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REVIEW

Sensory Eye Dominance: Relationship Between Eye and Brain

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Abstract: Eye dominance refers to the preference to use one eye more than the fellow eye to accomplish a task. However, the dominant eye revealed can be task dependent especially when the tasks are as diverse as instructing the observer to sight a target through a ring, or to report which half-image is perceived more of during binocular rivalry stimulation. Conventionally, the former task is said to reveal motor eye dominance while the latter task reveals sensory eye dominance. While the consensus is that the motor and sensory-dominant eye could be different in some observers, the reason for it is still unclear and has not been much researched. This review mainly focuses on advances made in recent studies of sensory eye dominance. It reviews studies conducted to quantify and relate sensory eye dominance to other visual functions, in particular to stereopsis, as well as studies conducted to explore its plasticity. It is recognized that sensory eye dominance in observers with clinically normal vision shares some similarity with amblyopia at least at the behavioral level, in that both exhibit an imbalance of interocular inhibition. Furthermore, sensory eye dominance is probably manifested at multiple sites along the visual pathway, perhaps including the level of ocular dominance columns. But future studies with high-resolution brain imaging approaches are required to confirm this speculation in the human visual system.

Keywords: amblyopia, binocular combination, binocular rivalry, excitatory-inhibitory balance, plasticity, stereopsis

Introduction

The efficiency of a component that makes up a pair of bilateral organs could either be equal, better or worse than its other component. This could lead to a preference for using the more efficient component, which is then referred to as the dominant component. For example, if one prefers to accomplish tasks such as viewing through a monocular with the right eye, then the right eye is labelled as the dominant eye.

Systematic mentions of eye dominance probably began roughly 500 years ago even though phenomenological observations of having an eye preference date back much earlier.[47] Closer to our recent history, there was a flurry of scientific activities that developed different methods for measuring eye dominance in an attempt to understand its basis.[5,10,24,31,32,45,46] Walls[45] for instance, listed twenty five different behavioral measurement criteria for determining the dominant eye. He further divided these criteria into five different groups and discussed the credibility of each group of criterion. These early researchers also noticed that not all criteria/tests revealed the same eye as the dominant eye, suggesting that dominance is likely task specific.

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Coren and Kaplan[4] further pared down the various tests into three categories: sighting dominance, sensory dominance and acuity dominance. Acuity dominance refers to the predominant use of the eye with the better quality image [better visual acuity (VA)]; this type of dominance may become more important in patients with ocular diseases. Sensory eye dominance (SED) testing is usually conducted by having the observer view a binocular rivalry (BR) target. On the other hand, sighting eye dominance testing is usually implemented by having the observer sight a target through a hole or a ring, or point to a target, in turn with each hand. Perhaps because sighting dominance testing is performed while the observer is using his/her hands (e.g. holding a card or pointing to a target), the test is also referred to as a motor eye dominance test. Most works on eye dominance e.g.[14,37,46,52,57] have found that sensory and motor eye dominance are not always correlated. But how these various classifications of eye dominance are related is still unclear. Also, unclear is the neural underpinnings of eye dominance.[30,35,40]

The present review focuses on eye dominance researches conducted in the last two decades, which are mainly related to sensory eye dominance. While the questions surrounding eye dominance remain unchanged, recent works have greatly benefited from the pioneering works of the twentieth century. This, coupled with the technological advances over the last few decades might bring us closer to unravelling the puzzle of eye dominance. Currently, most measurements of SED are based on two binocular visual phenomena, namely, binocular rivalry (BR) and binocular phase combination. We begin below with descriptions of SED measured with both types of binocular visual percepts.

Sensory Eye Dominance Measured with the Binocular Rivalry Stimulus

The popular adoption of computerized systems for generating visual stimuli to conduct psychophysical experiments about twenty years ago permitted more precise control of stimulation and quantitative measurements of the behavioral responses. This allowed our laboratory to measure SED with a novel "BR balancing" method, where we presented paired BR half-images (dichoptic red and green-oriented gratings) with six combinations of luminance levels. The stimulus duration was 333 msec and the stimuli were presented using the method of constant stimuli.[37] For each trial with a set right and left eye luminance combination, the observer indicated whether he/she saw more red or green-oriented grating. The luminance of the grating pair that led to the observer seeing about equal portions of right and left eye gratings was taken as the balance point. The difference in balance points between the two eyes, i.e. the luminance levels required to see about equal proportions of both eyes' gratings provides a quantitative measure of SED. We found our observers exhibited different amounts of SED, from being almost balanced, to mild (< 0.2 log unit) and moderately imbalanced (> 0.5 log unit). This finding of substantial variation in SED was surprising to us at that time, as these observers were young adults with clinically normal binocular vision (VA of 20/20 or better in each eye and at least 40 arc sec stereopsis with the high-contrast Titmus stereo test).

