Use of Visual Flicker in Remediation of Intermittent Central Suppression Suggests Regionalization of Vision

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Abstract
Intermittent Central Suppression (ICS) has been shown to be a likely cause of reading problems both in a correlational study and a cervical trauma report. The cervical trauma created both ICS and reading problems. Elimination of the ICS eliminated the reading problem. The present article proposes that the Lateral Geniculate Nucleus is the neural structure of ICS. Preliminary results are reported treating ICS with alternating visual flicker using electronically controlled liquid crystal lenses to alternately occlude at an average 6.7 Hz to override and minimize ICS. The same method demonstrated a positive effect on reading at 13 Hz.

Key Words
intermittent central suppression, visual flicker, lateral geniculate nucleus, whiplash, electronic rapid alternate occlusion

A variable sensory defect in vision should cause a problem with consistent, reliable perception of detail. Such a problem would likely include reading. But, prior to discussing that suggestion, some fundamentals of visual function deserve discussion.

Our two-eyed anatomy suggests three possible ways of seeing: monocular, binocular, and bi-ocular vision. Monocular vision - seeing with one eye - would, for this discussion, include strabismus and amblyopia. At the opposite end of the seeing spectrum is binocular vision. Binocular vision implies two eyes seeing simultaneously, adding their information in the brain into one combined image. Somewhere between monocular vision and binocular vision lies bi-ocular sight: simultaneous two-eyed sight, but without alignment (that is, diplopia).

Obviously, diplopia (bi-ocular vision) would be the most confusing of these three ways to see. Conversely, true binocularity should be the most desirable. The maximum amount of information should be going to the brain when binocularity is stable.

But, is there anything terribly wrong, anything terribly confusing, about monocularity - if it is stable? Certainly not as much information is going to the brain. And, stereopsis will suffer. But, if the monocularity is stable, it shouldn’t necessarily create visual confusion. If that premise is correct, then strabismus and amblyopia (both monocular conditions as defined above) shouldn’t necessarily affect reading, a detail intensive task. And, the literature on the effects of strabismus and amblyopia on reading supports that they don’t have a large effect on reading. By extension, monocularity doesn’t interfere with reading.

The second fundamental concept to address is the sensory feedback setup for fixation and aim. The visual system uses an error detection system. Sensory feedback of fixation error and subsequent corrective movements position the target of regard at the macula. Intact visual sensation provides that feedback. The continual error detection and correction produces a vergence fluctuation within Panum’s area.

Proposal of a Model for Visual Confusion
As a model to explore possible visual confusion, consider intermittently, possibly alternately, interfering with the sensory feedback in one eye for just a few seconds at a time. That interference will be repetitive, but will be limited to the central visual area, perhaps the central one to four degrees of visual angle. The periphery, then, remains intact. In this model, one eye loses visual sensation in the central few degrees for two or three seconds, then vision returns. Both eyes see centrally for two or three seconds, and then one eye again loses its central sensory feedback for a similar amount of time. And, that cycle will repeat continuously. Throughout these cycles, the periphery remains intact with its larger Panum’s areas. How might vision be affected in this model?
During binocular, aligned periods, there should be no problem; no interference in vision. When one eye's central vision is suppressed, creating monocularly, there should be no problem. Again, no confusion in vision. But, during this central suppression, monocular phase, the aim of the suppressed eye can drift slightly off target since feedback is reduced. The near periphery is still intact, limiting the error.

In this model, after two or three seconds, the central suppression resolves, and both eyes are seeing simultaneously again. If any aiming error has occurred during the suppression, this patient now has bi-ocularity and visual confusion from this slight misalignment. One degree off aim would be a two or three letter error in standard print. An overt strabismus is not necessary for visual confusion.

With both eyes seeing, the error detection mechanism should take over and the aiming error should be corrected. But, since this model defined this sequence as repetitive, the cycle continues. In this model, in a very real sense, the loss of central sensation through this intermittent central suppression is not the problem. Monocularly is not confusing. That the suppression resolves is the problem. Any misaim produces bi-ocularity. And this misaim and bi-ocularity is repetitive. Any repetitive misaim and correction should create variable visual confusion depending on the precise amount of misaim. This confusion should affect small words, targets and details, as well as likely affecting some depth discriminations.

