Developmental Dyslexia: A Motor-Articulatory Feedback Hypothesis

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Reading is mediated by parallel and widely distributed modular systems. There are, therefore, multiple loci in these systems where dysfunction may lead to developmental dyslexia. However, most normal children learn to read using the alphabetic system. Learning to use this system requires awareness that words are comprised of a series of speech sounds (phonological awareness) and the knowledge of how to convert letters (graphemes) into these speech sounds (phonemes). Most dyslexic children have deficient phonological awareness and have difficulty converting graphemes into phonemes. Studies of patients with acquired lesions who are unable to convert graphemes into phonemes, as well as positron emission tomographic studies of normal subjects, suggest that the left inferior frontal lobe is important in phonologic reading. Phonetic gestures are represented in the brain as invariant motor commands that program the articulators. Phonologic reading may activate the left inferior frontal lobe because grapheme-to-phoneme conversion requires activation of these motor-articulatory gestures. Dyslexic children are unaware of the position of their articulators during speech. The inability to associate the position of their articulators with speech sounds may impair the development of phonological awareness and the ability to convert graphemes to phonemes. Unawareness of their articulators may be related to programming or feedback deficits.

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Developmental dyslexia is a common and disabling disorder that has been diagnostically defined as a discrepancy between the acquisition of reading skills and other intellectual abilities, providing that this reading disability is not caused by environmental conditions (inadequate education), sensory deficits, or acquired neurological disorders. Recently, the discrepancy definition has fallen into disfavor and reading level, independent of intellectual ability, has been used as the diagnostic definition [1]. Although there have been substantial gains in our knowledge about dyslexia, the neurobiological and neuropsychological basis of this disorder is as yet not fully defined. There are also many unresolved questions as to whether dyslexia is a specific pathological entity. For example, Shaywitz and colleagues [2] have provided evidence that dyslexia occurs along a continuum and is the "tail of a normal distribution of reading ability." Although epidemiological studies suggest that dyslexia is not an all-or-none phenomenon, Shaywitz and colleagues [2] suggested that dyslexia is still a pathologic phenomenon, but, like hypertension, it occurs in varying degrees of severity. Evidence that dyslexia is a familial-hereditary disorder [3, 4] provides support that dyslexia is a pathological entity.

Although there are probably several subtypes of dyslexia with different neuropsychological and neurobiological characteristics, we will focus on one type. When children learn to read they often use the alphabetic system, learning the speech sounds that are associated with different letters. A failure to develop this reading strategy is called phonological dyslexia. The purpose of this study is to put forth an efferent or "motor-articulatory feedback" hypothesis for one type of developmental dyslexia—phonological dyslexia. However, prior to presenting this hypothesis we will briefly discuss some other current hypotheses.

Visual Hypotheses

Livingstone and coworkers [5] showed that people with developmental dyslexia have decreased evoked potentials to rapid, low-contrast visual stimuli, but normal responses to slow, high-contrast stimuli. In primates, fast, low-contrast visual stimuli are processed by the magnocellular subdivision of the visual system and slow, high-contrast stimuli are mediated by the parvocellular system. When Livingstone and coworkers [5] compared the lateral geniculate nuclei of subjects with dyslexia to those of controls, they found abnormalities of the magnocellular division but not of the parvocellular system. When reading using the alphabetic system, one has to perceive form (letters) from background stimuli and record their location and their relationships to other stimuli. Although there may be a subgroup of dyslexic children that are reading impaired because of dysfunction in the visual system, most dyslexic children have no difficulty naming letters, and compensated dyslexic subjects can read by the whole-word method. Reading whole words may be a more complex task visually than reading isolated letters. Dyslexic children also perform like controls on tasks that require the processing of visual–spatial information.

