

PEDIATRIC NEUROLOGY BRIEFS

A MONTHLY JOURNAL REVIEW

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

Vol. 15, No. 1

January 2001

LEARNING AND ATTENTION DISORDERS

NEUROLOGICAL BASIS OF DYSLLEXIA

Evidence of a neurological basis for developmental dyslexia is reviewed by a researcher from the Cognitive Neurology Laboratory, Department of Neurology, Marseille, France. Beginning with the *neuroanatomical* findings (cortical malformations - ectopias and dysplasias, and absence of normal asymmetry of the planum temporale) of the Boston school (Galaburda et al, 1985, 1989; Cohen et al, 1989), and interhemispheric corpus callosal deficits (Hynd et al, 1995); subsequent areas of investigation covered include the central role of *phonological* disorders (grapheme-to-phoneme conversion deficit) in dyslexia, an insensitivity to auditory frequency awareness and modulation (Manis et al, 1997); the demonstration of a *visual processing* deficit (the 'magnosystem' theory (Livingston et al, 1991), and 'dysideitic' (Boder, 1973) subgroup); the '*temporal (rate)-processing*' theory (Tallal and Piercy, 1973), the dyslexics' inability to process rapidly successive auditory or visual stimuli (a 'dyschronia' (Llinas, 1993)); the contribution of *electrophysiological* studies (event-related potentials, mismatch negativity, P300, and N400 (Taylor, 1995)), brain *functional imaging* (magnetoencephalography (Salmelin et al, 1996), functional MRI, PET during phonological tasks), and a working hypothesis and method of remediation based on the temporal processing deficit theory. Evidence points to a multi-system deficit related to temporal processing. The brain of dyslexics is unable to process stimuli presented in rapid succession, which accounts in part for the perceptual, motor, and cognitive impairments frequently associated with learning disorders. Recently proposed training methods include acoustically modified speech to correct the temporal processing deficit described in dyslexics (Merzenich et al, 1996; Tallal et al, 1996). (Habib M. The neurological basis of developmental dyslexia. An overview and working hypothesis. *Brain* Dec 2000;123:2373-2399). (Repond: Dr Michel Habib, Centre de recherche, Institut Universitaire de Geriatrie, 4565 Ch Queen Mary, Montreal (QUE), Canada H3W 1W5).

COMMENT. *Developmental dyslexia* is defined as a neurologically-based, often familial, disorder which interferes with the acquisition and processing of

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language. *Dyslexia* is one of several distinct learning disabilities. It is a specific language-based disorder of constitutional origin, characterized by difficulties in single word decoding that reflect insufficient phonological processing. (See Millichap JG. Attention Deficit Hyperactivity and Learning Disorders. PNB Publ, Revised 2001, for definitions and references to dyslexia, LD, and ADHD). Several methods of reading remediation have been reviewed by educators, including multisensory, modified alphabets, language-experience, individualized reading, synthetic phonics, linguistic decoding, and phonics systems (Millichap N, 1986). The multisensory approach to treatment of dyslexia is the most usual remediation employed by educators (Pavoni B, 2000).

The theory that dyslexia is due to an immaturity of cerebral function is supported by the pathological and neuroimaging evidence of developmental cerebral anomalies and electrophysiological studies. The anatomical location of the anomalies strongly supports the "phonological-linguistic," or deficient speech sound and decoding theory of dyslexia (Denckla MB, In Progress in Pediatric Neurology II. PNB Publ, 1994;p174). The left temporal-parietal area appears to be most critical in location of normal reading ability, but additional areas of the left hemisphere may be involved also. Some reports of acquired dyslexia following surgery on the brain have involved the left frontal lobe. A "disconnection theory" for dyslexia, involving impaired relays between the anterior and posterior areas of the left brain, has been proposed based on PET studies (Paulesu et al, 1996).

Dyslexia occurs in 5 to 10% of school children, at all levels of intelligence, from superior to low normal. Dyslexia may be an isolated abnormality or may be associated with other learning disabilities. Reading and spelling disability overlaps with ADHD and shows similar genetic characteristics but different brain localizations. Anatomically, left hemisphere deficits underly reading and other learning disabilities, whereas the right frontal lobe is involved in ADHD.

DEVELOPMENTAL RISKS OF INATTENTIVE BEHAVIOR

The developmental functioning, social, and environmental backgrounds of a community-based, epidemiological sample of 7-year-old children with pure inattentive behavior (I-subtype, n=31, 1.3% of sample) were compared to that of children with pure overactive behavior (HI subtype, n=31) ADHD-Combined type (n=31) and a control group at the Maudsley Hospital, London, UK. A 2-item inattention subscale was derived from the Conners' (1969) questionnaire, namely "Fails to finish things" and "Inattentive, easily distracted." A cutoff score of 3 or more on these items was employed as the definition of inattentiveness. Other measures included parent and teacher interviews, general psychometric tests, Digit Span subtest of the WISC-R, Continuous performance task, paired-associates learning task, a Matching Familiar Figures Test, and objective measures of attentive behavior.

Inattentive behavior was significantly correlated with low self-esteem and need for repeated instructions in school, lower verbal IQ and general cognitive functioning, poor reading scores, and lower language related skills. Whereas the HI and Combined-ADHD groups showed more conduct and social-interaction problems, these outcomes were not encountered in the inattentive group. The fathers of inattentive children were more likely to have a low occupational status. (Warner-Rogers J, Taylor A, Taylor E, Sandberg S. Inattentive behavior in childhood: Epidemiology and implications for development. Jrnl of Learning Disabilities Nov-Dec 2000;33:520-536). (Respond: Dr Jody Warner-Rogers, Child Neuropsychiatry Clinic, Children's Outpatient Department, Maudsley Hospital, Denmark Hill,