**Intermittent Central Suppression: A Review**

As a theory, this may explain some reading problems such as variable errors reading small words, but is this suppression model realistic? Both temporal and spatial characteristics of non-strabismic suppression need evaluation to see if they fit the model. A most often tested visual characteristic, refractive error, probably has no real influence on this discussion. Figure 1 shows refractive error in non-strabismic suppressors. Figure 2 shows the same data with refractive data from Helveston, et al., superimposed, showing virtually the same contour. Helveston's data were used to suggest vision does not affect reading, whereas the data from Hussey is only from patients showing non-strabismic suppression, two-thirds of whom specifically complained of reading problems.

This refractive error data and the following test comparisons are from my 1990 study of non-strabismic suppression. Files of 60 non-strabismic patients who showed suppression on the American Optical projected distance vectographic chart in routine examinations were evaluated retrospectively to look at characteristics of the suppressions, the testing, and refractive error. These patients had all been tested with some of the most commonly used suppression tests from the suppression and reading literature: the Worth 4-dot test, the Jampolsky 4-prism test, and Wirt stereopsis, in addition to being tested vectographically both at dis-
Borish Near Card

Enlarged Modified Diamond Target

Figure 3. Schematic of original Borish Near Card showing the modification of the diamond target to test for ICS.

Diagnosis of Intermittent Central Suppression
Commonly Used Tests Compared to Vectographic Refraction

n=60

100%
90%
80%
70%
60%
50%
40%
30%
20%
10%

Wirt Stereopsis
Worth 4-dot loss of lights
Worth with fusing anomalies
Jampolsky 4-prism
4-prism with questionable responses
Bisected Diamond

Figure 4. ICS seen on vectographic binocular refraction was used as a reference criterion for comparison of diagnostic effectiveness.1

tance (AO chart) and at near (modified Borish Near Card, above).

The suppression tests typically used in the literature have often been strabismus-derived tests. Perhaps more important than the derivation of the tests is that they have been used often as quick one-look, correct response/incorrect response tests rather than being used to evaluate sensation over time. Suppression on the distance vector graphic chart was chosen as a reference criterion for test comparisons simply because the typical suppression response of the projected target “disappearing” and turning when a patient is gazing at the externally observable unchanging target shows the suppression in a way that can be seen by any attending observer, and is therefore difficult to contest.3

Figure 3 shows the (original) Borish Near Card modified with Polaroid overlays over the small diamond positioned inferiorly.1,6,7 The diamond is somewhat less than two degrees wide visually. If this target is viewed through polarized analyzers and either eye suppresses, that side will turn black so that the underlying letters can’t be seen. If the suppression resolves, the black side will clear, allowing the letters to be seen.

Figures 4 and 5 show that strabismus suppression tests don’t correlate well to the reference criterion of the distance vector graphic chart, or to each other when used to diagnose non-strabismic on-and-off intermittent central suppression (ICS).1 The modified Borish Near Card correlated well with the vectographic chart reference criterion, suggesting it tests effectively for ICS, and so should provide information on both temporal and spatial characteristics of ICS. Figure 6 shows those temporal characteristics, roughly agreeing with the model of a two- or three-second on/off cycle.1

Figure 7 shows some spatial characteristics of the same target card. By patient report, non-strabismic ICS typically will show losses on the fixed diamond target, but not simultaneous losses of other polarized portions of the Borish Near Card — some as close as 1.5° away from the diamond. Strabismus will cause loss of the acuity groups 2° to 5° away from the diamond or fixation disparity targets, but ICS typically will not.

