National Library of Canada

Acquisitions and Bibliographic Services Branch

395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale du Canada

Direction des acquisitions et des services bibliographiques

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

Canada

Sleep Patterns and Eye Movement Density During REM Sleep in Reading Disabled Children

by

Lise Mercier

Thesis submitted to the School of Graduate Studies and Research of the University of Ottawa in partial fulfillment of the requirements for the Doctor of Philosophy Degree in Clinical Psychology.

ISBN 0-315-80033-X

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

à mes parents,

envers qui je serai toujours

reconnaissante

ABSTRACT

In the present study, sleep characteristics in reading disabled (RD) children were recorded to examine suggested relationships among sleep, maturational and cognitive processes. Subjects were thirty-nine 8-10 year old boys [15 controls (Cs), M= 9.2, SD= 0.6 yrs; 24 RD, M= 9.0, SD = 0.5 yrs, (Boder criteria)]. Reading disabled children were classified as: a) dysphonetic [N=8; (auditory-sequential processing deficits)]; b) dyseidetic [N=8; (visual-simultaneous processing deficits)]; or c) nonspecific [N=8; (absence of cognitive impairments)]. Sleep was recorded in the laboratory for four consecutive nights (2 adaptation, 2 baseline) using standard polysomnography. All groups exhibited variations across nights reflecting adaptation to the sleep laboratory, although these seemed attenuated in the RD subtypes relative to the Cs. Group comparisons (with nights 3-4 collapsed) were undertaken between Cs and: 1) RD children pooled into one group; and, 2) the three RD subtypes. Relative to the Cs, RD children showed: 1) more stage 4 sleep ($p < .009$); 2) less REM sleep ($p < .02$); 3) an extended initial NREM cycle ($p < .009$), composed of greater absolute amounts of stages 2 ($p < .03$) and 4 ($p < .005$); and, 4) a longer REM onset latency ($p < .009$), also

composed of more minutes of stages 2 ($p < .05$) and 4 ($p < .003$). Subtype analyses revealed that differences in REM sleep, initial NREM cycle duration and REM onset latency were largest among Cs and nonspecifics ($p < .05$; $p < .05$; $p < .01$, respectively). Eye movement density (EMD) analyses revealed that, with the exception of the initial REM period, in which the RD children (pooled) exhibited higher mean values than the Cs ($p < .05$), no significant group differences were noted over all REM periods, across the first 4 REM periods or for each individual REM period. The sleep profile observed in RD children, (the nonspecifics in particular), was characterized by a significantly extended initial NREM cycle with increased amounts of stage 4 sleep. This may reflect the influence of an underlying maturational delay, which decreases the functional quality of stage 4 sleep, resulting in a decelerated restitution process in RD children. The overall absence of differences between groups in EMD suggest that the presence or nature of its relationship to information processing in RD children remains unclear. The subtype differences observed were not expected, given Boder's description of the nonspecific subtype and suggest that her interpretation of the behavioral profile of these children may need revision.

CURRICULUM STUDIORUM

Lise Mercier was born March 31, 1962 in Ottawa, Ontario. She completed her secondary education at Ecole Secondaire De-La-Salle, in Ottawa, Ontario, in 1980. In June 1984, she received the Bachelor of Arts (Honours) degree from the University of Ottawa, Ottawa, Ontario. The title of her Honours thesis was La motivation au rendement chez les enfants canadiens-français et canadiens-anglais.

ACKNOWLEDGMENTS

The completion of this degree, and in particular of this thesis, has been a long and arduous process, but one of which I am proud and grateful to have completed. Many people have been there with me along the way and I would like at this time to express my gratitude to them. I wish to thank: Terry Pivik, for providing me with solid training in sleep research; Keith Busby, not only for his great help with data acquisition and analysis, but for being a constant and loyal source of support through very difficult times; and Joseph De Koninck, a caring and generous teacher, whose dedication to his students has been instrumental in helping me bring this project to completion. I am also very grateful to: Ken Campbell, for his insightful comments and for helping me through my statistical neurosis; Pierre Mercier and Dwayne Schindler, for their patience and help regarding statistical analyses; Monique Lortie-Lussier, for her continued interest in my studies; Ralph Nevins, not only for sharing his computer genius but for teaching me a lot about life; Stephen Butler, for his participation in data

acquisition and interesting conversations; Stella Cowley, for doing such a wonderful job with subject recruitment and screening; Sarah Dufour and Carole Lamarche, for their much needed help with crucial last minute data crunching; Kirk Brant, for helping me with the figures; Denise Grenier, for spending time at the library so that I wouldn't have to; and, Sonia Marcotte, for helping with the typing of this manuscript. I also want to thank the children and parents who participated and devoted so much of their time to make this project a reality.

Maintaining my sanity would not have been possible without the love and support of a very special family and very special friends. I am forever grateful to: my father, for being everything a loving and caring "Dad" should be; my mother, whom I miss dearly, for teaching me more about myself than she will ever know; my sister Adele, my role model, for believing in me especially when I no longer believed in myself, my brothers Paul and Louis and their families, for providing me with much needed playful distractions; and, to my friends: Gregory Christ, for making me laugh and letting me cry and for providing me with very special memories of what

a real friendship should be; Jennifer Miles, for standing by me and understanding; Janet Mullington, for her warm smile and for making the last days of thesis writing more bearable; Margo Lemelin, for being there when I needed her the most; Jocelyne Mongeon, for growing up with me and not away from me; Nicole Robillard, for being so consistently true to herself; Marie Saikaley, for her endless supplies of hugs and bagels; Marcel Viens, my wacky neighbor, for providing me with entertainment and arguments; David Latour, for his broad shoulders and for showing me that there is life after the Ph.D.; Marta Young, for her unending support and for sharing so much of herself with me; Serge Lessard, for being so generous with his time; Gilles and Marc Hébert, for welcoming me with open arms; Francine Tougas, for feeding me, mothering me and encouraging me from day one; and, with much affection, to Dave Pereira, for his love and support and for being a living proof that it is indeed never too late for a happy childhood. Finally, I want to express a very special thank you to Kerry Lawson, for caring enough to listen and for helping me find the strength to write my own script. I bring a part of you with me.

This research was supported by grants from The Hospital for Sick Children and The Ontario Mental Health Foundations awarded to Terry Pivik.

Table of Contents

Table of Contents

Table of Contents

List of Tables

List of Tables

List of Figures

List of Figures

CHAPTER 1: REVIEW OF THE LITERATURE

Specific developmental reading disabilities constitute possibly the most debilitating of learning disorders both in terms of prevalence and negative personal, social and behavioral consequences (Hynd & Cohen, 1983). It is estimated that 5 to 15% of the school-age population is afflicted by this disorder (Taylor & Taylor, 1983). A more precise estimate is difficult to obtain because of the uncertainty, contradiction and controversy still surrounding the definition and diagnosis of this entity (Adelman & Taylor, 1986; Algozzine & Ysseldyke, 1986; Epps, Ysseldyke & Algozzine, 1983,1985; Hooper & Willis, 1989).

Defining Specific Developmental Reading Disabilities

Over the past thirty years, this syndrome has gained increased attention as illustrated by the large amount of research devoted to this topic (Boder 1971a, 1971b, 1973; Critchley, 1972; Hooper & Willis, 1989; Pirozzolo, 1979; Rourke, 1985; Silver & Hagin, 1990;

Vellutino, 1979). Such research has revealed that many factors, such as low intelligence, inadequate education, serious physical, psychological and social problems, and severe brain damage, are associated with reading and spelling problems (Benton, 1975; Hynd & Cohen, 1983; Vellutino, 1977). Especially perplexing to investigators, however, are children who, although normal in other respects, still experience great difficulty in learning to read and spell (Vellutino, 1977). This is not to suggest that specific reading disabilities cannot co-exist with abnormal intellectual, emotional, educational or physical functioning, but rather they are termed 'specific' because they are not primarily caused by such factors (Boder, 1973; Boder & Jarrico, 1982). The acceptable definitions of learning, and more specifically reading, disabilities provided by professional agencies and groups (for listings see Critchley, 1972; Hooper & Willis, 1989; Hynd & Cohen, 1983) attempt to rule out handicapping factors, whether physical, mental, emotional, environmental and/or educational, which could possibly interfere with the process of learning to read or write (Boder, 1973; Hynd & Cohen, 1983). It

is thought that this elimination of associative factors by exclusion provides assurance that the reading difficulties are not an expression of these extrinsic variables (Benton, 1977; Boder, 1973; Boder & Jarrico, 1982). These definitions also attempt to highlight variables which may contribute to learning or reading disabilities, such as an underlying central nervous system (CNS) dysfunction, and most reflect researchers' presumption of a neurologic etiology (Hooper & Willis, 1989; Hynd & Willis, 1988). However, these exclusionary definitions remain quite imprecise in their description of what learning and reading disabilities actually are and, as highlighted by Epps et al. (1983, 1985) and Hooper and Willis (1989), this lack of clear, guiding operational criteria limits their use in clinical and research settings.

Homogeneity versus Heterogeneity of the Syndrome

One area of considerable controversy which has blurred the understanding of reading disabilities centers around their definition as a single or multiple syndrome entity. Many investigators have argued for a

unitary explanation and have attempted to uncover one underlying causal factor explaining the reading deficits (Doehring, Trites, Patel, & Fiedorowicz, 1981; Hooper & Willis, 1989; Malatesha & Dougan, 1982; Rourke, 1985). During the 1970's, however, the true complexity of the problem gradually became recognized. Research on cognition, language, reading, neurology and neuropsychology (reviewed in Doehring et al., 1981; Hooper & Willis, 1989) revealed the intricate interactions between these processes and offered much support for the conceptualization of learning and reading disabilities as a heterogeneous clinical entity (Doehring et al., 1981; Hooper & Willis, 1989), a conceptualization currently held by most researchers and clinicians working with this clinical population. The various unitary explanations proposed were too narrow and simplistic to provide a complete understanding of the syndrome. Nevertheless, these explanations provided fundamental ideas which have guided research attempting to characterize the multiple factors underlying specific learning and reading disabilities (Hooper & Willis, 1989). Following is a brief presentation of the various unitary factors

thought to be responsible for the disorder. [More complete reviews can be found in Doehring et al. (1981) and Hooper & Willis (1989)].

Specific Reading Disabilities as a Homogeneous Entity

Abnormal Cerebral Lateralization

Perhaps the most prominent unitary explanation of developmental learning disabilities is the absence of normal lateralization of processes, examples of which include incomplete lateralization and maturational lag in the development of cerebral lateralization (Hooper & Willis, 1989; Obrzut, 1988). The suggestion of abnormal lateralization is based upon Orton's (1937) original suggestion that a lack of hemispheric dominance is responsible for left-to-right confusions, a condition known as "strephosymbolia". Neurophysiological techniques, such as dichotic listening, have been frequently used to test the incomplete lateralization hypothesis (Bryden, 1970; Hynd & Obrzut, 1981; Obrzut, Hynd, Obrzut, & Leitgeb, 1980; Obrzut, Obrzut, Bryden, & Bartels, 1985; Sparrow, 1969; Witelson & Rabinovitch, 1972; Yeni-Komshian, Isenberg, & Goldberg, 1975; Zurif & Carson, 1970). Most of these reports found no significant differences

between normal and dyslexic readers, with both groups demonstrating a Right-Ear-Advantage (REA) for verbal material, suggesting left-hemispheric lateralization for processing linguistic information (Bryden, 1970; Sparrow, 1969; Yeni-Komshian et al., 1975). Others have found that disabled readers possess a left lateralization of linguistic processes but to a lesser degree than normal readers (Hynd & Obrzut, 1981; Obrzut et al., 1980; Obrzut et al., 1985). Taken together, these results are thought to suggest that language is lateralized to the left hemisphere in both normal and reading disabled children, thus offering no support for the incomplete lateralization hypothesis. However, they also suggest that "the efficiency of the language processor is less functional in learning disabled children" (Obrzut, 1988, p. 572). There remains a lack of consensus, however, regarding the role of cerebral lateralization in reading disability (Obrzut & Hynd, 1981).

The second hypothesis mentioned above, i.e., a maturational lag, is based upon Lenneberg's (1967) suggestion that the two hemispheres are equipotential at birth but that the left becomes more specialized for

language with increasing age. At a general level, this hypothesis predicts that learning disabled children present with a developmentally delayed central nervous system. More specifically, the development of left hemispheric specialization for linguistic processing is maturationally delayed and this negatively impacts on their ability to learn to read (Hooper & Willis, 1989; Obrzut, 1988).

Dichotic listening techniques have also been used to test the maturational lag hypothesis. Some researchers (Bakker, 1973; Bakker, Smink, & Reitsma, 1973; Bakker, Teunissen, & Bosch, 1976; Satz, Rardin, & Ross, 1971; Satz & Van Nostrand, 1972; Sparrow & Satz, 1970) have compared REA differences in younger and older normal and dyslexic readers. Their results support the notion of increased lateralization with age and that this developmental process is slower in reading disabled children. Such findings have been used by these authors as evidence for a maturational lag in learning disabilities and, as will be seen in a later section, such a concept has been incorporated in some learning disability classifications (Satz et al., 1971). Other investigators, however, (Hynd et al.,

1979; Hynd & Obrzut, 1981; Obrzut, Hynd, Obrzut, & Pirozzolo, 1981) have demonstrated that, although reading disabled children perform more poorly than normal readers, both groups show an overall REA. They could not find evidence of increasing lateralization with age and attributed the group differences to attentional factors (Hynd et al., 1979; Obrzut, 1988).

As reviewed in Hynd and Willis (1988), there is also increasing evidence at anatomical and behavioral levels which support the notion that the hemispheres are lateralized for function at a very early age. Therefore, despite the fact that this hypothesis is still viewed as a possible underlying factor in learning disabilities (Satz et al., 1971), the maturational lag hypothesis, as it relates to the development of cerebral lateralization, has received limited support and is viewed by some as untenable (Hooper & Willis, 1989; Hynd & Willis, 1988; Obrzut, 1988).

Visual-Perceptual Deficits

Some researchers have postulated visual-perceptual process deficits in specific developmental dyslexia which interfere with the normal processing of visual

information (Doehring et al., 1981; Silver & Hagin, 1966; Vellutino, 1979). An important factor in reading ability is the nature of eye-movement patterns (Rubino & Minden, 1973), and while reading normal or proficient readers execute a series of saccades, fixations and regressions (Pirozzolo, 1979; Rayner, 1978), resulting in a repetitive "staircase pattern" (Pavlidis, 1981). Reading disabled children, however, exhibit deficient eye movement patterns during reading, e.g., more fixations, regressions and return sweep inaccuracies than normal readers (Mosse & Daniels, 1959; Pavlidis, 1981; Pirozzolo, 1979, 1983; Rayner, 1978; Rubino & Minden, 1973; Tinker, 1958).

Visual-Auditory (Intermodal) Processing Deficits

Visual-auditory processes are also considered important for efficient reading permitting the translation of printed symbols into oral language (Resnick, 1979). The associative process between visual and auditory information is thought to be deficient in reading disabled children, rendering them incapable of transforming the written language into oral language (Birch & Belmont, 1964; Rubenstein, Lewis, & Rubenstein, 1971).

The role of possible disorders in auditory processes used in recoding the written language to spoken language has been investigated (Doehring et al., 1981; Tallal, 1980). Reading disabled children are thought to exhibit auditory-perceptual deficits related to the difficulty in learning sound-symbol relationships.

Linguistic Processing

Fox and Routh (1980) and Liberman and Shankweiler (1979) focus upon more specific deficits in linguistic processing as a possible unitary cause of reading disabilities. They postulate that reading achievement is correlated with the ability to segment spoken words into phonemes, and that dyslexic children lack the necessary awareness of phonemic segmentation to analyze words as strings of phonemes.

It is also suggested that deficits at higher linguistic levels could help explain reading disabilities (Doehring et al., 1981). Vogel (1977) suggests that poor readers have deficient morphological ability. Thus, they may not be making efficient use of the semantic and syntactic clues provided in the morphology of written language, resulting in greater

difficulty in combining the smallest units of meaning, the morphemes, into words. Vellutino (1979) presents a verbal processing theory which posits that verbal coding mechanisms are used for processing information in short-term memory prior to deposit in long-term memory. Efficient reading thus depends upon this ability to code information verbally, and, according to this theory, reading disabled children are not proficient in this process. They have short-term and long-term memory deficits because they lack facility in employing linguistic devices to aid recall.

Specific Reading Disabilities as a Heterogeneous Entity

As mentioned earlier, there has been an increasing realization that specific developmental dyslexia does not constitute a homogeneous diagnostic entity. As a result of this awareness, various methods have been used to identify subtypes of disabilities. These approaches may be generally classified as statistical and nonstatistical. A brief and selective review of investigations using these classification methods is presented below. [More complete reviews can be found in Doehring et al. (1981) and Hooper & Willis (1989)].

Statistical Classifications

The first major attempt to use statistical techniques to subtype reading disabled children was undertaken by Doehring and Hoshko (1977). Using the Q-technique of factor analysis, they defined three subtypes of children with reading problems. The first subtype - Type O - linguistic deficits - consisted of children with oral reading problems. This group possesses poor oral word, phrase and sentence reading skills but good silent reading, visual matching and visual-auditory matching skills. A second subtype - Type A - intersensory integration deficits - included children with intermodal association problems who are poor in rapidly matching spoken and printed letters, words and syllables (auditory-visual matching) but good at visual scanning. Finally, the third subtype - Type S - phonological deficits - consisted of children with sequential relation problems. These children are near normal in visual and auditory-visual word and syllable matching, but experience difficulty in the rapid perception of sequences of graphemes and phonemes.

Petrauskas and Rourke (1979) have attempted to classify subtypes of reading disabilities in terms of

neuropsychological performance. They administered 44 neuropsychological tests assessing tactile, sequencing, motor, visual-spatial, auditory-verbal and abstract-conceptual skills. Using the Q-technique of factor analysis they classified reading disabilities into three subtypes. These are as follows: Type 1, the largest subtype, consisting of children with mildly impaired verbal coding, word blending, and immediate memory for digits who have moderately and severely impaired verbal fluency and sentence memory. These deficits provide clear evidence of a language disturbance with difficulties in auditory-verbal memory and auditory-perceptual skills; Type 2 combines children with mildly impaired verbal fluency and concept formation. They have moderately to severely impaired finger recognition and visual-spatial memory. They also appear to have sequencing difficulties. The authors hypothesize that this combination of problems reflects a dysfunction in the posterior region of the left hemisphere; and, Type 3 composed of readers with mildly impaired finger recognition and immediate memory for digits. They have mildly to moderately impaired verbal fluency, sentence memory and immediate visual-

spatial memory, and moderately to severely impaired concept formation. Their predominant deficits are in conceptual flexibility, motoric and verbal expressive and retentive skills.

Satz and Morris (1981) performed a cluster analysis of an unselected sample of 236 boys from which they have identified five subtypes, based on achievement and neuropsychological variables. The children found in subtype 1 (N=27), a global language impairment group, showed severe impairment on all language measures but normal performance on the non-language perceptual tests. Subtype 2 (N=14), was composed of readers with a specific naming disorder, being selectively impaired on the verbal fluency test. In subtype 3 (N=10), the children were severely impaired on all neuropsychological tests (both language and perceptual), resulting in a global language and perceptual (mixed) impairment group. Subtype 4 (N=23), a visual-perceptual-motor impairment group, was characterized by selective impairment on the non-language perceptual tests but normal performance on the language tests. Finally, subtype 5 (N=12) consisted of boys having no impairment on any of the

neuropsychological tests and average to superior performance on all cognitive tests. This group constituted an unexpected learning disabled subtype.

Satz and his co-workers (Fletcher, 1981; Fletcher & Satz, 1980; Fletcher, Satz, & Scholes, 1981; Satz & Morris, 1981; Satz, Rardin, & Ross, 1971; Satz & Sparrow, 1970) have integrated the subtypes with the notion that a maturational lag in the establishment of left hemisphere lateralization underlies reading disabilities by causing a delay in the rate of acquisition of developmental reading skills. The younger readers seem to be delayed in acquiring sensorimotor-perceptual skills, necessary for the processing of graphological and phonological attributes. They eventually master these skills, but do so at an older age than normal children and, thus, are delayed in acquiring the "older" verbal-conceptual skills necessary for the semantic and syntactic processing.

The statistical classifications have not, in themselves, provided answers to the major questions concerning reading disabilities. Although these approaches are not without flaws and limitations, they

have managed to specify some linguistic and neuropsychological characteristics of different subtypes (Doehring et al., 1981; Hooper & Willis, 1989), adding to the information provided by the nonstatistical classifications.

Nonstatistical (Clinical-Inferential) Classifications

What is recognized as the first nonstatistical classification scheme was elaborated by Kinsbourne and Warrington (1963). From this model, other attempts at nonstatistical classification have developed (Hooper & Willis, 1989). These researchers identified two clinical subtypes based on subjects' performance on the Weschler Intelligence Scale for Children (WISC). Their first subtype (N=6) was labelled "language retarded" and demonstrated great discrepancy (>20 points) between scores on the Verbal and Performance Scales (PIQ > VIQ), as well as receptive and expressive language difficulties. Subtype 2, labelled the "Gerstman group" exhibited the opposite pattern, with the discrepancy between the Verbal and Performance scales favoring the VIQ. They also had accompanying finger agnosia, arithmetic difficulties and left-right confusion, demonstrating their visual-perceptual deficits.

According to Mattis, French, and Rapin (1975), fluent reading requires adequate development of language, motor-speech blending fluency and visuospatial perception. Therefore, deficiencies in these processes impair acquisition of reading skills. On the basis of this underlying hypothesis, these authors have identified three non-overlapping subtypes. Group 1 constitutes a language disorder subtype with deficiencies in naming, listening comprehension, oral sentence repetition and speech sound discrimination; Group 2 - an articulatory and graphomotor dyscoordination subtype, consists of children having deficiencies in sound blending and graphomotor ability; and Group 3 combines children with a visual-perceptual disorder, having deficiencies in various visual, nonverbal abilities.

Rourke and Finlayson (1978) classified learning disabled children into three subtypes based upon performance on the Wide Range Achievement Test (WRAT). Subtype 1 - a reading-arithmetic-spelling (RAS) group - consisted of children who scored at least two years below grade-level (< 19th percentile) on the reading, arithmetic and spelling subtests of the WRAT. The

grade-level discrepancy between the various subtests was less than one year. Subtype 2 - a reading-spelling (RS) group - consisted of children who scored below the 15th percentile on both the reading and spelling subtests. The grade-level equivalent scores on both of these subtests were at least 1.8 years below the arithmetic score. The children in subtype 3 - an arithmetic (A) group - fundamentally exhibited the reverse pattern of subtype 2. Their scores on the reading and spelling subtests were at least two years above their grade level equivalent on the arithmetic subtest. Subsequent investigations have been successful in replicating and validating this classification scheme (Breen, 1986; Hooper & Willis, 1989; Nolan, Hammeke, & Barkley, 1983; Rourke & Strang, 1978).

Pirozzolo (1979) identified two subtypes of reading disabilities. The first subtype consists of auditory-linguistic dyslexics. These children exhibit deficits in verbal-learning ability and cannot remember the phonemic features of words. It has been suggested that this deficit might be caused by the slow transmission of linguistic information within the left

hemisphere, the pathways permitting the transmission of auditory-verbal information not being fully established (Pirozzolo & Rayner, 1979). These children have reading eye movement patterns that resemble those of a beginning reader (increased number of fixations, longer fixation durations, regressions and forward saccades intermixed). Pirozzolo (1979, 1983; Pirozzolo & Rayner, 1979) suggests that this eye movement pattern reflects a linguistic processing difficulty. The second subtype, visual-spatial dyslexia, consists of children presenting deficits in visual-spatial information processing abilities. These children show faulty eye movements because of a dysfunction in the spatial mechanism that guides the eye. The author concludes that this subtype results from abnormally slow visuo-motor processing in the left hemisphere, suggesting a dysfunction in the left visual association areas.

One popular nonstatistical classification and diagnostic approach has been elaborated by Boder (1971a, 1971b, 1973; Boder & Jarrico, 1982). As will be seen shortly, this typology seems to lend itself well to the study of cognitive processing in

developmental reading disabilities and is used to define the subtypes of reading disabled subtypes in this study. A more in-depth description of this scheme will now be presented.

Boder (1971b) observed "a consistent correlation ... between the reading and spelling of a dyslexic child" (p.302), and has identified four subtypes of reading disabilities on the basis of the reading-spelling patterns of the child. Three of these patterns characterize specific developmental dyslexia, where deficits in either or both gestalt and analytic cognitive processing (to be explained in the following section) are evident. It is thought that these deficits result from an underlying neurodevelopmental lag (Boder & Jarrico, 1982). Boder's three dyslexic subtypes are as follows:

Group 1: Dysphonetic dyslexia -- These children have difficulty in the integration of written symbols with their sounds, resulting in a disability in developing phonic word-analysis skills. They rely on sight alone when approaching written material. The typical misreadings of this group are word substitutions, based on the similarity of the visual

configuration (reading 'horse' for 'house'), and semantic substitutions ('funny' for 'laugh'). Their typical misspellings are phonetically incorrect ('sleber' for 'scrambled'). Sixty-three percent of all dyslexics are thought to be found in this subtype; Group 2: Dyseidetic dyslexia -- This subtype is composed of children showing weakness in visual perception and memory for letters and whole-word configurations, with resulting difficulty in developing word-analysis skills. Their typical misreadings are phonetic pronunciations of non-phonetic words ('talc' for 'talk'). Their typical misspellings are phonetically accurate ('tok' for 'talk'). These children are characterized by visuospatial letter and word reversals in both reading and writing. This group is the smallest of the three, constituting 9% of all dyslexics; Group 3 -- Mixed Dysphonetic-Dyseidetic dyslexia: Children in this group have difficulty developing both sight vocabulary and phonic skills. They have difficulty learning what letters sound and look like. Their typical misspellings are very bizarre, such as a single, inappropriate initial letter. In reading and spelling they are plagued with

visuospatial difficulties, confusing reversible letters (b-d;p-q;m-w) and letters with subtle graphic differences (h-n;v-y). They are virtually nonreaders and nonspellers. Twenty-two percent of reading disabled children are found in this group; and, Group 4: Nonspecific reading disabled children -- These children exhibit a normal reading-spelling pattern. Boder (1973; Boder & Jarrico, 1982) suggests that the reading-spelling problems in these children are not primarily caused by weaknesses in cognitive processing resulting from a neurodevelopmental lag, but rather by physical, mental, emotional, sociocultural and/or educational factors.

Six percent of the dyslexic children studied by Boder could not be classified within a subtype. They consisted of 'undetermined dyslexics', representing remediated dysphonetics and, occasionally, older and gifted dyseidetics (Boder & Jarrico, 1982).

Cognitive Processing in Dysphonetic, Dyseidetic and Mixed Dyslexia

As Boder (Boder & Jarrico, 1982) suggests: "The observation that the reading and spelling patterns of a given child are mutually predictive led to the

hypothesis that to a very substantial extent the same cognitive strengths and weaknesses underlie reading and spelling" (p.82). The author consequently attempted to identify cognitive strengths and weaknesses inherent to each subtype, and adopted the premise that two processes are essential to reading: visual (simultaneous) and auditory (sequential) (Aaron, 1982; Baron 1979; Das, Kirby, & Jarman, 1975; Meyer, Schvaneveldt, & Ruddy, 1974; Wiener & Cromer, 1967). Familiar words (in sight vocabulary) are recognized through visual processes as visual-gestalts. Unfamiliar words are recognized through auditory processes by phonetic analysis (Myklebust, 1965). In normal readers, there is an automatic interplay of both visual-gestalt and auditory-analytic information processes (Das et al., 1975). In the dyslexic child, however, this interplay is dissociated (Boder, 1971a, 1971b). Thus the dysphonetic's reading-spelling pattern suggests a major weakness in auditory-analytic (sequential) skills but a normal functioning of the visual-gestalt cognitive function. The dyseidetic child's reading-spelling patterns exhibit weakness in the visual-gestalt (simultaneous) function but a normal

functioning of auditory-analytic skills. The mixed dysphonetic-dyseidetic dyslexic's reading-spelling patterns indicate weaknesses in both information processing strategies. Using this framework, specific developmental dyslexia can be defined as "a reading disability in which the reading and spelling performance gives evidence of cognitive deficits in either the visual-gestalt function, or auditory-analytic function or both. A corollary of this definition is that when the reading-spelling pattern of poor readers gives no evidence of such cognitive deficits, the reading disability is regarded as nonspecific rather than dyslexic." (Boder & Jarrico, 1982, p.5).

