AN IDEALIZED EXPERIMENT ON THE EFFECT OF DECREASING MAGNOCELLULAR SIGNAL ON VISUAL SENSATION

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Abstract

Idealized or "thought" experiments can be used to investigate theoretical principles. The present idealized experiment uses a neurological rheostat to decrease magnocellular signal in an otherwise visually normal subject to probe for possible consequences of the magnocellular impairment that is often linked to reading problems (dyslexia). Reduced magnocellular signal should decrease the threshold for Troxler's perceptual fading. It is proposed that the outcome of this reduced magnocellular signal and reduced threshold for Troxler's fading is clinically determined as intermittent central suppression, as well as visually related dyslexia.

Key Words

Dyslexia, intermittent central suppression, lateral geniculate nucleus, magnocellular pathway, parvocellular pathway, Troxler's phenomenon, visually related dyslexia

INTRODUCTION

Idealized, or "thought experiments" are exercises in imagination and logic that can serve to direct future laboratory experimentation if the logic of the thought experiment is reasonable. For example, Albert Einstein in *The Evolution of Physics* used the thought experiment of observers inside and outside of a uniformly accelerating elevator to illustrate the effect of gravity on light in a discussion of general relativity.[1] This logical reasoning was prior to the ability to experimentally confirm some of those concepts. In fact, the introduction to the newer edition of *The Evolution of Physics*, written by Leopold Infeld, notes some of the experimental confirmation of the concepts that have occurred since the original writing in 1938, even after Einstein's death.[1] This type of consideration can provide a thread of logic for future investigation.

Such an idealized experiment might help illustrate possible visual consequences of a magnocellular (M) defect. We will construct a thought experiment that explores what might occur if we were able to attach a rheostat – a dimmer switch – to the M visual pathway. By doing that in thought, we seek to determine what we would measure clinically, but do not yet have the technology to experimentally verify.

The lack of a perfect M signal has often been associated with dyslexia. Thus, there is a motive to consider this line of reasoning, even though some dispute exists as to whether a link between M defect and dyslexia exists.[2-4] The conclusions, as in general relativity will await experimental verification or refutation. Nevertheless, anything that may provide information in treating the visual aspect(s) of dyslexia, and doesn't put patients at risk, is worth considering. In the following discussions, the terms *dyslexia*, or *visual dyslexia*, mean reading disabilities that have a visual component.[2]

EXPERIMENTAL DESIGN

Five premises will guide our experiment:

1) The first premise is that our subject has two eyes that function – at least initially – equally and simultaneously. There is no strabismus or amblyopia. That is, we start with adequately functioning binocularity. Our subject is a normal young reader. My argument for a young subject is that if, a deficient M signal produces a visual defect that results in a dyslexic symptom, the expression of that defect should be easily identifiable by a classroom teacher.

2) Second, *sight* in this experimental individual is brought about by two parallel visual pathways, the M and parvocellular (P) pathways.[2,5] Much has been written, and the experimental evidence is voluminous and profound on these pathways. I won't attempt a complete literature review here. It is also worth noting that some dispute exists on the precise functional differentiation of the pathways discussed below.[6]

As an overview, the P pathway is specialized for carrying detail and color information through the lateral geniculate

nucleus (LGN) to the cortex. The M pathway complements the P pathway information by carrying motion separately through the LGN to the visual (striate) cortex.[7]

During any light-adapted fixation, visual information about the object of regard is gathered at the retina and transmitted to these pathways. They travel together from the retina, and become separated at the dorsal Lateral Geniculate Nucleus (dLGN) and continue this separation to the striate cortex and beyond. The P pathway occupies the four more dorsal layers of the dLGN, to the striate cortex, then on to the temporal cortex. The M pathway occupies the two more ventral layers of the dLGN, then to the striate cortex, and on through portions of temporal cortex to the parietal cortex.[7,8]

I propose that *sight* occurs at the striate cortex, but that it is in the higher areas of the brain where these complementary streams of information are added together into the "technicolor motion picture" we recognize as *vision*. Both pathways are sensitive to brightness, shapes, stereopsis, and contrast to varying degrees. Both are involved in scotopic vision. The P pathway contributes detail, pattern, color and fine stereopsis to visual sensation. It has color opponency and shows binocular enhancement with color at the cortex, indicating P pathway binocular convergence.[8]

