The conventional wisdom on suppression as a cortical competitive inhibition is difficult to reconcile with the clinical picture of intermittent central suppression (ICS). Equally difficult is to reconcile the suggested link of ICS to reading problems (symptoms of dyslexia) with the hard science on the visual pathways in dyslexia. These phenomena, products of the worlds of hard science and of the clinic, can be linked into a combined view of visual sensation through the perceptual fading of Troxler's phenomenon. This view can explain more of what we see in ICS, as well as provide clinical measurement of magnocellular pathway defect and, therefore, of (visual) dyslexia. Other implications of this theory are discussed. **Key Words: dyslexia, intermittent central suppression, magnocellular pathway.**

“Fix the suppression first.” That advice came from Louis Jaques, Sr. early in the 1950s. Strauss and Immerman gave the reason for worrying about suppression in the 1960s when they found *macular suppression*, defined as “an involuntary, temporary suspension of vision in one or both eyes” in non-strabismic subjects, significantly linked to reading problems. This was the first distancing of the diagnosis of this type of suppression from strabismic/amblyopic suppression.

Louis Annapole refined the definition of this type of suppression, introducing the term **intermittent central suppression**. The term intermittent central suppression (ICS) parallels Strauss and Immerman’s macular (i.e., central visual area) suppression but is more descriptive of a non-constant suppression of the central area of vision without suggesting an anatomical location for the neurological defect. That is, the suppression is in the central area of vision, but the neurological defect is probably not at the retinal level “in” the macula itself. I added the caveat that we are defining a non-strabismic, non-amblyopic suppression with this terminology.

The reason to follow Jaques’ advice is the controversial suggestion that intermittent central suppression is related to reading problems. Contradictory literature on suppression and reading disputes that suggestion. I’ve contended this dispute is in part simply a function of testing. These contradictory studies most often use strabismus-derived suppression tests. I found that a group of common-to-the-literature classic strabismus-derived suppression tests missed diagnosing ICS, which could be seen with routine vectographic testing. In fact, these strabismus-derived suppression tests didn’t even agree with each other when testing
non-strabismics. Obviously, assuming these tests to be equally valid as diagnostic tests for any single condition (as evidenced by their diagnostic use in research) must be erroneous. The actual flaw in all this may simply be in assuming that strabismic and non-strabismic suppression are precisely the same entity and that, therefore, any recognized test for one is automatically valid for the other. Other comparative studies on suppression tests for non-strabismics have not been done.

As part of defining this “missing link,” besides trying to establish some valid, reliable testing, I started looking at some characteristics of ICS and non-strabismic suppressors. Data from two separate groups of ICS suppressors suggest a typical ICS suppression would be a 2- to 5-second suspension of central vision, repeating at least once during any given 10-second period. These ICS patients tended to have eye movement and accommodative deficiencies, but the refractive condition was typically minimal; that is, passing a standard vision screening or typical refraction would be common in this group. This finding stands in contrast to the commonly significant refractive conditions of strabismus and amblyopia.

As might be expected with a visual function supposedly related to reading problems, ICS suppressors tend to have specific reading complaints. This is evidenced by complaints expressed by two separate groups of suppressors. That suggests ICS diagnosis should be attempted more routinely; certainly testing should be done when patients complain about reading or with a positive history of cervical trauma (whiplash). The documented vectographic (or routine stereoscopic) test techniques can diagnose ICS during routine examination, and I have argued that routine testing will give us fuller information about this anomaly. We should continue to search for better diagnostic techniques, as well as improved therapies. I’ve also suggested, on the basis of target sizes used in this testing along with patient responses, that the suppression zone is perhaps the central 1–3° of vision. That may reflect a motion sensitivity-based region- alization of the visual field that would therefore (because flicker is motion in stimulus form) be accessible through alternating flicker. New diagnostic techniques as well as more precise therapies may come from this if this understanding is correct.

