# BINOCULAR VISUAL SENSATION IN READING A UNIFIED THEORY

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**Abstract**

*Current visual sensory theory focuses on the dual pathway nature of the visual system. Two pathways carry information from the eye to the brain, the parvocellular (detail and color) and magnocellular (motion) pathways. The magnocellular pathway has been implicated as a cause of dyslexia. Clinically, intermittent central suppression has been shown to be associated with reading problems. These two phenomena can be tied together by applying the perceptual fading of Troxler's Phenomenon. This leads to the hypothesis that intermittent central suppression is a clinical diagnosis of visually involved dyslexia.*

**Key Words**

*dyslexia, parvocellular pathway,intermittent central suppression, magnocellular pathway, Troxler's phenomenon*

The essence of binocularity is the combination of sensory inputs from the two eyes into a unified sensation in the brain. Researchers have sought to characterize both the nature and anatomical location of visual sensation. Some conflict, or at least lack of linkage, is often apparent between clinical and basic scientific literatures in this regard. Visual sensory input to the brain and the meaning of binocularity–especially as it relates to reading–is one of those areas lacking linkage.

The bulk of recent scientific research on vision and reading has involved exploration of the parallel visual pathways to the brain: the magno (M) and parvocellular (P) pathways.[1-63] Defects in the M- pathway have been linked to "dyslexia", or reading problems.[4,5,7,10-12,14-17,21-23,25-29,32-35,40-56,62-64] Much of the clinical research on visual sensory input to the brain has been concerned with dyslexia. Intermittent central suppression (ICS) has been linked clinically to reading problems.[65-69,74-82] The scientific literature on the M- pathway defect in dyslexia has been impressive in its approach, data, and technology; the clinical literature on ICS is impressive in its number of human subjects. Papers by Annapole,[68] Strauss and Immermann,[67] Hussey,[69,70,75,77,79,81] and Miller[85] show data derived from over 650 ICS patients; a number rivaling - if not significantly more than - the subjects in the entire world literature on visual pathways research.

Both of these areas have their associated questions. For example, the scientific research has struggled with investigating the specific effect a magnocellular pathway defect has on reading. Does the visible persistence presumably created by defective timing between M- and P- pathways cause dyslexia,[4,15,29,50,64] or is the problem, perhaps, the altered visual attention that is associated with both dyslexia and M-pathway problems?[52,53] Perhaps the effect is a combined one. Both dyslexia and amblyopia show motion deficiencies, presumably from a M- pathway defect.[32,47,57] Are those linked? If so, is a monocular M-pathway defect possible in amblyopia? If not, why does research indicate that amblyopia, with its motion defect, is unrelated to dyslexia?[70] By what method is a M-pathway defect corrected?[29,93] Since no M-pathway theory requires two eyes, is binocularity and binocular therapy even an issue? Could we simply improve the M-pathway in one eye with some monocular technique such as patching, leave the other eye unaltered and still improve reading?[40]

## An overview of the magno–and parvo–cellular pathways

This body of research shows that two major streams or pathways carry information from the eye to the brain. Using electrophysiological, motion perception and contrast sensitivity testing, these studies delineate the existence of two parallel visual pathways. Each carries a different form of visual information that complement to comprise the light adapted (cone) visual world we see.[63] The Parvocellular or Sustained or P-pathway primarily carries detail and color information. Its com-

plement, the Magnocellular or Transient or Motion or M-pathway carries motion (on a stimulus level, flicker) information in the same area of the visual field. These two information streams travel separately to the striate cortex and to different interpretive areas of the brain. It is the M- or motion information pathway that is consistently implicated as defective in dyslexia. The research literature on the parallel visual pathways is huge and profound and cannot be discounted out of hand. Interested readers are referred to more extensive summaries than here.[22,33,56,61]

## An overview of ICS

The research on ICS is largely clinical. As stated above, the ICS research is impressive in its numbers. In several studies, an average of about 80% of ICS patients complained specifically about reading.[70,78,80] It is probable that a number of these 500 or so patients would be classified as dyslexic, depending upon the criteria used. Three cases showing the genesis and one case showing both the genesis and remediation of ICS in whiplash cervical trauma have been reported. The full circle in reading complaint from no complaint to reading problems back to no complaint was seen concurrently with the appearance, and then correction of the ICS.[77]

