Article ▸ Is There a Common Aetiology for Dyslexia, Visual Stress, and Pattern Glare?

Robert J. Longhurst, BSc (Hons), MCOptom, London, United Kingdom

ABSTRACT

This paper discusses the current understanding of dyslexia – the many contrasting definitions of the condition and the numerous theories regarding its aetiology. Visual stress and pattern glare are also discussed, and an attempt is made to find a common link between dyslexia and these two syndromes. An argument is then made for the term dyslexia being a misnomer and dyslexia, visual stress, and pattern glare all being related in terms of a magnocellular deficit and/or a dorsal stream processing disorder. Coloured overlays and tinted lenses are also briefly discussed in the context of these disorders.

Keywords: dorsal stream, dyslexia, magnocellular pathway, Meares-Irlen syndrome, pattern glare, Pulfrich effect, synaesthesia, visual snow, visual stress, visual timing

Introduction

What is dyslexia?

Definition and Prevalence

On 7 November 1896, a case study of Percy, a bright, intelligent 14-year-old boy with "congenital word blindness," was reported in the British Medical Journal by a British physician from Seaford, East Sussex.[1] This case study came to the attention of an ophthalmologist in Stuttgart, Rudolf Berlin. Although the condition was thought to have been first identified by Oswald Berkhan in 1881, Berlin was the first to coin the term dyslexia six years later.[2] He used his new term to describe the boy's condition. Dyslexia derives from the Greek *dys,* which means bad, abnormal, or different, and *lexis,* meaning word, and it is now in common usage. Perhaps surprisingly, these are the earliest examples of dyslexia in history.

Thirty years after Berlin first coined the term dyslexia, another ophthalmologist, James Hinshelwood, gave the following definition for the condition: *a congenital defect occurring in children with otherwise normal and undamaged brains characterised by a difficulty in learning to read.*[3] It is notable that from this definition, Hinshelwood associated the condition as occurring exclusively in children. He also believed dyslexia to be a visual problem and described symptoms such as letter reversals and spelling difficulties. Even though the British Dyslexia Association (BDA) acknowledges that there are a number of opposing definitions for dyslexia, in 2007, its management board approved the following: *Dyslexia is a specific learning difficulty … characterised by difficulties with phonological processing, rapid naming, working memory, processing speed, and the automatic development of skills that may not match up to an individual's other cognitive abilities.*[4] Conversely, the British Psychological Society (BPS) defines the condition as being evident when a child's reading ability and spelling do not develop well, despite appropriate learning opportunities.[5] These opposing definitions can make formal diagnosis difficult. Glutting et al.[6] note that the diagnosis is usually based on an indication of underachievement, where academic performance is below that expected based on an individual's IQ. IQ tests may be unreliable,[7] and the prevalence of dyslexia is therefore uncertain. Some studies conclude that dyslexia is prevalent in 4% of the Western world,[8] whereas others believe that the figure may be closer to 10%.[9]

Aetiology

Further controversies abound regarding the aetiology of dyslexia. Originally seen as a visual problem, dyslexia is now widely accepted to be a neurological condition, perhaps with several subtypes. Castles and Coltheart identified at least two subtypes.[10] One is characterised by a specific difficulty using the lexical procedure, and another by a difficulty using the sublexical procedure. Anderson lists ten subtypes from the point of view of educational psychology, based on the presenting symptoms: dysnemkinesia, dysphonesia, dyseidesia, dysphoneidesia, dysnemkinphonesia, dysnemkineidesia, dysnemkinphoneidesia, dysnomia, double deficit, and dyscalculia.[11] A brief definition of each of these subtypes can be found in Appendix A.

From the late 1970s to the early 1990s, research into the cause of dyslexia focused on phonological deficits. It was postulated by many researchers that dyslexic readers have impairment in their ability to store and to retrieve phonemes, the constituent sounds of speech. Having reduced ability to link these phonemes to graphemes, the graphical representation of these sounds, hinders their ability to read fluently.[12-15] Theorists who favour the phonological approach to dyslexia hold various opinions regarding its nature. They do, however, agree on phonology being a key aspect and likely to result from a dysfunction to the brain's left hemisphere, specifically the perisylvian areas responsible for connecting phonological and orthographic representations: Broca's area, the parieto-temporal region, and the occipito-temporal region.