Boder's model has also permitted the elaboration of a diagnostic test - The Boder Test of Reading-Spelling Patterns (BTRSP - Boder & Jarrico, 1982) - which facilitates the grouping of reading disabled children into one of the various subtypes based on their reading (word recognition) and spelling performances.

Evaluation of the Boder Classification Scheme and of the BTRSP

Research evidence (reviewed in Hooper & Willis, 1989) has provided mixed support for both Boder's classification scheme and for the BTRSP. Studies looking at various neuropsychological factors, such as simultaneous-sequential processing, intrasensory integration and visual memory, have found differences between the dysphonetic and dyseidetic dyslexic subtypes (Aaron, 1978, 1982; Bauserman & Obrzut, 1981; Bayliss & Liversey, 1985; Dalby & Gibson, 1981; Menken, 1981; Nockelby & Galbraith, 1984). However, other studies investigating reading disabled children's performance on a variety of cognitive tasks (Holmes & Peper, 1977; Hooper & Willis, 1989) could not distinguish between the dysphonetic and dyseidetic subtypes, although differences in performance were obtained between a pooled dyslexic group, controls, and nonspecific reading disabled children. As well, investigations using dichotic listening techniques and hemiretinal tasks have also provided mixed support for Boder's classification. While some authors reported evidence of hemispheric differences between the

subtypes (Malatesha & Dougan, 1982), Aylward (1984) could not differentiate between the dysphonetics' and dyseidetics' performances.

Electrophysiological studies of EEG and event-related potentials (ERPs) have also provided mixed support for Boder's classification. Fried (1979) and Fried, Tanguay, Boder, Doubleday and Greensite (1981) studies ERPs in normal, dysphonetic and dyseidetic readers. They found that the dyseidetic and normal children demonstrate significantly greater waveform differences between ERPs to word and musical chords over the left as compared to the right hemisphere. The dysphonetics did not show such asymmetry, suggesting that the dysphonetic readers may not have normal left hemispheric specialization for auditory-linguistic information-processing. The dyseidetics, presumed to have skills in sequential processing, did not differ from normal readers on this respect. Fried (1979) and Fried et al. (1981) have not found evidence of a right hemisphere dysfunction thought to underlie the visual-spatial difficulties of the dyseidetics.

Rosenthal, Boder and Calloway (1982) have also reported hemispheric differences between dysphonetic

and dyseidetic subtypes. Leisman and Ashkenazi (1980), however, were unable to find any distinctions between reading disabled subtypes. Flynn and Deering (1989) found that during cognitive tasks dyseidetics exhibited increased theta activity in the left hemisphere in the area of the angular gyrus, a region thought to be important for phonetic and sequential processing of linguistic material. They have suggested that the dyseidetic children's reading impairments may stem from over-use of linguistic abilities rather than visual-gestalt deficits as suggested by Boder.

As mentioned previously, research findings regarding the validity and reliability of the BTRSP have also been mixed. Although some investigators have found the BTRSP to be both a reliable and valid diagnostic instrument (Camp & Dolcourt, 1977; Ginn, 1979; Whiting & Jarrico, 1980), others have identified problems which may limit its usefulness. Reynolds (1984), for example, found the BTRSP to be weak on many important psychometric concepts. He suggests that the lack of a standardized procedure regarding administration of the spelling test, as well as the lack of normative data and standard scores, contribute

to the inappropriateness of using this as a diagnostic tool for reading disabled children. Alexander (1984) raised serious concerns regarding the theoretical background upon which the BTRSP rests. She suggests that the test's exclusion of reading comprehension [not exclusive to this scheme, however (Hynd & Hynd (1984)] and failure to acknowledge the interactive aspects of reading, oversimplifies the reading process as well as the cognitive processing underlying reading and spelling. Overall, she believes that the BTRSP "promises much but delivers little" (Alexander, 1984, p. 532).

Finally, the precision with which the BTRSP appropriately diagnoses and subtypes children presenting specific reading disabilities has been under scrutiny. Some researchers have found that it frequently misdiagnoses children either through a high rate of false negatives or, more specifically, misdiagnosing severely deficient readers as nonspecifics (Lund, Yingling, Galin, Marcus, & Simons, 1984; Nockelby & Galbraith, 1984).

Clearly, these mixed results on both Boder's classification scheme and the BTRSP raise concerns

regarding their overall usefulness. As Hooper and Willis (1989) suggest: "Although Boder is commended for providing a systematic procedure to complement her subtyping model .... the efficacy of this test and the subtyping model from which it was derived await further clarification" (p. 133).

Learning Disabilities Classifications: Unresolved Issues

As can be seen from this review of the various statistical and nonstatistical typologies, at least three subtypes of learning disabilities are repeatedly identified: an auditory-linguistic subtype, a visual-perceptual subtype, and a combined or mixed subtype. This finding underscores the importance of viewing this clinical entity within a multidimensional framework. However, although the subtyping research has helped increase researchers' and clinicians' knowledge of learning disabilities, important issues regarding these classifications remain unresolved and limit the understanding of this disorder (Hooper & Willis, 1989; Hynd & Hynd, 1984; Rourke, 1985, 1991).

From the numerous classification models available, it is clear that there is still no generally accepted

typology. This lack of consensus, resulting in large part from the definitional issues previously mentioned, demonstrates the importance of resolving and clarifying the conceptual bases upon which the subtyping research rests (Hooper & Willis, 1989).

Another important issue, which limits the contribution of the subtyping research, is the degree to which underlying etiological factors are identified. Most classification schemes assume neurologic etiological factors (Boder, 1973; Boder & Jarrico, 1982; Hooper & Willis, 1989; Hynd & Willis, 1988) but, with the exception of Rourke (1987) who has proposed a theory concerning neuroanatomical factors underlying nonverbal learning disabilities, the various typologies do not provide information regarding what these specific factors might be (Hynd & Hynd, 1984). Therefore, "more research delineating specific structure-function linkages clearly is needed, particularly with respect to its application to learning disability subtypes" (Hooper & Willis, 1989, p. 28).

Neurological/Neurolinguistic Models of Reading Disabilities

One attempt to delineate "structure-function linkages" is evident in the elaboration of neurological/neurolinguistic models of reading and of reading deficits (Hynd & Hynd, 1984; Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990; Marshall & Newcombe, 1973, 1980; Sevush, 1983). These models are based on Luria's theory of functional brain organization (1970, 1980) and description of a functional system for reading which postulate that various cortical areas (left and right medial occipital lobes, angular gyrus, Wernicke's area, arcuate fasciculus, Broca's area) work in an integrated fashion at different stages in the process of reading. The models suggest that abnormalities or deficits at any point in any cortical region or neural pathway involved in the system for reading will make it less functional and result in a variety of reading deficiencies, such as those described in the subtyping typologies (Hynd & Hynd, 1984). Evidence in support of the neurological/neurolinguistic models can be found in electrophysiological and postmortem studies.

Electrophysiological Studies

Using brain electrical activity mapping (BEAM), Duffy and his colleagues, (Duffy, Burchfield & Lombroso, 1979; Duffy, Denckla, Bartels & Sandini, 1980; Duffy, Denckla, Bartels, Sandini, & Kiessling 1980) have found increased alpha and theta activity (which they have interpreted as being suggestive of relative underactivation) in Broca's area, the left temporal area, the left posterior quadrant (including Wernicke's area) and the mediofrontal lobes. Most of these areas are thought to be in the functional system for reading (Hynd & Hynd, 1984). Subsequent observations in dyslexic adults of hypometabolic activity in the left temporal region and in the left posterior quadrant during reading, offer some support for a notion of relative cerebral underactivation in this population relative to normal readers (Gross-Glenn, Duara, Yoshii, Barker, Chang, Apicella, Boothe, & Lubs, 1987, 1990).

Postmortem Studies

Cytoarchitectonic studies (Drake, 1968; Galaburda, Corsiglia, Rosen, & Sherman, 1987; Galaburda & Eidelberg, 1982; Galaburda & Kemper, 1979) have also

provided evidence in support of developmental abnormalities in dyslexic brains in the regions associated with the functional system for reading (Hynd & Hynd, 1984). Such studies have demonstrated that anomalies, such as symmetry of the left temporal plane, cortical dysgenesis, and fibromyelin plaques, are frequently present in dyslexic brains and may possibly result from disruptions in cellular migration and organization during the maturation of the brain (Hynd & Willis, 1988). Recent evidence (Galaburda, 1990; Geshwind & Behan, 1982; Pennington, Smith, Kimberling, Green, & Haith, 1987; Sherman, Rosen & Galaburda, 1989) suggests that these neurodevelopmental errors which may stem in part, from autoimmune disorders and/or from abnormalities in the cell death process, disrupt normal corticogenesis (Duane, 1991; Hooper & Willis, 1989; Hynd et al., 1990; Hynd & Hynd, 1984), and may possibly delay the maturation of the left hemisphere and disrupt the establishment of normal linguistic functioning (Geshwind & Behan, 1982). One recent magnetic resonance imaging investigation has documented significant morphological differences in the left planum temporale of dyslexic children relative to age-

matched normal controls and subjects with an Attention Deficit Disorder with Hyperactivity (Hynd et al., 1990). These authors postulate that absences of "significant differences between these groups in total brain area suggest that this regional variation in brain morphologic characteristics is due to a more specific deviation in brain ontogeny" (p. 922). Such an hypothesis implicates once again the possible role of maturational issues in reading disabilities but not in relationship to the development of cerebral lateralization per se, as previously discussed. Rather, it would seem to relate specifically to the maturation of the various cerebral regions (and to their various interconnections) thought to be implicated in the reading process and known to be subjected to pre and postnatal ontogenetic influence (Hynd & Willis, 1988).

Support for these neurological/neurolinguistic models is still limited, and many of the supportive studies have been criticized on methodological grounds (in particular those involving autoimmune factors - Hynd & Semrud-Clikeman, 1989; Taylor & Fletcher, 1983). Clearly, more research is needed before the neurologic

etiological factors underlying learning and reading disabilities are positively identified. Nonetheless, the models presented offer some insight into the possible relationship between structural and functional deficits encountered in learning and reading disabilities, and how these may be reflected in their particular subtypes. The incorporation of such ideas into the subtyping research seems necessary in order to provide a better explanation of the basis for learning disabilities as opposed to simply providing a description of the various deficiencies encountered. As Hooper and Willis (1989) aptly point out, however, Luria's theory, on which the neurological/neuro-linguistic models are based, stems from research undertaken in adults. As such, this limits the generalizations that can be made to a pediatric population in which brain-behavior relationships are more dynamic. Future subtyping models will need to incorporate such neurodevelopmental considerations.

Clearly, much progress has been made over the past twenty years with regards to empirical and clinical understanding of specific developmental learning disabilities. Perhaps the most noticeable advance has

been in the conceptualization of this disorder as a heterogeneous, rather than a homogeneous, clinical entity. As well, recent research efforts have attempted to identify and clarify possible neurologic etiological factors and relate these to the various learning disability subtypes. Despite this progress, however, more studies are required if greater understanding of learning and reading disabilities is to be achieved.

Research into the behavioral and etiological features associated with this disorder has focused on variations during wakefulness where the negative effects impact most noticeably. However, studies of sleep could prove useful since much is now known regarding normative aspects of sleep patterning and events, and these activities have been related to both biological substrates and developmental factors (Feinberg, 1974; Hobson & McCarley, 1977; Pivik, 1983; Roffwarg, Muzio, & Dement, 1966) which are important considerations in learning disability research.

The Study of Sleep in Specific Developmental Reading Disabilities

The study of sleep patterns and events provides the opportunity to obtain valuable physiological information free from the distracting variables of wakefulness and permits cross-state comparisons. Much is now known regarding the normative expression of sleep patterns and events in both adult and pediatric populations (Busby, Firestone, & Pivik, 1981; Carskadon, 1982; Coble, Kupfer, Taska, & Kane, 1984; Coble, Kupfer, Reynolds, & Houck, 1987; Feinberg, 1974; Williams, Karacan, & Hursch, 1974). Studies investigating sleep patterns and events have also been undertaken in pediatric clinical disorders such as autism (Ornitz, 1972, Ornitz, Ritvo, Brown, La Franchi, Parmelee, & Walter, 1969), hyperactivity (Busby et al., 1981; Haig, Schroeder, & Schroeder, 1974; Khan & Rechtschaffen, 1978; Stahl, Orr, & Griffiths, 1979), minimal brain damage (Feinberg, Hibi, Brown, Cavness, Westerman, & Small, 1974; Small, Hibi & Feinberg, 1971) and mental retardation (Petre-Quadens, 1972; Petre-Quadens & de Lee, 1970). Except for abstracted

preliminary findings (Mercier, Pivik, Busby, & Butler, 1988,1990; Sheldon, Spire, & Levy, 1990) there are no reports regarding electrographic sleep characteristics in children with reading disabilities. One study has been conducted dealing with cognitive (dreaming) behavior during rapid eye movement (REM) sleep in these children, and this study reported differences in the characteristics of dreams in reading disabled children relative to controls (Butler & Pivik, 1991). Another study (Hilakivi, 1987), which used subjective sleep questionnaires and sleep and dream home diaries, reported increased sleep difficulties and lower dream recall in learning disabled children compared to controls. The examination of sleep patterns in reading disabled children would therefore provide needed information regarding the organization of sleep in this pediatric clinical population, which can be used to address theoretical and empirical issues related to this population, such as physiological features (e.g., maturational delay) and cognitive deficits (impaired information processing capability). These particular issues will be considered below.

Sleep and Maturational Issues

The ontogenetic development and expression of sleep patterns in normal individuals is well documented (Carskadon, 1982; Coble et al., 1984, 1987; Feinberg, 1974; Pivik, 1983; Roffwarg et al., 1966; Williams et al., 1974) and indicates that the study of sleep behavior in reading disabled children could provide information relevant to suggested physiological bases for reading disorders such as maturational delay (Bakker, 1973; Fletcher et al., 1981; Geshwind & Behan, 1982; Hynd & Willis, 1988; Satz et al., 1971). Variables such as amounts of REM and Slow Wave (SWS) sleep and latency to the first REM period have been found to decrease with age, whereas eye movement density during REM sleep increases with age. Thus, these have been linked to central nervous system (CNS) maturation and function (Dittrichova, Paul, & Pavlikova, 1972; Feinberg, 1974; Feinberg & Evarts, 1969; Roffwarg et al., 1966; Williams et al., 1974). The relationship between sleep variables and CNS maturation is suggested by findings that REM sleep deprivation negatively impacts on brain maturation in some animals (Mirmiran, 1986; Oksenberg, Farber, Marks,

Speciale, Mihailoff, & Roffwarg, 1985; Oksenberg, Marks, Farber, Cobbey, Speciale, Mihailoff, & Roffwarg, 1986; Shaffery, Marks, Speciale, & Roffwarg, 1991). As well, differences in some sleep parameters have discriminated normal children from those presenting with a variety of developmental disorders. For example, an extended REM latency has been found in hyperactive relative to normal children (Busby et al., 1981), and reduced eye movement density during REM sleep was shown in autistic and mentally retarded children compared to normal children of the same age (Ornitz, 1972; Ornitz et al., 1969; Petre-Quadens, 1972; Petre-Quadens & de Lee, 1970). Therefore, variations in these more specific parameters in reading disabled children may be used as an indication of underlying maturational status.

Sleep and Information Processing (Cognitive) Issues

As previously mentioned, many sleep related variables have been linked to the maturation and function of the CNS, features which may be necessary for normal cognitive development (Busby & Pivik, 1983; Roffwarg et al., 1966). Many authors (De la Pena, Zarcone, & Dement, 1973; Dewan, 1970; Feinberg &

Evarts, 1969; Morruzzi, 1966) have postulated that sleep, REM sleep in particular, may be related to information processing. Many of these theories suggest that REM sleep serves an essential cognitive function.

One of the most influential theories linking REM sleep to information processing in humans has been elaborated by Dewan (1970). His Programming (P) Hypothesis suggests that new functional structures (programs) are established in the brain during REM sleep, permitting memory to be consolidated according to the individual's needs. This activity reflects an adaptive function of REM sleep which may enable individuals to cope with their changing environments.

De la Pena et al. (1973) put forth a Sensory Control for Information Processing (SCIP) Hypothesis for REM sleep. These authors suggest that sensory information flow during REM sleep compensates for the relative "sensory deprivation" occurring in mature individuals who, during wakefulness, process information with efficient cognitive structures. This compensatory function is thought to be illustrated by a positive relationship between the rapid eye movements of REM sleep and those of wakefulness, implying "a

correlation between certain parameters of waking perceptual-cognitive processes and REM sleep dreams, the nature of which is influenced in large part, by experiential or maturational (developmental) considerations" (De la Pena et al., 1973, p.499). The suggestion of a positive relationship between the rapid eye movements of REM sleep and waking saccades, however, has been debated. A similiarity between waking and REM sleep eye movements has been reported by several investigators (Herman, Barker, & Roffwarg, 1983; Herman, Erman, Boys, Peisir, Taylor, & Roffwarg, 1984; Jeannerod & Mouret, 1962). Jacobs, Feldman, and Bender (1971), however, have denied the similarity between eye movements in these two states. They suggest that eye movements in REM sleep are not random and are similar in every subject. Herman et al. (1983) have pointed out, however, that these authors did not monitor waking eye movements, thus making it unclear "whether eye movements in REM sleep are different from waking saccades under any or all conditions" (p.538). A more recent study by Aserinsky, Lynch, Mack, Tzankoff, and Hurn (1985) compared waking and REM sleep eye movements and found REM sleep eye movements to be

considerably slower than those of wakefulness.

Many investigations have attempted to determine the relationship between REM sleep and information processing. (Reviews of these studies can be found in McGrath & Cohen, 1978; Horne & McGrath, 1984; and Smith, 1985). One approach has been to examine the effects of REM sleep deprivation on learning or memory for a task. The majority of these studies suggest that REM sleep deprivation interferes with memory processing, and these findings have been used to argue for an information processing function for REM sleep (Fishbein & Gutwein, 1977, 1981; Greenberg, 1970, 1981; Hartmann, 1981; Pearlman, 1981; Smith & Kelly, 1988). However, the techniques used in REM deprivation experiments (pedestal or 'flower pot' techniques in animals for example), as well as the frequent awakenings, provoke increased levels of stress and anxiety. Therefore, such paradigms have been criticized since it is difficult to determine whether the negative effects on learning and memory are due solely to REM sleep deprivation or to some interactions with these potentially confounding variables (Albert, 1975; Fishbein & Gutwein, 1977; Horne, 1988; Smith,

1985; Vogel, 1975).

The relationship between REM sleep and information processing has also been investigated by examining changes in sleep parameters (REM sleep in particular) following various types of new learning experiences. Using this approach, partial support for theories linking REM sleep to information processing can be found in studies examining the psychophysiological effects of altered visual experiences on sleep. Initially, Zimmerman, Stoyva, and Metcalf (1970) demonstrated increased amounts of REM sleep in subjects wearing vision-distorting prism glasses during waking. De Koninck and Prévost (1991) and Prévost, De Koninck, Barry, and Broughton (1974) found increases in REM sleep and decreases in both horizontal and vertical rapid eye movement density in subjects wearing inverting prism goggles during wakefulness. These authors (De Koninck & Prévost, 1991; Prévost et al., 1974) interpreted these results as supporting both Dewan's (1970) P Hypothesis and De la Pena et al.'s (1973) SCIP Hypothesis. However, experiments conducted by Allen, Lewis, and Tagney (1972) and a later study by Zimmerman, Stoyva, and Reite (1978),

have not replicated these results. These authors did not find any effects of spatially altered vision on REM sleep and concluded that the information processing theories of REM sleep could not be firmly supported.

Stronger evidence in support of a link between REM sleep and information processing comes from investigations studying the effects of intensive learning on REM sleep. Animal studies, using a variety of learning tasks (Bloch, Hennevin, & Leconte, 1981; Smith, 1985; Smith & Butler, 1982; Smith & Lapp, 1986; Smith, Young, & Young, 1980), and human studies, using such paradigms as intensive language learning and intensive studying (Christ, Hébert, Weicklein, Mercier, & De Koninck, 1991; De Koninck, Christ, & Lorrain, 1987; De Koninck, Lorrain, Christ, Proulx, & Coulombe, 1989; De Koninck, Proulx, Healy, Arsenault, & Prévost, 1975; Smith & Lapp, 1991), have found significant increases in amounts of REM sleep following effective learning. Many of these investigations also found increases in number and density of rapid eye movements (Christ et al., 1991; De Koninck et al., 1987, 1989; Smith & Lapp, 1986, 1991), suggesting a possible relationship between these more specific REM parameters

and information processing. As previously mentioned, reduced rapid eye movement activity in REM sleep has been found in developmentally delayed autistic and mentally retarded children, both of whom display significant cognitive deficits (Ornitz, 1972; Ornitz et al., 1969; Petre-Quadens, 1972; Petre-Quadens & de Lee, 1970). Such results seem to offer additional support for a possible relationship between EMD and efficient cognitive processing. In what seem to be contradictory findings, Busby and Pivik (1983) observed a reduction in eye movement density during REM sleep in superior IQ children. They suggested, however, that the relationship between eye movement and information processing may not necessarily be linear, and that the reduced intensity of eye movements in these subjects may reflect more efficient information processing during both wakefulness and sleep. Whatever the direction of the relationship, however, there is support linking not only REM sleep per se, but the phasic events of this stage as well, to information processing (Christ et al., 1991; Smith & Lapp, 1991).

The preceding reviews have underscored the complexity of the reading disability syndrome and the

important information which a study of sleep patterns and events in this disorder could provide.

The investigation to be described applies psychophysiological sleep methcdology to the study of reading disabled children. An inescapable conclusion from the review of the reading disability literature is that this is a heterogeneous syndrome. Consequently, this study classifies reading disabled children into subtypes according to the scheme of Boder (Boder & Jarrico, 1982). This investigation will also provide additional information regarding the research and clinical usefulness of this classification scheme and of the related BTRSP.

Hypotheses

The preceding review of the literature leads to the formulation of the following hypotheses:

1) Basic sleep patterns (sleep stage amounts) and associated parameters (sleep onset latency, total sleep time, wake after sleep onset, sleep efficiency, cycle durations - with the exception of the initial NREM cycle - see hypothesis 2) will not differ significantly

across groups.

This hypothesis is suggested by: studies in allied clinical groups - hyperactive children in particular (Busby et al., 1981) - which did not document any significant differences in baseline sleep patterns between these children and normal controls.

2) REM latency will be significantly extended in reading disabled relative to control subjects. More specifically, dysphonetic and dyseidetic children will exhibit a longer REM latency relative to nonspecific reading disabled and control children.

This hypothesis is suggested by: a) the frequently proposed maturational delay in specific developmental dyslexia (Bakker, 1973; Bakker et al., 1973; Boder & Jarrico, 1982; Geshwind & Behan, 1982; Hooper & Willis, 1989; Hynd et al., 1990; Hynd & Hynd, 1984; Satz et al., 1971). Since REM latency becomes shorter with maturation (as illustrated by the longer REM latency in young children compared to older children - Coble et al., 1984; Feinberg, Koresko, & Heller, 1967; Roffwarg et al., 1966), it is therefore expected that maturationally delayed children will exhibit an extended REM latency relative to controls; and, b) the

noted extended REM latency in hyperactive children (Busby et al., 1981).

3) Dysphonetic, dyseidetic and nonspecific reading disabled children will show reduced eye movement density (EMD) in REM sleep compared to controls, and dysphonetics and dyseidetics will exhibit reduced eye movement density relative to the nonspecific reading disabled children.

This hypothesis is suggested by: a) the postulated relationship between EMD in sleep and waking information processing capabilities (Christ et al., 1991; De Koninck et al., 1987; De Koninck & Prévost, 1991; De la Pena et al., 1973; Petre-Quadens & de Lee, 1970; Smith & Lapp, 1991) and the reported cognitive dysfunction in specific developmental dyslexia (Boder, 1971a, 1971b, 1973; Boder & Jarrico, 1982; Pirozzolo, 1979; Rourke, 1985); and, b) the observed decrease in EMD in children with developmental disorders (Ornitz, 1972; Ornitz et al., 1969; Petre-Quadens, 1972; Petre-Quadens & de Lee, 1970) and the proposed maturational delay in specific developmental dyslexia (Bakker, 1973; Bakker et al., 1973; Geshwind & Behan, 1982; Hooper & Willis, 1989; Hynd et al., 1990; Hynd & Hynd, 1984;

Satz et al., 1971).

Reading disabled children are thought to exhibit inefficient and/or deficient eye movements while engaged in the cognitive task of reading (Pavlidis, 1981, 1983; Pirozzolo, 1979, 1983; Pirozzolo & Rayner, 1979). During REM sleep, a state positively related to information processing (De la Pena et al. 1973; Dewan, 1970), it is therefore expected that the dysphonetic and dyseidetic children will exhibit reduced EMD (similar to that found in reading - Pirozzolo, 1979), which reflects their information processing deficits.

Although the reading difficulties of the nonspecific reading disabled children are not thought to result from a physiologically based cognitive dysfunction (as is suggested for the specific developmental dyslexics - Boder, 1971a, 1971b, 1973; Boder & Jarrico, 1982; Pavlidis, 1981, 1983; Pirozzolo, 1979), they nonetheless experience certain difficulties in processing visual material. In light of this, it is expected that EMD in REM sleep will be reduced in this group but to a lesser extent than that of the dysphonetic and dyseidetic children.

CHAPTER 2: METHODOLOGY

Subject Selection

Thirty-nine eight to ten year-old boys served as subjects. Fifteen of these boys were control (C) subjects (mean age= 9.2 years, SD= 0.6). They fell within the normal range of intellectual and academic functioning and were free from psychological, learning and medical disorders. A second group of twenty-four boys (mean age= 9.0 years, SD= 0.5) presented with specific reading disabilities (RD; criteria to be defined in Procedures section). They had an estimated IQ $\geq$ 80 and were free from emotional problems, sensory impairments, physical handicaps or gross brain damage. All subjects were unmedicated and free from sleep disorders. Control subjects were selected from the local school system. The RD children were recruited from the local school system (learning disabled classes), and the Children's Hospital of Eastern Ontario. The experimental procedures were fully explained to all subjects and their parents and informed consent was obtained prior to participation in

the study. Subject group characteristics are presented in Tables 1 and 2.

Procedure

Testing and Selection Procedure

Specific factors in subject selection were controlled by test measures obtained during one session of daytime testing requiring approximately 1.5 hours per subject.

The short form WISC-R (Kaufman, 1976) was used for the assessment of intellectual functioning of all subjects. An estimated Full Scale IQ $\geq$ 80 was required to include a child in the study.

The Boder Test of Reading-Spelling Patterns (Boder & Jarrico, 1982) was used to determine the presence, or absence, of a reading disability. The performance of the RD children on this test provided the basis for their allocation into one of three reading disabled subtypes defined by Boder (1971a, 1971b, 1973), i.e., a) dysphonetic [DP (n=8); mean age= 9.1, SD= 0.6]; b) dyseidetic [DE (n=8); mean age= 8.7, SD= 0.6]; and c) nonspecific [NS (n=8); 9.2, SD= 0.5). The choice of

Table 1

Mean (SD) Age, I.Q. and Reading Quotient for Control (C) and Reading Disabled (RD) Pooled Groups

Variable	Group	
	C	RD
Age	9.2 (0.6)	9.0 (0.5)
I.Q. - Short Form WISC-R*	117.2 (7.3)	101.7 (10.3)
Reading Quotient*	142.0 (16.4)	79.4 (7.7)

note: in this and in subsequent tables asterisks (*) denote main effects.

*p<.0001.

Table 2

Mean (SD) Age, I.Q. and Reading Quotient for Control and Reading Disabled Subtyped Groups

Variable	Group			
	C	DP	DE	NS
Age	9.2 (0.6)	9.1 (0.6)	8.7 (0.6)	9.2 (0.5)
I.Q. - Short Form WISC-R*	117.2 (7.3)+	106.3 (7.5)	98.0 (11.2)+	100.8 (11.2)+
Reading Quotient*	142.0 (16.4)+	82.0 (6.3)+	75.1 (7.1)+	81.1 (8.6)+

note: in this and in subsequent tables asterisks (*) denote main effects; crosses (+) denote post-hoc pair-wise differences. DP = dysphonetic group; DE = dyseidetic group; NS = nonspecific group.