I test for ICS as part of my routine examination in virtually all patients. I was able to diagnose ICS caused by whiplash cervical trauma because I had routine ICS diagnostic data prior to the cervical trauma. These remain the only documented cases where an optometrist could be in the right place at the right time with the right pre-existing information to see a suppression develop where no suppression had been seen in a previous examination. Other examples will occasionally follow if routine examination for ICS is followed in a relatively short time frame (after initial vision and ICS examination) by re-examination of the same patient after that patient has suffered whiplash.

In my routine examination, I use standard and modified stationary polarized targets at near and distance. When viewed through the phoropter and appropriate lenses, a patient with ICS will report the target will either black out or disappear (depending on the specific target) intermittently, indicating the suppression. This type of suppression occurs intermittently (“on and off” repeatedly) in the central area of vision, and 80%–90% of patients alternate. A typical patient exhibiting ICS behavior would suppress the central area of one eye for approximately 2 seconds, and then that eye’s picture would return. Either the other eye would then suppress immediately, or after a similar period of 2 or 3 seconds, either eye would suppress again. This entire sequence repeats, suggesting this is ongoing, typical visual behavior. In a typical routine diagnosis, I will spend 30–90 seconds timing the suppressions, sometimes video documenting a patient hand-signaling the suppression sequence.

In one of the documented whiplash patients, the complete “loop” of cause, effect, remediation, and recovery was shown: ICS appeared time-linked to and, therefore, an apparent consequence of whiplash trauma. Concurrently, reading suffered. Specific antisu suppression therapy eliminated the ICS, and reading then returned to subjective pre-trauma levels. This appearance of suppression occurred without other identifiable anomalies that might be expected after trauma, such as visual field defect. Without a field defect, tra-
motic involvement of the visual cortex seems unlikely. If we cast doubt on cortical involvement in a suppression because of how these suppressions developed, we must now reconsider the current cortical "fight or fuse" theories of suppression.

As a result of this ICS diagnosed after cervical trauma (without other vision defects developing concurrently with the ICS), I suggested the area of the LGN would be a logical locus of the ICS suppression. This follows from the anatomy: the LGN straddles the brain stem. Whiplash has been shown to damage the brain stem, so suggesting the LGN as the locus of the damage that created the suppression and, therefore, of ICS itself follows. 10 This finding suggests that ICS is an afferent neurological defect, an afferent postretinal, pre-cortical neurological defect linked to reading problems. I've pushed this concept further recently by suggesting that ICS is, in fact, the clinical diagnosis of magnocellular pathway defect and, therefore, "visual dyslexia" as defined by magnocellular defect. This afferent neurological defect tends to be associated with reading problems (symptoms of dyslexia) in the 650 or so ICS cases in the literature.

I've suggested a new theory of non-strabismic ICS in two recent articles in *Journal of Behavioral Optometry*. 11, 12 This new theory is simply an attempt to reconcile the ICS I see daily in private practice with all the hard sciences on vision in dyslexia and for suppression. As part of that literature reconciliation, the patient group has to be again defined: this group is non-strabismic and non-amblyopic. They typically are complaining about reading problems (symptoms of dyslexia). Often, they have minimal refractive errors, so a history of passing a vision screening or even a routine eye examination is common.

For a theory to be accepted, it must explain observable events. The conventional wisdom on suppression, well-supported in the literature on strabismus and amblyopia, is that suppression is a competitive inhibition at the cortex. The thought is that bilateral signals either fight or fuse at the cortex. It's important to realize that this literature comes not based on the populations of patients with straight eyes with equal aequities (ICS) discussed above but from strabismus and amblyopia research. However, the assumption in the literature is usually that whatever applies to strabismus and amblyopia will also apply to non-strabismics/non-amblyopes.

