BINOCULAR VISUAL SENSATION IN READING II

IMPLICATIONS OF A UNIFIED THEORY

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Abstract

Both magnocellular pathway defects and intermittent central suppression show links to dyslexia. The prior paper suggested a theory of intermittent central suppression based on a magnocellular pathway defect. This paper expands on that theory and suggests some further implications particularly for reading, amblyopia and the need for further research.

Key Words

amblyopia, dyslexia, intermittent central suppression, lateral geniculate nucleus, magnocellular, parvocellular, strabismus, VEP

Introduction

My previous paper presented a theory to explain how both magnocellular (M) pathway defects and non-strabismic intermittent central suppression (ICS) can have a close relationship to reading problems, that is, dyslexia.[1] In this view, a M pathway defect would cause repetitive fading of the detailed or high frequency information carried by the parvocellular (P) pathway through the same mechanism that mediates Troxler's perceptual fading. This repetitive perceptual fading would be manifested clinically as (ICS). The significance of this hypothesized link between M pathway defects and ICS is that ICS then becomes a clinical diagnosis of a M pathway defect and also, therefore of (visual) dyslexia. The theory can be summed up simply: "If the M-pathway fails, the P-pathway fades."

This view should be verifiable by observing the behavior of fixation during a suppression. Since ICS is by definition non-strabismic, any non-suppressed fixation would start with bilateral alignment on the target of regard. This M pathway theory of suppression proposes that a defective M motion signal either allows or produces the (Troxler's) perceptual fading. If the M pathway is intact and fully functional and is not involved in the condition of ICS, then we would expect no misalignment of the visual axes (drift in aim) during a suppression. This would be consistent with Cornsweet's finding of no fixation drift during a Troxler's fade as found in his image stabilization experiments.[2] Cornsweet presumably found no drift in aim because an intact M pathway would restore the previously faded image since this (Troxler's) fading of the image is controlled by the M pathway signal.[3] If, however, a M defect produces ICS - or perhaps, if the M pathway is defective in association with ICS - then during the suppression, with its lack of a perceptible visual image (faded P- and impaired M-signals), we would expect a fixation drift, the magnitude of which is dependent on the phoria. This drift would be not possible when there is an intact magnocellular pathway (as Cornsweet found), so any fixation drift must be associated with a defective magnocellular pathway. Verification of this hypothesis should be pursued.

In addition to involvement of the M pathway in ICS, my theory suggests the existence of a binocularity detector that determines the amount of enhancement of the M signal at the Lateral Geniculate Nucleus (LGN). (This theory could be argued almost as easily from an inhibition perspective rather than enhancement, or from a combination enhancement/inhibition perspective.) I cannot propose a specific location for the binocularity detector other than a location at a higher cerebral level than the LGN. The binocularity detector is suggested to me by research indicating that Troxler's perceptual fading is very difficult to elicit in monocular patients.[1] One complication from theorizing the existence of the "binocularity detector" that works on the basis of enhancement is that we must leave open the possibility that the actual neural defect in visual dyslexia is in this signal enhancing area, rather than at the LGN. The LGN would remain the site of the anomalous