The questions associated with ICS are somewhat more practical, clinical questions than with the M-pathway literature. (Practical questions, however, often point toward answers in structure and physiology.) For example, why do anti-suppression techniques incorporate motion in a binocular field such as plucking a Brock string to increase its effectiveness in treating suppression?[63] Why can the suppression in strabismus and amblyopia be diagnosed using stereopsis, but ICS cannot,[70] if they in fact are both suppressions? If ICS creates an obstacle for reading, shouldn't a patch eliminate the visual confusion and therefore, any associated reading problem disappear since the confusion-producing eye is now out of action? How can present suppression theories explain the alternation and intermittency seen in ICS (by definition a non-strabismic condition)? Or, are strabismic suppression and non-strabismic ICS neurologically different entities?[78] If so, how do we explain different neurological entities that are both defined by a lack of visual sensation in otherwise anatomically normal eyes and present identically on specific binocular tests (except for the intermittency of ICS)? If suppression is a competitive inhibition, how do we explain ICS arising from whiplash?[83,102] What sort of injury produces increased inhibition as its only manifestation?

I propose that since both the M-pathway and ICS literatures deal with reading problems, dyslexia included, it is not unreasonable to propose that they should be linked in some manner. If vision has any effect on reading, is it possible that they can be unified into a more general view of visual sensation? If not, one area of research must be seriously questioned. This paper will attempt to combine these disparate views into a unified view of what a suppression is, giving us a more complete look at the neurology of binocularity.

## Magnocellular Pathway Research

During any light-adapted fixation, the target of regard is seen and analyzed by two neural pathways that add together to produce a person's visual world. These pathways maintain some separation through the dorsal Lateral Geniculate Nucleus (dLGN) and on to the striate cortex. The P- pathway occupies the four more dorsal layers of the dLGN, to the striate cortex, then proceeds to the temporal cortex. The M- pathway occupies the two more ventral layers of the dLGN, then proceeds to the striate cortex, and on through the medial temporal lobe to the parietal cortex.

Both pathways, to different degrees, are sensitive to brightness, coarse shapes, coarse stereopsis, and detect contrast in low spatial frequency targets. Both are involved in scotopic vision.[18,19] The P- pathway contributes detail, pattern and color to visual sensation. It has color opponency and shows binocular enhancement with color at the cortex, indicating P-pathway binocular convergence.[39] Importantly, it is the P- pathway that carries fine stereopsis. Along with a lack of fine stereopsis, then, anisometropic amblyopia is associated with a loss of P-pathway function and neurons.Also, since M-pathway responses are available very early,[96-99] and since stereopsis continues to develop into adulthood,[58] I propose that, while both pathways develop over time, the M-pathway is functional earlier than the P-pathway and that the P-pathway may develop later than the M-pathway.[56]

As might be expected, the information carried by the P-pathway is a function of anatomy. The receptive field centers are smaller and have stronger antagonistic surrounds. However, the off-response is weak, giving a more sustained response. So, response to non-moving detail is good. That is, the P-pathway is responsible for acuity. At least early in the visual system, the P-pathway may be without an inhibitory apparatus.[2]

The P-pathway accounts for 80% of ganglion cells in the optic nerve. P-cells concentrate toward the fovea, comprising 91% of the ganglion cells representing this area. P-cells continue into the periphery, but decrease in relative density with increasing eccentricity, and comprise 40 to 45% of the ganglion cells in the periphery.[31]

The M- pathway, in contrast, has design characteristics benefiting detection of motion. Color (wavelength) opponency is not present, but the M-pathway may be relatively enhanced by shorter wavelengths (blue).[50] Receptive fields are larger than in the P-pathway; latencies are shorter and axon diameters larger. Response is movement dependant. On a stimulus level, then, the M-pathway is flicker-dependant, and flicker can differentiate the two pathways at the LGN.[2,48] This response to flicker is post-retinal.[1] Responses are transient, not sustained as is the P-pathway. The M-pathway is suppressed during saccades so the visual world doesn't rush by with each saccade.[36] The M-pathway is involved in pursuits.[33] It responds best to high temporal frequency targets (flicker) with low spatial frequency (large/coarse). All cone types and rods feed into the M-pathway, and, thanks in large part to the shorter latencies, information is processed and sent quickly to the LGN and then to the cortex. M-pathway neurons are injured first in glaucoma because of the larger axon size. Alzheimer's disease affects the M-pathway and the decline of the M-pathway parallels a loss of smooth pursuits. That loss of M-pathway ganglion cell neurons in the optic nerve is also reflected in a loss of contrast sensitivity.[27]

