The Science of Reading and Dyslexia

Sally E. Shaywitz, MD, and Bennett A. Shaywitz, MD

It is a great honor to present this lecture, which honors the extraordinary contributions to scholarship and teaching of Leonard Apt, MD a true renaissance man. For more than four decades, Leonard Apt has contributed both singular discoveries and a continuous stream of research findings that have significantly advanced our knowledge in the field of pediatrics and pediatric ophthalmology. In the words of Leonard Apt, "In academic medicine, I have been free to question and investigate new theories. Years from now, I'd like to be remembered as a doctor who never stopped asking 'Why?'"

Dyslexia is characterized by an unexpected difficulty in reading in children and adults who otherwise possess the intelligence, motivation, and schooling considered necessary for accurate and fluent reading.[2] Historically, dyslexia in adults was first noted in the latter half of the 19th century, and developmental dyslexia in children was first reported in 1896. Patients with dyslexia were frequently seen by ophthalmologists, who called the disorder "word blindness." James Hinshelwood, a Scottish ophthalmologist (Figure 1), who elaborated the disorder in children, noted that these children were often exceptionally smart except for their inability to read and proposed special education programs as treatment.[5]

Recent epidemiological data indicate that, like hypertension and obesity, dyslexia fits a dimensional model. In other words, within the population, reading ability and reading disability occur along a continuum, with reading disability representing the lower tail of a normal distribution of reading ability.[6,7] Dyslexia is perhaps the most common neurobehavioral disorder affecting children, with prevalence rates ranging from 5% to 10% in clinic- and school-identified samples to 17.5% in unselected population-based samples.[2] Previously, it was believed that dyslexia affected boys primarily[8]; however, more recent data indicate there is no sex predilection for dyslexia.[9-11] Longitudinal studies, both prospective[12,13] and retrospective,[14-16] indicate that dyslexia is a persistent, chronic condition; it does not represent a transient "developmental lag" (Figure 2). Over time, poor readers and good readers tend to maintain their relative positions along the spectrum of reading ability.[12]

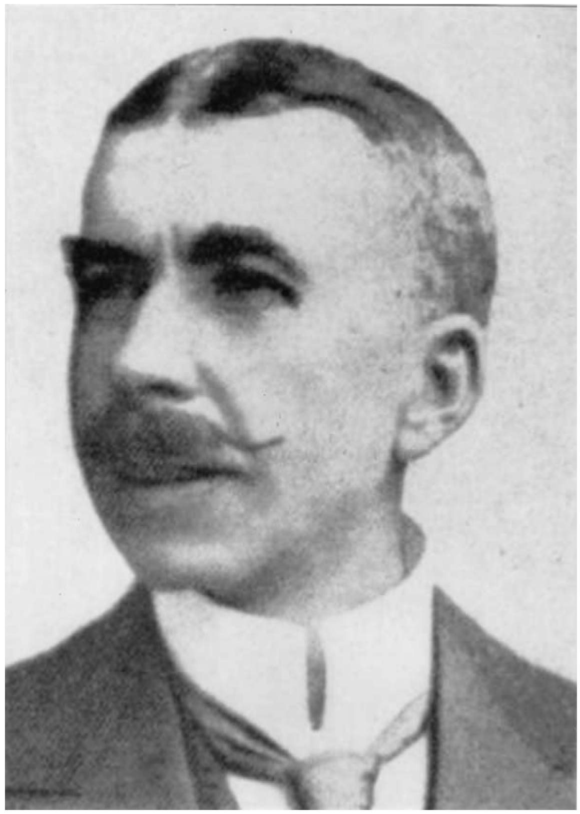

FIG 1. James Hinshelwood, a pioneering Scottish ophthalmologist who was the one of the first physicians to describe the clinical picture of dyslexia as well as to promise a coherent plan of management.

Dyslexia is both familial and heritable.[17] Family history is one of the most important risk factors, with 23% to 65% of children who have a parent with dyslexia reported to also have the disorder.[18] The prevalence rate among siblings of affected persons is approximately 40%, and among parents of affected individuals ranges from 27% to 49%.[17] This provides opportunities for early identification of affected siblings and often for delayed but helpful identification of affected adults. Linkage studies implicate loci on chromosomes 6 and 15,[19,21] chromosome 1, and—most recently—on chromosome 2[23] for the transmission of phonological awareness deficits and subsequent reading problems. Whether the differences in the genetic loci represent polygenic inheritance, different cognitive paths to the same phenotype, or different types of dyslexia is not clear.

From the NICHD–Yale Center for the Study of Learning and Attention, New Haven, CT.
Portions of this article were previously published in, and are similar to, other reviews by the authors.[1-4]
Submitted March 11, 2002.
Revisions accepted December 16, 2002.
Reprint requests: Sally E. Shaywitz, MD, Department of Pediatrics, Yale University School of Medicine, PO Box 333, New Haven, CT 06510-8064.