As stated above, the AO distance vectographic chart can be used to observe ICS.1,5 The same chart and its various sub-tests serves as the target for distance portions of the routine vision examination. These same ICS spatial questions then can be addressed in the distance testing paradigm. For example, Figure 8 shows a schematic of the distance vectographic chart clockdial cylinder test target. The projected chart measures just under 3° in visual angle. Commonly, a quadrant or similarly sized area of this target will disappear, rather than the whole, just as the suppressed area on the Borish card will spare the acuity targets. An estimate of the size of the suppression zone based on the diamond and distance vectographic charts would be perhaps 1° to 3° of visual angle. Again, this fits our model of a small central suppression zone.

The previously determined temporal and spatial characteristics discussed above suggest sensory feedback in the central vision is suspended repeatedly for about two seconds at a time, spaced by similar time periods of simultaneous sight. Patient descriptions and responses suggest the paracentral area (or near periphery) remains intact during the suppression. During the suppression period, the theorized
aiming errors should be reflected in reduced fixation constancy. This has been demonstrated in studies of fixation/vergence errors. Those errors would be expected to be small, certainly less than a frank strabismus. As this repetitive central suppression - aiming error - aiming recovery sequence occurs, the model predicts some interference in detail seeing such as reading.

**Clinical Evidence**

Although current optometric literature seldom refers to ICS, Louis Jacques wrote in 1950 and '56, that suppression was the first order of business in fixing a problem in binocularity. Strauss and Immermann studied 142 school children and found a positive link of non-strabismic "macular suppression" to reading problems in 1964. In 1997, Hussey documented the full circle of cause, effect, remediation, and recovery with ICS and reading complaints. In three whiplash victims, suppression was either created or worsened by the whiplash. And those patients had new reading complaints similar to the reading complaints of school children. One of the patients had therapy to eliminate the suppression, and the reading complaints resolved. The weight of evidence, including demonstration of cause and effect, supports ICS as a sensory problem that will negatively affect reading. If the evidence suggesting ICS is a problem is accepted, then it is incumbent on behavioral optometry to correct the problem.

**Use of Flicker for Treatment**

One method of treatment for the suppression of strabismus and amblyopia used historically is flicker. Use of Merrill Allen's translid binocular interactor (TBI Trainer) is well documented, and variations on the instrument are still available commercially. The TBI Trainer treated constant suppression with lights alternately flickering at 9 Hz. Allen has suggested that alternation is necessary. Bilateral strobing may in fact embed anomalous correspondence.

Allen does not discuss non-strabismic suppression. But, by extension, perhaps flicker can be used to treat ICS. And, Hussey has used electronically controlled liquid crystal alternating occlusion lenses mounted in goggles for such treatment.

In treatment, it is worth remembering that flicker is merely the fundamental visual stimulus underlying motion detection.

**Phi Coefficients**

<table>
<thead>
<tr>
<th>Phi Coefficients of Suppression Tests</th>
<th>4-dot Worth with Luster Stares</th>
<th>4-dot Worth with Luster Stares</th>
<th>4-prism with questionable responses</th>
<th>4-prism Stereopsis &lt; 40 arcsec</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common</td>
<td>0.07</td>
<td>0.07</td>
<td>-0.02</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>0.46</td>
<td>0.11</td>
<td>0.05</td>
<td>-0.08</td>
</tr>
<tr>
<td></td>
<td>0.16</td>
<td>-0.06</td>
<td>0.08</td>
<td>-0.12</td>
</tr>
</tbody>
</table>

Phi \( \phi \):
- \(< 0.30 \) shows weak relationship
- \(0.30 < \phi < 0.60\) shows moderate correlation
- \(> 0.60\) shows strong relationship

**Average Duration of Suppression**

\( n=60 \) subjects

**Average Frequency of Suppression**

\( n=60 \) subjects

**Figure 5.** Comparison of suppression tests showing standard suppression tests don't agree in diagnosing ICS. From Figure 4 it can be seen that vectographic binocular refraction and the Borish diamond test do agree.

**Figure 6.** Rough timing of ICS on/off cycle.

If flicker can be shown to override or treat suppression, and if it is the fundamental stimulus of motion detection, then it strongly suggests that all anti-suppression therapies should involve motion. Merely staring at a red-green visual target probably does not change the neurology underlying the suppression.