*p<.0001. +p<.01.

these subtypes permitted a comparison between RD children with and without specific cognitive impairments. Briefly, the presence of a specific (dysphonetic or dyseidetic) reading disability was confirmed when: a) the child's reading level was at least one year below his grade level; and, b) there was a discrepancy between the child's performance on the spelling test and on the reading test. Typically, the performance on the spelling test was lower than that on the reading test (50% or more of the Known Words - words in the sight vocabulary - were misspelled).

The presence of a nonspecific reading disability was confirmed when: a) the reading retardation was usually less than two years below grade level; and, b) the child had a normal reading-spelling pattern, not reflecting cognitive deficits in either the visual or auditory modality.

Sleep Recordings

Subjects' sleep patterns were recorded for four consecutive nights on which total bedtime (TBT) was limited to a maximum of nine hours per night. These recordings allowed for adaptation to sleep recording conditions (nights 1 and 2) and provided an adequate

database for baseline sleep evaluations (nights 3 and 4). Electroencephalographic activity recorded from central (C3/A2) and occipital (O1/A2) placements was used for sleep staging. Direct coupled bipolar horizontal and vertical electrooculographic (EOG) activities were recorded from electrodes placed at the outer canthus of each eye (horizontal) and on the superior and inferior orbital ridge of one eye (vertical), respectively. Facial muscle activity (electromyogram: EMG) was recorded from two electrodes placed over the orbicularis oris muscle. The inter-electrode impedance was <5 kOhms for all electrode pairs. Polygraphic data were recorded on paper write-out (Grass model 78D Polygraph) at a chart speed of 10 mm/sec and stored on magnetic tape (Hewlett Packard 8868A tape recorder).

Data Analysis

Polygraphic recordings were coded and independently scored according to standardized criteria (Rechtschaffen & Kales, 1968) by two individuals with previously established scoring reliability (> .9) The

following variables were determined: sleep onset latency (SOL), total sleep time (TST), total wake time (TWT), sleep efficiency index (SEI; TST/TBT), sleep stages 1,2,3,4 and REM, latency to the first REM period, total movement time (MT), wake after sleep onset (WASO) and REM and NREM sleep cycle lengths. REM cycles were defined as total time recorded from the onset of the first REM period to the onset of the second REM period, from the onset of the second REM period to the onset of the third REM period, etc. NREM cycles were calculated using the duration of total time recorded from the onset of stage 2 in the first cycle to the onset of stage 2 in the second cycle, from the onset of stage 2 in the second cycle to the onset of stage 2 in the third cycle, etc (a method similar to the one described by Feinberg & Floyd, 1979). Both REM and NREM cycles were calculated with WASO included and deleted.

Eye movement density during REM sleep was measured by calculating the number of 2-sec epochs containing at least one eye movement ($\geq 5^{\circ}$ visual angle; $\geq 40^{\circ}$ /sec velocity) divided by the total number of 2-sec epochs of REM sleep (Aserinsky, 1969). Separate density

measures were calculated as a function of night, for the first four REM periods pooled, and for individual REM periods.

Group comparisons for sleep patterns and parameters and EMD characteristics were made using one-way analyses of variance (BMDP2V -nights 3 and 4 collapsed) and, when appropriate, post hoc Tukey tests. To investigate whether the results obtained varied as a function of whether reading disability is considered as a homogeneous or a heterogeneous entity, and to see if various subtype differences in sleep patterns and EMD could be shown, group comparisons were made: 1) between C subjects and all RD children pooled into one group [RD (pooled), N=24]; and, 2) between C subjects and the three subtypes of RD children [RD (subtyped) - dysphonetic (DP): N=8; dyseidetic (DE): N=8; and nonspecific (NS): N=8].

CHAPTER 3: RESULTS

Adaptation Effects

Previous investigations have documented that both adults (Agnew, Webb, & Williams, 1966: Rechtschaffen & Verdone, 1964; Schmitt & Kaelbling, 1971) and children (Busby et al., 1981; Carskadon, Keenan, & Dement, 1987; Coble et al., 1987) undergo a period of adaptation to the sleep laboratory environment which decreases the overall quality of sleep on night 1 relative to subsequent recording nights ("first-night effect" - Agnew et al., 1966). Adaptation effects are usually illustrated by increases across nights in TST, SEI, and amounts of REM sleep and decreases across nights in TWT, SOL, WASO, and stage 1 sleep.

Two-way analyses of variance having one between factor [groups: C - RD (pooled); and C - RD (subtyped)] and one within-group factor [nights: nights 1,2,3 and 4] were undertaken to document the presence of adaptation effects. (Figures 1 to 7 illustrate these results). Since group main effects were not observed for TST (C - RD (pooled) and C - RD (subtyped), results

Figure 1
Mean Values of Total Sleep Time Across Nights

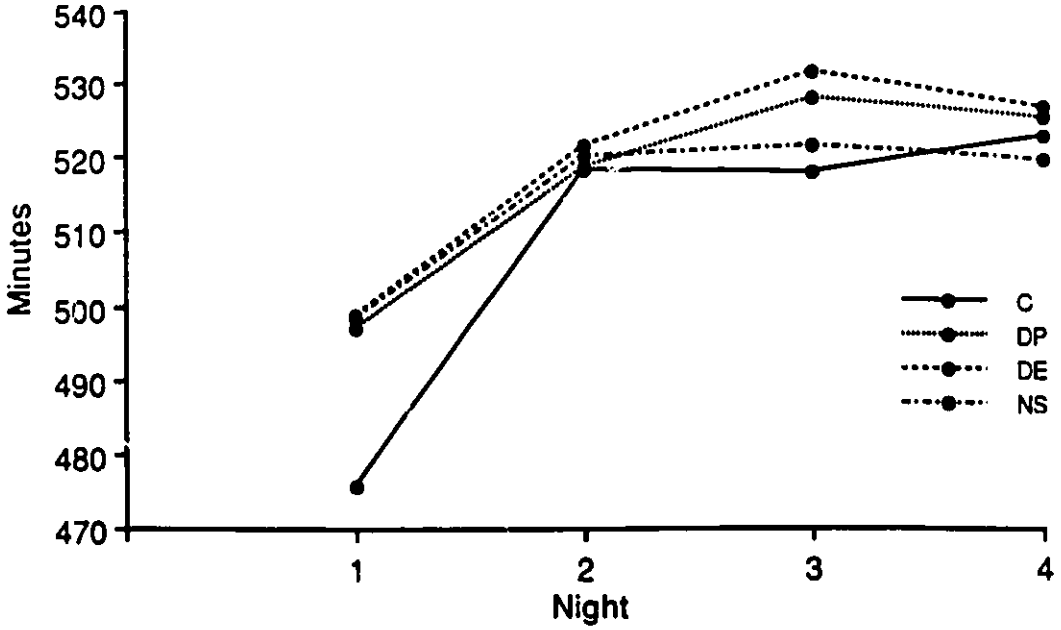


Figure 2
Mean Values of Sleep Onset Latency Across Nights

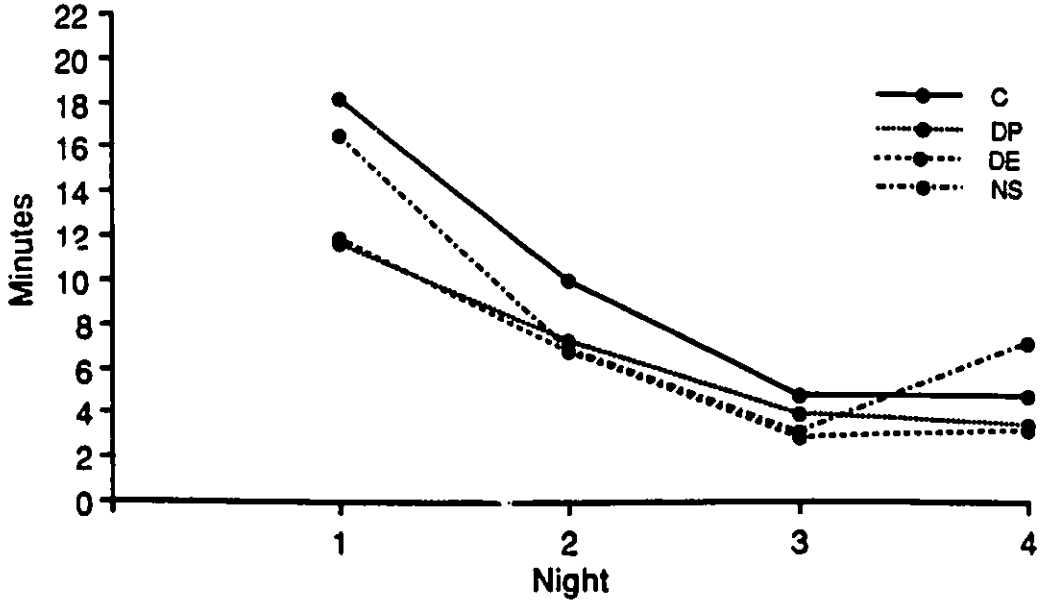


Figure 3
Mean Percentage Values of Sleep Efficiency Across Nights

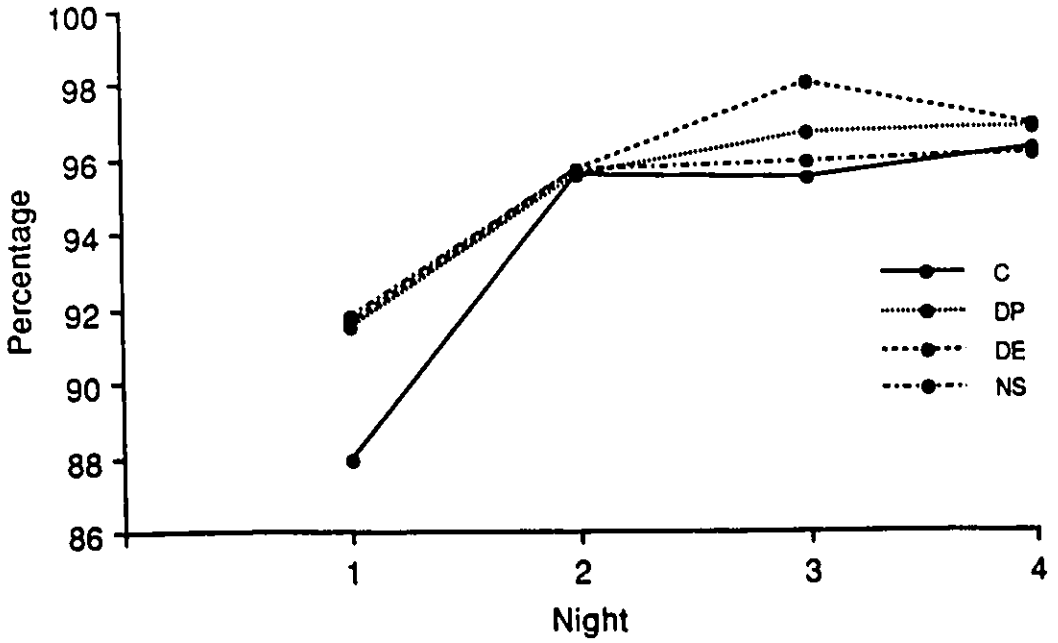


Figure 4
Mean Percentage Values of Total Wake Time Across Nights

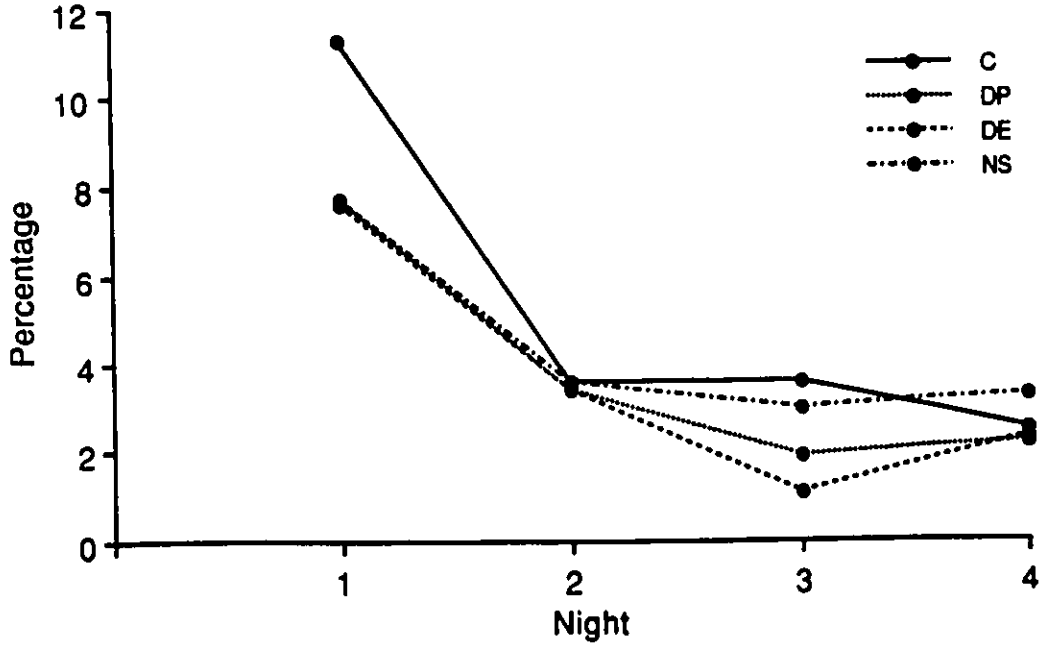


Figure 5

Mean Percentage Values of Wake Time After Sleep Onset Across Nights

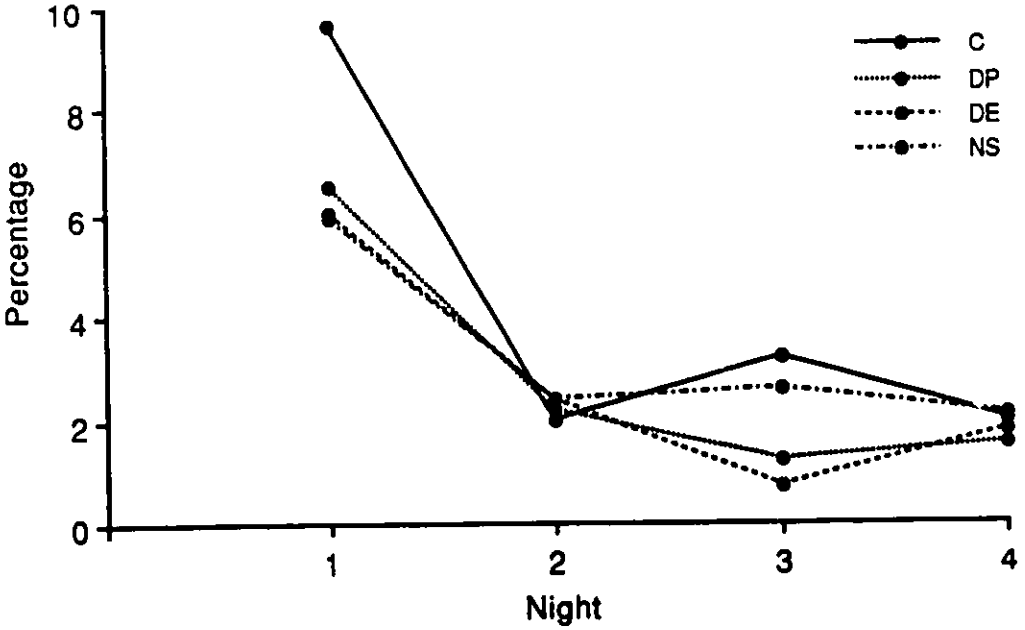


Figure 6

Mean Percentage Values of Stage 1 Across Nights

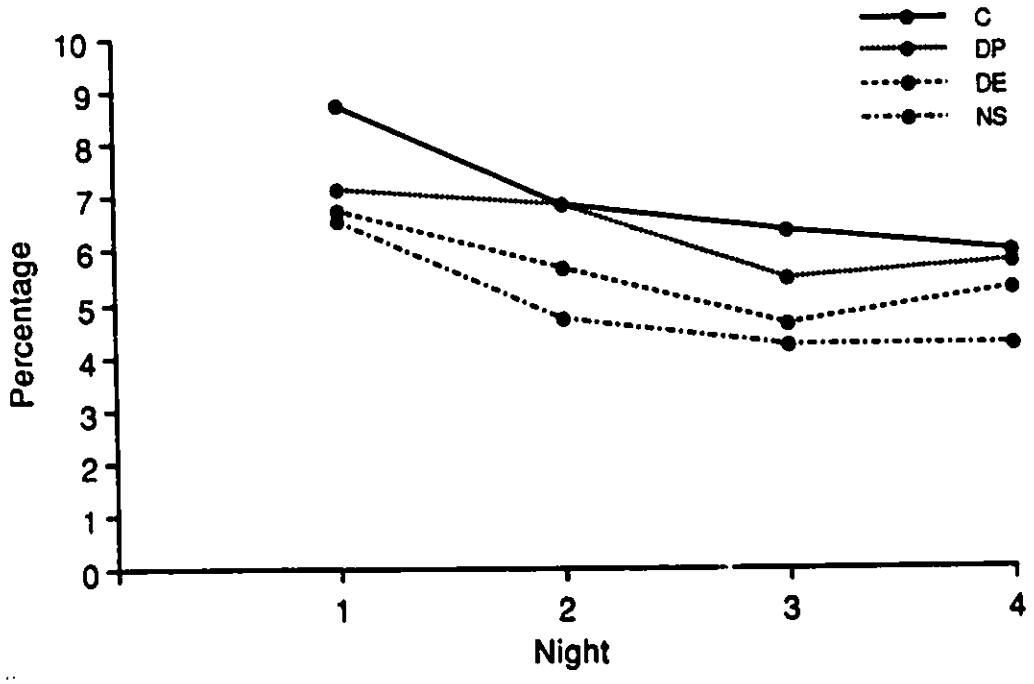
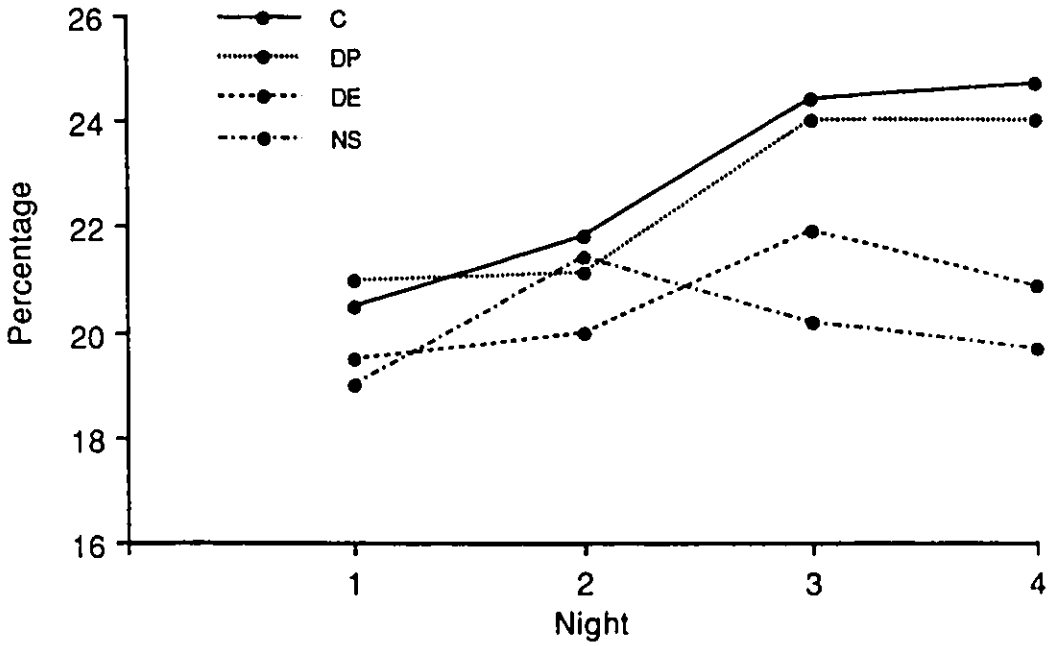


Figure 7
Mean Percentage Values of REM Sleep Across Nights



regarding the various sleep stages and parameters are reported in relative (%) values.

C - RD Pooled

Significant main effects of night, indicating a period of adaptation to the recording environment, were obtained for the following variables: TST [$F(3,111)= 43.47$, $p<.0001$]; SOL [$F(3,111)= 16.59$, $p<.0001$]; SEI [$F(3,111)= 41.03$, $p<.0001$]; TWT [$F(3,111)= 43.56$, $p<.0001$]; WASO [$F(3,111)= 28.05$, $p<.0001$]; stage 1 sleep [$F(3,111)= 12.22$, $p<.0001$]; and REM sleep [$F(3,111)= 11.14$, $p<.0001$]. A significant interaction was observed for TST [$F(3,111)= 3.41$, $p<03$], revealing that the C had a greater increase in TST from night 1 to nights 2-4 than did the RDs. No significant night differences were noted on any of the variables when only nights 3 and 4 were analyzed.

Post-hoc mean comparisons on the variables showing a significant night effect revealed the following relationships for both C and pooled RD subjects ($p<.05$): TST - night 1 < nights 2,3,4; SOL - night 1 > nights 3,4; SEI - night 1 < nights 2,3,4; TWT (%) - night 1 > nights 2,3,4; WASO - night 1 > nights 2,3,4; S1 - night 1 > nights 3,4. REM revealed a different

relationship within each group: C - night 1 < nights 3,4 and night 2 < night 4; RD - night 1 < night 3.

C - RD Subtyped

Similar significant main effects of night were obtained for the following variables: TST [$F(3,105)= 31.03$, $p<.0001$]; SOL [$F(3,105)= 12.66$, $p<.0001$]; SEI [$F(3,105)= 29.86$, $p<.0001$]; TWT [$F(3,105)= 32.19$, $p<.0001$]; WASO [$F(3,105)= p<.0001$]; S1 [$F(3,105)= 10.47$, $p<.0001$]; and REM [$F(3,105)= p<.0002$]. Significant night differences were not noted on any of these variables when only nights 3 and 4 were compared.

Post-hoc mean comparisons on the variables showing a significant night effect revealed that this effect was attenuated for some variables in RD (subtyped) relative to controls. The following relationships were noted in the controls ($p<.05$): TST - night 1 < nights 2-3-4; LSO: night 1 > nights 3-4; SEI: night 1 < nights 2-3-4; TWT - night 1 > nights 2-3-4; WASO - night 1 > nights 2-3-4; S1 - night 1 > nights 3-4; REM - night 1 < nights 3-4, night 2 < night 4. The following relationships were noted for both the dysphonetic and dyseidetic RD subtypes ($p<.05$): TST - night 1 < night 3,4; SEI - night 1 < nights 3-4; TWT - night 1 > 3-4.

No significant differences were observed for SOL; WASO; S1; and REM. Post-hoc tests revealed no significant differences on any of the variables showing a main night effect for the nonspecific RD children.

Since adaptation effects were observed, and the values obtained on some sleep stages and parameters on night 2 were still not characteristic of fully adapted sleep, only nights 3 and 4 (collapsed) were examined further in subsequent group comparisons.

Hypothesis 1

One-way analyses of variance were undertaken to provide information regarding baseline sleep architecture in C and RD children. Summary data for all nights are presented in Tables 3 and 4 [C and RD (pooled)] and Tables 5 and 6 [C and RD (subtyped)]. Again, results pertaining to sleep stages and parameters are reported in percentage values since no group differences were documented for TST. Mean percentage values of sleep measures for nights 3-4 (collapsed) are presented in Tables 7 [C and RD (pooled)] and 8 [C and RD (subtyped)]. Results from

Table 3

Means (SD) of Sleep Measures in Minutes (C and RD Pooled)

Variable	Group	Night 1	Night 2	Night 3	Night 4
TBT	C	541.4 (2.6)	542.5 (3.1)	542.6 (1.1)	542.7 (1.0)
	RD	543.7 (1.2)	543.9 (4.3)	543.9 (5.6)	542.9 (1.8)
TST	C	475.7 (40.5)	518.4 (13.3)	518.1 (22.3)	522.9 (15.0)
	RD	498.0 (19.9)	520.2 (4.3)	526.9 (11.5)	524.1 (11.1)
SOL	C	18.2 (24.6)	10.0 (6.5)	4.7 (5.2)	3.5 (3.4)
	RD	13.4 (8.4)	7.0 (5.4)	3.3 (2.7)	4.8 (4.4)
TWT	C	61.2 (41.0)	19.5 (13.7)	19.7 (22.2)	13.6 (15.0)
	RD	41.8 (20.1)	18.9 (14.0)	10.8 (10.6)	14.1 (10.3)
WASO	C	43.2 (35.0)	10.1 (11.6)	15.3 (22.1)	10.1 (13.4)
	RD	30.0 (20.2)	12.2 (12.8)	7.7 (9.5)	9.3 (9.8)
S1	C	40.7 (14.6)	35.3 (13.0)	32.7 (10.4)	31.1 (10.7)
	RD	33.4 (17.4)	29.5 (9.1)	24.8 (12.1)	26.3 (10.9)
S2	C	236.2 (22.4)	257.9 (29.9)	251.5 (24.4)	248.5 (27.5)
	RD	230.1 (32.4)	245.1 (37.5)	246.1 (36.6)	245.6 (38.5)
S3	C	33.9 (8.4)	37.1 (13.7)	36.3 (14.1)	35.1 (12.7)
	RD	42.5 (19.0)	40.4 (18.0)	43.8 (18.3)	43.9 (18.3)
S4	C	66.9 (22.2)	75.0 (18.2)	71.1 (23.2)	78.9 (14.4)
	RD	93.3 (32.2)	97.1 (30.5)	96.2 (29.3)	95.6 (26.9)
REM	C	98.1 (22.2)	113.2 (16.6)	126.6 (21.6)	129.3 (29.8)
	RD	98.8 (12.8)	108.5 (15.6)	116.0 (17.8)	113.2 (19.5)
NREM	C	377.6 (29.9)	406.3 (21.1)	391.5 (26.7)	393.6 (24.9)
	RD	399.2 (17.5)	411.6 (18.1)	410.9 (17.8)	411.3 (20.0)
SWS	C	100.8 (23.9)	112.1 (24.4)	107.3 (20.3)	114.0 (20.3)
	RD	135.8 (42.8)	137.4 (39.4)	140.0 (39.2)	139.4 (40.6)
MT	C	4.5 (2.6)	4.3 (1.8)	4.7 (2.2)	5.6 (2.9)
	RD	3.9 (2.3)	4.8 (3.3)	4.6 (3.7)	4.2 (2.2)

note: S1 = stage 1; S2 = stage 2; S3 = stage 3; S4 = stage 4; MT = movement time.

