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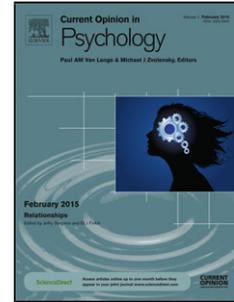
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Attention Deficits in Amblyopia

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Abstract:

Amblyopia is a neuro-developmental abnormality associated with deficits in a broad range of both low- and high-level visual tasks. This is particularly true in strabismic amblyopia where fixation is unstable and there is an increased frequency of microsaccades. In light of the close association between eye movements and attention, we propose a novel hypothesis: that the cost of unstable fixation in amblyopia is a deficit in selective attention. The increased latency for saccades and manual response time with amblyopic-eye viewing is consistent with attention being distracted by unwanted fixational eye movements. We review other attention deficits in amblyopia and discuss whether they are explained by fixation instability, or whether they involve a form of neglect or suppression of the visual input from the amblyopic eye.

Amblyopia ("lazy eye") is a common developmental abnormality that results in a loss of visual acuity in one eye that cannot be corrected by glasses or contact lenses. Typically, the acuity of the fellow eye is normal. Historically, the amblyopic deficit has been attributed to neural losses in early visual cortex, primarily V1 [1,2,3**]. While damage to both striate and extra-striate cortex undoubtedly contributes to the amblyopic loss, in this review we present a body of evidence that response latency is significantly impacted in amblyopia, suggesting a broader deficit than visual processing. Response latency has frequently been used as a metric of attention in studies ranging from the classic Posner cueing tasks [4] to performance in visual search tasks. We propose that the increased latency for saccades and manual

responses in amblyopia is due to an attentional deficit arising from poor fixation stability and an increased frequency of microsaccades, and review work that suggests that these factors impact the dynamics and the spatial allocation of selective attention.

Attention and eye movements

Goldberg and Wurtz [5] demonstrated a strong association between eye movements and attention. They showed that cells in the superficial layers of the superior colliculus respond more strongly when the animal attends to that location and prepares a saccade to a stimulus within the response field, compared to when the animal fixates or prepares a saccade to a location outside the response field. Rizzolatti and colleagues [6**] proposed the "premotor theory of attention" whereby attention is tightly linked to the saccade goal and the same cortical networks control attention as well as eye movements. This bold proposal is supported by neurophysiological studies in awake behaving primates as well as functional neuroimaging studies in humans that show that the same cortical structures in frontal, parietal and extrastriate cortex are involved in overt as well as covert attention [7-11].

The association between eye movements and attention is evident even when observers are required to maintain fixation and attend covertly to a cued location. Hafed and Clark [12*] showed that during a covert attention task, observers tend to make small eye movements (microsaccades with amplitude < 1°) in the direction of the cue. Furthermore, stimuli that appear at a location congruent with the direction of a microsaccade are discriminated better than those at incongruent locations, further supporting the link between saccadic eye movements and attention ([13], even when observers are required to fixate and attend covertly.

The attentional cost of fixational instability

Individuals with amblyopia, particularly those with strabismus, have poor fixation stability in their amblyopic eye [14, 15-16, 17*, 18-20]. In tasks that require fixation, the position of the amblyopic eye tends to drift from fixation and the frequency and amplitude of microsaccades is higher than for the fellow eye. This is particularly true of strabismic amblyopes, regardless of whether the viewing is binocular or with the amblyopic eye alone [17*, 21]. Critically, Chung et al. [14] used retinal imaging in a scanning laser ophthalmoscope to show that the greater error magnitude (the difference between the landing position of the microsaccade relative to the locus of the fixation marker on the retina) was the largest contributor to fixation instability of the amblyopic eye in strabismus. So rather than correcting for drift, the increased error magnitude often took the eye further from its intended landing position. For strabismic amblyopes the drift and associated microsaccades are mostly horizontal [22; also see 14 Fig. 1], whereas for anisometric amblyopes the drift patterns are more like a random walk, so the net deviation from fixation is smaller (23; 14).