There is an abundance of evidence supporting the phonological theory, including brain imaging studies,[16-21] and it remains the most widely supported theory. One study conducted by Robertson showed that when Broca's area was

stimulated, some patients had letter reversals and could not distinguish between pig and big when asked.[22] Fawcett and Nicholson summarise the phonological processing impairment as an impairment in the ability to detect and to process speech sounds, limiting the skills that are necessary for reading, such as the ability to sound out words.[23]

While the evidence for the phonological theory is compelling and has been said to provide a near complete explanation of the problems that dyslexic children experience,[23] it does not address visual processing difficulties or the motor difficulties associated with poor handwriting. Castles and Coltheart also argue that poor phonological awareness may be a result of poor reading and not a cause of it.[24] Over the last few decades, therefore, some other theories have been postulated. Perhaps the most convincing theory put forward in opposition to the phonological theory is the magnocellular deficit theory,[25] although some find it as an explanation of dyslexia to be highly controversial.[26]

The magnocellular theory refers to the role of the magnocellular pathway (M), the parvocellular pathway (P), and how they may be affected in dyslexia. These two major pathways of the visual system lead from the retina and terminate in the Lateral Geniculate Nucleus (LGN), which is found in the thalamus of the brain. Each pathway carries different information. The magnocellular pathway, or transient pathway, carries coarse-grain information and information about the location and movement of objects, whereas the parvocellular pathway carries fine-grain information, useful for the recognition and identification of objects. The magnocellular deficit theory originally proposed that at least in certain types of dyslexia, the magnocellular pathway was disrupted, meaning that it was unable to suppress the action of the parvocellular pathway sufficiently. Without this suppression, activity from one fixation would linger and affect activity from the next fixation,[27] causing confused images and disordered reading.[28-29] Recent research, however, has found the original theory to be untenable, as the parvocellular system is now known not to undergo any such suppression with eye saccades;[30] it is the magnocellular system itself that is supressed. The theory has therefore been modified[25,31] to relate magnocellular deficits to defective motion sensitivity, leading to binocular instability, and to some of the symptoms of dyslexia. These symptoms are remarkably similar to those of a condition referred to as visual stress.[32,33] Indeed, those supporting the magnocellular theory do not make reference to visual stress as being a discrete condition.

Proponents of the magnocellular theory cite post-mortem studies showing abnormalities in the brains of dyslexic readers.[28,34-36] Further, as Stein pointed out, in addition to the magno cells of the LGN being affected, magno cells of the Medial Geniculate Nucleus (MGN) can equally be affected.[31] These cells are thought to be responsible for processing rapidly changing auditory inputs. This would account for auditory impairments in dyslexia, in addition to the visual and motor impairments, encompassing nearly every apparent issue found in those with dyslexia. Indeed, de Luca et al. believe that the theory also provides the best explanation for the difficulty with convergent eye movements that dyslexic readers seem to possess.[37] The author of this paper concurs and believes that the magnocellular theory is the most persuasive in terms of dyslexia, visual stress, and pattern glare.

What are visual stress and pattern glare?

Visual stress, sometimes referred to as pattern-related visual stress (PRVS), is a type of visual perception problem that affects the way the brain processes visual information and causes discomfort, such as asthenopia.[38] It is also known as Scotopic Sensitivity Syndrome (SSS) or Meares-Irlen Syndrome (MIS). Visual stress was described by Olive Meares, a schoolteacher working in New Zealand in 1980, and Helen Irlen, a psychologist working in California in 1983. Meares detailed symptoms such as visual perceptual distortions, eyestrain, and headaches, which sufferers experience while reading and that are alleviated by using coloured filters.[39]

Jeanes et al. estimate the prevalence of visual stress to be about 20% of the population,[40] and it is believed that the condition accounts for a proportion of dyslexic readers independent of the phonological deficit model, which is most commonly used to account for dyslexia.[41] Visual stress is indeed very common in dyslexic readers, with the incidence being at least 40%.[42,43]

While the magnocellular theory can be used to account for visual stress and dyslexia, it has also been hypothesised that visual stress results from cortical hyperexcitability caused by pattern glare.[44] It is also believed that, in dyslexia, the effects of visual stress are made worse because dyslexic readers tend to adopt reading strategies involving increased visual scrutiny, making them more sensitive to pattern glare. Indeed, pattern glare can result from the physical characteristics of print,[44,45] and for dyslexic readers, reading may be a letter-mediated rather than a whole-word-mediated process.[46]

