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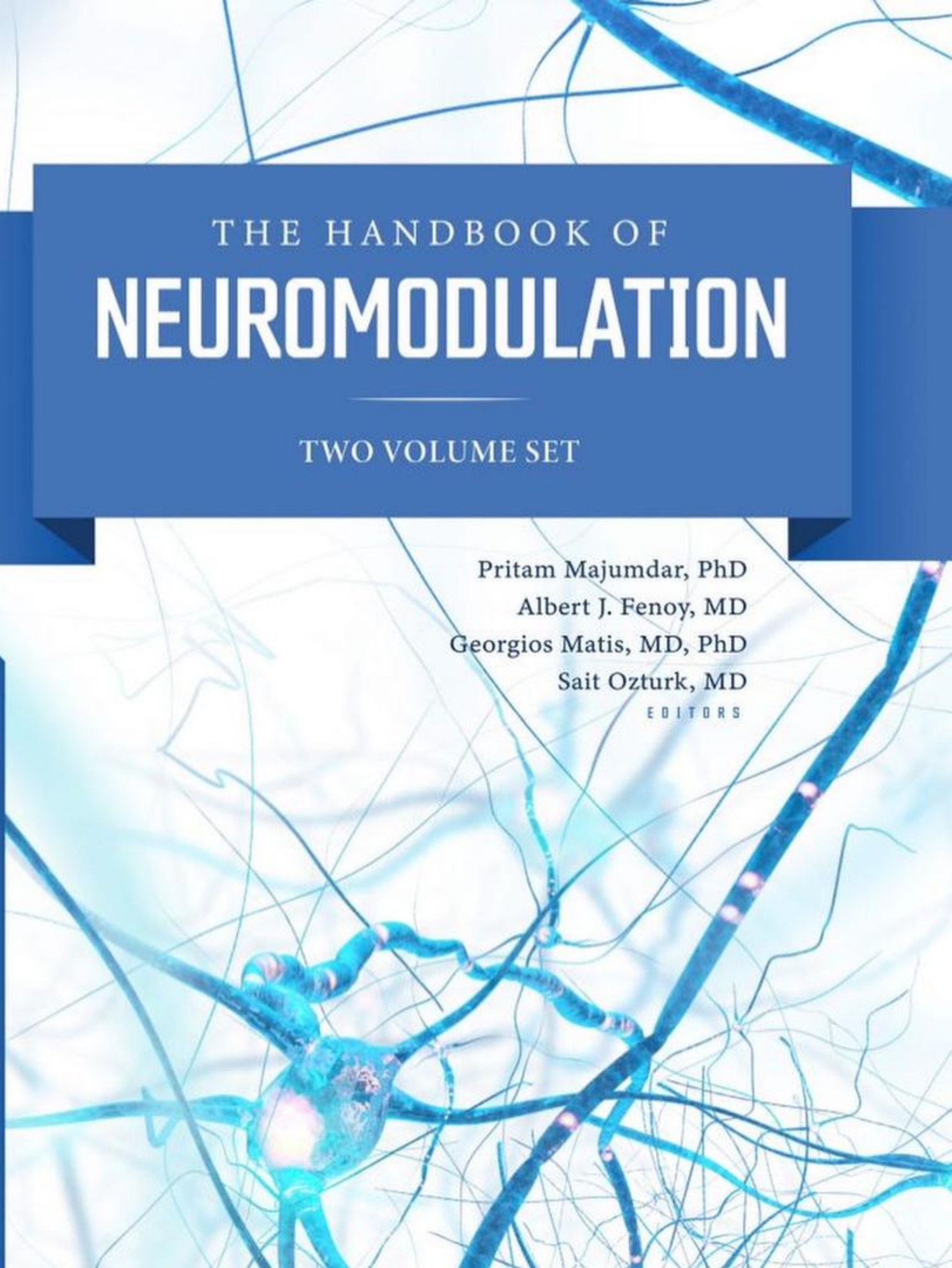

Chapter 19

Deep Brain Stimulation for Obsessive Compulsive Disorder

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Abstract

OCD is a common psychiatric disorder represented by a diverse group of symptoms, including constant or recurring intrusive anxiety-generating thoughts, which is known as obsession associated with some compulsive ritualistic, repetitive behavior (Association, 2013; Sadock, 2015). To reduce the anxiety associated with obsession, the patient feels driven into a specific compulsive act, but it does not help all the times and may even worsen the pre-existing anxiety (Sadock, 2015). These obsessions and compulsions can be so time-consuming as to interfere with a patient's social life and activities (Sadock, 2015).

According to DSM-V diagnostic criteria for OCD, the patient should have time-consuming (more than 1 hour per day or interfering with normal functioning) obsessions or compulsions or both of them which are and not attributable to another medical condition or substance abuse and defined as below:

Obsessions are defined as constant or recurring intrusive thoughts or urge that mostly cause anxiety or distress, and the patient attempts to ignore or neutralize them with some other thoughts or by performing a specific action. Compulsions are repetitive behaviors or mental acts that, because of obsessions patient feels compelled to perform. These behaviors are aimed to reduce the patient's anxiety basis upon an unrealistic connection, and most of the time, even worsen it (Association, 2013).