1091-8531/2003/$35.00 + 0
doi:10.1016/S1091-8531(03)00002-8

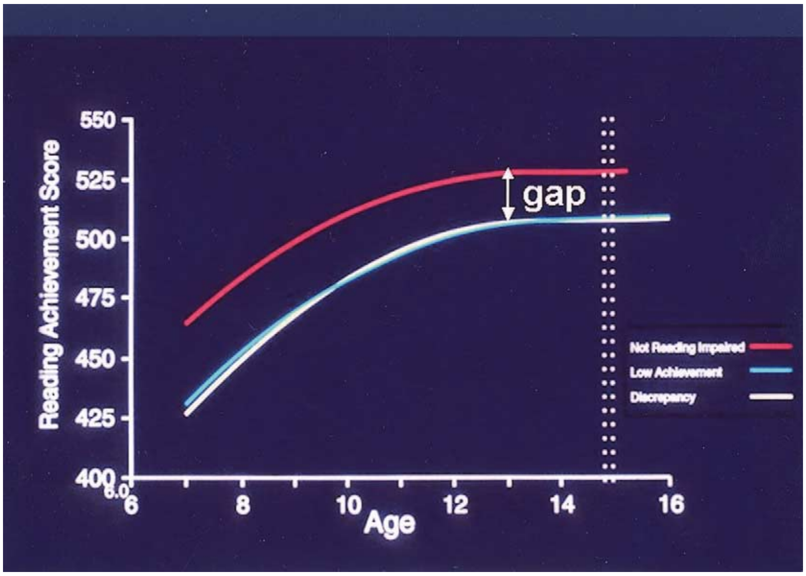

FIG 2. Trajectory of reading skills over time in nonimpaired and dyslexic readers. Dyslexic readers are defined both by a discrepancy between ability and achievement (discrepancy) and low achievement. The y-axis shows Rasch scores (W scores) from the Woodcock-Johnson reading test,[70] and the x-axis shows age in years. Reading scores of both dyslexic and nonimpaired readers improve with age, but the gap remains. Thus, dyslexia is a deficit and not a developmental lag. (Adapted and reprinted with permission.)

PATHOPHYSIOLOGY

Cognitive Influences

The Phonological Deficit Hypothesis. There is now a strong consensus among investigators in the field that the central difficulty in dyslexia reflects a deficit within the language system, although other systems and processes may also contribute to the difficulty. The language system is conceptualized as a hierarchical series of components. At higher levels are neural systems engaged in processing, eg, semantics, syntax and discourse; at the lowest level is the phonological module dedicated to processing the distinctive sound elements that constitute language. The functional unit of the phonological module is the phoneme, defined as the smallest discernible segment of speech; for example, the word "bat" consists of three phonemes: /b/ /ae/ /t/ (buh, aah, tuh). To speak a word, the speaker retrieves the word's phonemic constituents from his internal lexicon, assembles the phonemes, and then utters the word. Conversely, to read a word, the reader must first segment that word into its underlying phonological elements. The awareness that all words can be broken down into these basic elements of language (phonemes) allows the reader to decipher the reading code. To read, a child must develop the insight that spoken words can be pulled apart into phonemes and that letters in a written word represent these sounds. Results from large and well-studied populations with reading disability confirm that in young school-age children,[24,25] as well as in adolescents,[26] a deficit in phonology represents the most robust and specific[28] correlate of reading disability. Such findings form the basis for the most successful and evidence-based interventions designed to improve reading.[28]

Implications of the Phonological Model of Dyslexia. Basically, reading comprises two main processes—decoding and comprehension. In dyslexia, a deficit at the level of the phonological module impairs the ability to segment the written word into its underlying phonological elements. As a result, the reader experiences difficulty, first in decoding the word and then in identifying it. The phonological deficit is domain-specific, ie, it is independent of other, nonphonological linguistic abilities. In particular, the higher-order cognitive and linguistic functions involved in comprehension—such as general intelligence and reasoning, vocabulary, and syntax—are generally intact. This pattern, a deficit in phonological analysis contrasted with intact higher-order cognitive abilities, offers an explanation for the paradox of otherwise intelligent people who experience great difficulty in reading.

According to the model, a circumscribed deficit in a lower-order linguistic (phonological) function blocks access to higher-order processes as well as the ability to draw meaning from text. The problem is that the affected reader cannot use his or her higher-order linguistic skills to access the meaning until the printed word has first been decoded and identified. For example, an individual who knows the precise meaning of the spoken word, "apparition," will not be able to use her knowledge of the meaning of the word until she can decode and identify the printed word on the page and will appear not to know the word's meaning.

The Phonological Deficit in Adolescence and Adult Life. Deficits in phonological coding continue to characterize dyslexic readers even in adolescence; performance on phonological processing measures contributes most to discriminating between dyslexic and average readers as well as between average and superior readers.[26] Children with dyslexia neither spontaneously remit nor demonstrate a lag mechanism for "catching up" in the development of reading skills. That is not to say that many dyslexic readers do not become quite proficient in reading a finite domain of words that are in their area of special interest, eg, words that are important for their careers. For example, an individual who is dyslexic in childhood but who in adult life becomes interested in molecular biology, will learn to decode words that form a minivocabulary important in molecular biology. Such an individual, although able to decode words in this domain, still exhibits evidence of early reading problems when having to read unfamiliar words, which may be done accurately but not fluently and automatically.[16,26,29-33] In adolescents, the rate of reading as well as facility with spelling may be most useful clinically in differentiating average from poor readers. From a clinical perspective, these data indicate that as children approach adolescence, a manifestation of dyslexia may be a slow reading rate; in fact, children may learn to read words accurately, but they will not be fluent or automatic, reflecting the lingering effects of a phonological deficit.[33] Because they are able to read words accurately, albeit very slowly, dyslexic adolescents and young adults may mistakenly be assumed to have "outgrown" their dyslexia

Data from studies of children with dyslexia who were followed up prospectively support the notion that in adolescents, the rate of reading as well as facility with spelling may be most useful clinically in differentiating good from poor readers in students in secondary school, college, and even graduate school. It is important to remember that these older dyslexic students may be similar to their unimpaired peers on untimed measures of word recognition yet continue to suffer from the phonological deficit that makes reading less automatic, more effortful, and slower. For these readers with dyslexia, the provision of extra time is an essential accommodation; it allows them the time to decode each word and to apply their unimpaired higher-order cognitive and linguistic skills to the surrounding context to get at the meaning of words that they cannot entirely or rapidly decode. More will be said about this below in the section on management.

