Article ▸ Increases in Binocularity Periods with Treatment of Intermittent Central Suppression Contradict Suppression as Solely Inhibitory

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ABSTRACT

Intermittent central suppression (ICS), an intermittent loss of central visual sensation, has been associated with reading problems. In a prior report, ICS was treated using electronic rapid alternate occlusion as a stand-alone home therapy in a group of Job Corps students (n=26). The results from that study showed a decrease in suppression, a decrease in symptoms, and an increase in reading level (n=18). A further analysis of the decreased length of timed suppression periods versus the increased length of timed binocular periods suggests that the actual changes do not match changes that would be expected if suppression were exclusively an inhibition. If suppression is entirely inhibitory, anti-suppression therapy should decrease the inhibition, decreasing the length of suppression periods. However, decreasing an inhibition should not necessarily increase the length of non-inhibited, normal-sight binocular periods. The actual changes in suppression versus binocular periods in this group, however, showed an increase in length of binocular periods that was greater than the decrease in suppressed periods. This change is difficult to reconcile with the view of suppression as solely a cortical inhibition.

Keywords: binocularity, dyslexia, intermittent central suppression, suppression

Introduction

Suppression of binocular vision (ICD 368.31) is a defect affecting the sensory combination of two eyes' images into actual binocularity (simultaneous two-eyed, bi-foveal vision). Suppression remains a primary focus of the investigation of visual sensation in strabismus and amblyopia. However, abnormal binocular sensation in the form of suppression of binocular vision also occurs in the absence of strabismus and amblyopia. This is known as intermittent central suppression (ICS).[1,2] As the name implies, ICS is a repetitive on-and-off form of loss of central visual sensation.[3] This suppression intermittency contrasts with the more constant suppression of strabismus and amblyopia.

It is probably fair to say that the vast majority of research on the sensory defect of suppression has focused on strabismus and amblyopia.4 A fairly descriptive term for the accepted reason suppression develops in strabismus and amblyopia, diplopiaphobia, was applied by Bielschowski in the 1940s.5 That term suggests that diplopia and visual confusion from misaligned eyes would be biologically unacceptable, producing rivalry and then a phobic response cortically. That phobic response would trigger neural inhibition to suppress one image, preventing diplopia and therefore preventing visual confusion.[4-6] As described by the term diplopiaphobia, suppression is a logical way for the visual system, or the cortex, to deal with diplopia.

Clinically that all makes sense, but on closer inspection, explaining suppression as entirely a cortical inhibition can prove unsatisfying. Abhorrent diplopia does not always trigger suppression, or we would not bother using prism following a trauma-produced diplopia. If a suppression developed fairly quickly after trauma, or maybe, in a decompensating

convergence insufficiency, we would expect diplopia to be transient, rather than constant. However, sudden-onset diplopia in an adult, diplopia that apparently can't produce a constant suppression, will justifiably lead to cascades of diagnostic tests.[7] Confusion-compensating cortical-inhibition suppression seems selective in its appearance.

If diplopia-triggered rivalry gets some of the blame for the genesis of suppression, the suspected mechanism would involve a rivalrous visual alternation that leads to one eye being "chosen" and therefore the fellow eye being "unchosen" and suppressed. Certainly, the diplopia from a traumatic strabismus or a decompensating convergence insufficiency would be expected to produce rivalrous conditions. Since rivalry experiments routinely use adults as subjects,[8,9] we can safely assume that it can happen in adults. Consequently, we would assume that conditions are suitable to produce rivalry with any sustained diplopia. Further, the ability to "drive" one eye's image to the point of excluding the other eye's image can be seen in experiments (adult subjects, again) on continuous flash suppression and binocular switch suppression.[10,11] Therefore, rivalry should take over and cause inhibition to develop fully as constant suppression in newly-diplopic adults. However, even with the "neurological parts" present for both rivalry and to have one eye dominate, diplopia only sometimes causes such complete inhibition at the cortex that a suppression develops. Perhaps this occurs only in children at a neurologically early, developmentally sensitive period.

