been identified, including endplate acetylcholine and AChR deficiencies, a slow-channel syndrome, and defects in resynthesis of ACh and kinetics of AChR.

The common feature of CMS is a defect in the neuromuscular junction with exercise-induced weakness of skeletal muscle. In the initial report, cases presented with ptosis, weak cry, and generalized weakness. Diagnosis was delayed until 1 to 4 years after birth, when ptosis and external ophthalmoplegia were the most prominent signs, and generalized weakness and respiratory difficulty were mild in degree. Response to cholinergic drug therapy was poor, and ophthalmoplegia was unrelieved. Except for the resistance to treatment, CMS with *DOK7* mutations has an unusual clinical phenotype.

SLEEP DISORDERS

DYSSOMNIAS AND PARASOMNIAS

The prevalence of dyssomnias and parasomnias was determined in a longitudinal study of a sample of 2223 infants born in 1997-98 in the province of Quebec, Canada, and reported from the Sleep Disorders Center, Sacre-Coeur Hospital, University of Montreal. Self-administered questionnaires were completed by the mothers when the children reached 2.5 years of age, and at intervals up to 6 years. Dyssomnias manifested by frequent night wakings (-/>1 per night) and difficulty falling asleep at night (-/>30 min) were reported in 36.3% and 12.2% children at age 2.5 years, respectively. At age 6 years, the percentage with frequent night wakings had decreased steadily to 13.2%, and the percentage with difficulty falling asleep had decreased significantly to 7.4%. Between 2.5 and 6 years, the percentage with difficulty falling asleep had first increased to 16.0% at ages 3.5 and 4 and then decreased to 10% at age 5 and to 7.4% at age 6.

The overall prevalence of parasomnias from 2.5 to 6 years was as follows: somnambulism, 14.5%; sleep terrors, 39.8%; somniloquy (talking during sleep), 84.4%; enuresis, 25.0%; bruxism, 45.6%; and rhythmic movements, 9.2%. Sleep terrors were more frequent at ages 2.5, 3.5, and 4 than at ages 5 and 6. Rhythmic movements (body-rocking and head-banging) were more prevalent at age 2.5 than at all later ages. Boys outnumbered girls (ratio 2:1) with somnambulism and enuresis, but no gender differences were observed for prevalence of other parasomnias. Persistent somnambulism was correlated with persistent sleep terrors (P<.01) and persistent somniloquy (P<.001). Persistent sleep terrors were correlated with persistent somniloquy (P<.001), and with frequent night wakings (P<.001).

Children with persistent dyssomnias had higher separation anxiety scores, and had been put to bed already asleep in a greater proportion.at 2.5 years. Sleep-onset difficulties were associated with low socioeconomic status (SES), but not with co-sleeping or separation anxiety. Neither dyssomnia was associated with ADHD. Persistent parasomnias were associated with separation anxiety. They had little impact on sleep duration. Persistent somnambulism was related to a high ADHD score. Sleep terrors were related to a recent divorce of parents. Persistent rhythmic movements were related to insufficient SES and maternal depression. (Petit D, Touchette E, Trembley RE et al. Dysomnias and Parasomnias in early childhood. **Pediatrics** May 2007;119:e1016-e1025). (Respond: Jacques Montplaisir MD, PhD, Sleep Disorders Center, Sacre-Coeur Hospital, 5400 Gouin Blvd W, Montreal, Quebec, Canada H4J 1CS).

COMMENT. The prevalence of night wakings and sleep-onset problems is high in preschool children but symptoms lessen by 6 years of age. Parasomnias are also prevalent in early childhood and are associated with separation anxiety. Sleep terrors are associated with somnambulism, somniloquy, and frequent night wakings.

Night terrors and nocturnal frontal lobe epilepsy (NFLE). The differentiation of parasomnias and NFLE may be difficult, and the two diagnoses may co-exist. Night terrors in early childhood are sometimes followed by NFLE in school-age children. The Frontal Lobe Epilepsy and Parasomnias (FLEP) scale (Derry CP et al. **Epilepsia** 2006;47:1775-1791; Derry CP et al **Arch Neurol** 2006;63:705-709) is considered reliable in distinguishing these disorders. A video-polysomnographic recording may be necessary to confirm the diagnosis in some cases (Tinuper P et al. **Epilepsia** 2007;48:1033-1034).

CONGENITAL DEVELOPMENTAL ANOMALIES

CONGENITAL OCULAR MOTOR APRAXIA

The clinical and neuroradiological findings, and long-term intellectual prognosis in 10 patients (4 boys and 6 girls) with congenital ocular motor apraxia (COMA) are reviewed by researchers at Tottori University, Yonago, Japan. Age at first examination was 4 months to 5 years, and follow-up ranged from 4 to 37 years (mean 17.3 years). COMA was diagnosed by impaired voluntary, saccadic eye movements, and head thrusting movements in the horizontal direction. A representative patient, a boy examined at the age of 7 months and sitting had head thrust movements. He looked at objects obliquely, with eyes deviated laterally. He walked late at 22 months, but speech developed normally. At 5 years of age, he was examined because of persistent unsteady gait and abnormal eye movements. Apart from the tandem ataxia, impaired eye movements, and a left external strabismus, the remainder of the neurologic exam was normal. Brain CT showed dilatation of the fourth ventricle and hypoplasia of the cerebellar vermis. At follow-up at age 7 years, tandem gait had improved, but titubation of the head and signs of OMA, including head thrust and excessive blinking at visual tracking, persisted. At 37 years old, he is an independent, working adult.

Typical oculomotor findings of COMA (locking-up during manual spinning and impaired optikokinetic nystagmus) were elicited in all patients, excessive blinking in 5, strabismus (7), and oblique lateral glance in 8. Atypical signs, including rotatory or jerky nystagmus and limitation in upward gaze, were present in 4 patients, and were often accompanied by intellectual disabilities. Delay in walking occurred in all patients, 4 at 2 years of age and 6 walked later. Ataxia was common in the later-walking group, and was complicated by speech problems and intellectual retardation. Neuroimaging revealed dilatation of the fourth ventricle in 8, hypoplasia of the cerebellar vermis in 6, and 'molar-tooth' sign at the midbrain in 3. None had a family history of COMA, but 3 had family members with mental retardation. (Kondo A, Saito Y, Floricel F et al. Congenital ocular motor apraxia: Clinical and neuroradiological findings, and long-term intellectual prognosis. **Brain Dev** August 2007;29:431-438.) (Respond: Dr Akiko Kondo, Division of Child Neurology, Institute of Neurological Sciences, Tottori University, 36-1 Nishi-Cho, Yonago 680-8504, Japan).