Neurobiological Influences

To a large degree, these advances in understanding dyslexia have informed and facilitated studies examining the neurobiological underpinnings of reading and dyslexia. Historically, as early as 1891, the French neurologist Dejerine[34] suggested that a portion of the left posterior brain region is critical for reading. Beginning with Dejerine, a large body of literature on acquired inability to read (alexia) described neuroanatomic lesions most prominently centered in the parietotemporal area (including the angular gyrus, supramarginal gyrus, and posterior portions of the superior temporal gyrus), a region considered pivotal in mapping the visual percept of the print onto the phonological structures of the language system.[35-37] Another posterior brain region, this more ventral in the occipitotemporal area, was also described by Dejerine[38] as critical to reading ability. More recently, a range of neurobiological investigations using postmortem brain specimens,[39] brain morphometry,[40] and diffusion tensor magnetic resonance imaging (MRI)[41] supports the belief that there are differences in the temporo-parieto-occipital brain regions between dyslexic and nonimpaired readers.

FUNCTIONAL BRAIN IMAGING

Rather than being limited to examining the brain in an autopsy specimen or measuring the size of brain regions using static morphometric indices, functional imaging offers the possibility of examining brain function during performance of a cognitive task. In principle, functional brain imaging is quite simple. When an individual is asked to perform a discrete cognitive task, that task places processing demands on particular neural systems in the brain. Meeting those demands requires activation of neural systems in specific brain regions, and those changes in neural activity are in turn reflected by changes in brain metabolic activity, which in turn are reflected, for example, by changes in cerebral blood flow and in the cerebral utilization of metabolic substrates such as glucose. Functional MRImaging (fMRI) promises to supplant other methods (eg, positron emission tomography) because of its ability to map the individual brain's response to specific cognitive stimuli. Because fMRI is noninvasive and safe, it can be used repeatedly, which makes it ideal for studying humans, especially children.

Converging evidence using functional brain imaging in adult dyslexic readers also shows a failure of left hemisphere posterior brain systems to function properly during reading[42-52] as well as during nonreading visual processing tasks[53,54] (Figure 3). In addition, some functional brain imaging studies show differences in brain activation in frontal regions in dyslexic compared with nonimpaired readers; in some studies dyslexic readers' brains are more active in frontal regions,[42,47,48] and in other studies nonimpaired readers' brains are more active in frontal regions.[55-58]

Logan[59,60] proposed two systems critical to the development of skilled, automatic processing. One involves word analysis and operates on individual units of words such as phonemes, requires attentional resources, and processes relatively slowly. The other system operates on the whole word (word form), is an obligatory system that does not require attention, and processes very rapidly. Converging evidence from a number of lines of investigation indicates that Logan's word analysis system is localized within the parietotemporal region, whereas the automatic, rapidly responding system is localized within the occipitotemporal area and functions as a visual word form area.[61-65] The visual word form area appears to respond preferentially to rapidly presented stimuli[66] and is engaged

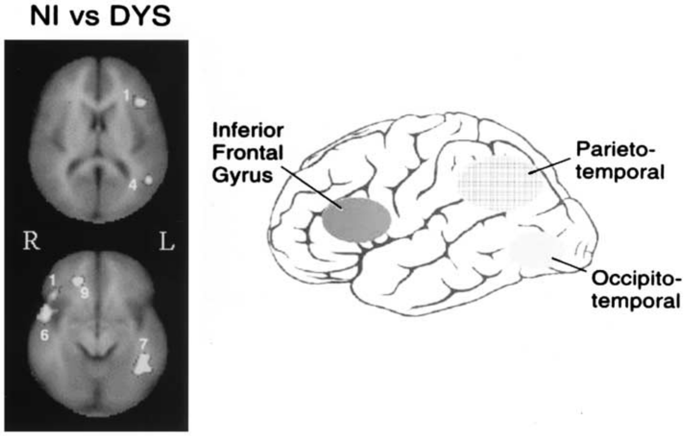

FIG 3. Disruption in reading systems in dyslexic readers. Axial images from superior and inferior axial slices showing the reading systems. Red and yellow colors show regions where nonimpaired (NI) readers are more active than are dyslexic readers (DYS). These regions involve left hemisphere sites in the inferior frontal gyrus,[1] the parieto-temporal region,[4] and the occipitotemporal region.[7] Right-hemisphere sites are also more active in NI readers, involving the inferior frontal gyrus,[1] the parietotemporal region,[6] and the middle frontal gyrus.[9] (Data from Shaywitz et al.[49])

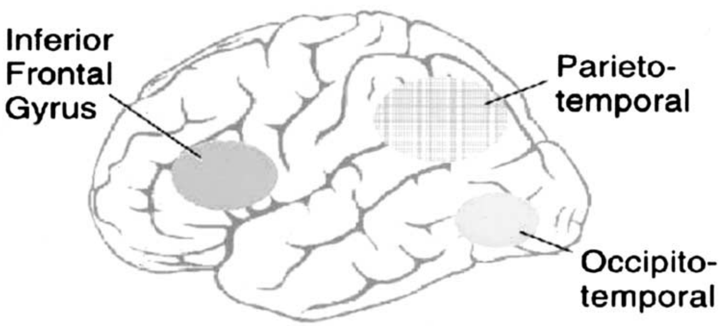

FIG 4. Neural systems used for reading. Converging evidence indicates three important systems in reading, all primarily in the left hemisphere. These include an anterior system and two posterior systems: (1) anterior system in the left inferior frontal region; (2) dorsal parietotemporal system involving angular gyrus, supramarginal gyrus, and posterior portions of the superior temporal gyrus; and (3) ventral occipitotemporal system involving portions of the middle temporal gyrus and middle occipital gyrus.

even when the word has not been consciously perceived.[63] It is this occipitotemporal system that appears to predominate when a reader has become skilled and has bound together as a unit the orthographic, phonological, and semantic features of the word (Figure 4).