Diplopia, then, does not always produce suppression. Similarly, it does not always precede the development of suppression, at least in non-strabismics. Non-strabismic ICS has been documented as occurring after whiplash in adults.[12] If we accept the rivalry explanation for suppression development, then data from these injured but non-strabismic suppressors may limit possible explanations for the suppression development. The whiplash victims could have been strabismic immediately after the trauma, producing short-term rivalry. These patients did not complain of diplopia post-trauma, but perhaps the psychological portion of the trauma prevented them from recognizing diplopia and also a very, very short-term rivalry that subsequently triggered a rivalry-produced suppression. That theory falls short in explaining the trauma-produced strabismics who don't develop a compensating suppression, requiring prism or patching to stop the diplopia. These whiplash victims were adults, as are many traumatic strabismics. As stated above, test subjects in rivalry experiments are usually also adults. The same neural pathways are present in the whiplash sufferers as in the diplopic traumatic strabismics. Diplopia is abhorrent, whether present momentarily or for the rest of a lifetime. These whiplash victims present a challenge and a contrast to diplopiaphobia-produced suppression, the conventional wisdom.

The diplopiaphobia model breaks down further when we consider other attributes of non-strabismic, non-amblyopic intermittent central suppression. Not only does ICS not, as far as is known, have diplopia as a triggering condition, but recent evidence suggests that, rather than eliminating diplopia and visual confusion as is presumed to happen in the suppression of strabismus, the suppression of ICS might actually produce visual confusion that harms reading ability. As the image from either eye intermittently is lost during the ICS suppression periods, and then regained during bilateral-sight "binocular" periods, any error in eye aiming would demand correction to avoid strabismus-like visual confusion. Any correcting vergence movement to avoid that visual confusion would have to be repeated whenever a suppression period resolved with an aiming error of whatever small degree. Logically, visual tasks requiring constancy in visual detail such as reading might be

Table 1: Pre- and post-vision therapy data with significance levels

	Pre-Therapy	Post-Therapy	Change	p<<
Suppression Periods (sec)	2.5±1.1	1.02±1.04	-1.48±1.36	0.0001
Binocular Periods (sec)	2.8±2.0	6.8±3.3	+3.9±3.3	0.0001
COVD Checklist Full Score	50.6±19.2	32.7±14.1	-17.96±17.65	0.0001
COVD Checklist Reading Questions[2]	30.4±9.2	17.4±8.3	-13.1±11.5	0.0001
TABE Reading Scores (grade level)	4.9±1.7	8.6±2.4	+3.7±2.6	0.0001

Table 2: Confidence intervals for pre-post therapy changes (nos. rounded)

	Pre-Post Therapy Difference	95% Confidence Interval of the Difference Lower	I	Upper
Suppression Periods (sec)	-1.5±1.4	-2.0		-0.9
Binocular Periods (sec)	+3.9±3.3	2.6		5.3
COVD Checklist Full Score	-18±17.6	-25.1		-10.8
COVD Checklist Reading Questions[2]	-13.1±11.5	-17.7		-8.4
TABE Reading Scores (grade level)	+3.7±2.6	2.4		5.0

The subject is signaling his suppression sequence – hand up when half the diamond is black.

affected. Conversely, correction of the ICS might be expected to improve symptoms of reading difficulty, as well as reading scores. This is a strikingly different scenario than diplopiaphobia presents with its underlying theoretical rationale as a reducer of visual confusion, almost the polar opposite.

To attempt to clarify the differences between ICS and a diplopiaphobia suppression, the data from a recent paper[2] on remote treatment of ICS supporting improvement in both symptoms and reading scores with reduction of ICS are reiterated here. Beyond these prior results, a supplemental data analysis from those patients is presented, suggesting that suppression as an exclusively inhibitory cortical defect may be in error.

