

## **Dyslexia and Neurobiology**

### **The anatomy of Reading: The Eye and the Brain Connection.**

*“For decades, it has been well established that one child in five has serious reading difficulties. Many systems of special teaching have been tried. This book details a new revolutionary concept together with a new curative therapy.*

*To put the theory in its simplest terms, systems of reading and language show up when there is conflict between one side of the brain and the other to gain language dominance. In the human brain the language area is usually located in either the left or the right side of the brain. Likewise, man is either left handed or right handed. Normally, dominance in the brain begins as soon as a baby learns to speak, and theoretically missing any stage of development between creeping and walking and seeing and writing creates a problem in reading.*

*Accordingly, for the poor reader, the method in this book suggests re-imprinting the early stages of brain dominance, by repeating in the brain the early steps in creeping and crawling and other physical exercises that develop the receptivity of the language area of the brain.”*

“A New Start for the Child with Reading Problems” 1970.

At the British Association for the Advancement of Science, Festival of Science in York, 2007, Professor Simon Liversedge from the University of Southampton revealed that after research into the mechanics of reading, the link was established between the way the eyes worked in conjunction with the brain for the brain to fuse the different images from the eyes together. He concludes that the ocular system uses information from both eyes, rather than the brain suppressing one image and only processing the other. Professor Liversedge reports on his work that, “ A comprehensive understanding of the *psychological* processes underlying reading is vital if we are to develop better methods of teaching children to read and offer remedial treatments for those with reading disorders such as dyslexia”.

The key to understanding of the underlying problems of why so many children fail to read to their age levels is to accept that when children are able to it is to be assumed that all the neurological connections between the sensory gathering system ,eyes, are correctly connected to the central nervous system . Conversely where there exists an inability to read, it must be assumed that a lesion exists in those areas of the brain which control language comprehension, the angular gyrus. It also means that we may have to assume that any part of the visual system from the eye to the macular in both the left visual cortex and the right visual cortex (areas 17) have been compromised as a result of neural impairment.

In their paper” Biological basis of Dyslexia: A maturing perspective”, authors Saviour and Ramachandra working out of the University of Mysore Human Genetics Laboratory and published in Current Science, Vol.90, No 2, 25 January 2006, accessed [www.ias.ac.in/currsci/Jan252006/168.pdf](http://www.ias.ac.in/currsci/Jan252006/168.pdf) , suggests, that “substantial evidence from multidisciplinary research suggests that dyslexia is a disorder of genetic origin with a basis in the brain. Many genetic studies indicated that different loci are involved in genetic predisposition of dyslexia”. The authors conclude in their synopsis that “identification of the specific genetic variants may bring a comprehensive explanation of the etiology of Dyslexia”.

The main body of the paper goes on to discuss the Genetics of Dyslexia, to expand their suppositions of subtle brain anomalies associated with Dyslexia, as well as discussing Cerebella theory of Dyslexia and Magnocellular theory of Dyslexia to explain the possible etiology of Dyslexia.

In the case for Cerebella Theory the authors cite” indirect evidences of cerebellar dysfunction in dyslexics which include delayed motor milestones such as crawling, walking and a characteristic clumsiness” further suggesting “ morphological and metabolic alterations in the cerebellum of dyslexics which relate to reading skills, motor skills and handedness. Cerebellar activity is ipsilateral to handedness, and right-handed people have a larger proportion of the right cerebella grey matter”.

A more in depth analysis of the case for Cerebella theory can be found in [www.theautismcentre.co.uk](http://www.theautismcentre.co.uk) article “Reading and the Autistic Child”.

The case for magnocellular theory of dyslexia is proposed on the evidence that “Psychophysical and anatomical studies provide increasing evidence that 75% of dyslexics exhibit visual processing abnormalities that may confine to particular portions of the visual system”, the authors going on to say that

“Magnocellular dysfunction is not restricted to the visual pathways but also in the other sensory modalities, auditory, tactile motor and phonological abilities”.

On this basis remedial action for reading problems requires a multisensory approach, reading article in [www.theautismcentre.co.uk](http://www.theautismcentre.co.uk) published June 2005 entitled “The 3 R’s Reading Righting Rehabilitation”.

In 1970 Dr Carl Delacato published his book “A new start for the child with reading problems—a manual for parents” ISBN 0-679-50765-5, which proposes the rationale for rehabilitation on the evidence suggested by the authors of this January 2006 paper being referred to in this document, a forerunner to today’s “Movement Therapy”

The following extract from Dr Antonio Parisi’s book “Children who do not look you in the eye—The secrets of autistic behaviour” outlines the mechanics of the workings of the visual system to reinforce the case for magnocellular theory relating to dysfunction, “Making sense of the senses”

“Children are able to juggle objects, defy gravity. But what was most astonishing is the fact that they seemed to pay no attention whatsoever to what he was doing during these moments. Their eyes are elsewhere. Children spend hours in this way, sometimes throwing things across the room or from the balcony, or leaving them neatly arranged at the edge of the table. It is amazing the deceptive nature of the behavior of autistic children. That way of looking askance at things was not a sign of lack of interest, but the only way children could actually see things. By looking out of the corner of their eyes they perceived the object. The juggling act was actually a way of focusing on it, seeing it better.