Table 4

Means (SD) of Sleep Measures in Percentage (C and RD Pooled)

Variable	Group	Night 1	Night 2	Night 3	Night 4
TWT	C	11.3 (7.6)	3.6 (2.5)	3.6 (4.1)	2.5 (2.8)
	RD	7.7 (3.7)	3.5 (2.6)	2.0 (1.9)	2.6 (1.9)
WASO	C	9.6 (8.5)	6.8 (2.6)	3.2 (4.9)	2.0 (1.9)
	RD	6.1 (4.4)	5.7 (1.8)	1.5 (1.9)	5.9 (2.1)
SEI	C	87.9 (7.5)	95.6 (2.5)	95.5 (4.2)	96.3 (2.7)
	RD	91.6 (3.6)	95.7 (2.7)	96.9 (2.3)	96.6 (2.0)
S1	C	8.7 (3.5)	6.8 (2.6)	6.3 (2.0)	5.9 (2.1)
	RD	6.8 (3.6)	5.7 (1.8)	4.7 (2.4)	5.0 (2.1)
S2	C	49.8 (4.4)	49.6 (5.0)	48.6 (4.7)	47.6 (5.5)
	RD	44.3 (10.5)	47.0 (6.8)	46.7 (6.8)	46.8 (7.1)
S3	C	7.2 (2.1)	7.1 (2.6)	7.1 (2.9)	6.7 (2.4)
	RD	8.6 (3.8)	7.8 (3.6)	8.3 (3.5)	8.3 (3.5)
S4	C	13.9 (3.9)	14.5 (3.6)	13.6 (4.2)	15.1 (2.7)
	RD	18.6 (6.2)	18.6 (5.9)	18.3 (5.5)	18.2 (5.1)
REM	C	20.5 (3.8)	21.2 (3.2)	24.4 (4.0)	24.7 (5.3)
	RD	19.8 (2.3)	20.8 (2.9)	22.0 (3.2)	21.6 (3.6)
NREM	C	79.5 (3.8)	76.8 (6.8)	75.6 (4.0)	75.3 (5.3)
	RD	80.2 (2.3)	79.0 (3.1)	78.0 (3.2)	78.4 (3.6)
SWS	C	21.1 (4.9)	21.7 (4.7)	20.7 (3.4)	21.8 (3.8)
	RD	27.2 (8.3)	26.4 (7.7)	26.6 (7.3)	26.6 (7.8)
MT	C	0.8 (0.5)	0.8 (0.3)	0.9 (0.4)	1.0 (0.5)
	RD	0.7 (0.4)	0.9 (0.6)	0.9 (0.7)	1.2 (1.9)

Table 5

Means (SD) of Sleep Measures in Minutes (C and RD Subtyped)

Variable	Group	Night 1	Night 2	Night 3	Night 4
TBT	C	541.4 (2.6)	542.5 (3.1)	542.6 (1.1)	542.7 (1.0)
	DP	543.3 (1.3)	542.8 (1.3)	545.9 (9.6)	542.6 (1.7)
	DE	544.3 (1.3)	545.1 (5.0)	542.1 (3.8)	543.6 (1.8)
	NS	543.6 (1.0)	543.9 (5.8)	543.6 (1.2)	542.3 (1.9)
TST	C	475.7 (40.5)	518.4 (13.3)	518.1 (22.3)	522.9 (15.0)
	DP	497.1 (27.3)	518.8 (13.2)	527.8 (9.1)	525.6 (9.8)
	DE	498.8 (18.3)	521.6 (12.9)	531.3 (7.9)	527.0 (11.5)
	NS	498.3 (14.9)	520.2 (16.5)	521.6 (15.2)	519.7 (11.9)
SOL	C	18.2 (24.6)	10.0 (6.5)	4.7 (5.2)	3.5 (3.4)
	DP	11.7 (8.3)	7.3 (5.8)	3.9 (3.0)	4.0 (2.7)
	DE	11.9 (4.0)	6.8 (3.9)	2.9 (2.4)	3.2 (2.9)
	NS	16.5 (11.4)	6.9 (6.7)	3.1 (2.9)	7.1 (6.3)
TWT	C	61.2 (41.0)	19.5 (13.7)	19.7 (22.2)	13.6 (15.0)
	DP	42.0 (27.8)	18.2 (13.5)	10.4 (9.9)	12.2 (8.9)
	DE	41.4 (18.4)	18.6 (13.8)	6.0 (7.2)	12.4 (10.4)
	NS	42.0 (14.9)	19.7 (16.5)	16.1 (12.6)	17.7 (11.8)
WASO	C	43.2 (35.0)	10.1 (11.6)	15.6 (22.1)	10.1 (13.4)
	DP	30.9 (27.5)	11.5 (11.1)	6.5 (8.3)	8.3 (8.3)
	DE	29.8 (17.9)	12.4 (13.5)	3.4 (5.3)	9.2 (10.3)
	NS	29.3 (16.1)	12.8 (15.3)	13.3 (11.9)	10.4 (11.8)
S1	C	40.7 (14.6)	35.3 (13.0)	32.7 (10.4)	31.1 (10.7)
	DP	34.8 (17.5)	35.3 (8.7)	28.3 (16.2)	29.8 (9.7)
	DE	33.2 (22.9)	28.9 (9.8)	24.2 (12.6)	27.1 (15.3)
	NS	32.4 (12.8)	24.3 (5.4)	21.8 (5.8)	22.0 (5.3)
S2	C	236.2 (22.4)	257.9 (29.9)	251.5 (24.4)	248.5 (27.5)
	DP	222.7 (32.3)	238.8 (33.7)	240.9 (30.0)	234.1 (28.4)
	DE	233.6 (39.5)	236.9 (47.6)	243.2 (44.8)	244.7 (48.1)
	NS	234.1 (27.3)	259.6 (29.3)	254.3 (36.9)	257.9 (37.7)

(table continues)

Table 5

Means (SD) of Sleep Measures in Minutes (C and RD Subtyped)

Variable	Group	Night 1	2	3	4
S3	C	33.9 (8.4)	37.1 (13.7)	36.3 (13.1)	35.1 (12.7)
	DP	40.3 (13.8)	44.2 (14.2)	45.3 (13.7)	41.6 (12.3)
	DE	38.7 (25.4)	41.3 (25.4)	41.2 (25.4)	44.8 (26.8)
	NS	48.9 (16.6)	35.6 (13.1)	44.8 (16.2)	45.3 (14.9)
S4	C	66.9 (22.2)	75.0 (18.2)	71.1 (23.2)	78.9 (14.4)
	DP	95.1 (36.4)	91.4 (36.4)	86.6 (28.0)	93.9 (25.6)
	DE	96.3 (39.4)	110.8 (32.5)	106.6 (30.6)	99.9 (34.9)
	NS	88.4 (22.2)	89.1 (18.7)	95.4 (29.7)	92.8 (21.7)
REM	C	98.1 (22.2)	113.2 (16.6)	126.6 (21.6)	129.3 (29.8)
	DP	104.3 (13.8)	109.2 (12.8)	126.7 (13.5)	126.2 (18.6)
	DE	97.4 (13.5)	104.1 (21.6)	116.1 (18.6)	110.5 (22.3)
	NS	94.6 (10.3)	112.1 (11.5)	105.3 (15.6)	102.9 (9.1)
NREM	C	377.6 (29.9)	406.3 (21.1)	391.5 (26.7)	393.6 (24.9)
	DP	392.8 (20.1)	409.6 (17.9)	401.1 (16.4)	399.4 (20.4)
	DE	401.0 (16.0)	417.3 (20.1)	415.2 (17.6)	416.5 (20.9)
	NS	403.7 (16.5)	408.1 (16.5)	416.3 (17.4)	417.9 (14.7)
SWS	C	100.8 (23.9)	112.1 (24.4)	107.3 (20.3)	114.0 (20.3)
	DP	135.4 (45.3)	135.6 (38.1)	131.9 (35.9)	135.5 (33.1)
	DE	134.6 (53.1)	151.8 (52.4)	147.8 (51.8)	144.8 (56.8)
	NS	137.3 (34.0)	124.7 (21.8)	140.3 (30.5)	138.1 (32.3)
MT	C	4.5 (2.6)	4.3 (1.8)	4.7 (2.2)	5.6 (2.9)
	DP	4.3 (2.0)	5.5 (2.9)	3.6 (2.4)	4.8 (2.0)
	DE	3.9 (2.8)	4.6 (4.4)	4.8 (4.7)	4.1 (2.7)
	NS	3.4 (2.3)	4.1 (2.1)	5.6 (3.7)	3.7 (2.1)

Table 6

Means (SD) of Sleep Measures in Percentage (C and RD Subtyped)

Variable	Group	Night 1	Night 2	Night 3	Night 4
TWT	C	11.3 (7.6)	3.6 (2.5)	3.6 (4.1)	2.5 (2.8)
	DP	7.7 (5.1)	3.4 (2.5)	1.9 (1.8)	2.2 (1.7)
	DE	7.6 (3.4)	3.4 (2.5)	1.1 (1.3)	2.3 (1.9)
	NS	7.7 (2.7)	3.6 (3.0)	3.0 (2.3)	3.3 (2.2)
WASO	C	9.6 (8.5)	6.8 (2.6)	3.2 (4.9)	2.0 (1.9)
	DP	6.5 (6.2)	2.2 (2.2)	1.2 (1.5)	1.5 (1.5)
	DE	6.0 (3.7)	2.4 (2.7)	0.7 (1.0)	1.8 (2.0)
	NS	5.9 (3.2)	2.4 (2.8)	2.6 (2.4)	2.1 (2.3)
SEI	C	87.9 (7.5)	95.6 (2.5)	95.5 (4.2)	96.3 (2.7)
	DP	91.5 (5.0)	95.6 (2.4)	96.7 (2.3)	96.8 (1.9)
	DE	91.6 (3.3)	95.7 (2.9)	98.0 (1.2)	96.9 (1.9)
	NS	91.8 (2.7)	95.7 (3.2)	95.9 (2.7)	96.1 (2.3)
S1	C	8.7 (3.5)	6.8 (2.6)	6.3 (2.0)	5.9 (2.1)
	DP	7.1 (3.7)	6.8 (1.8)	5.4 (3.2)	5.7 (1.9)
	DE	6.7 (4.6)	5.6 (1.9)	4.7 (2.4)	5.2 (3.0)
	NS	6.5 (2.7)	4.7 (1.2)	4.2 (1.2)	4.2 (1.0)
S2	C	49.8 (4.4)	49.6 (5.0)	48.6 (4.7)	47.6 (5.5)
	DP	45.0 (7.1)	46.1 (6.8)	45.6 (5.4)	44.5 (4.9)
	DE	41.1 (16.1)	45.3 (8.4)	45.8 (8.4)	46.4 (9.1)
	NS	47.0 (5.2)	49.6 (5.0)	48.7 (6.8)	49.4 (6.5)
S3	C	7.2 (2.1)	7.1 (2.6)	7.1 (2.9)	6.7 (2.4)
	DP	8.1 (2.5)	8.6 (2.8)	8.6 (2.6)	7.9 (2.4)
	DE	7.8 (5.2)	7.9 (5.1)	7.8 (4.8)	8.5 (5.1)
	NS	9.8 (3.4)	6.8 (2.6)	8.6 (3.2)	8.7 (2.9)
S4	C	13.9 (3.9)	14.5 (3.6)	13.6 (4.2)	15.1 (2.7)
	DP	19.0 (6.6)	17.5 (6.8)	16.4 (5.3)	17.9 (4.9)
	DE	19.3 (8.0)	21.3 (6.5)	20.1 (5.7)	18.9 (6.5)
	NS	17.7 (4.1)	17.0 (3.4)	18.3 (5.6)	17.9 (4.4)

(table continues)

Table 6

Means (SD) of Sleep Measures in Percentage (C and RD Subtyped)

Variable	Group	Night 1	Night 2	Night 3	Night 4
REM	C	20.5 (3.8)	21.2 (3.2)	24.4 (4.0)	24.7 (5.3)
	DP	21.0 (2.1)	21.1 (2.5)	24.0 (2.6)	24.0 (3.5)
	DE	19.5 (2.4)	20.0 (4.0)	21.9 (3.4)	20.9 (4.1)
	NS	19.0 (2.0)	21.4 (2.1)	20.1 (2.8)	19.7 (1.8)
NREM	C	79.5 (3.8)	76.8 (6.8)	75.6 (4.0)	75.3 (5.3)
	DP	79.0 (2.1)	79.0 (2.5)	76.0 (2.6)	76.0 (4.0)
	DE	80.5 (2.4)	80.1 (4.0)	78.1 (3.4)	79.1 (4.1)
	NS	81.0 (2.0)	78.0 (2.5)	79.8 (2.8)	80.2 (1.7)
SWS	C	21.1 (4.9)	21.7 (4.7)	20.7 (3.4)	21.8 (3.8)
	DP	27.1 (8.1)	26.0 (6.9)	25.0 (6.7)	25.8 (6.4)
	DE	27.0 (10.9)	29.2 (10.6)	27.8 (9.7)	27.5 (10.7)
	NS	27.5 (6.6)	23.9 (4.1)	26.9 (5.6)	26.6 (6.5)
MT	C	0.8 (0.5)	0.8 (0.3)	0.9 (0.4)	1.0 (0.5)
	DP	0.8 (0.3)	1.0 (0.5)	0.7 (0.4)	2.0 (3.3)
	DE	0.8 (0.5)	0.9 (0.8)	0.9 (0.9)	0.8 (0.5)
	NS	0.7 (0.4)	0.8 (0.4)	1.0 (0.7)	0.7 (0.4)

Table 7

Means (SD) of Sleep Measures for Nights 3-4 in Percentage (C and RD Pooled)

Variable	Group	
	C	RD
TWT	3.1 (2.6)	2.3 (1.5)
WASO	2.6 (3.0)	1.6 (1.4)
SEI	95.9 (2.8)	95.7 (1.7)
S1*	6.1 (1.7)	4.9 (2.0)
S2	48.1 (4.3)	46.8 (6.5)
S3	6.9 (2.3)	8.3 (3.3)
S4****	14.3 (2.7)	18.2 (5.0)
REM**	24.5 (4.0)	21.8 (3.0)
NREM**	75.5 (3.9)	78.2 (3.0)
SWS***	21.2 (3.2)	26.6 (7.3)
MT	1.0 (0.4)	1.0 (1.1)

*$p<.05$. **$p<.02$. ***$p<.01$. ****$p<.009$.

Table 8

Means (SD) of Sleep Measures for Nights 3-4 in Percentage (C and RD Subtyped)

Variable	Group			
	C	DP	DE	NS
TWT	3.1 (2.6)	2.1 (1.2)	1.7 (1.5)	3.1 (1.6)
WASO	2.6 (3.0)	1.4 (1.1)	1.2 (1.4)	2.3 (1.7)
SEI	95.9 (2.8)	96.8 (1.3)	97.5 (4.5)	96.0 (2.0)
S1	6.1 (1.7)	5.5 (2.0)	4.9 (2.6)	4.2 (0.8)
S2	48.1 (4.3)	45.1 (4.7)	46.1 (8.3)	49.1 (6.1)
S3	6.9 (2.3)	8.2 (2.4)	8.1 (4.9)	8.7 (2.5)
S4*	14.3 (2.7)	17.2 (4.7)	19.5 (5.9)	18.1 (4.7)
REM**	24.5 (4.0)+	24.0 (2.8)	21.4 (3.1)	20.0 (1.7)+
NREM**	75.5 (3.9)+	76.0 (2.8)	78.6 (3.1)	80.0 (1.7)+
SWS	21.2 (3.2)	25.4 (6.3)	27.6 (10.0)	26.7 (5.7)
MT	1.0 (0.4)	1.4 (1.8)	0.8 (0.7)	0.9 (0.5)

*$p<.05$. **$p<.009$. +$p<.05$.

the C - RD (pooled) comparisons are presented below and are followed by those from the comparisons of Cs to subtyped RDs.

C - RD Pooled

Sleep Stages and Parameters

The first hypothesis, which postulated an absence of group differences in basic sleep patterns and parameters was not confirmed. A significant difference was noted on the following variables: Stage 1 - with Cs exhibiting increased amounts relative to the RDs [$F(1,37)= 4.24$, $p<.05$]; Stage 4 - with the RD children displaying greater amounts than the Cs [$F(1,37)= 7.69$, $p<.009$]; Slow Wave Sleep (SWS): with the RDs having greater amounts than Cs [$F(1,31)= 7.12$,$p<.01$]; REM sleep - with the Cs having increased amounts relative to the RDs [$F(1,37)= 6.10$, $p<.02$]; and, NREM sleep - with RD children exhibiting greater amounts relative to the Cs [$F(1,37)= 6.09$, $p<.02$]. No significant differences were noted on the following sleep parameters variables: LSO; TST; WASO; and SEI. In general, therefore, C subjects showed more REM than the RDs while the RDs showed more stage 4 than the Cs. Mean percentage values of sleep stages for both groups

on nights 3-4 collapsed are illustrated in Figure 8.

Since these differences were noted, additional analyses were undertaken to investigate the distribution of stages 4 and REM across the night in both groups. Baseline nights were divided into thirds and the relative amounts of stages 4 and REM within each third of the night for both groups were calculated. As can be seen in Figure 9, both groups displayed decreasing amounts of stage 4 across each third of the night. No marked between-group differences were found for amounts of stage 4 sleep within each third of the night. However, RD children did exhibit a tendency towards having more stage 4 sleep in the last third of the night relative to C subjects [$F(1,37)= 3.43$, $p<.07$]. As Figure 10 reveals, both groups displayed increasing amounts of REM sleep across thirds of the night. No significant group differences were found for amounts of REM sleep within each third of night.

Cycle Analyses

REM and NREM Cycle Lengths

Since a group difference in the duration of the initial NREM cycle was postulated, analyses were

Figure 8
Mean (SEM) Percentage Values of Sleep Stages

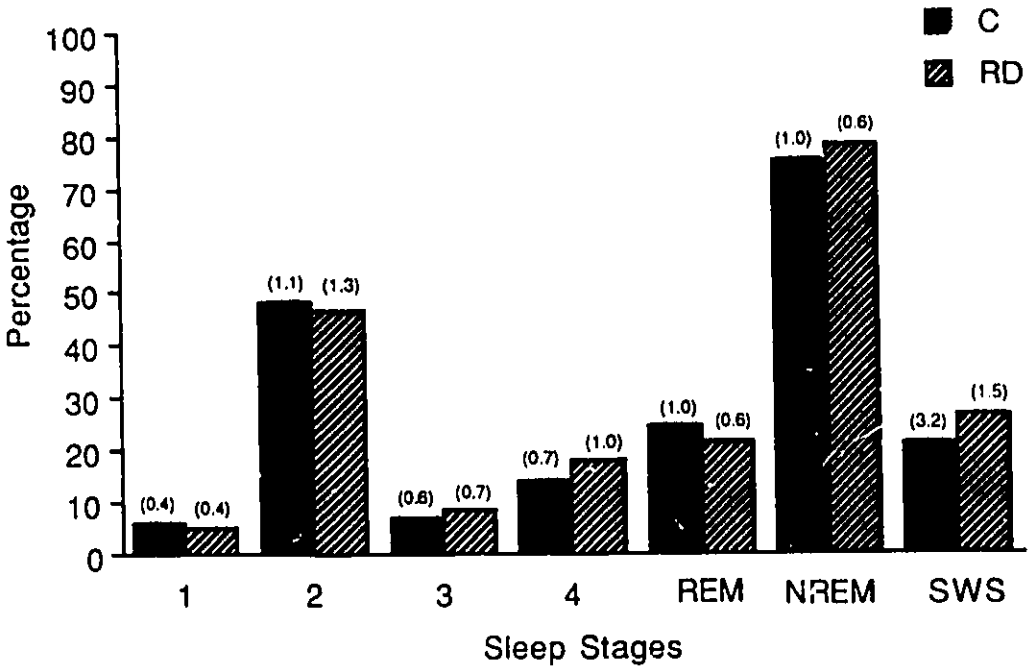


Figure 9
Distribution of Stage 4 Sleep Across Thirds of Night

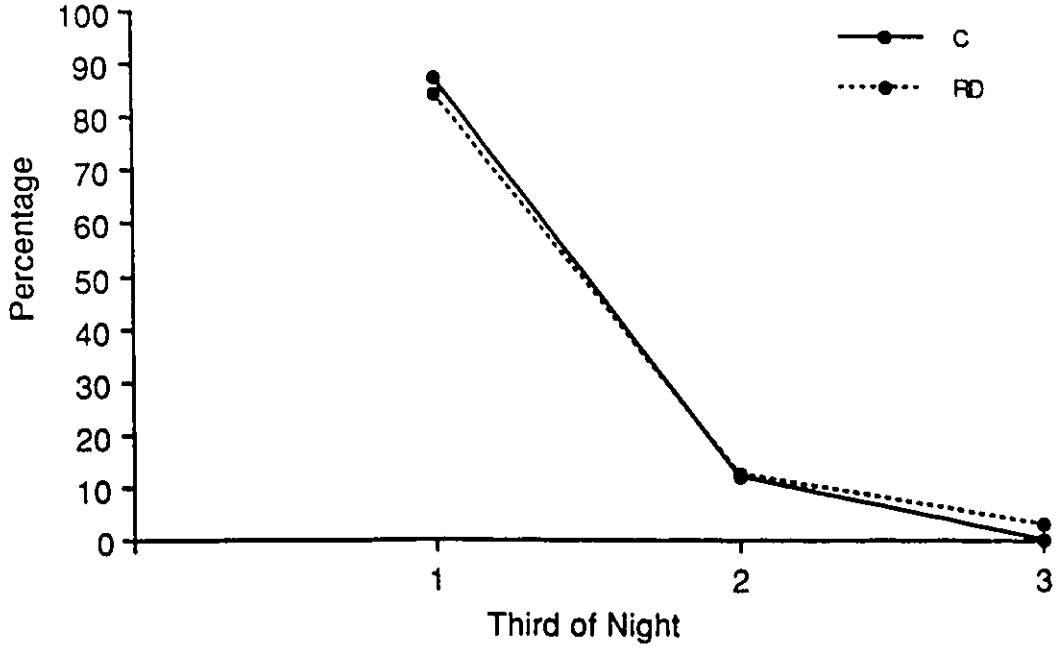
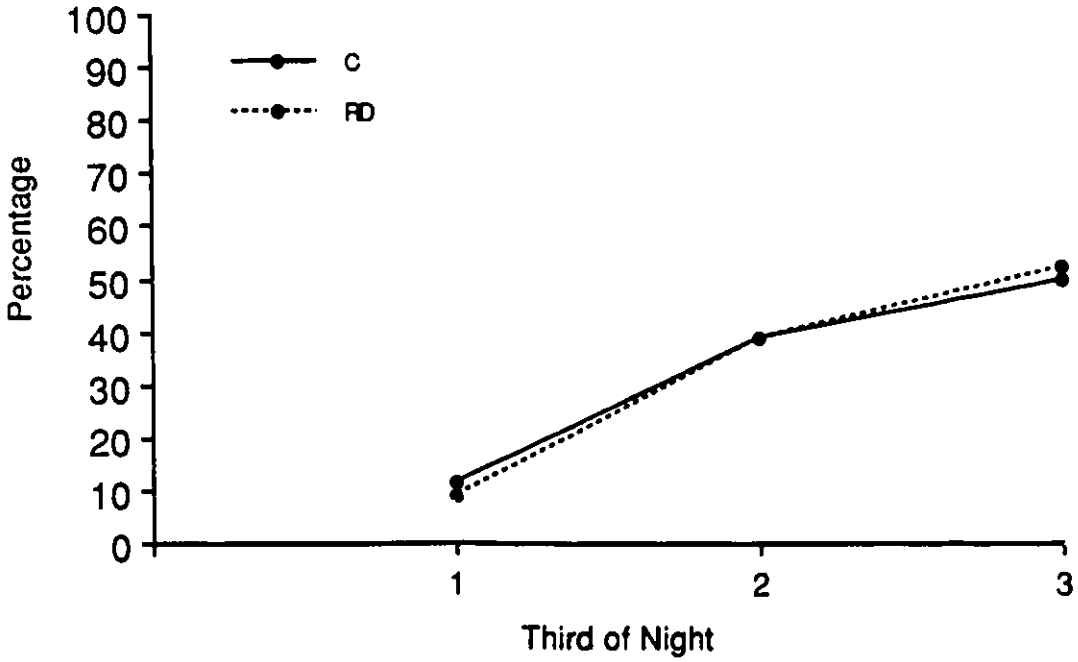


Figure 10
Distribution of REM Sleep Across Thirds of Night



undertaken to provide information regarding REM and NREM cycle lengths in C and RD children. All subjects completed a minimum of three REM and NREM cycles. Most had completed at least four REM cycles (C: 15/15 subjects; RD pooled: 15/24 subjects) and four NREM cycles (C: 15/15; RD: 18/24). Therefore, analyses pertaining to individual REM and NREM cycles were limited to the first four cycles. These analyses were conducted with WASO included (real-time) and deleted. Since the results obtained were the same in both conditions, only those relating to real-time REM and NREM cycle analyses are presented. Mean durations of REM and NREM cycle lengths in both groups (nights 3-4 combined) are presented in Figures 11 and 12, respectively.

No significant differences were noted for any REM cycle lengths. However, as postulated, a significant difference was observed for the duration of the first NREM cycle with RD subjects displaying a longer cycle than Cs [$F(1,37)= 7.60$, $p<.009$]. No significant differences were observed for the duration of NREM cycles 2, 3 and 4.

Figure 11
Mean (SEM) Durations of REM Cycles

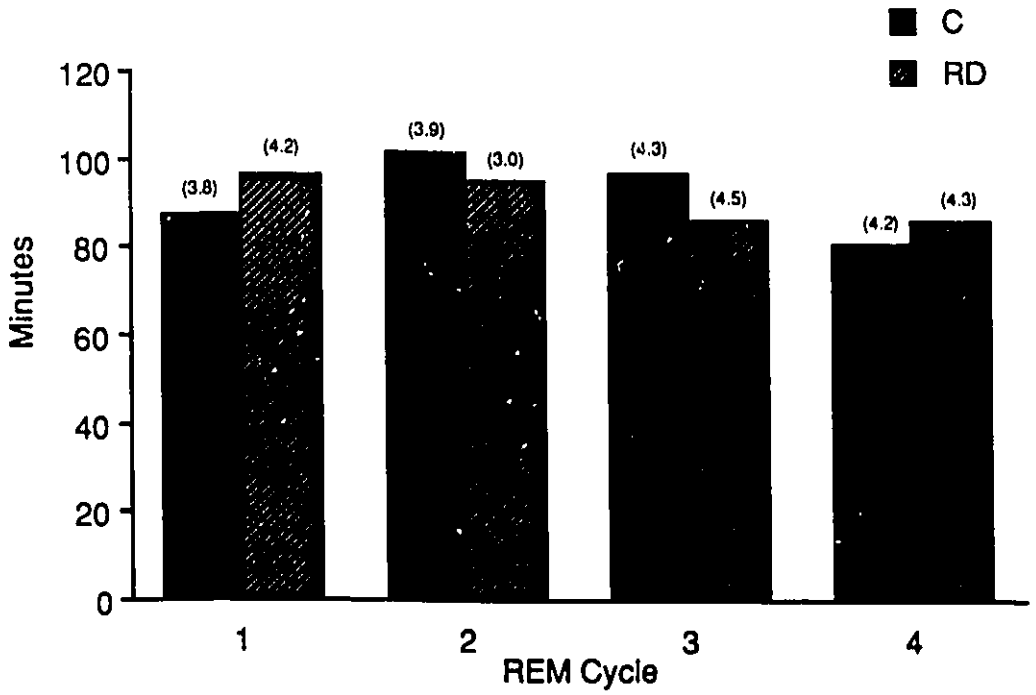
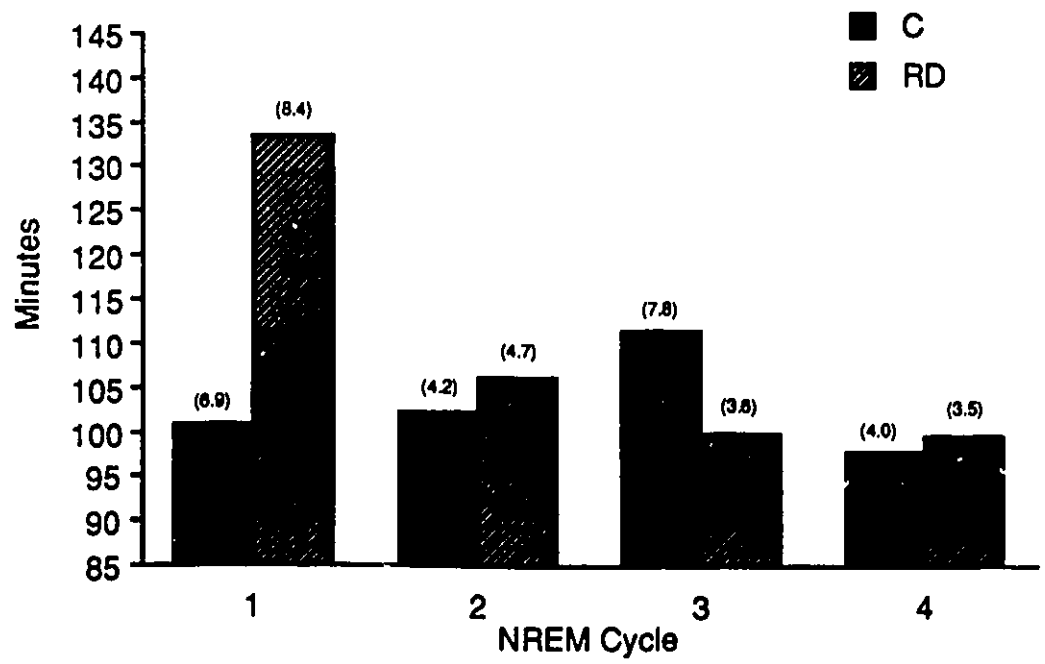


Figure 12
Mean (SEM) Durations of NREM Cycles



Stage composition of the first NREM cycle and remainder of the night

Since a significant difference was observed for the duration of the first NREM cycle, analyses examining the composition of this cycle and the remainder of the night were undertaken, in which the absolute and relative amounts of sleep stages and parameters which make up these time periods in both groups were computed. These data are presented in Tables 9-12.