Meares also believed that certain patterns were responsible for visual stress. She believed that the white gaps between the letters, words, and lines were responsible for the perceptual anomalies, including words appearing to blur, to double, and to jump. She believed that the gaps formed patterns, which some children found aversive, and gave rise to visual stress.[39] More recent research suggests that she was correct; those sensitive to striped patterns are much more likely to suffer from visual stress and dyslexia.[47]

Fernandez and Wilkins found that images in art and nature having more power near 3 cycles per degree visual angle caused discomfort in some people.[48] They also found that sinusoidal gratings of this spatial frequency could trigger migraines[49] and, for those with photosensitive epilepsy, seizures.[50] The white gaps between lines of text form a striped pattern of a similar spatial frequency and can cause similar discomfort.[51] Additionally, the stripes from the letter strokes themselves

have a spatial frequency that fall within the range that causes perceptual distortion.

Those with pattern glare tend to have frequent headaches and migraines and report distortions involving motion, shape, and colour when exposed to gratings of a spatial frequency of 2 to 10 cycles per degree. For some, the same distortions seen in these gratings also manifest in text,[52] showing an obvious link between pattern glare, reading discomfort, and the perceptual distortions associated with visual stress and dyslexia. Evans, et al. found further empirical evidence implicating pattern glare in the aetiology of visual stress,[53] and more recent studies have excluded other potential mechanisms for the syndrome.

Hollis & Allen were very interested in this research and also found that people who read more quickly through coloured overlays (i.e., those with visual stress) see more distortions in striped patterns.[54] Other research has shown that blood oxygenation in the visual cortex is increased in response to stripes with spatial frequencies in the 2-5 cpd range, especially in area V2 of the brain, and that the difference is particularly large in those who have symptoms of visual stress.[55]

Coloured Overlays and Precision-tinted Lenses

Coloured overlays are sheets of coloured plastic that may be placed over text to facilitate reading in those with visual stress. Their mechanism of action is not fully understood, but their success in alleviating symptoms of visual stress is well documented, especially in children with headaches.[56] Precision-tinted lenses worn as spectacles are individually prescribed to those with visual stress following clinical tests to determine an individual's specific colour requirement. Wilkins et al. determined that the appropriate colour needs to be determined precisely for a given individual in order for their maximum rate of reading to be achieved.[57] This colour is selected after testing with appropriate instrumentation, such as the Intuitive Colorimeter or the Jordan Read-Eye. It is thought that yellow filters specifically may increase the responsiveness of the magnocellular system, which may otherwise be sluggish, by eliminating the inhibitory blue input from S-cones.[58] Indeed, yellow filters have been shown to improve convergence and accommodation in children with reading difficulties.[59] One randomised controlled trial showed that yellow filters significantly improved reading in dyslexic readers.[60] The same trial showed that blue filters can also be beneficial. This has been explained in terms of specialised photosensitive ganglion cells of the retina, which contain a pigment (melanopsin) that is responsive to blue light. These specialised cells terminate in the suprachiasmatic nucleus of the hypothalamus, the control centre of our circadian clock. During arousal, the magnocellular nerve cells are also stimulated. In addition to reading speed being increased, poor sleep patterns are reduced! Serendipitously, migraine headaches are also eased, perhaps due to the cortical pain system also being controlled by the suprachiasmatic nucleus. There are no theoretical explanations as to why other colours may facilitate reading in dyslexic readers, but the colours yellow and blue are insufficient for all dyslexic readers,[61] and it has been suggested that trial tints need to exceed 1000 colours of varied hues.[57] As such, there are various tinting systems in place for visual stress sufferers, which cover a multitude of colours, such as those employed in Irlen lenses or in Hoya's VDEX lenses.

Pattern Glare Test

The Pattern Glare test was produced in 2001 by Arnold Wilkins and Bruce Evans. It is an A5 laminated booklet consisting of three sinusoidal gratings, each of a particular spatial frequency. Assuming a working distance of 40cm, pattern 1 has a low spatial frequency of 0.3 cpd, pattern 2 has a spatial frequency of 2.3 cpd, and pattern 3 has a spatial frequency of 9.4 cpd. Pattern 2, having a spatial frequency close to that at which contrast sensitivity is greatest (i.e., 2-5 cycles per degree visual angle) and with each stripe subtending approximately 10 minutes of arc, would be expected to elicit the most perceptual distortion in those prone to pattern glare. Those prone to pattern glare would also be more likely to be affected by visual discomfort in everyday life and have visual stress. Conversely, where viewing pattern 3 provokes the most perceptual anomalies, an individual is likely to have low visual discomfort, and the nature of the distortions experienced may differ considerably from the nature of the distortions in the mid-spatial frequency grating. Evens and Stevenson determined normative values for pattern glare: people with an abnormal degree of pattern glare would report >3 distortions on the 3 cpd grating and >1 distortion on the '3-12 difference,' i.e., the difference in the number of perceptual distortions reported for pattern 2 and pattern 3.[62] A diagnosis of pattern glare can be made where one of these conditions is met.