Ten percent of retinal ganglion cells in the optic nerve are M-cells. The M-pathway is represented in, and density is great-

est at the fovea, but still is only 5% of the ganglion cells connected there.[20] Its absolute density declines with retinal eccentricity, but the relative density increases to 20% of ganglion cells in the periphery.[31] The M-cells in the fovea are sensitive to fine enough motion that the small fixational eye movements should produce a M-pathway response.[2] Motion opponency is seen in the medial temporal lobe M-pathway cells, prior to this pathway proceeding to the parietal cortex.[58]

It might logically be expected that, if only one of these pathways were to be found defective in reading disability, it would be the P-pathway since reading letters involves seeing detail and pattern. Nevertheless, it is the motion-sensitive M-pathway that is consistently implicated in reading disability (dyslexia). The defect is probably early in the visual pathway, likely a post-retinal, pre-cortical defect,[10,15,46,54,55] likely in the central visual area.[15,38,43] This suggests the visual defect in dyslexia is a disturbance in basic visual processing, not in "perception."[7,12] Since the differences between normal and disabled readers occur at the first level of visual processing, some influence on processing at any subsequent level should be expected. As a post-retinal/pre-cortical defect, the LGN must be suspected as the location of the M-pathway defect in reading disability. The pulvinar has also been suggested, a structure intimately associated with visual attention.[52,53] The pulvinar is continuous laterally with the LGN, separated by only a slight line of demarcation.[101] Since only 20% of dLGN input is retinal, an extra-retinal signal setting gain in the M-pathway is likely.[36] The suspected M-pathway defect in reading disability is illustrated by figure 1.The reason a M-pathway defect might affect reading, and specifying the therapy for any suspected M-pathway defect have been a bit elusive. Pursuits are affected by M-pathway defects, so pursuits might be part of the therapy. But, pursuits are not involved in the act of reading, nor are eye movements necessarily part of the reading problem.[49] A defect in the M-pathway might disrupt the saccadic suppression and create visible persistence; a visual image that persists with subsequent saccades, creating a smeared resultant image. That does occur clinically, but apparently not in more than 25% of reading complainers.[29,64]

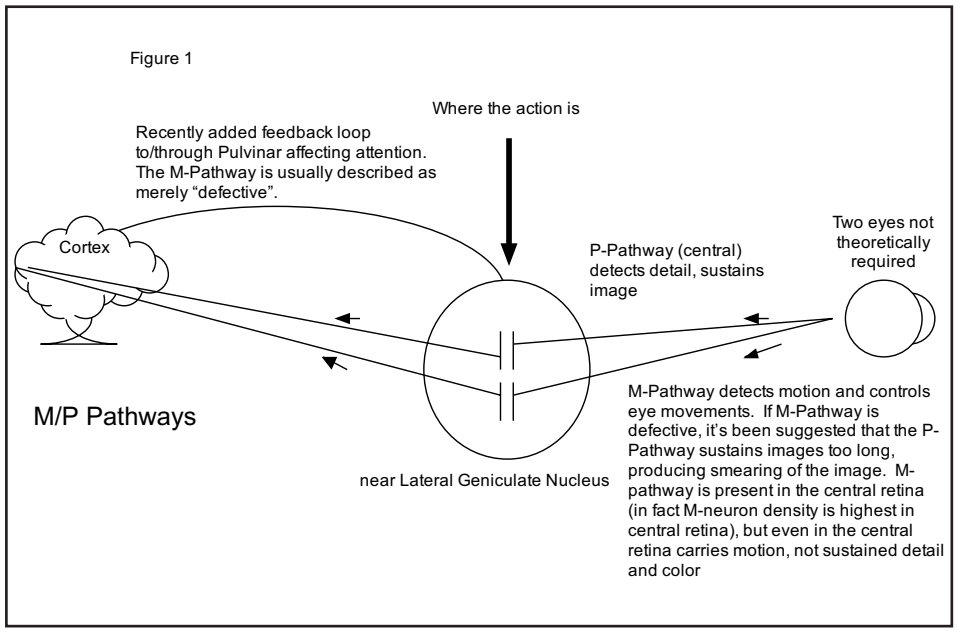


*Figure 1.*

Visual attention is affected by M-pathway deficiency. The M-pathway input to visual attention is more robust than the P-input and the former pathway is responsible for "priming" visual attention. Outside the "attentional spotlight," visual processing is inhibited. Disabled readers have a narrower, weaker attentional spotlight with a stronger inhibitory surround. Does this affect saccadic programming? Saccades require a shift in visual attention, so they may well be negatively affected in a M-pathway deficiency. The reduction in M-pathway function in ageing may lead to difficulty in attending to central visual stimuli and then to reading deficits.[52,53]

