

Dyslexia

Medical fact or fiction?

Following last month's media coverage of controversial claims about the 'myth' of dyslexia (OT 23/09/05), Professor Arnold Wilkins discusses current theories and treatment strategies.



From a medical viewpoint, dyslexia is a congenital and developmental condition associated with neurological anomalies in the brain. From an educational viewpoint, the term was used to refer to a person with selective, persistent and significant problems with reading, writing, spelling and sometimes mathematics and musical notation, where these problems were not explained by a general intellectual deficit. It has proved difficult to show any differences between reading difficulties in individuals with dyslexia and those without, however, and the requirement for discrepancies between reading and other abilities has now been dropped. It has become even more difficult to define the term dyslexia, but the reading problems, spelling difficulties etc, continue to cause concern and controversy regardless of the label attached to them.

Research focus

Much attention has been paid to the magnocellular deficits of vision that some individuals with dyslexia reveal. Vidyasagar¹ goes so far as to assert, "It has long been suspected that the basic lesion in dyslexia may be a M (magnocellular) impairment in the visual pathway", citing many papers²⁻¹¹. These studies found psychophysical and neuro-imaging evidence that the magnocellular pathways showed impaired function in some (not all) individuals with dyslexia.

Few of these studies have included other individuals with cognitive or neurological impairment for comparison with the dyslexic group, and were they to have done so, magnocellular deficits would almost certainly have been discovered – not only in the dyslexics but in the other patient groups as well, including those whose reading is quite normal. Patients with migraine, for example, have recently been reported as showing magnocellular deficits¹²⁻¹⁴ with some exceptions¹⁶.

The strongest research evidence that a patient population is impaired on a particular function comes when there is a double dissociation. For example, if Patient Group 1 shows impairment of function A and not B, whereas Group 2 shows impairment of function B but not A, it is impossible to attribute either impairment, A or B, to a non-specific general deficit. A double dissociation has

never been demonstrated with respect to magnocellular deficits in dyslexia, for example, a parvocellular deficit in one patient group and magnocellular deficit in another.

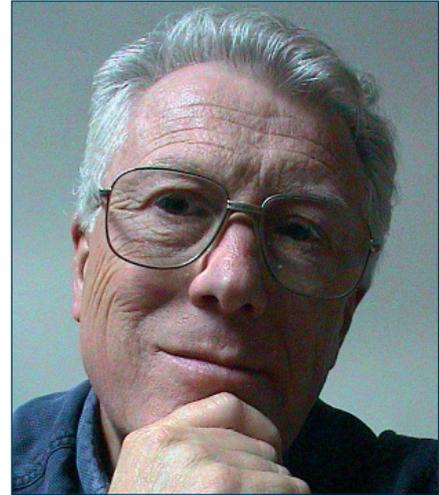
With one notable exception in the case of Williams' syndrome¹⁷, the studies have shown deficits exclusively in magnocellular and not parvocellular function and the deficits are usually demonstrated simply by comparing dyslexic and normal readers. This is unfortunate, because it would appear that magnocellular deficits are quite non-specific and occur in several disorders, including Williams' syndrome and migraine. They may arise simply because the magnocellular system is more fragile in some way, more susceptible to disease and trauma of whatever kind, perhaps because of the greater metabolic rate of larger cells.

Although there is plenty of convergent evidence for a magnocellular deficit in some individuals who have reading difficulties, there is no evidence that magnocellular deficits are exclusively associated with dyslexia or with reading difficulties, and there is clear evidence to the contrary. Until the results of longitudinal studies become available, the idea of a link between reading difficulty and magnocellular deficits relies on studies showing a correlation between magnocellular deficits and reading ability, when other more general abilities are statistically allowed for¹⁸⁻²¹.

The way in which statistical allowance is best made is controversial. Nevertheless, the idea that magnocellular deficits are in some way responsible for dyslexia has gained many adherents, and much attention has been given to how a magnocellular deficit could lead to a reading disability^{1,22-24}.