Analyses examining the stage composition of the first NREM cycle indicated that RD children had greater absolute amounts of stages 2 and 4 within this time period relative to the Cs [$F(1,37)= 5.03$, $p<.03$ and $F(1,37)= 8.99$, $p<.005$, respectively]. No differences were noted for either absolute amounts or relative amounts of other sleep stages and parameter within the first NREM cycle.

Analyses examining the stage composition of the remaining time in bed (rest of the night) revealed a significant difference in absolute amounts of REM sleep, with the Cs exhibiting more of this stage within this time period than the RDs [$F(1,37)= 8.54$, $p<.006$].

Table 9

First NREM Cycle Composition: Means (SD) of Sleep Measures in Minutes (C and RD Pooled)

Sleep Measure	Group	
	C	RD
WASO	0.8 (1.7)	0.5 (1.2)
S1	2.4 (4.6)	2.6 (2.5)
S2*	23.2 (13.6)	36.0 (19.3)
S3	12.1 (7.6)	13.8 (7.5)
S4**	53.6 (12.5)	67.3 (14.7)
REM	8.1 (5.1)	12.6 (10.1)

*$p<.03$. **$p<.005$.

Table 10

First NREM Cycle Composition: Means (SD) of Sleep Measures in Percentage (C and RD Pooled)

Sleep Measure	Group	
	C	RD
WASO	0.6 (1.3)	0.3 (0.6)
S1	1.8 (2.1)	1.3 (0.9)
S2	21.5 (7.5)	24.8 (8.4)
S3	11.8 (4.7)	9.5 (4.0)
S4	56.7 (12.9)	54.5 (13.0)
REM	7.8 (4.4)	7.9 (4.2)

Table 11

Remainder of Night Composition: Means (SD) of Sleep Measures in Minutes (C and RD Pooled)

Sleep Measure	Group	
	C	RD
WASO	12.0 (12.6)	8.0 (7.0)
S1	24.8 (7.9)	20.1 (9.3)
S2	226.1 (21.4)	209.2 (34.5)
S3	23.7 (9.5)	29.8 (16.1)
S4	21.5 (12.1)	27.8 (20.7)
REM*	120.0 (19.0)	101.8 (18.9)

*$p<.006$.

Table 12

Remainder of Night Composition: Means (SD) of Sleep Measures in Percentage (C and RD Pooled)

Sleep Measure	Group	
	C	RD
WASO	2.8 (3.1)	2.0 (1.8)
S1	5.7 (1.7)	5.0 (2.2)
S2	52.7 (4.3)	52.3 (7.8)
S3	5.4 (2.1)	7.3 (3.8)
S4	4.8 (2.6)	6.7 (5.0)
REM	27.5 (3.8)	25.2 (3.8)

No group difference in other sleep stages and parameters were noted. No significant differences were obtained when relative amounts of sleep stage and parameters making up this time period were computed.

C - RD Subtyped

Sleep Stages and Parameters

The comparison of baseline sleep architecture in Cs and the three subtypes of RD children revealed significant differences on the following variables: stage 4 [$F(3,35)= 2.91$, $p<.05$]; REM sleep [$F(3,35)= 4.49$, $p<.009$]; and, NREM sleep [$F(3,35)= 4.47$, $p<.009$]. Post-hoc mean comparisons revealed that the nonspecific group had decreased amounts of REM sleep relative to the Cs ($p<.05$) and increased amounts of NREM sleep relative to the Cs ($p<.05$). No pair-wise differences were observed for stage 4 sleep, suggesting that the effect is not attributable to any one subgroup but to the combined effects of the pooling of RD subjects. No other difference on any other sleep stage or sleep parameter was significant. Mean percentage values of sleep stages for Cs and RD subtypes (for nights 3-4 combined) are presented in Figure 13.

Additional analyses were undertaken to investigate

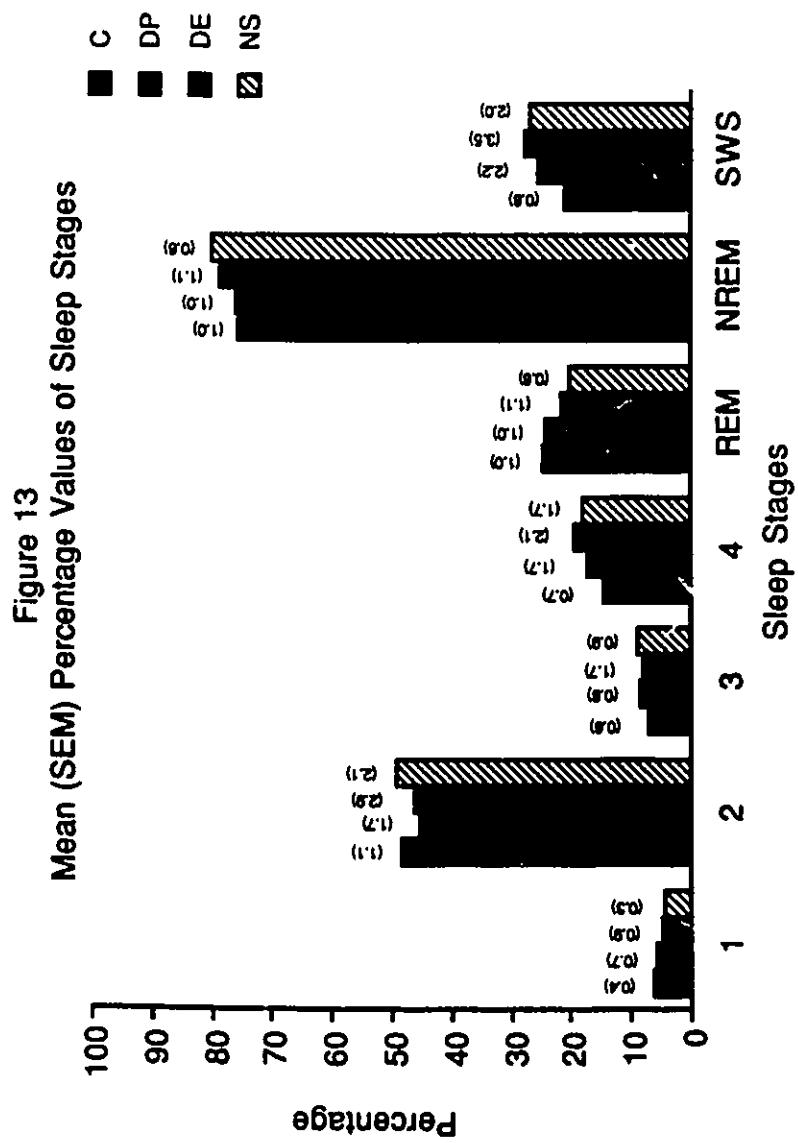


Figure 13
Mean (SEM) Percentage Values of Sleep Stages

the relative distribution of stages 4 and REM across thirds of night. As revealed in Figure 14, all groups displayed decreasing amounts of stage 4 sleep across the night. No significant group differences were found for relative amounts of stage 4 sleep within each third. However, the group difference observed in the last third of night approached significance, suggesting that some RD children (dyseidetics and nonspecifics in particular) tended to exhibit relatively more stage 4 sleep later in the night compared to the Cs and dysphonetics [$F(3,35)= 2.48$, $p<.08$]. The data presented in Figure 15 suggest that all groups had increasing relative amounts of REM sleep across the thirds of night. A significant difference was noted for amounts of REM sleep in the middle third of the night [$F(3,35)= 2.83$, $p<.05$], with the nonspecific group displaying increased amounts relative to the Cs ($p<.05$). No other group differences were observed in the first and last thirds of the night.

Cycle Analyses

REM and NREM cycle lengths

Every subject completed a minimum of three REM and NREM cycles. Most completed at least four REM cycles

Figure 14
Distribution of Stage 4 Sleep Across Thirds of Night

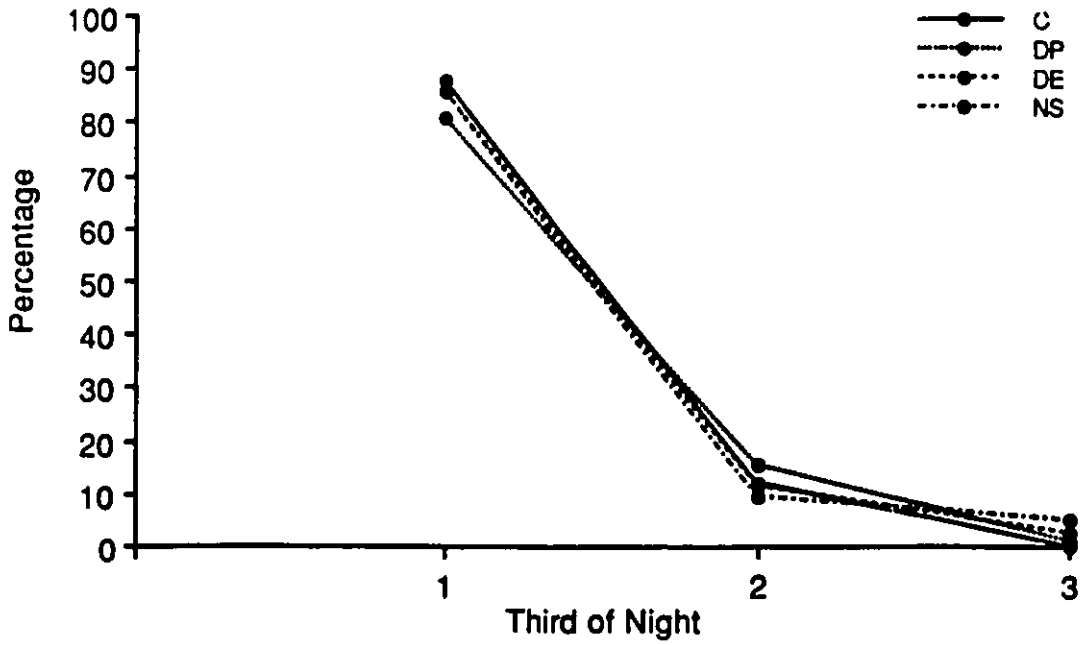
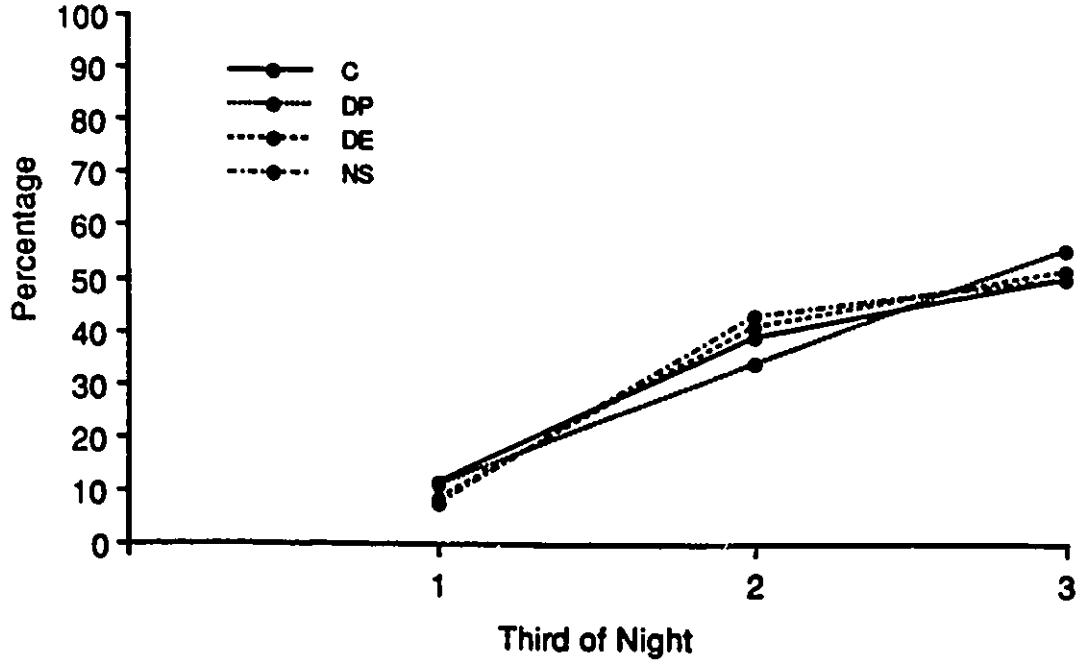


Figure 15
Distribution of REM Sleep Across Thirds of Night



(dysphonetic: 5/8 subjects; dyseidetic: 6/8 subjects; nonspecific: 4/8 subjects) and four NREM cycles (dysphonetic: 5/8; dyseidetic: 7/8; nonspecific: 6/8). Therefore, analyses pertaining to individual REM and NREM cycles were limited to the first four cycles. Again, the inclusion or exclusion of WASO in the cycle length analyses did not change the results obtained. Therefore, only those relating to real-time REM and NREM cycle analyses will be presented. Mean durations of REM and NREM cycle lengths for all groups (nights 3-4 collapsed) are presented in Figures 16 and 17, respectively.

No significant group differences were observed for the duration of any REM cycles. Significant differences were obtained, however, for the duration of the first NREM cycle [$F(3,35)= 5.53$, $p<.003$] with the nonspecifics exhibiting a longer cycle than Cs and dyseidetics ($p<.05$). No other specific pair-wise differences were noted.

Stage composition of the first NREM cycle and remainder of the night

Again, since a significant difference was observed for the duration of the first NREM cycle, analyses

Figure 16
Mean (SEM) Durations of REM Cycles

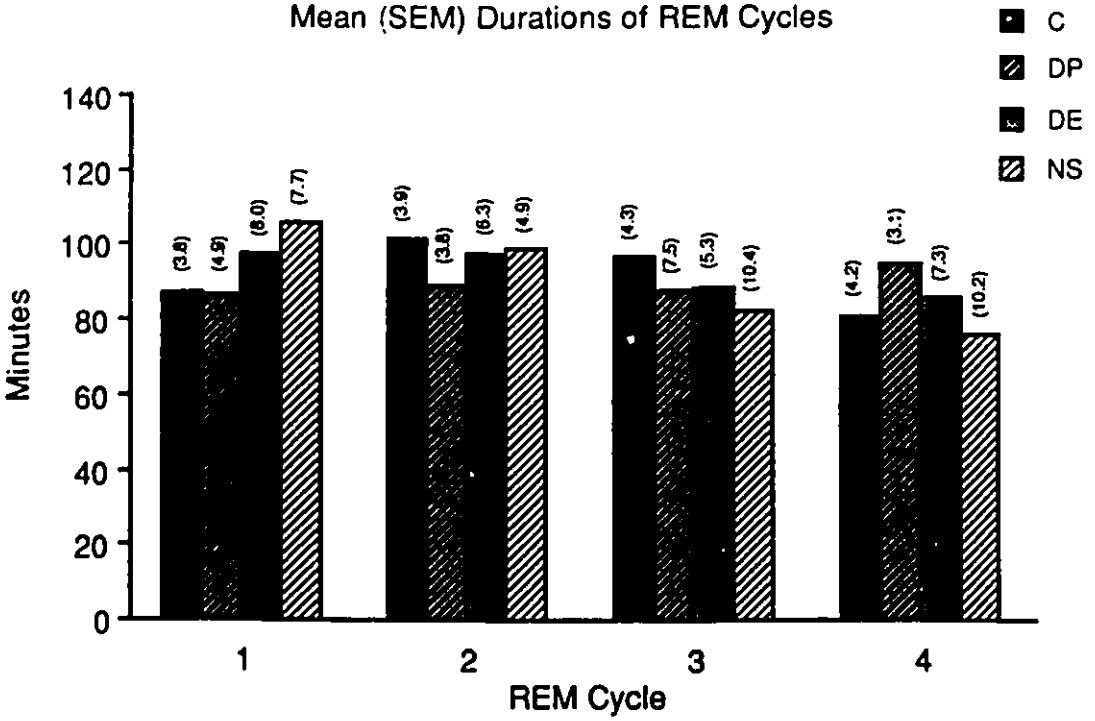
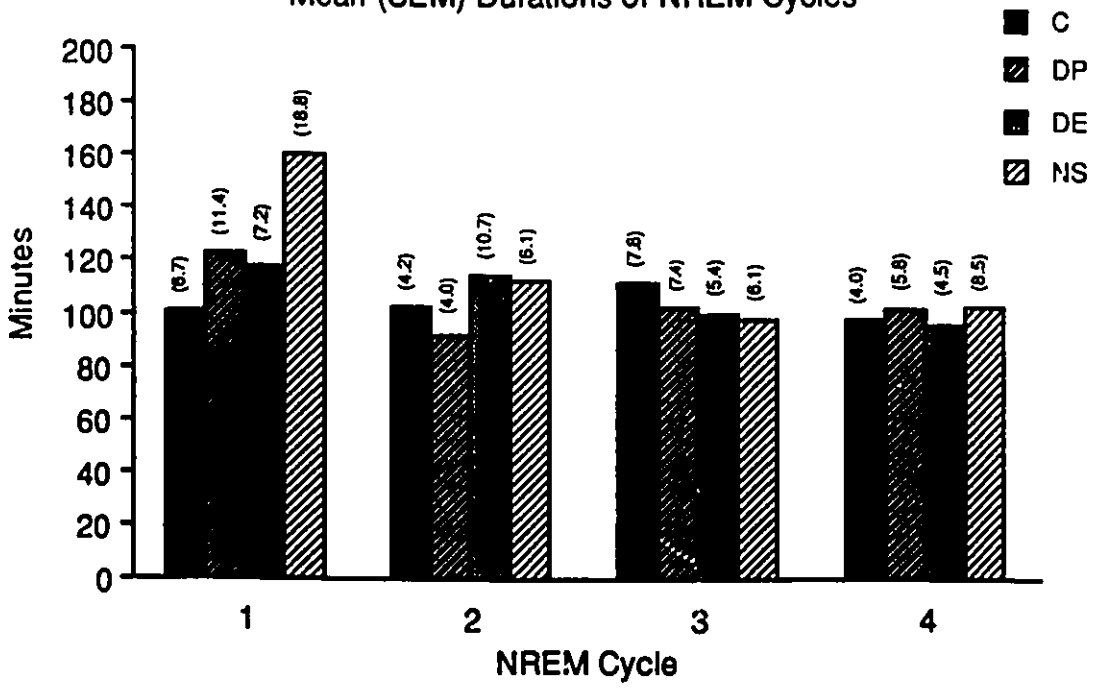


Figure 17
Mean (SEM) Durations of NREM Cycles



examining the composition of this cycle and the remainder of the night were undertaken. The absolute and relative amounts of sleep stages and parameters making up these time periods for each group are presented in Tables 13-16.

Analyses examining the stage composition of the first NREM cycle identified significant differences in absolute amounts of stages 2 and 4 [$F(3,35)= 3.75$, $p<.02$; and $F(3,35)= 4.05$, $p<.01$, respectively]. Post-hoc comparisons revealed that the nonspecifics exhibited more of these sleep stages within this time period relative to Cs ($p<.05$). No other specific pair-wise differences were identified for absolute or relative amounts of sleep stages and parameters within the first NREM cycle.

Analyses examining the stage composition of the remainder of the night revealed a significant difference in absolute and relative amounts of REM sleep [$F(3,35)= 7.76$, $p<.004$; and $F(3,35)= 3.88$, $p<.02$, respectively]. Post-hoc mean comparisons revealed that nonspecifics displayed more of this sleep stage within this time period than did Cs and dysphonetics ($p<.05$). No other significant pair-wise differences were

Table 13

First NREM Cycle Composition: Means (SD) of Sleep Measures in Minutes (C and RD Subtyped)

Sleep Measure	Group			
	C	DP	DE	NS
WASO	0.8 (1.7)	1.0 (0.2)	0.1 (0.3)	1.3 (1.9)
S1	2.4 (4.6)	1.6 (1.6)	1.7 (0.9)	3.0 (2.8)
S2*	23.2 (13.6)+	34.7 (21.5)	26.8 (21.5)	46.5 (21.3)+
S3	12.1 (7.6)	11.8 (4.2)	11.1 (6.7)	18.5 (9.3)
S4**	53.6 (12.5)+	61.0 (18.0)	68.3 (11.1)	72.7 (13.6)+
REM	8.1 (5.1)	11.9 (7.6)	8.2 (4.0)	17.8 (14.5)

*$p<.02$. **$p<.01$. +$p<.05$.

Table 14

First NREM Cycle Composition: Means (SD) of Sleep Measures in Percentage (C and RD Subtyped)

Sleep Measure	Group			
	C	DP	DE	NS
WASO	0.6 (1.3)	0.1 (0.2)	0.1 (0.2)	0.7 (1.0)
S1	1.8 (2.1)	1.1 (0.8)	1.2 (0.6)	1.6 (1.3)
S2	21.5 (7.5)	26.8 (11.6)	21.7 (6.4)	25.9 (6.2)
S3	11.8 (4.7)	9.6 (4.0)	8.7 (4.3)	10.2 (4.3)
S4	56.7 (12.9)	52.8 (15.2)	61.3 (8.0)	49.5 (13.3)
REM	7.8 (4.4)	8.3 (3.5)	6.5 (3.1)	9.0 (5.7)

Table 15

Remainder of Night Composition: Means (SD) of Sleep Measures in Minutes (C and RD Subtyped)

Sleep Measure	Group			
	C	DP	DE	NS
WASO	12.0 (12.6)	7.3 (5.9)	6.2 (7.1)	10.6 (7.8)
S1	24.8 (7.9)	23.4 (10.0)	20.9 (11.6)	16.0 (3.8)
S2	226.1 (21.4)	202.7 (23.1)	215.8 (37.3)	209.1 (43.4)
S3	23.7 (9.5)	32.0 (15.9)	30.7 (22.5)	26.6 (8.9)
S4	21.5 (12.1)	26.8 (10.8)	35.0 (26.0)	21.5 (22.5)
REM*	120.0 (19.0)+	114.5 (15.1)+	105.0 (16.1)	85.7 (13.6)+

*$p<.004$. +$p<.05$.

Table 16

Remainder of Night Composition: Means (SD) of Sleep Measures in Percentage (C and RD Subtyped)

Sleep Measure	Group			
	C	DP	DE	NS
WASO	2.8 (3.1)	1.8 (1.5)	1.5 (1.6)	2.8 (2.1)
S1	5.7 (1.7)	6.0 (2.8)	5.2 (3.1)	4.3 (1.5)
S2	52.7 (4.3)	49.2 (4.8)	51.8 (8.7)	55.9 (8.7)
S3	5.4 (2.1)	7.6 (3.5)	7.3 (5.4)	7.1 (2.6)
S4	4.8 (2.6)	6.4 (2.4)	8.0 (6.0)	5.7 (5.9)
REM*	27.5 (3.8)+	27.8 (3.8)+	25.0 (3.7)	22.9 (2.2)+

*$p<.02$. +$p<.05$.

identified in absolute or relative amounts of any other sleep stages and parameters making up this time period.

In summary, RD children (pooled) showed more stage 4 sleep than Cs, while Cs showed more REM sleep than RDs. Subtype analyses revealed that the stage 4 difference was not attributable to any one subtype, but to the effects of pooling RD children. The REM sleep differences, however, were strongest between the C and nonspecific groups of children. The first hypothesis, suggesting no group differences in sleep stages was, therefore, not confirmed.

Analyses examining the distribution of sleep stages 4 and REM across thirds of night revealed that both C and RD children (pooled and subtyped) displayed a relatively normal distribution of these stages across the night. Nonspecific and dyseidetic subjects, however, did show a nonsignificant tendency towards having relatively more stage 4 sleep later in the night relative to Cs.

Cycle analyses revealed that, with the exception of the initial NREM cycle, there were no group differences in REM and subsequent NREM cycle lengths.

As suggested in the first hypothesis, RD children exhibited an extended first NREM cycle relative to the Cs. However, the nonspecific subtype exhibited the longest first NREM cycle, which did not confirm the hypothesized subtype difference.

Analyses investigating the stage composition of the initial NREM cycle revealed greater absolute amounts of stages 2 and 4 within this time period in all RD children relative to Cs. Again, subtype analyses revealed that these differences were largest between the nonspecific subtype and the Cs. When the stage composition of the remainder of the night was computed, Cs showed more minutes of REM sleep within this time period than did the RDs (pooled). Subtype analyses suggested that both Cs and dysphonetics had more REM sleep (minutes and percentage) during the rest of the night relative to the nonspecifics.

Therefore, these results suggest the presence of some group differences between C and RD children (pooled) in baseline sleep architecture (stages 4 and REM) and first NREM cycle length and composition. Many of these group differences, however, in particular the ones pertaining to the initial NREM cycle, seem to

be attributable to one subtype of RD children - the nonspecific group.

Hypothesis 2

One-way analyses of variance were undertaken to document the presence of group differences in latency to the first REM period. The analyses were undertaken twice: once with WASO included (real-time) and again with WASO deleted. Since the results obtained in both conditions did not differ, only those pertaining to real-time analyses are now presented. Results pertaining to the C - RD (pooled) comparisons are presented first, and are followed by those pertaining to the comparisons of Cs to the three subtypes of RD children.

C - RD Pooled

REM Latency

The second hypothesis, suggesting a global group difference in REM latency was confirmed. A significant difference was noted, with RDs exhibiting an extended latency relative to the Cs [$F(1,37)= 7.50$, $p<.009$]. Analyses were conducted to verify whether this extended

REM latency in RD children was associated with the length of their first REM period and/or the number of REM periods experienced across the night. No significant differences were noted on either of these REM sleep variables. These data are presented in Table 17. When latency to the NREM sleep stages were computed (defined as the duration of total bed time recorded from lights out to the onset of stages 1,2,3 and 4), no significant differences were obtained. Mean latencies to NREM and REM sleep stages for both groups are presented in Figure 18.

REM Latency Interval Composition

Since a significant difference in REM latency was observed, analyses examining REM latency interval composition in each group were undertaken in which the absolute (minutes) and relative (percent) amounts of sleep stages and parameters found within this time period were computed. Although it is recognized that there is significant overlap in stage composition between the first NREM cycle and the initial REM latency interval, analyses were nevertheless conducted to detail any differences. These data are presented in

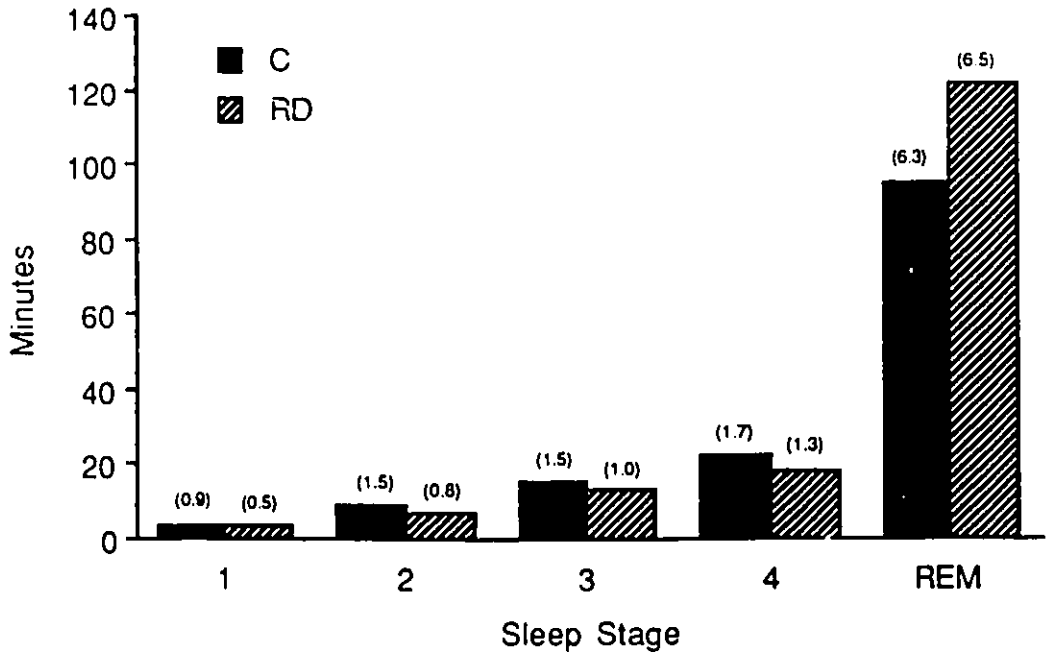
Table 17

Means (SD) of REM Latency-Related Variables (C and RD Pooled)

Variable	Group	
	C	RD
Latency (min)*	94.6 (24.4)	121.2 (32.1)
Length of First REM Period (min)	8.1 (5.1)	12.6 (10.1)
Number of REM .eriods	5.5 (0.5)	5.1 (0.7)

*p<.009.

