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Opinion Open Access

P300 for Depression: An Underestimated Neurophysiological Tool

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Opinion

Inspite of all the latest advancements in the treatment of depression, it remains a challenge for treatment so much so that 30-40% cases will be diagnosed as treatment resistant depression due to unresponsiveness for two adequate trials of psychopharmacological treatment [1]. The fact that this figure has remained constant over past several years irrespective of the developments in treatment forces us to think that depression may be a more complicated issue than what we have presumed so far. In fact, it seems that depression is not a single condition but a common manifestation of multitude of different kinds of underlying abnormalities affecting the brain function. This issue of heterogeneity has been recently addressed by Fried [2] where he describes the fallacies of presuming all the kinds of depression as a single entity. Now an important question arises that if depression is really a heterogeneous entity, then how do we evaluate the individual differences of this condition? Clinically we can only observe the symptoms presented at a cross-sectional level. Thus we cannot actually differentiate the underlying heterogenity of this condition merely by clinical/scale based assessments. In such a scenario, neurophysiological data can be of immense help by which we can observe the differences at the level of neural functioning. Unfortunately, depression has not been looked adequately from the neurophysiological standpoint. An important neurophysiological tool in this respect has been the event related potential P 300. In this brief opinion article, we stress upon the importance of using ERPs especially P300 as an additional tool to assess depression both for diagnostic and prognostic purposes. To strengthen our point, we will highlight some important studies conducted in past decade. A literature review reveals that P300 has been used in depression mainly for two purposes- 1) Diagnostic and 2) Prognostic. We have covered these uses in detail elsewhere [3]. Here we provide only an overview.

Regarding the diagnostic use, P300 evaluation of depressed patients was prominent in 1980s-90s. However, subsequently its use decreased drastically perhaps because the concept of depression was evolving at the time and the decrease in P300 amplitude found over several studies [4] was attributed simply to the attention/decision making process deficits. However, a notable aspect of those studies was that P300 amplitudes and latencies was not uniformly affected in all depression patients but in fact showed a wide range of variation [4] the reason of which unfortunately has not been explored adequately even to this date. Current trend has been focused on individual symptom evaluation using P300 rather than the whole condition of depression. For example, Urretavizcaya et al. [3] investigated the differences between the event related potentials of normal individuals and melancholic patients. They observed that a significantly higher latency as well as ERP waveforms like N100, N200, P300 and significantly lower P300 amplitude was produced in melancholic group as compared to the healthy controls. However, the latencies of the N400 or their inter latencies showed no differences. However, the authors could not find any associations between these abnormalities and the clinical variables. They mentioned that all the subjects of this study were inpatients and not random, with a severe subcategory of depression and the value of average age was also high. Therefore, these findings could not be generalized. Similarly, Karaaslan et al. [5] investigated the possible differences in the P300 component of event-related potentials in depressed patients in relation to psychotic features and to check the effect of treatment if any on these changes. They found that the pre-treatment P300 latencies were significantly prolonged in both type of patients i.e. with and without psychotic features as compared to controls. However, the patients with psychotic features exhibited significantly decreased pre-treatment P300 amplitudes and not in those without psychotic features. Vandoolaeghe et al. [6] used P300 to specifically study cognitive status of depressed patients. They observed significantly higher P300 latency and P200 amplitudes in the patients suffering from major depression with normal cognition when compared to normal volunteers. However, significantly lower P300 latency was found when these patients were compared with major depressed patients having cognitive impairment, although, AERP components showed no significant changes upon subchronic treatment with antidepressants. These findings suggest that P300 parameters can contribute significantly to assessing some specific dimensions of depression like psychosis and cognition.

The other use of P300 in depression has been to aquire a prognostic evaluation/treatment response. Interestingly, the prognostic/treatment response issue of depression is also intricately intertwined with the question whether the P300 changes in depression are an indicators of state/trait markers. Regarding the effect of treatment on P300, several studies have shown that with adequate treatment, the P300 amplitudes and latencies recover [7]. Similarly, Murty et al. [8] found that P300 amplitudes were smaller in depressed patients and this abnormality normalized after recovery. Karaaslan et al. [5] observed that after being treated for depression, delayed P300 latencies in patients and decreased P300 amplitude in the patient group with psychotic features became normalized. These studies results show that the delayed P300 can be best understood as the state marker of depression. Based on these findings, they concluded that prolonged P300 latency can be considered a state marker for major depressive episode. The prognostic value of P300 on the other hand is a more complicated issue. An interesting study in this context was conducted by Jaworska et al. [9] who observed that non-responders to antidepressant treatment had smaller baseline P3a/b amplitudes than responders and healthy controls. Their weekly assessment model gave weekly predictions of the extent of depression by rating changes till as far as 12 weeks. ERP

measures showed correlation with clinical changes in males and with behavioral measures in females. These results reinforce the idea that a positive antidepressant response is associated with greater (or control-like) baseline P3a/b amplitudes. Similarly, Vandoolaeghe et al. [6] found that those who did not respond to the antidepressant therapy had significantly higher pretreatment P300 latency and P200 amplitude as compared to the treatment responders and normal volunteers. The authors concluded that major depression is accompanied by delayed P300 latency as well as increased P200 amplitude and may predict a nonresponsive to subsequent anti-depressive therapy.

To conclude, the academic interest in the status of P300 in depression condition needs to be re-kindled which can serve a multitude of functions including diagnosis, treatment response as well as prognostication. Especially the studies on treatment resistant depression would be rewarding in this respect.

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