

# SPECULATIONS ON THE NATURE OF VISUAL MOTION OPTOMETRIC IMPLICATIONS

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## Abstract

*Recent research suggests two major neural pathways carrying visual information, the parvocellular (detail and color) and the magnocellular (motion) pathways. The magnocellular pathway has been implicated in reading disabilities. By analyzing the nature of visual motion it might be possible to find efficient ways to improve magnocellular function. I present a discussion suggesting visual flicker is motion in stimulus form. Repetitive flicker should improve magnocellular function, and by extension reading disabilities and intermittent central suppression. Such a stimulus should be applied centrally since magnocellular cell density is greatest centrally.*

## Key Words

*Intermittent central suppression, magnocellular pathway, parvocellular pathway, reading disabilities, visual motion, visual flicker*

## Introduction

**T**he perception of visual motion is fundamental to human survival. One only needs to imagine driving to work in the morning or walking on a crowded street without the assistance of visual motion to appreciate its importance. Recently, the understanding of motion perception has been expanded by research exploring an aspect of the visual involvement with dyslexia.

This research reveals that there are two major pathways carrying visual information from the retina to the brain. They are the parvocellular (P) and the magnocellular (M) pathways. The former pathway is most responsive to stimulation of high spatial frequency, while the latter to high temporal frequency. Put another way, the P pathway is essentially responsible for detail and color, while the M pathway complements that with motion. The M pathway has also been found to be selectively responsive to flicker, that is, light or visual stimulation of high temporal frequency.<sup>1-3</sup>

The recent interest in motion perception, then, is prompted by studies showing the M pathway is more functionally deficient than the P pathway in dyslexia.<sup>4,5</sup> Consequently, I propose that if the motion pathway is deficient in dyslexia, then perception of motion may have a role in reading problems. With this in mind, it should be clinically beneficial to more fully analyze, and more fully understand the nature of motion itself. The goal would then be to devise more precise motion stimuli to en-

hance its perception. If feasible methods of simulating motion sensation in the M pathway can be found, perhaps we can more effectively treat M defects, and, by extension, those reading problems that are at least partially caused by these defects. These methods could be particularly valuable for children who are deemed to be “at risk” for dyslexia and other reading problems.

## Understanding visual motion

I propose that the etiology of M defects includes congenital or developmental deficiencies, and neurological disease or injury. For example, I’ve suggested intermittent central suppression (ICS) is a function of an inadequate “wake-up” signal in a deficient M pathway, and have documented ICS caused by whiplash cervical trauma.<sup>6,7</sup> I then speculated that ICS occurs at the level of the Lateral Geniculate Nucleus (LGN). Prior research suggests the LGN is also the locus of M defects in dyslexia.<sup>8</sup> On this basis I propose that in the following discussion, we will not consider M pathway defects at the receptor level. In this discussion, all receptors are normal, even though they might be connected to a defective M pathway.

In order to better understand the nature of visual motion, I propose the following exercise. Figure 1, panel A represents a group of retinal receptors. I take the liberty of considering the intervals between the receptors as simply spaces of regular width rather than the usually considered “off”-surrounds of receptor cells with an on-center.<sup>9,10</sup> We should now imagine that all receptors are “off.” A continuously shining (not flickering) very thin bar of

light (one receptor in width) travels into the eye. That bar then moves across the field of retinal receptors (Figure 1, Panel A) from top to bottom. As the light strikes an individual receptor, that receptor switches “on.” As the light bar moves to the space between receptors, all receptors are again “off” (Figure 1, Panel B). As the light bar moves to the next receptor, that receptor switches to “on,” while the prior and other receptors remain “off” (Figure 1, Panel C). This action proceeds as each receptor remains “off”, but will respond by being turned “on” in its proper turn by the bar of light (Figure 1, Panels D and E).

I then propose that visual motion is essentially a series of “on” signals followed by “off” in these individual receptors as a light stimulus sweeps across the field of receptors in a regular fashion: “on-off-on,” etc. The sensation of continuing motion would then entail the ongoing repetition of the whole process with a series of similar bars of light moving across the same receptor field, stimulating each receptor in its proper turn. This would produce repetitive “on” signals in each of those receptors, followed by that individual signal “turning off” repetitively. It should be noted that the above does not fully account for the current level of knowledge about retinal morphology; this includes retinal cell interconnections that produce receptive fields with centers responding (for example) “on,” but surrounding oppositional response areas switching the response to “off.”<sup>9,10</sup> However, the proposed thinking is presented as basic to understand the nature of visual motion and can serve to develop appropriate therapeutic stimulation.

We now consider the response of the individual M receptor under a different condition: Figure 2, Panels A and B depict a similarly sized light beam as in figure 1 now being sent through a very small aperture to a single receptor. If we continue to alternately open and close the aperture, we repeatedly stimulate that single receptor. I propose this to be flicker. The question, then, is “*for this single receptor, is there any difference between this flickering stimulus and the prior motion stimulus, so long as the rate of flicker matches the speed of the prior “on”-“off” motion stimulus at that receptor?*” My answer is either none, or very little. Said more succinctly, flicker is motion in stimulus form. This is consistent with the research that in-

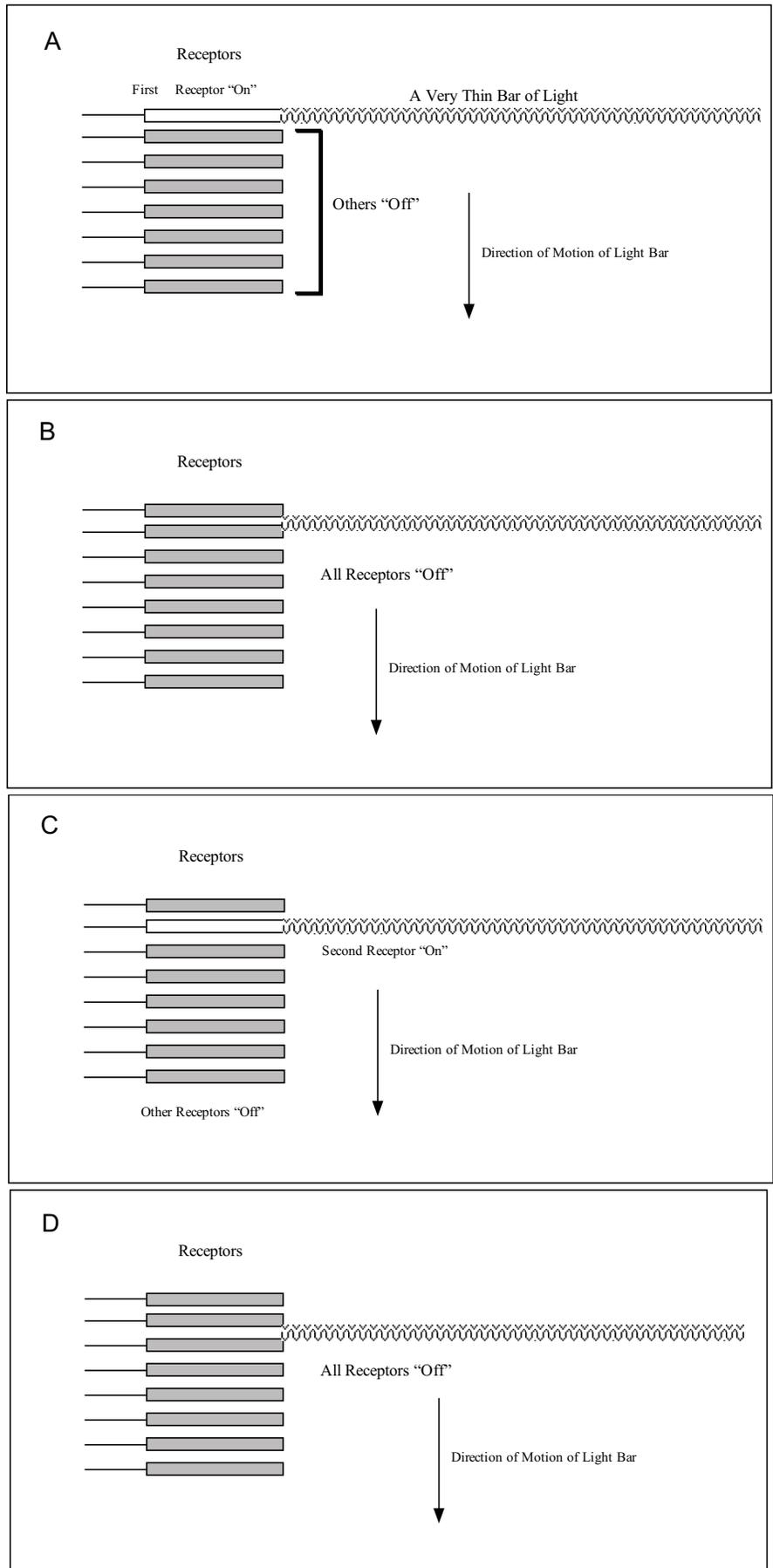


Figure 1, A, B, C, D.

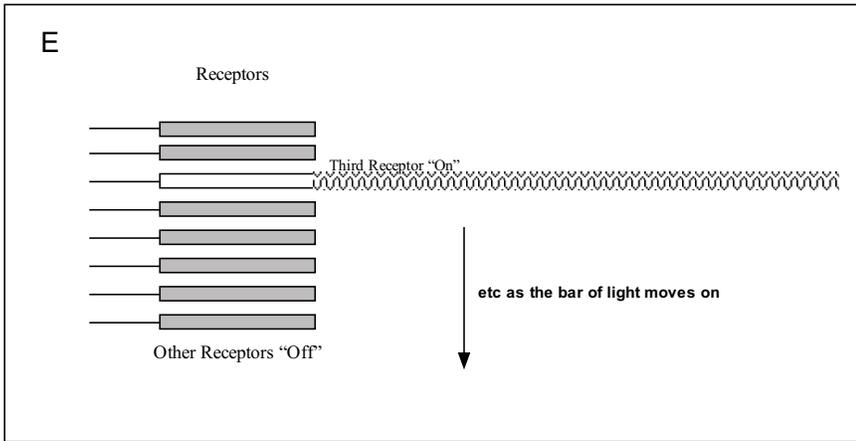


Figure 1, E.

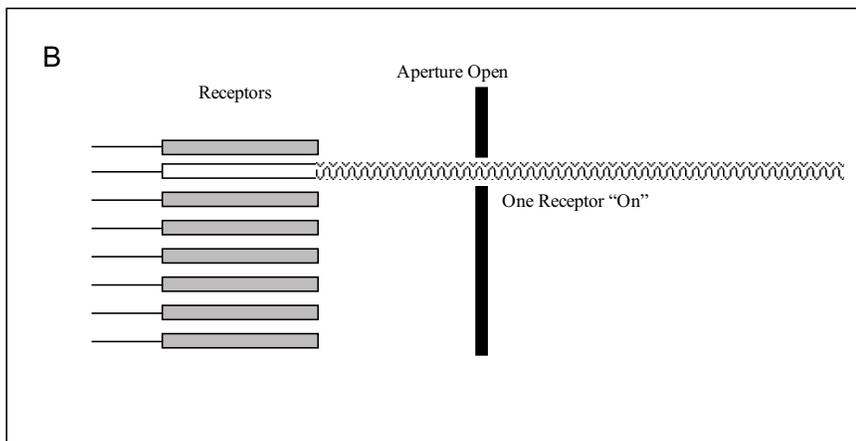
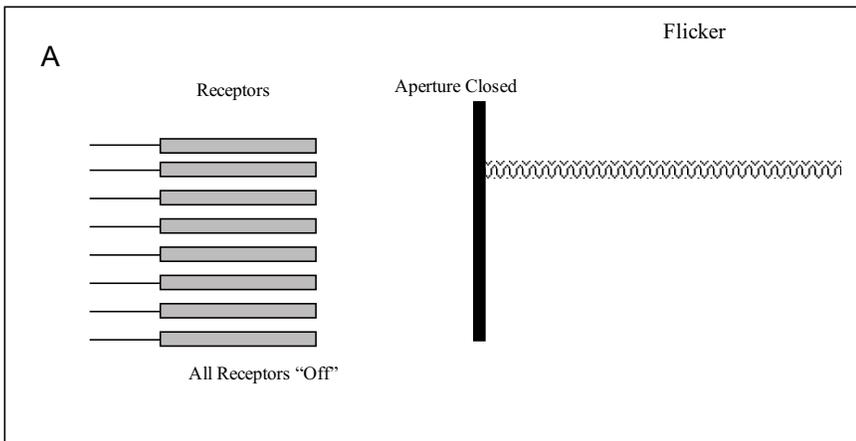


Figure 2, A, B.

icates the type of stimulation to which the M pathway is most sensitive.<sup>1-3</sup> It suggests the P and M pathways can be differentiated at the LGN based on their flicker responses: that the M pathway is more responsive to flicker.<sup>1</sup> This offers the possibility of selectively using flicker as a visual motion stimulus for the M pathway.<sup>1</sup>

Further, if that particular receptor is “tuned” to detect motion at a particular speed, then we can choose appropriate rates of flicker corresponding to that speed (temporal frequency). We can pick that receptor to maximally stimulate based on its temporal frequency tuning.

If instead of considering the response of an individual receptor, we consider motion and flicker responses across the ret-

ina, some differential in speed tuning fits our knowledge of flicker fusion and what I’ve observed in treatment.<sup>5,11</sup> That is, that the central retina is tuned to a slower speed of motion than the para-central and peripheral retinal areas. The central receptors that are part of the motion detection pathway are sensitive to small movements in detailed objects such as watching the cursor move on a computer monitor, while the more peripheral motion receptors are most sensitive to fast, large, abrupt stimuli such as a moving vehicle on the side of your car. Moving, then to a broader format in our previous discussion, one can imagine that instead of a single receptor, we now stimulate a broader group of receptors, clustered according to this differential in speed tuning - or sensitivity to frequency of the flicker - of those receptors (Figure 3). Clinically, this larger aperture might represent a liquid crystal lens changing from black to clear.

A corollary question that bears on this notion is the question of whether a binocular or simultaneous bilateral motion stimulus is optimal. My experience suggests that bilateral stimulation is most effective. This could mean flicker, which alternates between the eyes at a pace that is perceived centrally as a continuous binocular signal. However, we will ignore justifying that suggestion here; interested readers who want to explore that suggestion are referred to a fuller discussion of the pathways.<sup>4</sup> But, if this concept of motion proposed above is fundamentally correct, appropriately designed visual flicker stimuli should affect change in the M pathway. By extension, if the view that ICS is caused by a defective or deficient M pathway is accurate, then the same flicker stimulation should reduce or eliminate ICS.

### THE M-P and central-peripheral dualities

Frequently in lectures I have attended and in personal conversations I have had, the P-M duality seems to be equated with the central – peripheral duality of vision; that the P cells are virtually exclusively found centrally, while the M cells are virtually exclusively found in the periphery. However, recent research on M and P retinal cell density doesn’t support this concept. M cell density peaks in the fovea.<sup>12-14</sup> Figure 4 is my schematic graph of M-P cell density based on the 1994 work of

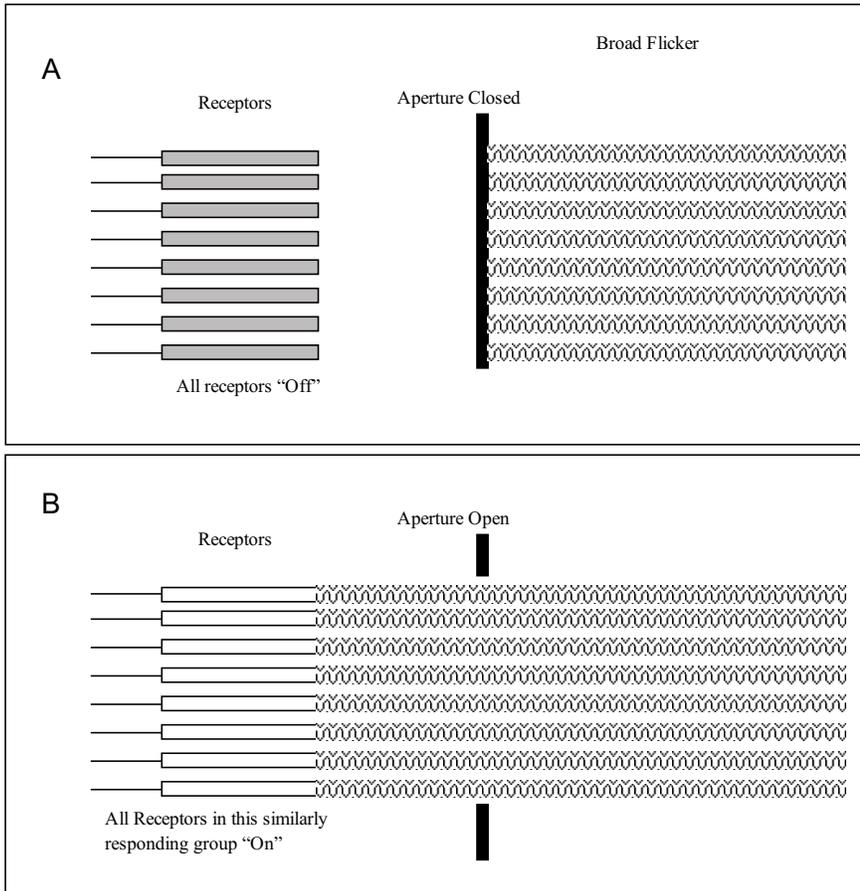


Figure 3, A, B.

Figure 4B shows that both M and P cells are present in the central and peripheral retina. In absolute terms of cell density, it is evident that there is a significant predominance of P cells in both locations. While the density of both types of cells decrease with increasing eccentricity, the P cell predominance continues at all locations.

Further, there is increasing evidence that implicates faulty M pathway functioning as a contributor to dyslexia and reading problems.<sup>3-5</sup> As clinically feasible methods are devised to enhance M pathway function, these therapies will need to include stimulation of the central retina where these cells are most densely populated (Figure 4B). Improvement of functioning of the M pathway in retinal areas eccentric to the para-fovea will probably have little impact on reading. In terms of the important area is comprised in the span of perception.<sup>15</sup> It is likely that there is faulty processing of the visual information to the right side of fixation. This is the domain of the M pathways which "set the table" by providing global pattern information prior to the arrival of the final visual details via the P channel."<sup>16</sup> In terms of ICS, these M pathways provide the "on" signal to keep the P pathway from being suppressed.

### Conclusions

Inadequate function of the M pathway has been found to be a correlate of some types of reading problems. This pathway is most sensitive to visual motion. I have made the case that flicker might be a clinically reasonable method to simulate motion. Consequently, this type of stimulation might be effective to remediate reading problems. Clinical research should be undertaken to determine the optimal means to use flicker in this regard. I further suggest that the stimulation should be applied at the central retinal area where the M cells are most prevalent and are indeed where the visual component of reading is most involved. I have made the hypothetical case that flicker is visual motion in stimulus form, in a sense, a "pure" motion stimulus. Further, if flicker is motion and if the M pathway primarily carries motion and if flicker can be shown to eliminate intermittent central suppression, then it is possible that the M pathway and ICS are linked.

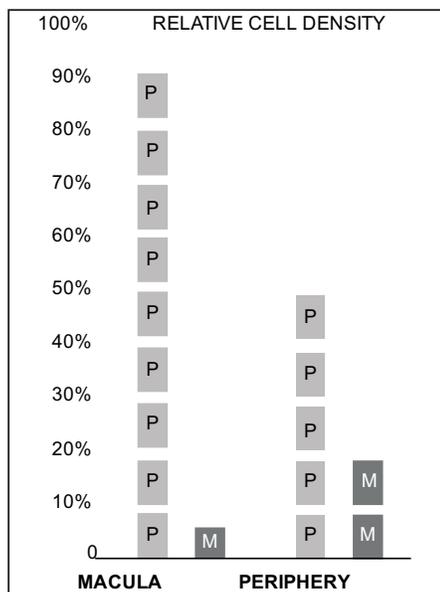


Figure 4, A.

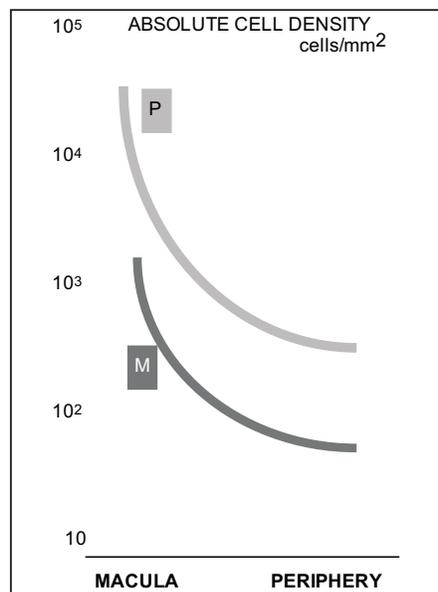


Figure 4, B.

Dacey.<sup>14</sup> The "periphery" in the graphs is 16mm from the macula.

Figure 4A represents the cell density in relative terms. It shows that, while the M cells gain in density at the periphery, it is relative only to its previous proportion

to P cells at the macula. This does not support the proposition that the periphery is the exclusive domain of M cells and that these should be totally equated with peripheral vision.

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Date accepted for publication:

September 3, 2003