legs syndrome (RLS) and their relation to disturbed sleep. Another prospective study of RLS and sleep and the involvement of the thalamus[2] showed that zolpidem, which is known to reduce thalamic GABA,[3] led to striking symptomatic improvement.

We saw 2 patients with medication-induced akathisia; the first patient took haloperidol and the other venlafaxine. Both patients showed marked response to zolpidem prescribed incidentally for insomnia. They elected to stay on their psychotropic medication due to the beneficial effect on their mental state.

Akathisia and RLS are clinically indistinguishable.[4] The mainstay of treatment for akathisia is withdrawal of the offending agent. However, in some cases, the illness may be resistant to other medications and so the offending agent must be continued. Pharmacologic treatments for akathisia are of limited effectiveness and the evidence base is poor.[5]

Together with the authors' observations, these findings provide support for a broader model of akathisia and RLS, incorporating the thalamus and GABA/glutamate system.

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SUBCORTICAL EPILEPSY?

Joe Verghese, Isabelle Rapin, Bronx, NY: Badawy et al.[1] described the cerebellar origin of seizures and explored why experts disagree about seizure origin. It is clear that current findings lack detail, do not include determinations between myoclonus and epilepsy, and many cerebellar pathologies are outlined.

We described a case in which myoclonus correlated with selective dentate nucleus degeneration.[2] We considered cerebellar seizures but offered an alternate explanation for previously described symptomatology.[1] We followed this child from age 2 to her death at age 6. She had type III Gaucher disease with splenomegaly, stridor, ataxia, oculomotor apraxia, and disabling spontaneous and action myoclonus. She had myoclonic jerks causing falls and episodes of myoclonic status that superficially resembled generalized seizures but without alteration of consciousness or postictal state. Intelligence was preserved. Two EEGs and CT scans at ages 3 and 4 were normal. At autopsy, there was no neuronal, perivascular, or meningeal storage but dentate nuclei neurons had degenerated without other brainstem abnormalities.[2] The myoclonus may have resulted from selective toxic effects on dentate neurons of psychosine, a neurotoxin produced by an alternate pathway to the blocked glucosylceramide degradation.[3] More detailed electrophysiologic study and well-defined neuropathology are needed before events are accepted as epileptic seizures arising from cerebellum.

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Author disclosures are available upon request (journal@neurology.org).

Subcortical epilepsy?

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Neurology 2014;82;373
DOI 10.1212/01.wnl.0000443818.22087.0d