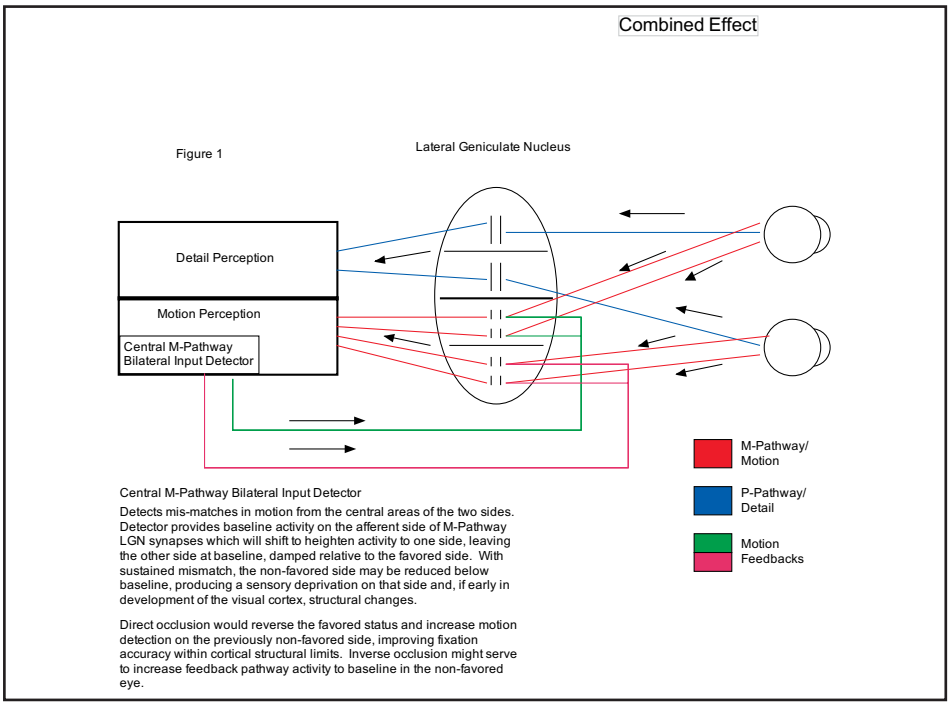

Figure 1

action, however, since this is a post-retinal, pre-cortical phenomenon.[1] Figure 1 shows what might be a very rough summary of the pathways involved in producing magnocellular intermittent central suppression as suggested in this magnocellular theory.

If my theory of suppression proves to be accurate, it has implications for a number of research and vision development issues. However, to be accepted, a theory must account for research findings that are not concordant with any of the theory's assumptions. In this regard, the previous paper partially addressed amblyopia.

The purpose of this paper is to apply the M theory of suppression to address some speculations involving suppression, the research on M pathway defects, dyslexia and further thoughts on amblyopia. I also propose research that is required to test these speculations.

The Conventional Wisdom: Competitive Inhibition

It is productive to juxtapose my theory with the present conventional wisdom on suppression; that is, that suppression is a function of competitive inhibition.

Most of the work on suppression has dealt with amblyopia or strabismus and is descended from the pioneering work of Hubel and Wiesel[4-6] as well as Duffy, et al.[7] In this view, a deprivation (for example, lid suture in kittens) causes LGN and cortical changes. Ventral LGN cells show cell atrophy, but normal receptive fields. Dorsal LGN cells become sluggish and show a larger reduction in cell area than the ventral cells. Apparently, the cortex is wired for binocularity early, and the ventral pathway through the LGN is available early. Late occlusion based deprivation can shift cortical cell sidedness. Intravenous use of bicuculline to eliminate inhibition can restore some of the bilaterality of the cortical cells.[7] The thought, then, is that the signals from the two sides either fuse or fight at the cortex as a result of inhibition.[8,9]

A salient point from this body of research is that all layers of the LGN, even the atrophic layers, continue to grow after birth. So, taken with the difference in effects lid suture produces on ventral and dorsal layers, these experiments suggest : (a) The ventral LGN layers are wired to the cortex early. (b) The dorsal layer develops later and can be more affected by an early insult or deprivation. (c) Even with lid suture, the VEP is present, but changed; the early VEP is present, but the late VEP is absent.[5] (d) Although monocular deprivation produces cell changes at the LGN, bilateral deprivation does not.[6] (e) Late occlusion shows a cortical, not a LGN mechanism is available that shifts sidedness of cortical binocular cells.[5] This supports the notion of a central "binocularity detector," although whether its action is at the LGN not known.

Amblyopia

The traditional competitive inhibition model of suppression immediately runs into a problem in amblyopia. Amblyopia, with its constant suppression, is associated with motion defects, a M pathway dysfunction.[10] And, it is defined by an acuity deficit and is associated with reduced stereopsis, both carried by the P pathway.[11-13] So, for amblyopic suppression to exist with these pathway deficits, it must involve a two-part suppression; one suppression mechanism for each of these two relatively neurologically separate pathways; or, perhaps, a double developmental deficit. The M theory of suppression simplifies this somewhat.