Two important points about these parallel visual pathways are: M-cells at the LGN are never truly silenced; they always have some background activity.[9] Second, both M and P cells have their highest density at the fovea.[10,11]

It is important to understand that the M and P duality does not represent the central versus peripheral vision conflict. The P pathway accounts for 80% of ganglion cells in the optic nerve. Ten percent of retinal ganglion cells in the optic nerve are M cells. Density of both pathways is greatest at the fovea, but only 5% of the ganglion cells connected there are M cells. Both pathways decline in absolute density with retinal eccentricity, but the relative density of M increases to 20% of ganglion cells in the periphery; the M pathway becoming relatively more important in the periphery. Meanwhile, the P pathway constitutes the overwhelming majority of cells centrally, and the somewhat less overwhelming majority of cells peripherally.[11] From this I propose that a deficit in either pathway would be expressed most strongly centrally. The M cells in the fovea are sensitive enough to fine motion that the small fixational eye movements should produce a M pathway response *in an intact M pathway.*[12]

As might be expected, the information carried by each pathway is a function of anatomy and physiology; P receptive field centers are smaller and have stronger antagonistic surrounds than M receptive field centers. However, the off-response of the P centers is weak, giving a more sustained response to relatively stable and discrete visual information. Thus, the P pathway is responsible for acuity (detail). M receptive fields are larger than in the P pathway, latencies are shorter and axon diameters larger. M response is more movement dependant. On a stimulus level, then, the M pathway is flicker-dependant, since visual flicker can be considered a motion stimulus.[13]

3) The third premise is that *vision* is a higher cerebral function. As stated in premise #2, it is in the higher areas of the brain where the complementary streams of information from the M and P pathways are further processed and added together to complement each other. Since pooled activity of large numbers of neurons predicts behavior, this summation of visual information in higher cortical centers with any other visual information streams might be referred to as *vision* or *visual perception*.[14] Further, the original *sight* information has to arrive at those higher centers intact. Inadequate or erroneous visual functioning at the sub-cortical level should influence visual processing at any subsequent (higher) level.[15]

4) The fourth premise requires a consideration of ***Troxler's perceptual fading***.

First reported in 1804, Troxler's Effect originally referred to the temporary and irregular fading or disappearance of a small object in the visual field that is steadily and centrally fixated.[16] This fourth premise requires an intact Troxler's mechanism to be operative in our idealized experimental individual. Further, it operates post-retinally and pre-cortically, most likely at or near the LGN.[17] This fading is a loss of detail and color (carried by the P pathway). It occurs when motion (carried by the M pathway) is removed from the visual stimulus.

One report of early studies on the phenomenon stated that 2.5 seconds of ultra-steady fixation was required to trigger a Troxler's fade.[18] Consequently, if enough conscious control to produce 2.5 seconds of steady central fixation is required in a non-impaired visual system, it unlikely that Troxler's is the result of a lack of attention. Nevertheless, Lou has proposed that the fade may be modified by attention.[19]

It is very important to understand that in these experiments[18,19] on Troxler's dyslexic subjects were not used and testing (by definition in Troxler's) was monocular. I propose a predictable consequence of impaired motion detection in this type of experiment would be a reduction in the time necessary to produce a Troxler's fade: If approximately 2.5 seconds of steady fixation in a normal subject under monocular conditions is necessary to reduce M signal enough to produce a Troxler's fade, it is reasonable to expect that with an impaired M signal less fixation time would be required to trigger a Troxler's fade.

