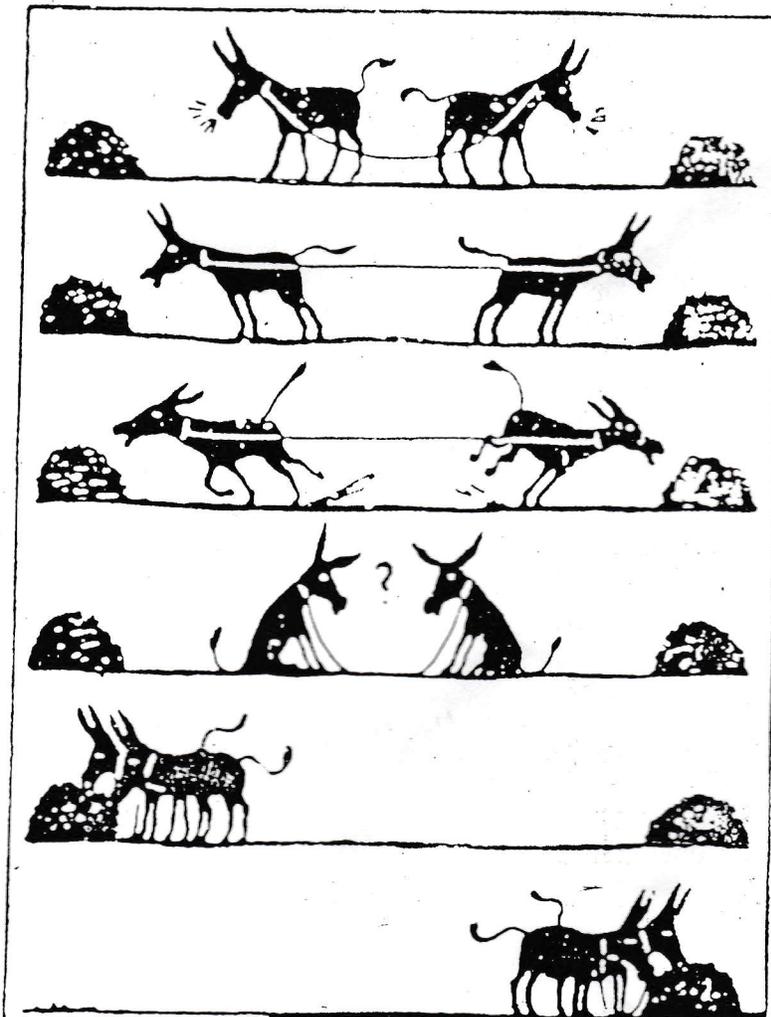


# OPTOMETRY TODAY

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## No Need for Words

*This illustration from Ottica Italiana needs no explanation, and it seemed appropriate to publish it at this time of the year to remind everyone of the need to work together in order to attain objectives! Please also see Editorial on page 104.*

# If the M-pathway fails, the P-pathway fades

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I've suggested a new theory of non-strabismic intermittent central suppression (ICS) in two recent articles in *Journal of Behavioral Optometry*.<sup>1,2</sup> This new theory is simply an attempt to reconcile what I see daily in private practice with the hard sciences on dyslexia and on suppression. We'll start by defining the patient group: 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.

In my private practice, I commonly diagnose intermittent central suppression during routine examination<sup>3</sup>. As one of several stationary polarized near or distance targets during routine examination is 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, indicative of the suppression. This type of suppression occurs intermittently ("on and off" repeatedly), is in the central area of vision, and 90% of patients alternate. A typical ICS patient would suppress the central area of one eye for approximately two seconds, then that eye's picture would return. Either the other eye would then suppress immediately, or after a similar period of two or three seconds, either eye would suppress again. This entire sequence, then repeats. In a typical routine diagnosis, I will spend 30 to 90 seconds timing the suppressions, sometimes video documenting the suppression.

