

The contribution of foveal activation to the oculomotor gap effect



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HIGHLIGHTS

- We investigated the origin of the gap effect.
- Previous research has suggested the gap effect to be foveal specific.
- We used a gaze-contingent fixation adaptation of the fixation offset task.
- The gap effect is mediated by more than foveal specific factors alone.
- Lateral interactions in the superior colliculus also contribute to the gap effect.

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ABSTRACT

Saccade initiation is facilitated when there is no physical stimulus at the start position of the saccade. There have been numerous explanations for this 'gap effect', the most prominent one being the facilitated release from active fixation when no visual information is present. Attributed to potential fixation sensitive neurons in the superior colliculus, previous research has suggested the gap effect to be a foveal specific effect. The aim of the present study was to investigate whether the gap effect is strictly a foveal effect by using a gaze-contingent eccentric fixation adaptation of the fixation offset paradigm. Results show that, although the gap effect is diminished under eccentric viewing conditions, it is still significantly present. This suggests that the gap effect is mediated by more than foveal specific factors alone. We argue that lateral interactions in the superior colliculus may also contribute to the gap effect.

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1. Introduction

Saccades are the rapid movements of the eyeball to direct the fovea onto an object or region of interest. In many situations in daily life a saccade will depart from a location at which a visual object is presented. It is, however, also possible to start a saccade from an 'empty' location, i.e., a location without any strong visual information, like an empty spot on a wall. When these two situations are simulated in an experimental situation, results have shown that saccade initiation is facilitated in the condition in which the saccade is initiated from an empty location [6,18]. This has been termed the 'gap effect' and reflects the shortening of saccade latencies if a fixation point disappears before a target appears (gap condition) compared to when the fixation point is still present at target

presentation (overlap condition). This reduction in saccade latency is maximum when the fixation point disappears 200–300 ms before target presentation, but is also observed when the fixation point disappears simultaneously with the presentation of the target [18].

It has been hypothesized that the gap effect involves two components [7]: one involving generalized response preparation due to the warning effect evoked by the offset of the fixation point [16,17] and one involving a facilitated release from active fixation. This facilitated release is caused by the lack of any visual presentation at fixation, making it easier to end the current fixation and initiate the saccade [3,5,11,15]. Support for the facilitated release was provided by neurophysiological recordings in the superior colliculus (SC), an area in the midbrain strongly associated with oculomotor programming [3,11]. Before a saccade can be initiated, fixation related neurons in the rostral pole of the SC are deactivated. The offset of the fixation point prior to a saccade facilitates this deactivation, resulting in a reduced saccade latency [3,11]. Indeed, the fixation offset causes the discharge of fixation related neurons to drop to about 65% of their initial rate. Furthermore, the changes in the discharge rate of rostral pole fixation neurons which occur

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when a fixation point is turned off are known to reflect the time course of the gap effect [3], providing further evidence for a colliculus driven explanation of the gap effect [although for a different explanation of the functioning of rostral neurons, see [9]].

Assuming that the gap effect is indeed mediated by SC fixation cells, Fendrich and colleagues [4] hypothesized that the effect should be reduced or eliminated by the use of an eccentric (non-foveal) fixation anchor, especially when there is no generalized response preparation. With such an anchor, there is no visual stimulation of the fixation cells in the SC, so any offset of the fixation anchor should not evoke a gap effect. To exclude the influence of any generalized response preparation, they also included a zero gap condition, in which the fixation anchor disappeared simultaneously with the target presentation. Indeed, when subjects fixated the empty centre of a square formed by four points with an eccentricity of more than 2°, saccade latencies were similar between the zero gap condition and the overlap condition. In contrast, a reduction was observed with a foveal fixation area (i.e., the corners of the square forming the eccentric fixation point were only 1° from the fovea). Based on these results, they reasoned that the gap effect could be attributed to the facilitated shutdown of colliculus rostral pole fixation neurons.