In a subsequent study also on clinically normal observers, we modulated the contrast of the BR stimuli (instead of luminance) using the QUEST method in conjunction with the BR balancing method to measure SED. Depending on the observer's response, either seeing more of the oriented grating from the left or right eye, the grating contrast of the tested eye was adjusted using QUEST method. In this way, the balance point was obtained for each eye and the difference between the balance points provided a measure of SED. SED was measured locally at seventeen different retinal locations at eccentricities of 0° (fovea), 2° and 4°.[53] We found the sign and magnitude of SED varied across the retinal areas tested for each individual observer. This suggests a localized neural interaction that is location specific determines the balance between binocularly corresponding retinal regions. Nevertheless, the foveal SED tends to be correlated with the average parafoveal SED, further suggesting that adjacent corresponding regions are more similarly affected.

We have since measured SED on amblyopic patients using the BR balancing method.[39] We found that amblyopes have much larger magnitudes of SED (about 1.0 log unit or larger) than observers with clinically normal vision. Altogether, our psychophysical data suggest there exists a continuum of SED that may be tied to the binocular visual status of the observers. But it is unknown if the underlying neural mechanisms giving rise to the behavioral measures of SED continuum are the same in the amblyopic and non-amblyopic populations.

It should be noted that other than the BR balancing method, there are other ways to test SED that are also based on instigating BR. For example, Yang et al,[57] stimulated one eye with dynamic noise while the fellow eye was stimulated with the target stimulus (i.e. non-identical BR stimulus). The observer's task was to indicate when

he/she first saw the target, which was initially suppressed by the dynamic noise. The eye that was quicker to see the target was determined to be the sensory-dominant eye.

The sensory-dominant eye can also be determined by instructing the observer to respond, via key-presses, to the instantaneous percepts while viewing a BR stimulus for an extended period of time (e.g. 30–60 sec). With this BR tracking method, the dynamics of BR alternation are recorded. The eye that sees its image for a longer duration of time and/or more frequently is the sensory-dominant eye.[6,27,53,54,55] Studies from our laboratory have shown that the same eye is found to be the sensory-dominant eye with the balancing and tracking methods.

It should be noted that while the indices (magnitudes or extents) measured to reveal sensory eye dominance in the various studies mentioned above might be different (e.g. contrast, luminance, response time, or predominance), they are all based on stimulation of BR. Furthermore, the duration of BR stimulation could be varied. It has been shown that the typical BR stimulus, e.g. a pair of orthogonal grating discs, is able to instigate BR after a stimulus presentation duration of 150 msec or longer.[43,50] Conceivably, using a longer BR presentation duration exposes the visual system to more top-down attentional and cognitive influences,[17,36] which in turn, could influence the SED measured.

Sensory Eye Dominance Measured with the Binocular Phase Combination Stimulus

In an attempt to explore how binocular contrast signals combine, Ding and Sperling,[9] designed a pair of dichoptic stimulus whose half-images were horizontal gratings with a slight phase shift relative to one another. This suprathreshold binocular summation stimulus has since been referred to as the binocular phase combination stimulus, as opposed to a (suprathreshold) binocular rivalry stimulus wherein the half-images have orthogonal orientations. Various laboratories have used the binocular phase combination stimulus to measure SED.[8,19,59] For simplicity, we have coined the SED revealed by the binocular phase combination stimulus as SED_{combo}, and SED measured by the BR stimulus as $SED_{inhibition}$.[12]

But do the binocular phase combination and BR stimuli produce similar behavioral measures of SED? After all, measuring SED_{combo} is based on potentially fusible horizontal grating stimuli whereas measuring $SED_{inhibition}$ is based on rivalrous stimuli (typically orthogonal gratings). Our laboratory investigated this by testing the same observers with both types of stimuli while matching as much as possible the parameters and designs of both stimuli.[12] We found SED measured with both stimulus types was, for the most part, similar in sign and magnitude. We also found both types of SED having a stronger correlation with stereopsis than with contrast sensitivity. These findings suggest a common origin for interocular imbalance in the two different binocular processes and that both types of SED are significant factors in impeding stereopsis. Various contrast gain control models of binocular phase combination[1,8,20] would suggest that SED_{combo} could possibly be caused by a stronger inhibition to the weak eye by signals from the fellow eye. It would be interesting to explore if these theoretical inhibitory networks are also responsible for binocular rivalry and $SED_{inhibition}$. Currently, models of BR do not address SED, and models of binocular phase combination are based on stimulation of horizontal orientation channels in both eyes that do not carry horizontal binocular disparity information.

