It is widely recognised that long-term neuroleptic treatment can cause tardive dyskinesia. This syndrome is thought to be due to hypersensitivity of nigrostriatal dopamine receptors. It has been suggested (Davis & Rosenberg, 1979) that an analogous syndrome of tardive psychosis might be caused by supersensitivity of mesolimbic dopamine receptors following long-term neuroleptic treatment. This syndrome, like tardive dyskinesia, could be apparent during drug treatment but would become more obvious after withdrawing drugs. Patients alleged to have this syndrome have been described clinically (Chouinard & Jones, 1980).

Curson et al (Journal, May 1985, 146 474–480) in their follow-up study of the MRC fluphenazine maintenance trial in schizophrenia reported the unexpected finding that patients who had spent the greatest percentage of their time taking neuroleptic drugs had the highest relapse rate. In a recent statistical overview of clinical trials of maintenance therapy with fluphenazine, Teicher & Baldessarini (1985) suggested that doses of the order of 50 mg fortnightly are "antitherapeutic" for 50% of patients, and that much lower doses are more effective. Both of these observations are consistent with the hypothesis that high exposure to fluphenazine causes tardive psychosis in some patients. The great majority of patients included in placebo-controlled studies of long-term neuroleptic treatment, including those in the MRC study, had received neuroleptic drugs before entering the trial. Some of these patients could have developed tardive psychosis even before entering the studies.

Whilst the existence of tardive psychosis is not yet definitely proven, it is clearly a possibility which should be kept in mind in the interpretation of research studies of maintenance neuroleptic treatment.

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References