Chapter 10
Neurocognitive Mechanisms for Detecting Early Phase of Depressive Disorder: Analysis of Event-Related Potentials in Human Brain

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ABSTRACT

This chapter discusses neurocognitive mechanisms in terms of latency and amplitudes of EEG signals in depression that are presented in the form of event-related potentials (ERPs). Reviewing the available literature on depression, this chapter classifies early P100, ERN, N100, N170, P200, N200, and late P300 ERP components in frontal, mid-frontal, temporal, and parietal lobes. Using auditory oddball paradigm, most of the studies testing depressive patients have found robust P300 amplitude reduction. Proposing EEG methods and summarizing behavioral, neuroanatomical, and electrophysiological findings, this chapter discusses how the different tasks, paradigms, and stimuli contribute to the cohesiveness of neural signatures and psychobiological markers for identifying the patients with depression. Existing research gaps are directed to conduct ERP studies following go/no-go, flanker interference, and Stroop tasks on global and local attentional stimuli associated with happy and sad emotions to examine anterior cingulate cortex (ACC) dysfunction in depression.

INTRODUCTION

Depression is an activity of abhorrence with a sad mood that severely affects human personality, behaviour, language, thoughts, emotions and sagacity of well-being (Schnaas, 2003). Studies on cognitive theories of depression posit that depressive disorder patients always suffer from the cognitive dysfunctions and impairments, dysregulation of emotions, inhibitory processing, deficits in working memory, and faster response to negative life events. Patients with depressive disorder are characterized with a sad mood, lack

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of interests, distrust, destructive belief of self, lack of motivation, communicative passivity, and suicidal thoughts McCabe et al., 2018). They largely show the incompetency to enhance optimistic and positive environmental stimuli for controlling their sad emotion and negative mood. These adverse cognitive and affective syndromes have severe effects on brain activity, leading to neuro-degeneration and disorders. In this regard, a state of mind that is characterized by apprehension, sorrow, grief, irritation, anger, hopeless, and guilty, is called a depressed mood (Rottenberg, 2005). When this type of mood occurs frequently after remission and controls day-to-day activities of the patients, by following a pathological stability at the neurocognitive level, it is categorised as a major depressive disorder (MDD) (Goodwin and Jamison, 2007; Taylor et al., 2007). Therefore, the screening and recognition of pathophysiological symptoms, based on the neurocognitive signatures, are some of the challenges that are not only to trace the origin, progress and recurrence of the depressive disorder, but also to prevent and treat the recurrence of it in a regular interval. The aim of this chapter is to discuss the various symptoms and types of depression, methodological insights and neurocognitive mechanisms to capture the severity of depression.

The course of depressive illness is unpredictable and episodic in nature, even patients feel fine between acute depressive episodes. Most of the cases, the longevity, number and patterns of episodes help to classify different types of depression. Considering the duration or phase of symptomatic mental illness, the depressive disorder is categorised into seven sub-categories such as (i) onset phase with a unipolar and single depressive episode, (Penninx et al., 2011; Spijker et al., 2002), (ii) recurring phase of episodes with major depression (Boschloo et al., 2014; Angst et al., 2009), (iii) chronic phase of episodes with dysthymia (Sansone et al., 2009), (iv) bipolar and manic-depressive disorder (Simon et al., 2008; Baik et al., 2018; Bertolote et al., 2004; Lejoyeux et al., 2010), (v) psychotic and delusional depression (Korn et al., 2014; Tondo et al., 2014; Rapinesi et al., 2015) and (vi) seasonal affective disorder (Winthorst et al., 2017). Before applying any therapeutic solution, patients use to go through the psychometric and neurocognitive diagnostic procedures for classifying these types of depressive disorder. Currently, there is a little explanation and discussion relating to the neural deficit and neurocognitive mechanisms that recognise the different types of depressive disorder.

Neural Deficit and Cognitive Impairments

The neural networking system of MDD is related to the function of multi-domain cognitive damage including working memory function, mental imagery and schema, attention span, executive function, and perceptual speed (Chen et al., 2013; Chen et al., 2015). In this direction, various brain imaging techniques are used to diagnose depression and provide an understanding of the essential underlying mechanisms leading to neural dysfunction and cognitive impairments in human (Christoffel et al., 2011). For example, the dysregulation of synaptic plasticity (Cruz-Martín et al., 2010; Hayashi-Takagi et al., 2010; Penzes et al., 2011), neurological ailments (Akram et al., 2008; Bingol and Sheng, 2011), and cognitive deficiency (Dumitriu et al., 2010) are recognised in different types of psychiatric disorder. The reduction and disruption of spine synapses that connect between neurons, contributes to the depressive vulnerability. Therefore, the disruption in synaptic plasticity, reduction in the volume of hippocampus and activity of brain regions are correlated with the depression in the clinical population (Woolley et al., 1990). Pizzagalli (2014) and Wacker et al., (2009) demonstrated that the reduction of NAc volume and NAc responsiveness to rewarding stimuli are directly linked with anhedonia, which is considered as a core symptom of depression. Anhedonia has also been proposed as a biomarker for the recovery of depression (Hasler et al., 2004). Previous studies have also recognised the regions of the brain such as