



Levodopa-induced dyskinesias in Parkinson's disease : current knowledge and future scenarios /

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Monografía

Levodopa-induced dyskinesias (LID) are abnormal movements that occur during the period of maximal benefit of parkinsonian symptoms when levodopa concentration in the brain is highest. LID are mainly choreic in nature and represent a very common problem in up to 80% of Parkinson's disease (PD) patients treated with levodopa. Current knowledge highlights the critical involvement of the striato-thalamo-cortical pathways, which are characterized by abnormal physiological overactivity. However, despite many recent advances in genetic and pharmacological fields, the pathophysiological mechanisms underlying LID are still a matter of debate. In the last few years, advances in the neurophysiological and neuroimaging fields have provided alternative scenarios for understanding the neurobiological mechanisms of LID. Indeed, several lines of evidence support the notion that other structures, outside traditional striato-thalamo-cortical pathways, are strongly involved in the LID. In particular, the cerebello-thalamic circuitry as well as intra-cortical connections between the premotor cortex (particularly supplementary motor area) and the inferior frontal cortex, would seem to play a key role in the dysfunctional pathophysiological model of LID. This topic aims to pool the most recent advances in the phenomenology and pathophysiology of levodopa-induced dyskinesias. We aim to compile original research papers, review articles, technical reports and commentaries that cover this topic broadly. Researchers with interest in clinical aspects (and particularly management strategies, e.g. deep-brain stimulation), neuroimaging (using either functional or structural MRI approaches), neurophysiology (transcranial magnetic stimulation (TMS)) of LID and other hyperkinetic movement disorders (i.e., dystonia, Huntington's disease, Gilles de la Tourette's syndrome), are encouraged to contribute to this topic. Again, articles concerning animal models of PD will be particularly appreciated.

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