Figure 18
Mean (SEM) Latencies to Sleep Stages



Tables 18 and 19.

Significant differences were found for absolute amounts of stages 2 and 4 with the RD children exhibiting more of these sleep stages within this time period relative to the Cs [$F(1,37)= 4.25$, $p<.05$; and $F(1,37)= 10.55$, $p<.003$, respectively]. No other differences were found on any sleep stage and parameter variables (absolute amounts) within the REM latency period.

A significant difference for relative amounts of stage 1 was noted, with Cs having increased amounts relative to the RDs [$F(1,37)= 9.29$, $p<.004$]. No significant differences were noted on relative amounts of any other sleep stage and parameter within the latency interval to the first REM period.

C - RD Subtyped

REM Latency

Although overall a significant difference was noted [$F(3,35)= 5.37$, $p<.004$], the specific subtype differences suggested in hypothesis 2 were not those observed. Post-hoc mean comparisons revealed that the nonspecific group had an extended REM latency relative to the controls ($p<.01$). No other significant pair

Table 18

REM Latency Composition: Means (SD) of Sleep Measures in Minutes (C and RD Pooled)

Sleep Measure	Group	
	C	RD
WASO	1.1 (2.4)	0.9 (2.0)
S1	6.4 (3.8)	4.7 (2.6)
S2*	21.6 (13.9)	33.9 (20.4)
S3	11.5 (7.9)	13.7 (7.5)
S4**	53.5 (12.7)	67.8 (13.8)

*$p<.05$. **$p<.003$.

Table 19

REM Latency Composition: Means (SD) of Sleep Measures in Percentage (C and RD Pooled)

Sleep Measure	Group	
	C	RD
WASO	1.2 (2.4)	0.5 (1.1)
S1*	6.6 (3.5)	4.0 (1.9)
S2	20.8 (8.1)	25.3 (10.5)
S3	11.4 (4.9)	10.5 (4.4)
S4	59.5 (13.6)	59.7 (14.0)

*$p<.004$.

-wise differences were found. A significant difference was observed for number of REM periods [$F(3,35)= 3.98$, $p<.02$], with the nonspecifics displaying a reduced number of REM periods relative to both Cs and dyseidetics ($p<.05$). Although no significant difference was obtained for length of the first REM period, group differences did approach significance [$F(3,35)= 2.79$, $p<.06$] with the nonspecifics displaying the longest first REM period. These data are summarized in Table 20. No significant differences were noted for the latency to any NREM sleep stage. Mean latencies to NREM and REM sleep stages for all groups are presented in Figure 19.

REM latency interval composition

REM latency composition data pertaining to the C - RD (subtyped) analyses are presented in Tables 21 and 22. A significant difference was found for absolute amounts of stage 2 sleep [$F(3,35)= 3,43$, $p<.03$], with the nonspecifics showing more of this stage of sleep within this time period than the Cs ($p<.05$). No other significant group differences were identified by post-hoc mean comparisons. A significant difference was also noted on absolute amounts of stage 4 sleep

Table 20

Means (SD) of REM Latency-Related Variables (C and RD Subtyped)

Variable	Group			
	C	DP	DE	NS
Latency (min)**	94.6 (24.4)++	112.3 (26.0)	108.5 (18.3)	142.8 (39.5)++
Length of First REM Period (min)	8.1 (5.1)	11.9 (7.6)	8.2 (4.0)	17.8 (14.5)
Number of REM Periods*	5.5 (0.5)+	5.4 (0.7)	5.4 (0.6)	4.6 (0.7)+

*p<.02. **p<.004. +p<.05. ++p<.01.

Figure 19
Mean (SEM) Latencies to Sleep Stages

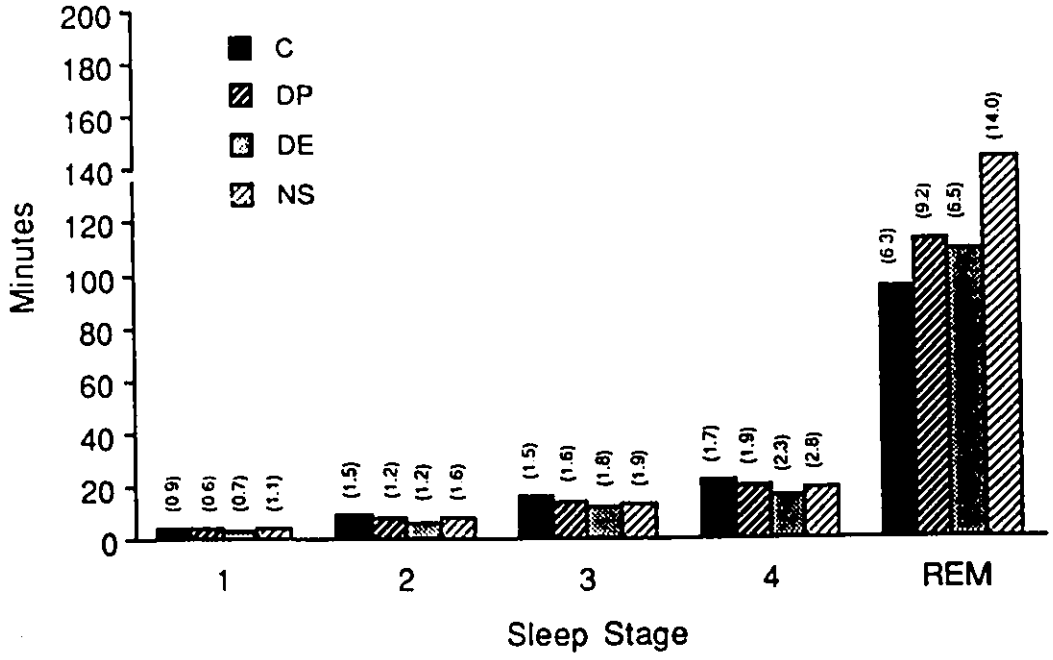


Table 21

REM Latency Composition: Means (SD) of Sleep Measures in Minutes (C and RD Subtyped)

Sleep Measure	Group			
	C	DP	DE	NS
WASO	1.3 (2.4)	0.1 (0.3)	1.0 (2.8)	1.5 (1.8)
S1	6.4 (3.8)	5.1 (2.9)	3.8 (2.3)	5.3 (2.7)
S2*	21.6 (13.9)+	33.0 (22.7)	24.1 (9.6)	44.7 (22.7)+
S3	11.5 (7.9)	11.7 (4.0)	11.0 (6.7)	18.4 (9.4)
S4**	53.5 (12.7)+	62.0 (16.4)	68.3 (11.0)	73.0 (12.8)+

*p<.03. ** p<.009. +p<.05.

Table 22

REM Latency Composition: Means (SD) of Sleep Measures in Percentage (C and RD Subtyped)

Sleep Measure	Group			
	C	DP	DE	NS
WASO	1.2 (2.4)	0.1 (0.2)	1.0 (1.6)	0.8 (1.1)
S1*	6.6 (3.5)	4.7 (2.1)	3.6 (2.1)	3.7 (1.6)
S2	20.8 (8.1)	26.8 (13.7)	20.8 (7.3)	28.4 (9.2)
S3	11.4 (4.9)	10.2 (3.6)	9.3 (4.6)	12.1 (4.9)
S4	59.5 (13.6)	58.2 (17.6)	66.0 (10.3)	55.0 (12.4)

*p<.03.

[F(3.35)=4.51, p<.009], with the nonspecifics having increased amounts relative to the Cs(p<.05). Post-hoc mean comparisons did not identify any other significant group differences for this and other sleep stages and parameters (absolute amounts) within the REM latency period.

A significant difference was obtained on relative amounts of stage 1 sleep [F(3,35)= 3.24, p<.03]. Post-hoc tests did not identify any significant pair-wise differences. No significant differences were observed on any other sleep stage and parameter (relative amounts) within the REM latency period.

In summary, as suggested by the second hypothesis, RD children (pooled) displayed an extended REM latency relative to Cs. Contrary to the hypothesis, however, subtype analyses revealed that the nonspecific subtype displayed the most extended latency relative to the Cs. This was not the expected subtype difference. Although the extended REM latency did not affect the length of the first REM period or the number of REM periods experienced by the RD children pooled into one group, the extended latency did seem to affect the subsequent REM sleep of the nonspecific subtype. These children

experienced an overall reduced number of REM periods relative to Cs, and displayed a tendency toward a longer first REM period relative to other subjects.

Analyses examining REM latency composition revealed that RD children (pooled) displayed more minutes of stages 2 and 4 within this time period relative to Cs. This difference, however, was greatest between nonspecifics and Cs. The Cs exhibited relatively more stage 1 during this time period when compared to the RDs (pooled), but did not demonstrate increased amounts of stage 1 relative to any specific RD subtype.

These results suggest, therefore, an overall difference in REM latency and REM latency composition between Cs and RD children. Generally, however, it is the nonspecific subtype which seems to differ the most from the controls.

Hypothesis 3

One-way analyses of variance were undertaken to document the presence of group differences on EMD measures (nights 3-4 collapsed). Results of the

comparisons of Cs to RD children (pooled) are presented first, followed by those comparing Cs to the three RD subtypes.

C - RD Pooled

Eye Movement Density

The third hypothesis, suggesting reduced EMD in RD children relative to Cs, was not confirmed. Mean REM density was first computed for all REM periods and no significant group differences were noted. Since individual subjects experienced a different number of REM periods, REM density was then calculated across the first four REM periods (since there was insufficient data for an analysis of five or more REM periods). Again, no significant group difference was obtained in REM density measures across these four REM periods.

To examine changes in EMD across the night, mean REM density measures were computed for each of the REM periods. A significant difference was noted on the first REM period, with RDs exhibiting higher mean EMD than Cs [$F(1,37)= 4.09$, $p<.05$). No significant differences were obtained for EMD measures of REM periods 2, 3 and 4. Table 23 summarizes the data pertaining to these EMD measures.

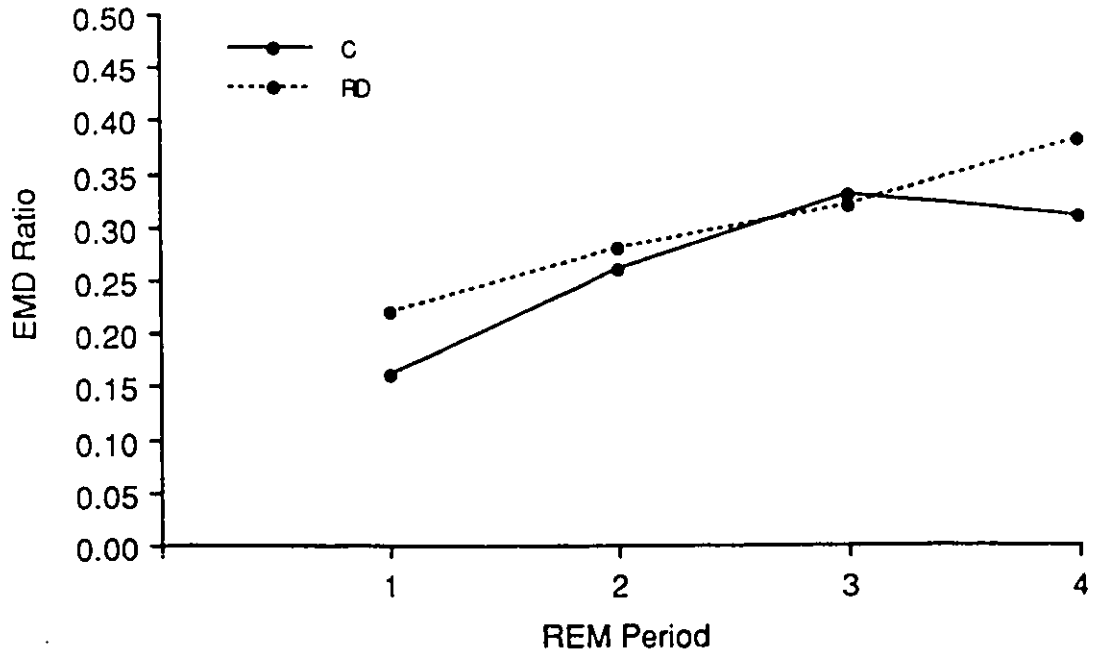
Table 23

Means (SD) of REM Density Measures (C and RD Pooled)

Measure	Group	
	C	RD
Grand Total	0.334 (0.086)	0.341 (0.091)
Total of First Four REM Periods	0.322 (0.080)	0.337 (0.092)
REM Period 1*	0.156 (0.076)	0.224 (0.113)
REM Period 2	0.259 (0.079)	0.280 (0.105)
REM Period 3	0.335 (0.126)	0.318 (0.120)
REM Period 4	0.310 (0.109)	0.376 (0.119)

*$p<.05$.

Figure 20
Variation in Eye Movement Density (EMD) Across the Night



Within-group variations in EMD across the night

Analyses were undertaken to determine within-group variations in EMD across the night (first 4 REM periods - see Figure 20). A significant difference in EMD across REM periods was noted in the Cs, [$F(3,42)= 11.76$, $p<.0003$; quadratic trend, ($p<.04$), (no significant cubic trend)], with EMD measures in REM period 1 being significantly lower than those of REM periods 2 ($p<.05$), 3 and 4 ($p<.01$). A significant difference in EMD across REM periods was observed in RD subjects, [$F(3.69)= 20.56$, $p<.0001$, linear trend, ($p<.0001$), no significant quadratic or cubic trend)], with increasing REM density across the night. Post-hoc mean comparisons revealed the following relationships for EMD measures: Cs - REM period 1 < REM periods 2 ($p<.05$), 3 and 4 ($p<.01$); and, RDs - REM period 1 < REM periods 2 ($p<.05$), 3 and 4 ($p<.01$); REM period 2 < REM period 4 ($p<.01$); REM period 3 < REM period 4 ($p<.05$).

C - RD Subtyped

Eye Movement Density

The specific subtype differences suggested in the third hypothesis were not observed. No significant differences were observed on mean EMD measures computed

for all REM periods, the first 4 REM periods or any individual REM periods. The nonspecifics did display, however, a nonsignificant tendency toward increased EMD in the first REM period, relative to the Cs. These data are presented in Table 24.

Within-group variations in EMD across the night

A significant difference in EMD across REM periods was observed in dysphonetic [$F(1,7)= 8.56$, $p<.002$; linear trend, $p<.006$, no significant quadratic or cubic trend], dyseidetic [$F(1,7)= 11.01$, $p<.02$; linear trend, $p<.002$, no significant quadratic or cubic trend] and nonspecific [$F(1,7)= 4.38$, $p<.02$; linear trend, $p<.001$, no significant quadratic or cubic trend] subjects, with increasing EM density across the night. Post-hoc mean comparisons revealed the following relationships for EMD measures: dysphonetics - REM period 1 < REM periods 3-4, ($p<.05$); REM period 2 < REM period 4, ($p<.05$); and, dyseidetics - REM period 1 < REM period 4, ($p<.01$). Despite an increasing linear trend, no significant differences in EMD measures across REM periods were observed in the nonspecific subjects (see Figure 21).

In summary, therefore, no significant differences

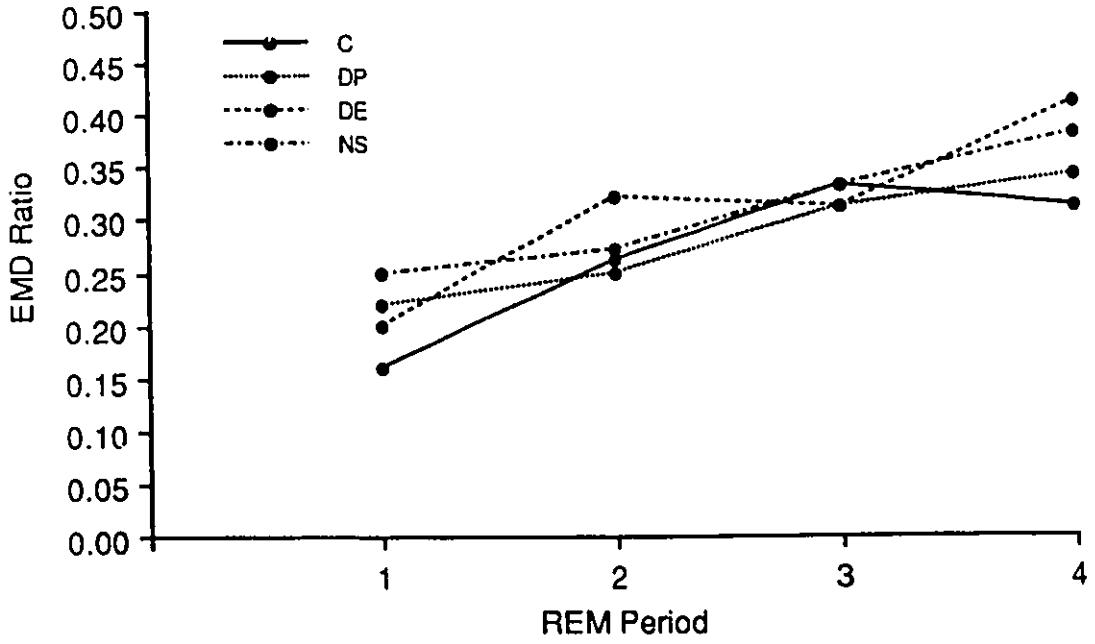
Table 24

Means (SD) of REM Density Measures (C and RD Subtyped)

Measure	Group			
	C	DP	DE	NS
Grand Total	0.334 (0.086)	0.313 (0.056)	0.361 (0.079)	0.348 (0.128)
Total of First Four REM Periods	0.322 (0.080)	0.306 (0.068)	0.354 (0.071)	0.337 (0.130)
REM Period 1	0.156 (0.076)	0.219 (0.051)	0.199 (0.071)	0.252 (0.181)
REM Period 2	0.259 (0.079)	0.251 (0.074)	0.316 (0.065)	0.272 (0.154)
REM Period 3	0.335 (0.126)	0.308 (0.091)	0.312 (0.108)	0.333 (0.164)
REM Period 4	0.310 (0.109)	0.340 (0.083)	0.412 (0.109)	0.377 (0.158)

Figure 21

Variation in Eye Movement Density (EMD) Across the Night



were noted between C and RD children (pooled or subtyped) when EMD was measured for all REM periods, and across the first 4 REM periods. With the exception of the initial REM period, in which the RD children (pooled) exhibited higher mean EMD than the Cs, no significant differences were noted when REM density measures were computed for each of the REM periods.

Generally, within-group analyses revealed significant increases in EMD from REM period 1 to REM period 4 in Cs and RDs (pooled). Subtype analyses revealed significant increases in EMD from REM period 1 to REM period 4 in dysphonetic and dyseidetic subtypes. In nonspecifics, EMD increases from REM period 1 to REM period 4 were present but not significant.

Contrary to the hypothesis EMD measures did not greatly discriminate C from RD children. Within-group analyses revealed that all groups generally displayed increased EMD across the night, but that this increase was somewhat attenuated in the nonspecific group.

CHAPTER 4: DISCUSSION

Adaptation Effects

As presented in the previous chapter, C and pooled RD subjects exhibited across night increases in total sleep time, sleep efficiency index, and REM sleep and across night decreases in sleep onset latency, total wake time, wake time after sleep onset and stage 1 sleep. These observed night differences suggest that the children underwent a period of adaptation to the sleep laboratory environment with lower overall quality of sleep on night 1 relative to the subsequent nights - ("first night effect" - Agnew et al., 1966; Rechtschaffen & Verdone, 1964; Schmitt & Kaelbling, 1971). They also provide additional evidence for the presence of adaptation effects in normal preadolescent children (Busby et al., 1981; Carskadon et al., 1987; Coble et al., 1987) and provide data regarding their presence in RD children as well.

Subtype analyses, however, revealed that although present, these adaptation effects seem attenuated for some variables in dysphonetic and dyseidetic children

and for all variables in nonspecific RD children. The "first night effect", therefore, seems weaker in the subtypes of RD children relative to the Cs, and possible subtype differences in adaptation are suggested by the relatively greater attenuation of these effects in the nonspecific children. These differences should be kept in mind throughout the discussion since many of the sleep architecture differences, although present in the RD pooled group, are greatest in the nonspecific subtype. The possible implications of such a subtype difference for Boder's typology and diagnostic instrument will be reviewed in the last section of this chapter.

Although the possible attenuation of adaptation effects in subtypes of RD children may be well within the normal range of variation, it may also suggest that these subjects habituate more rapidly than Cs to a variety of experimental situations. Perhaps the frequent assessments they undergo, in addition to the remedial training they receive in special education classes make them feel somewhat more at ease in such situations. However, hyperactive children, who are also subjected to frequent assessments, exhibited

"normal" adaptation effects relative to Cs (Busby et al., 1981). This suggests that, despite some degree of habituation to experimental situations, this factor is likely not solely responsible for the attenuation of adaptation effects in RD subtypes.

Alternatively, RD children may display, as of the first night, an increased physiological need for sleep relative to Cs and may therefore experience overall less disrupted sleep on night 1 than do the Cs.

One possible factor for this is that, contrary to what was previously suggested, RD children experienced higher levels of "experiment-related" anxiety prior to their participation in the study than did the Cs. Perhaps this increased anxiety disrupted sleep on the nights preceding the experiment, rendering them somewhat sleep deprived. The accumulated fatigue resulting from such sleep deprivation may have facilitated their adaptation to the laboratory setting and resulted in less disrupted sleep on night 1. However, if experiment-related anxiety did contribute to adaptation differences, it could be expected that their sleep on night 1 would have been more disrupted as they were actually in the anxiety-provoking

situation.

Since no sleep log or anxiety scales were obtained from the subjects, it is difficult to ascertain whether or not the RD children in general, and the nonspecifics in particular, did in fact experience more experiment-related anxiety than did Cs, and whether this resulted in sleep deprivation. Certainly, future research with this or similar populations should include such measures to control for such potentially confounding variables. Nevertheless, prior to their participation in the study, subjects were screened for obvious emotional problems and any child exhibiting disruptive levels of anxiety regarding the sleep experiment was excluded. Only two children (one C and one RD) were excluded because of this factor. As well, clinical observations during both the selection procedure and during the sleep recording sessions did not suggest that the RD children were more anxious than the Cs regarding sleeping in the laboratory. Although increased amounts of experiment-related anxiety may have been a factor, it is likely not the most salient one.

Another possibility is that the RD children suffer

from longer term sleep insufficiency resulting in a general increased need for sleep. This factor would appear to facilitate their adaptation to the recording environment relative to children who habitually obtain adequate amounts of sleep. However, the validity of such an explanation, as well as the possible causes underlying such chronic sleep inadequacy cannot be verified solely on the basis of data pertaining to adaptation effects.

Sleep Architecture (Hypotheses 1 and 2)

Sleep architecture results revealed general differences between RD and C subjects in amounts of stages 4 and REM sleep, and in duration and stage composition of the initial NREM cycle and REM onset latency. The lack of significant group differences in NREM-REM cycle length over the remainder of the night focuses attention upon the initial NREM sleep cycle.

Description of the Initial NREM Cycle and REM Onset Latency

The initial NREM cycle and REM onset latency in RD children showed increased minutes of stage 4 and stage

2 sleep relative to the Cs. This was especially evident in the nonspecific subtype. The increased absolute amounts of stage 4 sleep seem to be the factor which particularly extends the duration of the initial NREM cycle and may contribute to the build up of REM pressure, as evidenced by the nonsignificant tendency towards a longer first REM period in the RD children. Such a tendency contradicts previous documentation in adults of a shortened REM period following an extended NREM cycle (Feinberg, 1989; Feinberg, Koresko, & Heller, 1967). The extended initial NREM cycle of the RD group may provoke an overall decrease in REM sleep, as suggested by the significantly reduced REM sleep percentage in RD children and their tendency towards displaying a reduced number of REM periods (a reduction found to be significant in the nonspecifics). Thus, the REM sleep reduction evident in RD children may be relevant to the information processing deficits thought to be present in this population (Boder, 1971a, 1971b, 1973; Boder & Jarrico, 1982; Pirozzolo, 1979; Rourke, 1985).

Differences in REM sleep amounts were greatest between the Cs and nonspecific RD children. However,

specific information processing deficits are not believed to underlie the reading difficulties exhibited by this latter group (Boder, 1971a, 1971b, 1973; Boder & Jarrico, 1982). This finding may suggest that, contrary to Boder's findings, cognitive impairments are present in this group that could not be identified by the Boder Test of Reading-Spelling Patterns. This speculation would support the findings of Lund et al. (1984) and Nockelby and Galbraith (1984) who reported that the BTRSP frequently misdiagnoses severely dyslexic children as nonspecifics. Although such a conclusion is premature at this point, this finding does suggest that the nature of the postulated relationship between REM sleep and the specific subtype differences in cognitive style is unclear. As well, whether the relationship between REM sleep and information processing is a function of overall duration of REM sleep, or REM sleep intensity, or both, remains undetermined.

Factors Affecting Initial NREM Cycle Duration and REM Onset Latency Interval

Various factors are known to affect the duration of the initial NREM cycle and REM onset latency. For

example, conditions such as major affective disorder, narcolepsy and schizophrenia have been associated with reduced REM onset latency (Hiatt, Floyd, Katz, & Feinberg, 1985; Kupfer & Ehlers, 1989; Kupfer & Foster, 1972; Kupfer & Thase, 1983; Montplaisir, Billiard, Takahashi, Bell, Guilleminault, & Dement, 1978; Zarcone, Benson, & Berger, 1987). An additional factor related to the duration of the initial NREM cycle is total sleep or slow wave sleep deprivation. Investigations examining the effects of such deprivation on recovery sleep suggest that most of the lost stage 4 sleep is recovered (Agnew, Webb, & Williams, 1964; Berger & Oswald, 1962; Feinberg, 1974; Moses, Johnson, Naitoh, & Lubin, 1975; Webb & Agnew, 1971). More recent investigations suggest that this stage 4 rebound occurs mainly within the first NREM cycle, thus extending its length, and demonstrating the special plasticity of this first cycle (Feinberg, Floyd, & March, 1987, 1991; Travis, Maloney, Means, March, & Feinberg, 1991). Duration of the initial NREM cycle and REM onset latency, as well as the amounts of stage 4 sleep found within this time period, are also influenced directly by maturation (Feinberg, 1974,

1989; Kupfer & Ehlers, 1989; Roffwarg et al. 1966). A short REM onset latency is usually documented in neonates and infants. As sleep consolidates and begins to occupy primarily the nocturnal hours, REM onset latency and, as such, the duration of the initial NREM cycle increase (Coons, 1987; Coons & Guilleminault, 1982; Hoppenbrouwers, 1987; Parmelee & Stern, 1972). Normative sleep studies have documented the presence of an extended initial NREM cycle in post-napping younger children relative to older pre-adolescent, adolescent and adult populations (Coble et al., 1984, 1987; Feinberg, 1974; Feinberg et al., 1967: Feinberg & Floyd, 1979) with much of this time period being spent in stage 4 sleep. Thus, these studies demonstrate that during post-napping years, the initial NREM cycle and REM onset latency, as well as the amounts of stage 4 sleep found within these periods decrease with maturation. Both maturational and sleep deprivation issues may underlie the extended initial NREM cycle and REM onset latency in the RD children relative to the Cs.