Taking advantage of the M-pathway sensitivity to short wavelengths, blue filters have been used for some improvement in both eye movements and reading.[29,41,50,51] However, the papers exploring this wavelength relationship note two problems: Lack of a "simple and reliable procedure to diagnose,"[41] and lack of "specific vision therapy procedures"[29] to treat a M-pathway defect. This certainly puts a cloud over our ability to deal clinically with this defect either diagnostically or therapeutically. Because of this struggle to explain the specific effect on reading, the M-pathway explanation of reading disability is not without detractors.[43,91-93]

## Clinical Research on Intermittent Central Suppression (ICS)

This body of research began in the 1960s.[65,66] Notable for its numbers of documented affected patients and the consistently high frequency of reading complaint in those patients, the link has been made from reading problems to ICS as a causal factor. Problems in acceptance of ICS as a cause of reading problems have often had to do with diagnostic confusion with strabismic suppression. Those studies which use strabismus and amblyopia tests to assess suppression in reading disability find no association; those that use stereoscopic or vecto- graphic testing and acknowledge the time-course of ICS usually find an association.[70,85] The early literature, while not suggesting a precise mechanism for its interference with reading, simply specified that the suppression be remediated as the first order of business in treating binocularity problems. Also obvious in the early literature is a lack of specific diagnostic criteria.[65-68]

Strauss and Immerman first defined ICS as "an involuntary, temporary suspension of vision in one or both eyes"[66] (also recently termed "an intermittent alternating central scotoma"[82]) in non-strabismic subjects. It is a repetitive loss of visual sensation in the central area of vision in patients without strabismus or amblyopia. This is seen as a loss of detail (acuity) in a non-moving test target.[69,70,80] The central area of vision will be suppressed for an average of two to five seconds, two or more

times every ten seconds.[70,81] As such, screening-type suppression tests, many of which were designed to evaluate strabismus, don't evaluate visual sensation over time; they only require a momentary one-time response and are likely to yield a false negative in diagnosing a suppression when strabismus and amblyopia are absent. One suppression test consistently poor in diagnosing intermittent central suppression is stereopsis as measured with the Titmus dot test.[70] Here, an "incorrect" response can be "corrected" when the depth effect of the stereoscopic target becomes apparent at some time during the testing since non-strabismic suppression varies through time. ICS patients tend to have eye movement and accommodative deficiencies, but refractive errors tend to be moderate. Thus stereopsis and refractive error are not good predictors of non-strabismic suppression (ICS), unlike the commonly significant refractive errors of strabismus and amblyopia.[79]

Routine examination for ICS, as documented elsewhere, allowed diagnosis of ICS caused by whiplash cervical trauma and stands as the only documentation of the genesis of suppression.[77] The complete "loop" of cause, effect, remediation and recovery was shown when ICS appeared as a time-linked apparent consequence of the whiplash cervical trauma and concurrently reading suffered. Treatment of the ICS with anti-suppression therapy eliminated the suppression and returned reading to (subjective) pre-trauma levels.

Based on this cervical trauma induced ICS, Hussey suggested the area of the LGN as a logical locus of the suppression, making ICS an afferent visual defect that interferes with reading. As with the suggestion of a M-pathway defect being a post-retinal/pre-cortical defect, some significance accompanies any suggestion of a possible afferent visual sensory defect not associated with refractive error: A negative effect on reading in the presence of such a visual defect can logically be expected. As an LGN defect, higher levels of visual processing must be affected in ICS, just as the suggestion was made that, as a post-retinal/pre-cortical defect, magnocellular pathway defects in dyslexia must affect processing at higher levels.[7,12,79]

Figure 2 illustrates the suggested defect in ICS. Hussey has suggested a mechanical explanation for the reading interference.[70] Other authors have simply assumed that a binocularity problem might cause reading and perceptual problems.[65-68] According to Hussey, repetitive loss of central visual sensation could interfere with fixational stability. Then some aiming error would occur, followed by superimposition of letters as the ICS resolved after two to five seconds and two-eyed sensation resumed for another seconds-long period. Again, this on-off cycle of central visual sensation repeats over time. In sum, these clinical datasets suggest ICS is an afferent sensory defect in vision that would logically be expected to interfere with central visual tasks such as reading.

