ECT in Parkinson’s disease. Changes in motor symptoms, monoamine metabolites and neuropeptides

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Summary. Electroconvulsive therapy (ECT) was given to 16 non-depressed, non-demented patients with advanced Parkinson’s disease (PD). In all the patients an antiparkinsonian effect was seen, lasting for 18 months in one patient, 3–5 months in seven patients, and a few days to four weeks in eight patients. After ECT the levels of homovanillic acid and neuropeptide Y in cerebrospinal fluid (CSF) were significantly increased. The eight patients with long lasting motor improvement after ECT had significantly lower CSF-3-methoxy-4-hydroxyphenylglycol compared to the group with short lasting improvement.

Five patients developed transitory mental confusion after ECT. In these patients, and in no others, a high albumin-ratio was found already before ECT was given – an indication of blood CSF barrier damage.

Our results suggest that ECT is valuable in patients with drug refractory PD or PD with intolerance to antiparkinsonian drugs.

Keywords: Parkinson’s disease, electroconvulsive therapy, monoamine metabolites, neuropeptides.

Introduction

The introduction of levodopa (L-dopa) therapy has increased life quality and expectancy but not the ultimate prognosis in patients with Parkinson’s disease (PD). L-dopa has contributed to a higher prevalence of older, chronically disabled patients, many of whom also being resistant or intolerant to drug therapy (Abrams, 1989). Therapeutic methods such as new drugs, stereotactic surgery, surgical implant of foetal neural or autologous adrenal medullary tissues into the brain have failed to solve the problem of managing the disease. In the literature there were at the time of writing some 35 reports relating to PD and ECT, mostly case reports. Only six report on effects of ECT on PD without psychiatric comorbidity (Faber and Trimble, 1991), one of them as a
single case report and four as open studies of consecutive PD patients given ECT for severe symptomatology. Members of our group performed the sixth study including 11 patients in a double blind study with sham ECT as a control (Andersen et al., 1987). Altogether the six reports comprise 34 patients of whom 22 patients were reported as showing improvements up to a maximum of 41 weeks. Of all 35 reports on ECT and PD, only one failed to show some positive response in PD symptoms (Ward et al., 1980).

The aims of the present study were to

- study monoamine and neuropeptide levels in CSF before and after ECT
- look for biochemical and clinical predictors for a long lasting antiparkinsonian effect
- estimate the frequency of patients showing an antiparkinsonian response to ECT
- estimate the duration of the antiparkinsonian effect of ECT.

**Material and methods**

The diagnosis of PD was based on the following criteria, all who had to be fulfilled:

- History or presence of at least one of three classical signs – tremor, rigidity, and hypokinesia.
- History of insidious progression.
- No history of pharmacologically induced parkinsonism.
- Absence of atypical neurological signs.
- A significant improvement of PD symptoms on L-dopa treatment in adequate dosage.

Only non-depressed, non-demented patients with advanced PD were included in the study. All had disabling parkinsonian symptoms during long term L-dopa treatment (i.e. drug refractory patients) or inability of accomplishing antiparkinsonian pharmacotherapy due to side effects. Sixteen patients, seven women and nine men, aged 70.3 ± 8.5 (M ± SD) (range 60–87 years), were included in the study. The duration of the disease was 16.9 ± 8.6 years and the duration of L-dopa treatment 10.0 ± 5.1 years. Levodopa was given as monotherapy to five patients and in combination with bromocriptine, selegiline or orphenadinum to eight, three and one patients, respectively. Levodopa therapy had been withdrawn due to side effects in two patients more than one year before the study. The patients had been on a constant pharmacotherapy, considered optimal, for at least one month before ECT was given and this therapy was kept unchanged when the ECT sessions started. Before ECT was given, the Hoehn and Yahr (Hoehn and Yahr, 1967) clinical stage was 3.6 ± 0.6 (range 3–5) and the Webster (Webster, 1968) scores during the best part of the day 15 ± 3.4 (range 9–21). The patients on levodopa therapy all had fluctuating symptomatology but marked “on-off” was seen only in two of them. Repeated ratings were performed when the patients were in a favourable state, and the “best” ratings and results are reported. Before the first ECT the full Gottfries-Bråne-Steen scale (GBS) (Gottfries et al., 1982) was used to exclude dementia and the Montgomery-Asberg-Depression Rating Scale (Montgomery and Asberg, 1979) to exclude mental depression.

The study was approved by the Ethical Committee of the Faculty of Health Sciences, Linköping University. All patients had given their informed consent to participate.

**ECT procedure**

The patients were premedicated with atropine sulphate (0.5 mg subcutaneously) 0.5 hour before the ECT. All treatments were given at 8–10 a.m. Light anaesthesia was given by