Levodopa has been the basis of treatment for Parkinson’s disease (PD) for more than 40 years. During the last decades researchers have strived to optimize levodopa formulation to minimize side effects, enhance its bioavailability in the central nervous system and finally achieve stable concentrations in the plasma. Current strategies include concomitant administration with inhibitors of dopa-decarboxylase and catechol-O-methyltransferase to prolong the peripheral levodopa half-life and increase bioavailability in the brain. Clinical findings suggest that continuous dopaminergic stimulation that more closely approximates physiological stimulation may delay or prevent the development of motor fluctuations and dyskinesias. However, “around-the-clock” constant rate administration theoretically carries the risk of causing refractory off periods associated with severe immobility and hyperpyrexia. The antidyskinetic effects of continuous dopaminergic stimulation may be related to desensitization which could theoretically lead to a reduction in the amplitude of motor response. It has not yet been proven unequivocally (although some studies hint at it) continuous dopamine stimulation postpone or minimises future genesis of fluctuations in the clinical setting. However, in a very recent trial it was investigated the rotigotine transdermal system for the management of motor function and sleep disturbances in Parkinson’s disease. The most common adverse events were application site reactions (24%), somnolence and hallucination (3% each), nausea and fall (12% each) and dizziness and dyskinesia (11% each). Twelve subjects (14%) discontinued (due to adverse events), most commonly application site reactions and peripheral edema. Hence, the beneficial effects of rotigotine transdermal system on motor function and sleep disturbances were sustained for up to 1 year (Trenkwalder et al. 2012). In another recent study the most common adverse event seen with transdermal delivery of rotigotine are local skin reactions, which may lead to a treatment discontinuation in approximately 8% of patients (Sprenger and Powere 2012).

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