ANTICONVULSANT SUPPRESSION OF POSTNATAL NEUROGENESIS IN LABORATORY ANIMALS

The effects of phenobarbital and diazepam on cell proliferation and neurogenesis were studied in newborn rats followed for 6 months, in a study at University of Dresden, Germany; Medical University, Lublin, Poland; University Medicine Berlin, Germany; and Solvay Research Laboratories, Weesp, The Netherlands. The N-methyl-D-aspartate antagonist MK801, and the GABA subtype A agonists phenobarbital and diazepam administered to infant rats on postnatal days 6-10 caused reduced numbers of neurons in the hippocampal dentate gyrus at postnatal day 15. No apoptosis was demonstrated. At age 6 months, phenobarbital-treated rats had fewer neurons in the dentate gyrus and performed worse than saline-treated littermates in water maze learning and memory task. Blockade of N-methyl-D-aspartate receptor-mediated excitation and enhancement of GABA subtype A receptor activation impair cell proliferation and inhibit neurogenesis in the immature rat brain. These findings raise concerns about the frequent use of phenobarbital in the treatment of neonatal seizures. (Stefovska VG, Uckermann O, Czuczwar M, et al. Sedative and anticonvulsant drugs suppress postnatal neurogenesis. **Ann Neurol** Oct 2008;64:434-445). (Respond: Dr Ikonomidou, Department of Pediatric Neurology, Children's Hospital, Medical Faculty Carl Gustav Carus, University of Technology Dresden, Fetscherstrasse 74, 01307 Dresden, Germany).

COMMENT. Neurogenesis in the hippocampal dentate gyrus is at its peak during the first week of postnatal life and declines progressively after day 9 in newborn rats. Phenobarbital administered in the first week to 10 days results in reduced neurogenesis at 2 weeks postnatally and impairment of learning and memory at 6 months, equivalent to adult life. These effects result in decreased hippocampal volume, reduced neuronal densities in the dentate gyrus, the CA1-hippocampus, and the cingulate cortex. These observations call for caution regarding the use of NMDA receptor antagonists and GABA-a agonists in neonatal, pediatric, and obstetric medicine.

SLEEP DISORDERS

SLEEP TERRORS IN TWINS

In an attempt to clarify the genetic and environmental causes of sleep terrors in childhood, reasearchers in Canada followed 390 pairs of monozygotic and dizygotic twins by assessing the frequency of sleep terrors at 18 and 30 months of age using a questionnaire administered to the biological mothers. The prevalence of sleep terrors was 36.9% at 18 months and 19.7% at 30 months. Boys and girls were affected equally. The polychoric correlations were 0.63 monozygotic and 0.36 dizygotic at 18 months and 0.68 and 0.24 at 30 months. Sleep terrors were best explained by a genetic and non-shared environmental, 2-component model. At 18 months, genetic factors accounted for 43.7% and non-shared environmental factors for 56.3% of the phenotypic variance; at 30 months, these proportions were 41.5% and 58.5%, respectively. (Nguyen BH, Perusse D, Paquet J, et al. Sleep terrors in children: a prospective study of twins. **Pediatrics** Dec 2008; 122:e1164-e1167). (Respond: