

language impairment exhibited a significant lack of left lateralization in core language regions (inferior frontal gyrus-opercularis, inferior frontal gyrus-triangularis, supramarginal gyrus and superior temporal gyrus). Between group comparisons revealed a left hypoactivation of Wernicke's area during the responsive naming task, and a right hyperactivation of the anterior insula, inferior frontal gyrus and head of caudate during a phonological task. Developmental dysphasia is associated with atypical lateralization and functioning of core language areas. (de Guibert C, Maumet C, Jannin P et al. Abnormal functional lateralization and activity of language brain areas in typical specific language impairment (developmental dysphasia). **Brain** 2011;134:3044-3058). (Respond: Dr Clement de Guibert, Sciences du langage, Universite Rennes 2, Place H Le Moal, CS 24307, F-35043 Rennes Cedex, France. E-mail: clement.deguibert@univ-rennes2.fr).

COMMENT. The etiology of specific language impairment is unclear; an interaction between genes and environmental risk factors may affect the anatomo-functional development and organization of the brain language network (Rapin I et al, 2003). fMRI and other studies show that an abnormality of brain development is bilateral and not confined to the left hemisphere.

INFANTILE ENCEPHALOPATHIES

KCNQ2 ENCEPHALOPATHY AND NEONATAL SEIZURES

Researchers from centers in Belgium and Australia analyzed 80 patients with unexplained neonatal or early infantile seizures and associated psychomotor retardation for KCNQ2 mutations. Seven different heterozygous *KCNQ2* mutations were found in eight patients (8/80; 10%); six mutations arose de novo. No *KCNQ3* mutations were found. The eight *KCNQ2* encephalopathy patients had onset of intractable seizures with tonic component in the first week of life. Seizures resolved by age 3 years but the children had profound or severe psychomotor impairment. EEG at onset showed a multifocal epileptiform activity. Early brain MRI showed hyperintensities in the basal ganglia and thalamus that later resolved. *KCNQ2* screening should be included in the diagnostic workup of refractory neonatal seizures of unknown origin. (Weckhuysen S, Mandelstam S, Suls A, et al. *KCNQ2* encephalopathy: Emerging phenotype of a neonatal epileptic encephalopathy. **Ann Neurol** Oct 2011;published on line, 10.1002/ana.22644).(Respond: Prof Peter De Jonghe, University of Antwerp, Belgium. E-mail: peter.dejonghe@molgen.vib-ua.be).

COMMENT. Mutations in the voltage gated K(+) -channel gene *KCNQ2* cause benign familial neonatal convulsions, the majority having a favorable outcome. (Singh NA et al. **Brain** 2003;126:2726-2737) Reports of patients with a poor outcome are infrequent. Dedek K and associates (**Epilepsy Res** 2003;54(1):21-27) of Hamburg, Germany reported two children in an Italian family with neonatal convulsions and *KCNQ2* mutations, the index patient having a poor outcome and therapy-resistant epilepsy. The present report is supportive of the association of some *KCNQ2* mutations with an infantile epileptic encephalopathy complicated by psychomotor impairment and refractory seizures.