To verify the conclusion that foveal stimulation is indeed crucial to observe the gap effect, we employed a gaze-contingent eccentric fixation in which fixation was controlled peripherally and there was a zero ms gap between the offset of the fixation point and the onset of the target. So, although the fixation anchor was presented at the centre of the screen, the fovea was pointing to a peripheral location. Therefore, it should not matter whether the corners of the square forming the eccentric fixation point were 1° or 3° from the centre of this fixation anchor, because in both conditions there was no foveal activation as the fovea was pointed to an empty location. We compared the results of participants performing this 'peripheral' condition to the results of participants performing a 'central' condition in which the fixation anchors were presented at the centre of the screen and participants were required to look directly at it. For the central group, the 1° condition reflected foveal activation whereas the 3° did not. We therefore expected to replicate the findings of the zero gap manipulation of Fendrich and colleagues [4] for the central group in that a gap effect should only be observed for the condition with foveal activation. However, if foveal activation is indeed crucial to observe the gap effect, there should be no gap effect for both conditions in the peripheral group.

2. Method

2.1. Participants

For this study we recruited a total of 20 healthy participants with normal or corrected to normal vision (mean age: 27 years; 9 males). Ten participants performed the standard fixation offset paradigm and the other ten performed the gaze contingent version.

2.2. Fixation offset paradigm

To test fixation characteristics we used a fixation-offset paradigm [14]. All trials started with a drift check to ensure that calibration was still accurate. Participants viewed a display containing an eccentric anchor surrounding an *unmarked* centre (located at the centre of the display) with a background luminance of 32.07 cd/m². The eccentric fixation anchors consisted of four black crosses (0.64° × 0.64°) and were presented on the corners of an unmarked square. The distance from the crosses to the centre of the screen was either 3° or 1°. This way, participants viewed a fixation stimulus in which there is either foveal stimulation (1° fixation anchor) or

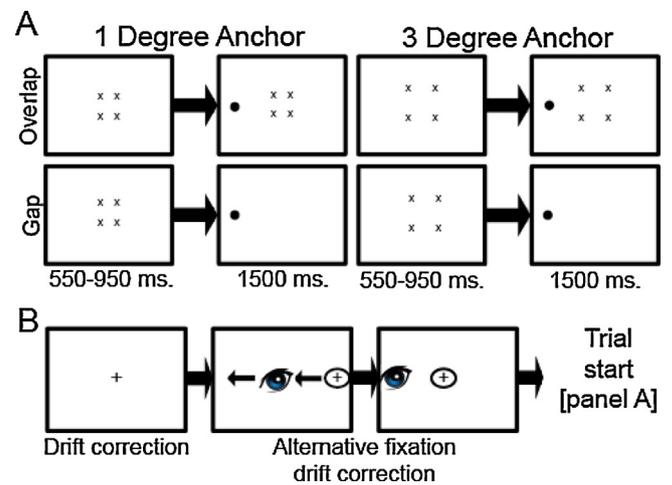


Fig. 1. Schematic overview of the two fixation offset paradigms used. Panel A shows the regular fixation offset paradigm. Prior to the start of each trial a drift check is performed after which the eccentric anchors are presented at either 1° or 3° of eccentricity. Next, a target is presented whereby in the gap condition the anchors disappear at target onset and in the overlap condition they remain visible. Panel B shows the adaptation made to the original paradigm. After the initial drift check a peripheral gaze-contingent fixation cross is shown and participants are instructed to align this cross with a central fixation cross using their eye movements. After stable alignment of these fixation the regular fixation offset paradigm is initiated with the only difference that targets are presented above, below or to the left of the true eye position as opposed to relative to the central fixation.

no foveal stimulation (3° fixation anchor). After a pseudo-random interval (between 550 and 950 ms.), a black target circle appeared (diameter of 1.43°). Simultaneously, the fixation stimulus disappeared (zero gap condition) or remained on the screen (overlap condition). See Fig. 1 for an overview. Participants were instructed to fixate at the *unmarked* centre until the target dot appeared, and subsequently were to move their eyes as fast as possible to the target circle. The target display was presented for 1500 ms. Afterwards all objects were removed from the display. The experiment consisted of 240 experimental trials and 24 practice trials.