Relationship Between Sensory Eye Dominance and Visual Functions

Investigating what monocular and binocular functions are compromised when one has SED can help identify the neural site(s) involved. To do so, one can compare the observers' SED with their monocular contrast sensitivity, suprathreshold brightness perception, visual acuity and stereopsis. Generally, it was found that while some observers' SED could be attributed to weaker monocular signals, this was not always the case.[12,37,53,57]

A stronger correlation exists between SED and stereopsis.[7,12,37,53] Stereo threshold was lower and stereo reaction time was faster in observers with little or no SED. Furthermore, using suprathreshold stereo half-images of different interocular contrast, we found observers had lower stereo thresholds and shorter stereo reaction time when the non-sensory-dominant eye viewed the higher contrast half-image. This suggests a stimulus-compensating effect.[13] Overall, the findings of correlation between SED and stereopsis but not with monocular visual functions point to a binocular basis of SED, possibly with an imbalance of interocular inhibition at its core.

The Neural Loci of Sensory Eye Dominance

Since most binocular visual functions are processed in a distributed manner, there is probably no one locus that

could account for SED, at least for that which is measured using the BR stimuli.[36,38,44] Consistent with this view, single unit recordings in monkeys[25] and fMRI imaging in humans[60,16,51] showed neural activities related to BR in extrastriate cortices, striate cortex, and lateral geniculate nucleus. But it is unknown if the activation signals reflect feedforward or feedback interactions.

Nevertheless, it is reasonable to suggest that the BR phenomenon demonstrates the retention of the eye-of-origin signature albeit at an unconscious level.[36] An indirect physiological evidence for this is from Xu et al[56] optical imaging study. They imaged anesthetized monkeys' V1 activities while stimulated with BR stimuli, and were able to show activity changes in the ocular dominance columns. The authors further provided several lines of evidence that the activity changes were consistent with the known BR dynamics.

From the foregoing, it is tempting to suggest that SED measured with the BR method could be gleaned from the structures and/or activities of the ocular dominance columns with the brain imaging approach, in a manner similar to those groundbreaking works by Hubel and Wiesel in animal models of amblyopia.[21,48,49] However, there are species-specific and technological barriers to overcome before we could explore this possibility in human. Namely, we would need a high-resolution imaging system that is capable of revealing possibly minute differences in activities in the human ocular dominance columns.

Long-Term Modification of SED in Adults and Possible Link to Ocular Dominance Plasticity

While the theoretical significance of SED can be traced to classical debates of the nature of binocular vision, it is also of clinical significance because SED is correlated with reduced stereopsis. Our laboratory thus devised a perceptual learning paradigm, called the "push-pull" paradigm, to investigate if training with the push-pull paradigm could reduce SED and improve stereopsis.[52] The push-pull paradigm simultaneously causes inhibition to the dominant eye and excitation to the non-dominant eye at corresponding retinal areas. We proceeded to show that 7–10 days of training (over ten sessions) of non-amblyopic adult observers were sufficient to reduce SED and improve stereopsis. As a control, the same observers were exposed to a "push-only" paradigm at different retinal locations, where the dominant eye was not stimulated while the non-dominant eye was excited ("push"). We found little change in SED and stereopsis with the "push-only" paradigm over the same training duration. That the "push-pull" paradigm was more effective highlights the role of strenghtening inhibition of the non-dominant eye onto the dominant eye in order to rebalance the eyes. Subsequently, we implemented the push-pull paradigm on amblyopic observers and found the same outcomes.[39] Both studies also found the positive outcomes to be retained months after the end of training, indicating induction of long-term visual plasticity in the adult population.

