Small eye movements during fixation

The case of postsaccadic fixation and preparatory influences

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Abstract

Describing human eye movement behavior as an alternating sequence of saccades and fixations turns out to be an oversimplification because the eyes continue to move during fixation. Small-amplitude saccades (e.g., microsaccades) are typically observed 1-2 times per second during fixation.

Research on microsaccades came in two waves. Early studies on microsaccades were dominated by the question whether microsaccades affect visual perception, and by studies on the role of microsaccades in the process of fixation control. The lack of evidence for a unique role of microsaccades led to a very critical view on the importance of microsaccades.

Over the last years, microsaccades moved into focus again, revealing many interactions with perception, oculomotor control and cognition, as well as intriguing new insights into the neurophysiological implementation of microsaccades.

In contrast to early studies on microsaccades, recent findings on microsaccades were accompanied by the development of models of microsaccade generation. While the exact generating mechanisms vary between the models, they still share the assumption that microsaccades are generated in a topographically organized saccade motor map that includes a representation for small-amplitude saccades in the center of the map (with its neurophysiological implementation in the rostral pole of the superior colliculus).

In the present thesis I criticize that models of microsaccade generation are exclusively based on results obtained during prolonged presaccadic fixation. I argue that microsaccades should also be studied in a more natural situation, namely the fixation following large saccadic eye movements. Studying postsaccadic fixation offers a new window to falsify models that aim to account for the generation of small eye movements. I demonstrate that error signals (visual and extra-retinal), as well as non-error signals like target eccentricity influence the characteristics of small-amplitude eye movements.

These findings require a modification of a model introduced by Rolfs, Kliegl and Engbert (2008) in order to account for the generation of small-amplitude saccades during postsaccadic fixation. Moreover, I present a promising type of survival analysis that allowed me to examine time-dependent influences on postsaccadic eye movements. In addition, I examined the interplay of postsaccadic eye movements and postsaccadic location judgments, highlighting the need to include postsaccadic eye movements as covariate in the analyses of location judgments in the presented paradigm.

In a second goal, I tested model predictions concerning preparatory influences on microsaccade generation during presaccadic fixation. The observation, that the preparatory set significantly influenced microsaccade rate, supports the critical model assumption that increased fixation-related activity results in a larger number of microsaccades.

In the present thesis I present important influences on the generation of small-amplitude saccades during fixation. These eye movements constitute a rich oculomotor behavior which still poses many research questions. Certainly, small-amplitude saccades represent an interesting source of information and will continue to influence future studies on perception and cognition.

Zusammenfassung

Die Beschreibung des Blickbewegungsverhaltens als eine sich abwechselnde Folge von Sakkaden und Fixationen stellt eine starke Vereinfachung dar, denn auch während einer Fixation bewegen sich die Augen. Typischerweise treten Bewegungen von kleiner Amplitude (z.B. Mikrosakkaden), 1-2 mal pro Sekunde während einer Fixation auf.

Frühe Studien zu Mikrosakkaden wurden von Fragen bezüglich des Einflusses von Mikrosakkaden auf die visuelle Wahrnehmung, und Studien zu der Rolle von Mikrosakkaden bei der Fixationskontrolle dominiert. Fehlende Evidenz für eine Rolle, die ausschließlich Mikrosakkaden zufällt, führten zu einer sehr kritischen Betrachtung von Mikrosakkaden. In den letzten Jahren rückten Mikrosakkaden wieder mehr in den Fokus. Vielerlei Zusammenhänge mit Wahrnehmung, okulomotorischer Kontrolle und Kognition, sowie neue Erkenntnisse bezüglich der neurophysiologischen Implementierung von Mikrosakkaden konnten aufgedeckt werden.

In den letzten Jahren wurden verschiedene Modelle der Mikrosakkadengenerierung vorgestellt. Auch wenn sich diese in ihren exakten Mechanismen unterscheiden, so teilen sie doch die Annahme, dass Mikrosakkaden in einer topographisch organisierten motorischen Karte für Sakkaden ausgelöst werden. Diese Karten beinhalten eine Repräsentation für klein-amplitudige Sakkaden im Zentrum der Karte (mit dem rostralen Pol der colliculi superiores als neurophysiologische Implementierung).

In der vorliegenden Arbeit kritisiere ich, dass Modelle der Mikrosakkadengenerierung ausschließlich auf Resultaten langanhaltender prä-sakkadischer Fixation beruhen. Ich führe an, dass Mikrosakkaden in einer natürlicheren Situation untersucht werden sollten, nämlich während der Fixation nach einer großen Sakkade. Die Untersuchung postsakkadischer Fixation bietet eine neue Möglichkeit Modelle der Mikrosakkadengenerierung zu falsifizieren. In den Studien zeige ich, dass Signale über den Fehler in der Sakkadenlandeposition (visuelle und extra-retinale), sowie fehler-unabhängige Signale, wie die Zielreiz-Exzentrizität, einen entscheidenden Einfluss auf kleine Sakkaden haben.

Diese Resultate erfordern Modifikationen an dem kürzlich eingeführten Modell von Rolfs, Kliegl und Engbert (2008), um die Generierung von kleinen Sakkaden auch während der postsakkadischen Fixation erklären zu können. Darüber hinaus präsentiere ich eine viel versprechende Ereigniszeitanalyse, die uns erlaubt zeitabhängige Einflüsse auf das postsakkadische Blickbewegungsverhalten zu untersuchen. Außerdem untersuche ich das Zusammenspiel von postsakkadischen Augenbewegungen und postsakkadischen Positionsurteilen. Dabei wird die Bedeutung von postsakkadischen Augenbewegungen als Kovariate in den statistischen Analysen betont.

Ein zweites Ziel dieser Arbeit besteht darin Modellvorhersagen bezüglich vorbereitender Einflüsse auf die Mikrosakkadengenerierung zu untersuchen. Die Ergebnisse, hinsichtlich eines signifikanten Einflusses des *preparatory set* auf die Mikrosakkadenrate unterstützt die wesentliche Modellannahme, dass erhöhte fixationsbezogene Aktivität zu einer größeren Anzahl an Mikrosakkaden führt.

In der vorliegenden Arbeit präsentiere ich wichtige Einflüsse auf die Generierung von kleinen Sakkaden während der Fixation. Diese Augenbewegungen stellen ein vielseitiges okulomorisches Verhalten dar, welche weiterhin zahlreiche Fragen mit sich bringen und sicherlich zukünftige Studien zu Wahrnehmung und Kognition beeinflussen werden.

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Chapter 1

General introduction

Eye movements have been extensively used to study human cognition in a variety of tasks including simple responses to upcoming targets, visual search, reading, and scene perception (Rayner, 1998, 2009).

In Figure 1.1, a subject's eye movement behavior is shown while viewing a picture of a natural scene. It becomes evident that regions of interest are fixated in order to allow visual information uptake of that region. For a certain time, the image needs to be fixed on the retina in order to allow slow responding retinal photoreceptors to receive a stable sensory input and transmit relatively unblurred visual information (Land, 2012; Walls, 1962). At some point later, the eyes move away and fixate another region of the image. Such observations of human eye movement behavior led to the simplistic view that eye movement behavior can be described by an alternating sequence of saccades and fixation. Visual processing is thought to take place during fixation and saccades bring the eyes very quickly to a new location in the visual scene.

This view of human eye movement behavior is immediately challenged by the observation that the eyes continue to move during fixation. These eye movements together are referred to as fixational eye movements and they consist of three different eye movement types, namely drift, tremor and microsaccades (for recent reviews see Martinez-Conde, Otero-Millan, & Macknik, 2013; Rolfs, 2009).

Drift refers to the slow movement of the eyes during fixation. Tremor is another type of fixational eye movement which is superimposed on the drift movement. While the amplitude of the tremor is very small, there is a high frequency which can vary considerably between subjects and different eye-tracking setups (Barlow, 1952; Rolfs, 2009).

A third type of fixational eye movement is referred to as microsaccades (Dodge, 1907; Hafed, 2011; Martinez-Conde, Otero-Millan, & Macknik, 2013; Rolfs, 2009; Steinman et al., 1973). These are fast eye movements with an amplitude that is typically smaller than one degree of visual angle (see Figure 1.2). They occur during fixation, interrupting the drift movement, with an average rate of 1-2 microsaccades per second. Both, monocular and binocular microsaccades can be observed, with the majority of microsaccadic events being binocular (Rolfs, 2009). In human subjects, microsaccades are mainly oriented in horizontal and vertical direction (Engbert, 2006).

In recent years, the similarity between large saccades and microsaccades has been stressed (for detailed review see Hafed, 2011; Rolfs, Kliegl, & Engbert, 2008). Most

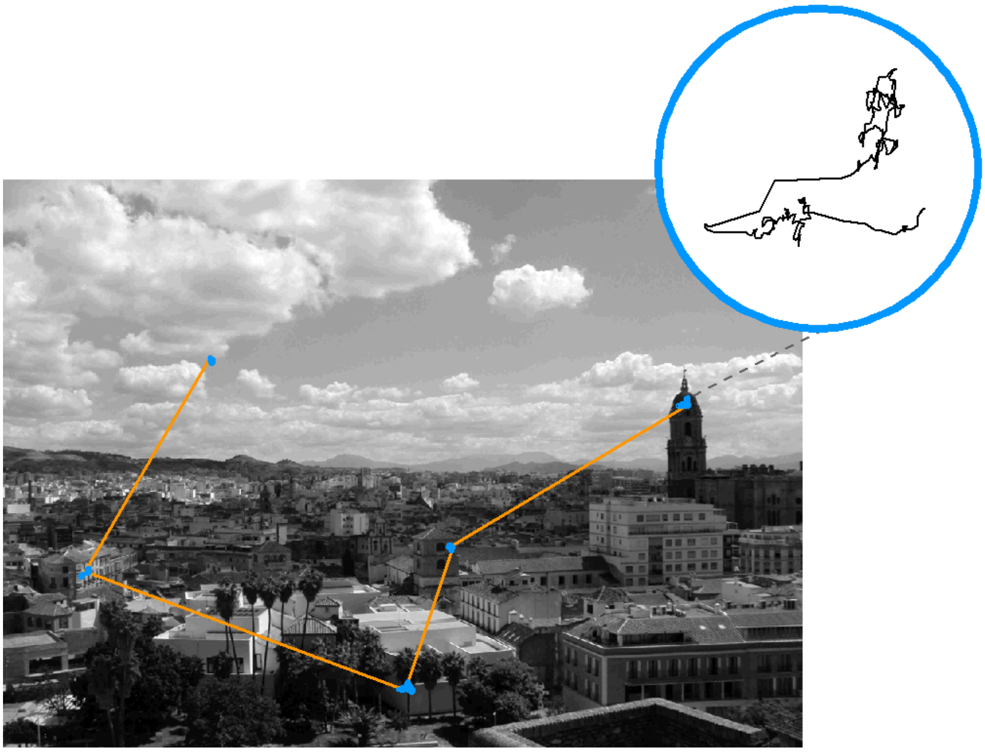

Figure 1.1: Fixation positions and saccadic eye movements during viewing of a natural scene. Periods of fixation are depicted in blue and saccades in orange. The first fixation is magnified in the upper right corner showing the eye movement trajectory during fixation.

notably, both microsaccades and large saccades fall on the main sequence (Zuber, Stark, & Cook, 1965) which describes the linear relationship between saccade amplitude and peak velocity. Alike for large-scale saccades, microsaccades change the eye position with a high velocity profile that displays characteristic acceleration and deceleration periods (Hafed, 2011). Also interesting, around both microsaccades and large-scale saccade, thresholds for visual perception are elevated (Beeler, 1967; Hafed & Krauzlis, 2010), therefore demonstrating that similarities between microsaccades and large saccades go well beyond kinematic properties. These similarities between microsaccades and large saccades suggest that both may be generated by the same oculomotor mechanisms and that both have similar functions just on a different scale.

