Pediatric Neurology Part I: Chapter 32. Angelman syndrome (Handbook of Clinical Neurology)

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Angelman syndrome combines severe mental retardation, epilepsy, ataxia, speech impairment, and unique behavior with happy demeanor, laughing, short attention span, hyperactivity, and sleep disturbance. Occurrence has been calculated at 1:20000 to 1:12000 constituting about 6% of all children with severe mental retardation and epilepsy. The physical “prototype” includes microcephaly with flat neck, fair skin and hair, wide-spaced teeth, and open mouth with tongue protrusion. Epilepsy is characterized by atypical absences, erratic myoclonus, and occasional tonic–clonic seizures. EEG demonstrates high-amplitude 2–3Hz delta activity with spike and slow-wave discharges and sleep-activated generalized epileptiform discharges. Sodium valproate, benzodiazepines, and priacetam are frequently used and effective. Development is generally slow, the majority attaining independent walking in the first 2.5–6 years. Vocabulary is limited to a few single words with superior speech and object apprehension. The condition is due to a lack of expression of the UBE3A gene on chromosome 15q. Maternal deletions of 15q11-13 produce the most pronounced phenotype (65–70% of probands), uniparental disomy and imprinting center mutations (10%), and UBE3A point mutations (11%) produce milder phenotypes.

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