Later experiments explored Troxler's by optically stabilizing the retinal image, thereby removing all motion from the retinal image. These experiments found that lack of sufficient signal in the motion (M) pathway is responsible for Troxler's perceptual fading. Therefore, Troxler's effect is neural, and not photochemical or retinal.[20] When an image is stabilized on the retina, that image fades.[21] Color disappears quickly.[22] If the formerly stabilized image is moved, the image reappears instantly.[23]

With the above information and assumptions, I propose that Troxler's may be more of an "on-off" switch in its operation, rather than a slow fade-away and equally slow image return. Higher processing may affect that time course. Lou suggested a cortico-thalamic feedback loop for attention to affect Troxler's.[19] Interestingly, I have previously suggested, based on the early studies of Troxler's in which one-eyed subjects were found to be "markedly resistant to Troxler disappearances," that some sort of M signal enhancement at the LGN must exist.[2,21] Therefore, given that monocularity and original strength of M signal may affect how quickly a Troxler's fade has been demonstrated under laboratory conditions, if we proceed to the clinical binocu-

lar testing conditions, and from normal subjects to dyslexics, the amount of steady fixation time necessary to produce the decrease in M signal to trigger fading may be greatly reduced.

In the normal eye, fixation drifts keep the image alive. The high frequency fixation tremor (physiological nystagmus) does not. [17] Also, saccades don't keep the target image alive. Importantly, drifts should produce an increase in M signal, whereas that same M pathway is suppressed during saccades.[12,24] In fact, given that drifts prevent Troxler's fading and that Troxler's is a result of lack of M signal, then drifts *must* increase M activity above baseline. Although the minimal M signal to prevent a Troxler's fade is unknown, apparently when that minimal signal is reached, the P pathway fades until sufficient M signal is established to switch the P-pathway back "on", and the image instantly returns. Accommodation apparently makes little difference in Troxler's, but accommodation fades to a rest position during a Troxler's perceptual fade.[20]

To summarize the small fixational eye movements and their effects on Troxler's fades: Only fixation drifts raise M activity enough above baseline to reverse a Troxler's fade. Physiological nystagmus apparently does not involve enough neurological units to affect a Troxler's fade.[17] During correctional saccades, as in any saccade, the M activity is suppressed, so a Troxler's fade is not reversed.

The role of attention in Troxler's perceptual fading remains open. As discussed above, Lou reported that attention can affect a Troxler's fade. [19] I again propose that lack of attention is not likely responsible for the fades, since 2.5 seconds of ultra-steady fixation was originally required to produce a fade; that is, if enough attention is applied to carefully watch for a fade, the same attention can't simultaneously be deficient to produce a Troxler's fade. This question of attention does have some bearing on the clinic: In clinical practice when looking at possible Troxler's effects, i.e., intermittent central suppression (ICS), I propose it would be the unusual child who could dramatically modify test results with attention, but a very attentive adult may alter results somewhat. Lou's experiment[19] may also add some information about why suppressions are not "black-outs", but rather "drop-outs" of sensation as will discussed below. The same mechanism of visual consciousness that fills in everyone's natural blind spot may fill in a suppressed area.

Table 1. The Five Premises for the Idealized Experiment.

1.	Subject is two-eyed, without strabismus or amblyopia, i.e. "bonocular"
2.	Two parallel visual pathways: Magnocellular and Parvocellular
3.	"Sight" occurs in the Striate Cortex "Vision" occurs at higher cerebral locations
4.	The mechanism for Troxler's Effect is functional
5.	Fisation drift occurs in the absence of a visual "picture"

5) The fifth and last premise for our idealized experiment is that fixation drift occurs when no visual signal–no "picture"–is present. The simplest way to conceive of this premise would be to consider the cover-uncover test. Phorias will manifest on the cover test because one eye no longer has an image to match with the other. Without two images to fuse, control over drift in eye aiming is gone. We measure that as a phoria on the alternating portion of the cover test. If a visible phoria manifests on the unilateral cover-uncover test, when the cover is removed, the formerly covered and now turned eye will make a non-saccade vergence movement to return to alignment (with a normally binocular individual).

Fixation is accomplished with a servomechanism separate from the control system for general direction of gaze.[25] Saccades, drifts and physiological nystagmus (or fixation tremor) work to hold aim within Panum's fusional area (roughly 10 minutes of arc centrally[19]). The servomechanism for fixation, similar to the servomechanism for accommodation, produces a "hunting" for accuracy.

