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COMMNT. The prevalence of sleep terrors is high in infants (37% at 18 months) and decreases by one half to approximately 20% by 30 months of age. Genetic factors play an important role in the etiology of this early childhood parasomnia, accounting for >40% of the phenotypic variance for both 18- and 30-month-old twins. The role of non-shared environmental factors is also significant, >55% of the variance at both 18 and 30 months. Night terrors have a combination genetic-environmental etiology, but to date, no specific genes have been identified.

GENETICALLY DETERMINED EEG FINGERPRINT OF SLEEP

The influence of genetic factors on the individual profile of sleep electroencephalographic (EEG) power spectra at the 8 to 16 Hz frequency range during non-rapid eye movement (NREM) sleep was determined by recording 40 monozygotic and dizygotic twins during sleep. The study performed at the University of Rome and various international centers found that this EEG fingerprint of sleep showed a greater similarity in monozygotic than dizygotic pairs, with a 96% estimate of heritability. (De Gennaro L, Marzano C, Fratello F, et al. The electroencephalographic fingerprint of sleep is genetically determined: a twin study. Ann Neurol Oct 2008;64:455-460). (Respond: Dr De Gennaro, Department of Psychology, Section of Neuroscience, University of Rome “Sapienza,” Via dei Marsi, 78, 00185 Rome, Italy. E-mail: luigi.degennaro@uniroma1.it).

COMMENT. Healthy humans have a unique profile of the sleep electroencephalographic (EEG) power spectra at the 8 to 16 Hz frequency range during non-rapid eye movement (NREM) sleep. This fingerprint allows discrimination between individuals with a probability of 92% (De Gennaro L et al, 2005). These authors have shown that individual differences in this EEG fingerprint of NREM sleep are genetically determined. A genetic contribution has already been demonstrated for the awake-resting EEG alpha power, and also, for many sleep disorders, including night terrors, narcolepsy, obstructive sleep apnea, restless legs syndrome, and Kleine-Levin syndrome.

NEUROMUSCULAR DISORDERS

HAND INVOLVEMENT IN CHARCOT-MARIE-TOOTH DISEASE 1A

Hand strength, function and disease-related symptoms were determined in 84 children, aged 2-16 years, with Charcot-Marie-Tooth disease type 1A (CMT1A) at University of Sydney, Children’s Hospital at Westmead, and Royal Children’s Hospital, Parkville, Australia. Hand weakness and dysfunction was present from the earliest stages of the disease and tended to worsen with age throughout childhood. Poor handwriting, weakness, pain and sensory symptoms also worsened with age. (Burns J, Bray P, Cross LA, North KN, Ryan MM, Ouvrier RA. Hand involvement in children with Charcot-Marie-Tooth disease type 1A. Neuromuscul Disord Dec 2008;18:970-973). (Respond: Dr Joshua Burns,