The sleep profile observed in RD children, and in the nonspecific group in particular, is characterized

by an extended initial NREM cycle and REM sleep latency, increased global amounts of stage 4 sleep and decreased amounts of REM sleep, increased minutes of stage 4 in the initial NREM cycle, and decreased stage 1 sleep. This appears to be consistent with increased need for stage 4 sleep and may result from possible long term sleep deprivation. Perhaps RD children are not consistently obtaining adequate amounts of sleep. In addition to altering their sleep patterns, chronic sleep deprivation would likely increase their level of daytime sleepiness, which may affect attention, memory, alertness and cognitive functioning, and possibly exaggerate existing deficits (Carskadon et al., 1987; Carskadon & Dement, 1987). Whether or not increased sleepiness contributed to the results obtained in this study remains unknown, as overall levels of sleepiness and possibly related napping behaviors were not monitored. Such measures would be necessary to document physiological and behavioral evidence of chronic sleep deprivation. Furthermore, the degree to which daytime sleepiness is problematic is not reported in the RD literature. Therefore, evidence of long term sleep deprivation, as it relates solely to

the total sleep time usually exhibited by these children, is presently inconclusive.

The sleep recording protocol which limited total bed time (TBT) to 9 hours, may not have provided the subjects with an adequate amount of sleep, as suggested by norms published by Williams et al. (1974). However, various factors argue against such an experimentally imposed sleep deprivation as being the source of the results. Firstly, although no sleep log was obtained by which the usual TBT of the subjects could be documented, the schedule followed during the sleep recording period was fairly consistent with the children's regular schedule at home (parent's report), as well as with TBT allowed in more recently published norms of sleep in pre-adolescent children (Coble et al., 1984, 1987; Palm, Persom, Elmquist, & Blennow, 1989). Secondly, if the recording protocol provided the subjects with a substantially reduced sleep period relative to their usual amount, a stage 4 sleep rebound would have been expected to occur in later recording nights as is normally observed during recovery sleep following consecutive nights of deprivation (Agnew et al., 1964; Feinberg et al., 1987, 1991; Moses et al.,

1975; Travis et al., 1991). As illustrated in Tables 5 and 6, minutes and percentage values of stage 4 sleep in all groups are consistent across the four nights, demonstrating the absence of such a rebound. Finally, an experimentally imposed sleep deprivation would have been evident in all groups, as TBT and TST were consistent across all subjects. The increased need for sleep in the RD children that has been suggested, especially in the nonspecifics, may not result from any known externally imposed sleep deprivation. Rather, it may reflect an underlying endogenous process specific to these children which may be a function of a maturational delay (Bakker, 1973; Bakker et al., 1973; Boder & Jarrico, 1982; Geshwind & Behan, 1982; Hooper & Willis, 1989; Hynd et al., 1990; Hynd & Hynd, 1984; Satz et al., 1971).

Possible Influence of Maturational Delay on the Functional Quality of Sleep

As reviewed in Chapter 1, recent evidence suggests neurodevelopmental errors, ocurring pre- and postnatally in RD children, may disrupt normal neuronal migration and organization, and overall corticogenesis. Such disruptions may delay the maturation of the left

hemisphere, or more precisely of the cortical areas and interconnections thought to be implicated in the reading system, and interrupt the normal establishment of linguistic functioning (Duane, 1991; Geshwind & Behan, 1982; Hooper & Willis, 1989; Hynd et al., 1990; Hynd & Hynd, 1984; Hynd & Willis, 1988). This maturational delay may render RD children somewhat more similar to younger normal children than to age-matched normal peers. As such, they may exhibit an increased need for stage 4 sleep. Interestingly, stage 4 percentage values obtained for the RD children in this study are similar to those reported in 6-7 year-old children and are higher than those obtained for normal 8-10 year-old pre-adolescents (Coble et al., 1984, 1987). However, one important consideration is that the postulated delay in corticogenesis and the disruptions in neuronal organization in RD children, affect the degree of speed and accuracy with which they process information, as is suggested by their deficits. As such deficits are not present in younger normal children, it may not be accurate to completely attribute the observed results to such a maturational parallel. Perhaps such a maturational delay in

corticogenesis, which affects neuronal efficacy, slows cerebral restitution, a process thought to be afforded by stage 4 sleep (Borbély, 1982; Feinberg, 1974, 1990; Feinberg & Evarts, 1969; Horne, 1988, 1989). As such, this particular functional quality of stage 4 sleep may be decreased in RD children relative to Cs. Therefore, increased amounts of this stage of sleep may be required to compensate for the extended restitution process in these children. Although these children may not suffer from actual sleep deprivation, they may be deprived of some of the functional benefits of stage 4 sleep.

The tendency exhibited by the RD children towards displaying increased amounts of stage 4 sleep later in the night relative to Cs, may also be indicative of a decelerated restitution process. Perhaps this process cannot be completed during the initial NREM cycle due to a build-up of REM pressure which may trigger the first REM period. Exaggerated stage 4 sleep requirements may not be met during the initial cycle and the extension of stage 4 across the night may reflect an attempt to compensate. However, such an explanation is tentative as the amounts of stage 4

sleep in the last third of the night were not significantly higher than those observed for the Cs.

Presently, however, it is not clear why stage 2 sleep, which primarily constitutes the remainder of the initial NREM cycle in these children, was not sacrificed to allow for greater amounts of stage 4 sleep during this time period. This finding may illustrate slowed functioning of cortical processes which generate stage 4 sleep, but may also be related to a possible function of stage 2 sleep. Although quantitatively different, stage 2 sleep may serve a qualitative function similar to the one postulated for stage 4 sleep (Feinberg & Evarts, 1969). Thus, this stage of sleep may be maintained because of its possible functional significance in the restitution process.

The present results cannot confirm the structural and morphological cerebral substrates that have been related to RD deficits (Galaburda et al., 1987; Galaburda & Eidelberg, 1982; Galaburda & Kemper, 1979; Hooper & Willis, 1989; Hynd et al., 1990; Hynd & Hynd, 1984). The extent to which such issues can be elucidated on the basis of sleep-related EEG measures

is limited. However, they do provide evidence regarding underlying abnormal physiological features in RD children, perhaps as a function of a delayed maturational process. In addition, the subtype differences observed suggest that a different degree of maturational delay may be involved in the various subgroups, and support the importance of the homogeneous subtyping.

However, this interpretation is speculative. Firstly, the results obtained are based on visually-scored sleep EEG. More direct evidence of cortical underactivation at various cortical sites in RD children, relative to Cs, would necessitate quantitative analyses of the EEG parameters. Such analyses would facilitate the documentation of absolute and relative delta density differences between these groups of children and may provide more direct support for the view that decelerated cortical processes is related to RD deficits.

Additionally, studies documenting the effects of sleep deprivation on subsequent sleep parameters, as well as the neurological models of reading disabilities proposed in the literature, stem from research

undertaken in adult populations. Therefore, the extent to which these findings, as well as their possible interrelationships, can be generalized to children must be done with caution.

Finally, factors such as immunological abnormalities may also be relevant to the results. Some evidence suggests that corticogenesis may be disrupted by autoimmune factors (Galaburda, 1990; Geshwind & Behan, 1982). A relationship between the immune system and slow wave sleep (SWS) has been postulated. Some investigations suggest that SWS might serve to sustain the normal functioning of this system (Horne, 1988; Krueger, Walter, & Levin, 1985; Moldofsky, Lue, Eisen, Keystone, & Gorzynski, 1986). In part, the increased presence of stage 4 sleep in RD children may result from an attempt to compensate for potential immunological deficiencies. However, evidence pertaining to the presence and role of immunological factors in reading disabilities, stemming mainly from studies documenting increased prevalence of allergies, immune disorders of the digestive tract, and thyroid dysfunctions in RD populations, is still limited (Galaburda, 1990; Geshwind & Behan, 1982; Hynd

et al., 1990; Lubs, Rabin, Carland-Saucier, Wen, Gross-Glenn, Duara, Levin, & Lubs, 1991). Any conclusions regarding their possible relationship to the observed results are premature at this point.

Eye Movement Density (Hypothesis 3)

Global Measures of Eye Movement Density

The lack of significant group differences in global EMD measures stands in contrast to the anticipated lower EMD levels in RD children relative to Cs and to the hypothesized subtype differences. Such findings do not seem concordant with the positive association (both theoretical and empirical) of EM activity during REM sleep with information processing and CNS maturation and their possible relevance to normal cognitive development (Christ et al., 1991; De Koninck et al., 1975, 1987; De Koninck & Prévost, 1991; De la Pena et al., 1973; Dittrichova et al., 1972; ; Petre-Quadens & de Lee, 1970; Petre-Quadens, de Lee, & Remy, 1971; Smith & Lapp, 1991).

Such discrepant findings may result, in part, from methodological factors. The EMD measures in this

present investigation were based upon the number of two second epochs containing at least one eye movement. This measure may not have been sensitive to density differences between C and RD children. A more discrete EMD measure, such as individual horizontal and vertical eye movements, may provide different results.

In addition, the quantity of information processed by RD children during prior wakefulness was not measured. Therefore, it is not clear whether the results obtained represent their baseline EMD levels, or are a reflection of possible increases in information processing demands (i.e., remedial training). Although it would be methodologically tedious to do so, the manipulation and measurement of the quantity of information processed during prior wakefulness may be necessary to clarify this issue.

Alternatively, however, EMD during REM sleep may not linearly reflect waking information processing capabilities, a possibility previously suggested by Busby and Pivik (1983). Indeed, the many investigations attempting to empirically document such a relationship have employed experimental protocols and conditions which access different modalities and levels

of information processing. Therefore, the relationship between EMD in REM sleep and information processing may not simply depend on increased or decreased overall cognitive capabilities. It may also depend on the precise nature of the cognitive capabilities being examined, (i.e., mental retardation, autism, reading disabilities, normal or superior intelligence) as well as the conditions under which these were required and measured (i.e., intensive learning). Thus, the results suggest that the presence or nature of a relationship between EMD and information processing in these particular reading disabled children may not be elucidated under the present experimental conditions.

Eye Movement Density Across the Night

Analyses of EMD across the night revealed more EMD in RD (pooled) relative to C subjects during the first REM period. A tendency for this effect to be greatest between the nonspecific RD and C children was observed. Such a finding in children who exhibit an increased presence of stage 4 sleep seems inconsistent with the documented reduction in EMD with greater sleep depth (Aserinsky, 1969; Feinberg, 1974; Feinberg, Floyd, & March, 1987). As well, it does not conform to De la

March, 1987). As well, it does not conform to De la Pena's SCIP hypothesis (De la Pena et al., 1973) which would predict a decrease in EMD in these children since, during wakefulness, they do not process information which is task specific (i.e., reading) with efficient cognitive structures. However, as mentioned previously, the presence and nature of the relationship between EMD and information processing in these children remains unclear. It may be loosely speculated that this particular result stems from the combined effects of the delayed REM onset and the decreased overall amounts of REM sleep in these children relative to Cs. Perhaps the higher levels of EMD observed solely during the first REM period represent an incomplete compensatory mechanism for their relative REM deprivation.

Within-Group Variations in Eye Movement Density Across REM Periods

Additional analyses pertaining to within-group variations in EMD across REM periods, however, may be more relevant to the RD children's possible increased need for stage 4 sleep, reflecting underlying maturational processes, than analyses of global EMD

measures. Overall, these analyses revealed that C children displayed a pattern similar to that originally reported by Aserinsky (1969; 1973), and subsequently by Feinberg (1974) and Busby et al. (1981) in young adults and children.

Aserinsky (1969; 1973) reported that EMD gradually increases across REM periods and peaks following 7.5-10 hours of sleep. After this, EMD measures remain relatively stable. The pattern displayed by the C children is similar, as illustrated by the obtained significant quadratic trend. EMD measures increase from the first to the third REM period and remain relatively unchanged through REM period 4. Such a plateau in EMD measures may be related to possible sleep satiety in these children, as suggested by Aserinsky (1969). The RD children, however, (pooled and subtyped), displayed an increasing linear trend in EMD across REM periods, and thus their values do not reach a peak value in the third REM period as they do in the Cs. Such a pattern suggests, in light of Aserinsky's view, that these children may not have reached their necessary quota of sleep, and as such, may be a marker of long term sleep deprivation, one

possibility presented earlier. Again, as data pertaining to potential levels of daytime sleepiness were not obtained, such an interpretation remains tenuous. However, sleep satiety in these children may not depend solely upon the total quantity of sleep obtained, but also on the overall functional quality of sleep. Perhaps, their EMD pattern across the night is a function of the stage 4 inefficiency and may reflect an uncompleted restitution process.

Both the dysphonetic and dyseidetic subtypes generally show a significant difference in EMD measures from REM period 1 to REM period 4. The differences observed in the nonspecifics, however, were not significant. This observation may support the earlier suggestion of a greater attenuation of the restitution process in this particular subtype. However, the precise relationship between EMD profile across REM periods and the specific cognitive impairments in the various subtypes, remains unclear.

Implications for Boder's Typology and The BTRSP

The sleep-parameter modulations observed suggest the presence of physiological features in RD children -- possibly related to a delayed maturational process -- which underlie the various cognitive deficits observed in this population. However, the nature or presence of any relationship between sleep-related parameters and the specific information processing deficits exhibited by these children remains unclear.

The global sleep-related differences observed between C and RD children (pooled) suggest that such differences may be present in all RD children but to differing degrees. As well, there are common features in the sleep architecture and eye movement activity of the three subtypes which render them more similar to each other than to Cs. Nevertheless, subtype analyses revealed that the greatest variations occur between Cs and one subgroup of RD children in particular - the nonspecifics. This finding is consonant with the heterogeneous nature of the RD population, and may reflect differences in the degree to which maturational

processes may be delayed in the individual subtypes. As such, these results underscore the importance of neurodevelopmental considerations in future RD subtyping research.

The particular subtype differences obtained in sleep seem surprising in light of Boder's description of the nonspecific RD subgroup. As reviewed in Chapter 1, the reading and spelling difficulties exhibited by this group do not result primarily from cognitive deficits stemming from neurodevelopmental delay, but rather from physical, mental, emotional, sociocultural and/or educational factors. The prognosis for normal reading achievement in these children is excellent if such factors can be alleviated (Boder, 1971b; Boder & Jarrico, 1982). Therefore, according to Boder, the nonspecific RD children are essentially similar to normal readers. However, the sleep-related measures obtained in this present study reveal that, of the three subtypes, the nonspecifics differ the most from Cs.

The significant differences obtained for the nonspecific subtype do provide a measure of reliability for the typology in that this group differs not only

from Cs, but also from the other subtypes. However, these same differences also suggest that Boder's interpretation of the behavioral profile of these children, based upon their reading-spelling pattern, may need revision. As such, the previously mentioned concerns regarding the validity and clinical usefulness of the BTRSP (Alexander, 1984; Reynolds, 1984) and, more precisely, the reported frequent misdiagnoses of severely dyslexic readers as nonspecifics (Lund et al., 1984; Nockleby & Galbraith, 1984) must be given serious consideration.

The BTRSP may not be sensitive or comprehensive enough to detect and identify cognitive impairments of some children, resulting in their misdiagnosis as nonspecifics. Presently, however, not enough is known regarding the neuropsychological characteristics of this particular group to make inferences about potential underlying deficits. A complete neuropsychological and academic achievement test battery may be necessary to isolate cognitive impairments disrupting their ability to develop efficient reading and spelling skills, and may help determine the degree to which this particular group of

children constitutes a homogeneous dyslexic subtype.

Certainly, the clinical, social, and personal consequences resulting from misdiagnosing possibly severely deficient readers as nonspecifics warrants the use of complete and accurate psychodiagnostic procedures. These consequences also underscore the importance of continued research into the relationships between physiological and cognitive features of RD subtypes in order to obtain the most accurate and discriminating screening measures.

REFERENCES

Aaron, P. G. (1978). Dyslexia, an imbalance in cerebral information processing strategies. Perceptual and Motor Skills, 47, 699-706.

Aaron, P. G. (1982). The neuropsychology of developmental dyslexia. In R.N. Malatescha & P. G. Aaron (Eds.), Reading disorders: Varieties and treatment (pp. 5-67). New York: Academic Press.

Adelman, H. S., & Taylor, L. (1986). The problems of definition and differentiation and the need for a classification schema. Journal of Learning Disabilities, 19, 514-520.

Agnew, H. W., Webb, W. B., & Williams, R. L. (1964). The effect of stage four sleep deprivation. Electroencephalography and Clinical Neurophysiology, 17, 68-70.

Agnew, H. W., Webb, W. B., & Williams, R. L. (1966). The first night effect: An EEG study of sleep. Psychophysiology, 2, 263-266.

Albert, I. B. (1975). REM sleep deprivation. Biological Psychiatry, 10, 341-351.

Alexander, P. A. (1984). Enlarging the gap between theory and practice: A review of the Boder Test of Reading-Spelling Patterns. School Psychology Review, 13, 529-533.

Algozzine, B., Ysseldyke, J. E. (1986). The future of the LD field: Screening and diagnosing. Journal of Learning Disabilities, 19, 394-398.

Allen, S. R., Lewis, S., & Tagney, J. (1972). The effects of distorted visual input on sleep. Psychophysiology, 9, 498.

Aserinsky, E. (1969). The maximal capacity for sleep: Rapid eye movement density as an index of sleep satiety. Biological Psychiatry, 1, 147-159.

Aserinsky, E. (1973). Relationship of rapid eye movement density to the prior accumulation of sleep and wakefulness. Psychophysiology, 48, 309-318.

Aserinsky, E., Lynch, J. A., Mark, M. E., Tzankoff, S. P., & Hurn, E. (1985). Comparison of eye motion in wakefulness and REM sleep. Psychophysiology, 22, 1-10.

Aylward, E. H. (1984). Lateral asymmetry in subgroups of dyslexic children. Brain and Language, 22, 221-231.

Bakker, D. J. (1973). Hemispheric specialization and stages in the learning to read process. Bulletin of the Orton Society, 23, 15-27.

Bakker, D. K., Smink, T., & Reitsma, P. (1973). Ear dominance and reading ability. Cortex, 9, 301-312.

Bakker, D. J., Teunissen, J., & Bosch, J. (1976). Development of laterality reading patterns. In R. M. Knights & D. J. Bakker (Eds.), The neuropsychology of learning disorders: Theoretical approaches (pp. 207-220). Baltimore: University Park Press.

Baron, J. (1979). Orthographic and word specific mechanisms in children's reading of words. Child Development, 50, 60-72.

Bauserman, D. N., & Obrzut, J. E. (1981). Spatial and the temporal matching ability among subgroups of disabled readers. Contemporary Educational Psychology, 6, 306-313.

Bayliss, J., & Liversey, P. J. (1985). Cognitive strategies of children with reading disabilities and normal readers in visual sequential memory. Journal of Learning Disabilities, 13, 326-332.

Benton, A. L. (1975). Developmental dyslexia: Neurological aspects. In W. J. Friedlander (Ed.), Advances in neurology (Vol. 7, pp. 1-47). New

York: Raven Press.

Benton, A. L. (1977). Some conclusions about dyslexia. In A. L. Benton & D. Pearl (Eds.), Dyslexia: An appraisal of current knowledge (pp. 453-476). New York: Oxford University Press.

Berger, R. J., & Oswald, I. (1962). Effects of sleep deprivation on behavior, subsequent sleep, and dreaming. Journal of Mental Science, 108, 457-465.

Birch, H., & Belmont, L. (1964). Auditory-visual integration in normal and retarded readers. American Journal of Orthopsychiatry, 34, 852-861.

Bloch, V., Hennevin, E., & Leconte, P. (1981). The phenomenon of paradoxical sleep augmentation after learning: Experimental studies of its characteristic and significance. In W. Fishbein (Ed.), Sleep, dreams and memory (pp. 1-18). New York: SP Medical & Scientific Books.

Boder, E. (1971a). Developmental dyslexia: A diagnostic screening procedure based on three characteristic patterns of reading and spelling. In B. Bateman (Ed.), Learning disorders (Vol. 4, pp. 298-342). Seattle: Special Child Publications.

Boder, E. (1971b). Developmental dyslexia: prevailing diagnostic concepts and a new diagnostic approach. In H. Myklebust (Ed.), Progress in learning disabilities (Vol. 2, pp. 293-321). New York: Grune & Stratton.

Boder, E. (1973). Developmental dyslexia: A diagnostic approach based on three atypical reading-spelling patterns. Developmental Medicine and Child Neurology, 15, 663-687.

Boder, E., & Jarrico, S. (1982). The Boder Test of Reading-Spelling Patterns: A diagnostic screening test for subtypes of reading disability. New York: Grune & Stratton.

Borbély, A. A. (1982). A two-process model of sleep regulation. Human Neurobiology, 1, 195-204.

Breen, M. J. (1986). Cognitive patterns of learning disability subtypes as measured by the Woodcock-Johnson Psychoeducational Battery. Journal of Learning Disabilities, 19, 86-90.

Bryden, M. P. (1970). Laterality effects in dichotic listening: Relations with handedness and reading ability in children. Neuropsychologia, 8, 443-450.

Busby, K., Firestone, P., & Pivik, R. T. (1981). Sleep patterns in hyperkinetic and normal children. Sleep, 4, 366-383.

Busby, K., & Pivik, R. T. (1983). Sleep patterns in children of superior intelligence. Journal of Child Psychology and Psychiatry, 24, 587-600.

Butler, S., & Pivik, R. T. (1991). Dreaming in reading disabled children: Formal features. Dreaming, 1, 193-209.

Camp, B., & Dolcourt, J. (1977). Reading and spelling in good and poor readers. Journal of Learning Disabilities, 10, 300-307.

Carskadon, M. A. (1982). The second decade. In C. Guilleminault (Ed.), Sleeping and waking disorders: Indications and techniques (pp. 99-125). Menlo Park, Cal.: Addison-Wesley.

Carskadon, M. A, & Dement, W. C. (1987). Sleepiness in the normal adolescent. In C. Guilleminault (Ed.), Sleep and its disorders in children (pp. 53-66). New York: Raven Press.

Carskadon, M. A., Keenan, S., & Dement, W. C. (1987). Nighttime sleep and daytime sleep tendency in preadolescents. In C. Guilleminault (Ed.), Sleep and its disorders in children (pp. 43-52). New York: Raven Press.

Christ, G., Hébert, G., Wecklein, M., Mercier, P., & De

Koninck, J. (1991). Language learning, dreams and REM sleep: An attempt to replicate. Sleep Research, 20A, 253.

Coble, P. A., Kupfer, D. J., Reynolds, C. F., & Houck, P. (1987). EEG sleep of healthy children 6 to 12 years of age. In C. Guilleminault (Ed.), Sleep and its disorders in children (pp. 29-41). New York: Raven Press.

Coble, P. A., Kupfer, D. J., Taska, L. S., & Kane, J. (1984). EEG sleep of normal healthy children. Part 1: Findings using standard measurement methods. Sleep, 7(4), 289-303.

Coons, S. (1987). Development of sleep and wakefulness during the first 6 months of life. In C. Guilleminault (Ed.), Sleep and its disorders in children (pp. 17-27). New York: Raven Press.

Coons, S., & Guilleminault, C. (1982). The development of sleep-wake patterns and non-rapid eye movement sleep stages during the first six months of life in normal infants. Pediatrics, 69, 793-798.

Critchley, M. (1972). The dyslexic child. London: Heinemann.

Dalby, J., & Gibson, D. (1981). Functional cerebral lateralization in subtypes of disabled readers. Brain and language, 14, 34-48.

Das, J. P., Kirby, J. R., & Jarman, R. F. (1975). Simultaneous and successive syntheses: An alternative model for cognitive abilities. Psychological Bulletin, 82, 87-103.

De Koninck, J., Christ, G., & Lorrain, D. (1987). Intensive language learning and REM sleep: More evidence of a performance factor. Sleep Research, 16, 201.

De Koninck, J., Lorrain, D., Christ, G., Proulx, G., & Coulombe, D. (1989). Intensive language learning and increases in REM sleep: Evidence of a performance factor. International Journal of

Psychophysiology, 8, 43-47.

De Koninck, J., & Prévost, F. (1991). Le sommeil paradoxal et le traitement de l'information: Une exploration par l'inversion du champ visuel. Revue Canadienne de Psychologie, 45, 125-139.

De Koninck, J., Proulx, G., Healy, T., Arsenault, R., & Prévost, F. (1975). Intensive language learning and REM sleep. Sleep Research, 4, 150.

De la Pena, A., Zarcone, V., & Dement, W. C. (1973). Correlation between measures of the rapid eye movements of wakefulness and sleep. Psychophysiology, 10, 488-500.

Dewan, E. (1970). The programming (P) hypothesis for REM sleep. In E. Hartmann (Ed.), Sleep and dreaming (pp 295-307). Boston: Little, Brown & Co.

Dittrichova, J., Paul, K., & Pavlikova, E. (1972). Rapid eye movements in paradoxical sleep in infants. Neuropediatrie, 3, 238-257.

Doehring, D. G., & Hoshko, I. M. (1977). Classification of reading problems by the Q-technique of factor analysis. Cortex, 13, 281-294.

Doehring, D. G., Trites, R. L., Patel, P. G., & Fiedorowicz, C. A. M. (1981). Reading disabilities: The interaction of reading, language and neuropsychological deficits. New York: Academic Press.

Drake, W. E. (1968). Clinical and pathological findings in a child with a developmental learning disability. Journal of Learning Disabilities, 1, 486-502.

Duane, D. D. (1991). Biological foundations of learning disabilities. In J. E. Obrzut & G. W. Hynd (Eds.), Neuropsychological foundations of learning disabilities: A handbook of issues, methods and practice (pp. 7-27). San Diego:

Academic Press.

Duffy, F. H., Burchfiel, J. L. & Lombroso, C. T. (1979). Brain electrical activity mapping (BEAM): A method for extending the clinical utility of EEG and evoked potential data. Annals of Neurology, 5, 309-321.

Duffy, F. H., Denckla, M. B., Bartels, P. H. & Sandini, G. (1980). Dyslexia: Regional differences in brain electrical activity by topographical mapping. Annals of Neurology, 7, 412-420.

Duffy, F. H., Denckla, M. B., Bartels, P. H., Sandini, G., & Kiessling, L. S. (1980). Dyslexia: automated diagnosis by computerized classification of brain electrical activity. Annals of Neurology, 7, 421-428.

Epps, S., Ysseldyke, J. E., & Algozzine, B. (1983). Impact of different definitions of learning disabilities on the number of students identified. Journal of Psychoeducational Assessment, 1, 341-352.

Epps, S., Ysseldyke, J. E., & Algozzine, B. (1985). An analysis of the conceptual framework underlying definitions of learning disabilities. The Journal of School Psychology, 23, 133-144.

Feinberg, I. (1974). Changes in sleep patterns with age. Journal of Psychiatric Research, 10, 283-306.

Feinberg, I. (1989). Effects of maturation and aging on slow wave sleep in man: Implications for neurobiology. In A. Wauquier, C. Dugovic, & M. Radulovacki (Eds.), Slow wave sleep: Physiological, pathophysiological, and functional aspects (pp. 31-48). New York: Raven Press.

Feinberg, I. (1990). Function(s) of slow wave sleep. In M. H. Chase, & T, Roth (Eds.), Slow wave sleep: Its measurement and functional significance (pp. 65-68). Los Angeles: Brain Information Service/Brain Research Institute.

Feinberg, I., & Evarts, E. V. (1969). Changing concepts of the function of sleep: Discovery ofintense brain activity during sleep calls for revision of hypotheses as to its function. Biological Psychiatry, 1, 331-348.

Feinberg, I., & Floyd, T. C. (1979). Systematic trends across the night in human sleep cycles. Psychophysiology, 16, 283-291.

Feinberg, I., Floyd, T. C., & March, J. D. (1987). Effects of sleep loss on delta (0.3-3 Hz) EEG and eye movement density: New observations and hypotheses. Electroencephalography and Clinical Neurophysiology, 67, 217-221.

Feinberg, I., Floyd, T. C., & March, J. D. (1991). Acute deprivation of the terminal 3.5 hours of sleep does not increase delta (0-3 Hz) electroencephalograms in recovery sleep. Sleep, 14, 316-319.

Feinberg, I., Hibi, S., Brown, M., Cavness, C., Westerman, G. & Small, A. (1974). Sleep amphetamine effects in MBDS and normal subjects. Archives of General Psychiatry, 31, 723-731.

Feinberg. I., Koresko, R. L., Heller, N. (1967). EEG sleep patterns as a function of normal and pathological aging in man. Journal of Psychiatric Research, 5, 107-144.