The Job Corps Study

Clinical data were collected from 26 young adult (age 19.7±1.6 years) Job Corps students enrolled at an isolated Job Corps educational and vocational site. Those students had been diagnosed with ICS and then were treated at the Job Corps site with electronic rapid alternate occlusion as the only form of anti-suppression and vision therapy. Diagnosis occurred at a private clinic, but distance between the clinic and the Job Corps site in combination with transportation constraints required treatment on site at Job Corps. Students were to wear liquid crystal goggles alternating at 5 Hz while sitting reading (literally, reading anything printed) starting at five minutes per session working up to 40 minutes per session over an average six month treatment period. Student reports of usage, though imperfect, suggested total usage of roughly thirty therapy-hours, similar to usage reported by Miller et al.[13] Three

to five sessions per week during Job Corps education weeks (education weeks alternate with trade instruction weeks at Job Corps) were requested as well as follow-up examination when allowed by Job Corps' schedule. Data from 26 students are summarized here, including decrease in suppression periods, increase in binocular periods, decrease in symptoms as measured by the COVD symptom checklist, and improved reading scores (n=18).[2]

Table 1 shows the previously reported pre- and post-therapy suppression timings, symptom and reading scores, as well as the significance levels (p-values) for the changes. Table 2 shows 95 percent confidence levels as shown in the original paper. These data reflect both diagnostic characteristics of ICS and therapeutic changes after treatment of ICS.

Looking first at diagnostic, or pre-therapy, characteristics, a typical intermittent central suppression patient in this group would show an "on-off" cycle of suppression of about 2- to 3-second suppression periods spaced by 3- to 4-second periods of bilateral sight, or maybe loosely, binocular periods.

The temporal characteristics of the suppression periods make this group diagnostically very similar to groups reported on prior to this. Both suppression period length and binocular period length overlap with timing in a prior video-documented and -timed group.[3]

In addition, pre-therapy data suggest that ICS does negatively impact vision. Possibly more accurately, this intermittent break in sensory binocularity apparently does reduce stability of visual sensation and therefore increases confusion in visual perception. If, as expected in diplopiaphobic strabismic suppression, visual confusion were reduced, we might expect low visual symptom levels as measured by Quality of Life checklists. We see the opposite in this group. With COVD QOL checklist scores of 20 or higher considered indicative of visual problems, the Job Corps scores averaging 30 denote a high symptom level.[14]

If visual confusion is actually increased in ICS, another symptom we might expect would be impaired reading. The pre-therapy 5th grade reading level in the 18 adult students who reported reading level scores supports the suggestion that ICS interferes with reading, a sustained, detail-heavy visual task. We must acknowledge that many things can affect reading scores, including educational opportunities and family situations. But on a diagnostic level, these data support the description of typical ICS as an intermittent (on-and-off) suppression of central vision with a temporal pattern of 2- to 3-second suppressions spaced by 3 to 4 seconds of bilateral sight and that those intermittent suppressions interfere with reliable detection of detail, increasing symptoms and impairing reading efficiency.

Post-therapy data illustrate some therapeutic effect from the treatment. Suppression periods decreased, binocular periods increased, symptoms decreased, and reading scores increased. The reading scores in this group improved by almost four years over the 6-month treatment period. Data from Table 2 support pre- to post-therapy changes as being real and significant. As shown by these data, if ICS is treated, in this case by electronic rapid alternate occlusion at a 5Hz pace, we can expect that as the suppression periods decrease and binocular periods increase, symptoms should improve. Assuming proper reading preparedness, we might expect some improvement in tested reading level as well. The curious outcome of this treatment group is not that length of suppression periods decreased since the argument has been made that treatment should reduce suppression, but that periods of binocularity, perhaps more accurately bilateral visibility, increased more than suppression periods decreased. The average increase in time which patients