The crux of our proposal is that this fixational instability manifests as an attention distractor [24]. The typical coupling of attention and eye movements implies that when a microsaccade occurs, attention is tied to the location of saccade goal [25**]. With the increased frequency of microsaccades in amblyopia, attention is being continuously shifted from one location to another, and is less available to respond to the appearance of a stimulus. This causes delays when the amblyopic observer is asked to maintain fixation on a marker and respond as quickly as possible to the appearance of a stimulus, as vigilance is compromised by frequent microsaccades [26]. This distracting component of attention could apply to all motor latencies, including eye and hand. Another factor that affects saccadic latency is the refractory period for saccades of about 150 ms [27,28], which places a lower bound on how quickly the eye can move following a preceding microsaccade. Previous work with non-amblyopic observers shows that microsaccades increase the initiation time of a saccade: saccadic latency increases by 40 ms when microsaccades occur within 150 ms before the presentation of a saccade target [29]. Thus, the increased frequency of microsaccades in amblyopia could add a further delay to saccadic latency as the eye is more likely to be in a saccade-related refractory period at any given time.

Increased Saccadic latencies in amblyopia

Both saccadic and manual latencies are slower in strabismic amblyopia [15,24,30-35, 36**]. Von Noorden [37] was the first to show increased latency of manual responses when viewing a central (rather than peripheral) target with the amblyopic eye. He also showed that the delay is even longer when the amblyopic eye is tested under conditions of binocular fixation. This additional delay may be the result of interocular suppression of the amblyopic eye by the dominant fellow eye [37]. The delay in both saccadic and manual reaction time is strongly correlated with the reduction in acuity in the amblyopic eye [24,32]. However, the increased delay is not simply a consequence of reduced acuity or contrast sensitivity. For example, Levi et al. [31] studied this manual latency difference as a function of contrast and fit the data with a power function as predicted by Pieron's law [38,39]. Their study showed that while response times decreased with increasing contrast in both eyes, the amblyopic eye needed higher contrast to reach asymptotic response time. McKee et al. [24] considered the possibility that the higher contrast required by the amblyopic eye might simply reflect its lower sensitivity and that the latency vs. contrast functions would superimpose if the latency were plotted against multiples of contrast threshold in each eye (effective contrast). They replotted one observer's data from Levi et al. [32] and showed that while the functions for the two eyes almost overlapped, there was still an irreducible difference in the asymptotic latency for manual response times, with the amblyopic eye having a 46 ms longer latency. To investigate this persistent difference thoroughly, Gambacorta et al. [36**] examined latency for saccades and hands when the effective contrasts in the two eyes were equated. They measured the latency vs. effective contrast for both anisometropic and strabismic amblyopes and found that equating contrast causes the latency vs. effective contrast function to superimpose for anisometropic and some strabismic amblyopes, especially for manual latencies.

However, for other strabismic amblyopes, the amblyopic eye has an additional delay with respect to the non-amblyopic eye of about 75 ms for saccades and a delay of about half of this amount for manual latencies, Moreover, Gambacorta et al. [31**] also showed that saccadic and manual reaction times are highly correlated (> 0.9) in the amblyopic eye, which indicates that common factors are producing a delay in both motor systems. They argue that the common factor involves attention whereby the increased frequency of microsaccades in strabismic amblyopes distracts attention from events such as target onset, resulting in an increase of both saccadic and manual latencies. The frequent microsaccades also increase the time that the amblyopic eye is in a refractory period from a previous saccade. Thus, saccadic latency suffers from two factors both due to microsaccades — distracted attention and the motor refractory period – and therefore exhibits a greater delay than manual latency.