Fishbein, W., & Gutwein, B. M. (1977). Paradoxical sleep and memory storage processes. Behavioral Biology, 19, 425-464

Fishbein, W., & Gutwein, B. M. (1981). Paradoxical sleep and a theory of long-term memory. In W. Fishbein (Ed.), Sleep, dreams and menory (pp. 147-182). New York: SP Medical & Scientific Books.

Fletcher, J. M. (1981). Linguistic factors in reading acquisition: Evidence for developmental changes. In F. J. Pirozzolo & M. C. Wittrock (Eds.), Neuropsychological and cognitive processes in reading (pp. 261-294). New York: Academic Press.

Fletcher, J. M. & Satz, P. (1980). Developmental changes in the neuropsychological corrolates of reading achievement: A six-year longitudinal follow-up. Journal of Clinical Neuropsychology, 2, 23-27.

Fletcher, J. M. Satz, P., & Scholes, R. J. (1981). Developmental changes in the linguistic performance correlates of reading achievement. Brain and Language, 13, 78-90.

Flynn, J. M., & Deering, W. M. (1989). Subtypes of dyslexia: Investigation of Boder's system using quantitative neurophysiology. Developmental Medicine and Child Neurology, 31, 215-223.

Fox, B., & Routh, D. K. (1980). Phonemic analysis and severe reading disability in children. Journal of Psycholinguistic Research, 9, 115-119.

Fried, I. (1979). Cerebral dominance and sybtypes of developmental dyslexia. Bulletin of the Orton Society, 29, 101-112.

Fried, I., Tanguay, P., Boder, E., Doubleday, C., & Greensite, M. (1981). Developmental dyslexia: Electrophysiological corroboration of clinical subgroups. Brain and language, 12, 14-22.

Galaburda, A. M. (1990). The testosterone hypothesis: Assessment since Geshwind and Behan, 1982. Annals of Dyslexia, 40, 18-38.

Galaburda, A. M., Corsiglia, J., Rosen, G. D., & Sherman, G. F. (1987). Planum temporale asymmetry: Re-appraisal since Geshwind and Levitsky. Neuropsychologia, 25, 853-868.

Galaburda, A. M., & Eidelberg, D. (1982). Symmetry and asymmetry in the human posterior thalamus: II. Thalamic lesions in a case of developmental dyslexia. Archives of Neurology, 39, 333-336.

Galaburda, A. M., & Kemper, T. L. (1979). Cytoarchitectonic abnormalities in developmental dyslexia: A case study. Annals of Neurology, 6,

94-100.

Geshwind, H., & Behan, P. (1982). Left-handnedness: Association with immune disease, migraine and developmental learning disorders. Proceedings of the National Academy of Sciences of the U. S. A., 79, 5097-5100.

Ginn, R. (1979). An analysis of various psychometric typologies of primary reading disabilities. Unpublished doctoral dissertation, University of Southern California.

Greenberg, R. (1970). Dreaming and memory. In E. Hartmann (Ed.), Sleep and dreaming (pp. 258-267). Boston: Little, Brown & Co.

Greenberg, R. (1981). Dreams and REM sleep - an integrative approach. In W. Fishbein (Ed.), Sleep, dreams and memory (pp. 125-145). New York: SP Medical & Scientific Books.

Gross-Glenn, K. Duara, R., Yoshii, F., Barker, W. W., Chang, J. Y. Apicella, A., Boothe, T., & Lubs, H. A. (1987). PET-scan studies during reading in dyslexic and non-dyslexic adults. Neuroscience Abstracts, 15, 371.

Gross-Glenn, K. Duara, R., Yoshii, F., Barker, W. W., Chang, J. Y. Apicella, A., Boothe, T., & Lubs, H. A. (1990). PET-scan reading studies: Familial dyslexics. In G. T. Pavlidis (Ed.), Dyslexia: A neuropsychological and learning perspective (Vol , pp. 109-118). New York: Wiley.

Haig, J. R., Schroeder, C. S., & Schroeder, S. R. (1974). Effects of methylphenidate on hyperactive children's sleep. Psychopharmacologia (Berl.), 37, 185-188.

Hartmann, E. (1981). The functions of sleep and memory processing. In W. Fishbein (Ed.), Sleep, dreams and memory (pp. 111-124). New York: SP Medical & Scientific Books.

Herman, J. H., Barker, D. R., & Roffwarg, H. P. (1983).

Similarity of eye movement characteristics in REM sleep and the awake state. Psychophysiology, 20, 537-543.

Herman, J. H., Erman, M., Boys, R., Steigman, L., Taylor, M. E., & Roffwarg, H. P. (1984). Evidence for a directional correspondance between eye movements and dream imagery in REM sleep. Sleep, 7, 52-63.

Hiatt, J. F., Floyd, T. C., Katz, P. H., & Feinberg, I. (1985). Further evidence of abnormal non-rapid eye-movement sleep in schizophrenia. Archives of General Psychiatry, 42, 797-798.

Hilakivi, L. (1987). Sleeping and dreams in learning-disabled boys. Acta Psychiatrica Scandinavia, 76, 103-105.

Hobson, J. A., & McCarley, R. W. (1977). The brain as a dream state generator: An activation-synthesis hypothesis of the dream process. The American Journal of Psychiatry, 134, 1335-1348.

Holmes, C. L., & Peper, R. J. (1977). An evaluation of the use of spelling error analysis in the diagnosis of reading disabilities. Child Development, 48, 1708-1711.

Hooper, S. R., & Willis, W. G. (1989). Learning disability subtyping: Neuropsychological foundations, conceptual models, and issues in clinical differentiation. New York: Springer-Verlag.

Hoppenbrouwers, T. (1987). Sleep in infants. In C. Guilleminault (Ed.), Sleep and its disorders in children (pp. 1-15). New York: Raven Press.

Horne, J. (1988). Why we sleep: The functions of sleep in humans and other mammals. Oxford: Oxford University Press.

Horne, J. (1989). Functional aspects of human slow wave sleep (hSWS). In A. Wauquier, C. Dugovic, & M. Radulovacki (Eds.), Slow wave sleep:

Physiological, pathophysiological, and functional aspects (pp. 109-118). New York: Raven Press.

Horne, J. A., & McGrath, M. J. (1984). The consolidation hypothesis for REM sleep function: Stress and other confounding factors - a review. Biological Psychology, 18, 165-184.

Hynd, G., & Cohen, M. (1983). Dyslexia: Neuropsychological theory, research, and clinical differentiation. New York: Grune & Stratton.

Hynd, G. W., & Hynd, C. R. (1984). Dyslexia: Neuroanatomical/neurolinguistic perspectives. Reading Research Quarterly, 19, 482-498.

Hynd, G. W., & Obrzut, J. E. (1981). Development of reciprocal hemispheric inhibition in normal and learning-disabled children. Journal of General Psychology, 104, 203-212.

Hynd, G. W., Obrzut, J. E., Weed, W., & Hynd, C. R. (1979). Development of cerebral dominance: Dichotic listening asymmetry in normal and learning disabled children. Journal of Experimental Child Psychology, 28, 445-454.

Hynd, G. W., & Semrud-Clikeman, M. (1989). Dyslexia and brain morphology. Psychological Bulletin, 106, 447-482.

Hynd, G. W., Semrud-Clikeman, M., Lorys, A. R., Novey, E. S., & Eliopulos, D. (1990). Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. Archives of Neurology, 47, 919-926.

Hynd, G. W., & Willis, W. G. (1988). Pediatric neuropsychology. Orlando: Grune & Stratton, Inc.

Jacobs, L., Feldman, M., & Bender, M. B. (1972). Are the eye movements of dreaming sleep related to the visual images of the dreams? Psychophysiology, 9(4), 393-401.

Jeannerod, M., & Mouret, J. (1962). Etude des

mouvements oculaires observés chez l'homme au cours de la veille et du sommeil. Société de Biologie de Lyon, 25, 1407-1410.

Kaufman, A. J. (1976). A four-test short form of the WISC-R. Contemporary Educational Psychology, 1, 180-196.

Khan, A., & Rechtschaffen, A. (1978). Sleep patterns and sleep spindles in hyperkinetic children. Sleep Research, 7, 137.

Kinsbourne, M., & Warrington, E. K. (1963). Developmental factors in reading and writing backwardness. British Journal of Psychology, 54, 145-146.

Krueger, J. M., Walter, J., & Levin, C. (1985). Factor S and related somnogens: An immune theory for slow wave sleep. In D. J. McGinty, R. Drucker-Colin, A. Morrison, & P. L. Parmeggiani (Eds.), Brain mechanisms of sleep (pp. 253-269). New York: Raven Press.

Kupfer, D. J., & Ehlers, C. L. (1989). Two roads to rapid eye movement latency. Archives of General Psychiatry, 46, 945-948.

Kupfer, D. J., & Foster, F. G. (1972). Interval between onset of sleep and rapid eye movement sleep as an indicator of depression. Lancet, 2, 684-686.

Kupfer, D. J. & Thase, M. E. (1983). The use of the sleep laboratory in the diagnosis of affective disorders. In J. Davis, J. Maas (Eds.), Affective disorders II: Advances in diagnosis and treatment (pp. 107-124). Washington, DC: American Psychiatric Press.

Leisman, G., & Ashkenazi, M. (1980). Aetiological factors in dyslexia: IV. Cerebral hemispheres are functionally equivalent. Neuroscience, 11, 157-164.

Lenneberg, E. H. (1967). Biological foudations of

language. New York: Wiley.

Liberman, I. Y., & Shankweiler, D. (1979). Speech, the alphabet and teaching to read. In L. B. Resnick & P. A. Weaver, Theory and practice of early reading (Vol 1, pp. 109-12). Hillsdale N. J.: Erlbaum.

Lubs, H. A., Rabin, M., Carland-Saucier, K., Wen, X. L., Gross-Glenn, K., Duara, R., Levin, B., & Lubs, M. L. (1991). Genetic bases of developmental dyslexia: Molecular studies. In J. E. Obrzut & G. W. Hynd. Neuropsychological foundations of learning disabilities: A handbook of issues, methods, and practice (pp. 49-77). San Diego: Academic Press.

Lund, P. E., Yingling, C. D., Galin, D. Marcus, M., & Simms, H. (1984, February). Validity studies of the Boder Test of Reading-Spelling Patterns. Paper presented at the Twelfth Annual Meeting of the International Neuropsychological Society, Houston, Texas.

Luria, A. R. (1970). Functional organization of the brain. Scientific American, 222, 66-78.

Luria, A. R. (1980). Higher cortical functions in man. New York: Basic Books.

Malatesha, R. N., & Dougan, D. R. (1982). Clinical subtypes of developmental dyslexia: Resolution of an irresolute problem. In R. N. Malatesha & P. G. Aaron (Eds.), Reading disorders: Varieties and treatments (pp. 69-92). New York: Academic Press.

Marshall, J. C., & Newcombe, F. (1973). Patterns of paralexia: A psycholinguistic approach. Journal of Psycholinguistic Research, 2, 175-197.

Marshall, J. C., & Newcombe, F. (1980). The conceptual status of deep dyslexia: A historical perspective. In M. Coltheart, K. Patterson, & J. C. Marshall (Eds.), Deep dyslexia, (pp. 1-21). Boston: Routhledge & Kegan Paul.

Mattis, S., French, J. H., & Rapin, I. (1975). Dyslexia in children and young adults: Three independent neuropsychological syndromes. Developmental Medicine and Child Neurology, 17, 150-163.

McGrath, M. J., & Cohen, D. B. (1978). REM sleep facilitation of adaptive waking behavior: A review of the literature. Psychological Bulletin, 85, 24-57.

McKeever, W. F., & Van Deventer, A. D. (1975). Dyslexic adolescents: Evidence of impaired visual and auditory language processing. Cortex, 11, 361-378.

Menken, G. A. (1981). Comparison of auditory disabled and non-auditory disabled children on a time/speech compression task. Unpublished doctoral dissertation, University of Southern California.

Mercier, L., Pivik, R. T., Busby, K., & Butler, S. (1988). Sleep patterns in reading disabled children: An extension. Sleep Research, 17, 76.

Mercier, L., Pivik, R. T., Busby, K., & Butler, S. (1990). Sleep patterns in reading disabled children: Subtype analyses. Sleep Research, 19, 106.

Meyer, D. E., Schvaneveldt, R. W., & Ruddy, M. G. (1974). Functions of graphemic and phonemic codes in visual word-recognition. Memory and Cognition, 2, 309-321.

Mirmiran, M. (1986). The importance of fetal/neonate REM sleep. European Journal of Obstetrics, Gynecology, and Reproductive Biology, 21, 283-291.

Moldofsky, H., Lue, F. A., Eisen, J., Keystone, E., & Gorczynski, R. M. (1986). The relationship of interleukin-1 and immune function to sleep in humans. Psychosomatic Medicine, 48, 309-318.

Montplaisir, J., Billiard, M., Takahashi, S., Bell, J.

R., Guilleminault, C., & Dement, W. C. (1978). Twenty- four-hour recording in REM-narcoleptics with special reference to nocturnal sleep disruption. Biological Psychiatry, 13, 73-89.

Morruzzi, G. (1966). The functional significance of sleep with particular regard to the brain mechanisms underlying consciousness. In J. C. Eccles (Ed.), Brain and conscious experience (pp. 345-388). New York: Springer.

Moses, J. M., Johnson, L. C., Naitoh, P., & Lubin, A. (1975). Sleep stage deprivation and total sleep loss: Effects on sleep behavior. Psychophysiology, 12, 141-146.

Mosse, H. L., & Daniels, C. R. (1959). Linear dyslexia. American Journal of Psychotherapy, 13, 826-841.

Myklebust, H. (1965). Development and disorders of written language: Picture-story language test. New York: Grune & Stratton.

Nockleby, D. M., & Galbraith, G. G. (1984). Developmental dyslexia subtypes and the Boder Test of Reading-Spelling Patterns. Journal of Psychoeducational Assessment, 2, 91-100.

Nolan, D. R., Hammeke, T. A., & Barkley, R. A. (1983). A comparison of the patterns of the neuropsychological performance in two groups of learning disabled children. Journal of Clinical Child Psychology, 12, 13-21.

Obrzut, J. E. (1988). Deficient lateralization in learning-disabled children: Developmental lag or abnormal cerebral organization? In D. L. Molfese & S. J. Segalowitz (Eds.), Brain lateralization in children: Developmental implications (pp. 567-589). New York: Guilford Press.

Obrzut, J. E., & Hynd, G. W. (1981). Cognitive development and cerebral lateralization in children with learning disabilities. International Journal of Neuroscience, 14, 139-

145.

Obrzut, J. E., Hynd, G. W., Obrzut, A., & Leitgeb, J. L. (1980). Time-sharing and dichotic listening asymmetry in normal and learning-disabled children. Brain and Language, 11, 181-194.

Obrzut, J. E., Hynd, G. W., Obrzut, A., & Pirozzolo, F. J. (1981). Effect of directed attention on cerebral asymmetries in normal and learning disabled children. Developmental Psychology, 17, 118-125.

Obrzut, J. E., Obrzut, A., Bryden, M. P., & Bartels, S. G. (1985). Information processing and speech lateralization in learning disabled children. Brain and Language, 25, 87-101.

Oksenberg, A., Farber, J., Marks, G., Speciale, S., Mihailoff, G., & Roffwarg, H. P. (1985). Testing the role of rapid eye movement (REM) sleep in the development of the CNS. Sleep Research, 14, 76.

Oksenberg, A., Marks, J., Farber, J., Cobbey, K., Speciale, S., Mihailoff, G., & Roffwarg, H. P. (1986). Effect of REM sleep deprivation during the critical period of neuroanatomical development of the cat visual system. Sleep Research, 15, 53.

Ornitz, E. M. (1972).Development of sleep patterns in autistic children. In C. D. Clemente, D. P. Purpura, & F. E. Mayer (Eds.), Sleep and the maturing nervous system (pp. 363-381). New York: Academic Press.

Ornitz, E. M., Ritvo, E. R., Brown, M. B., La Franchi, S., Parmelee, T., & Walter, R. D. (1969). The EEG and rapid eye movements during REM sleep in normal and autistic children. Electroencephalography and Clinical Neurophysiology, 26, 167-175.

Orton, S. T. (1937). Reading, writing and speech problems in children. New York: Academic Press.

Palm, L., Persson, E., Elmquist, D., & Blennow, G. (1989). Sleep and wakefulness in normal

preadolescent children. Sleep, 12, 299-308.

Parmelee, A. H., & Stern, E. (1972). Development of states in infants. In C. D. Clemente, D. P. Purpura, & F. E. Mayer (Eds.), Sleep and the maturing nervous system (pp. 199-228). New York: Raven Press.

Pavlidis, G. Th. (1981). Do eye movements hold the key to dyslexia? Neuropsychologia, 19, 57-63.

Pavlidis, G. Th. (1983). The "dyslexia syndrome" and its objective diagonosis by erractic eye movements. In K. Rayner (Ed.), Eye movements in reading (pp. 441-466). New York: Academic Press.

Pearlman, C. A. (1981). Rat models of the adaptive function of REM sleep. In W. Fishbein (Ed.), Sleep, dreams and memory (pp. 37-45). New York: SP Medical & Scientific Books.

Pennington, B. F., Smith, S. D., Kimberling, W. J., Green, P. A., & Haith, M. M. (1987). Left-handedness and immune disorders in familial dyslexics. Archives of Neurology, 44, 634-639.

Petrauskas, R., & Rourke, B. (1979). Identification of subgroups of retarded readers: A neuropsychological multivariate approach. Journal of Clinical Neuropsychology, 1, 17-37.

Petre-Quadens, O. (1972). Sleep in mental retardation. In C. D. Clemente, D. P. Purpura, & F. E. Mayer (Eds.), Sleep and the maturing nervous system (pp. 383-417). New York: Academic Press.

Petre-Quadens, O., & de Lee, C. (1970). Eye movements during sleep: A common criterion of learning capacities and encocrine activity. Developmental Medicine and Child Neurology, 12, 730-740.

Petre-Quadens, O., de Lee, C., & Remy, M. (1971). Eye movement density during sleep and brain maturation. Brain Research, 26, 49-56.

Pirozzolo, F. J. (1979). The neuropsychology of

developmental reading disorders. New York: Praeger Publishers.

Pirozzolo, F. J. (1983). Eye movements and reading disability. In K. Rayner (Ed.), Eye movements in reading (pp. 499-509). New York: Academic Press.

Pirozzolo, F. J., & Rayner, K. (1979). Cerebral organization and reading disability. Neuropsychologia, 17, 485-491.

Pivik, R. T. (1983). Order and disorder during sleep ontogeny: A selective review. In P. Firestone, P. McGrath, & W. Feldman (Eds.), Advances in behavioral medicine for children and adolescents (pp. 75-102). New York: Lawrence Erlbaum.

Prévost, F., DeKoninck, J., Barry, W., & Broughton, R. (1974). Inversion of the visual field and REM sleep: Experimental reconciliation of the P hypothesis and the SCIP hypothesis. Sleep Research, 3, 89.

Rayner, K. (1978). Eye movements in reading and information processing. Psychological Bulletin, 85, 618-660.

Rechtschaffen, A, & Kales, A. (Eds.). (1968). A Manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. (NIH Publications, No., 204). Washington, DC: Public Health Service, U.S. Govt. Printing Office.

Rechtschaffen, A., & Verdone, P. (1964). Amount of dreaming: Effects of incentive, adaptation to the laboratory and individual differences. Perceptual and Motor Skills, 19, 947-958.

Resnick, L. B. (1979). Theories and prescriptions for early reading instruction. In L. B. Resnick & P. A. Weaver (Eds.), Theory and practice of early reading (Vol. 2, pp. 321-338). Hillsdale N. J.: Erlbaum.

Reynolds, C. R. (1984). Psychometric characteristics of the Boder Test of Reading-Spelling Patterns:

Take one giant step backwards. School Psychology Review, 13, 526-529.

Roffwarg, H. P., Muzio, J. N., & Dement, W. C. (1966). Ontogenetic development of the human sleep-dream cycle. Science, 152, 604-619.

Rosenthal, J., Boder, E. & Calloway, E. (1982). Typology of primary developmental dyslexia: Electrophysiological evidence for its construct validity. In R. N. Malatesha, & P., Aaron (Eds.), Reading disorders: Varieties and treatments (pp. 93-120). New York: Academic Press.

Rourke, B. P. (1985). Overview of learning disability subtypes. In B. P. Rourke (Ed.), Neuropsychology of learning disabilities: Essentials of subtype analysis (pp. 3-14). New York: Guilford Press.

Rourke, B. P. (1987). Syndrome of nonverbal learning disabilities: The final common pathway of white-matter disease/dysfunction. The Clinical Neuropsychologist, 1, 209-234.

Rourke, B. P. (1991). Neuropsychological validation studies in perspective. In B. P. Rourke (Ed.), Neuropsychological validation of learning disability subtypes (pp. 379-385). New York: Guilford Press.

Rourke, B. P., & Finlayson, M. A. J. (1978). Neuropsychological significance of variations in patterns of academic performance: Verbal and visual-spatial abilities. Journal of Abnormal Child Psychology, 6, 121-133.

Rourke, B. P., & Strang, J. D. (1978). Neuropsychological significance of variations in patterns of academic performance: Motor, psychomotor, and tactile-perceptual abilities. Journal of Pediatric Psychology, 3, 62-66.

Rubenstein, H., Lewis, S. S., & Rubenstien, M. A. (1971). Evidence for phonemic recoding in visual word recognition. Journal of Verbal Learning and Verbal Behavior, 10, 645-657.

Rubino, C. A., & Minden, H. A. (1973). An analysis of eye movements in children with a reading disability. Cortex, 9, 217-220.

Satz, P., & Morris, R. (1981). Learning diability subtypes: A review. In F. J. Pirozzolo & M. C. Wittrock (Eds.), Neuropsychological and cognitive processes in reading (pp. 109-141). New York: Academic Press.

Satz, P., Rardin, D., & Ross, J. (1971). An evaluation of a theory of specific developmental dyslexia. Child Development, 42, 2009-2021.

Satz, P., & Sparrow, S. (1970) Specific developmental dyslexia: A theoretical reformulation. In D. J. Bakker, & P. Satz (Eds.), Specific reading disability: Advances in theory and method (pp. 17-40). Rotterdam: University of Rotterdam Press.

Satz, P., & Van Nostrand, G. K. (1972). Developmental dyslexia: An evaluation of a theory. In P. Satz & J. J. Ross (Eds.), The disabled learner (pp. 46-62). Lisse: Swets and Zeitlinger.

Schmitt, H. S., & Kaelbling, R. (1971). The differential laboratory adaptation of sleep parameters. Biological Psychiatry, 3, 33-45.

Sevush, S. (1983, February). The neurolinguistics of reading: Anatomic and neurologic correlates. Paper presented at the Eleventh Annual Conference of the International Neuropsychological Society, Mexico City.

Shaffery, J. P., Marks, G. A., Speciale, S. G., & Roffwarg, H. P. (1991). The ontogenetic hypothesis of REM-sleep, ponto-geniculo-occipital (PGO) waves and developmental plasticity: A preliminary report. Sleep Research, 20A, 216.

Sheldon, S. H., Spire, J. P., & Levy, H. B. (1990). REM sleep eye movements in reading disabled children. Sleep Research, 19, 34.

Sherman, G. D., Rosen, G. F., & Galaburda, A. M. (1989). Animal models of developmental dyslexia: Brain lateralization and cortical pathology. In A. M. Galaburda (Ed.), From reading to neurons (pp. 389-404). Cambridge: MIT Press.

Silver. A. A., & Hagin, R. A. (1966). Maturation of perceptual functions in children with specific reading disability. The Reading Teacher, 19, 253-269.

Silver, A. A., & Hagin, R. A. (1990). Disorders of learning in childhood. New York: John Wiley & Sons.

Small, A., Hibi, S., & Feinberg, I. (1971). Effects of dextroamphetamine sulfate on EEG sleep patterns of hyperactive children. Archives of General Psychiatry, 24, 369-380.

Smith, C. (1985). Sleep states and lerning: A review of the animal literature. Neuroscience & Behavioral Reviews, 9, 157-168.

Smith, C., & Butler, S. (1982). Paradoxical sleep at selective times following training is necessary for learning. Physiology & Behavior, 29, 469-473.

Smith, C., & Kelly, G. (1988). Paradoxical sleep deprivation applied 2 days after the end of training retards learning. Physiology & Behavior, 43, 213-216.

Smith, C. & Lapp, L. (1986). Prolonged increases in paradoxical sleep following acquisition of an avoidance task. Sleep, 3, 67-81.

Smith, C., & Lapp, L. (1991). Increases in number of REMs and REM density in humans following an intensive learning period. Sleep, 14, 325-330.

Smith, C., Young, J., & Young, W. (1980). Prolonged increases in paradoxical sleep during and after avoidance task acquisition. Sleep, 3, 67-81.

Sparrow, S. (1969). Reading disability and

laterality. Proceedings of the 77th Annual Convention of the Americal Psychological Association.

Sparrow, S., & Satz, P. (1970). Dyslexia, laterality and neuropsychological development. In D. J. Bakker & P. Satz (Eds.), Specific reading disability: Advances in theory and method (pp. 41-60). Rotterdam: Rotterdam University Press.

Stahl, M. L., Orr, W. C., & Griffiths, W. J. (1979). Nocturnal levels of growth hormone in hyperactive children of small stature. Journal of Clinical Psychiatry, 40, 225-227.

Tallal, P. (1980). Auditory temporal perception, phonics and reading disabilities in children. Brain and Language, 9, 182-198.

Taylor, H. G., & Fletcher, J. M. (1983). Biological foundations of "specific developmental disorders": Methods, findings and future directions. Journal of Clinical Child Psychology, 12, 46-65.

Taylor, I., & Taylor, M. M. (1983). The psychology of reading. New York: Academic Press.

Tinker, M. A. (1958). Recent studies of eye movements in reading. Psychological Bulletin, 55, 215-231.

Travis, F., Maloney, T., Means, M., March, J. D., & Feinberg, I. (1991). Acute deprivation of the terminal four hours of sleep does not increase delta (0-3 Hz) electroencephalograms: A replication. Sleep, 14, 320-324.

Vellutino, F. R. (1977). Toward an understanding of dyslexia: Psychological factors in specific reading disability. In A. L. Benton & D. Pearl (Eds.). Dyslexia: An appraisal of current knowledge (pp. 61-111). New York: Oxford University Press.

Vellutino, F. R. (1979). Dyslexia: Theory and research. Cambridge, Mass.: MIT Press.

Vogel, G. W. (1975). A review of REM sleep deprivation. Archives of General Psychiatry, 32, 749-761.

Vogel, S. A. (1977) Morphological ability in normal and dyslexic children. Journal of Learning Disabilities, 12(1), 41-49.

Webb, W. B., & Agnew, H. W. (1971). Stage 4 sleep: Influence of time course variables. Science, 174, 1354-1356.

Wiener, M., & Cromer, W. (1967). Reading and reading difficulty: A conceptual analysis. Harvard Educational Review, 37, 620-643.

Whiting, S., & Jarrico, S. (1980). Spelling patterns of normal readers. Journal of Learning Disabilities, 13, 40-42.

Williams, R. L., Karacan, I., & Hursch, C. J. (1974). Electroencephalography (EEG) of human sleep: Clinical applications. New York: Wiley.

Witelson, S. F., & Rabinovitch, M. S. (1972). Hemispheric speech lateralization in children with auditory-linguistic deficits. Cortex, 8, 412-426.

Yeni-Komshian, G. H., Isenberg, D., & Goldberg, H. (1975). Cerebral dominance and reading disability: Left visual field deficit in poor readers. Neuropsychologia, 13, 83-94.

Zaidel, E. (1985) Introduction. In D. F. Benson & E. Zaidel (Eds.), The dual brain: Hemispheric specialisation in humans (pp. 47-63). New York: The Guilford Press.

Zarcone, V. P., Benson, K. L., & Berger, P. A. (1987). Abnormal rapid eye movement latencies in schizophrenia. Archives of General Psychiatry, 44, 45-48.

Zimmerman, J., Stoyva, J., & Metcalf, D. (1970). Distorted visual feedback and augmented REM sleep. Psychophysiology, 7, 298.

Zimmerman, J., Stoyva, J., & Reite, M. (1978). Spatially rearranged vision and REM sleep: A lack of effect. Biological Psychiatry, 13, 301-316.

Zurif, E. B., & Carson, G. (1970). Dyslexia in relation to cerebral dominance and temporal analysis. Neuropsychologia, 8, 351-361.