CHAPTER 23

Management of Psychiatric Symptoms in Parkinson’s Disease

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A. Introduction

Most reviews of the psychiatric problems seen during the chronic therapy of parkinsonism have focused primarily on nonaffective psychosis and related disorders. This chapter will include both these disorders and alterations in affect or mood.

B. Depression

Depression has been associated with Parkinson’s disease since the original description by James Parkinson. The potential misdiagnosis of parkinsonian akinesia and facial amimia as depression has led some clinicians to caution against the diagnosis of depression on the basis of motor symptoms alone. Nonetheless, from 1922 through the present, clinical evaluations of patients with Parkinson’s disease have found symptoms of depression in 20%-93% of patients interviewed (MINDHAM 1970; PATRICK and LEWY 1922).

Depression has traditionally been divided by etiology into two types: (1) reactive, in which depressive symptoms occur as a result of an identifiable cause; and (2) endogenous, in which symptoms have no identifiable contributory environmental factors. This view of depression has led to the argument that depression in Parkinson’s disease is simply a reaction to chronic disability, and is not an inherent part of the disease process itself.

Undoubtedly, some parkinsonian patients with symptoms of depression are exhibiting a reaction to their chronic disability. If this were the sole etiology of depressive symptoms in Parkinson’s disease, however, symptoms would be expected to be maximal at either the time of diagnosis (when the patient must adjust to the presence of a chronic disease) or at a time when significant disability occurs. Moreover, patients with Parkinson’s disease and patients with other disabilities would be expected to have a similar prevalence of depression. The relationship of depression to the underlying disease process in Parkinson’s disease has been addressed by several investigators.

In one group of studies, the prevalence of depressive symptoms in patients with Parkinson’s disease was compared with a control group of patients with other illness. HORN (1974) compared paraplegics, parkinsonian patients, and persons with no identified illness who were similar to each other in age, sex, and socioeconomic level. He found a significant increase in depressive symp-
toms in the Parkinson's disease groups when compared with either paraplegics or normal subjects. Similar findings have been obtained when comparing patients with Parkinson's disease with those with other disabilities, such as hemiplegia or amputation, as well as patients with medical illnesses. These multiple observations of an increased prevalence of depression in Parkinson's disease as compared with a variety of other disorders suggest that depression may be endogenous rather than reactive in most parkinsonian patients (Robins 1976).

Several other investigators have explored the relationship between the onset of parkinsonian symptoms and the onset of depression. If depressive symptoms were merely a reaction to either the diagnosis or the progressive disability of Parkinson's disease, they would be expected to be maximal around the time of diagnosis. Indeed, in several studies, depressive symptoms occurred frequently during the year after the diagnosis of Parkinson's disease was made, suggesting that reaction to the diagnosis of a chronic illness played some role in depression in many patients with Parkinson's disease (Patrick and Lewy 1922; Mindham 1970). Several other studies, however, using retrospective techniques, have reported depression to occur in 34%-42% of patients before Parkinson's disease was ever diagnosed (Mindham 1970; Mjones 1949; Mayeux et al. 1981). These latter data support the postulate that patients with Parkinson's disease are more likely to experience depression, and that this mood abnormality may be an intrinsic part of the disease process.

There may be a neurochemical rationale for the increased frequency of depressive symptoms in Parkinson's disease. The major chemical deficit in this disease, and the one most clearly related to the motor disability, is loss of dopaminergic neurons in the substantia nigra. Pathologic alterations in Parkinson's disease include all pigmented nuclei, however, and accompanying pharmacologic deficits of all biogenic amines occur. Although dopamine is probably not an important neurotransmitter in most depressive syndromes, decreased activity of other biogenic amine neurotransmitter systems, norepinephrine and serotonin, have frequently been implicated. Both of these neurotransmitters are present in decreased concentration in patients with Parkinson's disease, and depressive symptoms in Parkinson's disease may be the result of these biochemical defects. Mayeux et al. (1984a, 1986a) have reported a correlation between decreased metabolites of serotonin in the spinal fluid and depression in patients with Parkinson's disease. They found that CSF 5-hydroxyindoleacetic acid (5-HIAA) was lower in depressed than nondepressed parkinsonian patients and was related to both psychomotor retardation and loss of self-esteem. An open trial of L-5-hydroxytryptamine (5-HT) alleviated depression in six of seven patients, and this alleviation was associated with increased CSF 5-HIAA in three patients (Mayeux et al. 1986b).

The treatment of depression in Parkinson's disease is currently under active clinical investigation. Levodopa and other dopaminergic agents, while of clear benefit to the primary symptoms of Parkinson's disease, do not alleviate symptoms of depression. For this reason, depression can be the major source of disability for the patient with a good therapeutic response to antiparkinso-