Would the contribution of microsaccades to delays in saccadic latency be minimized if the demand for steady fixation were removed, by turning off the fixation point prior to test target onset, as in the 'gap' effect? Strabismic amblyopes do show the normal 'gap' improvement in saccadic latency in both their normal and amblyopic eyes, but the interocular difference in saccadic latency remains essentially unchanged by the introduction of a gap between fixation point and target (36**). Perhaps the pattern of drift followed by corrective saccades occurs in strabismic amblyopes, even in the absence of a fixation target. In fact, Schor and Hallmark [19] observed large drifts and saccades when their strabismic observers attempted to maintain fixation in darkness with their amblyopic eyes.

Other attentional deficits in amblyopia

Does poor contrast sensitivity and the distraction of attention by the increased frequency of microsaccades account for all reaction time delays, or could there be other factors that impose a general sluggishness in the processing of input from the amblyopic eye? For instance, we have shown that the time for a cue to reach peak effectiveness is delayed in strabismic amblyopia [40]. This is true for both peripheral and central cues. The delay for the amblyopic relative to the fellow eye was greater under binocular presentation, when the stimuli could appear randomly in either eye, compared to monocular presentation, when the stimuli appeared in one eye for a block of trials. However Roberts et al. [41] found no significant delay in response time for the amblyopic vs. the fellow eye for either anisometropic or strabismic participants. Differences in the stimuli used may explain the different pattern of results: the stimuli in Roberts et al. [41] were unmasked and observers were not under any time pressure to respond, resulting in latencies greater than 1 s. More importantly only 4 of the 14 participants were amblyopic, and of these 4 amblyopic participants, only one was strabismic.

The attention deficits due to fixation instability in strabismic amblyopia may not be the whole story. It is clear that amblyopia is associated with impaired performance at tasks that require attention such as multiple-object tracking [42] or detecting the change in direction of motion of multiple trajectories, particularly when the magnitude of change is small [43]. Studies suggest that in the case of strabismic amblyopia, attending to a cue produces a smaller enhancement for input in the amblyopic eye. Hou and coworkers [44] measured SSVEP responses to inputs in the amblyopic and fellow eyes in striate and extra-striate cortical areas while participants were cued to attend to a stimulus on the right or the left. In their study, strabismic amblyopes showed smaller attentional modulation to a cue than did controls and V1 responses were diminished for inputs to the amblyopic eye [44]. Extra-striate responses were diminished for inputs in both the amblyopic and fellow eyes. It is unclear whether this reduced gain is related to the common delay that exists for saccadic and manual reaction times or whether there is an additional mechanism that down-weights input from the amblyopic eye so that it is in a state of attentional neglect [45]. In another study that examined the effect of an attentional cue Pham, Carrasco and Kiorpes [46] measured direction discrimination as a function of contrast in monkeys with amblyopia. The animal was either validly cued to the location of the single vertical grating among seven patches of different orientations, or provided an uninformative neutral cue. Performance in amblyopic monkeys in response to an attentional cue increased both contrast gain and response gain. However, for strabismic monkeys a valid cue in the amblyopic eye did not increase response gain to the level of the fellow eye with a neutral cue. Thus, while the amblyopic eye can benefit from an attentional cue, the response to the cue does not compensate for the lower sensitivity of the amblyopic eye. The Hou et al. [44] and the Pham et al. [46] studies suggest that the input from the amblyopic eye may be habitually down weighted.

Other studies, such as undercounting features in a numerosity judgment task [47] when viewing with the amblyopic eye also point to deficits of attention. One possibility is that when the number of features is large (>5), observers are unable to switch attention to another cluster to subitize and estimate the number within the limited display time using their amblyopic eye. Sharma et al. [47] specifically tested whether extending the trial interval from 200 ms to 1 sec and removing the mask alleviated the undercounting deficit. The undercounting deficit decreased slightly but did not quite reach the performance of the fellow eye, suggesting that perhaps there maybe more than a temporal switching cost to the attention deficit in amblyopia.