Although saccades and fixations are often regarded as antagonistic players in human eye movement behavior, microsaccades occurring during fixation were less extensively studied than saccades and eye movements in general. Catching up in terms of more studies on fixational eye movements will certainly improve our understanding of processes underlying fixation, but also cognition in general, as microsaccades may be used as easily accessible information which has been shown to be influenced by cognitive processes (e.g., attention).

The present thesis will focus on one type of fixational eye movement, namely microsaccades. Although microsaccades have been noticed at least more than 100 years ago for the first time (Dodge, 1907), only relatively few studies were concerned with the role of microsaccades in visual perception and eye movement control. To some degree, this might be due to a missing precise definition of microsaccades which led to differences in

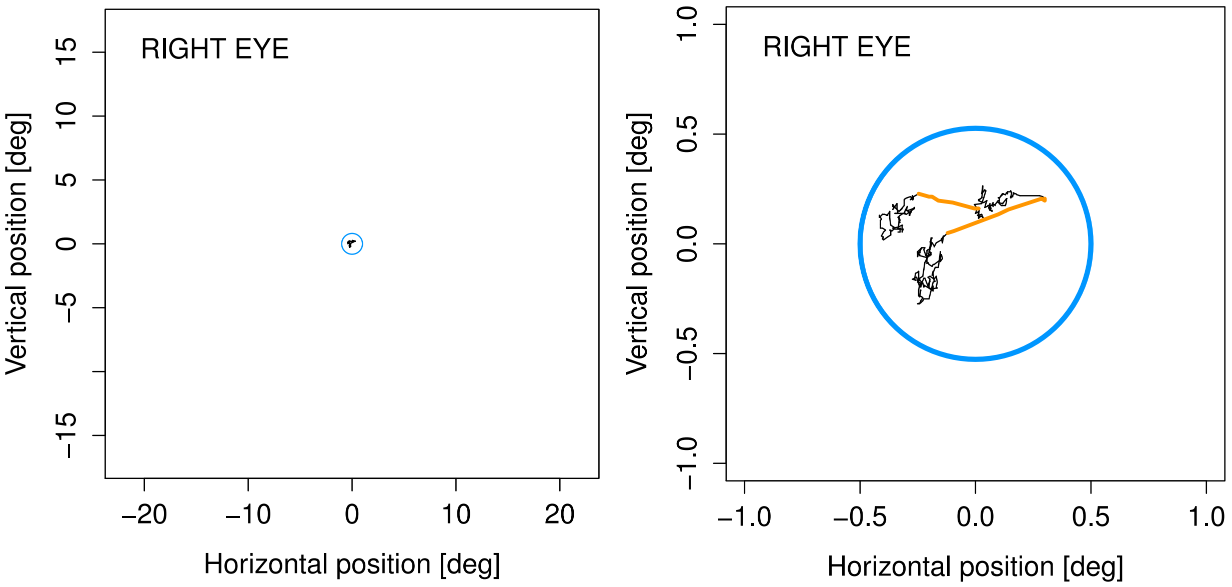

Figure 1.2: Exemplary eye movement trajectory of the right eye during fixation. **Left panel**, the border of the fixation point is displayed in blue while the eye movement trajectory is indicated in black. **Right panel,** eye movement trajectory after magnification. Microsaccades are highlighted in orange. Microsaccades were detected binocularly, although only the trajectory of the right eye is shown.

the assumed amplitude range of microsaccades. Moreover, whether microsaccades play a significant role at all has been repeatedly questioned (Collewijn & Kowler, 2008) which represented a further obstacle for research on microsaccades. This changed dramatically over the last decade with many studies examining the role of microsaccades in human sensory and cognitive processing.

In the present thesis, I will address two aspects concerning microsaccades. First, I will highlight that microsaccades do not only occur during presaccadic fixation. Indeed, studying the generation of small-amplitude eye movements like microsaccades during postsaccadic fixation represents a far more natural situation because fixations are typically preceded by a large saccadic eye movement. Nevertheless, this requires to study the classical field of secondary saccades (which are small-amplitude eye movements following large saccades). In chapter 3-6 we will use a recent framework of microsaccades in order to account for the generation of small-amplitude eye movements during postsaccadic fixation.

Second, I am going to study whether top-down preparatory processes affect microsaccades during presaccadic fixation. Preparatory influences have been widely studied in oculomotor research. Neurophysiologically motivated models of saccade generation were quite successful in predicting the effect of preparation on behavior. But the neurophysiological grounding of these models needs to be rejected after accumulated evidence which requires to revisit the influence of preparatory processes in the oculomotor system. Interestingly, a new framework allows us to derive predictions how preparatory influences affect microsaccades which will be tested in chapter 7.

In the beginning of the next chapter, two classic fields of microsaccade research will be reviewed, which deal with the influence of microsaccades for vision and the role of microsaccades for the correction of position error. Then I will proceed to the neurophysiology of the oculomotor system and recent evidence concerning the implementation of microsaccades in the oculomotor network. The neurophysiology of the oculomotor system has fundamental implications for models of eye movement behavior. Consequently, new evidence concerning the role of the superior colliculus for the generation of microsaccades had a tremendous impact on models of (micro-)saccade generation. In addition, the most important models dealing with the generation of microsaccades will be introduced. Furthermore, I will review the most prominent topic of the last decade on microsaccade research which focused on the relationship between microsaccades and attention.

Following the review I will elaborate on the goal of the present thesis which focuses first on the generation of small saccades during postsaccadic fixation, and second on the influence of top-down preparatory processes on microsaccade generation.

Chapter 2

Small-amplitude eye movements during fixation

2.1 Microsaccades and visual perception

Fixational eye movements cause a corresponding movement of an image across the retina. In order to study the role of fixational eye movements for vision several studies aimed to develop an experimental setup that allowed stabilization of a retinal image (Ditchburn & Ginsborg, 1952; Riggs & Ratliff, 1952; Yarbus, 1967) and therefore removing the effect of fixational eye movements on the retinal position of an image. Using such retinal stabilization techniques, the image will always fall on the same location of the retina and the influence of a stable retinal image on visual perception can be studied.

The results from early studies of retinal stabilization were surprisingly clear-cut in such a way that the stabilized image quickly disappeared from visual perception (Ditchburn & Ginsborg, 1952, Heckenmueller, 1965; Riggs & Ratliff, 1952; Yarbus, 1967). At this time, the demonstration of perceptual fading as a consequence of retinal stabilization constituted strong support for a fundamental role of fixational eye movements in visual perception.

In the beginning, retinal stabilization was realized by an optical system attached to a contact lens (Ditchburn & Ginsborg, 1952; see also Pritchard, 1961). This approach was accompanied by critical reflections whether the retinal image was really stable when using contact lenses because the lenses were subject to the risk of slippage (Barlow, 1963). Such critique resulted in new approaches to achieve retinal stabilization. First and foremost, the consequences of making entoptic structures visible (e.g., blood vessel, capillary) were studied (Campbell & Robson, 1961; Coppola & Purves, 1996; Drysdale, 1975; Ratliff, 1958; Sharpe, 1971, 1972). Those entoptic structures are part of the eye and consequently they form a stable retinal image. The results based on this alternative approach showed that the perceived entoptic structures fade from visual perception within only a few seconds; therefore providing additional support for the role of fixational eye movements for visual perception by means of counteracting perceptual fading.

In recent years, it has been made use of video-based eyetracking and gaze-contingent display changes in order to further examine the role of fixational eye movements under the condition of a stabilized retinal image (Poletti & Rucci, 2010; Rucci et al., 2007; Santini et al., 2007). In line with results from previous retinal stabilization experiments, video-based eyetracking with gaze-contingent display changes can demonstrate that stabilized images fade from perception. In addition, this technique has a strong advantage, namely that even unexperienced subjects can be tested with this setup (Poletti & Rucci, 2010).

Perceptual fading does not only occur under conditions of retinal stabilization. Indeed it was noted by Troxler (1804) more than 200 years ago that peripheral objects can fade from perception. In a series of experiments by Clarke (Clarke, 1957, 1960, 1961; Clarke & Belcher, 1962) it was assumed that fading after retinal stabilization and Troxler fading may have a common cause, namely neural adaptation (Martinez-Conde et al., 2006). Thus, the established finding of a fading image could be explained as a result from absent retinal image motion. Ditchburn et al. (1959) proposed that microsaccades might be very well suited to restore the image by causing a shift of the retinal image. They showed that a sudden (microsaccade-like) movement of a stabilized stimulus on the retina causes an immediate perceptual reappearance of the stimulus. Importantly, a drift-like movement of the stimulus was not effective in restoring the percept. However, these results remained controversial as other studies came to opposing results (Gerrits & Vendrick, 1970, 1974; see also below).

Martinez-Conde et al. (2006) were the first to directly examine whether microsaccades are involved in the restoration of a faded image in the Troxler illusion. The authors hypothesized that fading in the Troxler illusion should be preceded by a time interval characterized by low retinal image motion as the result of fewer microsaccades. In contrast, reappearance of the faded stimulus should be preceded by an increasing number of microsaccades which cause small shifts of the retinal image. To test their hypothesis, subjects fixated a centrally presented fixation point while viewing a peripherally located gabor stimulus. Subject's eye positions were recorded while they had to indicate the visibility (fading vs. intensifying) of the stimulus. The key finding of this study was a strong relationship between microsaccade statistics and visibility of the peripheral target. As predicted, microsaccade rate, probability and mean amplitude decreased before fading and increased before the reappearance of the peripheral target. Thus, this study provided additional support for the often suggested role of microsaccades in restoring faded images.

Troxler fading is not only observed for static objects in peripheral vision. Indeed, it has also been reported that flickering stimuli, moving stimuli and also foveal stimuli can fade during prolonged fixation (Magnussen et al., 2001; Schieting & Spillmann, 1987; Stürzel & Spillmann, 2001, see also Martinez-Conde et al., 2006).

Consequently it is straightforward to examine whether microsaccade statistics show the same behavior in various versions of the Troxler illusion as reported by Martinez-Conde and colleagues (2006). Indeed, a recent study examined the influence of microsaccade on the visibility of both foveal and peripheral targets (McCamy et al., 2012). Alike for peripheral targets, the authors observed a decrease in microsaccade rate before a fading foveal stimulus and the reverse pattern before reappearance of the visual stimulus. These results are strong evidence against the critical note that microsaccades may not be relevant for foveal vision (Collewijn & Kowler, 2008; Kowler & Collewijn, 2010; Kowler, 2011).

The usefulness of microsaccades for vision has been proposed already very early (Ditchburn, Fender, & Mayne, 1959), but from the very beginning this claim was also accompanied by authors who doubted that microsaccades serve a special role in perception (for recent critique see Collewijn & Kowler, 2008).

First, an important argument against a special role of microsaccades criticized that retinal stabilization experiments were conducted in a very artificial laboratory setting which excluded head and body movements (e.g., by using chin rest and bite board). So why should fixational eye movements in general be necessary to produce retinal image motion when other sources of movement (body or head movements) are available (Kowler & Steinman, 1980; Skavenski et al., 1979; Steinman & Collewijn, 1980; Steinman et al., 1973)?

