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Letter to the Editor

Treatment of Palatal Myoclonus With Sumatriptan

We read with interest the recent report by Scott and colleagues[1] describing the case of a patient with essential palatal myoclonus (EPM) who was successfully treated with either oral or subcutaneous administration of sumatriptan.[1] The authors also pointed out that, in contrast to this EPM patient, a small oral dosage of sumatriptan was ineffective in a second patient with symptomatic palatal myoclonus (SPM).[1] Based on these findings, Scott and colleagues questioned whether EPM and SPM differ pharmacologically or whether the failure of sumatriptan in SPM may simply have been related to an inadequately small dosage.[1]

We recently reported in *Movement Disorders*[2] the case of a patient who developed palatal myoclonus (PM) (also called palatal tremor)[2,3] while he had a primary intestinal lymphoma. A remarkable feature was that PM was absent at rest and triggered only by action, such as attempts at speaking or swallowing. Blink reflex study revealed an enhanced recovery curve after stimulation of either side. Magnetic resonance imaging findings in the brain were strongly indicative of hypertrophic degeneration of the inferior olivary nuclei, which are presumed to be the pacemaker of SPM.[3] In that patient, we tested the effect of several drugs—including alcohol, clonazepam, sumatriptan, and valproate—on both blink reflex and PM. Only clonazepam was effective in reducing action PM and normalizing the blink reflex recovery curve as well.[2] Most importantly, sumatriptan had no clinical or neurophysiological effect, even if an adequate (6 mg) subcutaneous dosage was injected.

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All these findings suggest that sumatriptan does not affect SPM regardless of the dosage and the character of myoclonus (that is rest or action PM). Therefore, the hypothesis that SPM and EPM represent pharmacologically distinct disorders seems more likely. Accordingly, an exhaustive review of PM patients provides evidence that EPM and SPM are two separate clinical entities[4] arising from different pathophysiological mechanisms.[3,4] In SPM, there is presumably a brain stem-release phenomenon resulting from deactivation of GABAergic neurons within the olivary nuclei.[3] On the other hand, the inferior olive does not play a role in EPM, whose pathophysiological characteristics are still unknown.[3] As stated by Scott and colleagues,[1] the striking efficacy of sumatriptan may support a major role of serotonin receptors in the pathogenesis of EPM.

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