Rate-of-Reading Tests

There are many rate-of-reading tests. The Wilkins Rate of Reading Test (WRRT) was designed specifically to assess the impact of coloured overlays on reading speed and is commonly used in clinical practice. The WRRT comprises four passages of text that have been designed to be visually stressful. Each passage consists of ten lines of fifteen randomly ordered words to eliminate syntactic and semantic cues. The fifteen words are of high frequency in English children's books and therefore are easily read by even those with weak vocabularies.[63] Participants in the test are asked to read the passages as quickly as possible without making errors. The number of correct words read in one minute (WPM) determines the rate of reading.

In research, other methods of assessing rate of reading are often employed. One method is the forced scrolling technique, wherein words are scrolled horizontally across a computer screen at an increasing rate until a participant is unable to read them. Similarly, another method is the Rapid Serial Visual Presentation (RSVP) test, wherein words are rapidly presented at one location on a screen. Again, the speed of presentation

is increased until the participant is no longer able to read the words.

Each technique described above produces different rates of reading. It is therefore important to consider this when critiquing research where more than one technique for determining rate of reading may have been used. For example, the RSVP testing gives a much higher reading speed than do scrolling methods.[64]

Critical Review

The Prevalence of Pattern Glare in Dyslexia

Due to there being no universally agreed definition of dyslexia and therefore its nature of being hard to diagnose, any research into its prevalence is open to criticism. Moreover, any attempts to link the condition to pattern glare can be met with equal opposition. Although aware of this, Evans et al. looked for correlations between pattern glare and dyslexia, testing 39 dyslexic children and 43 controls for the condition.[47] Both a 4.2 cpd grating and a 24 cpd grating were shown to the participants for 10 seconds, and seven predetermined questions were asked about visual anomalies. The anomalous visual effects reported were then summed. While the sum of the distortions reported by the dyslexic group viewing the 4.2 cpd grating was significantly higher than that of the control group, indicating pattern glare, the same held true for the 24 cpd grating. Since this pattern has a high spatial frequency, it should not have elicited any visual perceptual distortions in the dyslexic group. When this was taken into account, the results did not indicate any correlation between dyslexia and pattern glare; the dyslexic group was just as likely to have pattern glare as the control group. It is this author's opinion, however, that this finding is perhaps due to the way in which the pattern glare test was conducted. Indeed, Conlon believes that asking specific questions and then summing the perceived distortions is a very unreliable measure of pattern glare and that using a visual discomfort rating scale version of the Rasch model provides a more sensitive measure.[65] Conlon's research also found that those with moderate pattern glare may be more sensitive to higher frequencies than those with severe pattern glare. The study should be repeated using a pattern of very low spatial frequency as a control.

Conlon found that high scorers on the visual discomfort scale reported a great number of unpleasant effects when exposed to a 2.5 to 4 cpd square-wave pattern. Many unpleasant distortions were also reported when the high-scoring participants were asked to view a page of text with a global fundamental spatial frequency of 4 cpd. Conversely, those who had a low score of visual discomfort and no apparent pattern glare reported few discernible distortions. These findings directly correlate difficulties reading text of specific parameters with pattern glare. It can be further hypothesised that, in at least some cases of dyslexia, pattern glare may play at least a contributory role in the symptoms experienced by sufferers and their poorer reading ability.

Brown suggested that dyslexia is a heterogeneous condition.[66] Christenson et al., however, pointed out that studies involving dyslexia may include "a co-mingling of subjects who may have dyslexia and/or other causes of poor reading (e.g., vision problems and attention deficits),"[32] making such suggestions contentious. White et al. compared 23 dyslexic children with 22 control children matched for age and nonverbal IQ, assessing literacy in addition to phonological, visual, auditory, and motor abilities.[41] They found comorbidity between dyslexia and other problems, including eight with visual stress. Only five had literacy only problems. This number was not dissimilar to those found to have problems with phonology, perhaps suggesting multiple aetiologies of dyslexia. This author would, however, suggest one aetiology of dyslexia, citing dyslexia as a condition with diverse symptoms that may not always be present, which is only suggestive of multiple aetiologies. There may, however, be several causal factors in terms of reading difficulties.

