COMMENT. Epidemiological studies of the antecedents of CP demonstrate a strong association between premature birth and CP. In the classical description of cerebral palsy by Little (Little WJ, **Trans Obstet Soc Lond** 1862;3:283-344), the influence of abnormal parturition, difficult labor, premature birth and asphyxia neonatorum are emphasized. In the US National Institutes of Health Neurological Collaborative Perinatal Project (NCPP), 27% of all cases of CP are attributed to low birth weight (<2501g) and short gestation (<36 weeks). (Ellenberg J, Nelson K. **Am J Dis Child** 1979;133:1044-1048). However, the majority of children with CP are of normal birth weight and term gestational age. The NCPP data show that even infants of normal birth weight (>2500g) who are born prematurely (<36 weeks) are at higher risk of CP than those of normal weight born at term (>37 weeks). True prematurity is more important than intrauterine growth retardation in infants at risk of CP. (Freeman JM. Ed. **NIH Publication** 1985-1149)

Preterm birth is an established risk factor for CP, but the relevance of gestational age in the term range, where most of CP occurs, is rarely documented. In near-term infants, the risk of CP is highest with gestational ages slightly lower (37 or 38 weeks) or greater (42 and 43 weeks) than term. The interpretation of the Norway data is difficult for several reasons (eg. variation in prevalence with gestation over different time periods (Suzuki J et al. **No To Hattatsu** 2009; 41(4):279-283) and with different subtypes–spastic, dyskinetic, ataxic, or causes [Self L, Shevell MI. **J Child Neurol** 2010 Mar 11 (Epub ahead of print]). Furthermore, CP is not a disease entity, but a general descriptive term for a non-progressive motor deficit of early onset.(Freeman, 1985). The data obtained from epidemiological studies represent only an association of risk factors with CP and are of limited clinical significance. As the authors conclude, the biological mechanisms for these risk factors must be determined before intervening clinically in the gestational age at delivery.

SEIZURE DISORDERS

CLINICAL CORRELATIONS OF EEG MIDLINE SPIKES

The frequency, clinical characteristics and outcome of children with isolated midline spikes in the EEG, and comparison of those with and without epilepsy, were analyzed by researchers at Children's Hospital, Boston, MA. In a total of 12,000 EEGs performed from 2005 to 2009, 69 had Cz-Pz discharges as the only epileptiform abnormality. Midline spikes were defined as focal epileptiform discharges localized to or of highest amplitude at one of the vertex scalp electrodes, Cz or Pz. Midline spikes were differentiated from transient vertex waves of sleep by their occurrence during the awake stage as well as sleep. EEGs with discharges at additional sites were excluded. The prevalence of isolated midline spikes was 0.54%; 45 (65%) were in boys. Median age was 6 years (range, 4 mo -23 yrs); 2-3 yr-olds were the most common age group (16 cases), followed by the 4-5 yr-olds (11 cases). Forty-three (62%) had a history of seizures, and 26 (37%) without epilepsy presented with symptoms suggestive of seizures. *In the group with seizures*, Cz and Pz spikes occurred in 33% (14 of 43 cases); Cz spikes only in 49% (21 of 43 cases), and Pz spikes only in 18% (8 of 43 cases). Seizures were partial in 25 (58%); complex partial in 22 (51%), and focal myoclonic in 3. MRI was

abnormal in 56% cases with seizures, and 26% of those without (P<0.05). The group with seizures was 5 times more likely to develop new discharges at additional sites than the nonepilepsy group. *In the nonepilepsy group*, 46% had developmental delay, 19% pervasive developmental disorder, and 15% had ADHD; these disorders were significantly more frequent in the nonseizure group than in those with seizures (P<0.05). Computerized source analysis in 12 random cases showed that isolated midline spikes were localized to the midline convexity, similar to the EEG localization by surface electrodes. (Vendrame M, Tracy M, Das R, Duffy F, Loddenkemper T, Kothare SV. Clinical correlations of midline spikes in children. **Epilepsy Behav** 2010;18(8):460-465). (Respond: Sanjeev V Kothare MD, Children's Hospital Boston, Fegan 9, 300 Longwood Ave, Boston, MA 02115. E-mail: Sanjeev.Kothare@childrens.harvard.edu).

COMMENT. The authors conclude that isolated midline spikes do not invariably signify an increased susceptibility to seizures, but also occur in normal children and in children with developmental and behavioral problems. A repeat EEG in a child with midline spikes and epilepsy is more likely to show new discharges in additional sites.

NEURONAL NETWORKS AND CONTINUOUS SPIKES AND WAVES DURING SLOW SLEEP

Haemodynamic changes associated with epileptic activity were investigated at Christian-Albrechts-University, Kiel, Germany, using simultaneous acquisitions of EEG and functional MRI in 12 children with symptomatic and cryptogenic continuous spikes and waves during slow sleep (CSWSS). MR findings were compared to electric source analysis, and all patients showed spike-related positive (activations) and negative (deactivations) blood oxygenation-level-dependent changes (P<0.05). The activations involved bilateral perisylvian region and cingulated gyrus in all cases, bilateral frontal cortex in 5, bilateral parietal cortex in 1, and thalamus in 5 cases. Electrical source analysis demonstrated a similar involvement of perisylvian regions, independent of the area of spike generation. Spike-related deactivations were found in precuneus, parietal cortex and medial frontal cortex in all patients and in caudate nucleus in 4. Independent of the cause, CSWSS cases were characterized by activation of a similar neuronal network: perisylvian region, insula and cingulate gyrus. The activations correspond to both initiation and propagation pathways. (Siniatchkin M, Groening K, Moehring J, et al. Neuronal networks in children with continuous spikes and waves during slow sleep. **Brain** Sept 2010;133(9):2798-2813). (Respond: E-mail: m.siniatchkin@pedneuro.uni-kiel.de).

COMMENT. CSWSS (or electrical status epilepticus during sleep) is an age related (peak at 4-5 yrs old) epileptic encephalopathy characterized by interictal epileptic discharges during >85% non-REM sleep, and receptive language, cognitive, and behavioral disorders. Seizures occur during sleep or while awake, partial, generalized tonic-clonic or myoclonic. CSWSS is distinguished from Landau-Kleffner syndrome by the continuous bilateral and diffuse spike-wave activity, persisting through all slow sleep stages. (Browne, Holmes 2004) Four children with epilepsy and CSWSS, followed between 18 mos and 4 years of age, showed a pattern of behavioral and cognitive