Pediatric Neurology Part I: Chapter 67. Lennox-Gastaut syndrome and epilepsy with myoclonic-astatic seizures (Handbook of Clinical Neurology)

Anna Kaminska, Hirokazu Oguni

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Among nonsymptomatic epilepsies exhibiting several types of generalized seizures in children two syndromes were progressively identified: epilepsy with myoclonic–astatic seizures (MAE) and nonsymptomatic Lennox–Gastaut syndrome (LGS). Various approaches based on etiology, electroclinical semiology, and mathematical analysis have progressively helped to distinguish these two conditions. Both conditions preferentially affect boys. The course is stereotyped in MAE, characterized by progressive worsening of epilepsy, usual pharmacoresistance at onset and tonic–clonic seizures, myoclonus and frequent episodes of myoclonic status epilepticus. EEG shows 3Hz spike wave bursts characteristic of idiopathic generalized epilepsy together with slowing of the tracing. In LGS, major seizures are mainly atypical absences and tonic seizures with 0.5–2Hz slow spike-waves and eventually focal anomalies. Prognosis in both syndromes ranges from recovery without sequelae to pharmacoresistant epilepsy that has improved over the past 2 decades with the new generation antiepileptic compounds. Iatrogenic factors may contribute to the poor prognosis, mainly in MAE. Pathophysiology remains speculative for both syndromes: although both share factors of brain maturation, MAE is probably mainly related to genetic predisposition whereas LGS results from some unidentified cortical brain malformation. In unfavorable cases, there may therefore be a continuum between both syndromes. They need to be distinguished from other epilepsy syndromes and inborn errors of metabolism that begin in the same age range: atypical idiopathic benign epilepsy, frontal lobe epilepsy with secondary bisynchrony, ring chromosome 20, ceroid lipofuscinosis, and nonsymptomatic late-onset spasms.

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