Pattern glare is related to visual stress,[62] and it can be assumed that pattern glare is just as prevalent as visual stress in those considered to be dyslexic, approximately 35% according to White et al.[41] As mentioned above, in this particular study, 23 dyslexic children were tested. They were identified through a formal assessment by the Dyslexia Institute and had well-documented histories of reading disability. This hopefully allays any concerns regarding the definition of dyslexia and whether the children in the study were indeed dyslexic readers.

McConkie and Zola found that those with dyslexia are more sensitive to the global percept of the page and are unable to direct their visual attention sufficiently to the words in order to read fluently.[45] This can be used as an argument against the phonological theory of dyslexia and supports the notion that other, primarily visual, factors play a significant role in the reading difficulties associated with dyslexia. Further supporting this, studies have demonstrated that low-frequency words are more difficult to process,[67,68] showing a requirement for a very discerning visual attention. Those suffering visual deficits, perhaps particularly those concerned with pattern glare, would find this discernment difficult.

While Evans concluded that there was no correlation between pattern glare and those thought to be dyslexic,[62] it is this author's belief that this would not be the case when the changes discussed above were made to the study's methodology and where a greater number of dyslexic participants were investigated, specifically those of the varying subtypes of the condition. For example, the possibility of a bimodal distribution of flicker threshold in the dyslexic group was briefly discussed by Evans, although considered unlikely.[62]

Many experts seem to agree that phonological deficits are the causal factor of dyslexia, but this does not explain the cases of dyslexia where there are other isolated causal factors indicated, such as those seemingly of a cerebellar origin[69] or those concerned solely with the visual system.[70-72] This author believes that pattern glare could be cited as a causal factor in

dyslexia and would be found to be highly prevalent in dyslexia where it is tested for using the methodology Conlon proposes.

Eye Movements

Ocular motor abnormalities have also been suggested as a causal factor in dyslexia, and this author concurs. One study found orthogonal second saccade latencies in those with the condition,[73] and another found poor smooth pursuit movements.[74] The magnocellular theory of dyslexia would account for such ocular motor abnormalities. Stein explains that the magnocellular system is responsible for signaling the eyes to move at the correct time in order to read.[31] If there is a problem with this system, eye movements are affected, leading to a retinal slip, where the image of the text moves off the fovea. Reading would become impaired in this case.

These theories have been rebutted by subsequent research where eye movements were minimised. Yu et al. used flashcard techniques to determine reading speed rather than scrolling techniques.[75] Contrast sensitivity function, which also relates to pattern glare, has been put forward as an alternative theory. This would still, however, support a magnocellular aetiology.

The parallel letter recognition model is now accepted in terms of reading and proposes that we recognise the individual constituent letters of a word simultaneously, rather than the whole word or the individual letters separately, which was once thought to be the case.[76] The parallel letter recognition model is supported by research into eye movements, which shows that our eyes jump from word to word (saccades) rather than making a smooth movement.[77] Further research has shown that we usually fixate in the middle of a word[78] for roughly one-third of a second[79] before making a ballistic movement to the next word.

Depending on the size of the print and viewing distance, the fovea can accommodate approximately 10 letters with a single fixation.[80] This information, combined with parafoveal information, is used to determine the position of the next fixation. Occasionally, small words (e.g., "and") are skipped where they can be readily processed by means of textual context, a navigational process[81] whereby saccades permit the brain to develop an overall image of a page of text.[82] Anecdotally, the author of this paper believes that the same process may be involved in scanning faces and instantly recognising facial expressions, which may explain why in our clinics many dyslexic readers often struggle with facial and expression recognition and have symptoms of prosopagnosia and prosopometamorphopsia.

