was recorded using silver-silver chloride disc electrodes attached with collodion at Cz, using linked earlobes for references and forehead for ground. All sites were cleaned with acetone and abraded to maintain resistance below 3 k Ω . EOG was recorded from above the left eye. Amplifier gains were set at 10,000, with a band pass of 0.1–30 Hz, and digitized at 512 samples/s for 800-ms epochs (of which the first 100 ms were prestimulus activity).

P300 amplitude and latency were defined as the higher positive point which occurs after 250 ms on the rare tone wave minus the amplitude of the mean wave from the frequent tone.

Statistical Analysis

P300 parameters of PTSD and non-PTSD subjects were compared using analysis of variance.

Results

P300 amplitude was significantly lower in the 16 PTSD subjects as compared to the 10 subjects without PTSD (8.8 ± 2.4 vs. 13.7 ± 0.9 μ V, $F = 38.6$, $DF = 1,25$, $p < 0.001$). In contrast, P300 latency did not significantly differ between the two groups (315.9 ± 19.3 vs. 310.4 ± 25.3 ms, $F = 0.4$, $DF = 1,25$, $p = 0.46$). No gender differences were observed in P300 amplitude or latency (fig. 1).

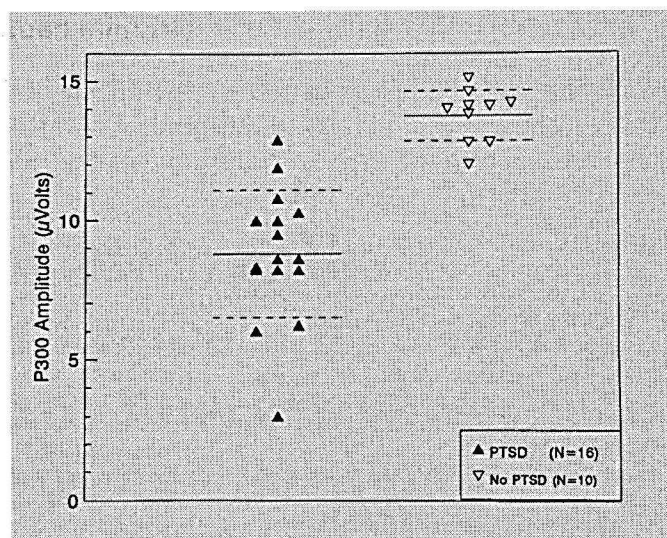


Fig. 1. P300 amplitude distribution among subjects with or without PTSD.

Discussion

The major finding of the present study is that the victims of psychological trauma who fulfill DSM-III-R criteria for PTSD exhibit a reduced P300 amplitude as compared to victims without PTSD. These results confirm the information processing disturbances recently reported in PTSD [7].

From a cognitive point of view, P300 has been linked to categorization and information processing. More precisely, it reflects the context updating of memory and has been considered as the comparator between relevant and irrelevant information [4]. The reduced P300 obtained in our PTSD subjects could be interpreted as an impairment in the discrimination of relevant and irrelevant information, or more generally as attentional deficits.

Moreover, P300 amplitude has also been related to some psychological variables such as expectancy and stimulus significance. Unsignificance of the stimuli could be responsible for the observed P300 reduction and would be in agreement with the psychological numbing described in PTSD [9].

Of particular interest is also the lack of difference in P300 latency between the two groups. Since P300 latency reflects the evaluation of the time of the stimulus, these results suggest that information is normally processed in PTSD. The lack of N1 and N200 data however prevents us from analyzing the first steps of information processing.

Finally, several limitations in the design of the study should be acknowledged. First, by using only one recorded channel, no information on topographic distribution was available. Second, the lack of independent rating of intrusive, numbing and hyperarousal symptoms prevents

us to specifically assess possible relationships between P300 modifications and certain aspects of psychopathology. Future studies using larger samples will be necessary to deal with these important issues.

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