Recognition of these systems allows us to suggest an explanation for the brain activation patterns observed in dyslexic children. We suppose that rather than the smoothly functioning and integrated reading systems observed in nonimpaired children, disruption of the posterior reading systems results in dyslexic children attempting to compensate by shifting to other, ancillary systems, eg, anterior sites such as the inferior frontal gyrus and right hemisphere sites. The anterior sites, which are critical in articulation,[42,67,68] may help the child with dyslexia develop an awareness of the sound structure of the word by subvocalizing, ie, by forming the word with the lips, tongue, and vocal apparatus, thus allowing the child to read, albeit more slowly and less efficiently than if the fast occipitotemporal word identification system were functioning. The right hemisphere sites may represent the engagement of brain regions that allow the poor reader to use other perceptual processes to compensate for poor phonological skills. A number of studies of young adults with childhood histories of dyslexia indicate that although they may develop some accuracy in reading words, they remain slow, nonautomatic readers.[15,16]

DIAGNOSIS

The diagnosis of dyslexia is basically no different than that for any other medical disorder. Guided by knowledge of the presumed underlying pathophysiology, the clinician seeks to determine through history, observation, and psychometric assessment, if there are (1) unexpected difficul-

TABLE 1. Clues To Dyslexia In School-Age Children*

History
Delayed language
Problems with the sounds of words (trouble rhyming words, confusing words that sound alike)
Expressive language difficulties (mispronunciations, hesitations, word-finding difficulties)
Difficulty naming (difficulty learning letters of alphabet and names of numbers)
Difficulty learning to associate sounds with letters
History of reading and spelling difficulties in parents and siblings
Reading
Difficulty decoding single words
Particular difficulty reading nonsense or unfamiliar words
Inaccurate and labored oral reading
Slow reading
Comprehension often superior to isolated decoding skills
Poor spelling
Language
Relatively poor performance on tests of word retrieval (name the pictured item)
Relatively superior performance on tests of word recognition (point to the pictured item)
Poor performance on tests of phonological awareness
Clues most specific to young children at-risk for dyslexia
Difficulty on tests assessing: knowledge of the names of letters, the ability to associate sounds with letters, and phonological awareness
Clues most specific to bright young adults with dyslexia
Childhood history of reading and spelling difficulties
Accurate but not automatic reading
Very slow performance on timed reading tests (eg, Nelson-Denny Reading Test)
Penalized by multiple choice tests

*Clues are based on history, observations, testing, or a combination of all three.
Reprinted with permission.

ties (for age, intelligence, or level of education) in reading and (2) associated linguistic problems at the level of phonological processing. There is no one single test score that is pathognomonic of dyslexia. As with any other medical diagnosis, the diagnosis of dyslexia should reflect a thoughtful synthesis of all the clinical data available. What the clinician is seeking is converging evidence of a phonologically based reading disability as indicated by a disparity between the individual's reading and phonological skills in contrast to his or her intellectual capabilities, age, or level of education. Dyslexia is distinguished from other disorders that may prominently feature reading difficulties by the unique, circumscribed nature of the phonological deficit, one not intruding into other linguistic or cognitive domains. How reading and language are assessed will reflect the age and educational level of the patient (Tables 1 and 2).

Diagnosis at School Age

Presenting complaints most commonly center about school performance, eg, the child is not doing well in school, and often parents and teachers do not appreciate that the child's poor academic performance stems from a

TABLE 2. Types of Tests Useful in Identifying Children At Risk for Dyslexia at School Entry

Letter identification (naming letters of the alphabet)
Letter—sound association (eg, identifying words beginning with the same letter from a list: doll, dog, boat)
Phonological awareness (eg, identifying word that would remain if a particular sound were removed: if the /k/ sound was taken away from "cat")
Verbal memory (eg, recalling a sentence or a story that was just told)
Rapid naming (rapidly naming a continuous series of familiar objects, digits, letters, or colors)
Expressive vocabulary or word retrieval (eg, naming single-pictured objects

Reprinted with permission.

reading difficulty. Thus, an evaluation for dyslexia should be considered in all children presenting with school difficulties, even if reading difficulty is not the chief complaint. As with most other medical disorders, the history is critical to the diagnosis of dyslexia. Clinicians need to develop a sense of the developmental pattern demonstrated by children with dyslexia. Overall, the ontogeny of dyslexia is that of a child who may have had a delay in speaking, who did not learn letters by kindergarten, and who did not begin to learn to read by first grade. The child progressively falls behind, with teachers and parents puzzled as to why such an intelligent child may have difficulty learning to read. The reading difficulty is unexpected with respect to the child's ability, age, and/or grade. Even after acquiring decoding skills, the child generally remains a slow reader. When teachers are not informed, they may unnecessarily pressure or hurry the student. Dysgraphia is often present and accompanied by laborious note taking. Self-esteem is frequently affected, particularly if the disorder has gone undetected for a long period of time. Learning-disabled children are likely to have encountered negative test-taking experiences where there was a disparity between their knowledge and their test scores, especially on timed tests, and thus tend to exhibit more test anxiety than nondisabled peers. Test scores may thus be artificially depressed as a result of such anxiety. Adults with strong histories of dyslexia who have compensated for their reading disability demonstrate good accuracy in reading but are less automatic. Compensated dyslexics take longer to apply their decoding skills and thus are slower readers; however, given sufficient time, they score very well on tests of reading comprehension.

Assessment of Reading

Reading is assessed by measuring decoding and comprehension. In the school-age child, one important element of the psychometric evaluation is how accurately the child can decode words, ie, read single words in isolation. This is measured with standardized tests of single real-word and pseudoword reading such as the Woodcock-Johnson PsychoEducational Battery–Revised (WJ-R) and the Woodcock Reading Mastery Test. Difficulties often also emerge on tests of spelling, which depend on these same abilities. Reading fluency should be assessed by reading aloud using the Gray Oral Reading Test. This test consists of 13 increasingly difficult passages, each followed by 5 comprehension questions. Word-reading efficiency may be assessed using the Test of Word Reading Efficiency, a test of speeded reading of single words. This test measures how many words, from a list that gradually increases in difficulty, the child can read in 45 seconds.