Where whole pages of text are processed in this fashion, readers will become increasingly susceptible to the effects of pattern glare, perhaps explaining why symptoms of visual stress and pattern glare are often not immediate and only become manifest after prolonged periods of reading. Dyslexic readers are also prone to these effects, and the magnocellular deficit theory may be used as an explanation. Specifically, this author believes that the dorsal cortical stream, to which the magnocellular neurons project, may be affected. Palmer et al. also believe that the dorsal pathway in the brain is concerned with the spatial selection and sequencing required for orthographic processing in visual word recognition.[83]

Crowding

Crowding is a well-documented phenomenon wherein the legibility of letters is diminished due to the presence of neighbouring letters in the peripheral visual field.[84] When flanking letters, for example, are too close to the target letter, it is harder to identify that letter.

Martelli et al. showed that crowding accounts for 60% of the reading slowness in dyslexic readers.[85] Children with dyslexia can be more susceptible than others to the effects of crowding.[86] Bouma and Legein[87] and Moores et al.[88] substantiated this finding, the latter additionally finding that an increased number of distractors affect attention in those with dyslexia, further hindering reading. Researchers have been unable to explain these findings fully, which this author believes can also be explained in terms of dorsal processing. This theory derives from the idea that the dorsal stream controls attentional mechanisms,[89] although this view would be vehemently rejected by Skottun and Skoyles.[90]

Despite their criticisms, the belief is held that crowding (and other distracting phenomena) will be of greater significance to readers with dyslexia due to compromised processing. Dorsal stream disorders themselves may lead to an extreme form of crowding.[91] Indeed, inadequate dorsal processing would lead to an inability to process spatial attention information as readily as it is for those without a dorsal processing disorder.

Other Comorbidities

In addition to dyslexia, visual stress, and pattern glare, there are other possible comorbidities, including frontal headaches and migraine, allergies and sensitivities, and even some types of stomach problems. This is based on empirical data. This author has also found that, when specifically asked, a large number of dyslexic readers complain of problems with their jaw or with mastication. This would suggest that, in at least some cases of dyslexia, the trigeminal nerve could be affected. The vagus nerve may be indicated where there are stomach problems, and it could be hypothesised that dorsal stream impairment may lead to problems with one or both of these nerves. Of course, this is based solely on this author's own observations, and hypotheses would need to be developed and tested. It could be that the dorsal stream carries information from a greater number of senses than is currently thought to be the case, perhaps with each of the senses having their own carrier wave.

By far, the most common comorbidity is with visual processing. Specifically, when tested using a medical pendulum, visual timing seems to be implicated in nearly all cases of reading difficulties associated with dyslexia. It is thought that visual timing is, at least to some degree,

mediated by the dorsal stream,[92] further implicating a dorsal stream disorder in the aetiology of dyslexia.

Other comorbidities may be synesthesia (perhaps where the aforementioned carrier waves have incorrect modulations), simultanagnosia, tinnitus, changes in auditory volume as someone talks, reduced convergence and accommodation, and hemispatial neglect. A disproportionate number of patients with dyslexia also complain of micropsia, macropsia, pelopsia, or teleopsia, suggestive of Alice in Wonderland syndrome.

Many of these comorbidities seem to be linked in terms of a magnocellular and/or dorsal stream processing disorder. Indeed, many of these conditions are found in stroke patients or in those with traumatic brain injuries, where one of these pathways is thought to be affected.

The Pulfrich Phenomenon and Visual Timing

The Pulfrich phenomenon describes the situation when the visual signal from one eye is processed at a different speed than that of the other. This phenomenon can be observed in patients with cataract, for example, but it can also be thought to occur spontaneously. In this author's experience, this spontaneous Pulfrich seems to be prevalent in virtually all cases of reading impairment thought to be caused by dyslexia.

This difference in visual timing between the two eyes can be significant, causing problems with movement, distortion of moving images, and difficulty determining speed of movement, and it may be a factor in strabismus and amblyopia. Since 1922, it has been known that changing the visual stimulus through tinted lenses alters visual timing, suggesting a reason why tinted lenses may be beneficial to those dyslexic readers with visual timing problems contributing to their symptoms.

Cross-sensory problems

In clinical situations, this author has found that visual processing is sometimes out of sync with other sensory processing and that this asynchrony is not unusual in dyslexia. Indeed, Sela affirmed that visual and auditory processing cross-modal integration is asynchronistic among dyslexic readers.[93]

This can cause the mistiming of hearing, which may explain phonological aspects. Lip-sync issues can cause issues with concentration, and the McGurk effect, in which speech is perceived as distorted, may explain problems with comprehension and language learning.