I've diagnosed this ICS apparently developing as a result of whiplash cervical trauma. Other research on whiplash and vision suggests the brain stem is injured in whiplash. Since the lateral geniculate nucleus (LGN) straddles the brain stem, I suggested the LGN is a logical location for the acquired ICS<sup>4</sup>. These patients with acquired ICS showed symptoms similar to the reading problem children with ICS display. Importantly, this would define ICS as a post-retinal/pre-cortical neurological defect;

that is, an afferent defect. This neurological defect tends to be associated with reading problems (symptoms of dyslexia) in the 650 or so ICS cases in the literature.

In order for a theory to be accepted, it must explain observable events. The conventional wisdom on suppression, well supported in the literature, is that suppression is a competitive inhibition at the cortex. The thought is that signals either "fight or fuse" at the cortex. It's important to realize that the conventional wisdom in this area comes from strabismus and amblyopia research, NOT from a population of straight eyed patients with equal acuities, part of the definition of ICS. However, the assumption in the literature is usually that whatever applies to strabismus and amblyopia will apply to non-strabismics/non-amblyopes.

This cortical competitive inhibition has some problems when trying to explain ICS. If cortical competitive inhibition is the mechanism for non-strabismic ICS, then it must explain

- 1) the alternation of ICS
- 2) the intermittency
- 3) stereopsis in ICS, and
- 4) the finding of ICS after whiplash.

How does a competitive fight or fuse mechanism explain the almost rhythmical intermittency of ICS? And, how would it explain the alternation? Wouldn't one side prevail, as in amblyopia? What mechanism is there in "fight or fuse" that would fight (suppress) on one side for two or three seconds, then fuse as the suppression resolves and both eyes see again for a similar time period, then re-fight 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) usually remains normal. How would competitive inhibition, using the same mechanism that decreases stereopsis in strabismus and amblyopia, retain stereopsis 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 affect on the neurology as would be required with cortical competitive inhibition? And, this ICS shows without the scotomata that might be expected with cortical trauma, which should be the site of neural changes according to the conventional wisdom. The clinical entity ICS is at odds with the conventional wisdom of suppression.

The next question is how to reconcile the clinical entity ICS, which apparently affects reading negatively, 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 that 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", usually simply defined as problems with reading. This literature suggests that a post-retinal, pre-cortical motion detection defect is present in dyslexia (reading problems). This literature defines two primary visual pathways: 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 this can be found elsewhere, see reference 1 for a short review and a listing of some other reviews.) It is worth noting at this point 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 decrease in relative density with increasing eccentricity, being 40 to 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, while 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 an M-pathway deficit to be expressed most strongly centrally.

The persistent question with reading problems being blamed on an M-pathway motion deficit is simply: what is the mechanism? To further complicate this picture, if ICS is related to reading problems as I and others have suggested, how does that fit in to the M-pathway deficit picture, since the patient is viewing a non-moving target which should, therefore, *apparently* not intimately involve the deficient M-pathway? How can all this be put together into a cohesive picture?

To develop the cohesive picture, the components should be assembled: patients with reading problems, no strabismus or amblyopia, intermittent loss of central visual sensation (ICS), a magnocellular - or motion - pathway deficit, all of this suggested as a post-retinal, pre-cortical defect. To this picture we will add one other known quantity: Troxler's Effect or Phenomenon.

Troxler's Effect was first documented late in the 1800s. It was originally noted as "the temporary and irregular fading or disappearance of a small object in the visual field during steady fixation"<sup>5</sup>. This fading is a loss of detail and color (carried by the Parvocellular pathway). It occurs when motion is removed from the visual stimulus. The early explanations for the fading included angioscotomata and receptor fatigue. Later work expanded to include image stabilization experiments, finding that Troxler's "perceptual fading" is a function of lack of signal in the motion pathway, not receptor fatigue. The suggested location of this action is the lateral geniculate nucleus; that is, this Troxler's fading of detail is a post-retinal, pre-cortical consequence of lack of magnocellular signal.