Every bit as important as treating with flicker is the possibility that flicker can be used to explore the visual system being treated. For example, Hussey has documented the treatment of ICS with alternate flicker using liquid crystals alternating at an average 6 Hz. As part of the...
testing for a treatment frequency, the rate of flicker is altered to minimize suppression while the patient works on a chiroscopic tracing. Although true elimination of suppression is certainly not immediate, overriding the suppression can be immediate (by definition in this test paradigm). Hussey, in fact, reported on Robert, a 32-year-old male who showed ICS that could be temporarily overridden with 5.5 Hz alternation when gazing at vectographic test targets. What does that immediacy of effect with the flicker of rapid alternate occlusion suggest? Does it provide any direction in the discussion of structure?

Whiplash cervical trauma can create a suppression. Other studies suggest the brain stem is the site of damage in whiplash. If the visual pathways are assumed to be involved in suppression, since the Lateral Geniculate Nucleus (LGN) straddles the brain stem, a logical hypothesis is that the LGN is the site of the suppression. This hypothesis is consistent with recent functional magnetic resonance imaging report showing physiological differences in dyslexics are precortical. If correct, this would infer ICS is neurologically.

Could suppression actually be the product of a blocking input from either a higher or lower center? If a blocking input from a higher or lower center is responsible for ICS, then the direct stimulation from alternating 6 Hz flicker seems unlikely to produce an instantaneous observable effect in overriding suppression. The visual input would require a cortical loop through processing centers. Yet, as was demonstrated with the alternate flicker chiroscopic testing, the instantaneous feedback defines the optimal anti-suppression treatment frequency. In fact, the effect is linked to suppression behavior closely enough that the best individual treatment frequency is bracketed by constantly altering the flicker alternation frequency and eliciting verbal feedback. This immediacy of effect near 6 Hz in overriding and treating suppression suggests a local effect rather than a neural loop to higher centers. This, however, has not been studied. That should not be extrapolated to exclude input to the LGN from other areas. I have found some adult patients can override suppression with attention. That effect typically doesn’t last long, of course. But, if this theorized location is correct, if ICS is located at the LGN, that means suppression is an afferent defect. Suppression occurs prior to the cortex, prior to the “black box.”

Gallaburda, et al., found changes in the neighboring Medial Geniculate Nucleus (MGN) in dyslexics and proposed a model to explain these findings suggesting these changes were secondary to cortical changes. If we can generalize about brain stem involvement from these MGN dyslexia models to this LGN suppression and reading problem model, the LGN model would seem to contradict the Gallaburda hypothesis. It seems unlikely that suppression would be secondary to cortical changes from whiplash when no other brain trauma is apparent; when whiplash cervical trauma has been linked to brain stem damage; when the suppression appeared concurrently with trauma; and when the suppression can be eliminated and normal visual function restored with the apparently local therapeutic effects of alternating flicker. Taking the immediacy of the flicker effect on suppression together with the suggestion that suppression is centered at the LGN, then logically LGN synapses must be considered a most likely location for abnormal or damaged structure.

**Methods and Results Treating Intermittent Central Suppression**

Computer controlled liquid crystal alternating occlusion lenses mounted in goggles were used to treat ICS. Using the immediacy of the flicker effect on ICS, the best anti-suppression alternation frequency was found by bracketing during chiroscopic tracing tasks searching for minimal suppression. ICS treatment frequency data were collected first on a group of 89 non-strabismic ICS patients undergoing anti-suppression therapy in a private optometric practice. Figure 9 shows the average ICS treatment frequency for alternating flicker using liquid crystal lenses is 6.7 Hz (standard deviation = 1.6 Hz). Since Allen’s suppression treatment frequency of 9 Hz falls outside the 1.6 Hz standard deviation, the ICS treatment frequency is different from the TBI Trainer treatment frequency. Assuming the ICS data is correct, this difference in frequencies may imply either that the 9 Hz frequency is in error, or, more likely, that suppression in strabismus is a different phenomenon from ICS.