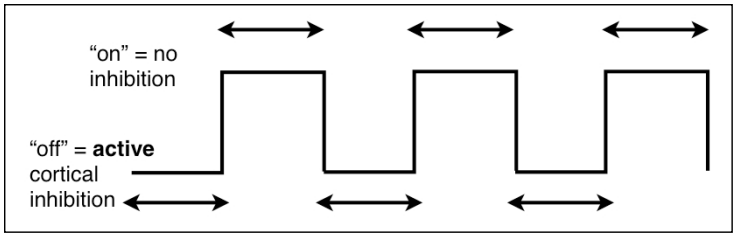

***Figure 1**: Black arrows represent original time in "on" or "off" phase of ICS*

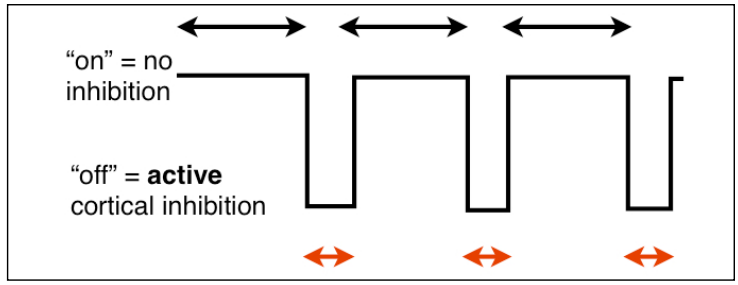

***Figure 2:** Red arrows represent anticipated post-therapy changes*

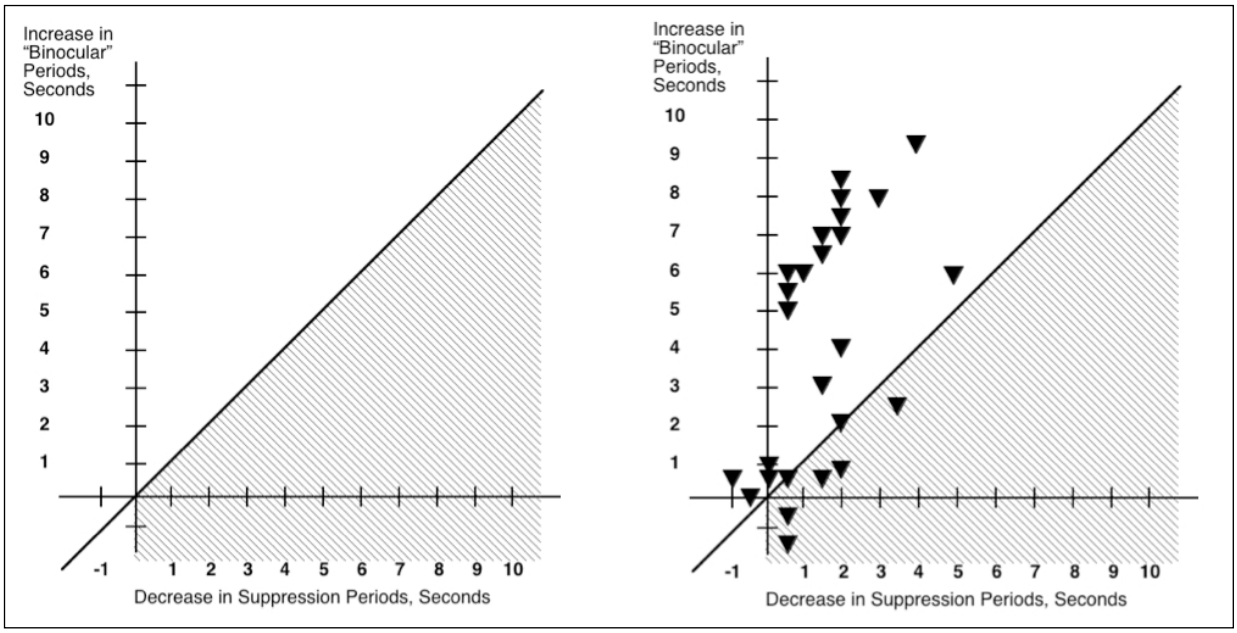

***Figure 3a and 3b:** Shaded area in both figures shows where data points are expected, Figure 3b shows actual data points.*

spend in periods of binocular vision (bilateral visibility) was larger than the average decrease in suppressed-period time. This change deserves consideration.

Suppression Period Changes

As treatment occurs, the periodicity of the suppression would be expected to change. If we assume a cortical suppression as in diplopiaphobic suppression, some predictability might be expected in the change in the on-off periodicity. That is, if we assume that suppression is entirely a cortical inhibition, then we would expect changes in that inhibition with treatment. That (presumed) cortical inhibition is reported by patients during the vectographic examination as a change in target visibility, as targets disappearing or blacking out.[1-3] Reducing active cortical inhibition should decrease that interference in target visibility. Clinically we would then expect reported suppression periods to be shorter.