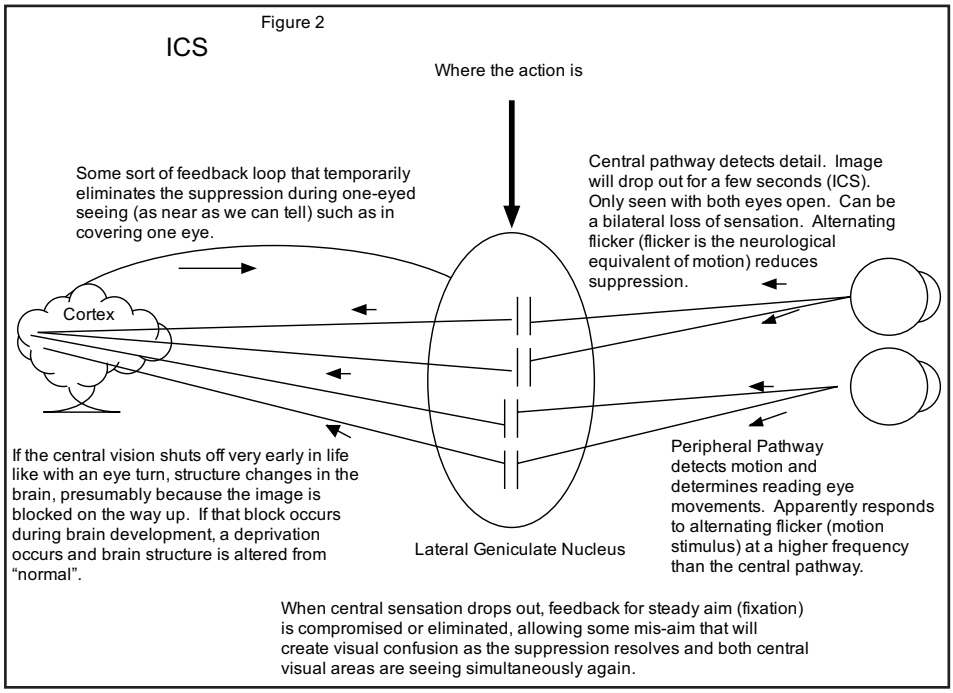


*Figure 2.*

But, how can that be reconciled with the research on the M- pathway defect in dyslexia, especially since most ICS tests have little to do with motion? It's worth remembering here that most anti-suppression techniques require or benefit from target motion. Suppression has been treated clinically with alternating flicker.[71,75,79,83-85] Flicker is merely motion in stimulus form.[64] Still, at first blush, these two areas seem poles apart.

## Troxler's Effect: History and Research

In 1804 it was noted that if a subject could hold his eye very still in viewing a target monocularly, that is, remove motion from what he sees, the image would fade: Troxler's Effect or Phenomenon.[89] Later experiments with image stabilization showed the same effect: if a retinal image could be externally stabilized, it faded. The two phenomena came to be considered the same effect, the disappearance of the image sometimes referred to as "perceptual fading."[86,92]

When an image is stabilized on the retina; again, when motion is removed from the image, it fades. Color fades quickly (suggestive of P-pathway involvement). Complex images are somewhat more persistent.[86] The image can regenerate in part or whole, but motion causes "instant" reappearance.[89] The average length of the disappearances varies from an estimate of just over 3 seconds[92] to 6.41 seconds.[87]

The effect occurs both foveally and peripherally.[88] Drifting eye movements, more so than saccades, counteract this fading and keep the image intact centrally; but drifts are not as quickly effective in generating reappearance peripherally as foveally.[89] This may explain why Troxler's Effect was noted first as a peripheral phenomenon. High frequency fixation tremor that scans over about 1/2 cone diameter is not sufficient to bring the image back, since three neural units must respond to break the effect.[86,87] Flicker in the range of 1 to 2 Hz will keep the image alive, but 25 Hz flicker has no effect. Reappearance occurs quickly if the target is