This claim was substantiated by a study examining fixation during free head movements and artificially imposed head movements (Skavenski et al., 1979). The authors demonstrated that fixation during head-unrestrained experiments result in strong retinal image motion. Interestingly, eye movements largely compensated for artificially imposed oscillatory head movements. Thus, the remaining retinal image motion might be sufficient to counteract perceptual fading (Skavenski et al., 1979, see also Collewijn & Kowler, 2008; Steinman & Collewijn, 1980). This problem led Martinez-Conde and colleagues (2006) to study also the effect of microsaccades on the Troxler illusion during free viewing, allowing subjects to move their head. Indeed, under the condition of free head movements, it was still observed that microsaccades significantly contributed to the visibility of peripheral objects during prolonged fixation.

Second, assuming that fixational eye movements are indeed involved in restoring faded images, it is not clear whether this can be attributed solely to microsaccades, or whether drift and tremor also counteract visual fading. Fixational eye movements need to bring a visual stimulus to new retinal photoreceptors in order to overcome fading caused by retinal fatigue. Reports on the spatial extent which is produced by the different types of fixational eye movements show that both microsaccades and drift movements may generate sufficient motion of the retinal image (see Rolfs, 2009).

A clever experimental setup was introduced to study the influence of external stimulus motion on counteracting visual fading. A stimulus was stabilized on the retina, so that fixational eye movements did not result in changing retinal images. In addition, the stimulus could be moved in a controlled fashion in order to allow examination of movement characteristics on the visibility of the stabilized stimulus (Ditchburn, Fender, & Mayne, 1959; Krauskopf, 1957; for detailed review see Rolfs, 2009). As already mentioned above, Ditchburn and colleagues (1959) presented stimuli that had either drift-like, microsaccade-like or tremor-like motion while the rest of the image was stabilized with respect to the retina. The results were clear in such respect that only microsaccade-like motion caused reappearance of the faded image (see also Ditchburn, Drysdale, & Drysdale, 1977).

However, additional research on that topic produced controversial results. Gerrits and Vendrick (1970, 1974) used a retinal stabilization setup including an electric motor that enabled them to simulate movements of an object similar to the previously mentioned studies. In contrast to the results observed by Ditchburn, Fender, and Mayne (1959), Gerrits and Vendrick postulated that drift-like motion was most effective in restoring the object after visual fading. In another series of experiments (Kelly, 1979, 1981) it was shown that superimposed drift-like movements were most effective in restoring contrast sensitivity to gratings which were stabilized on the retina. These experiments highlight the possible role of drift movements for visual perception and strongly question a predominant role of microsaccades in counteracting visual fading.

It can be concluded from these controversial findings, that fixational eye movements cause a constantly changing visual input. This is indeed necessary in order to prevent visual fading. Accumulating evidence supports the view that both microsaccades and drift movements contribute to the retinal image refresh. Nevertheless, this issue is far from being solved. In a recent study by McCamy and colleagues (2012), the authors used sophisticated analyses in order to determine whether drift or microsaccades were more effective in restoring an image after visual fading. The authors concluded that microsaccades were more effective than drift (and larger microsaccades are more effective than small microsaccades) in counteracting visual fading, insisting that microsaccades play an outstanding role for visual perception.

Third, assuming that microsaccades were indeed the most effective movement counteracting retinal fatigue (McCamy et al., 2012), it does not necessarily mean that there is a causal relationship between fading and subsequent triggering of microsaccades (Cornsweet, 1956; Poletti & Rucci, 2010, but see Engbert & Mergenthaler, 2006).

Recent studies by Rucci and colleagues (Ko, Poletti, & Rucci, 2010; Kuang et al., 2012; Rucci et al., 2007) addressed additional controversial topics related to the role of fixational eye movements and vision. First, the authors were interested in the role of fixational eye movements for visual perception of fine spatial details. To this end, subjects had to judge the orientation of a tilted grating which was embedded in noise. The experiment was run under the condition of retinal stabilization and also under normal viewing. While for low frequency gratings, the percentage of correct judgments was about the same in the stabilized and unstabilized condition, performance varied strongly for high frequency stimuli. More specifically, performance was strongly improved in the unstabilized as compared to the stabilized condition. While this study, emphasizes the role of fixational eye movements on the discrimination of high spatial frequency gratings in general, it is not possible to derive any further conclusions on the specific contribution of the different types of fixational eye movements on the improved perception of fine spatial detail. Recently, this result received further substantiation by showing that fixational eye movements strongly influence the spatiotemporal input to the retina (Kuang et al., 2012). In this study natural images were presented to subjects during prolonged fixation. Using the eye movement trajectory during fixation, the authors constructed movies how the images moved across the retina. Typically, the spectral power of a static natural scene strongly decreases at high spatial frequencies. Analysis of the reconstructed movies showed that fixational eye movements caused the spectral power to be equal across a wide range of spatial frequencies; therefore accentuating the contribution of high spatial frequencies (Kagan, 2012).

Second, Ko, Poletti, and Rucci (2010) revisited the role of microsaccades in a high visual acuity task. The general hypothesis is that microsaccades may have a similar function as large saccades just on a different scale, namely guiding the eyes to informative locations in the visual scene. To test this hypothesis, subjects' eye positions were recorded while they were asked to thread a needle in a virtual environment. In this study, it was nicely demonstrated that microsaccades moved the eyes back and forth between the two most important locations in the task, the tip of the thread and the eye of the needle. This result convincingly refuted earlier findings on that topic which doubted the role of microsaccades in such high acuity task (Bridgeman & Palca, 1980; Winterson & Collewijn, 1976).

This selective review on microsaccades (and fixational eye movements in general) and vision demonstrates that early results on the possible role of fixational eye movements were often contradicted by subsequent studies. Although it can be concluded that fixational eye movements certainly are crucial for visual perception it is often very controversial whether specific functions can be solely attributed to one specific type of fixational eye movement. Another important topic is the possible role of fixational eye movements for control of the fixation position. As we will see in the following section, the scientific course of this topic shows similar characteristics as highlighted for the topic of fixational eye movements and vision in the current section.

2.2 Microsaccades and control of the fixation position

While the problem of fixation control is easily stated the corresponding debate turned out to be highly controversial, sometimes mirroring aspects of the debate on the role of microsaccades for visual perception. Ditchburn and Ginsborg (1953) noticed that in the light of constantly moving eyes during fixation, a control mechanism is necessary in order to keep the eyes on a particular point, therefore counteracting accumulating position error over time. Such a control mechanism could theoretically be realized by microsaccades and/ or drift which bring the eyes back to the intended fixation position.

A study by Cornsweet (1956) was set out to directly assess the role of microsaccades and drift for the control of fixation position. In this experiment a flickering stimulus was shown under retinal stabilization or under normal viewing condition. The author was interested to determine whether microsaccades and drift were triggered by either large fixation error, perceptual fading or even non-visual factors. Neither drift rates nor microsaccade rate were correlated with disappearance time-fractions. This result speaks against the idea that perceptual fading triggers microsaccades or drift movements. In addition, Cornsweet showed that drift rates were similar under normal viewing condition and stabilized viewing, which also speaks against a potential role of drift movements for correcting target displacement. Finally, an increasing drift interval was associated with an increase in fixation error therefore supporting the view that drift movements are not involved in the control of fixation position.

However, microsaccade rate was dependent on displacement. More specifically, microsaccades were more likely to be generated after increasing displacement. A positive correlation between the amount of displacement and microsaccade amplitude further supports the idea that microsaccades correct for fixation error. Also, in the stabilized condition fewer microsaccades were observed. Cornsweet (1956) concluded that oculomotor instability allows drift movements to increase the displacement of the retinal image.

Increasing displacement in turn increases the probability that a corrective microsaccade will bring the eyes back to the intended fixation position.

A study by Nachmias (1959) challenged the view that microsaccades are the only type of fixational eye movement which is involved in the compensation of fixation error. In this study, recording of the horizontal and vertical eye movement component allowed the author to study the direction of microsaccade occurrence. Microsaccades compensated for accumulated fixation error in some directions. Nevertheless, microsaccades were not necessarily triggered by large fixation error. Nachmias proposed that the probability of microsaccade generation increased with increasing time since the last microsaccade. Most interestingly, drift seemed to compensate for fixation error in those directions where microsaccades failed to do so, a result which was replicated in a subsequent study (Nachmias, 1961). These studies together suggest that compensation of fixation error is not a unique function of microsaccades. Slow control mechanisms (e.g., the compensation of fixation error by drift movements) have been studied in more detail (for review see Rolfs, 2009). At this point I want to emphasize that the existence of slow control mechanisms has been used to argue that microsaccade do not serve a special role in the control of the fixation position (Kowler & Steinman, 1980).

Engbert and Kliegl (2004) addressed the conflicting role of fixational eye movements for control of fixation position using a sophisticated random walk analysis. In their analysis the authors demonstrated that the trajectory of fixational eye movements deviates from Brownian motion. Importantly, eye displacement as a function of time is best described by a subdivision of fixational eye movements into a short time scale and a long time scale. Over a short duration, fixational eye movements increased displacement (e.g., larger position error). In contrast, over a long time scale fixation error was reduced. The authors pointed out that these results can reconcile that microsaccades are both error correcting and error producing (Engbert & Kliegl, 2004). Error production on a short time scale may help to counteract retinal fatigue while on a long time scale microsaccades correct for fixation position error.

The debate on the role of microsaccades in the control of fixation position and their role in vision by counteracting retinal fatigue dominated the research on microsaccades. Understanding their role and function for vision and oculomotor control distracted from the possibility that microsaccades can be used as a source of information during fixation to study all kinds of cognitive processes. Assuming that microsaccade statistics (e.g., frequency, orientation) are sensitive to experimental modulations in cognitive tasks may provide us with a powerful tool to use eyetracking for studying human cognition during prolonged fixation.

2.3 The oculomotor system

When we study eye movements and their implementation in the oculomotor system, it is also important to describe the muscles that produce the rotations of the two eyes. Each eye is under the control of three pairs of extraocular muscles. While horizontal eye movements are controlled by the lateral and medial rectus muscles, vertical and oblique eye movements are the result from combined contractions of the superior and inferior rectus muscles as well as the superior and inferior oblique muscles (Sparks, 2002).

The muscles receive their input from a burst generator circuit that is located in the reticular formation (for detailed reviews see Fuchs, Kaneko, & Scudder, 1985; Moschovakis, Scudder, & Highstein, 1996; Scudder, Kaneko, & Fuchs, 2002; Sparks, 2002). Noteworthy, neurons of the burst generator with horizontal vs. vertical orientation preferences originate in different regions within the burst generator circuit.

One important group of neurons in the burst generator consists of omnipause neurons (OPNs). These neurons have the intriguing characteristic to fire tonically during fixation and stop firing during saccadic eye movements (Sparks, 2002). Another (more heterogenous) group of neurons is referred to as burst neurons. These neurons produce a burst of activity before saccade onset. The burst of activity from excitatory burst neurons (EBNs) is tightly coupled to saccade onset as they provide the main input to motor neurons that control the extraocular muscles. Importantly, saccade amplitude, duration and velocity all depend on the burst characteristics in terms of spike number, burst duration and peak firing rate (Sparks, 2002).

The transformation of activity from long-lead burst neurons (LLBNs) into specific characteristics of the saccade is not as strong as in the case of EBNs. LLBNs also generate saccade-related bursts of activity but in addition they discharge already prior to these bursts at a low frequency (Everling et al., 1998; Keller, 1974). LLBNs project excitatory input to EBNs while fixation-related activity from OPNs inhibits firing of EBNs. This triangle between OPNs, LLBNs and EBNs constitutes the major signaling pathway within the burst generator circuitry whose output controls the extraocular muscles.

