Physical Coordination and Dyslexia

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Introduction

A number of years ago I taught Jeremy, an intelligent, enthusiastic seven-year old. His writing and reading were well below his chronological age level. He also seemed to be physically awkward, which hampered his participation in sports. Arising from this teaching experience I want to examine the connections between dyslexia and physical coordination, by reviewing literature from neurology and neuropsychology. Neurology is the scientific study, or knowledge of the nerves and the nervous system. Neuropsychology describes how the neurological organization of the brain influences the way people think, feel and act.

Typically, between 5% and 20% of youngsters have poor physical coordination. Among children with learning disabilities this figure rises to between 18% and 60% (Cratty, 1996). "Dyslexia is a specific inability to learn to read at an age-appropriate level despite adequate opportunity, training and intelligence." It affects approximately 5% of people (Banich, 1997 p.476). Denckla (1985) reminds us that coordination can refer to ability in sports or to more specific, clinical tests (such as copying an image). She finds minimal correlation between these two types of coordination. In this paper I am interested in connections between any kind of physical coordination and dyslexia.

Neurology

Predictably, one of the most common correlates of development dyslexia is language impairment. However, non-linguistic correlates of dyslexia have also been found. Denckla (1985) found a high level of perceptual-motor and fine motor impairments in a large group of dyslexic children.

However, Denckla subsequently changed her view about the links between motor skill deficits and dyslexia (Fawcett, Nicolson and Dean, 1996). This contributed to the now widely accepted phonological explanation of dyslexia. The phonological model or phonological deficit hypothesis is based on the premise that children with dyslexia have difficulty in understanding the phonemic structure of spoken words. This impairs decoding and makes word identification difficult. Based on functional magnetic resonance imaging (fMRI), Shaywitz (1996) suggests a 'tentative neural architecture' for reading a printed word. She identifies three stages involved in reading:

(a) Letter Identification. This takes place in the extrastriate cortex within the occipital lobe (see image, adapted from Gazzaniga et al. - missing).

(b) Phonological Processing (converting letters into the corresponding phonemes (language sounds)). This takes place in the inferior frontal gyrus (left hemisphere only for men, both hemispheres for women)

(c) Making meaning. This takes place in the middle and superior temporal gyri of the brain.

She sees the process as hierarchical where meaning cannot be made if there is a deficit at level (a) or (b). This is consistent with the view that pupils with dyslexia could comprehend text if they could decode it. According to this model, reading is a function of the cerebral cortex.

However, the phonological explanation does not consider a number of features of people with dyslexia. These include problems in visual processing, reduced information processing speed and impairments in "automatic balance" (cited in Fawcett et al. 1996). The role of the cerebellum in modulating muscles, controlling speech, posture, the eyes and other functions has been well documented (e.g. Banich, 1997). This makes it an obvious place to start looking when considering links between physical coordination and dyslexia. Brain imaging studies which show that the cerebellum is more active during cognitive tasks, such as coming up with a verb to match a noun, than when simply repeating a verb, have inspired debate about the role of the cerebellum in cognition (Barinaga 1996).

Fawcett et al. (1996) hypothesize on a possible link between cerebellar damage and dyslexia. Typical symptoms of cerebellar dysfunction are dystonia (problems with muscle tone) and ataxia (disturbance in posture). Standard theories of dyslexia don't predict difficulty in these tasks. Using clinical cerebellar tests, Fawcett and Nicolson (1999) found that over 95% of children with dyslexia have weaknesses in either postural stability or muscle tone or both. While this implicates the cerebellum in reading difficulties, the authors say that it is premature to locate the difficulties of dyslexic children in the cerebellum alone.

Fawcett and Nicolson(1992) cite another reason to move the dyslexia research focus from phonological issues alone, to those of wider cognition. In a study of 49 15-year-old and 11-year-old children (dyslexic and normal), those with dyslexia performed worse than controls on balance and on a combination of balancing and performing a task. The authors interpret this as a general learning deficit where children with dyslexia have a problem in making a task 'automatic' ('automatization deficit') and they say that phonological problems may be a symptom of this deficit.

Using MRI we can attempt to explain the unpredicted cerebellar dysfunctions in dyslexic individuals. Schmahmann uses MRI images to show that the cerebellum's role is greater than its traditional motor role. He claims that it may contribute to sensory, affective, autonomic and cognitive functions, as well as motor functions. Whereas Schmahmann studies the general picture, Rae et al.(1998) obtained magnetic resonance spectra from 14 dyslexic men (aged 20-41) and 15 controls. They found that dyslexic men have a higher neuronal density in the right cerebellum hemisphere and a lower neuronal density than controls in the left cerebellum. This finding also implicates the cerebellum in dyslexic dysfunction.

In other research, Wolff, Michel, Ovrut and Drake (1990) cite a 1937 hypothesis that speech and skilled movements (such as bimanual coordination) share neural mechanisms of timing precision and serial order control. More specifically

dyslexic children have significantly greater difficulty than .. normal readers in maintaining the correct tempo, prosody and rhythm in language, reading and writing, and other skilled manual actions (p.349)

These skills require timing precision, serial ordering and response frequency and are collectively described by Wolff et al. as "temporal resolution."