The last few decades have seen significant molecular and genetic advancements in models of ocular dominance plasticity in juvenile and mature animals. For example, works by Hensch et al,[18] and Harauzov et al,[15] point to cortical inhibition as an important factor in controlling cortical plasticity, respectively, in juvenile and adult rodents. Furthermore, Maya Vetencourt et al[61] showed that application of fluoxetine, a selective serotonin reuptake inhibitor that reduces GABAergic inhibition, reinstated ocular dominance plasticity in adult rats and promoted recovery of visual functions. They further theorized that fluoxetine shifted the intracortical excitatory-inhibitory (E/I) balance, which triggered the plasticity. It is thus tempting to reason that the reduction in SED after undergoing the push-pull training, which excites (push) one eye and inhibits (pull) the fellow eye, is related to resetting of similar processes of E/I balance in the human visual system – although one has to take species differences into consideration.

Other laboratories have also implemented various binocular-based perceptual learning methods to improve vision in amblyopic adults. Li et al,[26] implemented a dichoptic training method where the amblyopic eye was excited with high contrast images and the fellow eye excited with low contrast images at non-corresponding retinal areas. Although they did not measure SED, they used a dichoptic global motion stimulus to measure the signal-to-noise ratio for seeing coherent motion in order to determine suppression depth. They found reduced suppression in the amblyopia eye and improved stereopsis. Ding and Levi[62] trained their amblyopic observers using a perceptual learning method that combined monocular and binocular disparity cues. Though they did not measure SED with the BR method, they reported improved stereopsis after the training. Altogether, these studies reinforce the notion that the adult binocular visual system retains its plasticity.

Short-Term Monocular Deprivation Temporarily Boosts Sensory Dominance of Previously Deprived Eye

It has been reported that patching an eye for about 2 hrs (short-term deprivation) of non-amblyopic adult observers led to it becoming more dominant after the patch was removed.[27,58] This intriguing phenomenon is observed when SED was measured with either the BR or binocular phase combination methods. To reveal the characteristics of short-term deprivation, Min et al[33,34] investigated if the boost in SED could be increased (cumulative effect) with longer deprivation duration or repeated deprivations over several days. Interestingly, they found no cumulative effect, which suggested to them that the phenomenon acts in an all-or-none fashion. Separately, to test whether the acetylcholine neuromodulator enhances visual system's plasticity during short-term deprivation, Sheynin et al,[42] administered a single dose of 5mg donepezil (cholinergic inhibitor) before commencing the experiment. They found that the cholinergic dosage acted to reduce the magnitude and duration of the shift in dominance to the previously deprived eye.

Animal studies of amblyopia in the rodents have shown that physical activity promotes recovery in amblyopia.[22,41] To test if this applies to human observers, Lunghi and Sale,[29] tested two groups of non-amblyopic human observers who underwent short-term monocular deprivation. They found that the group whose deprivation was combined with physical activity (bicycling) exhibited enhanced deprivation effect compared to the group that did not have physical activity. However, in contrast to the finding by Lunghi and Sale,[29] Finn et al,[11] found no effect of physical activity in their group of adult non-amblyopic observers even though they used the same BR method to measure the change in SED. This latter study corroborates an earlier study by Zhou et al,[63] who used the binocular phase combination method to measure SED.

The short-term deprivation effect favoring improved dominance of the previously deprived eye has been attributed to homeostatic plasticity in the adult visual system, which is tied to reduced inhibition of the previously deprived eye. Bolstering this claim, Lunghi et al[28] used MR spectroscopy to show that resting GABA level in V1 decreased after deprivation. Furthermore, imaging studies showed that fMRI BOLD responses[2] and MEG responses[3] of the previously deprived eye were increased.

From a theoretical perspective, this observation of improved dominance of the previously deprived eye is thought to reflect a readjustment of the gain control mechanism between the two eyes. Further supporting this idea, Kim et al,[23] demonstrated that rather than temporarily occluding an eye, a similar effect could be obtained by causing the eye to be suppressed using the continuous flash suppression paradigm. This led them to suggest that rather than the occluding patch depriving the eye from pattern vision, the trigger for the change in eye dominance could be suppression of the short-term deprived eye.

Summary

This review has delved into knowledge acquired in recent decades on the topic of sensory eye dominance. Though we are still far from understanding the relationship between sensory and motor eye dominance, recent findings not only enrich our appreciation of at least one aspect of eye dominance but also offer novel way to reduce the sensory imbalance and improve stereopsis. On a larger scale, studies of sensory eye dominance might provide an insight into the neural mechanisms underlying the excitatory-and-inhibitory balance in normal visual processing and in amblyopia.

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Disclosure

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