The reticular formation receives two major inputs. First, the cerebellum projects to the reticular formation. Studies examining cerebellar lesions demonstrated decreasing accuracy of saccadic eye movements (Ritchie 1976; in more detail see Scudder, Kaneko, & Fuchs, 2002). The cerebellum is strongly interconnected in the oculomotor network (e.g., receives input from superior colliculus (SC), frontal eye fields, supplementary eye fields) and plays an important role in the control of voluntary eye movements (Robinson & Fuchs, 2001).

Second, the main input to the reticular formation is sent from the SC, a central node in the oculomotor system that integrates sensory and top-down information in order to compute the goal for the upcoming saccade. Following, I will review the SC in more detail and highlight its role in the generation of small and large saccadic eye movements.

The SC is located in the roof of the brain stem and consists of seven layers (for recent reviews see Gandhi & Katnani, 2011; Isa & Hall, 2009). It is one of the most vital nodes in the oculomotor system (see Figure 2.1), mediating the transformation of sensory signals into motor commands (Sparks & Mays, 1990; Stein & Meredith, 1993). The SC contains several types of neurons which can be classified as sensory neurons, neurons that convey motor signals, and neurons that are sensitive to both, sensory and motor signals.

Neurons located in the deeper layers of the SC (stratum griseum profondum, stratum album profondum) form a topographically organized saccadic motor map (Robinson, 1972; see Figure 2.2). Activity of these neurons is elevated before and during saccade execution. Importantly, the neuronal response is maximum for a specific saccade vector, meaning that each neuron has a preferred saccade amplitude and direction (Straschill

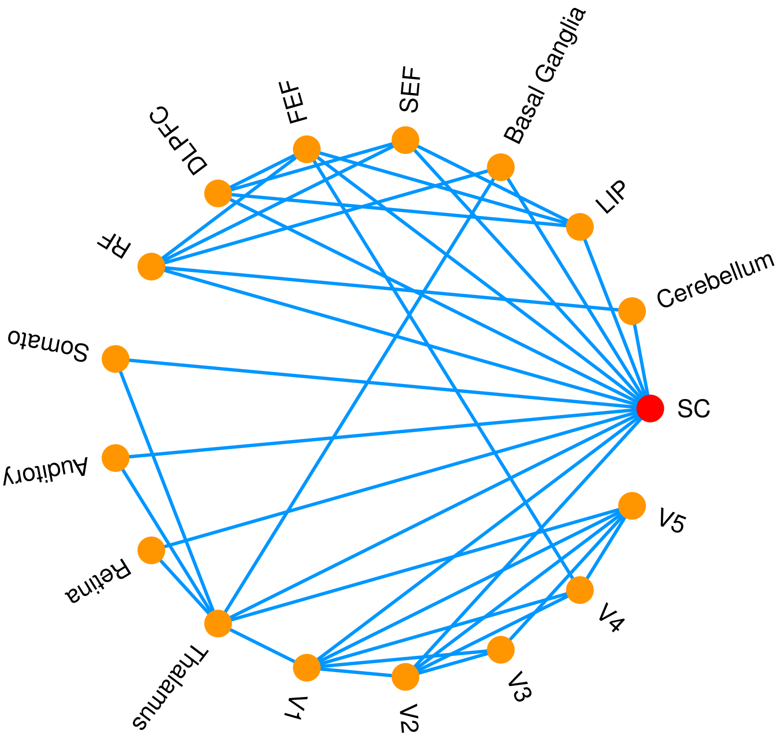

Figure 2.1: Illustration of important projections in sensory information processing including the most important nodes in the oculomotor network. The node of the superior colliculus is highlighted in red. Note, that only a selective number of projections has been displayed based on Gattas et al., 1997; Munoz & Everling, 2004; and Pierrot-Deseilligny et al., 1995.

& Hoffmann, 1970; Wurtz & Goldberg, 1972). Microstimulation of neurons in the deeper layers of the SC results in saccades with a vector of specific amplitude and direction (Robinson, 1972), therefore providing direct evidence for the role of these neurons in saccade programming. The saccadic motor map is topographically organized, meaning that neurons with preferences for similar saccade vectors are located next to each other (Sparks, Holland, & Guthrie, 1976), with an increasing saccade amplitude being located at increasingly caudal regions of the SC. Thus, saccade amplitude is continuously represented from small (rostral pole) to large amplitudes (caudal locations). Moreover, saccade amplitude is logarithmically represented within this map (Ottes, van Gisbergen, & Eggermont, 1986). Thus, a large amount of space in the deep layers of the SC is devoted to neurons with preferences for small saccade amplitudes.

In the superficial layers of the SC (stratum zonale, stratum griseum superficiale, stratum opticum) so called visuosensory neurons are located. These neurons respond to the presentation of visual stimuli in the contralateral visual field. Visual neurons of the left-hemispheric SC are firing in response to stimuli presented in the right visual hemifield and vice versa. Sensory neurons in the intermediate (stratum griseum intermedium, stratum album intermedium) and the deep layers respond to visual stimuli as well, but

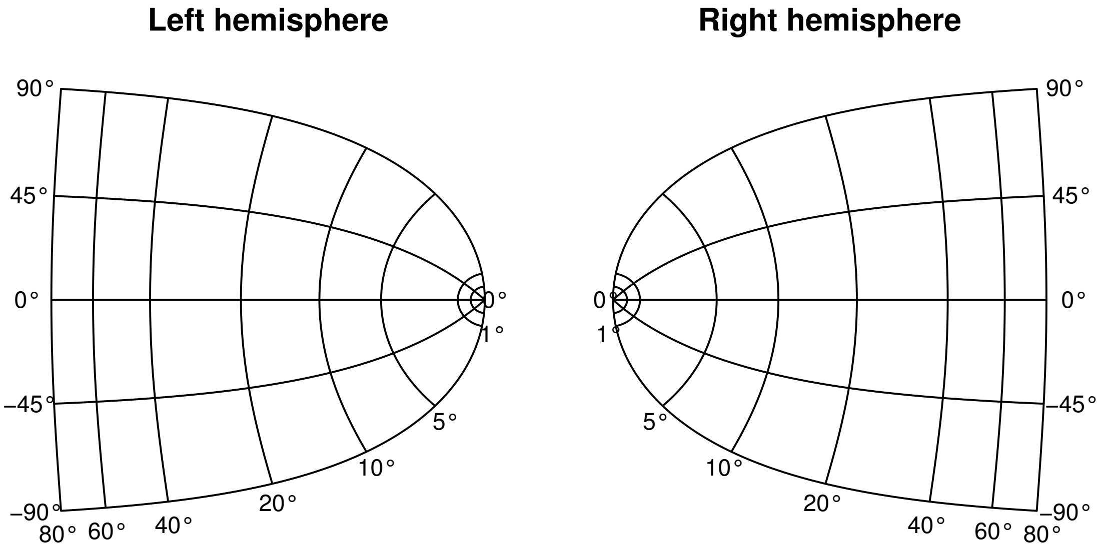

Figure 2.2: Saccadic motor map in the superior colliculus. Map for rightward saccades in the left superior colliculus and map for leftward saccades in the right superior colliculus. The maps are based on equations introduced by Ottes, van Gisbergen, & Eggermont (1986).

they are also sensitive to input from other modalities (e.g., auditory and somatosensory stimuli).

Following the work by Munoz and colleagues, it has been proposed that so called fixation neurons (FNs) form a fixation zone in the rostral pole of the SC (Munoz & Guitton, 1989, 1991; Munoz & Wurtz, 1993a, 1995). It has been observed that these neurons are tonically active during fixation but they stop (or at least reduce) firing during saccadic eye movements (Munoz & Guitton, 1991; Munoz & Wurtz, 1993a). Consequently, it has been thought that FNs can be regarded as a neural correlate of fixation. In contrast, neurons that program saccadic eye movements were referred to as saccade neurons (SNs). These neurons are typically found outside the rostral pole at caudal regions of the SC.

The antagonistic relationship between saccades and fixation was thought to be mirrored by the antagonistic activity of SNs and FNs. Inactivation of the rostral pole in the SC decreases the latency of large-scale saccades (Munoz & Wurtz, 1993b), while stimulation of the rostral pole results in the opposite effect, namely an increase in saccade latency for large-amplitude saccades (Munoz & Wurtz, 1993b). Such a reciprocal pattern of activation between FNs and SNs has been thought to be the result of lateral interactions within the SC (Isa & Hall, 2009; McIlwain, 1982; Meredith & Ramoa, 1998; Munoz & Istvan, 1998). Importantly, it has been assumed that FNs in the rostral pole of the SC project to OPNs (Büttner-Ennever et al. 1999; Gandhi & Keller, 1997; Paré & Guitton, 1994) which has been described already in the previous section.

The three basic elements that constitute the saccadic motor map, (1) SNs coding for specific saccade vectors, (2) FNs coding for fixation and (3) lateral interaction between FNs and SNs, largely determined models of saccade generation (Arai & Keller, 2005; Findlay & Walker, 1999; Kopecz & Schöner, 1995; Meeter, van der Stigchel, & Theeuwes, 2010; Trappenberg, Dorris, Munoz, & Klein, 2001; van Opstal & van Gisbergen, 1989; Wilimzig, Schneider, & Schöner, 2006). In general, models based on these three core assumptions were quite successful in predicting and explaining behavioral observations;

e.g. remote distractor effect (Walker et al., 1997), influences of distracters on saccade curvature (Sheliga, Riggio, & Rizollatti, 1994; van der Stigchel, Meeter, & Theeuwes, 2006; van der Stigchel, 2010) and the global effect in landing position (Bruell & Albee, 1955; Coëffé & O'Regan, 1987).

However, from early on several findings concerning the SC were not in line with such models of eye movement generation. First, cumulative evidence favors the representation of desired gaze position instead of saccade vector in the saccadic motor map (Bergeron & Guitton, 2000, 2002; Bergeron, Matsuo, & Guitton, 2003; Choi & Guitton, 2006, 2009). Second, the assumption of specific interactions between the fixation zone in the rostral pole and SNs in caudate regions of the SC has been criticized. Indeed, it has been shown that interactions between SNs and other SNs are not different to interactions between FNs and SNs (Basso & Wurtz, 1997; Munoz & Istvan, 1998). Third, although FNs and OPNs display similar discharge properties (e.g., both are active during fixation and stop firing during saccadic eye movements), physiological data demonstrated large discrepancies between the time-dependent discharge pattern of FNs and OPNs (Everling et al., 1998) which is strong evidence against the idea of direct projections from FNs to OPNs. Fourth, FNs in the rostral pole of the SC discharge a burst for small saccades (Gandhi & Keller, 1997; Krauzlis, Basso, & Wurtz, 1997). These results point to a continuous representation from very small to large gaze-position error in the deeper layers of the SC and speak against a model of SNs and FNs, linked by lateral interactions.

In a recent study by Hafed, Goffart and Krauzlis (2009), the authors demonstrated additional strong support for the idea that the rostral pole of the SC is involved in the generation of microsaccades which requires irrevocable rejection of the former FN assumption. The authors' evidence is twofold. First, they identified neurons in the rostral pole of the SC which were firing before and during microsaccades. Most importantly, these neurons were highly selective for microsaccade direction and amplitude (e.g., neurons were firing before and during contralateral, but not ipsilateral, microsaccades of 12 min-arc). Second, reversible inactivation of neurons in the rostral pole of the SC resulted in a reduced probability of microsaccade occurrence. Together these results demonstrate that neurons in the rostral pole of the SC are not FNs, but they are causally involved in the generation of microsaccades (Hafed, Goffart, & Krauzlis, 2009).

The conclusive results from Hafed, Krauzlis and colleagues concerning the causal role of neurons in the rostral pole of the SC for microsaccade generation represent a paradigm shift from models assuming reciprocal activation between FNs and SNs, to models assuming a saccadic motor map with a continuous representation of saccade amplitude (as initially hypothesized by Robinson, 1972) including the smallest possible eye movements in the rostral pole of the SC.