With an eye turn or anisometropia, some difference in the central retinal motion signal would be expected. Since the M pathway density is greatest in the central area, that area would be expected to carry a fair amount of weight at the "binocularity detector."[1] Further, that central detector would be expected to choose one signal (eye) to favor when faced with a wide disparity in signals that it could not adjust to be compatible with its signal enhancement (or inhibition) mechanism. Perhaps an early form of visual attention is involved. The other eye would relatively lose M input. This loss of M activity at an early enough developmental stage would lead to a relative lack of that pathway's development. The automatic victim of this lack of M activity and development would be the P pathway. The relative reduction in the M signal would promote loss of P signal through the mechanism of Troxler's fading. Since the P pathway is less developed at birth than the M, and since the available P signal is now impaired, we would expect some lack of development, leading to the loss of the P pathway seen in amblyopia.[11] This would be accompanied by a lack of development in acuity and fine stereopsis, both carried by P pathway.

But, a motion defect should also be expected. As the motion signal has been relatively boosted on one side, the other side should be relatively inhibited in its development. Since both pathways continue development over time, both would be affected by this interference in normal development; they would not develop fully. The M pathway might be expected to suf-

fer less since it is more fully developed at birth. But, both defects accompanying or constituting amblyopia are accounted for in the M theory of suppression. Thus, it is plausible that amblyopia is simply a congenital M defect that then leads to the domino-effect P and cortical developmental defects.

Along with our understanding of the dual pathways serving vision, the "binocularity detector" concept can provide an explanation for the positive effects occlusion (patching) may have on visual acuity. Patching would essentially use the same process that produces amblyopia to reverse amblyopia: Disparity in motion detected. That is, the unpatched eye would now have a stronger motion signal than the patched eye. Signal enhancement would follow on the side of the stronger M signal. With the dominant eye patched, the "binocularity detector" would relatively boost the M signal on the non-dominant unpatched side. This would have the effect of reducing any tendencies for perceptual fading, the phenomenon noted in monocularity.[17] The increased activity in the P pathway would be expected to make some new connections in the cortex, improving acuity. This is very similar to the scenario suggested by the more traditional competitive inhibition theory.

However, under binocular test conditions such as vectographic testing, the competitive inhibition theory would suggest a decrease in suppression on the same order of magnitude as the increase in acuity. For example, if the acuity were restored to 20/20 and equal to the formerly better eye, it is reasonable to expect a substantial decrease in competitive inhibition and therefore in suppression. This contrasts with the M theory that suggests that under binocular conditions, the favored eye would still be favored simply because its pathways had more completely developed earlier. Therefore, the suppression would likely be retained in some form, although the depth might be expected to be decreased by the improved connections in the two pathways as a result of the patching. So, as is seen clinically with binocular testing, the amblyopic suppression often remains although the acuity has improved. Further, since the M pathway is apparently more thoroughly developed earlier than the P pathway, we might expect the functional gains in the P pathway to surpass those in the more developed and therefore less malleable M pathway. So, we might expect a suppression on the originally amblyopic side similar to ICS rather than the original complete/constant suppression, since the underlying M defect would persist on the originally amblyopic side to some degree.

Indirect occlusion usually provides a case for competitive inhibition. By patching the poorer eye, the competition at the cortex is reduced. Modest improvements in acuity in the poorer eye might be expected. The M theory would predict strong facilitation on the favored side. Indirect occlusion might be expected to allow some normalization of that facilitation, producing mild decrease in the perceptual fading. Some improvement in the acuity might be expected, but, certainly, this is easier to explain if inhibition is thought to be involved.

An application of the M theory can raise some speculations about patching for amblyopia that require clinical research. Since both pathways develop over time, could injudicious patching, at an early or particularly sensitive time in development create a problem with development and therefore possibly even create visual dyslexia? Some earlier literature supports that strabismics and amblyopes are less likely to have reading problems, that is, visual dyslexia.[14] I propose that even though the amblyopic eye has a compromised M pathway (above), my theory suggests the M pathway development on the dominant side is normal, and perhaps even enhanced. This is simply a function of the "binocularity detector" favoring the dominant eye and facilitating the dominant eye's M-pathway. But, if it were possible to unintentionally interfere with continuing M development with early and long term occlusion, the effect of this interference in pathway development could result in a somewhat higher frequency of visual dyslexia among amblyopes who have been patched versus those who have not been patched. If such a trend was evident, group age commonalities in age at time of patching might increase our knowledge of the timing of pathway development.