However, since the "on" periods, or periods where visibility is not compromised by cortical inhibition, are just normal transmission of the visual signal, we would not necessarily expect some sort of change in the length of those unencumbered normal-transmission, normal-sight periods. Certainly, if suppression periods were eradicated, the normal-sight "on" periods would merge into a longer "on" period. However, in a group showing significant reduction but not necessarily an extinguishing of the suppression inhibitory periods, the expectation would be reduction in suppression periods, not lengthening of binocular bilateral-sight periods.

An oversimplification of this idea might be illustrated by viewing the original suppression as a square-wave on-off sequence (Figure

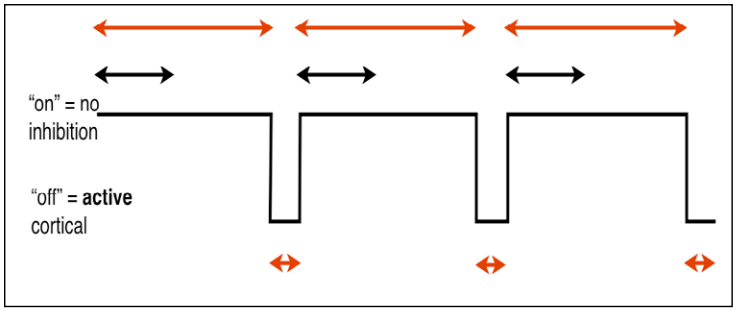

Figure 4: *Red arrows represent actual post-therapy changes vs. original timing (black arrows).* **CLICK HERE for an animation of the graphics described in this paper.**

1). The "on" segments would be periods of normally functioning vision. The "off" segments would be cortical active-inhibition segments during which normal visibility is suspended.

With anti-suppression therapy reducing the (presumed) active cortical inhibition, the "off" periods of active cortical inhibition should decrease (Figure 2).

The measured changes in individuals' "on" binocular periods versus the "off" (presumed) active inhibition periods from the prior study can be graphed. Figure 3a shows coordinate axes without data, labeled to plot increases in binocularity versus decreases in suppression. We would expect, if pre- to post-therapy changes are graphed as suggested in figure 3a, and if the decrease in suppression periods theoretically should be the greater change (Figure 2), the data points should be below the equality of change 45° line, and therefore in the shaded area of the figure. However, graphing the actual data shows a different story (Figure 3b) with the increase in binocularity periods exceeding the decrease in suppression periods. This suggests that the positive increase in visibility of targets versus decreasing inhibition (possibly masking[15]) of targets[16] may actually be the bigger share of anti-suppression therapy, at least absent strabismus and amblyopia.

A schematic representation of the actual outcome is shown in Figure 4. The simple square wave representation of the on-off sequence of ICS has changed to show both a significant decrease in suppression inhibitory periods as well as a significant increase in the binocular bilateral-visibility periods. The actual data show that the bilateral-visibility periods actually grew more than the suppression periods decreased in seconds.

The conventional wisdom on suppression defines suppression as a cortical inhibition. These data suggest perhaps some modification of the conventional wisdom is in order, at least in the case of non-strabismic, non-amblyopic ICS. Reduction in inhibition doesn't seem to explain the changes seen in this group in their ICS. However, if instead of defining all suppression as cortical inhibition, we instead define anti-suppression therapy as increasing visibility of the bilateral images, these data may make more sense. Perhaps when part of strabismus and amblyopia, this is related to the monocular amblyopic attenuator suggested at the lateral geniculate nucleus.[16] A subsequent paper will address this issue more thoroughly from a vision science perspective.

Conclusions

These data suggest that explanations for suppression beyond cortical inhibition may be necessary. On a clinical level, treatment of intermittent central suppression, whether by electronic rapid alternate occlusion or by more traditional therapies, can be beneficial to our patients. Increased periods of binocularity may decrease visual confusion that interferes with reliable sustained visual perception of central detail, decreasing visual symptom levels and possibly improving performance in measures such as reading levels.

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