Reasons for failure

Recent media coverage of dyslexia in the *Times Education Supplement* and on *Channel 4* has sought to dispel the 'myth' that dyslexia is due to a visual impairment. I would argue that the jury is still out. It is quite unclear what dyslexia is due to, and it is unlikely to be just one thing. Reading is a complex visual, phonological, linguistic and cognitive skill, and might be expected to fail for a large number of different reasons. It may be more appropriate to concentrate on the reasons



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for reading failure than on the classification of individuals with such failure.

Visual stress can certainly be one of the reasons for reading failure, although it would be foolish to claim that it is the only reason or even the most important one. However, treatment of visual stress is simple and quick and can make a large difference to the speed with which text is read²⁵. Using spectral filters to reduce the head pain with which reading is associated can sometimes turn a reluctant reader into an avid reader overnight, with consequences for reading acquisition.

Some 20% of unselected children in mainstream education (not simply those with reading difficulties) find coloured overlays of benefit, and the benefit is pronounced in 5%. Coloured filters do not provide a treatment for dyslexia – they provide a means of avoiding the visual stress with which reading is sometimes associated. Why are so many children affected by visual stress in this way? I believe there are several reasons.

One reason has to do with the lighting in schools. School classrooms are often very bright²⁵. They are usually lit with fluorescent lighting. Fluorescent lighting has long been known to cause headaches. It does so because of the continuous rapid flicker that occurs twice with each cycle of the electricity supply²⁶. The flicker is too rapid to be perceived, but it is resolved by the retina and affects the control of eye movements²⁷. Not all lighting installations flicker in this way. When fluorescent lamps are controlled by electronic ballast, the flicker is too rapid to be resolved by the visual system and headaches are reduced. The electronic ballast is more efficient, and costs much less to run. Unfortunately, it costs more to install, and

many schools are still fitted with the old fashioned flickering lighting. There is really no excuse for this because the lower costs of running the electronic ballast overcome the additional costs of installation in about two years.

Another reason why so many children are affected by visual stress is that their reading books are poorly designed. Children are required to read text that is of adult size long before they can see it clearly enough²⁸ to read it fluently for long periods²⁹. The text in children's reading schemes gets too small too quickly. We need reading schemes in which the text remains large, while the phonological and linguistic content advances in the usual way. Some children begin to read normally and yet start to fail at about age seven, when the text in their reading books becomes difficult for them to see comfortably.

The above refer to two ways in which the visual aspects of reading might be made less stressful, but they will not 'cure dyslexia'. Reducing the difficulties children have in distinguishing the component sounds of words may have a more important part to play in treating reading failure³⁰. Treatments of this kind have scientific backing and deserve governmental support.

Available support

Parents are understandably desperate to help their children, and partly as a result there are a large number of diverse treatments available on the market. Few of these treatments are supported by scientific evidence and some are quite bizarre. Governmental support is not appropriate for all the treatments that currently receive funding under the provision for student support, and the expenditure is not adequately monitored at governmental level.

Specialist teaching is expensive, and sometimes the only way for parents to obtain additional help in school is to obtain the 'diagnosis' of dyslexia. For those parents who have the resources to obtain it, the label can provide access not only to specialist help but also to extra time in examinations. Students formally diagnosed as having dyslexia are given up to 25% extra time in GCSEs, A levels, university degrees and vocational assessments. Those with similar reading difficulties that have not been recognised by the educational establishment have no such dispensation. There is no longer a level playing field for all candidates, including those with reading difficulty. There is simply no equitable way of calculating the extra time appropriate for a

particular individual in a particular examination, and no easy way of ensuring that all individuals that need such provision have similar access to it. Fortunately, new government interventions require schools to monitor and support any child whose reading is a cause for concern, regardless of diagnosis.

The label of dyslexia provides children who are failing at school with an 'explanation' of their difficulty. This is not an excuse for failure so much as a reason for specialist intervention. But it is also much more than this – it is a means of restoring a child's self-esteem, and should not be lightly set aside.

About the author

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References

For references email nicky@optometry.co.uk or visit www.optometry.co.uk/references.