For many autistic children the only way they can see is to move their heads sideways and look out of the corner of the eye.

The visual apparatus of human beings is quite complex: essentially it involves breaking elements down into separate entities and then recomposing the picture as it was. Of course we are not aware that this is what we do. Every time we look at an object, a person, the scenery, a group of people, neurons analyze color, form, depth and motion. The neurons that recognize color are all located in one area of the brain, and so on, for other functions.

There are five such vision areas in the brain that for simplicity’s sake we can call V1, V2, V3, V4 and V5.

The first area, V1, is also known as the layered or primary area. This area receives the images falling on the retina. These images are then passed to V2, which links up to the other areas making up the area of associative vision, or the pre-layered cortex.

Area V3 receives information from V1 and V2 concerning form. Areas V4 and V5 also receive information from V1 and V2, but V4 concerns form and color. While V5 concerns depth and movement.

Once V3, V4 and V5 have done their jobs, the message is sent back to V1 and V2 along pathways that integrate the information, so the image is recomposed.

It is as if the elements of a picture were poured into a funnel. Everything goes through the neck of the funnel and mixes together: color, form, depth and movement.

In order to see the world normally these areas must be perfectly balanced; any imbalance can cause distorted vision.

Serious damage in the V1 and V2 areas causes total blindness while damage in the pre-layered area (V3, V4 and V5) causes difficulties in perceiving form.

One of the world’s leading experts on vision in human beings, Semir Zeki, has noticed that people with brain injury in the pre-layered area, who therefore have difficulty identifying forms, tend to rotate their heads in order to create movement, or prefer objects which move such as objects seen on television. Typical behavior of autistic children is to tilt the head in order to look at things out of the corner of the eye. Another is to frenetically rock or rotate an object, or to watch television programs featuring things in rapid motion, such as the list of characters, actors and crew at the end of a film. They also like to see the same scene of a cartoon over and over again, or watch quiz shows like “The Price is Right” or “Wheel of Fortune.”

A lot of evidence concerning autistic children points to injury of the pre-layered cortex. This is not necessarily wrong, but there is also evidence of other types of disturbance such as the inability to distinguish between similar images, or to see small things on an object’s surface or to remember a visual experience such as scenery or the route along a road.

It is therefore more correct to say that autistic children process visual inputs differently. This different perception may be associated with injury in the occipital lobe (the area related to vision) and may involve one or more areas of vision.

A slight but widespread injury at an early age may not have serious immediate consequences but certainly alters the neurological organization of the central nervous system. To make things simpler we could say that the brain of a newborn child is like a building site where the engineer is running things. An injury is like a construction event that won’t change the scaffolding but causes all of the engineer’s orders to be carried out slightly wrong, with consequences that may make the building unsteady or unusable. It is as if the engineer were speaking a slightly different language from the builders following his orders. The inputs are skewed, making the building out of shape. The neurons—the builders in our metaphor—simply

carry out orders as best they can, building things the way they seem to have been told. The result in autistic children is a series of neurological maps that don't correspond with the real world, giving rise to antisocial and unusual behavior. In the case of vision, only VI has an efficient neurological organization at birth. This means that the baby uses this area immediately in order to see. But it also means that this area is the most exposed to injury during pregnancy. In addition, the primary visual cortex is just like the engineer on the building site: all other areas of vision are organized by VI despite the fact that the messages go through V2. If the orders coming from VI are in this rather odd language the rest of the visual apparatus is built wrong.

Lack of perception of color could be caused by injury to the specific area dealing with color perception (V4) or to the primary visual cortex (VI) failing to give the right messages to V4. If the only defect in vision were the perception of color, it would probably be correct to assume the injury has occurred in V4. But autistic children generally have many defects of vision, so it is far more likely that VI has been injured and the entire language of the engineer is off-cue. Usually V4 is unlikely to be injured since autistic children generally receive injury during pregnancy or in the first few months of life, and V4 practically does not exist at this time.

So the hypothesis is that autistic children receive injury to VI before or just after birth. Now we need to explain how this determines their behavior.

Let us recap what we know about the primary visual cortex: first, it works from birth. Second, it receives all visual inputs. Third, it communicates with all other areas. Fourth, serious injury causes total blindness. Now we can go backward over these points. Autistic children are very rarely blind usually their injury is not severe. The layered visual cortex (VI) sends information to all other areas, determining the way in which these areas develop. In order to do this, the neurons in VI must be able to recognize each visual input and understand where to send it. The nerve cells in VI that recognize a stimulus are contained in the layer. Nerve cells responding to movement are found in sub-layer IV c a, the cells recognizing forms in sub-layer IV c p and other cells responding to color and spatial orientation in layers II and III.

