Pain in Parkinson’s disease
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Pain is defined as an unpleasant or distressing sensory experience and has been recognized as a non-motor symptom in Parkinson’s disease (PD). Pain may occur in 40-50% of patients according to the sparse literature on this topic. It has also been reported that PD patients suffer from pain e twice as often as age-matched controls. In a minority of individuals it is even more prominent than motor disturbances. Pain in PD can be subdivided into several categories. The most common form of pain is due to the increase in muscle tone (rigidity), a finding which is similar to another form of increased muscle tone (spasticity) in multiple sclerosis. An increase in rigidity is correlated with decreased dopamine stimulation. Therefore many PD patients complain of pain when their levodopa levels are low, for example in the wearing-off or end of dose period (diphasic or early morning dyskinesia and others). High levodopa levels may, however, also induce pain which is sometimes the case in peak-dose dyskinesia. Apart from its association with these levodopa levels, pain can also be subclassified according to its quality. There is musculoskeletal pain, neuritic or radicular pain and dystonia-associated pain, akathitic discomfort, central and peripheral pain. Some PD patients present with genital and oral pain such as burning mouth. No other definable organic causes of pain apart from PD could be found in these patients PD. It is therefore tempting to speculate that dopamine has central analgesic properties in such patients. Furthermore, it could be demonstrated that the intake of levodopa increased pain threshold. A PET study showed abnormalities in the insular and prefrontal region and in the anterior cingulated cortexes of PD patients with pain. The introduction of levodopa normalised this condition. Sometimes, pain is associated with panic attacks and is when resolves the underlying cause of the pain is treated. The first step should be a careful exploration of the patient with respect to levodopa level-associated pain. In these patients, dopaminergic treatment, preferentially continuous dopaminergic stimulation, should be initiated. Moreover, there is also evidence for improvement of PD pain by cannabinoid-receptor antagonists, amantadine and opioids, but my own experience with non-steroid analgesics is not very encouraging.