Retinal image displacement is a feedback signal for position to fixate the target of regard with both eyes. Then retinal image motion provides the feedback for a negative feedback loop to keep the target of regard fixated accurately.[25] Retinal image velocity adds to this feedback. Therefore, motion detection and the M signal are an integral part of accurate fixation. Any interference in the retinal motion signal also decreases fixation accuracy.[25]

These five premises are summarized in table 1.

THE IDEALIZED EXPERIMENT

We now apply these five premises or experimental conditions to an idealized experiment. From the above discussion, we propose that an intact M signal of sufficient strength prevents a Troxler's fade of the P carried image. The M pathway always has baseline activity.[9] It is never truly silent, so it can respond even without P activity. That is evidenced by image stabilization experiments that used target movement to re-establish the image after a Troxler's fade had eliminated the (P carried) "picture."[18,23] Stimulation of the M pathway produces a signal increase beyond baseline; then activity will drift back toward baseline.[26]

We will now attach the rheostat to the M pathway bilaterally, anterior to the LGN. It will be set to reduce the M signal to just at, or barely below the minimum necessary to prevent Troxler's fading during a normal fixation in reading; this will mechanically impair the M pathway. A few milliseconds of decreased (or increased) stimulus is all that's required to further decrease (or increase) the M activity.[26] Our binocular subject now starts reading.

Fixating on the first word somewhat stabilizes the retinal image and decreases the M signal. Given that M is suppressed during saccades, our subject may actually start the fixation with M below the Troxler's minimum if a saccade preceded this first fixation. Since the signal was previously at the minimum to prevent a fade, our impaired subject's M signal now definitely drops below the Troxler's minimum with this first fixation. Again, only a few milliseconds of decreased stimulus (stable fixation) is required to reduce the M signal below our starting point[26]. We will assume some asymmetry in signal of one side relative to the other so one side will be affected first. As one M pathway's signal drops below the minimum to prevent fading, a Troxler's fade will rather quickly occur.

The eye whose signal has faded now has lost the intact picture. As per premise #5, fixation drift will occur without an intact picture. The image displacement

feedback on the faded side no longer exists, since the fade itself is in the P pathway. The P pathway does not send a signal to the higher visual centers. As the affected eye drifts off target, the M signal increases because of the motion of the drift (fixation drifts increase M signal; saccades and physiological nystagmus do not).

As the M signal increases because of the drift of the "faded" eye, enough motion will increase the M signal above the minimum for the Troxler's fade to be broken (again, M apparently can respond without an active P signal). The signal (intact image) carried by the P pathway is instantly restored. Unfortunately for our young reader, the aim of the formerly faded eye is no longer precisely aligned with its fellow. The image displacement feedback is now active since the formerly faded P pathway is now active. Our subject is "binocular" now, so a vergence movement will pull the disparate images back together; just as in the unilateral cover test, the formerly covered eye will converge to resume bilateral fixation (in the absence of strabismus). This non-saccade will continue the increased M signal, and, therefore, not allow another Troxler's fade. Both images are now intact with accurate aim re-established. The subject is now truly binocular. However, our young reader is still fixating the word, or perhaps is now fixating the next word in the test sentence.

Since the M pathway remains impaired because we haven't changed the rheostat, this sequence can and will repeat. Our subject, then, is constantly switching between four visual situations:

1) truly aligned binocularity
2) central "monocularity"
3) bilateral sight without precise alignment, and
4) bilateral sight with motion as the alignment error is corrected.

Since the other eye's pathway is similarly impaired, the repetition will occur on a timetable determined by the amount of impairment which is a function of the rheostat setting, and the relative impairment on the two sides. Obviously, the more significantly we impair the M signal - by setting our rheostat lower – the more conditions during which the alternate fading will occur. For example, if the impairment is intense enough, we might expect fading while tracking a fly ball in baseball,