The presence of nerve cells responding to all types of visual stimulus in VI means, this area of the brain is independent. In other words, with this area alone we can perceive forms, colors and movement. But this does not make for proper vision. We would find it difficult to focus on nearby objects, would see few differences in color and would only be able to see the outline of an object by moving it. We would see like a newborn child who only has the VI area of the brain available for seeing. But in the case of a newborn child, vision improves as the functions of the pre-layered cortex come into play.

The pre-layered cortex receives its orders from VI, which is organized in layers, each one responding to a particular stimulus and each transmitting input to other areas. The nerve cells in these areas activate a series of synapses that excite and inhibit, forming a kind of circuitry.

One of the circuits called the magnocellular circuit, transports information concerning depth and movement. Another called the parvocellular circuit, transports information concerning form and color.

The magnocellular circuit starts with some rather large ganglial cells (hence the name) connected to cells on the retina that recognize movement. The signal is then projected onto the VI sub-layer IV Cc a, where spiny star-shaped nerve cells respond to the stimulus, activating an exciter synapse. The message processed by these cells is then transmitted via V2 to V5 where specialist neurons further analyze the message.

The parvocellular circuit starts with some rather small ganglial cells (hence the name) connected to cells on the retina that recognize form and color. The signal related to form is projected to the sub-layer of VI, IV c p, and to some cells in layers II and III where the remaining cells respond only to color. The messages processed by these sub-layers are sent to V3 and V4 respectively.

The cells contained in sub-layer IVC b are exciters, but those in layers II and III excite a group of cells contained in layer VI of VI—smooth star-shaped nerve cells—which inhibit and modulate the effect of exciter cells. Two observations can be made at this point. The first is that the pathway carrying messages about movement and the pathway carrying a message about color and form are completely separate. The second observation concerns the nature of some cells in VI: they are generally exciter cells but are regulated by inhibitor cells. The message that is finally sent back to VI is the result of the balance between these two types of cells. If the pathways are separate, one may be injured without affecting the other. For example the parvocellular circuit may be damaged but the magnocellular circuit may be intact.

This is probably what occurs in autistic children.

The magnocellular circuit is the first to develop; the receptors or rods at the edge of the retina develop and detect movement. The ganglial cells creating the magnocellular circuit start from there. Autistic children seem to be able to detect movement perfectly well, almost too well. So it is unlikely that this circuit has been damaged. It is more probable that the processing of the image in the parvocellular circuit is affected.

The balance between inhibitor and exciter cells is very delicate. A slight injury to the visual cortex can have serious consequences. For example, if these cells are not perfectly balanced, the information processed in sub-layer IV c P and in layers II and III concerning the form and outline of objects, affects perception. The alteration is transmitted to V3, which then works on faulty input. It may be that V3 receives excessively detailed information about parts of an object, without providing the outline, so that the child is bombarded with information about single parts of objects without perceiving them as wholes. Autistic children with this vision

defect recognize people not from their faces but by the shape of their ears or their shoes. They are also able to notice tiny changes in two almost identical figures and to reproduce images effortlessly.

They can do jigsaw puzzles at a very astonishing speed and notice a tiny crumb on the floor.

Conversely, the effect of imbalance between inhibitor and exciter cells in the parvicellular circuit, may also send too little information to V3, so shapes are indefinite. Autistic children of this sort need to use special strategies to distinguish shapes.

It is well known that to distinguish the outline of an object motion is helpful. Autistic children are masters of this. Similarly, a child may place things along the edge of a table in order to make the edge clearer or touch things with the tips of the fingers. Another method is to create strong color contrasts. The way children played with saliva in front of the window was a way of creating light contrasts. With hands shade is created that fell over the eyes; the saliva had a prismatic effect, separating colors.

The way children continuously move their hands is a torment for parents. Even if they kept hands relatively still, they continue playing with things around him—a pen, a piece of paper, a leaf, to rock in front of their eyes.

Autistic children have problems perceiving depth. Every time they come close to a carpet they seem to raise their feet as though they were about to climb onto a step. A lack of depth perception, i.e. the inability to see how far away things are, creates enormous difficulties. It is like walking in mid-air, without having any idea of how far away the floor or the wall is. It makes stairs difficult to climb and the ceiling appearing to fall. Children with this difficulty often trip over colors and designs. One of the consequences of this problem is that in large open spaces autistic children often scream in order to receive an echo and calculate the distance to the walls. Or they throw things to see when they impact with the wall.

Our sense of depth is a complex process that requires the combination of information from both eyes. The information is sent from VI to V5 but it would be mistaken to think that V5 is the area where the information is definitively processed. Depth information has nothing to do with objects in motion. On the contrary, it often requires the use of a distant, stationary object. An injury to the primary visual area means that all information coming from this area may be erroneous, particularly if the sensory input comes from a stationary object. Therefore the correct sense of depth is one of the most common difficulties.”

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