Such a paradigm shift requires revisiting the reliable behavioral effects in eye movement research that were formerly explained by the antagonistic relationship between SNs and FNs. Those findings from experiments need to be explained within the new model frame. This in turn can result in specific predictions for characteristics of microsaccades in these experiments which further allows us to improve current models of eye movement generation.

The SC is an important structure in the oculomotor network because it receives input from many sub-cortical and cortical areas. The superficial layers of the SC receive their visual signal from two distinct pathways (for an overview see Munoz & Everling, 2004). First, there is a fast and direct pathway from the retina to the SC which is referred to as the retinotectal pathway. Second, the superficial layers in the SC receive input from the visual cortex (for an overview see Munoz & Everling, 2004), which provides more detailed and fine grained visual information than the signal from the retinotectal pathway. Besides input from visual processing streams, the SC receives sensory input from auditory (in cat see Paula-Barbosa & Sousa-Pinto, 1973) and somatosensory cortex (in rat see Drager & Hubel, 1976).

Important input to the intermediate and deeper layers of the SC stems also from widespread sources (for an overview see Munoz & Everling, 2004) including the basal ganglia, the lateral intraparietal cortex (LIP; Paré & Wurtz, 2001), and frontal regions including the dorsolateral prefrontal cortex (DLPFC), the frontal eye fields (FEF), and the supplementary eye fields (SEF). The DLPFC is crucial for suppressing reflexive and producing goal-directed eye movements (Guitton, Buchtel, & Douglas, 1985; Pierrot-Deseilligny et al., 1991), e.g., as demonstrated in the anti-saccade task. Both, FEF and SEF project also directly to the burst generator in the reticular formation (Munoz & Everling, 2004). The SEF is thought to be involved in performance monitoring (Stuphorn, Taylor, & Schall, 2000), and as recently shown in metacognition (Middlebrooks & Sommer, 2012). Alike the SC, the FEF contributes to the translation from visual signals into a command of motor orientation (for reviews see Schall, 2002). Noteworthy, lesion studies demonstrated that neither a lesion of the SC, nor a lesion of the FEF alone can abolish saccadic eye movements (Schiller, True, & Conway, 1980; Sparks, 1986). Only a combined lesion in SC and FEF has the effect that saccades to visual targets cannot be generated anymore (Schiller, True, & Conway, 1980).

An important line of research addresses the question whether attention can affect signal processing in the brain. In a typical experiment that studies attention, subjects are asked to respond to a cued stimulus (and ignore distracting stimuli). In many areas of the cerebral cortex, including the visual cortex (Desimone & Duncan, 1995; Moran & Desimone, 1985) as well as parietal and frontal areas (Corbetta & Shulman, 2002), it has been shown that the neuronal response to cued stimuli is higher than the neuronal response to un-cued or distracting stimuli (Reynolds & Chelazzi, 2004). Attentional modulation of neuronal firing is not restricted to the cortex. Sub-cortical structures, including the LGN (O'Connor et al., 2002) and SC (Ignashchenkova et al., 2004; Kustov & Robinson, 1996; Zénon & Krauzlis, 2012), are also part of the attention network. Given the evidence that attention has a direct influence on activity in the SC and therefore the distribution of activity within a saccadic motor map, it is straightforward to expect that covert shifts of attention can also influence the generation of microsaccades. Indeed the influence of attention on microsaccades represents one of the most fruitful fields in microsaccade research over the last decade. In the following section I will further review findings and controversies related to the link between microsaccades and attention.

2.4 The influence of attention on microsaccades

Studies by Hafed and Clark (2002) as well as by Engbert and Kliegl (2003) were the first studies to demonstrate that the orientation of microsaccades is informative about the allocation of covert attention. This finding launched a strong interest and a controversial debate in research on microsaccades. In the beginning of this section the central findings of Engbert and Kliegl (2003) will be reviewed and afterwards I will proceed to the current view on the link between microsaccades and shifts of covert attention.

Engbert and Kliegl (2003) made use of the classic attentional cueing paradigm which has been originally introduced by Posner (1980). Endogenous cues (e.g., arrows, colors) were centrally presented, indicating in 80% of the trials the correct position of the subsequently presented target (valid trials). Subjects were asked to move their eyes immediately to the new upcoming target location. Two important findings concerning the characteristics of microsaccades were reported in this study.

First, microsaccade rate followed a characteristic pattern. During fixation before cue onset about one microsaccade per second was observed. Following cue onset (about 100 ms after cue presentation), microsaccade rate dropped, reaching its minimum at about 150 ms after cue onset. This initial inhibitory phase was then followed by an increase of microsaccade frequency, resulting in strong microsaccadic enhancement at about 350 ms after cue onset. Afterwards, microsaccade rate decreased again and remained at a baseline rate of one microsaccade per second.

Second, microsaccade orientation turned out to be significantly influenced by the spatial allocation of covert attention. The average direction of microsaccades during the enhancement phase (350 ms after cue onset) was clearly shifted into the direction of the cued location. It has been concluded by the authors that microsaccade orientation offers a promising tool *to map the orientation of covert visual attention* (Engbert & Kliegl, 2003).

In a subsequent study by Laubrock, Engbert and Kliegl (2005) the authors examined the influence of covert attention both after endogenous color and exogenous flash cues on microsaccade statistics in a paradigm that allowed decoupling the mechanisms of response preparation and the shift of attention (Müller & Rabbitt, 1989). Again, cue- and time-dependent orientation effects were observed. Endogenous cues resulted in a small cue-incongruent orientation effect of microsaccades during a late time interval. In contrast, shortly after the presentation of exogenous flash cues, microsaccade orientation was strongly biased into the direction of the cued location. This early cue-congruent orientation effect was followed by a cue-incongruent effect in an interval between 250 and 600 ms after cue onset. In a very late interval between 600 and 800 ms after cue onset, microsaccades were again biased into the direction of the cued location. In summary, this study nicely demonstrates that microsaccade orientation is strongly influenced by the allocation of covert attention. Moreover, this influence follows a complex pattern which strongly depends on the type of cue used in the experiment.

Nevertheless, there is also evidence against the idea that eye movements during fixation are influenced by a shift of attention (Tse, Sheinberg, & Logothetis, 2002, 2004). One possible explanation for the null finding was given by Rolfs and colleagues (2004) arguing that the large number of display changes during these experiments (Tse, Sheinberg, & Logothetis, 2002, 2003) might have resulted in strong microsaccadic inhibition and therefore a too small number of microsaccades in order to demonstrate attention specific effects in microsaccade orientation.

Horowitz and colleagues (2007) examined whether fixational eye movements are causing attentional shifts, employing the same paradigm as reported by Engbert and Kliegl (2003). The rationale behind their analysis was that behavior (response times) should be better predicted by the orientation of microsaccades than by the cue itself, given the assumption that microsaccade orientation is an almost perfect index of covert attention. Horowitz and colleagues did not find any evidence for such a causal relationship of microsaccades and attention which led them to state that *fixational eye movements are not an index of covert attention* (Horowitz et al., 2007). In a commentary on this study, a strong correlation between spatial cue and microsaccade orientation was demonstrated (Laubrock et al., 2007). Moreover, the authors have shown that microsaccade orientation can account to some degree for the variance in reaction times after statistical control of the spatial cueing influence. Although microsaccades clearly are not the cause for an shift of attention, they still provide useful information about the allocation of covert attention (Laubrock et al., 2010).

Subsequent studies further elaborated the relationship between covert attention and microsaccades, providing by now a detailed and complex picture on the link between covert attention and microsaccades. Further evidence points to the bias of microsaccades to be oriented away from the exogenous cue (Betta, Galfano, & Turatto, 2007; Gowen et al., 2007; Turatto et al., 2007) and also a bias of cue-congruent microsaccades after endogenous cues (Gowen et al., 2007; Hermens & Walker, 2010).

An interesting approach was chosen by Pastukhov and Braun (2010) who hypothesized a relationship between the attentional demand and the number of microsaccades. In this study, the authors were able to show that the number of microsaccades decreased with increasing attentional load. At the same time the few observed microsaccades during high attentional demand were extremely informative about the location of spatial attention (Pastukhov & Braun, 2010).

So far, effects of attention on microsaccades have been well documented in human subjects. However, the implementation of microsaccades in the oculomotor system was mainly studied in monkeys. Thus, evidence from these lines of research in humans and monkeys do not necessarily translate one-to-one across species. Recently, Hafed, Lovejoy and Krauzlis (2011) bridged this gap and studied the effects of attention on microsaccade rate and orientation in monkeys.

In their task, two monkeys had to indicate the orientation of a briefly presented coherent motion pulse which was presented in one of four quadrants. Motion pulse onset was preceded by a cue that highlighted the relevant quadrant. Alike for humans, microsaccades in monkeys were strongly influenced by the allocation of attention. Microsaccades were oriented to task relevant locations, but the time-dependent orientation effects differed strongly between the two monkeys. Moreover, the orientation of microsaccades was correlated with performance in the visual task. In successful trial, microsaccades were more likely to be oriented in the direction of the attended quadrant than to any other quadrant. In unsuccessful trials, when the monkeys reported the orientation of the motion pulse in the opposite quadrant, fewer microsaccades were oriented to the cued than the opposite quadrant. These results show, alike for human subjects, that microsaccades are strongly correlated with the allocation of attention and the performance in a visual attention task.

In the following section I will further review the microsaccade signature after stimulus onset which has been initially observed by Engbert and Kliegl (2003). As already mentioned this microsaccade signature can be described by an early inhibitory phase starting around 100 ms after stimulus onset, followed by an enhancement phase and a late return to the baseline rate.

Importantly, Rolfs et al. (2005) showed that this microsaccade signature can also be evoked by presentation of auditory cues; therefore multisensory processing can influence microsaccade generation. Interestingly, Galfano, Betta and Turatto (2004) demonstrated that the presentation of uninformative exogenous, visual cues also results in the typical microsaccade signature. Together, these studies imply that a simple change of the stimulus configuration, possibly irrespective of stimulus modality (visual or auditory), can produce a microsaccade signature that is characterized by a sequence of inhibition, enhancement and return to baseline. Indeed, further experiments demonstrated the importance of stimulus characteristics (color contrast, luminance contrast) for the inhibitory phase of the microsaccade signature (Rolfs, Kliegl, & Engbert, 2008). Since its first observation, the microsaccade signature has been reported repeatedly by different authors and across many experimental paradigms showing that it is a basic response of the oculomotor system to the onset of a sensory event.

Engbert (2006) hypothesized that the evolution of the microsaccade signature in response to a sensory change can be explained by intrinsic processes within the SC. As already mentioned above, sensory neurons in the deeper layers of the SC respond to multisensory stimuli. Engbert (2006) assumed that incoming sensory information would increase the mean-field activation in the saccadic motor map, having the consequence that neurons in the rostral pole of SC will be inhibited stronger due to the inhibitory connections within the saccadic motor map; thus resulting in a strong decrease of microsaccade frequency. The inhibitory phase is very reliable while the subsequent enhancement phase is much more variable across studies. Engbert (2006) suggested that the early inhibitory phase is very likely to be caused by sensory information coming directly from the retina via the retinotectal pathway.

The subsequent enhancement phase in contrast is then thought to be the result of dynamic processes within the saccadic motor map (Engbert 2006, 2012). Furthermore, the late enhancement phase is thought to be modulated by higher cognitive processes, which is also reflected in the putative modulatory influence of FEF, DLPFC and BG. In an oddball task it has been observed that the enhancement phase can be even absent (Valsecchi & Turatto, 2007, 2009; Valsecchi et al., 2007; 2009), demonstrating again the variability of the enhancement phase across studies and experimental paradigms.