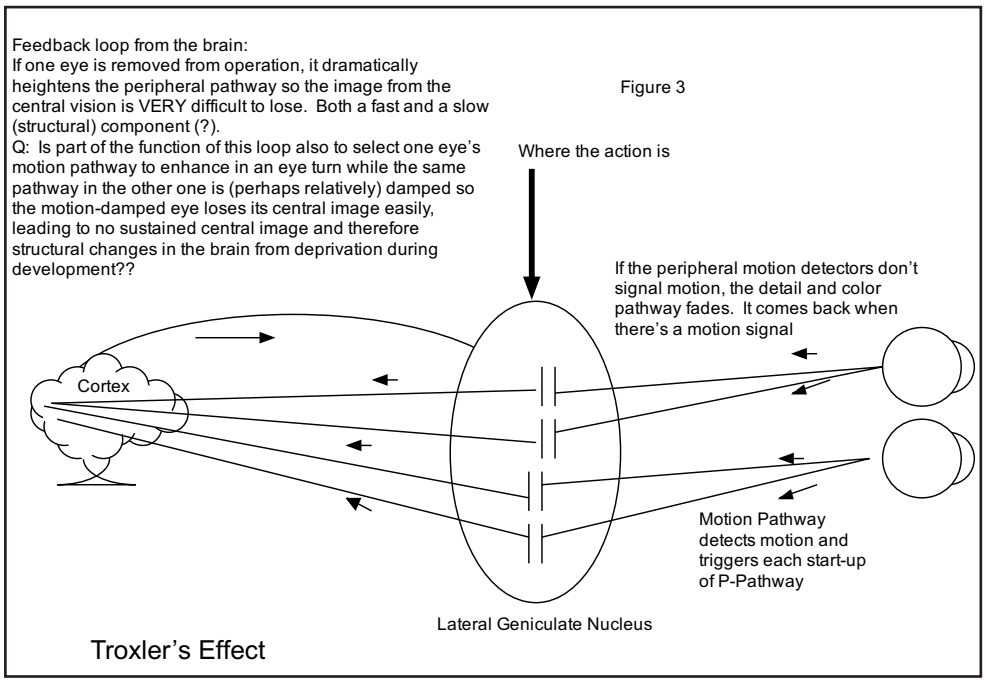


*Figure 3.*

moved to a fresh retinal area, less quickly if it is moved to a corresponding area, and most slowly if it remains in the original retinal area. Plus lenses make no difference in the image disappearance.[88]

Troxler's Effect is neural, not photochemical.[92] It is a post-retinal/ pre-cortical phenomenon.[88,91,92] The site is prior to where the accommodative controller receives its error signal. Retinal ganglion cells at the LGN are the likely site, so this is an early afferent visual pathway phenomenon. Ganglion cells responding to transient stimuli (i.e., the M-pathway) carry the message that breaks the fading, producing image reappearance.[92] Since the signal reestablishing sensation is apparently a M-pathway phenomenon, it is not surprising that saccades don't cause reappearance, since the M-pathway is suppressed during saccades.

Permanent loss of binocular neural interaction affects Troxler responses. That is, one-eyed subjects "were found to be markedly resistant to Troxler disappearances."[91] (Even superficially, this would appear to be advantageous, or a monocular individual might have the world fade from view if he didn't keep his eye moving.) Motion or a motion signal is necessary to keep the retinal image alive. Nevertheless, the target disappearance produced during a Troxler's fading is not a stimulus for production of saccades or drifts.[90] However, during the perceptual fading of Troxler's, accommodation is affected. The accommodative response deteriorates to its resting level during the perceptual fade, then returns to its prior level when the image returns.[92] Figure 3 shows a possible illustration of Troxler's Effect.

## A Unified View of Visual Sensation Relative to Reading

A large and impressive body of evidence suggests that if dyslexia is not produced by a defect in the motion-sensing M- pathway for vision, the defect has been shown to be present in a large number of dyslexics. At the same time, ICS continually shows an association to reading problems in the clinic. Can these two be reconciled?

In constructing a unified theory of visual sensation as it relates to dyslexia, let us take only two points on faith: first, the "perceptual fading" of Troxler's Effect is the same neurologically as the perceptual fading that occurs during intermittent central suppression. That is to say, loss of visual sensation is simply loss of visual sensation. The second point of faith or assumption, is Hussey's suggestion of the LGN as the location for ICS.

If these concessions are granted, then by considering the commonalities in the above three areas of research, I propose the following unified framework. I will use the term *visual* dyslexia simply to differentiate this visual sensory defect from other potential facets of a possibly larger dyslexia universe that, for example, might include a sensory defect entity we would term "auditory dyslexia." In visual dyslexia the M- pathway is defective. This is a post-retinal/ pre-cortical defect occurring somewhere near the LGN. This reduction in motion "message" allows the perceptual fading seen in Troxler's Effect (a post-retinal/pre-cortical effect) to occur. This perceptual fading is clinically diagnosed as ICS (a post-retinal/pre-cortical defect). Any wandering of aim (drift) will eventually produce enough motion signal so that the image will return; clinically evident as the intermittency of ICS. The repetitiveness of ICS is simply the same sequence of events repeated with a failing/ sputtering M- pathway. Strabismic suppression is the developmental result of very early ICS.[79] The trigger for this early strabismic ICS must be explained (probably in the context of unbalanced motion detection because of anisometropia or an eye turn), but since the perceptual fade in essence shuts off the later-developing P-pathway, normal development of this pathway and its binocular cortical neurons would not occur.