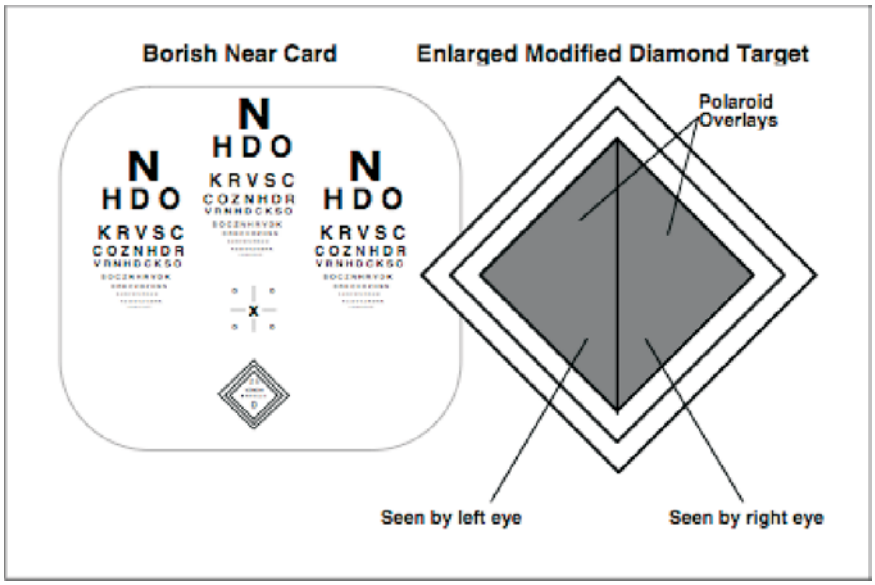

Figure 1. The modified Borish vectographic nearpoint card

or perhaps when judging distances when driving. We can also postulate that conditions such as Alzheimer's, that don't just set the rheostat lower but actually destroy some of the wiring, can cause similar, but much more dramatic visual distortions.[16,28]

This will be observable in the central visual area since the M pathway is most dense centrally (as is the P pathway). With a sufficiently defective M pathway, lapsing into a strabismus is certainly possible.[27] However, most of these defects would be expected to produce a repetitive fading that might be clinically identifiable.

CLINICAL IMPLICATIONS AND LIMITED VERIFICATION

If we now take this same experimental individual to the clinic, what might we observe? If we approximate the same level of fixation in the proper clinical test situation, we should observe the same phenomenon in this M impaired individual. An appropriate test target would use polarization, or a similar technique, to allow monitoring the individual's binocular status. Such a target would be the bisected diamond target (Figure 1). This is a modification of the Borish vectographic near point card.[a,29] See Figure 1.

The behavior we would expect to observe would be a repetitive fading to black of either side of the diamond. Fading to black would indicate a Troxler's fade has in fact occurred and the P pathway on the affected side is not transmitting the detail to be able to see the letters underneath the Polaroid overlays. The faded side would then clear as the Troxler's fade is broken. Then we would expect a repetition of this sequence of blackening and clearing of either side of the target. With a sufficiently mature patient who could understand and report what is seen, we should be able to diagnose this clinically. In fact, that type of sequence has been documented as intermittent central suppression (ICS).[30] The conventional wisdom describing suppression as a cortical "fight or fuse" phenomenon does not provide a plausible or convincing explanation for this continuing on-off sequence we define as ICS: That conventional wisdom presupposes strabismus and amblyopia.[2]

At this point, our experiment is finished. We have seen a clinical effect of a defective M signal at the LGN – intermittent central suppression. Fortunately for our experimental subject, we can remove our neurological rheostat and return the visual system to non-impaired status. That should return him to the same M signal strength that was present in the original Troxler's research that required extended ultra-steady fixation for a fade to be produced, i.e., this fading should not occur with normal M signal strength in normal reading.

In further evaluating our results we might take another cue from Albert Einstein and his thought experiment of the uniformly accelerating elevator. Einstein explored the relativistic effects of gravity on light by asking what the path of a light

beam would look like to two differently situated observers; one in a stationary frame of reference outside, and one inside the uniformly accelerating elevator.[1]

In this vein, we would solicit interpretations of the results by three observers, each in a different frames of reference: The clinician who reported the suppression produced by the M defect; an academician-scientist who is familiar with (and agrees with) the research on M defect and dyslexia; and the teacher who observes our subject reading with the induced M defect in the classroom.

