

## The P300 Event Related Potentials and its Implications in Psychiatric Disorders

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### Abstract

P300 wave is an event related potential which has been frequently used in various psychiatric and neurological disorders for research and clinical purposes. It is supposed to give an insight about the in-depth neuroanatomical and neurobiological underpinnings of various brain structures in different disorders. Nowadays, it is being used often in different psychiatric disorders as a part of neurophysiological/neurobiological studies. The P300 findings are varied across the several disorders which points towards the varied neurophysiological basis in different disorders. P300 in short provides an opportunity to detect the fine cognitive processes underlying the complex psychiatric disorders. It brings psychiatry more towards being 'biological'. This brief review is expected to give an overview of findings of P300 parameters in psychiatric disorders and also to provide its implications in these disorders.

**Keywords:** P300; Event –related potential; Psychiatry; Neurophysiology

### Introduction

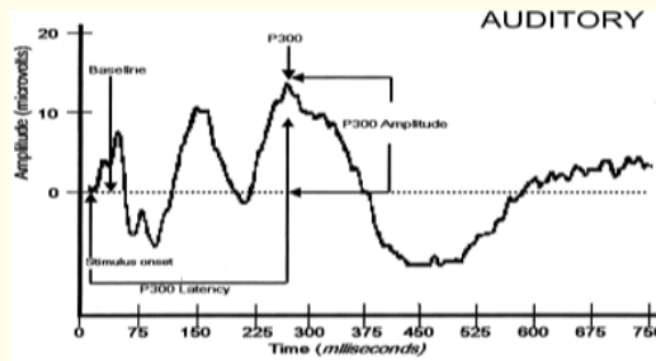
Biological research in psychiatry has advanced a lot since last 3 decades and has enhanced our understanding of certain abnormalities that underpin the major psychiatric disorders. Now a days more and more studies are being done in biological psychiatry so as to find out any genetic or neurobiological abnormalities behind the major disorders which is expected to reveal more and more unknown mechanisms behind development of such disorders.

P300 wave is one such much studied parameter in biological psychiatry. It is an event-related potential (ERP) that is generated and recorded in the central nervous system (CNS), following the presentation of a stimulus. The P300 wave is a component of ERP that was first described by Sutton, *et al.* in 1965. The P300 event-related brain potential is an index of endogenous cognitive processes, typically elicited by infrequent sensory stimuli that are either task relevant or novel. It receives its name from its appearance as a large vertex-positive component with peak latency approximately 300ms after stimulus presentation. Occurring after the obligate evoked potential response to the physical attributes of a stimulus, it reflects a variety of cognitive processes elicited by a change in the sensory environment. It is involved in the process of decision making, novelty processing and attention. It is associated with brain activity related to the engagement of attention especially orienting and involuntary shifts to changes in the environment. It is perhaps the most studied of the ERPs and usually elicited in most research settings by auditory and visual modalities. It has also been regarded as an endophenotype [1].

Debate still continues regarding the exact physiological process associated with P300 and its origins from CNS but it is being used frequently as a non-invasive procedure for studying various physiological correlates of cognitive processing in psychiatric disorders [2]. This article is intended to give a brief idea on various findings of P300 from various studies done so far in different psychiatric disorders and its implications in psychiatry.

**P300: Elicitation & Neuroanatomical basis:** The P300 potential is elicited by variants of the 'oddball' paradigm. The subject is required to respond physically or mentally to the target or the 'oddball' stimulus that is presented. This gives rise to a potential that can be measured by means of electrodes placed on the scalp. The P300 is measured by quantifying its amplitude (size) and its latency (timing). The amplitude is defined as the voltage generated as measured from the baseline amplitude to the largest positive peak of the P300 waveform

in the latency period. The latency is the time that is taken for the wave to reach its maximum positive peak within the latency period from the point of stimulus. (See Figure 1)



**Figure 1:** P300 amplitude and latency.

P300 is not an unitary phenomenon. It contains two subcomponents – P3a and P3b. P3a is a component of the time-locked (EEG) signals known as ERP. It arises as a result of incoming stimuli that replace the contents of working memory in the anterior cingulate cortex, the communication of which is transmitted to the infero temporal lobe representation mechanisms [3]. It is produced when a demanding stimulus commands a frontal lobe attention. P3b has been associated with brain activity related to the engagement of attention especially orienting, involuntary shifts to changes in the environment and novelty processing. It arises from memory storage operations in the hippocampal cortex with updated output to the parietal cortex [4]. It is produced when attention resources are allocated for the memory updating in the temporoparietal association cortex.