In this section, two important findings associated with microsaccades were reviewed. First, a complex time-dependent response of cue-congruent and cue-incongruent microsaccades after presentation of a spatial cue is observed. Second, in response to display changes the microsaccade rate shows a typical inhibitory phase which is followed by a more variable enhancement phase. Both findings need to be explained by models of microsaccade generation and therefore provide also a good test for models that will be reviewed in the next chapter.

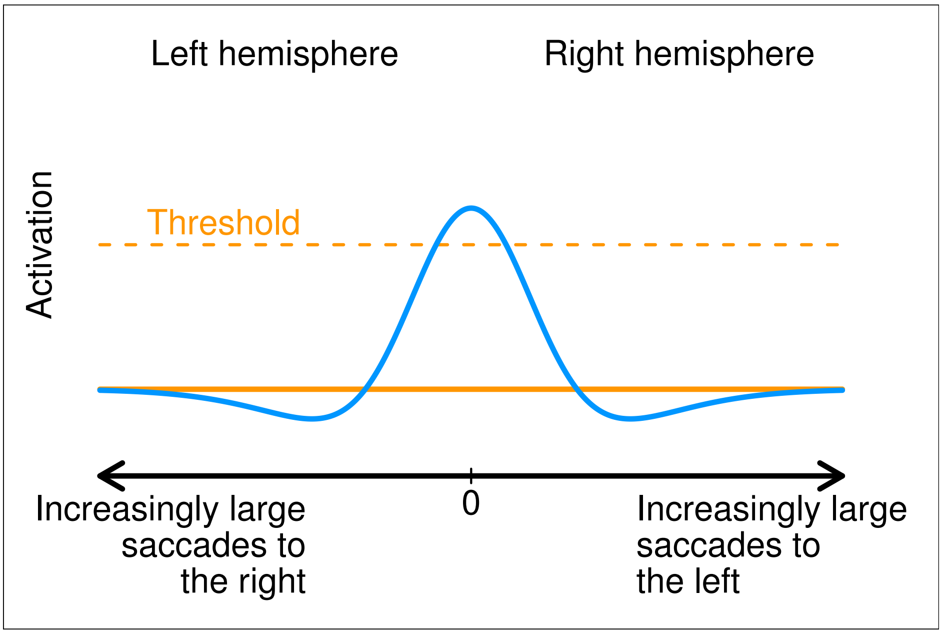

Figure 2.3: Model illustration adopted from Rolfs, Kliegl and Engbert, 2008. Microsaccades result from activation crossing the threshold at the center of the map. Rightward saccades are programmed in the left hemisphere and leftward saccades in the right hemisphere of the map.

2.5 Models of microsaccade generation

At first I present a qualitative model introduced by Rolfs, Kliegl and Engbert (2008) which assumes that microsaccades and large saccades are the outcome of activity surpassing a critical threshold in a motor map. In this motor map microsaccades are the result of activity above threshold in the center of the map (see Figure 2.3). Increasingly large saccades to the left are programmed at increasingly distant locations in the right hemisphere of the map while rightward saccades are programmed at an eccentricity-dependent site in the left hemisphere.

In addition to this topographical organization, the authors proposed that interactions within the motor map follow the principle of local excitation and global inhibition (Rolfs, Kliegl, & Engbert, 2008). This implies that neighboring sites in the motor map excite each other, while two locations separated by a large distance are reciprocally active, therefore inhibiting each other. The distribution of activity in the motor map is influenced by either inherent noise in the map, or from signals outside the motor map, namely endogenous or exogenous inputs.

In this model, large fixation related-activity in the center of the map results in a high microsaccade rate, while a smaller microsaccade rate is the result of decreasing fixation-related activity. This assumption is interesting because the consequence which follows from this assumption states that microsaccades can be used as read-out of fixation-related activity. Microsaccade direction is thought to be the result of activity arising in the motor map which shifts the overall distribution away from the center of the map. The authors argue that several sources can theoretically cause this shift of activation (e.g., neural noise, the goal to reduce fixation error, interacting influences from other locations in the motor map, see Rolfs, Kliegl, & Engbert, 2008).

With these basic assumptions the model can provide an explanation for the signature of microsaccadic inhibition (see chapter 2.4). Stimulus onset is thought to result in reduced fixation-related activation, therefore a decreasing microsaccade rate in response to stimulus onset is observed. At the same time activation increases at the site of the eccentric visual stimulus. Furthermore, the model correctly predicts that microsaccadic inhibition affects microsaccades with large amplitudes first; therefore the remaining microsaccades during the phase of microsaccadic inhibition are on average microsaccades of smaller amplitude (Rolfs, Kliegl, & Engbert, 2008).

Due to the assumption of global inhibitory interactions within the saccadic motor map, the model further predicts strong interactions between saccade and microsaccade generation. Large fixation-related activity results in a higher microsaccade rate. At the same time a strong fixation-related activation in the center will inhibit more distant locations in the map, where large eye movements are programmed. Thus, the model predicts longer saccade latencies when microsaccades occur in close temporal distance to a stimulus that instructs a subject to move the eyes to a target. Indeed, such an influence of microsaccades on subsequent saccade latency has been demonstrated (Rolfs, Laubrock, & Kliegl, 2006). In another study, the same authors confirmed an additional prediction from the model, namely that microsaccade-induced prolongation of subsequent saccade latency is largest for larger microsaccades (Rolfs, Laubrock, & Kliegl, 2008).

Alike the model by Rolfs and colleagues, Hafed, Goffart and Krauzlis (2009) emphasize the role of the rostral pole in the superior colliculus for microsaccade generation. In their computational model, goal locations are encoded in the superior colliculus, with foveal goal locations being represented in the rostral pole of the superior colliculus. Again, a one-dimensional map is assumed with the center representing foveal goal locations and activity being distributed around zero during steady-state fixation (Hafed, Goffart, & Krauzlis, 2009).

An important difference to the qualitative model proposed by Rolfs, Kliegl and Engbert concerns the mechanism of saccade triggering. Here, Hafed, Goffart and Krauzlis propose that microsaccades result when the *center of mass deviates sufficiently from zero*. Model simulation demonstrated that this model can account for the result of reduced microsaccade frequency after inactivation of neurons in one hemisphere of the superior colliculus (in close vicinity of the rostral pole).

Their basic assumption of microsaccades as the result of an imbalance in rostral activation in the superior colliculus can also be used to provide an explanation for the influence of attention on microsaccade orientation. Attention was modeled as increasing activation at a more caudal location in their motor map. This attentional modulation resulted in a shift of the overall activity distribution towards the attended stimuli with the consequence that activity had a higher probability to deviate significantly from zero, therefore triggering a microsaccade in the direction of the attended location.

Both models presented so far are very similar with respect to the central role of rostral pole activity in the superior colliculus for microsaccade generation. Also, both models assume that microsaccades are triggered after crossing a threshold. Recently, a mechanism for microsaccade generation has been proposed that is implemented in the neural circuitry between the SC and the burst generator (Otero-Millan et al., 2011). The proposed mechanism has the advantage that it does not need the assumption of a triggering signal that is send after threshold crossing. Otero-Millan and colleagues (2011) propose that microsaccades are the result of dynamic interactions between LLBNs and OPNs which receive input from the SC. The LLBNs and OPNs are connected with the SC. The strength of the signals from SC to LLBNs and OPNs depends on the specific location in the SC. The rostral pole projects strong input to OPNs and weaker input to LLBNs. In contrast, more caudal regions excite LLBNs more strongly than OPNs. During fixation, activation is increased in the rostral pole of the SC. Consequently, OPNs are active which inhibit LLBNs and BNs. When SC activation shifts away from the rostral pole, OPN-firing is reduced and activity of LLBNs increases again. At some point LLBN-firing is strong enough to overcome inhibition from OPNs and therefore can drive BNs which trigger the microsaccade. Thus, the authors provide a simple mechanism in the oculomotor system that can produce microsaccades without the assumption of a hypothetical threshold for microsaccade triggering.

Finally I would like to introduce a model proposed by Engbert and colleagues (see Figure 2.4) which is based on the concept of a self-avoiding random walk (Freund & Grassberger, 1992) in a potential (Engbert et al., 2011). Experiments have shown that the trajectory of fixational eye movements can be characterized by persistent behavior in a short time window and antipersistent behavior in a large time window (Engbert & Kliegl, 2004; Mergenthaler & Engbert, 2007). Thus on a short timescale, fixational eye movements have the tendency to continue their current movement direction (persistent behavior) and on a long timescale the tendency to move into the opposite direction (antipersistent behavior). The idea of fixational eye movements showing persistent and antipersistent behavior depending on the timescale fits nicely with the two assumed functions of trying to avoid retinal adaptation and the control of fixation position. Self-avoiding random walk can produce such a pattern of persistent and antipersistent behavior, therefore providing a potential framework for modeling of fixational eye movements.

In this model, a square lattice is assumed and a walker (representing the current eye position) moves within this lattice from site to site. At each point in time the walker moves to the neighboring site with lowest activation. Activation of the currently visited site increases while all other sites slowly decrease their activation back to a baseline level. The strong increase in activation at the current position of the walker causes the walker to avoid recently visited locations. A potential consequence of such a mechanism is that the distance between walker and center of the lattice strongly increases. Therefore, a quadratic potential is added which constraints the walker to move within a defined small area. In this model microsaccades are triggered by suprathreshold activation at the four neighboring sites surrounding the current position of the walker. Thus, if the walker visits a site with activation over the defined threshold, a microsaccade is executed to the site in the lattice with lowest activation (defined as sum of walker-generated activation and quadratic potential).

The model is able to produce an exponential distribution of inter-microsaccadic intervals (Engbert et al., 2011) which has been suggested by experimental data. Also, it has been shown in experiments that microsaccades and drift interact in such a way that slower drift is observed before microsaccades (Engbert & Mergenthaler, 2006). Indeed, the model captures also this behavior and even further predicts that larger areas covered

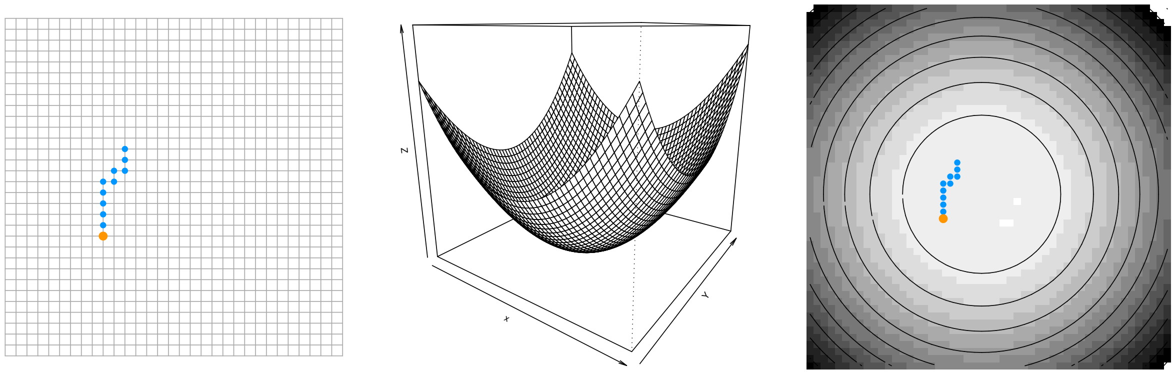

Figure 2.4: A computational model for fixational eye movements introduced by Engbert and colleagues, 2011, 2012. **Left panel**, walker on a lattice representing eye position during fixation. Current eye position is depicted in blue. Last ten eye positions are depicted in orange. **Middle panel**, quadratic potential which is added to the self generated pattern of activation. The quadratic potential constraints the maximum distance from the center which the walker can move to. **Right panel**, sum of self generated activation and quadratic potential in a heatmap. Low values are indicated in white, and high values in black. the walker's current (in orange) and recently visited sites (in blue) are added.

by drift will produce smaller microsaccades. Again the model prediction was supported by a re-analysis of experimental data (Engbert et al., 2011).