Diagnosis in Preschool and at School Entry

Currently, reading disabilities are not diagnosed until children are in third grade or approximately 9 years old. Good evidence indicates that it is possible to screen children as young as 4 to 5 years old to identify those at risk for reading disability, an identification based on poor reading relative to chronological age, ie, poor reading defined solely on the basis of low reading achievement. A history of language delay or of not attending to the sounds of words (trouble playing rhyming games with words, confusing words that sound alike), along with a positive family history, represent significant risk factors for dyslexia. The most helpful measures in predicting reading difficulties are those designed to assess, in general, phonemic awareness and phonological skills. Normed tests of phonological analysis for young children—for example, the Comprehensive Test of Phonological Processing—are now available. It consists of measures of phonological awareness, phonological coding and working memory, and rapid naming and has a national standardization on children ranging in age from 5 years to adulthood. For example, measures most predictive of later reading ability include the child's knowledge of letter sounds, the ability to blend sounds into words (done orally), and at the end of kindergarten, the ability to name letters rapidly. There is growing evidence that early identification and intervention in kindergarten and first grade 1 may substantially decrease the number of children requiring special services for reading disability (reviewed in[69]). These early identification procedures are sensitive but not specific and so tend to overidentify children with dyslexia. Such overidentification is reasonable given that the costs of delaying intervention are so great.

Diagnosis in Adolescents and Young Adults

The developmental course of dyslexia has now been characterized. First, dyslexia is persistent and does not go away; on a practical level, this means that once a person is diagnosed as having dyslexia, there is no need for reexamination after high school to confirm the diagnosis. Second, over the course of development, skilled readers become more accurate and more automatic in decoding; they do not need to rely on context for word identification. Dyslexic readers, too, become more accurate over time, but they do not become automatic in their reading. Residua of the phonological deficit persist so that reading remains effortful and slow, even for the brightest of individuals with childhood histories of dyslexia. Failure to either recognize or measure the lack of automaticity in reading represents, perhaps, the most common error in the diagnosis of dyslexia in accomplished young adults. It is often not appreciated that tests measuring word accuracy are inadequate for diagnosing dyslexia in young adults at the college, graduate, or professional school levels. For these individuals, timed measures of reading must be employed in making the diagnosis. However, few standardized tests for adult readers are administered under timed and untimed conditions; the Nelson–Denny Reading Test represents an exception. Reading measures commonly used for school-age children may provide misleading data in some adolescents and young adults because they assess reading accuracy but not automaticity (speed). In bright, young adults, the sine qua non of a diagnosis of dyslexia are (1) a history of phonologically based reading difficulties, (2) requirements for extra time on tests and for current slow and effortful reading, and (3) signs indicating lack of automaticity in reading. At all ages, and especially in young adults, dyslexia is a clinical diagnosis.

MANAGEMENT

Because physicians are frequently asked about various reading programs for dyslexia, they should understand the principal elements of an effective training program. These elements reflect an understanding of the reading process and why it is so difficult for children and adults with dyslexia to learn to read. The management of dyslexia demands a life-span perspective. Early on, the focus is on remediation of the reading problem. As a child matures and enters the more time-demanding setting of secondary school, the emphasis shifts to the important role of providing accommodations. The goal of effective intervention programs is to remediate the underlying problem in phonemic awareness; however, all too frequently the standard instruction provided through remediation is too little, too general, and too unsystematic. Most recently, based on the work of the National Reading Panel,[28] evidence-based reading intervention programs have been identified that provide instruction in the most important elements in reading: phonemic awareness, phonics, reading fluency, vocabulary, and reading comprehension strategies. To identify these programs, the National Reading Panel used the same methodology that has been recognized as the scientific standard and that has been used so successfully in providing evidence-based treatments for many other disorders affecting children. Taking each component of the reading process in turn, the intervention used with younger children and even with older children are programs to improve phonemic awareness, ie, the ability to focus on and manipulate phonemes (speech sounds) in spoken syllables and words. The elements found to be most effective in enhancing phonemic awareness, reading, and spelling skills include (1) teaching children to manipulate phonemes with letters; (2) focusing the instruction on 1 or 2 types of phoneme manipulations rather than multiple types; (3) teaching children in small groups; and (4) providing explicit instruction that directly teaches children how to identify, count, and manipulate the sounds in spoken words. The next step in teaching reading is to teach phonics, ie, to make sure that the beginning reader understands how letters are linked to sounds (phonemes) to form letter–sound correspondences and spelling patterns. In teaching phonics, it is critical to ensure that the instruction is explicit and systematic; phonics instruction enhances children's success in learning to read, and systematic phonics instruction is more effective than instruction that teaches little or no phonics. Furthermore, the effects of phonics instruction are substantial in kindergarten and first grade, indicating that systematic phonics programs should be implemented in these early grades.[28] The evidence indicates that kindergarten-age children who receive phonics instruction benefit in their ability to read and spell words, and first graders who are taught phonics are better able to decode and spell and show significant improvement in their ability to comprehend text. In contrast, older children in later primary grades receiving phonics instruction are better able to decode and spell words and to read text orally, but their comprehension of text is not significantly improved.

