Australian Orthoptic Journal 1989; 25: 7-9

DYSLEXIA IS A DAB WORD

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Abstract

Dyslexia is a word which is used inappropriately by the general public and has acquired a number of undeserved connotations. A brief historical review is presented, and modern theories concerning the cause of specific primary dyslexia are presented.

Key words: *Dyslexia, learning disability, reading disability, dysgraphia.*

Consider for a moment what it would be like not to be able to read public notices, to be intimidated by a page of print, or to be cut off from our immensely rewarding literary heritage. These problems are part of the daily experience of a dyslexic.

The disorder now known as dyslexia was first described by Kussmaul in 1877 as "word blindness", but the word "dyslexia" was coined by Berlin in 1887. It was the Scottish Ophthalmologist Hinshelwood[1] who first differentiated alexia from dyslexia in 1895.

Dyslexia must be differentiated from the other four learning disabilities — educational deprivation, intellectual impairment, disorders of special senses (deafness, blindness), and emotional disorders. The group of disorders known as dyslexia can be further sub-divided into specific primary dyslexia, hyperkinetic syndrome, dysphasia, dysgraphia, and dyscalculia. Because it has been loosely defined in the past, and because it has acquired connotations of dim wittedness, laziness, and delinquency in some quarters — dyslexia is a bad word. The proposed alternatives include dyssymbolia, specific reading disability, word blindness, and strephosymbolia — but all are too narrow or too oblique in meaning.

The remainder of this discussion concerns specific primary dyslexia, which is defined as, "a disorder manifested by difficulty learning to read despite conventional instruction, adequate intelligence, and sociocultural opportunities. It is dependent on fundamental cognitive disabilities which are frequently of constitutional origin".[2]

Dyslexia occurs in languages which progress from right to left as well as those which progress from left to right, but the lack of logical orthography in English means that dyslexics are identified more easily. It affects 15% of school age children, 50% of young unemployed, and 70% of juvenile offenders.[3]

Some factors argue a genetic cause — an incidence in males eight times that in females, higher incidence in those with dominant left hand or left eye, 100% concordance in identical twins, 33% concordance in dizygotic twins, and up to 88% incidence in relatives of those affected.

Other factors argue an acquired cause — a 50% higher incidence in the second half of large

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sibships, and a high incidence of major stress events in early life.

A large number of epiphenomena are described, including EEG abnormalities, colour agnosia, clumsiness, left right confusion, other abnormalities of language development, poor attention span, slowness and variability of performance when using other symbols. Like other syndromes there is a large variability between cases, but a common pattern unites them. Unfortunately some investigators have been distracted by these epiphenomena — and spurious new syndromes have been defined, such as minimal cerebral disfunction.

Medical attention has been predictably focused on the problem of "where is the lesion?" In the early 1900's the angular gyrus was suspected and indeed a number of acquired neurological conditions manifest reading difficulties. Lesions around the left angular gyrus due to posterior cerebral infarction, aneurysm, metastasis or meningioma, can cause alexia without either agraphia or the usual right homonomous hemianopia. This is due to disconnection of the occipital cortex from Wernicke's area. Bilateral lesions in the visual association areas cause prosopagnosia, simultanagnosia, or optic ataxia, depending on the site of the lesions.[4] While these may all produce reading difficulties, none are like dyslexia.

Galaburda et al in 1985[5] reported lack of the usual asymmetry in the planum temporale — and subsequent reports have added weight to the notion of failure of right hemisphere regression in dyslexics. This work supplants previous notions of delayed myelination in dyslexics.

The notion of a visual defect in dyslexics has persisted despite the recent ascendancy by theories supporting a primarily central or language problem. Pavlidis[6] reported saccadic abnormalities in dyslexics, and despite several excellent studies refuting his findings, the notion of saccadic problems has found a place in medical folklore. While dyslexics do demonstrate abnormal saccades when reading — a situation which requires higher processing — they perform quite normally when using a target such as flashing LED's in an array. A small number have abnormal saccadic intrusion when tracking a target at pursuit velocities of 5° per second.[7] Two more recent findings may be of note, especially when considered together. Lovegrove[8] reports that dyslexics have abnormalities in their transient ganglion cell function. It is known that during reading, saccades of approximately 25 m-seconds intrude between fixations of around 250 m-seconds. Each fixation absorbs a letter string of some 20 spaces — forming a precategorical mental icon in short term visual memory. However, the after image of the sustained cells is some 300 m-seconds, so it intrudes on sebsequent fixations. Transient retinal elements — Y cells and some W cells — have shorter retina to cortex conduction times and shorter after images.

Geiger and Letvin[9] reported that dyslexics have greater ability to detect a letter flashed in their extra foveal field, whereas non dyslexics detect a letter flashed in their foveal field better than dyslexics. It is known that the fovea has mainly sustained, or X type ganglion cells, while the extra foveal retina has mainly transient elements, with better temporal but poorer spacial resolution. Could it be that dyslexics fail to make the central adaptations necessary to enable rapid sequential fixations, and instead employ the strategy of using extra foveal retina, with its transient elements? In so doing, normal saccadic function, which serves to foveate the fixation target, would have to be altered, thus explaining the abnormal saccades.

Whatever the real answer may be, it is still safe to say that we do not yet know the site of the lesions.

Dyslexic patients usually present with poor school performance at second or third grade level, and may demonstrate certain characteristic features which help to confirm the diagnosis. There may be a positive family history, some difficulty with colour naming, crossed hand eye dominance, some history of retarded language development, left right confusion, a tendency to read letters on the chart which are not there, and skip other letters which are usually read, and some difficulty with compound cooperation such as is required for slit lamp and visual field examination. It is important to ensure that the child

has had an adequate educational opportunity, that he has had normal developmental milestones and does not have any hearing or visual impairments. Assuming emotional disturbances have been excluded, the diagnosis can be confidently made at this time. A record of reading age, handwriting, and a standard drawing test, is useful.

What is the management of dyslexia? In the past, both perceptual training and visual training have been advocated, and both have been systematically proven to have no benefit in the treatment of dyslexia. In 1971 the American Academy of Ophthalmology and the American Academy of Paediatrics made a strong statement to this effect. The use of coloured glasses has recently come into vogue, and was introduced into this country in 1985 following a report on the 60 Minutes television programme. Since that time no scientific evidence has been produced to support the use of this modality, and the Royal Australian College of Ophthalmologists, the American Academy of Ophthalmologists, and the American Optometrist Association have all made statements to this effect. In Western Australia those prescribing coloured lenses follow either the Irlen technique, and pay a five thousand US dollar per year franchise for the right to prescribe these lenses, or else the Meares technique. These lenses are aggressively marketed within the local schools, and it is fair to say that a great deal of time and money is being diverted from conventional treatment modalities by these coloured lens prescribers. The only treatment which confers proven advantage to dyslexic children is a very thorough and systematic educational programme, supplemented by additional remedial tuition. This is arranged after assessment by the local school guidance officer who is under the control of a district guidance officer, who is in turn controlled through the Education Support Branch of the Education Department of Western Australia.

There are also private clinics available.

The role of the Ophthalmologists and Orthoptists in the management of dyslexia is to treat any coincidental visual problem, make a firm diagnosis, advise the family of where best to seek assistance, and offer an informed opinion regarding the controversial treatment modalities. We must all make an effort to keep in touch with developments in this field as these families are extremely vulnerable to unscrupulous practitioners who are able to profit out of their misfortune.

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