This cortical competitive inhibition has some problems when trying to explain the characteristics of and test responses we see in ICS. If cortical competitive inhibition is the mechanism for non-strabismic ICS, it must explain 1) the alternation of ICS, 2) the intermittency, 3) normal Wirt stereopsis associated with ICS, and 4) the finding of ICS after whiplash. How does a competitive fight or fuse mechanism explain the often almost rhythmical intermittency of ICS? And how can it explain the alternation? Wouldn't one side prevail, as in amblyopia? What mechanism is there in fight or fuse that would fight (suppression) on one side for 2 or 3 seconds and fuse as the suppression resolves and both eyes see again for a similar time period (with or without alignment), and then refight only this time on the other side as the other eye suppresses, this happening repeatedly? All this occurs in ICS as stereopsis (as typically measured in the literature; often the Wirt dot test) remains normal more than 80% of the time. 5, 6 How would competitive inhibition, using the same mechanism that decreases stereopsis in strabismus and amblyopia, retain stereopsis at these high levels in ICS? Finally, is the question of ICS that develops after whiplash: What other condition can be found that—in trauma—specifically increases neurological inhibition (an increase in neurological inhibitory activity), as its only effect on the neurology, as would be required with cortical competitive inhibition? And, because the site of neural changes according to the conventional wisdom would have to be the visual cortex, how do we damage the cortex to produce this ICS without the scotomata that might be expected with cortical trauma? The clinical entity ICS stretches the credibility of the conventional wisdom on suppression, but only when strabismus and amblyopia are excluded.

Another area of confusion is what ICS has to do with dyslexia. How do we reconcile the clinical entity ICS and its apparent negative effect on reading, with the hard science on dyslexia? As a means of dealing with the terminology of dyslexia, I've come to use the term "visual dyslexia," simply to focus on the vision problem that can affect reading. Whether this
is an appropriate use of the term dyslexia or not, I will leave to others. However, it is worth noting that much of the visual pathways research uses the term dyslexia, often simply defined as problems with reading.

The hard science on dyslexia (reading problems) suggests that a postretinal, pre cortical motion detection defect is present and probably in part responsible for the dyslexia. Two primary visual pathways carry visual information through the LGN to the cortex: the parvocellular (P-) and the magnocellular (M-) pathways. The P-pathway primarily carries detail and color to the brain. The M-pathway is primarily responsible for motion. (Much more complete descriptions of the neurology can be found elsewhere.) It is important to understand that this does not represent a simple central-peripheral conflict. Both the M- and P-pathways are most densely represented centrally. The P-pathway accounts for 80% of ganglion cells in the optic nerve. P-cells concentrate more toward the fovea, being 91% of the ganglion cells representing this area. P-cells continue out into the periphery, but they decrease in relative density with increasing eccentricity, being 40%–45% of the ganglion cells in the periphery. Ten percent of retinal ganglion cells in the optic nerve are M-cells. The M-pathway is represented in and density is greatest at the fovea, but still is only 5% of the ganglion cells connected there. Its absolute density declines with retinal eccentricity, but the relative density increases to 20% of ganglion cells in the periphery. The M-pathway simply becomes relatively more important in the periphery, whereas the P-pathway is the overwhelming preponderance of cells centrally and the somewhat less overwhelming preponderance of cells peripherally. But, importantly, if M-cells are concentrated most densely centrally (as the P-pathway is also), then we would expect the effect of a M-pathway deficit to be expressed most strongly centrally.

The persistent question with reading problems being blamed on a M-pathway motion deficit is simply: what is the mechanism? If ICS is related to reading problems as I and others have suggested, the picture of vision and dyslexia is further complicated. How does ICS fit in to the M-pathway deficit picture? A M-pathway deficit would be expected to be seen producing some sort of motion perception problem. But, as stated above, ICS is diagnosed with the patient viewing a non-moving target, which should, therefore, apparently not intimately involve the deficient M-pathway (i.e., deficient motion detection). How can these apparently disparate pieces of the puzzle be put together into one sensible picture?

To develop the cohesive picture, the suggested component pieces of the whole must be defined separately: patients with reading problems, but without strabismus or amblyopia; repetitive, ongoing intermittent loss of central visual sensation (ICS); a magnocellular—or motion—pathway deficit, all of this suggested as a postretinal, pre cortical defect. To these pieces we will add one other known quantity: Troxler’s effect or phenomenon.