Acquired reading disorders, or alexia, are often associated with lesions in the dominant hemisphere in the region of the inferior parietal lobe. Since Déjerine [6] first described alexia from lesions in this area, it has been posited that the inferior parietal lobule in the re-

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gion of the angular gyrus contains the visual representations of learned words. Today these memory stores are called orthographic lexical representations and are posited to be stored in neuronal networks [7]. Galaburda and his coworkers [8] performed autopsy studies on several dyslexic men whose brains showed cortical anomalies including dysplasia and ectopia in the region of the left inferior parietal lobule. In addition, Rumsey and associates [9] studied severely dyslexic men with positron emission tomography (PET) and found there was reduced activation in the left inferior parietal lobes in the region of the angular gyrus.

Dyslexia may not be a homogeneous disorder. Although these anatomic abnormalities in the temporal and parietal lobes may account for developmental dyslexia in some patients, we do not believe they can account for the dyslexia seen in all reading disabled children. Studies of acquired alexic subjects demonstrate that there are patients who have more difficulty reading irregular words (words that cannot be sounded out by using a letter-by-letter strategy) such as "yacht" than reading regular words such as "blanket" and nonsense words such as "flig." This acquired alexia is termed orthographic, lexical, or "surface" alexia. Although there may be children with this lexical form of developmental dyslexia, we are focusing our discussion on children who are unable to use the alphabetic reading system or phonological dyslexia.

Auditory Hypotheses

Galaburda [10] demonstrated that people with dyslexia may have abnormalities in the auditory system that are similar to those found in the visual system. Because of their abnormalities in the magnocellular division of the medial geniculate nucleus, dyslexic children may have difficulties correctly perceiving low-contrast, complex sounds that require rapid discriminations. The perception and comprehension of speech does require rapid formant discriminations [11], and since the time of the report by Orton [12], it has been noted that dyslexic children often have associated speech and language disturbances. Although speech and language disturbances may be a contributory factor to developmental dyslexia, there are many dyslexic children who when learning to read have normal speech comprehension. For these children with normal speech comprehension, it would be difficult to understand how a defect in the auditory magnocellular system could fully account for their profound reading disorder. However, as we will discuss, learning to read may be aided by the development of phonological awareness, and phonological awareness may depend, in part, on the ability to hear words phoneme by phoneme. Defects in the auditory magnocellular system may impair this type of phonemic analysis.

Orton [12] posited that dyslexia was related to a failure to establish left hemisphere dominance. On an unselected sample of autopsied brains, Geschwind and Levitsky [13] demonstrated that the posterior portion of the superior temporal lobe (the planum temporale) was often larger on the left side than it is on the right side. More recently, using selective hemispheric anesthesia and magnetic resonance imaging (MRI) scanning, Foundas and colleagues [14] reported there was a close relationship between language dominance and asymmetries of the planum temporale. The planum temporale is part of the auditory association cortex and is probably synonymous with what has been termed Wernicke's area. Wernicke's area is critical in speech comprehension and appears to store lexical representations (the sound images of words). Galaburda and his co-workers [8] did postmortem studies of dyslexic men and also found dysplasia and ectopia in the posterior portion of the superior temporal lobe or in and around Wernicke's area. There was also a loss of the normal left/right asymmetry found in the planum temporale. Using MRI, several investigators have reported similar findings [15, 16]. However, as we discussed, many dyslexic children have normal speech comprehension. Therefore, a defect in an auditory input lexicon cannot fully account for the profound reading disturbance seen in some dyslexic individuals.

Phonological Dyslexia

There are patients who can read regular and irregular words much better than they can read nonwords. These acquired alexias are called phonological alexia and "deep" alexia depending upon whether the patient also makes semantic errors. Based on these studies of brain-damaged subjects, neuropsychologists have posited that there are a least two methods by which one can read, a lexical or whole-word method and a phonological method that requires conversion of graphemes to phonemes.