A second speculation is that if the theorized reliance on facilitation is accurate, we might also expect slightly superior (compared to normal eyes) motion detection on the dominant side in non-patched amblyopia. As the "binocularity detector" would adjust the afferent signal with enhancement rather than inhibition, the resultant slightly superior motion detection in the favored eye in a non-patched amblyopic individual would produce slightly less tendency for Troxler's fading than in "normal" eyes. Any such superiority in motion detection would support enhancement rather than inhibition as a signal modulation tool.

My last speculation is based on this scenario of pathway development and interference with development: I have implicated Troxler's perceptual fading for the lack of a P pathway signal and therefore a lack of P development. During a Troxler's fade, another "fade" occurs: The accommodative response also "fades" to its resting position.[3] Is it possible that the relaxation of accommodative posture is partially responsible for the excess hyperopia often accompanying amblyopia? That suggestion seems difficult to resolve experimentally, but may be worth discussion.

Competitive Inhibition—-Amblyopia and ICS

The traditional competitive inhibition theory of suppression also runs into problems with non-strabismic intermittent central suppression (ICS). I have found that most ICS patients alternate supressions between eyes, while still maintaining normal stereopsis (as typically clinically tested with devices such as the Wirt dot test[14]). This alternation is different than an alternating strabismus where one eye or the other will often be consciously "chosen" to be the sighting eye with a consequent loss of alignment expected from the other eye.

In ICS, one eye's picture is "lost" for two or three seconds, then that eye's picture returns and either the other eye suppresses in a direct alternation, or a period of bilateral sight will intervene for a similar period of time. Then either eye suppresses again.[14,15] All this happens without consciously "choosing" a sighting eye. In fact, during examinations I often tell patients to "relax and observe" what happens rather than trying to "make something happen." This sequence of visual events occurs in ICS, a condition shown to be associated with a normal distribution of refractive errors (minimal refractive errors), normal stereopsis and, by definition, with eye alignment. This, of course, is in contrast to strabismus and

amblyopia that tend to be associated with lack of stereopsis and more significant refractive errors.[14,16] So, for the traditional competitive inhibition model to explain the alternation of ICS requires an alternating inhibition to be interspersed with periods of non-inhibition.

A visual pathway plagued with static similar to varying static in an electrical or radio signal could account for some of this. However, because there is a pattern of a repetitive signal interruption (ICS suppression period) of two or three seconds followed by clear signals of similar time lengths (including the detail clarity producing fine stereopsis and normal or near normal acuities), that seems unlikely. And even with a static-impaired circuit, it's difficult to imagine sufficient static for the central image to be removed, as occurs in amblyopia, but would not interfere with stereopsis, generally considered defective in amblyopia. The same consideration applies to acuity, since a loss of central image must be associated with a loss of P message. However, my experience is that acuity is usually normal in ICS.[14,16]

Further complicating the problem is the above suggestion that both these entities are apparently associated with M pathway deficits. These can appear to be neurologically different entities, except that both must involve the M and P pathways. And, through those pathways, the intermittency and alternation of ICS must be explained.

The M theory of suppression can explain the alternation and intermittency in ICS. The original theory of the visual defect produced by ICS involved the loss of feedback for fixation during the suppression.[14] During the suppression period, small aiming errors occur because of a lack of fixational feedback. When the image is re-established, visual confusion would result from the resulting diplopia. This would be further complicated by motion as the formerly suppressed eye recovered correct alignment. The small area of the suppression zone would limit the degree of misalignment.[16]