First described in 1804, Troxler’s effect was originally defined as “the temporary and irregular fading or disappearance of a small object in the visual field during steady fixation.” This fading is a loss of detail and color (carried by the Parvocellular pathway). It occurs when motion is removed from the visual stimulus, in the early work simply by a subject developing ultrステady fixation. The early explanations for the fading included angioscotomat and receptor fatigue. Later work expanded into image stabilization experiments. Those later experiments found that Troxler’s “perceptual fading” is from lack of signal in the motion pathway, not receptor fatigue. The suggested location of this action is the lateral geniculate nucleus (i.e., this Troxler’s fading of detail is a postretinal, pre cortical consequence of lack of magnocellular signal).

Troxler’s effect provides the last piece of the puzzle to explain the mechanics of Troxler’s effect, ICS, and magnocellular deficit in reading problems. Too simply put, the magnocellular pathway is the “on switch” for seeing. If there is no—or perhaps a weak—magnocellular signal, the detail and color (P-pathway) fades. A deficient M-pathway doesn’t keep the P-pathway “awake,” allowing the P-pathway to fade. This will probably be seen most strongly or dramatically centrally because the cell density of both pathways is greatest centrally. So, a defective, or perhaps deficient, M-pathway causes the central visual detail to fade (i.e., if the M-pathway fails, the P-pathway fades). This is what we see as ICS.

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If this theory correctly ascribes the clinical entity ICS to M-pathway deficiency and, therefore, dyslexia, through Troxler's perceptive fading, it must more fully explain what we see in practice with ICS patients than does the conventional wisdom. For example, because this theory doesn't require cortical competitive inhibition of ICS, the effect of whiplash can be explained easily as disruption of synapses at the LGN. And, because there is no reason the P-pathway couldn't concurrently be neurologically intact in ICS (although getting inadequate instruction from the impaired "on switch," the M-pathway), stereopsis could be fine, depending on how tested. If stereopsis testing could be limited to the suppression "off" periods, perhaps some degradation would be found. But, during clinical testing, simply waiting for a suppression to resolve to an "on" period (probably while encouraging the patient to look more closely) should provide a stereopsis response representative of an intact P-pathway.

This ICS M-pathway theory can more easily explain the intermittency and alternation of ICS than the conventional cortical competitive inhibition wisdom. To explain the intermittency with the conventional wisdom, we have to imagine a cortical competitive inhibition that would be active for 2 seconds or so, then would (by what mechanism?) become inactive for a similar period of time, then would reactivate, this sequence to continue in a repetitive fashion as essentially a standard operational mode, not just an occasional occurrence. One can imagine an incomplete neurological control similar to static in a radio signal, but the continuing, almost rhythmical 2 or 3 seconds "on," 2 or 3 seconds "off" cycle is troubling if explained in so sketchy, so incomplete a fashion.

Or we might imagine some sort of neural capacitor that stores up charge and then periodically "fires." This is similar to how synapses work individually, but we now have to imagine that this neural capacitor works as a unit over the neurally dense P-pathway central vision at the cortex. Because synapses typically improve with simultaneous stimulation and firing, it seems likely that if this "capacitor" did exist, it would cease to exist as the area actually strengthened itself. How this would establish with trauma over a broad area of cortex is a problem. In addition, an injury that would disrupt cortical synapses to this point would also be expected to produce a field defect (or at least reduced sensitivity) and most likely a mild amblyopia. ICS is not associated with either.

If to these objections is added the alternation that affects 80%-90% of ICS patients, the conventional wisdom is in more trouble. What sort of a fight or fuse situation can be imagined that would not only alternate between fight and fuse but would then actually switch which signal wins the fight? These cortical mechanisms come up short in explaining what we see in ICS.

If we ignore the conventional wisdom as a possible mechanism for ICS (although competitive inhibition probably is involved to some extent in the detection and control of binocularity11), the sequence of events during an intermittent suppression can be explained fairly simply and readily by this magnocellular theory of suppression. If a Troxler's fade were to produce an image fade (a suppression) due to lack of magnocellular signal, we would expect that during the period of image fade the normal fixation lock would be decreased, or possibly absent centrally, Some drift in fixation would be likely. As the suppressed eye drifted off target, visual motion would be produced by the drift in aim and, therefore, the M-pathway signal would be increased from that motion induced by the drift. That increased M-signal would re-establish the image (P-).