In a study,Wolff (1993) tested bimanual coordination in 230 children, adolescents and adults with dyslexia. Although the findings were not equally discriminating at each age, dyslexic individuals had greater variation in inter-response intervals (IRI) than the age-matched controls had. Ultimately, he concludes that the hierarchical models are inadequate to explain the findings. He believes that to understand the 'complex behavioral variations' in dyslexia, a macroscopic rather than a microscopic view of brain functions must be taken and that it is not helpful to localize the temporal resolution deficit in any specific area of the nervous system.

Insights gained from MRI studies of adults and children with dyslexia suggest another possible cause for, my pupil Jeremy's, awkwardness when playing sports. Eden and Zeffiro (1998) show that PET and MRI studies have identified the Middle Temporal/Visual Cortex Area 5 (MT/V5 Complex) to be sensitive to motion. Banich locates the motion sensitive cells at the superior temporal gyrus (at the border of the parietal, temporal and occipital regions).

In the first study that shows a link between individual differences in brain activity and human motion perception, Demb et al (1998) found that when a group of dyslexic subjects were presented with moving stimuli their fMRI responses in the primary visual cortex and several extrastriate areas were significantly lower than control group responses. This suggests difficulties for dyslexic pupils in playing ball sports. This deficit happened in response to low mean lighting with stimuli of various contrasts. Interestingly, the dyslexic subjects were as good as or better than controls in response to "contrast-reversing" stimuli with higher lighting. This finding is consistent with a deficit in the magnocellular pathway. This strengthens Rae et al's findings that dyslexic individuals have such a deficit. The magnocellular layer is part of the lateral geniculate nucleus (LGN) which is located in one of the most complex relay centers of the brain, the thalamus. It is extremely sensitive to low light levels.

Linking this with other findings, the magnocellular system is associated with high temporal resolution. The MT/V5 complex provides input to the inferior parietal cortex and cerebellum, areas that are involved in visual motion processing. The areas mentioned here for motion perception, superior temporal gyrus and the extrastriate cortex overlap with Shaywitz's areas for phonological processing.

Neuropsychology

Wolff(1993) highlights the fact that although the individual skill components of reading can be broken up and even remediated with dyslexic children, severely impaired dyslexic pupils, typically, still have problems in organizing these skill elements into a complete task. It seems that the coordination failures of dyslexic pupils, in his study, to perform activities requiring bimanual coordination, are due to a combination of timing precision, serial ordering and response frequency factors. Wolff speculates that children with dyslexia may face their biggest challenge in integrating individual skills.

In addition, Shaywitz suggests that material should be presented in a meaningful way to pupils with dyslexia. Pupils with dyslexia typically have significant strengths in reasoning,

problem solving, concept formation, critical thinking and vocabulary. However, details must be unified by associated ideas or theoretical frameworks. Rote memorization, rapid word retrieval and multiple choice exams are particularly difficult for pupils with dyslexia.

In support of their argument and to link it to the phonological model, Fawcett et al.(1996) explain that cerebellar impairment would cause problems in young children learning to speak. For example, a child who is slow to develop control of speech muscles, will be less fluent in speaking, then articulation will take up more conscious resources and fewer resources will be available to process the auditory, phonemic structure of the words. This will cause a delay in the child's development of phonological awareness. They also point out that the cerebellar theory would explain why most children with dyslexia have such bad handwriting. It may also explain why spelling, which requires the combination of phonological skill and motor output, is "particularly resistant to remediation" (Fawcett et al., 1996 p.279).

Because weaknesses in the cerebellum seem to be so highly correlated with dyslexia, and cerebellar tasks also tap natural skills (as opposed to taught skills), much earlier diagnosis of dyslexia in young children is possible (Fawcett & Nicolson 1999).

Conclusion

The phonological model for explaining dyslexia is the dominant approach in current literature. This adopts a hierarchical view of the reading process and locates the process of reading in the cerebral hemispheres (left for men; right and left for women). Recent fMRI studies show that the magnocellular pathway in the LGN of dyslexic adults seems to be affected. Imaging studies also seem to suggest that the cerebellum is involved in reading, perhaps in coordinating tasks. Pupils with dyslexia seem to have an automatization deficit which is applicable to all areas of their learning. It seems that there is a relationship between movement and dyslexia. But, just as no single part of the brain is involved in motion, this is also true for reading. One caveat is that, because Demb's study took place with adults we are looking at compensated dyslexics. It points to a direction that future MRI studies of dyslexic children may take.

I have found no concrete evidence of physical therapy 'easing' dyslexia. The area is far too complex to suggest any direct causal link between physical coordination and reading or vice versa (Wolff, 1993). In fact, it is important to take a 'macroscopic' view to research into dyslexia, which includes considering the phonological model, the cerebellum, automatization deficit and the magnocellular pathway. The magnocellular finding suggests that the levels of luminance and of contrast in a stimulus will influence how it is processed. Higher luminance stimuli seem easier for dyslexic individuals to process. In teaching children with dyslexia, material should be presented in a rich, meaningful context.

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