### P300 findings in different psychiatric disorders

**Schizophrenia:** Since the initial reports of Callaway and colleagues of P300 deficits in schizophrenia, a large number of investigators have identified their topography, lateralization and neural basis. The results of these studies make the P300 event-related potential an excellent candidate endophenotype for understanding the genetic basis of the deficits [5]. Various researches based on P300 abnormalities in schizophrenia are noted down. In these studies, P300 refer to the P3b component elicited by target stimuli typically in an oddball paradigm.

- Reduction of P300 amplitude to target stimuli has been found in schizophrenia, more reliably for auditory than visual stimuli [6].
- P300 amplitude reduction is not due to increased latency variability in the patients of schizophrenia [6].
- Auditory P300 amplitude reduction has been shown in patients receiving medication [7] patients withdrawn from medication and in never medicated patients [8].
- Auditory P300 amplitude reduction has also been seen in patients in first episode of the illness [9].
- Delayed latencies have been reported in patients with longer duration of illness [10].
- Increased latencies of the P300 were proposed as a consequence of neuroleptic medications [11].
- Neuroleptic-free patients showed longer P300 latencies, which normalized after sufficient medications [12].
- P300 deviances described in schizophrenia are not caused by antipsychotics but are genuinely associated with the disease, either as a premorbid marker of risk, or as part of the schizophrenia syndrome [13].

Hence, Auditory P300 amplitude reduction may represent an enduring biological feature of schizophrenia that is relatively insensitive to clinical status or medication after the onset of the illness. However, Auditory P300 amplitude reduction is not specific to schizophrenia. It has also been found in other psychiatric disorders like depression and alcoholism. But the topographic distribution of P300 reduction might be specific to schizophrenia as found in several studies.

- Patients with schizophrenia show a greater P300 amplitude reduction over the left than over the right temporal lobe regions [14].
- Subsequent studies have shown that the lateralized P300 deficit appears in off-medication schizophrenic patients [15], in first-episode schizophrenic patients [9] and in never-medicated schizotypal subjects [16] when a nose reference [17] and a 64-channel recording [18] is used.
- Most of these studies show left<right temporal lobe P300 asymmetry. It was also suggested that left temporal lobe abnormalities may play a major role in schizophrenia, especially in the genesis of positive symptoms [19].
- Increased task difficulty may also reduce P300 asymmetry [20,21].

In a study comparing first-episode schizophrenic patients, first-episode affective psychotic patients, and age matched controls it was found that first-episode schizophrenic patients displayed smaller P300 amplitude over the left temporal region (T3) than did either first-episode affective patients or normal control subjects. The first psychotic episode affective disorder patients, although showing an overall P300 amplitude reduction, did not show a left < right asymmetry. These data suggest that P300 left temporal region abnormalities reported in chronic schizophrenia are present at the onset of the illness [9]. It has also been suggested that right maximum of the P300 and decreased amplitudes in schizophrenic patients is closely related to a set of negative symptoms [22,23], poor outcome [24], early onset of schizophrenia [24] and verbal dysfunctions [25,26]. Furthermore, this pattern indicates an increased rate of tardive dyskinesia [22]. Relapse to an episode of schizophrenia was more probable in patients with reduced amplitudes [27]. Thus, because of overlaps with healthy volunteers and lacking specificity, neither amplitude reductions nor latency prolongations could be used as diagnostic marker for schizophrenia until now.

**Acute and transient psychotic disorders (ATPD):** In a controlled study of P300-evoked responses in patients with cycloid psychosis, normal P300 topographies and latencies but significantly higher P300 amplitudes than normal controls were observed. This finding is suggested to be indicative of higher levels of arousal in the cycloid psychosis group [28,29]. A recent study which evaluated P300 parameters in ICD-10 diagnosed ATPD patients and compared with P300 parameters in patients with schizophrenia and healthy biologically unrelated controls revealed that while the schizophrenia group differed significantly from the ATPD group in all the P300 parameters i.e. amplitudes, latencies and reaction time, the ATPD group was found to have lower mean Pz latency and low mean reaction time as compared to healthy controls which may indicate hyper-arousability and heightened attention in ATPD patients. This study provided an insight that auditory P300 ERP could distinguish between ATPD and schizophrenia and may also be used as a tool for prediction of recurrence in ATPD [30].