The model has been recently extended in order to capture the signature of microsaccade rate in response to cue onset and also attention-specific modulations of microsaccade orientation (Engbert, 2012). The steepness of the quadratic potential is lowered in response to stimulus onset and returns to baseline following a time-varying function. Moreover, the attentional cues cause an asymmetric modulation of the quadratic potential. Finally, both signals (perceptual and attentional) together determine the threshold for microsaccade triggering. In this model, modulations of microsaccade rate (initial microsaccadic inhibition and subsequent microsaccade overshoot) are the result of changes in the slope of the quadratic potential. The lowered slope results in a decrease while the lowered threshold for microsaccade triggering causes an increasing microsaccade rate. Orientation effects in this model are the result of asymmetric deformations of the potential. Based on the specific values of the parameters, the model is able to capture complex patterns of microsaccade rate and time-dependent cue-congruent and incongruent orientation effects of microsaccades in an attention cueing paradigm.

The introduction of models that account for various characteristics of microsaccades constitutes a major step forward in our understanding of their implementation but also for our understanding of the oculomotor system in general. Consequently, models that focus only on the generating mechanisms of large saccades neglect a rich and complex oculomotor behavior. Models of microsaccade generation and fixational eye movements in general just started to evolve and will certainly generate new testable predictions in the future. The models presented so far strongly differ in their degree of complexity and the specificity of the derived predictions. As already mentioned, the first model by Rolfs, Kliegl and Engbert (2008) is a qualitative model which allowed to examine rather general principles but also generated a number of testable predictions. Although this model represents a very helpful framework, for the future it is of course desirable to use computational models that allow quantitative comparison of experimental data and model simulations.

The model by Hafed, Goffart and Krauzlis (2009) is computational and provides a possible mechanisms how microsaccades might result from activation in the superior colliculus. So far, the recently introduced dynamic computational model by Engbert (Engbert et al., 2011, 2012) constitutes the most successful approach which allows to derive precise predictions for microsaccades, drift and the influence of attentional cues on fixational eye movement statistics. In the present thesis, I will focus on the initially introduced model by Rolfs, Kliegl and Engbert (2008). Nevertheless, combining experimental data and predictions from recent computational models will very likely constitute a fruitful approach for future studies.

2.6 Secondary saccades

So far, I reviewed the significance of small eye movements during fixation for vision, the control of fixation position, attention and their implementation in models and the oculomotor system. In the present thesis I want to emphasize that the important findings and conclusions are based on results obtained under a very artificial condition, namely prolonged presaccadic fixation. Obviously, a fixation is typically preceded by a saccadic eye movement. Studying postsaccadic fixational eye movements consequently represents a far more natural condition in which to examine the importance and the generating mechanisms of fixational eye movements, and in our case more specifically of microsaccades.

From very early on it was noted that large primary saccades are followed by small eye movements which were referred to as secondary saccades (Dodge, 1903). Interestingly, secondary saccades have rarely been discussed in terms of microsaccades. An explanation of this phenomenon might be given by the fact that microsaccades were initially defined as saccadic eye movements with an amplitude smaller than 12 min-arc. In contrast, secondary saccades are more variable in the criterion of amplitude. Nevertheless, a high percentage of secondary saccades were often in the amplitude range of the classically defined microsaccades. Studying the literature of secondary saccades and microsaccades under the perspective that both may be the result of the same generating machinery promises synergistic effects with respect to the principles underlying the programming of small eye movements in general.

The scientific story of secondary saccades begins with the advent of eye tracking devices and the question how precise saccadic eye movements can be. In his pioneering work, Dodge (1903) noted that saccades to targets at an eccentricity of 40°displayed a systematic undershoot, therefore providing evidence that the target distance has been underestimated. A second movement was necessary to align eye and target location which was consequently referred to as secondary corrective movement. A systematic study of the relationship between the error in saccade landing position and target eccentricity demonstrated that the percentage of target undershoot increased with increasing target eccentricity (from 15°to 90°; see Hyde, 1959).

A widespread belief is that saccades tend to undershoot the target by 10% of the initial distance (Becker, 1972; Becker & Fuchs, 1969; Henson, 1978, 1979; Prablanc, Massé, & Echallier, 1978). It has been observed that target displacement during saccade flight, in order to correct for the anticipated saccadic undershoot, results in quick adaptation in such a way that primary saccades continue to undershoot the displaced target. This finding has been regarded as evidence for the idea that undershooting may be the preferred response mode of the oculomotor system.

The large majority of normally occurring saccades have amplitudes smaller than 10°(Bahill, Adler, & Stark, 1975). Despite this observation, the above listed studies on the accuracy of primary saccades focused on saccades to very distant targets. In subsequent years the accuracy of primary saccades to closer targets has been examined in more detail and many opposing results have been reported with respect to the landing position of these saccades. Henson (1979) stated that even primary saccades to close targets often undershoot the target. This finding was challenged by the range effect (Kapoula, 1985; Kapoula & Robinson, 1986), which assumes saccadic overshoot of close targets and saccadic undershoot of distant targets. In contrast to both concepts, Kowler and Blaser (1995) emphasized that saccades to close targets tend to be very precise in general.

Studies that focused on primary saccade landing position often examined secondary saccades, too. Those studies highlighted the corrective function of secondary saccades as they often tend to reduce the large oculomotor error remaining at the end of the primary saccade (Becker, 1972; Becker & Fuchs, 1969).

A classical finding concerning secondary saccades is the inverse relationship of secondary saccade amplitude and latency. Larger secondary saccades are associated with shorter secondary saccade latency. This finding supports the view that error-correcting factors influence secondary saccade programming in order to allow fast alignment of eye and target position. Another influential observation of these early studies is that the latency of secondary saccades was remarkably shorter than primary saccade latency (i.e., mean latency of 127 ms for secondary saccades). These rather short latencies led to first suggestions that other than visual feedback signals alone may be involved in the generation of secondary saccades (Becker & Fuchs, 1969; Weber & Daroff 1972). Indeed the role of extra-retinal signals for secondary saccade programs constitutes an important topic related to secondary saccades. In the present thesis it will also be tested whether extra-retinal signals, in form of an error-signal, influence the generation of secondary saccades.

A first study on the topic of non-visual signals and secondary saccades was conducted by Becker and Fuchs (1969). In their experiment, subjects had to generate a saccade to a target at an eccentricity of 40°which was extinguished prior to saccade onset. The authors observed secondary saccades despite the lack of a visual signal. Interestingly, the saccades to this very distant target in the dark were very precise, and subsequent secondary saccades were exclusively in the same direction as the primary saccade. The observation of secondary saccades despite the lack of visual information led to the conclusion that visual signals are not necessary to generate secondary saccades. In a subsequent study, the same experiment was conducted, but using smaller eccentricities including 10°, 20°and 30°of visual angle (Weber & Daroff, 1972). The observation of corrective secondary saccades in this experiment led Weber and Daroff to conclude that non-visual signals contribute to the programming of secondary saccades, suggesting a putative role of proprioception as important internal signal.

In contrast to these two early studies, Prablanc and Jeannerod (1975) did not find any secondary saccades when a postsaccadic visual target was omitted. Restoration of the postsaccadic visual stimulus for even a very short duration was able to elicit corrective secondary saccades. Based on these findings it was concluded that the incoming postsaccadic visual information plays a crucial role for the triggering of secondary saccades (Prablanc & Jeannerod, 1975). This finding was again challenged by a study showing in two subjects that under both conditions, with and without postsaccadic visual feedback, corrective secondary saccades can be produced (Shebilske, 1976).

In an additional study, again the role of an extra-retinal error signal for the triggering of secondary saccades has been examined. This time the latency was varied when the target has been removed during saccade flight (Prablanc, Massé, & Echallier, 1978). The authors observed secondary saccades following very large saccadic undershoot, but almost no secondary saccades after precise primary saccades. These results were obtained when the target was removed at target onset. Moreover, the few secondary saccades after large saccadic error were not very precise. The authors concluded that the extra-retinal error signal may not be very strong. Furthermore, it may be only involved in the programming of secondary saccades after very large saccadic error. In contrast, when the target was removed in the deceleration phase of the primary saccade, many corrective secondary saccades were observed (Prablanc, Massé, & Echallier, 1978). Thus, visual reafferent signals entering before the end of saccade seem to be used for programming of secondary saccades.

It can be seen that the debate on whether retinal and/ or extra-retinal signals trigger secondary saccades determined the debate on these small postsaccadic eye movements. Two further observations are important to mention in this context.

First, the latency of secondary saccades depends on the direction of secondary saccades (Deubel, Wolf, & Hauske, 1982; Henson, 1978). Secondary saccades in the same direction as the primary saccade have shorter latencies than secondary saccades into the opposite direction of the primary saccade (Deubel, Wolf, & Hauske, 1982). This observation is in agreement with an earlier explanation by Robinson (1973) who argued that keeping the target after the saccade in the same cortical hemisphere as before the saccade could result in processing benefits. This also constitutes a potential explanation why primary saccades often undershoot a target. Nevertheless, this finding also highlights that computation of a secondary saccade vector depends also on non-error-correcting influences.

Second, in a recent study it has been examined whether available visual background information influences programming of secondary saccades (Morel, Deneve, & Baraduc, 2011). In this experiment, target information and visual background information were manipulated while subjects performed a double-step saccade task. Noteworthy, for the main saccades visual information and background information was optimally integrated in order to generate an accurate sequence of the two saccades. Secondary saccades, in contrast, relied only on visual target information alone while background information was discarded (Morel, Deneve, & Baraduc, 2011).

What can we gain from the above reviewed studies? First, similar to studies on microsaccades also secondary saccades are discussed in terms of foveating a target.

Nevertheless, the signals that contribute to the programming of secondary saccades were strongly disputed. While the importance of postsaccadic visual feedback for the generation of secondary saccades has been repeatedly confirmed, there is a less coherent picture on the role of extra-retinal error signals. The often conflicting results suffer from several drawbacks of these early studies. Results concerning secondary saccades were often rather anecdotal and experiments included only very few subjects.

Secondary saccades have often been of interest under the perspective of error correction. Consequently, experimental tasks with very large eccentricities were chosen in which large saccadic errors could be observed. Obviously, it is important to study secondary saccades in their natural environment, meaning secondary saccades after primary saccades of normal length. Moreover, early studies did not treat saccadic error as a continuous variable. They often divided the accuracy of primary saccades into hypometric, normometric, and hypermetric saccades, without further clarification of the exact criterion that determines a normometric saccade. Thus, overall the above mentioned studies turn out to be very difficult to compare.

Microsaccades are small saccadic eye movements during fixation. Secondary saccades are small saccades during postsaccadic fixation (which can nevertheless be larger than the classical microsaccades during presaccadic fixation). Both are thought to be involved in the control of the fixation position (i.e., error reduction). Still, both types of eye movements are treated as separate topics in the eye movement literature although many secondary saccades meet the amplitude-criterion of microsaccades. Importantly, it has been stated that microsaccades may be a laboratory artefact, as they may not play a significant role in non-fixation tasks like free-viewing or visual search (Collwijn & Kowler, 2008). In a recent review it has been proposed to consider small saccadic eye movements during fixation between two regular saccades as microsaccades (Martinez-Conde, Otero-Millan, & Macknick, 2013). This suggestion is very one-sided, as it neglects the classical research field of secondary saccades by incorporating small secondary saccades into the increasing field of microsaccade research without even mentioning the field of secondary saccades.