**A Reading Effect**

As the above data regarding treatment of ICS indicate, rapid alternate occlusion with liquid crystal lenses has allowed exploration of different frequencies of alternation. Hussey also reported on a positive reading effect at about 13 Hz. Seventy percent of patients who had been treated for ICS either reported oral reading as just “easier” or were observed with improved oral reading. Often, the words held still better. The effect is often small; certainly small in comparison to eliminating ICS. But occasionally it is subjectively quite significant for individual patients.

Frequency data were collected from 62 patients undergoing anti-suppression therapy. Typically, the suppression had been eliminated before testing for the reading...
The best reading frequency primarily was determined by observation of oral reading and patient reporting. Patients read while wearing the alternate occlusion goggles and reported any effects. Early on, we reasoned that equalizing input in central and para-central regions might have a beneficial reading effect. As an experiment, an Amsler grid was used in an attempt to equalize central and paracentral flicker effects and observe any subsequent reading effect. The determination was difficult for some patients, but yielded the first positive reading effect, and also frequencies comparable to reading observation.

This positive effect is a problem for recent magnocellular pathway (M-Pathway) and parvocellular pathway (P-Pathway) theory. Alternation at 12 to 15 Hz should occupy the M-pathway and make any M-pathway deficiency worse. So, how can we account for this positive effect?

One explanation is that this is just patient and therapist excitement over a new instrument. But, we documented the effect only after eliminating any ICS in therapy also using the alternate occlusion goggles - an average 37 uses in therapy at an average 6.7 Hz. It seems unlikely that merely altering frequencies would induce the reading effect. Related to the excitement theory is the Hawthorne/Placebo Effect: Anything helps. If this were true, we would expect almost any frequency to help, and we certainly would not expect a close clustering of frequencies that produce the effect. However, Figure 10 shows that the ICS and Reading Effect frequencies are two distinct non-overlapping frequency bands. That suggests this is not a random frequency effect.

Another possible explanation for this positive reading effect is a practice effect. Obviously the patients have to read in order to display a positive reading effect. That's practice. But, Hussey discussed individual cases that are difficult to explain with the practice effect. Kirsten was a 9-year-old girl who used her finger to read, unless she was wearing goggles alternating at 12 Hz. The effect was repeatable. If theoggles were removed, she went back to using her finger. Diana was a 52-year-old woman who could find notes in her music more easily at 12 Hz. As an accomplished musician, the practice effect should have been exhausted long prior to this. Diana stated, and it was reported at this presentation that she would be willing to use the goggles at symphony practices... but, not the concerts. These individual cases suggest a practice effect can't explain the positive 12 to 13 Hz reading effect.

A third possible explanation would be that the strobe effect of alternation with liquid crystal lenses stops motion and thereby stabilizes the picture. In order for
that to work, the strobe would have to freeze-frame fixation tremors in such a way that vision is stable. That requires looking at the different fixation drifts and flicks.

Fixation micro-nystagmus scans over a few cones, so a strobe effect has nothing to gain. Drifts are about 200 msec in length. Alternating at 13 Hz cuts a second into 13 frames for each eye, and each frame, therefore, is about 38 msec (77 msec per cycle). So, drifts would be cut into two or three seeing frames. Simply chopping the drift into spaced segments seems unlikely to stabilize the image. And, drifts and flicks are irregular in occurrence, forcing a secondary question of how a regularly occurring strobe effect would stabilize an irregularly occurring event. It is improbable that the strobe effect can explain how flicker positively affected reading in seventy percent of these patients.

Yo and Wilson looked at perception of flicker and calculated that the visual system had three broadly tuned channels for perceiving flicker: 5.5, 12, and 22 Hz.

Figure 11 shows an adaptation of their tuning graphs. Interestingly, their 5.5 and 12 Hz flicker sensitivity curves cross at 9 Hz, the TBI Trainer and Bartley phenomenon frequency.