Fluency refers to the ability to read aloud with speed, accuracy, and proper expression. Although it is generally recognized that fluency is an important component of skilled reading, it is often neglected in the classroom. The most effective method to build reading fluency is guided oral reading, ie, reading aloud repeatedly to a teacher, an adult, or a peer, and then receiving feedback.[28] The evidence indicates that guided oral reading has a clear and positive impact on word recognition, fluency, and comprehension at a variety of grade levels and applies to all students, good readers as well as those experiencing reading difficulties. The evidence is less secure is for programs for struggling readers that encourage large amounts of independent reading, ie, silent reading without any feedback to the student. Thus, although independent silent reading is intuitively appealing, at this time the evidence does not support the notion that reading fluency improves. No doubt there is a correlation between being a good reader and reading large amounts; however, there is a paucity of evidence indicating that there is a *causal relationship*, ie, if poor readers read more they will become more fluent.

Fluency is of critical importance because text reading that is dysfluent is slow and may impair the child's ability to comprehend what has been read and, clearly, comprehending the text is the ultimate goal of reading. In contrast to teaching phonemic awareness, phonics, and fluency, interventions for reading comprehension are not as well established. In large measure, this reflects the nature of the complex processes influencing reading comprehension. Limited evidence indicates that the most effective methods to teach reading comprehension involve teaching such components as vocabulary as well as active interaction between reader and text, an interaction fostered by teachers who have the knowledge and skills to apply strategies designed to engage the student with the material.

One of the most exciting developments in reading and reading disability is the converging evidence that, in many cases, and if recognized very early (at ages 4 and 5 years), reading difficulties may be prevented. As early as kindergarten, and perhaps even in preschool, it is now possible to identify children at risk for word-reading difficulties on the basis of their performance on tasks that assess phonemic awareness and naming abilities (see above).[69] Even with the early identification and interventions designed to prevent reading disability, there may be a substantial number of children who will need the interventions discussed above.

Large-scale studies to date have focused on younger children; as yet, there are few or no data available on the effect of these training programs on older children. The management of dyslexia in students in secondary school, and especially college and graduate school, is based on accommodation rather than remediation. College students with a history of childhood dyslexia often present a paradoxical picture; they are similar to their unimpaired peers on measures of word recognition and reading comprehension, yet they continue to suffer from the phonological deficit that makes reading less automatic, more effortful, and slower. For young adults with dyslexia, the provision of extra time is an essential accommodation; it allows them the time to decode each word and to apply their unimpaired higher-order cognitive and linguistic skills to the surrounding context to get at the meaning of words that they cannot entirely or rapidly decode. Studies comparing performance of reading-disabled students with and without extra time show the latent potential of such students that never emerges unless they are provided with extra time to compensate for their lack of automaticity. In contrast, additional time makes little to no difference in the performance of nonimpaired readers. Although providing extra time for reading is by far the most common accommodation for people with dyslexia, other helpful accommodations include allowing the use of lap-top computers with spell-check programs, tape recorders, and recorded books (materials are available from Recording for the Blind and Dyslexic; 800-221-4792) in the classroom as well as providing access to syllabi and lecture notes; tutors to "talk through" and review the content of reading material; alternatives to multiple- choice tests (eg, reports or orally administered tests), and a separate, quiet room for taking tests. With such accommodations, many students with dyslexia are now successfully completing studies in a range of disciplines, including medicine.

People with dyslexia and their families frequently consult their physicians about unconventional approaches to the remediation of reading difficulties; in general, no credible data exist to support the claims made for these treatments (eg, optometric visual training, medication for vestibular dysfunction, chiropractic manipulation, and dietary supplementation).

In summary, there is now convincing scientific evidence to explain why some very smart people have trouble learning to read as well as to govern the management of children and adults who are dyslexic. Longitudinal studies, cognitive studies, and now neurobiological studies provide powerful proof of the enduring nature of the phonological deficit in even the brightest of dyslexics, including those who are accurate but not automatic readers. Key factors to keep in mind are (1) that dyslexia is a clinical diagnosis; (2) that in bright young adults, a family history of dyslexia and slow reading are the sine qua non of the diagnosis; and (3) that accommodations are as essential to these individuals as insulin is to a diabetic. There are now at least two Nobel laureates (Niels Bohr and Baruch Benacerref), as well as numerous other distinguished physicians and scientists, who are dyslexic. No doubt, there could be many more if otherwise bright and able dyslexic men and women were provided with the accommodations necessary to access their strengths on tests that serve as gate-keepers. It is the obligation of each physician to ensure that the diagnosis and management of children and adults with dyslexia are based on science, not on arbitrary and capricious dogma.