The lack of P300 asymmetries in this patient group indicates the absence of abnormal functional asymmetries in cycloid psychosis/ATPD. The hemispheric blood flow level co varied significantly with the degree of clinical symptoms, such that the more elevated the cortical blood flow was, the more behaviorally disturbed was the patient [31]. The finding of higher than normal amplitudes in cycloid psychosis/ATPD is consistent with the typically excellent prognosis of this group, including full recovery and complete restitution of social competence [29,30]. However, it is still not clear whether the elevated P300 amplitude is a trait among patients of acute psychosis or is a transient remainder of psycho-vegetative excitation during acute psychosis and further studies are needed to bring more light on this disorder.

**Depression:** Most of the studies on P300 in depressed patients have used a simple “oddball” technique and have found out that depressed patients have reduced P300 amplitude when compared to normal healthy controls. There exists some controversy as there are some studies which found out no significant difference in P300 latency between depressed patients and controls whereas in some researchers have found out that depressed patients have longer P300 latencies than controls [32]. Another study has found out that P300 amplitude is significantly reduced in unmedicated depressed patients but the reduction is not as large as compared to schizophrenic patients and it became normal after treatment [33]. A Study has also been done so as to compare P300 findings between patients of typical depression and atypical depression and it revealed though patients of both typical depression and atypical depression have reduced P300 amplitude but typical depression patients have longer mean latency as compared to atypical depression patients. However, no difference has been found between melancholic depression and simple mood reactive depression. It was also found that typical depression patients failed to show normal hemisphere asymmetry of P300 [34]. The reason behind such findings in patients with depression can be because of cognitive deficits present in such patients. These studies do reveal some evidence for the importance of dealing with heterogeneity of depressive disorders.

**Bipolar disorder:** In bipolar disorders, several studies have been done based on P300. In some studies no significant differences have been found between patients and healthy volunteers [35]. In a study by Strik, *et al.* in 1998 it was found that in patients with mania, P300 amplitudes and latencies were normal but more posterior positive peaks of the amplitudes were obtained which is explained by the fact that patients with mania have frontal disinhibition [36]. Anteroposterior P300 Topography findings in patients with acute mania with psychosis revealed anterior reduction i.e. frontal which showed left > right asymmetry. The frontal reductions in bipolar psychosis may suggest abnormalities in a hypothetical frontal generator which is suggestive of the altered frontal lobe functions in mania [37]. It has also been confirmed by PET study which found decreased right rostral and orbitofrontal activation in patients with mania [38]. Patients with major depressive episode in bipolar disorders have similar P300 findings as described in patients with unipolar depression.

**Anxiety disorders:** In patients with anxiety disorders several studies have been done which have revealed that P300 amplitude is reduced compared to healthy controls [39,40]. Electroencephalographic studies in patients with anxiety disorders have found abnormal symmetries of left-right brain function [41]. In a recent study which was intended to study auditory P300 ERP in such patients using dipole source analysis has revealed that patients with anxiety disorders have reduced P300 amplitude which is however less as compared to patients with depression. This study also found out that in these patients there is right greater than left hemispheric asymmetry of dipole magnitude that too more in temporal cortex. It suggested that patients with anxiety disorders may increase their efforts to enhance temporal dipole activity to compensate for a deficit in frontal cortex processing [42]. However, more studies are required in patients with anxiety disorders so as to gather more information.

**Obsessive and compulsive disorder (OCD):** Cortical hyperactivity have been proved to be the major pathogenetic factor in OCD from various neuroimaging studies. As P300 is related to cognitive and attention process which involve functioning of brain cortex, so several studies have been done on it in OCD patients. But these studies have conflicting results. In most of the studies, OCD patients have been found to have reduced P300 amplitude and shorter P300 latency especially when the difficulty of the task was increased as compared to healthy controls [43-46]. However, a study done by Sanz, *et al.* (2001) found OCD drug free patients to be having longer P300 latencies and lower P300 amplitude [47]. Another study which took into account the two different sub-components of P300 separately i.e. P3a and P3b revealed no difference in P3a findings between OCD and healthy controls but larger P3b amplitude, a shorter P3b latency and shorter reaction time in OCD patients [48]. From this we can conclude that since P3b is more related to attention and cognitive process, P3b abnormalities could be because of overfocussed attention, faster cognitive process and hyperarousal in OCD patients. All these studies done so far in OCD provide evidence behind the neurophysiological abnormalities in these patients and further broaden our understanding in this disorder.