Although my theory previously sought to provide a basis for the observable events in ICS, it did not attempt to explain the cause or mechanism of the intermittency.[1] The M theory can explain the sequence of events differently and perhaps more completely: The M defect would produce a perceptual fading (loss of the P pathway) that is clinically seen as ICS. Loss of the P message would remove the fixation lock (similar to the former theory) and fixation drift would occur depending on the phoria. As the line of sight drifted, enough motion would be detected to activate the M pathway, which in turn would activate the P pathway, and the image would be restored. Since the paracentral area is tuned to a higher rate of flicker[16] (i.e., greater sensitivity to speed), the amount of turn would likely be limited by this sensitivity similar to the original ICS theory. But, the magnitude of the aiming error would fundamentally be limited by motion detection rather than image displacement. Motion to reassume accurate bilateral fixation would produce some image "swim," similar to the original ICS theory. This whole sequence of events is primed for repeat since the underlying defect–the M pathway defect–is unchanged. The "binocularity detector" likely has a hand in how the alternation plays out. Notice also that, given a sufficiently defective M pathway, both central images theoretically could be lost simultaneously. This would be an impossibility in the competitive inhibition theory.[15]

Some Predictions Based on Theory

1. Intermittent central suppression is the clinical manifestation of M pathway deficit. As such, elimination of the suppression should mark improvement in that deficit. The probable locus of the defect is at or near the LGN. The likely changes with therapy would be at synapses.
2. I propose a syndrome of visual problems associated with ICS. Based on the Kotulak and Schor's research[3] showing that accommodation lags come to a resting state during Troxler's fading, we would expect some accommodative symptoms, problems, or perhaps instabilities. Eye movements would be expected to be deficient since the M pathway is involved in their control.[3,18] However, reduced stereopsis may or may not be diagnosed as part of the syndrome, depending on the point in time when the stereopsis test is carried out. If the test is presented during a suppression period, with its P pathway "dropout," stereopsis, should be reduced. However, if even fine stereo targets are presented during non-suppressed periods – or if the patient is given enough time for the suppression to resolve - stereopsis should register as normal. "Depth perception problems" could easily be a part of a patient's visual complaint, however, as stereopsis varies depending on the state of sensation at any given moment: Catching a baseball might be difficult. Also, in pursuits, since the target now involves motion, visible persistence is no longer contradictory to ICS since both are merely manifestations of a M defect.[18]
3. As an afferent defect, ICS would be expected to change perception and perception tests.[19-21] Perceptual testing and training should probably follow after treatment of the ICS.
4. If, as I propose, the underlying defect in ICS is in the M pathway, and is therefore a defect in motion detection, motion should be useful in its remediation. This includes visual flicker, which is motion in its simplest form; its stimulus form.[22,23] As suggested in the above discussion of patching and residual suppression, flicker might not be expected to improve binocular M function unless the theorized "binocularity detector" is included in the process. Alternating flicker, therefore, should be more effective in driving the M-pathway's neurology on both sides rather than bilateral simultaneous flicker which would not necessarily force the hypothesized "binocularity detector" from its possibly one-sided mode of operation.
5. I expressed concern above about whether injudicious patching might interfere with M development. The real thrust for research in this area is that it should be designed to improve our understanding of the sensitive time periods in M and P pathways development.
6. With ICS (and therefore visual dyslexia), there may be some potential positive benefit to patching. Given that the eyes are closer to equality in ICS than amblyopia, and given that I have theorized that ICS is caused by a M deficit rather than competitive inhibition, then anything that pushes M development without negatively effecting the function of the "binocularity detector" should help. Therefore,

alternate occlusion in visual dyslexia may have some positive effect.[24] That does not address the issue of whether there might not be more effective and comprehensive therapies. Plus, we might logically expect that after normal M development is completed (i.e., with neurological adulthood), patching might have less effect since it doesn't forcefully "drive" the neurology as a more active therapy might.

7. Lower rates of flicker should be more useful in overriding suppression than higher rates. This follows from data on Troxler's showing high rates of flicker (25 Hz) don't prevent Troxler's fading whereas low rates (1-2 Hz) do.[25] This is supported in data on alternating flicker for treating suppression.[16,22]
8. Alzheimer's disease is associated with a decrease in M neurophysiology.[26] By extension, ICS may encountered with increasing frequency in early Alzheimer's and ageing. This may explain some of the lack of interest in reading with ageing. My clinical impression is that an increase in ICS does indeed sometimes occur with ageing. However, this should be tested. It will require a sophisticated test paradigm since ICS testing is subjective and the picture is confused with other anomalies of ageing such as cataracts and macular degeneration.