Interpretation by the clinician

This individual has observed ICS to be present on our subject. Further, our idealized neurological rheostat was at the LGN. Therefore, this sensory defect we have created is afferent. Despite the fact that the defect is not cortical, we would expect a cortical effect in both sight and vision since, according to premise #3, both occur beyond the LGN at higher brain centers. At those higher visual centers, the result of this intermittent suppression or fading of the P signal would likely be interference in perception of detail. The constancy of the picture and perception at those higher centers will suffer as either side fades and then has to be realigned repetitively, as discussed above. Tasks requiring constancy of detail, such as reading, would likely be most negatively affected. With this knowledge, the clinician would likely predict that the child would show some of the reading disability symptoms identified with ICS. [30] I propose this would be a verification of my previous suggestion that M defect-produced ICS is the clinical sign of visual dyslexia.[2]

Interpretation by the academician-scientist

This individual accepts the scientific evidence linking M defect to dyslexia.[3-7] The scientist would view the M defect as a neurological problem that produces effects in the subject. Based on the evidence of M pathway defects in dyslexia, the scientist would predict our subject should be dyslexic to the extent that vision and the M defect are involved.[2,3,7]

Interpretation by the teacher

We intentionally chose a young subject hoping to make any performance effects of the experiment easier for an experienced educator to observe. Visual detail is being compromised as the P pathway repetitively becomes inactive then reactivates, i.e., the on-off suppression we clinically define as ICS. The repetitive alignment-misalignment-motion-realignment sequence suggested above would produce a variable picture in a reading task requiring visual constancy for accuracy. It is then likely that the teacher will observe some of the reading symptoms associated with ICS: The need to re-read for comprehension, loss of place, mixing up letters and words, and more problems reading to learn than is expected at the subject's particular age.[30]

In Einstein's experiment, the two observers would *dis*agree on the path of the light ray. However, in our experiment, all three of our observers agree. Our observers, from a clinical, intellectual and performance based frame of reference respectively, would all *agree* that our subject is prone to, or already exhibits some type of reading disability – that is, our subject could or would be called "dyslexic" within the frames of reference and definitions of these observers.

All three of our observers would also hope to improve the dyslexia, either to help the child or out of scientific interest. The positive implication for our experimental subject is we can remove our neurological rheostat and return the visual system to non-impaired status. In the real world of patients in the clinic, active vision therapy (including electronic rapid alternate occlusion[27]) remains the only documented treatment for this defect. As such, I propose that vision therapy *must* be considered the standard of care and generally accepted medical principles of medical care should define such treatment as medically necessary and appropriate.

Conclusions

This has been an idealized "thought" experiment re-asserting a theory presented in prior papers to explain the clinical manifestations in M defect, vision-related reading problems, ICS and visual dyslexia. The science presented would support this view, but certainly differences of opinion on even the science exist. [3-7] However, Troxler's perceptual fading needs further exploration in this area of vision and dyslexia. Some directions for future research have been previously presented.[28] A prediction of research results based on the above idealized experiment might be made, and if proven correct, would support this theory: We might expect a difference between Troxler's fade or image stabilization responses between those with ICS and those without ICS. Since Troxler's experiments were carried out monocularly, the suppression should not be an issue. But, since both ICS and Troxler's are theorized to be subserved by the M pathway, the impaired M pathway of ICS (represented above by the rheostat-impaired pathway) might be expected to produce a quicker Troxler's fade and possibily a longer lasting fade, or perhaps a fade that requires more motion stimulus to reverse the fade.

Another possible investigation would require a sophisticated observer who manifests ICS to try to document the theorized intermittent and variable vergence error: Use a sufficiently sensitive eye movement monitor along with a time-linked subject-activated recording device that the subject could activate as a suppression starts and stops. The eye movement monitor would then read minute changes in vergence posture and could be time-linked to the subject's recording of the ICS on-off cycle. We would expect vergence variations similar to those found by Bedwell, et.al. in cases of "reading difficulty", but we would expect some of those variations to be time-linked to the intermittent suppressions.[31]

The author has no financial or proprietary interest in the Borish vectographic near point card.

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