A phoneme is defined as the smallest unit of speech that distingushes one word from another. In English there is not a direct correpondence between letters in the alphabet and speech sounds or phonemes. For example, the letters "th" make one phoneme. Some letters are not pronounced but rather influence the pronunciation of other letters. Therefore, the letter or letters that symbolize one speech sound are called graphemes. In the grapheme-to-phoneme conversion system, one breaks apart or parses words and then finds the speech sound that is associated with each letter or groups of letters. In phonological and deep alexia this system is impaired (see Friedman and co-workers [17] for a review). One cannot read irregular words such as "yacht" by using the grapheme-to-phoneme conversion system, but rather one has to derive the pronunciation by using the whole-word or lexical route. In lexical or surface alexia this whole-word route is impaired. If parietal dysfunction was inducing all cases of develop-

mental dyslexia, then one would expect that reading-disabled children would have a lexical type of dyslexia. However, studies have demonstrated that most children with developmental dyslexia are impaired at nonword reading, which requires using the grapheme-to-phoneme conversion route. Many dyslexic children do learn to read. The term compensated developmental dyslexia has been used to describe people who are able to eventually learn to read adequately but who have to use the whole-word or lexical system. Typically these patients may have persistent difficulty in reading nonwords. Patients with persistent or uncompensated dyslexia as adults, such as those studied with PET scans by Rumsey and colleagues [9], may not have been able to compensate because they have parietal (angular gyrus) dysfunction.

Motor-Articulatory Feedback Hypothesis

We propose that when learning to read using the alphabetic system, one must learn to perform grapheme-to-phoneme conversions and these conversions require activation of motor-articulatory gestures. In adults an acquired inability to read nonwords (phonological and deep alexia) may be associated with a variety of lesions, but one of the most common areas to be injured in these patients is the anterior perisylvian region, including the sensorimotor cortex, premotor areas, and the pars opercularis and triangularis (Broca's area), areas critical for articulation. That these anterior perisylvian areas are important in reading is supported by functional imaging studies that demonstrate when normal subjects read these anterior perisylvian areas become activated [18]. This activation can be seen even when the subject is reading silently. The activation in the left inferior frontal region is greater when reading nonwords or pseudowords than when reading irregular words [19]. Further evidence that the anterior perisylvian region is important for grapheme-to-phoneme conversion comes from the study of an adult patient with a lesion in the anterior perisylvian region (Brodmann's area 6) who had reading epilepsy. The patient's seizures were provoked when the patient read using grapheme-to-phoneme transformations [20].

The concept that motor-articulatory gestures are important in perception is not a new one. There is an extensive literature on the motor theories of speech perception (see Sokolov [21], Liberman and Mattingly [22] for a review). The claim of this motor theory of speech perception is that the objects of speech perception are the intended articulatory gestures of the speaker. These gestures are represented in the brain as invariant motor commands that program the movements of the articulators. These articulatory gestures provide the basis for phonemic categories. Motor theories of speech perception propose that articulatory gestures are not only the elemental events of speech production but are also critical for speech perception. Therefore, to perceive a speech utterance one must perceive a specific pattern of intended gestures. According to Liberman and Mattingly [22], normally the conversion from the acoustic signal to articulatory gesture is automatic, the link being innately specified and, according to Sechenov (quoted by Sokolov [21]), muscular sensations produced by soundless movements of the tongue within the oral cavity result in the sound image of words being replaced by kinesthetic sensations.

The motor theory of grapheme-to-phoneme conversion shares some elements of the motor theory of speech perception. However, this link has to be learned rather than being innately specified. To read using the grapheme-to-phoneme conversion (alphabetic) system, one must know that words are comprised of a series of phonemes. Gleitman and Rozin [23] have noted that although spoken words can acoustically be broken into syllables, there is no indication of separate phonemes in the acoustic syllabic bundle. Based solely on the acoustic properties of spoken words, it would be difficult for a child to know that a spoken word (e.g., cat) has a series of phonemes. Therefore, before a child can learn to use the grapheme-to-phoneme conversion system, the child must first develop phonological awareness. The motor-articulatory feedback theory of speech perception may explain how one develops phonological awareness. According to this motor theory, the perception of spoken words is associated with the production of intended articulatory gestures. Therefore, for each phoneme in a word there is a new movement of the articulators. Awareness of the movements of the articulators would allow the child to parse a word into its component phonemes. If awareness of the position and movement of the articulatory apparatus is critical for phonemic parcellation of words and dyslexic children are unaware of the position and movements of the articulatory apparatus, they would be less able to perceive the low-contrast, rapid temporal presentation of the phonemic components of spoken words. This unawareness of the phonological composition of words may also account for some of the speech disturbances seen in these children. For example, dyslexic children often have difficulty rapidly repeating phonologically complex phrases and nonsense syllables [24, 25]. However, these speech–language deficits are often subtle and most dyslexic children do not need the service of school-based speech–language services for speech problems.

