PEDIATRIC NEUROLOGY BRIEFS
A MONTHLY JOURNAL REVIEW

J. GORDON MILLICHAP, M.D., F.R.C.P., EDITOR

Vol. 22, No. 1 January 2008

DEVELOPMENTAL DISORDERS

DEVELOPMENTAL OUTCOME OF VERY PRETERM INFANTS AT ADOLESCENCE CORRELATED WITH GREY AND WHITE MATTER ABNORMALITIES ON MRI

MRI data of brains of 218 adolescents (ages 14-15 years) born very preterm, < 33 weeks gestation (VPT), and 128 controls born at term were compared, using voxel-based morphometry, and the findings correlated with neurodevelopmental outcome in a study at King's College London Institute of Psychiatry and Centre for Neuroimaging Sciences, Maudsley NHS Trust, London, UK. VPT subjects showed reduced grey matter (GM) in temporal, frontal, occipital cortices and cerebellum and increases in adjacent GM predominantly in temporal and frontal lobes. White matter (WM) was decreased in the brainstem, internal capsule, temporal and frontal regions, and showed excesses in temporal, parietal and frontal regions. The areas showing increased and decreased GM and WM volumes were structurally associated. The greatest WM and GM alterations occurred in VPT individuals with evidence of periventricular hemorrhage and ventricular dilatation on neonatal ultrasound. VPT adolescents had lower scores than controls on measures of language and executive function and their cognitive function was more likely to be impaired (27% vs 14%, respectively; p=0.013). Gestational age was positively correlated with GM and WM volumes. Specific cognitive deficits and neurodevelopmental delay associated with VPT birth may be related at least in part to altered grey and white matter volumes. (Nosarti C, Giouroukou E, Healy E, et al. Grey and white matter distribution in very preterm adolescents mediates neurodevelopmental outcome. **Brain** January 2008;131:205-217). (Respond: Dr Chiara Nosarti, Department of Psychiatry, PO Box 63, King's College London Institute of Psychiatry, 16 De Crespigny Park, Denmark Hill, London SE5 8AF, UK).

PEDIATRIC NEUROLOGY BRIEFS (ISSN 1043-3155) © 2008 covers selected articles from the world literature and is published monthly. Send subscription requests ($68 US; $72 Canada; $75 airmail outside N America) to **Pediatric Neurology Briefs - J. Gordon Millichap, M.D., F.R.C.P.-Editor,** P.O. Box 11391, Chicago, Illinois, 60611, USA. The editor is Pediatric Neurologist at Children's Memorial Hospital and Professor Emeritus, Northwestern University Medical School, Chicago, Illinois.

PNB is a continuing education service designed to expedite and facilitate review of current scientific information for physicians and other health professionals. Fax: 312-943-0123.

COMMENT. Previous studies have shown that infants born very preterm (VPT), <33 weeks gestation and of low birth weight (<2500g), are at risk of hemorrhage and hypoxic-ischemic damage that results in dilated ventricles, loss of white matter, and enlarged subarachnoid space (Volpe JJ. **Pediatrics** 2003;112:176-180); (Inder TE et al. **J Pediatr** 2003;143:171-179). Subarachnoid fluid collections in very low birth weight infants, sometimes called "external hydrocephalus," may be associated with transient macrocrania and neurodevelopmental abnormalities (hypertonia and hyperreflexia) that resolve by 18 months of age. (Al-Saedi SA et al. **J Pediatr** 1996;128:234-236). Half of VPT adolescents show persisting brain abnormalities, with smaller cortical volumes and larger lateral ventricles compared to controls. (Cooke RW et al. **Arch Dis Child Fetal Neonatal Ed** 1999;81 (F):116-121).

Computational morphometry used to process MRI data in the present study identifies focal localized changes in GM and WM concentration. The findings suggest that alterations in grey and white matter volume demonstrated in temporal, frontal, cerebellar and other regions of brain may be responsible for the cognitive impairments found in adolescents born VPT. The cerebral developmental changes following VPT birth result not only in GM and WM loss, but also in cortical and subcortical tissue excesses, often in adjacent and structurally associated areas. Infants who experience the greatest degree of perinatal insult exhibit the most severe GM and WM alterations and have the highest risk of neurodevelopmental compromise. When VPT birth is complicated by severe brain injury, brain plasticity compensates for resulting cell loss, with production of extra cells and synapses that normally become 'pruned' during later development. These compensatory processes are particularly extensive in preterm infants showing the most severe neonatal ultrasound abnormalities.

Selective vulnerability varies with the stage of development of different brain regions. GM volume in frontal lobes increases during preadolescence, the prefrontal areas reaching full maturity in late adolescence. Temporal lobe GM development peaks in mid-adolescence. VPT children may show not only a global delay in brain maturation but also, differences in GM and WM volumes. Identification of these brain volume changes by MRI in early life may be used as a clinical marker of increased risk of cognitive impairment at a later age, and may lead to educational intervention.

ETIOLOGY AND TREATMENT OF DEVELOPMENTAL STAMMERING

The etiology and treatment of developmental stammering in childhood (DS, also called idiopathic stammering or stuttering) are reviewed by a speech pathologist and psychologist at the University of Reading, UK. Prevalence is estimated at 1 to 3% of the population. DS is distinguished from neurogenic stammering (secondary to stroke, tumor, or degenerative disease) and psychogenic stammering. DS usually develops in pre-school age groups, the mean age at onset of 4 years, with 75% cases beginning before age 6. The cause is multifactorial, demands placed on the child exceeding the capacity to manage speech and language variables. The evidence for a genetic component to stammering is strong, twin studies showing concordance in monozygotic twins of 75 – 89%. Some children with DS are linguistically advanced while others are delayed. A mismatch between motor speech and language abilities may cause impairment in fluency, when articulation skills are not