Referring again to the present findings for comparison shows two distinct frequency bands for two different effects. ICS treatment and the positive reading effect (Figure 10). These data also show that the first two frequencies found by Yo and Wilson are within the standard deviations of the average frequencies of 6.7 (ICS) and 12.9 (Reading) Hz found on a group of 62 patients. Statistically, there is no argument between 6.7 (± 1.6) Hz and 5.5 Hz, and no argument between 12.9 (± 1.3) Hz and 12 Hz. This apparently, at a minimum, is a clinical validation of Yo and Wilson’s numbers. Also, the two means are more than two standard deviations away from each other, showing these to be two distinct, separate channels. These data suggest this is not a random effect.

Discussion

Yo and Wilson used a weighting factor to explain flicker perception in the periphery. This leads to the possibility that these channels represent a regionalization of the visual system. Our previous discussion of target sizes in ICS points in the same direction (Figure 7): the approximately 6 Hz flicker region probably corresponds to the central area that shows ICS. Yo and Wilson’s weighting factor implies the 22 Hz channel is in the periphery. That would leave the 12 Hz region in the paracentral, or intermediate area, perhaps 2° to 5° from fixation. As Garzia points out, the paramacular region is responsible for cascading imagery. That certainly fits with the reports of reading just being easier with alternation at 12 to 13 Hz. Figure 12 shows one possible regionalization schematic, following a likely anatomical basis for this: M-cell and P-cell receptive field distribution through the retina.

If there were any accuracy to this idea of regionalization based on flicker, then what would we expect if we could drive the neurology? First, we would expect 6 Hz alternation to override ICS. Second, 12 Hz alternation should improve sensation in the paracentral area which, logically, should help localization of targets in tasks such as reading. Third, given the crossing of sensitivity curves at 9 Hz (Figure 12), Merrill Allen’s 9 Hz alternation frequency in stra-
bismus may make perfect sense with the larger suppression areas in strabismus.36

But, M/P theory says that shouldn’t happen. Unless, perhaps, the anatomical location of effect is synapses. This reading effect is not necessarily an instantaneous effect. There is an average nine uses before we document a definite positive response - that is, roughly 90 minutes of use. Similarly, perhaps in ICS, we should not necessarily expect an immediate improvement in reading with simple occlusion. Eighteen months of constant occlusion has been used for reading problems where suppression was part of the diagnostic criteria for treatment, supporting the notion of a delayed, or slowly developing effect.37 If the effect is at synapses, it should take time to effect structural change. Synaptic modification is fundamental to behavior change and results from repeated, synchronous stimulation of the postsynaptic neuron by presynaptic neurons; in this case provided by the alternating flicker. Those synaptic changes develop over time. And, if the paracentral area is responsible for saccadic programming, logically that programming should take some time to normalize. If accurate, this brings us full circle back to synapses, likely at the LGN.

Conclusions and Application to Strabismus

Table 1 summarizes differences between ICS and strabismic suppression. The thrust of this summary is that ICS and strabismic suppression are two different entities that share some diagnostic similarities. This is not how the literature on suppression and reading (dyslexia) treats the subject of suppression, so care should be taken in evaluating conclusions suggesting suppression has no effect on reading.1

The present study hypothesizes that non-strabismic suppression occurs at LGN synapses. ICS, therefore, is likely an afferent defect. What happens if the same defect (ICS) occurs in early strabismus? If an eye turns, and a suppression occurs as an afferent defect - prior to the cortex - then a deprivation is occurring that will affect the development of the brain.39 Hubel and Wiesel showed that two to three months of light and form deprivation produced cell atrophy and synapse abnormality.40 42 This leads us to the conclusion that anyone who says “Let’s watch” an esotropia in an infant may be fostering cortical deprivation and abnormal development. Strabismic suppression may be a combination of LGN synapse malfunction and resultant cortical abnormality from sensory deprivation.

An area for future research would be exploring if it is possible to use flicker perception to drive development of the visual neurology in early esotropia. The appropriate choice of flicker stimulus frequency may be useful to differentially stimulate central or peripheral visual areas. Recent technologies such as functional magnetic resonance imaging may someday be able to map any flicker effects at specific visual pathway locations such as the LGN or the visual cortex.

Note
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