References

1. Shaywitz S, Shaywitz B. Dyslexia. In: Swaiman K, Ashwal S, editors 3rd ed. Vol. 1. St. Louis (MO): Mosby; 1999. Pediatric neurology: principles and practice. p. 576-84.
2. Shaywitz S. Current concepts: dyslexia. N Engl J Med 1998;338(5): 307-12.
3. Shaywitz B, Pugh KR, Jenner A, et al. The neurobiology of reading and reading disability (dyslexia). In: Kamil M, Mosenthal P, Pearson P, Barr R, editors. Handbook of reading research Vol 3. Mahwah (NJ): Lawrence Erlbaum Associates; 2000. p. 229-49.
4. Shaywitz S, Shaywitz B, Pugh K, et al. The neuropsychology of dyslexia. In: Segalowitz S, Rapin I editors. Handbook of neuropsycholology. 2nd ed. Vol 7. Amsterdam: Elsevier. 2003.
5. Hinshelwood J. The treatment of word-blindness acquired and congenital. Br Med J 1912;2:1033-5.
6. Shaywitz SE, Escobar MD, Shaywitz BA, Fletcher JM, Makuch R. Evidence that dyslexia may represent the lower tail of a normal distribution of reading ability. N Engl J Med 1992;326(3):145-50.
7. Gilger JW, Borecki IB, Smith SD, DeFries JC, Pennington BF. The etiology of extreme scores for complex phenotypes: an illustration using reading performance. In: Chase CH, Rosen GD, Sherman GF, editors. Developmental dyslexia: neural, cognitive, and genetic mechanisms. Baltimore (MD): York Press; 1996. p. 63-85.
8. Finucci JM, Childs B. Are there really more dyslexic boys than girls?. In: Ansara A, Geschwind N, Albert M, Gartrell N, editors. Sex differences in dyslexia. Towson (MD): Orton Dyslexia Society; 1981. p. 1-9.
9. Flynn J, Rahbar M. Prevalence of reading failure in boys compared with girls. Psychol Sch 1994;31:66-71.
10. Shaywitz SE, Shaywitz BA, Fletcher JM, Escobar MD. Prevalence of reading disability in boys and girls: results of the Connecticut Longitudinal Study. J Am Med Assoc 1990;264(8):998-1002.
11. Wadsworth SJ, DeFries JC, Stevenson J, Gilger JW, Pennington BF. Gender ratios among reading-disabled children and their siblings as a function of parental impairment. J Child Psychol Psychiatr 1992; 33(7):1229-39.
12. Shaywitz BA, Holford TR, Holahan JM, et al. A Matthew effect for IQ but not for reading: Results from a longitudinal study. Read Res Q 1995;30(4):894-906.
13. Francis DJ, Shaywitz SE, Stuebing KK, Shaywitz BA, Fletcher JM. Developmental lag versus deficit models of reading disability: a longitudinal, individual growth curves analysis. J Educ Psychol 1996; 88(1):3-17.
14. Scarborough HS. Continuity between childhood dyslexia and adult reading. Br J Psychol 1984;75:329-48.
15. Felton RH, Naylor CE, Wood FB. Neuropsychological profile of adult dyslexics. Brain Lang 1990;39:485-97.
16. Bruck M. Persistence of dyslexics' phonological awareness deficits. Dev Psychol 1992;28(5):874-86.
17. Pennington BF, Gilger JW. How is dyslexia transmitted? In: Chase CH, Rosen GD, Sherman GF, editors. Developmental dyslexia: neural, cognitive, and genetic mechanisms. Baltimore (MD): York Press; 1996. p. 41-61.
18. Scarborough HS. Very early language deficits in dyslexic children. Child Dev 1990;61:1728-43.
19. Cardon LR, Smith SD, Fulker DW, Kimberling WJ, Pennington BF, DeFries JC. Quantitative trait locus for reading disability on chromosome 6. Science 1994;266:276-9.
20. Cardon LR, Smith SD, Fulker DW, Kimberling WJ, Pennington BF, DeFries JC. Quantitative trait locus for reading disability: Correction. Science 1995;268:1553.
21. Grigorenko EL, Wood FB, Meyer MS, et al. Susceptibility loci for distinct components of developmental dyslexia on chromosomes 6 and 15. Am J Hum Genet 1997;60:27-39.
22. Rabin M, Wen X, Hepburn M, Lubs H, Feldman E, Duara R. Suggestive linkage of developmental dyslexia to chromosome 1p34-p36. Lancet 1993;342:178.
23. Fagerheim T, Raeymaekers P, Tonnessen F, Pedersen D, Tranebjaerg L, Lubs H. A new gene (DYX3) for dyslexia is located on chromosome 2. J Med Genet 1999;36:664-9.
24. Fletcher JM, Shaywitz SE, Shankweiler DP, et al. Cognitive profiles of reading disability: comparisons of discrepancy and low achievement definitions. J Educ Psychol 1994;86(1):6-23.
25. Stanovich KE, Siegel LS. Phenotypic performance profile of children with reading disabilities: A regression-based test of the phonological-core variable-difference model. J Educ Psychol 1994;86(1): 24-53.
26. Shaywitz SE, Fletcher JM, Holahan JM, et al. Persistence of dyslexia: the Connecticut Longitudinal Study at adolescence. Pediatrics 1999; 104:1351-9.
27. Morris RD, Stuebing KK, Fletcher JM, et al. Subtypes of reading disability: Variability around a phonological core. J Educ Psychol 1998;90:347-73.
28. Report of the National Reading Panel. Teaching children to read: An evidence-based assessment of the scientific research literature on reading and its implications for reading instruction. Bethesda (MD): National Institute of Child Health and Human Development, National Institutes of Health); 2000.
29. Ben-Dror I, Pollatsek A, Scarpati A. Word identification in isolation and in context by college dyslexic students. Brain Lang 1991;40:471-90.
30. Bruck M. The adult functioning of children with specific learning disabilities: a follow-up study. In: Siegel I, editor. Advances in applied developmental psychology. Norwood (NJ): Ablex; 1985. p. 91-129.
31. Bruck M. Word-recognition skills of adults with childhood diagnoses of dyslexia. Dev Psychol 1990;26(3):439-54.
32. Bruck M. Outcomes of adults with childhood histories of dyslexia. In: Hulme C and Joshi RM, editors. Reading and Spelling: Development and disorders (Mahwah, NJ: Erlbaum) 1998. p. 179-200.