Visual timing may be linked to mapping problems, wherein dyslexic readers see things in the wrong position, which would explain why letters and words may displace, change sequence, or reverse and invert, all of which are symptoms of visual stress. Adequate mapping is also the basis of movement control both in terms of eye movements, which may explain why dyslexic readers have poor and often asymmetric convergence and reduced accommodation, and hand-eye co-ordination, which may explain clumsiness, especially since balance and the perception of ground may be affected. There could also be a contributory lower field defect.

Discussion

Dyslexia, Visual Stress, and Pattern Glare

Dyslexia has a number of opposing definitions, but many cite some aspect of reading disability. The term dyslexia itself refers to difficulty with words. Many subtypes of dyslexia have been postulated over many decades of research, and the condition is now considered to be heterogeneous, perhaps with multiple aetiologies. It is therefore difficult to diagnose.

The author of this paper challenges the current definitions of dyslexia and some of the ideas held by researchers, believing the term dyslexia itself to be a misnomer. It is possible that the vast majority of the symptoms of dyslexia can be explained in terms of a magnocellular processing disorder, a notion that is supported by multiple post-mortem studies. The dorsal stream, which receives significant input from the magnocellular pathway, may also be implicated. Indeed, the dorsal stream is highly involved in lexical recognition.[94] Since the dorsal stream is involved in many cortical functions, including those concerned with attention, eye movement, and auditory processing, a magnitude of likely comorbidities become explained. It can be argued, therefore, that a dorsal stream disorder may be a causative factor in dyslexia. Dorsal Stream Dysfunction (DSD) may be a more appropriate diagnosis than dyslexia in many cases, a term which seems somewhat narrow and restricted. It is this author's belief that DSD can be said to account for all of the symptoms encountered by the vast majority of dyslexic readers, encompassing all subtypes. It can also be argued that visual stress and dyslexia are synonymous, while accepting that not all of those diagnosed with dyslexia have the symptoms traditionally associated with visual stress (i.e., visual perceptual distortions), and vice versa.

It is proposed that dyslexia or DSD is a spectrum. The capability of the dorsal stream is on a continuum, and genetic and environmental factors play a role in determining an individual's position on this continuum; some will have adequately functioning dorsal streams and be asymptomatic, whereas others will not. Without adequate dorsal stream processing, individuals may develop a range of symptoms, some of which would be traditionally associated with dyslexia.

Similarly, DSD may be implicated in migraineurs and those who suffer with pattern glare. Pattern glare can affect many people to some degree, but only a proportion of these will have a severe aversion to patterns, particularly those patterns that fall within the 3-5 cpd range. This is the range where contrast sensitivity is greatest, which also seems to support the notion that the magnocellular pathway and dorsal stream are affected.

A link between dyslexia, visual stress, and pattern glare has already been shown, and it is this author's belief that the three are in fact synonymous, i.e., symptoms of the same condition, a spectrum disorder concerned with the dorsal stream. Not all symptoms may be present in any given condition; an individual may have DSD but not find 3-5 cpd patterns aversive, for example.

Evans et al. failed to correlate pattern glare and dyslexia[47] but may have had more success where a rating scale version of the Rasch model was used as Conlon proposed.[65] Pattern glare may be just one symptom of dyslexia and could be expected to be particularly prevalent in those with the visual perceptual distortions associated with visual stress. Visual stress itself has been shown to account for 35% of dyslexic readers.[41] It could be that 35% of those with DSD have the symptoms commonly associated with visual stress.

It is hoped that future research will firmly establish the possibility of a common aetiology for dyslexia, visual stress, and pattern glare, which may be possible with advancements in technology. Specifically, brain imaging may prove advantageous.

General Discussion

Dyslexia, visual stress, and pattern glare all relate to one another and are certainly not mutually exclusive conditions. Indeed, it is this author's belief that their aetiologies are all the same: magnocellular and/or dorsal stream dysfunction. Symptoms of such dysfunctions are wide-ranging, and it can be argued that pattern glare is one manifestation of the condition. Pattern glare is perhaps a causative factor in some of the reading difficulties associated with dyslexia. Several other apparent comorbidities have also been explained in terms of the dorsal stream. There may be a carrier wave system in place for all the senses, which can become confused. This may be particularly true in cases of synaesthesia or visual snow.

