Movement Disorders
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Letters to the Editor

Treatment of Palatal Myoclonus with Sumatriptan

To the Editor:

The pathophysiology of essential palatal myoclonus (EPM), which results in rhythmic, involuntary contractions of trigeminally innervated tensor veli palatini, is unknown. Our report of abolishment of EPM by treatment with the 5-hydroxytryptamine 1B/D (5-$HT_{1B/D}$) receptor agonist, sumatriptan, suggests a role for serotonin receptors in the trigeminal system in this disorder (1). In contrast to the marked effect of sumatriptan in EPM, we found no effect of sumatriptan in a case of symptomatic palatal myoclonus, which is produced by rhythmic, involuntary contractions of levator veli palatini, innervated by the facial and the ambiguus nerves. Studies of human and bovine cerebral blood vessels support localization of 5-HT_{1D} receptors to trigeminal neurons associated with these vessels (2).

In light of the hypothesis that 5-$HT_{1B/D}$ receptors in the trigeminal system may attenuate EPM, we note with great interest the recent report by Hoskin et al. (3) demonstrating that sumatriptan directly reduces transmission in trigeminal afferents in an animal model of migraine. Their studies show that the attenuation of trigeminal transmission by sumatriptan does not require mechanical distention of the vasculature, nor does it require sumatriptan to cross the blood–brain barrier. A similar effect of sumatriptan on 5-$HT_{1B/D}$ receptors associated with trigeminal innervation of tensor veli palatini or with its afferent sensory input could explain the rapid therapeutic effect of sumatriptan observed in EPM.

Studies of additional EPM patients, and of animal models of palatal myoclonus, are required to establish whether sumatriptan reproducibly attenuates EPM, and whether the effects of sumatriptan involve 5-$HT_{1B/D}$ receptors on afferent or efferent neurons in the trigeminal system.

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Movement Disorders in Multiple Sclerosis

To the Editor:

We feel encouraged by the review by Tranchant et al. (1) and the letter to the editor by Burn and Cartlidge (2) to report the occurrence of Lewy bodies (LBs) in the brainstem of three of 17 multiple sclerosis (MS) cases. A total of 16 patients had donated their brain and spinal cord to the Netherlands Brain Bank. None of the patients had parkinsonian symptoms. In a 17th case, the man had died of temporal lobe metastasis of a large cell pulmonary carcinoma complicated by subtentorial herniation: large sharply defined demyelinated lesions were found around the occipital horns, and small lesions in the cerebellum and other locations. Clinically, he never had symptoms pointing to MS or Parkinson's disease. In three cases, including the latter, LBs were found (Table 1).

Table 1 shows that in half of the cases with demyelinated plaques in which the deceased were between the ages 70 and 81, LBs were also found. Of course, this percentage may be mere chance, because LBs occur with increasing number in the elderly (3). It may also point to a causal or promoting relationship between MS and Parkinson's disease. This underlines the statement of Tranchant et al. that magnetic resonance imaging and pathological studies of MS patients with (but, in our opinion, also without) parkinsonian symptoms should be reported. Not only could critically placed MS lesions be detected, but also, by neuropathology, LBs and nigral cell loss.

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TABLE 1. *Substantia nigra*

Sex	Age at death	SN/LC	Remarks
			Lung carcinoma
♂	80	+/+	Brain metastasis
♀	72	−/+	MS, 28 years
♀	71	+/+	MS, 42 years
11♀, 3♂	81, 78, 70; 34–60	−/−	

+, −, Lewy bodies present or not present in substantial nigra (SN) or locus caeruleus (LC).

Paroxysmal Kinesigenic Dystonia Associated with a Medullary Hemorrhage

To the Editor:

Riley (1) presents an interesting case of paroxysmal kinesigenic dystonia associated with a remote medullary hemorrhage. The lesion appears to be juxtaposed to the dorsal surface of the left inferior olivary complex. Olivocerebellar climbing fibers traverse the medial lemnisci before passing through and above the contralateral principal olivary nucleus. Climbing fibers exert a powerful excitatory influence on Purkinje cells in cerebellar cortex. 3-acetylpyridine (3-AP) is a neurotoxin that destroys inferior olivary neurons (2). Rats treated with 3-AP develop generalized dystonia (3). Abnormal climbing fiber activity is also a critical feature of the genetically dystonic rat's motor syndrome (4). It is conceivable that, in Riley's patient, destruction or irritation of olivocerebellar pathways leads to paroxysms of abnormal cerebellar output and dystonia. Of course, reasonable alternative hypotheses may exist.

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4. LeDoux MS, Lorden JF. Single-unit activity of cerebellar Purkinje cells in the awake genetically dystonic rat. *Soc Neurosci Abs* 1996; 22:1629.

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