Once the image was re-established, the brain would likely require proper realignment of images for single binocular vision. Motion in the signal would be maintained during realignment. But after some period of alignment, because the deficient M-pathway is still deficient, this sequence of events would likely repeat, allowing—or causing—another fade. This explains the intermittency. Because (excluding trauma for the moment) the M-pathway defect would likely be a developmental deficiency, both sides of the M-pathway as a whole would probably be affected, meaning simply that alternation is likely as well as the intermittency. With trauma, one side could easily be injured more than the other, so we might see a more one-sided suppression. Unfortunately, in children especially, the patient
history probably won’t reveal all the potential whiplash-like trauma that might produce ICS, starting with birth trauma and proceeding through all the trials of an active childhood. If we accept the M-pathway dyslexia literature, the literature on Troxler’s perceptual fading, and my suggestion about ICS based on whiplash, then all of this occurs as an afferent sensory neurological defect. It’s worth noting here also, that, in theory, with a sufficiently defective M-pathway, we could actually see a “bilateral suppression” (i.e., both sides of an ICS target such as the bisected diamond target disappearing simultaneously). 6

I’ve suggested research proofs for this theory elsewhere, based on fixation behavior during suppressions. 11 This theory also suggests some of the behaviors and, therefore, complaints we should expect in patients with ICS and “visual dyslexia.” As a suppression occurs and some drift in aim occurs, some letter and word confusion would happen as the suppression resolved and both eyes were now seeing simultaneously, but not precisely aligned (1° off target corresponds to two or three letters in standard print). Visual confusion would result from the misalignment, and some movement would occur as the brain demanded realignment of the misaligned images. If my suggestion that the suppression zone is perhaps the central 1–3° of vision is accurate, smaller words would more likely be confused than larger, in part because relatively more of a small word will fit in the central suppression confusion area. Because small words might be expected to be affected more, textbooks would probably cause more confusion than novels simply because small words count more significantly for content in a text than in a novel.

Variability in how specific words are read would occur, depending on the fixation misalignment at the precise time the word is sighted. That variability would include some correct reading when aim is correct. So a common complaint from a parent might be about “memory”: A word appearing several times in a story would be read wrong. The parent would tell the child to “go back and look at it.” During that time of pondering the faulty word, the suppression would have time to resolve (along with faulty eye movements no longer complicating viewing), resulting in correctly reading the word. Moving on, this might happen again, only perhaps the word would be read inaccurately differently from the earlier mis-read. “Go back an look at it!” This would proceed with the suppression sequence happening at its own separate timing, irrespective of what is on the printed page. The basic level of familiarity with the specific word would also likely be involved. Accurate reading would occur occasionally simply because accurate alignment happened to randomly occur at the time that word again appeared in the story. At that point, the parent would think the child had finally learned. But when a later misalignment occurred as the reading continued and the same word was again misread (perhaps differently), the parent or teacher would be inclined to declare the child’s memory as defective, as the child runs for cover. It happens in my practice.

Other logical and probable consequences and behaviors could be discussed, but perhaps as important are the larger implications of this theory. First, a diagnosis of ICS can also be considered a diagnosis of “visual” dyslexia. Again, this term comes from the M-pathway literature that often uses the term “dyslexia” along with my desire to limit the term to vision. Because M-pathway defect is responsible for ICS, we also now have a measure of central M-pathway function. Conversely, as the suppression is eliminated with vision therapy (the only documented treatment), this implies measurable improvement in the M-pathway. Prior to this suggestion, M-pathway defect as well as improvement were clinically unmeasurable. 14 And yet, it is fairly common to hear at optometric educational meetings how we are treating magnocellular deficiencies. How can we suggest we are changing this pathway when we can’t measure it? This magnocellular theory of suppression provides for measurement, treatment, and remeasurement of the magnocellular defect.

Second, magnocellular defect producing ICS provides a theoretical means to produce the deprivation that could produce the constant suppression of strabismus and amblyopia. Cortical competitive inhibition in strabismus and amblyopia is well supported in the literature. But a motion pathway mediated afferent loss of visual sensation as the first step in sensory deprivation leading to maldevelopment could provide the mechanism to account
for that lack of development of one side. Obviously, more work needs to be done to determine if this is the mechanism.