The attentional blink is different in amblyopia [48]. The blink appears to be less finely tuned in the amblyopic eye, although the coarse (100 ms) sampling used in the study [48] makes it unclear whether it is delayed. Importantly, the pattern of errors is different than for controls. For the first target letter (T1), observers viewing with their amblyopic eye often report the letter that occurred before T1, suggesting delays in processing such that the previous letter was still being processed when T1 appeared. This effect is consistent with altered dynamics of spatial attention. However, when reporting the second

target letter (T2), amblyopic observers report letters that were never presented, suggesting an erroneous binding of letter features. This might be similar to a deficit in contour integration that we will discuss later, and might seem unrelated to the increased frequency of microsaccades, as only one letter was presented at a time. However, it is possible that the suppression associated with saccades prolongs the time required for evidence to accumulate to reach a perceptual decision [49]. Moreover, amblyopic individuals have longer integration times for higher spatial-frequency stimuli [50], and they show temporal crowding [51], both of which are consistent with frequent periods of suppression that might act like "time-outs".

Farzin and Norcia [52] reported that with the amblyopic eye viewing, choice response times to report the location of a gap (left/right) on a Landolt C are longer. They interpreted this as a consequence of attentional neglect; however an alternative explanation may be inappropriate spatial integration. Their finding, that a simple response-time task did not result in significantly longer delays with amblyopic-eye viewing, can be explained by the dependence of simple response time on stimulus contrast— the high contrast of the stimulus in Farzin and Norcia [52] probably made it clearly visible to the amblyopic eye of observers.

Contour integration and Crowding in Amblyopia

Normal observers can easily find a contour formed of roughly collinear Gabor patches presented amidst a dense background of randomly oriented patches. Amblyopic humans show difficulties in detecting contours in noise [53-58]. This deficit is not due to reduced visibility or increased positional uncertainty [59]. One possible explanation is that the amblyopic eye is impaired at using a contour as a cue. For control participants, Verghese [60] showed that parts of a contour act as a cue to other parts of the contour such that the presence of two or more aligned patches enhances contrast discrimination for a test patch that lies along the implicit path. This self-cueing effect is diminished in amblyopia [61]. While normal controls use a contour cue to find the test patch in an unknown location in noise and discriminate its contrast, amblyopic observers have great difficulty with the presence of noise, even when the test patch is in a fixed location. Interestingly, their contrast discrimination is unimpaired in the absence of noise. These studies suggest that spatial integration is impacted in amblyopia and may be due to the same underlying factors that result in abnormally large crowding zones in amblyopia. Crowding is the spatial pooling of information within a crowding zone that impairs the ability to detect an object in the periphery particularly when it is flanked by other objects. Crowding represents a bottleneck to object recognition and reading in amblyopia [62,63]. As noted above, in normal observers microsaccades can lead to shifts in attention; to the extent that similar attentional shifts occur when strabismic amblyopes make large microsaccades, this may result in increased crowding and substitution errors (i.e., mistakenly reporting a flanker rather than the target) because attention is erroneously directed at a flanker rather than the target [64]. For example, in a crowded acuity task with a 15 arcmin letter (20/60), a 15' microsaccade could result in fixation (and/or attention) on an abutting flank.

Conclusion

Several lines of evidence point to attention deficits in amblyopia. Here, we consider how much fixation instability and its contribution to distracting attention and increasing microsaccade-related refractory periods can explain these attention deficits. Clearly, attention deficits due to unstable fixation are highly consistent with increased latencies for both saccadic and manual latencies. It also possible that fixation-related attention deficits contribute to phenomena as diverse as undercounting, increased SOA for a cue to be effective, the attentional blink, contour integration and crowding deficits in amblyopia. However, there are clearly other results such as reduced response gain and reduced attentional modulation that suggest that fixation-related deficits are not the whole story, and that the attentional deficit in amblyopia may involve a habitual down-weighting of the input from the amblyopic eye, a form of attentional neglect.

Acknowledgments

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