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LETTER TO THE EDITOR

Paroxetine efficacy in stuttering treatment

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Stuttering is usually developmental, with onset at 4 to 5 years of age, and sometime may have a genetic component; among the possible cause of stuttering, response to conflicts, fears, neurosis, organic models, and learning models have been proposed (Andrews et al., 1983). Stuttering can be neurogenic (or acquired) as a result of stroke, head injury and degenerative or metabolic brain damage (Grant et al., 1999).

A variety of treatments for stuttering have been proposed both pharmacological and non-pharmacological (Kaplan et al., 1994). Controlled studies showed consistent stuttering improvement only with haloperidol (Brady, 1991), and to a lesser extent with clomipramine (Gordon et al., 1995). Paroxetine (a phenyl-piperidine derivate, and a potent and selective serotonin reuptake blocker) has been reported to be effective in cases of acquired stuttering (Schreiber and Pick, 1997; Turgut et al., 2002), and in stuttering with comorbid obsessive–compulsive disorder (OCD) (Murray and Newman, 1997). Here we describe the successful treatment with paroxetine of a patient with stuttering associated with OCD.

Case report

Mr B was a white, 30-year-old single man with a history of prominent stuttering since adolescence. He presented a speech dysfluency characterized by part-word repetitions, audible and silent prolongations, with difficulty in starting and sustaining air flow. When he had entered cognitive–behavioral psychotherapeutic treatment (CBT) 3 years earlier, he had been complaining of anxiety, intrusive thoughts, and poor social and scholastic functioning. At that time he was treated with CBT, which resulted in some decrease in the symptoms. At the time of his current presentation he had remained in psychotherapy. He complained of distress related to intrusive thoughts, which were persistent and difficult to suppress, and were interfering with his ability to concentrate in studying (he was an university student). Mr B was slowing down in taking exams and reduced his social contacts. Psychological evaluation and psychiatric consultation resulted in stuttering associated with OCD. Physical examination was normal; there was no history of brain injury, brain surgery or neurological disease, substance abuse or dependence and active medical conditions.

Treatment with, 20 mg/d paroxetine, was initiated, and weekly CBT continued. Treatment with paroxetine was chosen with the aim of treating the OCD symptoms and considering the possibility of improving stuttering as suggested from previous observations (Murray and Newman, 1997; Schreiber and Pick, 1997; Turgut et al., 2002). In this case the use of drugs with demonstrated efficacy for stuttering in controlled studies, like haloperidol (Brady, 1991) or clomipramine (Gordon et al., 1995) would not be of choice in OCD (haloperidol) or have less tolerability (clomipramine). After 1 month of paroxetine treatment, Mr B spontaneously noted a marked decrease in his stuttering. His therapist also observed the decrease in stuttering during their weekly sessions. Mr B also experienced a decrease in the frequency and intensity of his intrusive thoughts. After the first month, the paroxetine dose was increased to 30 mg/d in an attempt to further reduce residual stuttering and OCD symptoms. One year later, while still taking 30 mg/d, Mr B displayed marked improvement: the frequency and persistence of his stuttering and intrusive thoughts had decreased, and his social and scholastic functioning had substantially improved.

Discussion

Our case was improved after paroxetine treatment. This is further evidence that paroxetine seems effective in the treatment of stuttering in comorbidity with OCD, as previously reported (Murray and Newman, 1997). One possibility is that in some OCD cases stuttering could be a compulsive symptom responding to paroxetine. On the other hand, case reports on acquired stuttering (Schreiber and Pick, 1997; Turgut et al., 2002) also showed favourable response to drug treatment with paroxetine. The paroxetine efficacy in primary and secondary stuttering suggests that both may respond to the same pharmacological mechanism, even presenting different clinical characteristics (Grant et al., 1999). It has been suggested that paroxetine

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may produce stuttering improvement by a serotonin-mediated indirect dopaminergic effect (Schreiber and Pick, 1997; Turgut et al., 2002). In fact, the demonstrated efficacy of haloperidol (Brady, 1991) and paroxetine (Murray and Newman, 1997; Schreiber and Pick, 1997; Turgut et al., 2002) in stuttering supports the hypothesis of a possible dopaminergic–serotonergic interaction in the production of the symptomatological improvement.

Paroxetine may have a secondary dopaminergic effect via 5-HT_{2A} receptors that are specifically associated with dorsal raphe nucleus terminals (Deakin, 1991), which correspond closely to the distribution of dopamine terminals in the brain and show interactions with the nigrostriatal and the mesolimbic dopaminergic circuits (Hyttel and Larsen, 1985).

The possible influence of specific brain circuit dysfunction in the pathogenesis of primary stuttering should be investigated. Further controlled studies are required to establish the potential therapeutic effect of SSRIs in stuttering.

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