As suggested in Troxler's Effect where color (carried by the P-pathway) fades first, and by the experience with ICS targets, where non-moving detail fades, the P-pathway is the victim of the M-pathway defect. But, if the M defect occurs late enough in the development of the pathways or is not so complete that the P-pathway is not allowed to function and develop, there is no reason the P-pathway can't itself be essentially intact. Since "perceptual fading" is built into normal neural design, no abnormal P-pathway morphology or physiology is required for ICS (again, different from strabismic and amblyopic suppression). That would simply explain why ICS can be present with normal stereopsis. There is also no conflict with Cornsweet's finding that during Troxler's fades, aiming errors don't occur.[90] Again quite simply, Cornsweet was dealing with intact M-pathways responding to externally (experimentally) reduced target motion. Any motion stimulus/message would have produced an "instant" recurrence of the image, ending the experiment.[89] This, however suggests perhaps the easiest experimental confirmation for this magnocellular theory of

suppression: since Troxler's Effect research teaches that a M- pathway message will reestablish the faded image, then any perceptual fading (ICS) that is accompanied by aiming errors (fixation drift) during the perceptual fading MUST be associated with a M pathway defect. Without such a defect, any motion message will immediately reestablish the image. It might be argued that we're actually merely seeing Troxler's fading from inadvertent, but true, image stabilization without any M-pathway defect involvement in the clinical diagnosis of ICS. That would be unlikely since the majority of patients diagnosed with ICS are children and 2.5 seconds of image stabilization are necessary for Troxler's perceptual fading in the laboratory[88]–not likely in a young child in the clinic.

