Though controlled release forms of levodopa are available, they do not prevent or provide significantly prolonged benefit for motor complications [2,3]. This creates space for the development of infusion therapies” Prakash et al (2019).

Extract:

Motor complications result from the combination of multiple underlying factors. Reduced striatal dopaminergic terminal function plays a pivotal role in the variable response to levodopa therapy. With advancing dopaminergic terminal and dopamine transporter loss, striatal neurons slowly lose the ability to store dopamine in terminal vesicles. This, in turn, affects the neuron’s ability to buffer the synaptic dopamine levels with exogenous levodopa use [1, 2].

Additionally, intermittent oral levodopa leads to pulsatile stimulation of the degenerating striatal neurons. Such pulsatile delivery leads to changes at the receptor level that contribute towards fluctuations in clinical response. The oral levodopa bioavailability is also affected by its short half-life and unpredictable absorption. Other factors include delayed gastric emptying and impaired absorption across the intestine and blood-brain barrier [1, 2].

One potential solution aims to develop therapies that could deliver steady or continuous dopaminergic stimulation at the striatal level. Though controlled release forms of levodopa are available, they do not prevent or provide significantly prolonged benefit for motor complications [2,3]. This creates space for the development of infusion therapies. Advanced therapies currently available on the market include Levodopa-Carbidopa intestinal gel (LCIG), Deep Brain Stimulation (DBS) and Apomorphine pump.

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