When a child who is learning to read is confronted with a new printed word, the child must convert the letters in this word into phonemes. Although theoretically children should be able to learn to associate specific speech sounds (phonemes) with specific graphic representations (letters), according to this motor theory, children use the articulatory apparatus when learning to associate phonetic gestures with specific graphic representations. Therefore, learning to read would in-

volve coupling the specific articulatory gestures that are associated with specific graphemes. These articulatory gestures then can be easily coded into the speech sounds they ordinarily produce. The coupling of articulatory gestures with their auditory (phonemic) and visual (graphemic) representations is, perhaps, why children move their lips and tongue when learning to read. Sokolov [21] notes that several investigators have reported that the mechanical retardation of articulation by clamping the lips and tongue between the teeth hinders children in learning to read. As one becomes a skilled reader, one may be able to use articulatory gestures without making overt movements. However, studies using electromyograms of the tongue during silent reading does reveal activity [21]. In addition, even when adults with good reading skills are presented with novel nonwords they may revert to overtly moving their articulatory apparatus.

As we discussed, children with phonologic developmental dyslexia have the most difficulty reading nonwords and have a deficit in their ability to learn how to convert graphemes into phonemes. According to the articulatory feedback hypothesis, developmentally dyslexic children may be reading disabled because they are unable to spontaneously use articulatory gestures when attempting to convert graphemes to phonemes.

There are several studies that appear to support the articulatory feedback hypothesis of developmental dyslexia. Montgomery [26] studied dyslexic and normal children who were the same reading age (approximately 8 years) but different chronological ages. The children were presented with 10 different phonemes and 9 cartoons of midsagittal sections through the head that illustrated the position of tongue, teeth, and lips used to articulate these phonemes. After hearing a specific phoneme, the child was asked to repeat the phoneme and to point to the cartoon that best illustrated the position of the articulators when producing this phoneme. Montgomery [26] found the dyslexic children, when compared with controls, were severely impaired at this task. Because the children were able to normally repeat the phoneme, a deficit on this articulatory awareness task could not be explained by an auditory perceptual defect. Rather it appears that the children were unaware of the movements and position of their articulators when they were producing phonemes. This unawareness of their articulators when producing phonemes may be responsible for an inability to convert graphemes into phonemes. Magnusson and Naucler [27] demonstrated that phonological awareness is one of the best predictors of dyslexia, and unawareness of one's articulatory gestures may also account for impaired phonological awareness.

If awareness of the movement and position of one's own articulatory apparatus plays an important role in both phonological awareness and in the ability to convert graphemes into phonemes, then treatment of dyslexia should include speech therapy that teaches the dyslexic to be aware of the articulatory gestures. Alexander and her coworkers [28] studied 10 dyslexic children. These dyslexic childrens' phonological awareness skills were assessed with the Lindamood Auditory Conceptualization Test, and they demonstrated a marked impairment on this skill. The children were also tested with the Word Identification and Word Attack subtests of the Woodcock Reading Master Test [29]. The children were then trained using the Auditory Discrimination in Depth (ADD) program of Lindamood and Lindamood [30]. In this program subjects are first trained in oral motor awareness. This program uses proprioceptive and tactile information from the articulators and visual information from a mirror that allows the subjects to view their mouths while they make individual speech sounds or phonemes. When they make speech sounds they also receive auditory feedback. For example, when subjects make a /b/ sound and watch themselves in the mirror, they note that this sound is made by closing the lips and letting the air explode out of the mouth. Each one of the phonemes is also given a name that reinforces the motor characteristics of the articulatory gesture. For example, the bilabial plosives are called "lip poppers." Whereas a /b/ is a noisy lip popper (voiced), a /p/ is a quiet lip popper (unvoiced). After subjects discover what their articulators are doing when making speech sounds and they learn to describe and label the articulatory characteristics of the 39 phonemes used in English, they are taught to select cartoons of the articulatory apparatus that best represent each of the articulatory motor characteristics of the 39 phonemes. This training in oral awareness allows the subjects to become consciously aware of the distinctive position and movements used to produce phonemes. Following this oral awareness training, the subject receives phonological awareness training and learns to associate graphemes with the articulatory gestures that produce the target phonemes. Students then practice reading pseudowords of increasing complexity. Following training, not only did the children in the study by Alexander and colleagues [28] demonstrate a remakable improvement on tests of phonological awareness, but they also improved their performance on both the Word Identification and Word Attack Tests [29]. This latter task depends on grapheme-to-phoneme conversions, and with this training the subjects of Alexander and colleagues [28] improved so that they were in the normal range.

