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## ATTENTION DEFICIT AND LEARNING DISORDERS

### TRANSCALLOSAL MECHANISM OF MIRROR MOVEMENTS

The mechanism of mirror movements in 11 normal adults and 39 children (aged 4-11 years) was studied in the Department of Physiology, University College London, UK. Surface electromyographic (EMG) activity was recorded from the left and right first dorsal interosseous muscles (IDI) during voluntarily activated sequential finger-thumb opposition or repetitive index finger abduction. During unilateral opposition movements (IDIvol), mirrored EMG activity (IDImm) in the homologous muscle of the opposite hand was decreased with increasing age. In the younger children, involuntary EMG activity in the IDImm was associated with activity recorded from the IDIvol during a single finger-thumb opposition. In older children, age 11 years, and adults, mirrored activity was not always present and was only detected in the average of 25 sequential finger-thumb oppositions or during performance of forceful and the less familiar index finger abduction. Mirrored activity was variable in time of onset but could occur at the same time as the voluntary burst. The slowly conducting ipsilateral corticospinal projection was not responsible for the mirrored activity recorded. In adults but not in children, transcranial magnetic stimulation showed a significant decrease in the motor evoked potential (MEP) from one motor cortex, in response to the conditioning stimulus of the other motor cortex, consistent with a transcallosal effect. Slower conducting callosal fibers in children because of late myelination would prevent the transcallosal inhibitory mechanism. The bilateral activation of both motor cortices during unilateral voluntary tasks is suggested as the likely mechanism for mirror movements in healthy children. (Mayston MJ, Harrison LM, Stephens IA. A neurophysiological study of mirror movements in adults and children. Ann Neurol May 1999:45:583-594). (Respond: Dr Margaret I Mayston. Department of Physiology, University College London, Gower Street, London WC1E 6BT, UK).

COMMENT. Mirrored alternating movements of forearms, a normal finding in the neurologic examination of children younger than 7 years, is regarded as a "subtle" abnormality or "soft" sign in older children with attention deficit disorder. Pathologic mirror movements occur in children with congenital

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The editor is Pediatric Neurologist at Children's Memorial Hospital and Northwestern University Medical School, Chicago, Illinois.

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cerebral hemiplegia, Kallmann's syndrome, Klippel-feil syndrome, and dysplasias of the upper spinal cord. A delayed maturation of inhibitory cortical pathways is often suggested as the mechanism of mirror movements. The University College study localizes the mechanism of mirror movements in children to a delayed myelination of transcallosal inhibitory fibers, with consequent bilateral activation of cortico-spinal pathways originating from both motor cortices. Underdevelopment of the corpus callosum demonstrated by quantitative MRI in a group of children with ADHD supports the transcallosal mechanism of mirror movements in this syndrome. (see <a href="Progress in Pediatric Neurology III">Progress in Pediatric Neurology III</a>, PNB Publ, 1997;p212).

Precision grip and manipulation (grip-lift synergy) was examined in 13 children with spastic hemiplegia and unilateral brain lesions, in a study at the Karolinska Hospital, Stockholm, Sweden (Forssberg H et al. <u>Brain</u> June 1999;122:1157-1168). The developmental level of grip-lift synergy, a model of grasping and manipulation, was correlated with impaired dexterity and with extensive lesions in the contralateral cortex, white matter, thalamus, and basal ganglia. Perinatal lesions in specific cortical motor areas, not resulting in impaired dexterity, may be compensated for by other neural circuits. No specific localization was demonstrated by MRI to correlate with impaired dexterity in children with hemiplegic cerebral palsy. Instead, the memory for grip-lift synergy involves several cortical and subcortical regions.

### DYSCALCULIA AND ATTENTION DEFICIT SUBTYPES

The association of specific academic deficits with attention deficit disorder (ADD) subtypes was determined in 20 students (ages 8-12) with ADD with hyperactivity (ADD/H) compared to 20 with ADD without hyperactivity (ADD/noH), at the Department of Educational Psychology, University of Texas at Austin, TX. Using subtests of the Woodcock-Johnson Psycho-Educational Battery-Revised to determine group differences, scores for students with ADD/noH were significantly different on four of seven measures, and the Calculation subtest scores were significantly lower when compared to all other achievement subtests. By contrast, for students with ADD/H, no significant differences were found in six of seven analyses, although their Calculation subtest scores were lower than scores on the Applied Problems subtest. These results support the hypothesis that inattention has a specific and adverse effect on arithmetic computation skills. (Marshall RM, Schafer VA, O'Donnell L, Elliott J, Handwerk ML. Arithmetic disabilities and ADD subtypes: Implications for DSM-IV. I Learning Disabilities May/June 1999;32:239-247). (Respond: Richard M Marshall, Department of Educational Psychology, SZB 504, University of Texas at Austin, Austin, TX 78712).

COMMENT. Students with ADHD-Predominantly Inattentive Type are at increased risk for arithmetic calculation deficits. These findings have important implications for the diagnosis and treatment of ADHD and the recognition of ADHD subtypes as defined by the DSM-IV criteria. Children with ADD-inattentive type should also be examined for Gerstmann syndrome.

Gerstmann syndrome. Profound developmental dyscalculia and Gerstmann syndrome may occur in children with ADHD. Deficits in specific cognitive areas may involve visuo-spatial perception and parietal-occipital function. (See Millichap, Attention Deficit Hyperactivity and Learning Disorders, PNB Publ. 1998;pp82-86, for an account of Gerstmann syndrome in ADHD). Confirmation of the essential visuospatial origin and impaired mental manipulation of images is described in "a pure case of Gerstmann syndrome with a subangular lesion" (Mayer E et al. Brain June 1999;122:1107-1120). An adult