The lifetime prevalence of OCD has been estimated to be 2 to 3 percentage in the general population. This estimation has placed OCD as the fourth most common psychiatric diagnosis (Sadock, 2015).

The conventional therapeutic approach to OCD is based on medical and psychological therapies. Although combination therapy of SSRIs and ERP is useful in many patients, 40-60% of patients experience persistent symptoms, and about 20% of them are refractory to these conventional treatments (Hezel & Simpson, 2019; Hirschtritt, Bloch, & Mathews, 2017; Kim et al., 2011). In addition to the disturbance in their everyday social life, these

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Chapter 20

Deep Brain Stimulation for Depression

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Abstract

Ancient Greek used the term *melancholia* instead of depression. Melancholia consists of 2 parts —*melas* means black and *khole is* translated as bile; thus, melancholia means black bile. It comes from Ancient Greeks' belief indicating that the healthy body depends on the balance of 4 fluids and hormones—blood, yellow bile, dark bile, and phlegm (Karst, Friedman and Katz 1974, Jackson 1986). In *melancholia*, the balance of these 4 fluids is disturbed. A patient suffering from Melancholia developed particular mental and physical symptoms, as explained by Hippocrates in his book, *Amorphous* (Jackson 1986). Persian physicians extended the definition of Avicenna defined melancholia in his book, The Canon of Medicine, as a state of depressed mood (Haque 2004, TABEI, DR et al., 2004). In addition, he evaluated the relationship between depressive mood and several diseases. In the Canon of Medicine, he illustrated several treatments for depression such as herbal Persian medicine as antidepressants, aromatherapy, and music therapy (Khodaei, Noorbala et al., 2017).

In the 17thcentury, Robert Burton in '*The Anatomy of Melancholy*' illustrated that melancholia could affect different aspects of the patients' daily life such as sleep and social activities (Burton 1912).

Emil Kraepelin, a German psychologist, for the first time used the term depression which has been extracted from the Latin word 'deprimere' and defined it as a decrease in mood. In addition, he described different types of melancholia in various decades of life, for instance, involuntary melancholia in adulthood (Davison 2006). In 1860, a French psychiatrist, Louis Delasiauve, reported specific symptoms of depression (Berrios 1988).

Introduction

Major depressive disorder (MDD) is a rife psychiatric disease that is categorized as a mood disorder. In MDD, the patients experience at least 5 out of 9 symptoms: depressed mood, decreased energy, psychomotor retardation, diminished daily activity level, appetite variation,

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Chapter 22

Deep Brain Stimulation for Alzheimer's Disease

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Abstract

Alzheimer's disease (AD) is the most common type of dementia and as an age-related disease is increasing in prevalence as life expectancy has raised worldwide. In 2016 there were about 43.8 million individuals affected by dementia. This figure has more than doubled in the last 30 years (Nichols et al., 2019) and it is estimated that by the year 2050, one in every 85 persons will be affected by AD (Brookmeyer, Johnson, Ziegler-Graham, and Arrighi, 2007). Dementia is tremendously debilitating for affected individuals and places a huge burden on their families and caregivers. Alzheimer's disease has been known for a long time and scientific efforts to understand disease pathophysiology have been massive. Pathological hallmarks of the disease are accumulation of amyloid-β peptide in extracellular space, presence of intracellular tangles of the protein tau, and also neuritic plaques formation (Thakur, Kamboj, Goswami, and Ahuja, 2018). Neuronal cell degeneration in AD is widely distributed in the brain and is seen particularly in the hippocampus, entorhinal cortex, amygdala, deep subcortical nuclei such as the cholinergic basal nuclei, serotonergic dorsal raphe, and noradrenergic locus coeruleus. Moreover, their cortical association regions of the frontal, temporal and parietal cortices are shown to be affected (Kumar and Singh, 2015). The cholinergic hypothesis of AD is among the oldest explanations of disease pathophysiology. The key points of physiological abnormalities in AD are the expression of cholinergic receptors, acetylcholine release, and choline transport. Cholinesterase inhibitors are among the AD treatment front line (Blake, Terry, Plagenhoef, Constantinidis, and Liu, 2017).

Even though amyloid-β accumulation is a hallmark of the disease and several therapeutic strategies such as promoting amyloid clearance, preventing amyloid aggregation, amyloid based immunotherapy, and modulation of secretase enzyme were available but none were successful in demonstrating efficacy to cure or reverse the disease in clinical trials (Anand, Gill, and Mahdi, 2014).

The network disturbance hypothesis is also an important debate in the understanding of AD pathophysiology. Fornix as a part of the Papez circuit is shown to have disease-