The oral awareness study of Montgomery [26] and the treatment study of Alexander and coworkers [28] provide support for the motor-articulatory awareness theory of dyslexia.

Possible Causes of Unawareness

What remains unclear is why dyslexic children would be unaware of the position and movement of their articulators. There are several possibilities. There may be an articulatory or phonetic programming defect. Articulatory and phonetic production deficits have been reported in dyslexic children [31]. The presence of speech programming disorders suggests that their articulatory motor representations are disordered or poorly specified. However, these speech defects are not always strongly associated with dyslexia and are usually gone or minimal by school age when the child learns to read.

Maher and associates [32] reported aphasic patients who made frequent paraphasic errors when speaking but were unaware of their errors. When their speech was recorded and played back to them, they were easily able to recognize their errors. Studies of these patients suggest that they may not have recognized their on line errors because their speech was effortful and they may not have had residual attentional capacity to simultaneously speak and monitor their own speech. Therefore, perhaps dyslexic children also have difficulty simultaneously speaking and monitoring their articulators. By the term "unawareness" we mean a feedback failure rather than a failure to reach conscious awareness. Feedback failures may also be caused by sensory deficits. Although there is no evidence that dyslexic children have elemental sensory disorders of the articulatory apparatus, perhaps there is a defect in the magnocellular division of the ventral posterior medial nucleus of the thalamus (similar to that seen in the auditory and visual geniculate nuclei) that impairs tactile and proprioceptive feedback from the articulatory apparatus. A feedback failure may also account for the delay in developing articulatory motor representations. Although sensory deficits of this magnitude should also preclude the development of normal speech, perhaps the articulatory awareness required for reading is greater than that needed for speech.

Unawareness in a brain-damaged population is most commonly related to either inattention or to a representation deficit (see Heilman and colleagues [33]). One of us (A.W.A.) has noted that dyslexic children are often unaware of debris left around their mouth after eating. In monkeys, whereas lesions of both banks of the frontal arcuate sulcus (Brodmann's area 8) produce unawareness or neglect of extrapersonal space, Rizzolatti and collaborators [34] demonstrated that postarcuate lesions (Brodmann's areas 6 or perisylvian premotor cortex) produce inattention around the monkey's mouth. As we discussed, not only may anterior perisylvian lesions cause an impairment of grapheme-to-phoneme conversion, but Galaburda's postmortem study of dyslexic subjects also demonstrated anterior perisylvian anomalies. In a series of clinical studies, Bisiach and his coworkers [35] demonstrated that unawareness and inattention may be caused by a representational deficit, and Coslett (personal communication) demonstrated that a patient with a cerebral lesion had a representational deficit for his hand but not for space. Perhaps developmentally dyslexic children have a representational deficit for their articulatory apparatus (i.e., mouth, lips, tongue, and so on). Such a deficit would interfere with their awareness of their own articulators. This unawareness would impair the development of graphemic–articulatory–phonemic associations and induce developmental dyslexia.

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