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Tardive Dyskinesia

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ships, professional activities, etc. To get a grasp of all the possible consequences of dyskinesias, it appears very helpful to imagine oneself suffering from them.

Functional Impairment

Whereas even mild dyskinetic movements can cause psychosocial impairment, significant functional impairment and physical complications generally occur only with more severe forms of the disorder; physical disability may also be more frequent in dystonic than in classic tardive dyskinesia.

Problems with dentures are very common among patients exhibiting dyskinesias of the tongue and jaw. Many patients state that their dentures do not fit despite repeated adaptations by the dentist. Some patients even refuse to continue wearing dentures as they are afraid they might inadvertently swallow them. Continuous tongue movements may also cause traumatic ulcerations of the tongue and lips.

Impaired swallowing may be more frequent in patients with tardive dyskinesia than has been previously recognized (MASSENGIL & NASHOLD, 1970, CRAIG et al., 1982). Tongue, jaw and lip movements may interfere with articulation and result in slurred speech. Occasionally, spastic dysphonia has been observed in dyskinetic patients.

Dyskinesias of the diaphragm, too, may interfere with speech and give rise to grunting and groaning, and to an irregular, arrhythmic pattern of speech. Dyskinesias of the diaphragm may also lead to shortness of breath and – very rarely – to respiratory alkalosis.

Gastrointestinal disturbances (vomiting, aerophagia, episodic retching) as a part of the tardive dyskinesia syndrome have been described in case reports (CASEY & RABINS, 1978).

Dyskinesia, particularly dystonia, may severely interfere with motor skills. Impairment ranges from a reduced ability to execute fine motor tasks to an inability of patients to walk without aid. Gait disturbances can lead to falls and injuries; prolonged dystonic contractions may cause fixed postures. In some cases, the motor restlessness caused by tardive dyskinesia can result in weight loss (GARDOS et al., 1977 a).

In several cross-sectional epidemiological surveys (JESTE & WYATT, 1981; HAAG et al., 1987), there was a decrease in tardive dyskinesia prevalence in patients older than 70 years, possibly pointing to a reduced life-span of dyskinetic individuals. This interpretation is supported by findings of prospective studies by METHA et al. (1978) and McCLELLAND et al. (1986). One possible explanation for the

Table 8. Psychosocial and physical impairment by tardive dyskinesia.

Psychosocial
– feeling of embarrassment and guilt; shame; depression; social withdrawal
– handicapped in personal relationships, stigmatization by strikingly abnormal movements
– difficulty in professional activities
Physical
– denture problems, ulceration of the tongue, difficulty in swallowing
– dysarthria, spastic dysphonia
– respiratory disturbances
– gastrointestinal disturbances
– difficulty in motor function (fine motor skills, walking etc.); falls and injuries
– fixed postures
– increased mortality?

reported statistical association of tardive dyskinesia with increased mortality would be a direct causal connection (e.g. from an increased incidence of deaths by choking). Another possibility – and probably the more important one – is the existence of a common variable predisposing to both, tardive dyskinesia and increased mortality.

THE NATURAL COURSE OF TARDIVE DYSKINESIA

Initially, it was believed that tardive dyskinesia develops only after a minimum period of two or more years of continued neuroleptic exposure. But there is only little empirical evidence supporting the idea of such a minimum threshold for tardive dyskinesia development. In a prospective study, KANE and colleagues (1984) showed that the yearly incidence of tardive dyskinesia stayed fairly constant for the first seven years of treatment. There are case reports of an occurrence of tardive dyskinesia after as little as three months and sometimes after even shorter courses of treatment (CHOUINARD & JONES, 1979).

The study by KANE et al. cited above suggests that there may be important differences between cases with an early onset (less than two years of neuroleptic treatment) and cases with a later onset (after more than two years), since early-onset tardive dyskinesia was found to be associated with significantly lower maximum neuroleptic doses than late-onset tardive dyskinesia. This might indicate that early onset of