33. Lefly DL, Pennington BF. Spelling errors and reading fluency in compensated adult dyslexics. Ann Dyslexia 1991;41:143-62.
34. Dejerine J. Sur un cas de cécité verbale avec agraphie, suivi d'autopsie. Soc Biologie 1891;43:197-201.
35. Damasio AR, Damasio H. The anatomic basis of pure alexia. Neurology 1983;33:1573-83.
36. Friedman RF, Ween JE, Albert ML. Alexia. In: Heilman KM, Valenstein E, editors. Clinical neuropsychology. 3rd ed. New York: Oxford University Press; 1993. p. 37-62.
37. Geschwind N. Disconnection syndromes in animals and man. Brain 1965;88:237-94.
38. Dejerine J. Contribution a l'etude anatomo-pathologique et clinique des differentes varietes de cecite verbale. Memoires Soc Biologie 1892;4:61-90.
39. Galaburda AM, Sherman GF, Rosen GD, Aboitiz F, Geschwind N. Developmental dyslexia: four consecutive patients with cortical anomalies. Ann Neurology 1985;18(2):222-33.
40. Filipek P. Structural variations in measures in the developmental disorders. In: Thatcher R, Lyon G, Rumsey J, Krasnegor N, editors. Developmental neuroimaging: mapping the development of brain and behavior. San Diego, (CA): Academic Press; 1996. p. 169-86.
41. Klingberg T, Hedehus M, Temple E, et al. Microstructure of temporo-parietal white matter as a basis for reading ability: evidence from diffusion tensor magnetic resonance imaging. Neuron 2000;25: 493-500.
42. Brunswick N, McCrory E, Price CJ, Frith CD, Frith U. Explicit and implicit processing of words and pseudowords by adult developmental dyslexics: a search for Wernicke's Wortschatz. Brain 1999;122: 1901-17.
43. Helenius P, Tarkiainen A, Cornelissen P, Hansen PC, Salmelin R. Dissociation of normal feature analysis and deficient processing of letter-strings in dyslexic adults. Cereb Cortex 1999;4:476-83.
44. Horwitz B, Rumsey JM, Donohue BC. Functional connectivity of the angular gyrus in normal reading and dyslexia. Proc Natl Acad Sci U S A 1998;95:8939-44.
45. Paulesu E, Demonet J-F, Fazio F, et al. Dyslexia-cultural diversity and biological unity. Science 2001;291:2165-7.
46. Rumsey JM, Andreason P, Zametkin AJ, et al. Failure to activate the left temporoparietal cortex in dyslexia. Arch Neurol 1992;49:527-34.
47. Rumsey JM, Nace K, Donohue B, Wise D, Maisog JM, Andreason P. A positron emission tomographic study of impaired word recognition and phonological processing in dyslexic men. Arch Neurol 1997;54:562-73.
48. Shaywitz SE, Shaywitz BA, Pugh KR, et al. Functional disruption in the organization of the brain for reading in dyslexia. Proc Natl Acad Sci U S A 1998;95:2636-41.
49. Shaywitz BA, Shaywitz SE, Pugh KR, et al. Disruption of posterior brain systems for reading in children with developmental dyslexia. Biol Psychiatry 2002;52:101-10.
50. Simos P, Breier J, Fletcher J, Bergman E, Papanicolaou A. Cerebral mechanisms involved in word reading in dyslexic children: A magnetic source imaging approach. Cereb Cortex 2000;10:809-16.
51. Salmelin R, Service E, Kiesila P, Uutela K, Salonen O. Impaired visual word processing in dyslexia revealed with magnetoencephalography. Ann Neurol 1996;40:157-62.
52. Temple E, Poldrack R, Salidas J, et al. Disrupted neural responses to phonological and orthographic processing in dyslexic children: an fMRI study. Neuroreport 2001;12:299-307.
53. Demb J, Boynton G, Heeger D. Functional magnetic resonance imaging of early visual pathways in dyslexia. J Neurosci 1998;18: 6939-51.
54. Eden GF, VanMeter JW, Rumsey JM, Maisog JM, Woods RP, Zeffiro TA. Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. Nature 1996;382:66-9.
55. Corina D, Richards T, Serafini S, et al. fMRI auditory language differences between dyslexic and able reading children. Neuoreport 2001;12:1195-1201.
56. Paulesu E, Frith U, Snowling M, et al. Is developmental dyslexia a disconnection syndrome? Evidence from PET scanning. Brain 1996; 119:143-57.
57. Georgiewa P, Rzanny R, Hopf J, et al. fMRI during word processing in dyslexic and normal reading children. Neuroreport 1999;10:3459-65.
58. Gross-Glenn K, Duara R, Barker WW, et al. Positron emission tomographic studies during serial word-reading by normal and dyslexic adults. J Clin Exp Neuropsychol 1991;13(4):531-44.
59. Logan G. Toward an instance theory of automatization. Psychol Rev 1988;95:492-527.
60. Logan G. Automaticity and reading: perspectives from the instance theory of automatization. Read Writ Q: Overcom Learn Dis 1997; 13:123-46.
61. Cohen L, Dehaene S, Naccache L, et al. The visual word form area: spatial and temporal characterization of an initial stage of reading in normal subjects and posterior split-brain patients. Brain 2000;123: 291-307.
62. Cohen L, Lehéricy S, Chochon F, Lemer C, Rivaud S, Dehaene S. Language-specific tuning of visual cortex? Functional properties of the visual word form area. Brain 2002;125:1054-69.
63. Dehaene S, Naccache L, Cohen L, et al. Cerebral mechanisms of word masking and unconscious repetition priming. Nature Neurosci 2001;4:752-8.
64. Dehaene S, Le Clec'H G, Poline J, LeBiha D, Cohen L. The visual word form area: a prelexical representation of visual words in the fusiform gyrus. Neuroreport 2002;13:1-5.
65. Moore C, Price C. Three distinct ventral occiptotemporal regions for reading and object naming. Neuroimage 1999;10:181-92.
66. Price C, Moore C, Frackowiak RSJ. The effect of varying stimulus rate and duration on brain activity during reading. Neuroimage 1996;3:40-52.
67. Fiez JA, Peterson SE. Neuroimaging studies of word reading. Proc Natl Acad Sci U S A 1998;95(3):914-21.
68. Frackowiak R, Friston K, Frith C, Dolan R, Mazziotta J. Human brain function: New York Academic Press; 1997.
69. Torgesen J. The prevention of reading difficulties. J Sch Psychol 2002;40:7-26.
70. Woodcock RW, Johnson MB. Woodcock-Johnson Psycho-Educational Battery—Revised (WJ- R). Allen (TX): Developmental Learning Materials; 1989.