**Personality disorders:** Very few studies have been done in patients with personality disorders. It has been found that patients with personality disorders and healthy subjects who are extrovert in nature have high P300 amplitude [49,50]. Studies on patients with borderline personality disorder showed increased P300 latency [51]. Studies have also been done on schizotypal personality disorder which suggested normal P300 parameters [52] but a tendency towards reduced P300 amplitudes was found in central and parietal electrodes in separate study which studied P300 topography in these patients [16]. P300 studies in different personality disorders is lacking, so an effort should be made to do such studies in future which will further help the psychiatrists to unpin any biological abnormalities behind these disorders.

**Substance use disorders:** P300 has been used as an objective indicator of brain activity which is disrupted by acute effects of drug use [53]. Studies on P300 inpatients with alcohol use disorders have been done extensively and these patients have reduced P300 amplitude [54,55]. Initially it was thought that the P300 deficit was due to effect of alcohol on brain but later on it was found that even after a sufficient period of abstinence, this deficit still persisted [56]. In another study, it was found that sons of alcoholics who are naïve to alcohol have also low P300 amplitude [57]. A meta-analysis suggested that low P300 in unaffected first degree relatives of alcoholics may have a predictive value as an index of vulnerability for alcohol dependence [58]. However, P300 studies are very few in patients with opioid dependence. In a study done to examine P300 activity in patients who were dually dependent on cocaine and opioid revealed that prior to detoxification, no difference in P300 amplitudes were found between patients and healthy controls but after detoxification, P300 amplitude was found to low in patients as compared to healthy controls which again became normal after buprenorphine treatment [53]. An exploratory study done on P300 which compared P300 in opioid dependent patients, their first degree brothers and healthy controls revealed smallest amplitudes and longest latencies of P300 in opioid dependent patients and largest amplitudes and shortest latencies in controls whereas the brothers group had an intermediate position [59]. More extensive studies on these disorders will uncover further new and interesting findings in neurophysiological aspects of substance use disorders.

**Dementia:** P300 studies in patients with dementia of Alzheimer’s type had revealed low amplitudes at Cz, Pz and Oz electrodes [60]. A meta-analysis of P300 done on Alzheimer’s patients suggested that the amplitudes were unspecific but there were increased latencies which correlated with the decline in cognitive process in such patients [61]. Studies are lacking in patients with severe dementia may be because of the fact that such patients are incapable to carry out the procedure of oddball paradigm.

**Conclusions**

From all these studies, it is very clear that P300 is an important parameter in biological psychiatry and more and more researches on it are going on in various disorders. Many findings from different studies contradict each other and it appears that P300 has low reliability. But the reason behind these results could be because of overlapping sub-components P3a and P3b in P300 recordings which have different physiological process behind their generation. However, the findings of P300 in different disorders provide an interesting outlook to revisit these disorders so as to do more research and to understand neurophysiology behind them (See table 1).

| Psychiatric disorders   | P300 amplitude  | P300 latency  |
|---|---|---|
| Schizophrenia in all phases (acute/chronic/remitted/never medicated patients) | Low amplitude   | Increased latency   |
| Acute and transient psychotic disorders                                       | Higher amplitude as compared to normal; inconclusive results due to few studies | Decreased latency ; inconclusive results due to few studies       |
| Depression  | Low amplitude than controls   | Increased/longer latency; normalized after treatment              |
| Bipolar disorder  | Manic phase – normal amplitude<br>Depressive phase – low amplitude              | Manic phase – normal latency<br>Depressive phase – longer latency |
| Anxiety disorders   | Reduced amplitude which is less when compared with patients with depression     | Inconclusive findings   |

|   |   |   |
|---|---|---|
| Obsessive-compulsive disorder                           | Higher amplitude as compared to controls  | Longer latency                            |
| Personality disorders (PD) Borderline PD-Schizotypal PD | High amplitude<br>Tendency towards low amplitude  | Increased latency<br>Normal latency       |
| Substance Use disorders<br>Alcohol<br>Opioid            | Low amplitude<br>Low amplitude before detoxification which normalized after agonist treatment | Possibly longer latency<br>Longer latency |
| Dementia  | Non-specific verses low amplitude   | Longer latency                            |

**Table 1:** Summary of P300 findings in different psychiatric disorders.

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