2.3. Fixation offset gaze contingent paradigm

In the adapted peripheral paradigm, a para-foveal fixation cross was presented at 8° to the right side of the true fixation. This eccentric fixation was an offset of the eye-position as measured with the eye-tracker and was thus controlled by participants' eye-movement (e.g., gaze-contingent). Participants were instructed to move this alternative fixation point over a centrally located fixation cross, hold their fixation steady and press the spacebar. When this alternative fixation was stable within 2° of the central fixation cross, the trial started by removal of the central fixation point. Participants were instructed to keep their para-foveal fixation cross steady at moment the fixation cross disappeared. See Fig. 1B for a graphical overview. At the same time as the central fixation cross disappeared, fixation anchors were presented at either 1° or 3° eccentricity from the centre of the screen. These were exactly the same as in the original paradigm and were presented for the same pseudorandom interval (550–950 ms). After this a target was presented above, below or to the left of the true fixation and participants were instructed to move the eyes there as fast as possible and press the spacebar once they had done so. Also here, there was a zero gap and an overlap condition. Size of the target dots, fixation crosses and fixation anchors as well as all luminance ratios was kept the same as in the original paradigm. This task was programmed using PyGaze [2].

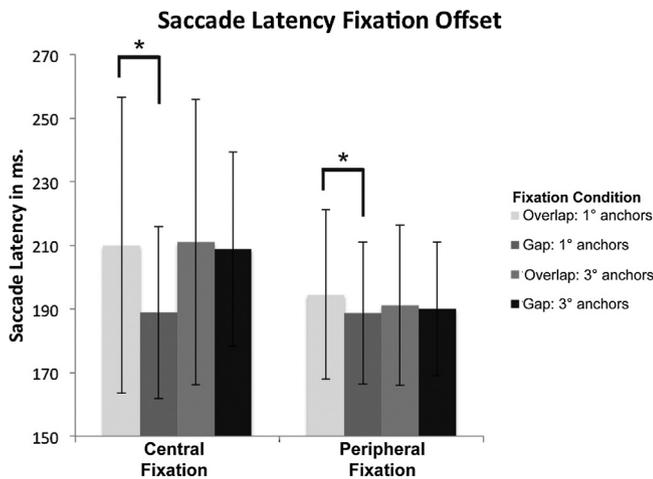


Fig. 2. Mean saccade latencies for both groups. For both groups, there is a clear fixation offset effect for the 1° fixation anchor, and no effect for the 3° fixation anchor. The fixation offset effect for the 1° fixation anchor is smaller for the peripheral fixation group compared to the central fixation group. Significant post hoc differences are marked with an asterisk. Error bars indicate standard deviations.

3. Results

Trials in which a saccade was made during the fixation phase of the paradigm (prior to target onset) were excluded from further analysis. Participants that made such saccades on more than 30% of the trials were excluded from analysis. If the saccade latency at target onset exceeded two standard deviations from the subject's mean saccade latency, those trials were excluded as well. In total this led to the exclusion of two participants (one in the central condition and one in the peripheral condition), leaving nine subjects in each group. In the central group 9.88% of trials were excluded on average and in the peripheral group 12.72% of trials were excluded on average based on these exclusion criteria.

We analyzed saccade latency using a $2 \times 2 \times 2$ repeated measures mixed analysis of variance (ANOVA) with factors anchor size (1° or 3°), offset type (gap or overlap) and group (central or peripheral). The $2 \times 2 \times 2$ mixed ANOVA showed a main effect of anchor size; $F(2,16)=13.455$, $p=0.002$, and a main effect of offset type; $F(2,16)=5.460$, $p=0.033$. In addition it revealed a significant interaction between anchor size and group; $F(2,16)=19.142$, $p<0.001$, between anchor size and offset type; $F(2,16)=72.131$, $p<0.001$; and a three-way interaction between all factors: $F(2,16)=29.782$, $p<0.001$.

To dissect this complicated three-way interaction, we performed a 2×2 ANOVA for each group separately with factors anchor size (1° or 3°) and offset type (gap or overlap).

For the central group, there was a significant main effect of anchor size $F(1,8)=28.347$, $p<0.001$, and a significant interaction between anchor size and offset type $F(1,8)=90.843$, $p<0.001$. In the peripheral group, the interaction between anchor size and offset type was at trend level: $F(1,8)=4.961$, $p=0.057$.

For both groups, the interaction between anchor size and offset type revealed a fixation offset effect in the 1° condition, with no effect in the 3° condition (see Fig. 2). Indeed, post hoc paired sampled t -tests showed that in the central group the saccade latency in the 1° gap condition was significantly faster compared to the 1° overlap condition: $t(8)=3.269$, $p=0.011$, while no such effect was present for the 3° overlap condition: $t<1$. The same holds for the peripheral group: saccade latencies in the 1° gap condition was significantly faster compared to the 1° overlap condition: $t(8)=2.952$, $p=0.018$. No effect was observed for the 3° overlap condition: $t<1$.

These results indicate that the fixation-offset effect in the 1° gap condition also occurs when people use a peripheral fixation anchor.

This effect was smaller, however, compared to the central group. When the fixation offset effect was transformed into a difference measure (the difference between the overlap and gap condition and subsequently the difference between this value between the three and 1° condition), a t -test between both groups revealed that the fixation offset effect was significantly stronger in the central group than in the peripheral group ($t(8)=5.866$; $p<.001$), explaining the three-way interaction between anchor size, offset type and group.

4. Discussion

In the present study, we investigated the contribution of foveal activation to the oculomotor gap effect. Previous studies have related the reduction in saccade latency in situations without foveal activation to a facilitated deactivation of fixation related neurons in the SC [3,4]. To test this hypothesis, we adopted a fixation anchor with and without foveal activation, using a square formed by four points with an eccentricity of either 1° (foveal activation) or 3° (no foveal activation). Indeed, when the conditions with and without foveal activation were compared under normal viewing conditions, a gap effect was only observed in the condition with foveal activation, replicating the results of Fendrich and colleagues [4]. The crucial condition, however, was performed by the peripheral group in which participants used a peripheral fixation under gaze-contingent control. For this group, both fixation anchors were not associated with foveal activation. Results still showed a small, but consistent, gap effect when a fixation anchor was used with eccentricity of 1°, but not when the eccentricity was 3°. So, although the gap effect was significantly smaller for the peripheral group, the significant reduction in saccade latency was still observed in a situation without foveal activation, rejecting the hypothesis that the gap effect is strictly due to a facilitated release from active fixation in situations in which the fixation point offsets simultaneously with target presentation.

One might argue that the difference in the gap effect might be influenced by differences in cognitive load between the two tasks. There are, however, two reasons why we consider an effect of an increased cognitive load unlikely. First, there was only a 3% difference in excluded trials between both groups, indicating that there was no strong difference in difficulty in keeping fixation between both tasks. Second, there was no significant difference in saccade latency between the two groups. As it is known that a higher cognitive load already influences simple pro-saccades [19], any influence of cognitive load on the current task should have been present in all conditions.

Our results suggest that the gap effect with a zero gap is mediated by multiple factors. Given that the gap effect was smaller with peripheral fixation than with direct fixation, visual stimulation of the fixation cells in the SC indeed seems to contribute to the gap effect. The results of the peripheral group clearly indicate, however, that this is not the sole factor underlying the gap effect. Previous studies have proposed that early general programming of the (oculo)motor response might be an additional factor [1,8,13,15]. These suggestions have been based on studies that have used an interval between the offset of the fixation point and the onset of the target. In these situations, the programming of the motor movement can already be initiated during the gap period. In the present study, we used a gap of zero milliseconds, ruling out the contribution of any general motor preparation. Furthermore, this explanation cannot account for the finding that the gap effect with peripheral fixation was only observed for the 1° fixation anchor.

The key to the increase in saccade latency for the 1° fixation anchor might lie in the lateral interactions in the SC. Within the motor map of the SC, connections between neurons are assumed to be mainly inhibitory, except for short connections which are

claimed to be excitatory [10]. For the 3° fixation anchor, activity evoked by the four corners of the anchor might be diminished due to the inhibitory connections between these remote locations. For the 1° fixation anchor, however, the peaks of activity associated with the four corners will result in a strong averaged peak at the intermediate location due to the local spread of excitatory activity. This strong peak of activity subsequently functions as a remote distractor, increasing saccade latencies to a remotely presented target [12].

In sum, our study has indicated that the visual stimulation of the fixation cells in the SC is not the sole explanation of the gap effect, but that lateral interactions in the oculomotor map also contribute to this multi-faceted phenomenon.

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