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Chapter 1

Introduction

Parkinson's disease

Parkinson's disease is a chronic progressive neurodegenerative disease affecting the motor, autonomic, cognitive, affective and sensory systems.[1] The motor symptoms are characterised by the progression of tremor, rigidity, bradykinesia and postural disturbances. Patients may suffer from drooling, excessive sweating, chronic constipation, and sleep disturbances as a result of autonomic nervous system dysfunction. The disorder mostly begins in later life with an average onset of 60 years of age. The prevalence of Parkinson's disease ranges from 84 to 164 per 100,000.[2]

The aetiology of Parkinson's disease is still unknown. Characteristic neuropathological findings in Parkinson's disease are neuronal loss, depigmentation of pigmented brainstem structures, and the presence of Lewy bodies.[3] The core symptoms are caused by dysfunction of the dopaminergic neurotransmission in the *substantia nigra-striatum system* due to degeneration of dopamine producing neurons. The substantia nigra and the striatum (caudate nucleus and putamen), together with the claustrum, globus pallidus, subthalamic nucleus, and the neurones of the ventral tegmental area form the basal ganglia. They were usually regarded as components of several largely segregated circuits serving motor, oculomotor, and cognitive functions.[4,5] It is now apparent that the basal ganglia system is a complex and widely distributed neuronal network comprising a markedly branched axon collateral network system by which the output nuclei of the basal ganglia can be influenced in multiple ways.[6]

There is still no cure for Parkinson's disease. The symptomatic treatment predominantly consists of adding-on the dopamine deficiency with levodopa (plus a peripheral decarboxylase inhibitor) or — and often in combination with — a dopamine agonist. These can bring about an important improvement of bradykinesia and rigidity, but they are less effective on tremor, and postural disturbances.

After 3 to 5 years of pharmacological treatment, problems with therapy may arise. The patient experiences variations in symptoms during the day and this necessitates levodopa dose adjustments. Subsequently, adverse effects may emerge, such as levodopa-induced involuntary movements or psychiatric complications, consisting of hallucinations and paranoid delusions. In the beginning, involuntary movements occur in a temporal pattern in relation to levodopa administration and they can be temporarily reduced with levodopa dose adjustments. This phenomenon of episodes with good levodopa effect — often with involuntary movements — alternating with episodes of re-emerging parkinsonian symptoms is called response fluctuations. With advancing disease, the response fluctuations frequently become abrupt oscillations in motor state, bear no clear temporal relationship with levodopa dosing and become unpredictable. This form of response fluctuations is referred to as 'on-off' fluctuations.

After 4 to 6 years of levodopa therapy 40 percent of patients experience response fluctuations, which increases to 70 percent after 9 or more years of levodopa therapy. Eleven to 15 years after disease onset, 55 percent of patients are severely disabled or dead.[7,8]

Stereotactic Pallidotomy

Stereotactic pallidotomy is a functional neurosurgical procedure during which a lesion is made in the globus pallidus by heating the tip of an electrode that — through a burr hole in the skull — has been placed in the globus pallidus. Stereotactic methods are used to localise the posteroventral part of the globus pallidus.[9,10]

In 1948, stereotactic pallidotomy was performed for the first time in a patient with Huntington's disease. Soon after that, the procedure was carried out for tremor and rigidity in patients with Parkinson's disease based on the work of Meyers and Fénelon.[11-13] By the mid-fifties the target for functional stereotactic neurosurgery in Parkinson's disease gradually moved to the ventrolateral thalamus, because the thalamus seemed to be a better target for tremor and was associated with less adverse effects.[14,15] After the introduction of levodopa in 1967, the number of stereotactic operations for Parkinson's disease declined dramatically and, occasionally, thalamotomy was done for tremor.

The recent revival of pallidotomy has occurred as a result of various factors. In the 1980's, there was an increasing awareness of the limitations and side effects of levodopa and dopaminergic agonists. In 1992 Laitinen et al. reintroduced pallidotomy reporting the results of 38 patients with advanced Parkinson's disease and posteroventral pallidotomy.[16] Their results were excellent; 92 percent of the patients experienced great alleviation of rigidity and hypokinesia; and 81 percent had complete or almost complete relief of tremor contralateral to the pallidotomy. Furthermore, involuntary movements — whether or not levodopa-induced — were reduced substantially by the operation. At approximately the same time, anatomic and physiological studies provided a new model for the organisation and function of the basal ganglia,[4] and although the model does not fully predict the effects of pallidotomy,[17] it provided a strong rationale for surgical intervention in the basal ganglia.

Aim and outlines

The main question of the thesis is: do patients with advanced Parkinson's disease benefit from stereotactic pallidotomy? Therefore, we started a retrospective study on the effects of unilateral pallidotomy in patients who had undergone surgery in our hospital (chapter 2). Based on these results

and the outcomes of several other cohort studies,[18,19] we decided to perform a randomised, single blind, multicentre trial to assess the efficacy of unilateral pallidotomy in advanced Parkinson's disease. Patients were randomised to unilateral pallidotomy within 1 month or to pallidotomy after the primary outcome assessment, 6 months later (chapter 3). In the same trial, cognitive and behavioural effects were studied (chapter 4). Eventually, 32 of the 37 patients enrolled in the trial had undergone a unilateral pallidotomy. In chapter 5, a follow-up of these patients is reported. Nineteen patients were followed for 12 months after surgery and 13 for 6 months. We investigated whether or not a relationship existed between lesion location and clinical outcome, and searched for preoperative patient characteristics predictive for good outcome. We evaluated the effects of bilateral pallidotomy in a retrospective study of patients who had undergone bilateral surgery in our hospital (chapter 6). Chapter 7 includes a systematic review dealing with the adverse effects of pallidotomy. The place of stereotactic pallidotomy in the treatment of advanced Parkinson's disease is discussed in chapter 8. A summary in English and Dutch concludes this thesis.

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