As all this is put together into a cohesive picture, we can develop a simple conception of the mechanics of Troxler's effect, ICS, and Magnocellular deficit in reading problems: The Magnocellular pathway is the "on switch" for seeing. If there is no 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, typically centrally since the cell density of both pathways is greatest centrally. So, a defective, or perhaps deficient M-pathway causes the central visual detail to fade; that is, if the M-pathway fails, the P-pathway fades. This is what we see as ICS.

Can this theory of ICS as a function of M-pathway deficiency more fully explain some of what we see in practice? It successfully combines ICS, M-pathway theory, and, therefore, dyslexia. And, since it doesn't involve competitive inhibition, the effect of whiplash can be explained easily as disruption of synapses at the LGN. Since the P-pathway is neurologically intact (although getting inadequate instruction from the impaired M-pathway), stereopsis should be fine, depending on how tested. If stereopsis testing could be limited to the suppression "off" periods, perhaps some degradation would be found.

Further, this ICS M-pathway theory can explain the questions that are difficult to explain with cortical competitive inhibition: the intermittency and alternation of ICS. Applying what we suspect about the sequence of events during a suppression, we would expect that during the period of image fade, the normal fixation lock would be decreased, or possibly absent centrally. Some fixation drift would be likely. As the suppressed eye drifted off target, the M-pathway signal would be increased from the induced motion of the drift. That increased M-signal would re-establish the image (P-). This sequence of events would likely repeat, since the deficient pathway is still deficient, allowing - or causing - another fade. This explains the intermittency. Since (excluding trauma for the moment) an M-pathway defect would likely be a developmental deficiency, the M-pathway as a whole would likely be affected, meaning simply that alternation is likely as well as the intermittency. It's worth noting here also, that, in theory, with a sufficiently defective M-pathway, we could actually see a "bilateral suppression", that is, both sides disappearing simultaneously. All of this probably occurs as an afferent defect if we are to accept the M-pathway dyslexia literature, the literature on Troxler's perceptual

fading, as well as my suggestion about ICS based on whiplash.

This also suggests some of the behaviors we should expect to see in 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 (one degree off target corresponds to two or three letters in standard print). Visual confusion would result from the misalignment, then some movement would occur as the brain demanded realignment of the non-aligned images. Smaller words would more likely be confused than larger, in part because relatively more of a small word will fit in the suppression confusion area. Therefore, texts would probably cause more confusion than novels simply because small words count more in 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.

Other logical consequences can 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. Since the M-pathway is now at fault in this, we also have a measure of 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 not measurable. How could we suggest we are changing this pathway when we couldn't even measure it?

Second, magnocellular defect producing ICS may provide a 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 could provide the mechanism to account for that development. Obviously more work needs to be done to determine if this is the mechanism.

Third, accommodative malfunction should be expected with ICS. During a Troxler's fade, accommodation "fades" toward a rest position. So, with ICS, 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, since 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 may be descending into a fractured, visually unstable world as a P-pathway without sufficient "on-switch" support of the M-pathway struggles to see. This will be difficult to test, since ICS tests are subjective, but may be worth discussing with family members. Perhaps it should be standard practice to verbally identify oneself when speaking with Alzheimer's sufferers since 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 Jacques of some 50 years ago.

### **Some Notes on Diagnosis and Treatment**

As stated above, I typically use vectographic targets at both distance and near in 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, we owe it to our patients to test the validity. The tests I've compared include stereopsis, the Worth 4-dot, color luster, the 4-prism test and vectographic tests. When dealing with non-strabismics and non-amblyopes, those tests: 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 vectographic tests.

The only documented treatment of ICS is vision therapy. Therapies will revolve around the neurology. We know we've got to force both eyes to see simultaneously, and we know motion is the underlying defect. So, both those should be combined in any suppression therapy. That would include dissociated rotations, cheirosopic tracings and VO stars. It would also include alternating flicker. On-off flicker is motion in stimulus form. Alternation at an appropriate pace is read by the brain as a continuous signal, and the flicker is a strong motion signal<sup>6</sup>. 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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