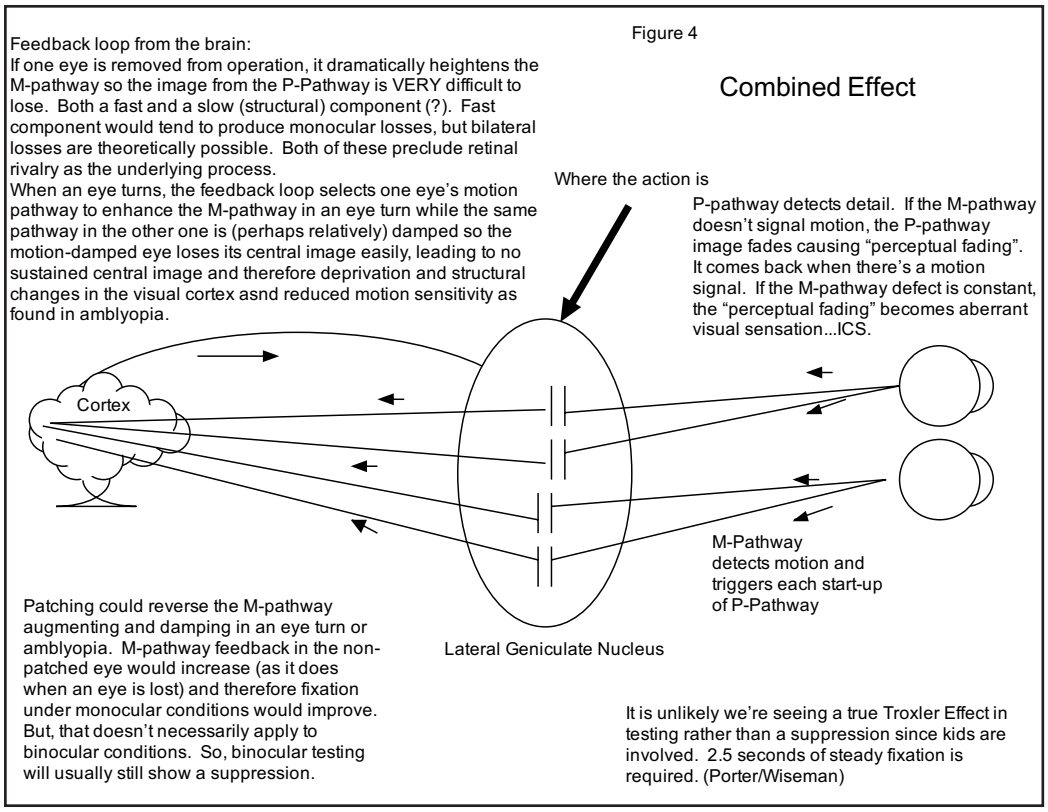


*Figure 4.*

If this view of visual sensation is accepted, a working neurological model of visual sensation and binocularity can be derived. Troxler's teaches us that a long-term adjustment in M input occurs in monocularity that makes perceptual fading very difficult. Troxler's research was done monocularly (by occlusion), so we know this is not a short-term adjustment. However, I propose from experience that occlusion momentarily suspends a suppression as the uncovered eye instantly assumes the role of actively seeing. Without this, monocular acuities would be impossible in amblyopia, for example. Therefore, some sort of a binocularity sensor must exist that "boosts" M signals at the LGN. Enough boost, probably developing over a period of time as when an eye is lost (the "adjustment period"), and Troxler's is difficult to produce. Figure 4 shows a schematic of binocular visual sensation: Dual parallel pathways carry motion in one set of neurons and detail and color in another. The relative signal strength of the motion pathway is read by a higher "binocularity center" which sends a modifying signal to the region of the LGN, either boosting or inhibiting the M signal. A defective M-pathway would allow perceptual fading (ICS). In monocularity and therefore an absence of a second competing visual signal to match, all boost would go to the surviving signal. As discussed above, this has various implications in strabismus and amblyopia, depending on age, that is, the stage of development when the eye turn occurs. Amblyopia can be viewed as ICS at an early developmental age in which the binocularity center influences relative M-pathway boost so that the non-chosen eye, through Troxler's perceptual fading, does not allow the P-pathway to develop normally.[17] The cortex simply does not develop normally because of an inconsistent signal during developmental periods. Since this more complete suppression of amblyopia relies on interfering with development of the P-pathway and therefore the cortex, this also means a mechanism is not available to completely suppress an eye in late acquired diplopia; although the "binocularity center" and its ability to somewhat boost a signal on one side might provide a mechanism to allow some form of "ignoring" the less favored image.

This notion of a boosted signal is at odds with present suppression theory.[83,102] And, in fact, the neurodynamics of the M signal modification at the LGN could be argued from either an inhibition or a facilitation point of view. Most of the information forming our theory of suppression as a competitive inhibition comes from the pioneering work of Hubel and Wiesel on visual deprivation.[83,96-99,102] This area will be discussed more fully in a subsequent paper on the implications of this M-pathway theory of suppression. But, it is worth noting for now that many of their findings can be reconciled with the M-pathway theory of suppression by noting that this pathway is present and fairly well wired at birth. The P-pathway develops more slowly and therefore later. The evidence of later development of stereopsis, again, supports this. Kulikowski and Tolhurst suggest that the P-pathway does not have the mechanism for inhibition and are supported by Boynton, et al. with their finding of the probable facilitation of the signal. [2,60] The M-pathway has much ability for modification (either inhibition or facilitation[36]) of its signal since it is never truly silenced, but maintains a background level of activity.[24]

## Conclusions

A new theory of binocularity has been suggested. The M-pathway theory of suppression combines the seemingly contradictory areas of dyslexia and this pathway, and reading problems and ICS. By applying the research on Troxler's Effect, it can be seen that a M-pathway defect producing a significantly weakened motion sig-

nal would produce a perceptual fading that would be seen clinically as intermittent central suppression. Conversely, any suppression based on M function and Troxler's fading would require motion to reestablish the image (permanently).

Implications of this theory will be discussed more fully in a later paper. However, if accurate, two profound conclusions arise: First, a diagnosis of intermittent central suppression signals a diagnosis of M-pathway defect. Conversely, elimination of the suppression signals improvement in that same M- pathway. Second, and perhaps even more profound, since M- pathway defects are so strongly associated with dyslexia, a diagnosis of ICS can be considered as a reliable diagnosis of visual dyslexia that can be made optometrically. Conversely, any change in the ICS made therapeutically must be viewed as a change in the status of the visual dyslexia. This still doesn't settle the question of what the precise reading- confounding effect is. But, the M-pathway schema with its involvement in dyslexia, Troxler's Effect, and therefore Intermittent Central Suppression suggests a summary statement: "If the magnocellular pathway fails, then the parvocellular pathway fades."

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