dopa adjustments, adjunctive drugs, and ultimately STN DBS may not result in perfect control, but sufficient to lead a reasonable life if other PD-related refractory problems do not intervene. We should shift our focus to problems that most compromise the lives of our patients with PD.¹

Finally, should levodopa be delayed, or the dose limited to avoid motor complications that progressively develop over many years? The treatment goal should be to maintain patients with PD in the mainstreams of their lives, including social, recreational, and occupational. It is certainly acceptable to initiate therapy with an alternative drug to levodopa. However, both clinical trial data and clinical experience indicate that levodopa is the most efficacious medication, and by a substantial extent. Clinical trials indicate that even with aggressive dopamine agonist treatment, few patients can go without levodopa beyond a few years. Thus, if other drugs prove insufficient, levodopa in necessary doses should be considered. If patients backslide into a sedentary existence from undertreatment, it is difficult to resurround them.

Furthermore, there is no evidence that you can save the best levodopa responses for much later. For practical and ethical reasons, this will never be addressed in a clinical trial. However, the early levodopa era experience (in patients with long durations of previously untreated PD)—is relevant; this taught us that the development of levodopa motor complications,⁵ as well as increasing parkinsonism disabili-

ty,⁸ reflects PD progression, rather than levodopa treatment duration.

Many independent clinical series reported substantially increased longevity time-locked to the advent of levodopa therapy.¹ If levodopa allowed patients to live longer, it can't be that bad!

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Disclosure: The author reports no disclosures.

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CORRECTION

A “nema” of hope in the treatment of late-onset nemaline myopathy

In the editorial “A ‘nema’ of hope in the treatment of late-onset nemaline myopathy” (*Neurology*® 2008;71:472–473) by Marinos C. Dalakas and Stephen A. Smith, reference 11 is incorrect. The correct reference follows:

Dalakas MC, Rakocevic G, McElroy B, et al. Alemtuzumab (CAMPATH 1-H) therapy in sporadic inclusion body myositis (sIBM) alters disease progression and suppresses endomysial inflammation. *Ann Neurol* 2007;62(suppl 11):S60–S61. Abstract.

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CORRECTION

Neurology 2008;71;1652
DOI 10.1212/01.wnl.0000339370.39213.01

This information is current as of November 10, 2008

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