Motor response to apomorphine and levodopa in asymmetric Parkinson's disease

The study of the motor response to levodopa in patients with asymmetric parkinsonism is of relevance to the changes in response to pharmacological treatment that evolve with disease progression and to the mechanism of action of levodopa. Pathological and neuroimaging studies show that asymmetry of clinical involvement in Parkinson's disease is caused by asymmetry of degeneration of neurons in the substantia nigra. Assuming that lateralised motor disability affecting upper limb motor function is a consequence of nigral degeneration in the contralateral half of the midbrain, comparison of body sides in patients with asymmetric motor disability allows direct comparison of two different stages in the progression of Parkinson's disease. If the duration of effect of exogenous levodopa is dependent on storage and synaptic release of dopamine in surviving nigral neurons, it should be possible to detect some difference in the duration of motor response between body sides in asymmetric cases.

Rodriguez et al.1 have evaluated asymmetry of motor response to levodopa and apomorphine in 14 cases and concluded that the motor response is more prolonged on the less affected body side. By contrast, our previous study of levodopa motor responses in asymmetric cases showed that the onset and wearing off of the motor response to levodopa occur simultaneously for each side of the body.2 There are differences in patient selection and methods that may be relevant to understanding this discrepancy.

Parkinson's disease often begins with exclusively unilateral involvement and patients then retain obvious motor asymmetry for the first few years of their disease course. Eventually, motor involvement becomes bilateral and clinical asymmetry is more subtle, although differences in upper limb motor function in accordance with first affected body side are still present in many cases. By choosing relatively early cases (untreated patients and patients with moderate motor fluctuations after a mean period of levodopa treatment of 4-9 years), Rodriguez et al were able to study a patient group with obvious asymmetric Parkinsonism. It needs to be recognised, however, that when the amplitude of motor response is relatively low, as it will be in milder cases, differences in motor scores before and after pharmacological agents will not be great and the precise determination of time points of onset and wearing off of motor response will be difficult. In these studies, the estimation of the duration of motor response on the less affected body side is more prone to error because of this factor. The low negative gradients of the lines representing wearing off phases on the motor function v time graphs presented by Rodriguez et al illustrate this difficulty.

By contrast, we studied patients with more advanced disease and well developed motor fluctuations, mean duration of levodopa treatment 9-9 years) who retained clinical asymmetry in accordance with the first affected body side. A large amplitude of motor responses and unequivocal wearing off phases allowed accurate measurement of duration of motor response for each body side, as represented by the graphs of individual patient data published with our results. We showed that such cases with advanced and bilateral Parkinson's disease should still retain asymmetry of neuronal degeneration in the substantia nigra with about 25% difference between nigral neuronal counts on opposite brainstem halves. Readers wishing to confirm that body sides do not fluctuate out of phase in advanced cases can do so by observing any patient with asymmetric disease onset and severe "on-off" motor oscillations.


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