Campbell and Robson identified many channels in the visual system that link to neurons in area V1 of the brain that have specific sensitivity to certain frequencies and orientations.[95] They propose that a mechanism in the brain must exist that collates all the information, integrating it, and that there are different channels used for letter identification than the channels used for the detection of gratings, which have no orientation tuning. This is a concept that is supported by research conducted by Solomon and Pelli,[96] but additional research needs to be conducted in order fully to understand these channels and any such mechanisms. Olzak and Thomas identified channels for both frequency and orientation,[97] but since letter recognition requires integration of information across these channels (i.e., both frequency and orientation information), there must be integral processes involved in the brain that we do not yet fully understand.

It is postulated that information from magnocellular and parvocellular cells is communicated to the dorsal and ventral streams in varying amounts, facilitating integration in V1. The dorsal stream is mostly concerned with magnocellular input, and where DSD exists, integration would be rendered difficult, causing symptoms commonly associated with dyslexia and other comorbid conditions. Such symptoms would include pattern glare, since grating information is largely processed by the magnocellular pathway and dorsal stream, regardless of any integration system that may be involved in higher visual function.

It has been found that coloured lenses can help in some cases of dyslexia. While current scientific knowledge can explain the use of blue and yellow filters, in terms of the magnocellular pathway and S-cone functioning, it is undeniable that other colours prove more beneficial for some dyslexic readers. Upwards of 1000 colours need to be tested to find the precise colour that works for any individual. It could be that stimulating the three cones in different ways through the use of different colour hues may stabilise the visual system somehow in a way that science does not yet understand. It could be that higher brain processes are involved. Future research will determine whether this is the case and to what extent.

Conclusions

There are a great number of conflicting opinions regarding dyslexia, with regard to both its definition and aetiology. The condition may be seen as being heterogeneous, perhaps with multiple aetiologies, or as a name given to a collection of symptoms that this author feels are caused by magnocellular pathway deficits and/or a dorsal stream disorder. The belief is held that dyslexia, visual stress, and pattern glare are synonymous and that DSD may be a more appropriate diagnosis, at least in the vast majority of cases. Trigeminal and vagus nerve problems may result from DSD, which may provide an explanation for some apparent comorbidities. Indeed, the dorsal stream may process more information than currently understood, and it is possible that carrier waves compartmentalise information, perhaps sometimes unsuccessfully.

The exact processes that are concerned with dyslexia are not yet fully understood. It is hoped that with advances in scientific technology, future research will help us develop a fuller understanding of dyslexia, its aetiology, and any possible treatments. Until then, dyslexia shall remain an elusive condition and an area of great interest and intensive study.

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Correspondence regarding this article should be emailed to Robert Longhurst, BSc (Hons), MCOptom, at longhurr@aston.ac.uk.

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The online version of this article contains digital enhancements.

Appendix A

Double Deficit
Deficit in grapheme-phoneme integration and the ability to recall the correct word quickly

Dyscalculia
Difficulty with mathematics

Dyseidesia
Deficit in the ability to perceive whole words as visual gestalts and match with auditory gestalts

Dysnemkineidesia
Deficit in the ability to develop motor gestalts for written symbols and in the ability to perceive whole words as visual gestalts and match with auditory gestalts

Dysnemkinesia
Deficit in the ability to develop motor gestalts for written symbols

Dysnemkinphoneidesia
Deficit in the ability to develop motor gestalts for written symbols, in grapheme-phoneme integration, and in perceiving whole words as visual gestalts and matching with auditory gestalts

Dysnemkinphonesia
Deficit in the ability to develop motor gestalts for written symbols and in grapheme-phoneme integration

Dysnomia
Deficit in the ability to recall the correct word quickly

Dysphoneidesia
Deficit in grapheme-phoneme integration and in the ability to perceive whole words as visual gestalts and match with auditory gestalts

Dysphonesia
Deficit in grapheme-phoneme integration

Fixation
Visual gaze on a single location

Grapheme
A letter of the alphabet

Magno cells
Magnocellular, M-cells, or magno cells; neurons located within the magnocellular layer of the lateral geniculate nucleus of the thalamus

Parvo cells
Parvocellular, P-cells, or parvo cells; neurons located within the parvocellular layers of the lateral geniculate nucleus of the thalamus

Phoneme
The constituent sounds of words

Saccade
The fast, jerky movement of the eye from one fixation to another

Synesthesia
A neurological problem in which stimulation of one sensory or cognitive pathway leads to automatic, involuntary experiences in a second sensory or cognitive pathway

Visual snow
A symptom of seeing snow or television-like static in parts or the whole of the visual field