   If this view is accurate, further M-pathway deterioration in Alzheimer's suggests some wide reaching inferences. As M pathway deterioration progresses, image fading in the P pathway would be more extensive. An advanced Alzheimer's patient may be descending into a completely unstable visual world as vision literally shuts off more and more frequently, perhaps even for longer periods of time. Perhaps memory is not always the problem in recognition of faces. The faces may actually not be visible in any normal sense. Family members need to understand the necessity of identifying themselves verbally at every visit, and not be surprised if recognition varies day to day.
9. One of the more complex issues might be what we should expect from monocular versus binocular VEPs. First, in a normally binocular individual, because of binocular convergence of the two monocular signals, we'd expect the binocular VEP to be somewhat larger than the monocular VEP, but probably not double.[27] With this binocular signal convergence and without necessity of any signal modification, we would expect normal binocularity to minimize excess pathway neural activity, the binocular VEP therefore somewhat less than a doubling of the monocular VEPs.[28] That is, normal binocularity should be the most physiologically efficient state.

   The question is more complex if ICS – and by extension, visual dyslexia – is present. In ICS and visual dyslexia as described in the M-pathway theory of suppression, we should expect to see a combination of effects. This theory suggests some facilitation of the signal is involved at the LGN. This theorized facilitation effect accounts for how, in loss of an eye, Troxler's fading is very difficult to achieve, but also accounts for lack of suppression effects during short-term occlusion. That is to say, use of an occluder suspends the suppression while the contralateral eye is covered; binocular testing is required to find suppression. Although some of the effects of a suppression might be seen, suppression itself isn't there when one eye is occluded. This facilitation effect, probably active at the LGN, is under control of the theorized "binocularity detector." Although much in the M theory of suppression can be argued from an inhibition standpoint rather than facilitation, it is more difficult to explain this permanent adjustment of sensation in true monocularity that would prevent Troxler's perceptual fading if we assume the binocularity control mechanism is inhibition.

   With a defective M pathway, both ICS and visual dyslexia would be expected. Overall pathway (and VEP) activity would be less than normal because of the defective M pathway and the loss of some P activity through Troxler's perceptual fading (that is, ICS). The P pathway would still show binocular signal convergence and summation so the binocular activity would again be somewhat less than a doubling of the monocular signals. However, the theorized "binocularity detector" would be at work trying to boost what it would read as a deficient and unstable signal in order to limit fading and equalize the two magnocellular channels. So, overall activity would be relatively larger in the binocular VEP as the "binocularity detector" boosted the signal as much as it could. The binocular VEP would still be less than a doubling of the monocular, but relative to the deficient monocular signals, we would expect the binocular signal to be somewhat greater than the ratio that would be expected with the "normal" monocular and binocular VEPs. This is simply because of the added activity involved in the boosting of the signal. In non-dyslexics, then, the binocular VEP signal would show less binocular enhancement than in visual dyslexics. But, because no neural deficit exists in non-dyslexics, the overall strength of the signals would be greater than in visual dyslexia. This is, of course, what the data show.[28]

Conclusions

The purpose of this paper has been to expand on the implications of the M theory of suppression. The full theory, suggested in the prior paper, suggests that M pathway deficits are responsible for ICS and visual dyslexia. In fact, ICS is the clinical diagnosis of visual dyslexia. Further, a "binocularity detector" is theorized that controls facilitation (and perhaps inhibition) of the M signal.

A number of speculations are raised by this theory. Some have been tested; some have not. But, as each suggestion from the theory is tested, the theory will stand or fall. Perhaps the most profound implication of the M theory of suppression is that clinical diagnosis of intermittent central suppression is the first, and to date, arguably the only clinically feasible test available to evaluate M pathway function.[29] And, clinical testing for ICS has been available to optometrists for a long time.

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Date accepted for publication: