Viewpoint ▸ Visibility, Suppression, and Implications for Downstream Visual Development

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ABSTRACT

Non-strabismic intermittent central suppression is a loss of visibility at the level of the lateral geniculate nucleus. When put into a developmental context, this implies that all amblyopia has a deprivation component.

Keywords: amblyopia, suppression, visibility amblyopia

Non-strabismic, non-amblyopic intermittent central suppression (ICS) has been the crux of the challenge I have in accepting the conventional wisdom of suppression development, diplopiaphobia. Reconciling the on-again, off-again intermittent loss of visual sensation that is ICS with cortical inhibition (often defined as masking at the cortex[1]) as the proposed visual sensation shut-down mechanism is a challenge. That inhibition must not only be intermittent, but alternating from side to side, (in 80% of ICS patients) uncontrolled by some sort of volitional switch in fixation as occurs sometimes in alternating strabismus. Those shortcomings led me to explore how vision science on visibility might apply to this intermittent type of suppression.[2] My starting point was that the visual neurology is, well, the visual neurology. That is, whether strabismus is present or not, the neurology responsible for the suppression is in its basic construction – or perhaps in its beginning construction – the same, assuming no gross genetic or trauma-caused defects. The conventional wisdom would hold that strabismus or anisometropia, through diplopiaphobia shuts down recognition of the visual signal on one side at the cortex. This is the definition of suppression.

If we redefine loss of the visual signal as a loss of visibility (Troxler's Perceptual Fading, possibly ICS[2]), that loss of visibility is positioned as a more afferent, maybe more primary, visual sensory defect rather than being entirely secondary to misalignment or anisometropia. If accurate, an early interruption of visibility (visual sensation) changes the time course of sensory anomalies from current thought. With diplopiaphobia, although unstated, there is always a sense that the organism actively decides over time that diplopia does not work well. The diplopia comes first. If instead the sensory loss becomes more primary, the time-frame to defect may contract. In neurological development and potential defects in development, time is of the essence. Exploring the consequences of loss of visibility in our view of the development of binocularity defects means putting that loss of visibility into the moving framework of time – inserting loss of visibility into the development of the visual neurology through time. But, moving the sensory defect from the cortex to the more afferent location of loss of visibility at or near the LGN also means any loss of visibility now affects development further downstream neurologically. Loss of visibility at the LGN, if developmentally early enough, has consequences for development at the cortex, versus the cortex interfering with its own development through masking.

Science fiction has used intervention in the fourth dimension (time) as a vehicle for stories for decades. Here we have real-life consequences through time from an early disturbance in information transfer.

Assuming that loss of visibility may be responsible for the loss of sensation of ICS

led to the first suggested visual-behavioral consequence that I termed visual dyslexia. In this view, loss of visibility contributes to such classic behavioral descriptions of dyslexia as reading letters and words backwards. Loss of visibility not only creates fixation instability, but also surprisingly strong filling-in of the area that has lost visibility.[3] Backwards could easily be a short word seen correctly with one eye paired with a filled-in area from the fellow eye further confused (and variably confused?) by fixation instability. That the filling-in is strong enough to create rivalry with the normal-seeing eye's image adds to the sense of loss-of-visibility variability in the central perception. This combination of a normal image conflicting with fixation-unstable filling-in could turn that short word into a variable visual mush – but a mush that has some normal characteristics from the non- suppressed eye's image. Perhaps seeing backwards should be replaced with confused image. Importantly, this would move some of the visual part of dyslexia from a cortical perceptual problem to a pre-cortical variable defect in the visual percept coming to the cortex.

In amblyopia, suppression has traditionally been considered entirely secondary to misalignment (diplopiaphobia) and/or to blur in refractive amblyopia, presumably mediated by an active sort-of organismic decision against diplopia or blur. Schor et al.[1] studied the 72 msec open time in his 7 Hz alternation paradigm in subjects with amblyopia and compared that with a common stimulus asynchrony in masking experiments to suggest masking at the cortex is responsible for the suppression. In masking experiments, a brief target stimulus can be made invisible if it is immediately preceded or followed by another (masking) stimulus. A masking stimulus to one eye can erase the target stimulus to the other. In much of the vision science, masking is studied by preceding or following a target stimulus at different inter-stimulus intervals to look at how the visual neurology responds.[4] The thrust for binocularity is that one eye's masking of the less favored fellow eye's image (weaker? turned? blurred?) might explain suppression on a neurological level. Masking could be a perfect explanation for suppression … except again for ICS. The intermittency and alternation are still pretty hard to explain with masking.

Loss of visibility actually might support development of a cortical masking suppression. Poletti and Rucci[5] and Komatsu[3] suggest that loss of visibility might be a loss of contrast to the point of disappearance. That certainly makes sense if the actual fade is a loss of parvocellular signal triggered by an insufficiently strong magnocellular signal. Stated eloquently, "if the M-pathway fails, the P-pathway fades."[6] Reducing contrast supports masking of the weaker signal.[7] A relatively brief loss of visibility (loss of contrast) on one side could trigger, or if not trigger, then at least facilitate, masking on the reduced-contrast side. That loss of contrast, however, is not at the cortical level where masking is thought to occur, but at the level of the Lateral geniculate nucleus (LGN). This signal loss, then, is afferent and would therefore, if early enough in development, deprive the visual cortex of the signal it needs to develop normally. The result would be impaired activity in the cortical visual neurology since "neurons that don't fire together, probably don't wire together." If accurate, this would inflict, at least to some degree, a deprivation component on the cortex in virtually all forms of amblyopia. This discussion ignores the initial trigger for the loss of visibility, the chicken or egg argument in the neural development.

We need to be careful with this on at least two counts. We need to not allow ourselves to use that as an excuse for lack of success in therapy. It is essential to pay attention to any developing neurology research that may suggest ways to restart development. This interference in visibility at the LGN may be what Hess has described as a monocular pathway attenuator at the LGN in amblyopia.[8]

Also worth exploring is whether anisometropic refractive error and amblyopia would become co-morbid secondary to signal dropout at the LGN rather than the amblyopia being entirely secondary to the anisometropia. Kotulak and Schor[9] suggest a (loss of visibility will have some effect on accommodation since the loss of visual signal occurs neurologically prior to accommodation control. The question is whether that could affect a developing visual system remains. Perhaps if the effect on input to accommodation is compromised early enough, emmetropization on one side might be affected and therefore loss of visibility would become part of the development of anisometropia in amblyopia versus the anisometropia being the trigger for all anomalies.

One of the parts of loss of visibility that has not been looked at extensively in any clinical sense is the filling-in that occurs with loss of visibility. Let's project that into a very early stage of neural development in an infant, remembering that perceptual filling-in is strong enough to create rivalry with the normally seeing side.[3] The filling-in is a cortical calculation, beyond V-1, apparently projected down to the LGN since rivalry occurs at the LGN.[10,11] Filling-in is just that – fill-in - filler – nothing with content, sort of like the middle section of that last movie you paid real money to see. Is it conceivable that such a strong filling-in with non-content at a very early stage of development could create real interference with such basic sensitivity as direction of motion in the affected eye? If so, could that contribute to some of the measurements we get in anomalous correspondence/anomalous projection? Not enough is known about perceptual filling-in to go much beyond speculation. Nothing that I have seen suggests anything about how early that filling-in develops, although this is the same cortical calculation process that fills in the optic nerve head's natural blind spot.Therefore, we might suggest that it is an early-established cortical function.

All these thoughts await experimental verification of some sort, but, if there is any accuracy at all, perhaps understanding these things will aid development of new efficacious, efficient and safe therapies.

References

1. Schor C, Terrell M, Peterson, D. Contour interaction and temporal masking in strabismus and amblyopia. Am J Optom Physiol Opt 1976;53:217-23. http://bit.ly/2vZeoj5
2. Hussey ES. Is anti-suppression the quest for visibility? Optom Vis Perfom 2015;3(1):33-41. http://bit.ly/2vU2qXJ
3. Komatsu H. The neural mechanisms of filling-in. Nature Reviews/Neuroscience 2006; 7:220-31. http://bit.ly/2L6GTjg
4. Macknik S, Livingstone M. Neuronal correlates of visibility and invisibility in the primate visual system. Nature Neuroscience 1998;1(2):144-9. http://bit.ly/2vTFdF7
5. Poletti M, Rucci M: Eye movements under various conditions of image fading. J Vis March 2010, Vol.10, 6. doi:10.1167/10.3.6. http://bit.ly/2L2boaf
6. Hussey ES. Binocular visual sensation in reading II: Implications of a unified theory. J Behav Optom 2002;13(3):66-70. http://bit.ly/2L20gKo
7. Foley JM. Forward–backward masking of contrast patterns: The role of transients. J Vis. 2011 Aug 25;11(9). pii: 15. doi: 10.1167/11.9.15. http://bit.ly/2L3SwHG
8. Huang PC, Baker DH, Hess RF. (2012). Interocular suppression in normal and amblyopic vision: Spatio- temporal properties. J Vis. 2012 Oct 31;12(11). pii: 29. doi: 10.1167/12.11.29. http://bit.ly/2L2OP58
9. Kotulak JC, Schor CM. The accommodative response to subthreshold blur and to perceptual fading during the Troxler phenomenon. Perception 1986;15:7-15. http://bit.ly/2vTRae2
10. Lee S-H, Blake R. V1 activity is reduced during binocular rivalry. J of Vision 2002; (618-626), http://bit.ly/2M2eRKd. http://bit.ly/2L1sWmV
11. Wunderlich K, Schneider KA, Kastner S. Neural correlates of binocular rivalry in the human lateral geniculate nucleus. Nat Neurosc 2005; 8(11):1595-1602. http://bit.ly/2L4XKmG

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