

# Parkinson Disease Treatment in Hospitals and Nursing Facilities: Avoiding Pitfalls

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## CME Activity

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## Abstract

The unique needs of patients with Parkinson disease challenge staff when such patients are admitted to hospitals or nursing facilities. Prolongation of the hospital stay, falls with injuries, fainting, or declining motor function may result from therapeutic misadventures or failure to anticipate common problems. Staff familiarity with Parkinson disease, and especially carbidopa-levodopa dosing and dynamics, may prevent such problems and streamline hospital and nursing home care.

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Parkinson disease (PD) is a common condition, estimated to affect about 1 million people in the United States. People with PD have unique problems and special medication needs. This can be a challenge to clinicians and nurses caring for them when they are hospitalized or admitted to a nursing facility. The consequences of therapeutic misadventures due to staff unfamiliarity with PD are potentially substantial. Prolongation of hospital stays due to PD-related problems is common but often avoidable. Patients with PD who are admitted to nursing facilities may experience major upheaval in their medication

management. Fortunately, a little knowledge can be quite helpful, and that is the focus of this article, which is directed to doctors, mid-level health care professionals, and nurses.

## BACKGROUND

Parkinson disease is a neurodegenerative disorder that notoriously causes slowness (bradykinesia), stiffness (rigidity), and a walking disorder typified by a stooped, shuffling gait with reduced arm swing; most, but not all, patients have a resting tremor. Unrecognized by many clinicians, however, are the nonmotor features of PD, which include anxiety, akathisia, depression,



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dysautonomia, as well as insomnia and other sleep disorders, to name a few.

Neurodegenerative disease with loss of the brain neurotransmitter dopamine underlies many of the motor and nonmotor symptoms of PD. The recognition that replenishment of brain dopamine with levodopa alleviates many PD symptoms was a revolutionary discovery. Despite 4 decades since the discovery of levodopa therapy, it still remains the most efficacious treatment for PD (as carbidopa-levodopa).

With advancing PD, the Lewy body neurodegenerative process extends beyond dopaminergic systems, causing dysautonomia (eg, orthostatic hypotension), cognitive impairment, and motor symptoms refractory to carbidopa-levodopa and related medications. Nonetheless, carbidopa-levodopa remains the foundation of treatment throughout the lifetime of those with PD, necessary for optimal quality of life. Basic knowledge of levodopa dosing and dynamics is crucial when caring for patients with PD who are hospitalized or admitted to nursing facilities.

### CARBIDOPA-LEVODOPA

Dopamine cannot cross the blood-brain barrier, whereas the amino acid precursor levodopa crosses via an amino acid transporter. When levodopa was initially introduced without carbidopa, nausea and vomiting were common adverse events, and extremely high levodopa doses were necessary. These problems were related to the conversion of levodopa to dopamine before entering the brain, ie, in the bloodstream. Prematurely converted dopamine in the circulation is excluded from the brain. However, it crosses in one limited region where the blood-brain barrier is patent: the brainstem nausea/vomiting center. The addition of carbidopa solved that problem. Carbidopa blocks the enzyme that converts levodopa to dopamine; it does this only outside the brain because it does not cross the blood-brain barrier. Carbidopa-levodopa remains the most efficacious drug for symptomatic PD treatment.

The antiparkinsonian effect from carbidopa-levodopa occurs in 2 patterns, termed the *long-duration response* and the *short-duration response*. The levodopa long-duration benefit builds up over about a week with stable dosing. This is typically the primary pattern during the first several years of PD. Patients treated with a stable

carbidopa-levodopa dosing scheme can be early or late with their doses or occasionally even skip a dose without consequence. If the carbidopa-levodopa is stopped, the deterioration may be delayed for days up to a week.

After several years of PD, a second response pattern superimposes, the short-duration response. This presumably occurs because of the progressive loss of dopamine neurons, ie, the cumulative beneficial effect cannot be “stored,” and the responses increasingly mirror the levodopa concentrations in the circulation. For example, those with a short-duration levodopa response may be unable to walk in the morning before taking their medications, only to note a normal gait an hour after carbidopa-levodopa administration. The short-duration effect typically spans 2 to 6 hours, with the response duration typically shortening over time. Higher levodopa doses do not substantially prolong the response. An appropriate treatment strategy for such short-duration responses is to identify the optimal dose (the individual dose producing the best effect 60-90 minutes later) and administer that dose at intervals matching the response duration. It is important to recognize that there is no arbitrary ceiling on the number of doses or tablets per day.<sup>1,2</sup>

Carbidopa-levodopa has few adverse effects, and they tend to reflect short-duration dynamics. Involuntary movements (chorea), known as dyskinesias, represent an excessive levodopa effect, typically appearing about an hour after a dose and lasting 2 to 4 hours. A sudden drop in the standing blood pressure (BP) may be provoked by individual doses of carbidopa-levodopa, with potential for syncope. Because of the short-duration levodopa dynamics, the BP may vary dramatically, depending on the time since the last carbidopa-levodopa dose. Nausea may be induced by levodopa, and like the other levodopa adverse effects, it tends to be time locked to the doses.

### OTHER DRUGS FOR PD

Drugs for the treatment of PD are listed in [Table 1](#); all work through dopamine mechanisms, except for the anticholinergic medications and amantadine. Although amantadine pharmacology was initially linked to dopamine, the primary activity is now recognized

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