Third, some sort of accommodative malfunction should be expected with ICS. During a Troxler’s fade, accommodation “fades” toward a rest position. So, with ICS mediated by magnocellular defect, we’d expect to see some accommodative variability and inaccuracy. We might also expect some changes in accommodation with the elimination of the ICS.

Fourth, ICS as a function of magnocellular defect may give us information on the visual world of those afflicted with Alzheimer’s disease. The M-pathway is selectively affected in Alzheimer’s. If this theory is accurate, then we should see some similarities in vision between the two groups except that because the pathway is damaged, not just deficient, in Alzheimer’s, we might expect an exaggeration of any vision problems. That may mean that Alzheimer’s patients are descending into a fractured, visually unstable world as a P-pathway without sufficient “on-switch” support of the M-pathway struggles to see. Where a visual dyslexic has a constantly changing visual perception, including changing and moving words, an Alzheimer’s sufferer may have an outright “blank-out” of vision, perhaps followed by broad sweeps of motion as vision is re-established. This will be difficult to test, because ICS tests are at present subjective, but it may be worth discussing with family members. Perhaps, it should be standard practice to verbally identify oneself when speaking with Alzheimer’s sufferers because they may, in fact, not be seeing you the same way they saw you at the last visit.

Fifth, as an afferent defect, ICS would be expected to affect all perception that occurs further up into the cerebrum. Therefore, in my opinion, ICS should be the first visual anomaly to be treated with vision therapy. This echoes the words of Louis Jaques of some 50 years ago.

Sixth, I would again suggest that routine examination is preferable in looking for ICS. As stated above, I typically use vectographic targets at both distance and near in my routine examination to diagnose ICS. The advantage, of course, is a broader look at ICS in the population. Routine examination allowed the discovery of ICS as a function of whiplash cervical trauma, simply because patients were given the same examinations before trauma during a routine vision examination and then again after the trauma so that a direct before and after comparison could be made. Other tests are available. The stereoscope with appropriate questioning can be a very sensitive ICS diagnostic instrument and has been a large part of ICS diagnosis in the literature. The VO Star can be used with careful interpretation. However, if these other tests are to be used, besides my basic criticism of their tendency to be “special tests,” we owe it to our patients to test their validity in ICS. The tests I’ve compared include Wirt stereopsis, the Worth 4-dot, color luster, the Jampolsky 4-prism test as well as routine vectographic tests. When dealing with non-strabismics and non-amblyopes, these “special tests”—Wirt stereopsis, the Worth 4-dot, color luster, and the 4-prism test—don’t agree with each other in diagnosing ICS, as well as diagnosing ICS at a much lower rate than the routine vectographic tests.

If this theory is correct, the neurophysiology described will teach us how to treat. It should not surprise anyone that with the suggested defect at LGN synapses and, therefore, a need to change those synapses for elimination of the suppression, the only documented treatment for ICS is active vision therapy. Therapies will revolve around the defect in neurology and the changing of those synapses. We know we’ve got to force both eyes to see simultaneously, and we know the underlying defect is in the motion pathway. So, both those should be combined in any suppression therapy. That would include dissociated rotations, cheiioscopic tracings of all sorts, and VO stars. That would not include simply looking at red-green targets. In addition because we’re suggesting altering synapses, although perhaps color would help in choosing or highlighting the specific pathway on which we want to perform a specific therapy, simply gazing at a non-moving target in a specific color or with a specific color of light would not be expected to change synapses.

Changing synapses in the magnocellular pathway at the LGN would include alternating flicker. On-off flicker is motion in stimulus form. I’ve suggested there may be a motion
sensitivity-based regionalization of the visual field that would, therefore, be accessible through different rates of alternating flicker (differing “speeds” of motion stimulus). The brain reads alternation at an appropriate pace as a continuous signal centrally, and the flicker is a strong motion signal. In all forms of anti-suppression therapy, it’s worth remembering that, according to this theory, we are either building or strengthening synapses at the LGN. This takes time.

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