In the present thesis I am interested to study the generation of small saccades during postsaccadic fixation. It will be proposed that small postsaccadic eye movements need to be explained by models that can also account for microsaccades during presaccadic fixation. More specifically, I examine the influence of visual and extra-retinal signals on a saccade motor map which produces small amplitude eye movements in the center of a two-hemispheric motor map (see chapter 2.5).

2.7 Preparatory influences on microsaccades

Preparatory processes related to eye movement behavior can take many forms. Temporal expectation can speed up saccadic responses to upcoming targets irrespective of target location. Also, spatial preparation can decrease saccadic reaction times to targets presented at a predictable location in the visual field. Indeed, preparatory processes vary strongly between various tasks (e.g. pro-saccade/ anti-saccade task).

Preparatory processes in eye movement tasks have been explained by excitation and inhibition of neuron populations in a saccadic motor map. While a decrease in saccadic reaction times was thought to be mediated by excitation of SNs and a simultaneous decrease of FNs, successful cancelation/ suppression of reflexive saccadic responses was thought to be accomplished by the reverse pattern of neural activation, namely increased firing of FNs and decreased firing of SNs.

I already reviewed the oculomotor system and its underlying neurophysiology, stating that models of saccade generation that build upon the idea of FNs have to be rejected (see chapter 2.3). Based on the recent insight that neurons in the rostral pole of the SC are coding for microsaccades, the implementation of preparatory processes in a saccadic motor map needs to be revisited. A new explanation is based on the idea that the amount of FN firing can be translated into fixation-related activation. This activation competes with saccade-related activation. Fixation-related activation should directly impact the number and characteristics of microsaccades (Rolfs, Kliegl, & Engbert, 2008), with increasing fixation-related activation resulting in more microsaccades.

So far, only a few studies addressed a possible influence of preparatory processes on the generation of microsaccades. Temporal expectations concerning the presentation of the target can influence response preparation. It has been shown that temporal expectation can improve the detection and discrimination of a stimulus (Nobre, Correa, & Coull, 2007). Betta and Turatto (2006) examined whether the number of microsaccades can be influenced by response preparation in a target discrimination task. The authors compared the microsaccade rate before stimulus onset in conditions with and without required manual response. They observed a significantly smaller microsaccade rate in trials with required manual response. This result was further substantiated by the observation that a reduced microsaccade rate was particularly present in trials with fast responses. In trials with slow responses, microsaccade rate was indistinguishable from the no-response condition. These results suggest that preparatory processes affect the generation of microsaccades.

It is also observed in tasks with saccadic responses that microsaccade rate decreases when time approaches the expected presentation of a target stimulus (Rolfs, Laubrock, & Kliegl, 2006), therefore demonstrating again the influence of preparatory processes on the frequency of microsaccades. Note that the link of increasing preparation and a decreasing microsaccade rate is predicted by a qualitative model of microsaccade generation (Rolfs, Kliegl, & Engbert, 2008). Response preparation results in decreasing fixation-related activity and consequently decreasing inhibition of saccade-related sites in the map. Given that spatial predictions are available, activation can build up at saccade-related sites in the saccadic motor map, therefore allowing faster saccadic responses. It has been hypothesized that microsaccades occurring in close temporal vicinity to the presentation of a go-signal should prolong saccadic reaction times because microsaccades are thought to be a correlate of strong fixation-related activity. Indeed this hypothesis has been confirmed for saccades to memorized and visually-guided saccades (Rolfs, Laubrock, & Kliegl, 2006).

An interesting side note of a decreasing number of microsaccades before a go signal concerns the suppression of visual signals around microsaccade onset (Beeler, 1967; Hafed & Krauzlis, 2010). A smaller number of microsaccades results in a reduced risk of visual suppression and therefore a stronger signal being available which in turn may allow faster processing and responding (Hafed, Lovejoy, & Krauzlis, 2011). Microsaccade-induced visual suppression constitutes also an important interpretation of prolonged saccadic reaction times following microsaccades. This explanation is in conflict with an explanation of a motor account that assumes inhibitory connections between fixation- and saccade-related sites within a saccadic motor map as driving source for such behavior (Rolfs & Ohl, 2011). This topic will be addressed in more detail in chapter 7.

Moreover, I will focus on the influence of the preparatory set on microsaccade statistics. The preparatory set refers to preparatory processes before a specific type of response (e.g., pro- vs. antisaccade) or the readiness to respond in general (Hebb, 1972; Evarts, Shinoda, & Wise, 1986). Importantly, these preparatory processes can begin long time before the presentation of a target, although the exact details of the response (e.g., amplitude, direction, type of response) are not clear at the given point in time (Hermens, Zanker, & Walker, 2010). Evidence for a significant influence of the preparatory set on human information processing is manifold including single-cell studies in monkey FEF and SC (Everling et al., 1999, Everling & Munoz, 2000).

Recently, Hermens, Zanker and Walker (2010) examined the influence of the preparatory set on microsaccade rate. They compared microsaccade rate in pro- vs. anti-saccade trials in different experiments manipulating the cue (endogenous vs. exogenous) and timing of response (delayed vs. immediate). In this study, microsaccade rate was higher in pro- as compared to anti-saccade trials when a delayed response to a peripheral target onset was required. This result is a first hint, that microsaccades are influenced by a preparatory set. However, in a condition with an immediate response saccade and endogenous cue presentation, no significant differences between pro- and anti-saccades were observed.

These results clearly need further support from future studies as the observed effect is rather week and opposite to the expected direction. Anti-saccade trials are thought to result in increased fixation-related activation. Consequently, the authors hypothesized a higher number of microsaccades in anti- than pro-saccades which turned out to be in the opposite direction. Nevertheless, this study provides a first hint on preparatory influences on the generation of microsaccades.

To sum up, research of preparatory processes on the generation of microsaccades is rare and needs more examination. Whether endogenously and exogenously triggered saccades share the same generating mechanisms has been subject to many studies. In this thesis, I will examine microsaccades in a preparatory interval and test whether microsaccade frequency differs before endogenously vs. exogenously triggered saccades. Indeed, such preparatory influences on the microsaccade rate is predicted from a model that assumes a link between fixation-related activity and the number of microsaccades (Rolfs, Kliegl, & Engbert, 2008), therefore providing an important test to falsify and further develop models of microsaccade generation.

Chapter 3

Secondary (micro-)saccades: The influence of primary saccade end point and target eccentricity on the process of postsaccadic fixation

3.1 Introduction

For optimal processing of our visual environment it is essential to bring areas of interest onto the fovea. This is achieved with rapid shifts of the eyes, so called saccades, which bring the eyes quickly to a new part of the visual scene. Visual information is then processed during fixation following the saccadic eye movement. Consequently, human eye movement behavior is often described as a sequence of alternating periods of saccades and fixation. This ignores at least two additional characteristics of eye movement behavior. First, a saccade towards a target in a visual scene often ends at some distance from the target position. Therefore, primary saccades are frequently followed by secondary saccades bringing the eyes to a position closer to the target location. Second, during the period of fixation the eyes are not motionless but instead show different specific movements which are referred to as fixational eye movements. One type of fixational eye movement which shares similar properties with large saccades are so called microsaccades (Hafed, 2011; Kowler & Steinman, 1980; Martinez-Conde et al., 2009; Rolfs, 2009; Steinman et al., 1973). So far microsaccades have mainly been studied during the initial period of fixation in an experimental trial. This experimental approach lacks insight into a more natural process of fixation which is the fixation following a saccade. Here we report that early (micro-)saccades following saccade execution are strongly influenced by target eccentricity and the magnitude of the saccadic error. Further, we provide new evidence that target under- and overshoot imply different consequences for the programming of secondary (micro-)saccades.

Microsaccades are defined by their amplitude (typically less than 1 degree of visual angle) and their high velocity. Similar to normal saccades, microsaccades fall on the main

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sequence (Zuber, Stark, & Cook, 1965) which describes the linear relationship of peak velocity and saccade amplitude. For a number of reasons microsaccades have moved into the focus of eye movement research (see Martinez-Conde et al., 2009; Rolfs, 2009): Microsaccades are a possible index of covert attention (Engbert & Kliegl, 2003; Laubrock, Engbert, & Kliegl, 2005), they may play an important role in counteracting visual fading (Engbert & Kliegl, 2004; Martinez-Conde et al., 2006); they afford new perspectives on the dynamics governing saccadic motor programs (Hafed & Krauzlis, 2010; Rolfs, Laubrock, & Kliegl, 2006; Rolfs & Ohl, 2011).

Despite recent advances in identifying neurophysiological mechanisms underlying the generation and consequences of microsaccades there is a strong need to further explore how microsaccades are embedded in the oculomotor system (Hafed, 2011). Recent models addressing the implementation of microsaccades highlighted the significance of the superior colliculus, a key brain structure for saccade programming (Hafed, Goffart, & Krauzlis, 2009; Rolfs, Kliegl, & Engbert, 2008). Examining secondary (micro-)saccades takes into account that a fixation is preceded by a saccade and therefore allows to examine potential influences related to primary saccade execution on eye movement behavior during postsaccadic fixation. Indeed, identifying the factors that contribute to the overall distribution of postsaccadic activity in the oculomotor system will further improve our understanding of microsaccade generation.

In the current study we determine how (1) the magnitude of the saccadic error, (2) under- or overshoot of the target by the primary saccade, and (3) target eccentricity influence the latency, amplitude and orientation of secondary (micro-)saccades. In the remainder of this study we will refer to the first (micro-)saccade occurring after a goal-directed saccade as secondary saccade. The factors which influence the fixational process conditional on a previous saccade have received surprisingly little attention in the literature, although they may constitute a crucial source to account for the variance of fixation durations in various tasks studying eye movement behavior. In the present study subjects were instructed to respond immediately with a saccade to an upcoming target and hold fixation after execution of the saccade until the end of a trial. Targets appeared to the left or right of the fixation point at two possible eccentricities resulting in four possible target locations.

3.1.1 Saccadic error

Saccades towards a target do not exactly land on the target. Indeed, there is a general tendency to undershoot a target by 10% of the target distance (Becker & Fuchs, 1969; Henson, 1978). Nevertheless, the overall accuracy of saccades strongly depends on the experimental procedure (Lemij & Collewijn, 1989). Saccades that miss the target position are usually followed by a secondary saccade bringing the target closer to the center of the fovea thereby reducing initial saccadic error (Becker & Fuchs, 1969; Becker, 1972; Hallett, 1978; Prablanc & Jeannerod, 1975; Weber & Daroff, 1972). These secondary saccades can occur shortly after the end of the primary saccade and it is argued that such error correction must rely to some degree on an internal signal (e.g., efference copy) as visual feedback is not available so quickly. The latency of secondary saccades with respect to their error-correcting amplitude is shorter for larger amplitudes of secondary saccades

(Becker, 1972; Prablanc & Jeanerod, 1975). Kapoula and Robinson (1986) also reported such a negative relationship between amplitude and latency for secondary saccades, including also eye movements with amplitudes well below one degree of visual angle; hence in the range of microsaccades. Therefore it is reasonable to expect that early secondary saccades (including microsaccades and large saccades) in our experiment will also be influenced by the magnitude of the saccadic error.

3.1.2 Under-/overshoot

Goal-directed saccades can either undershoot or overshoot a target. Hypometric and hypermetric saccades may trigger different processes. It is found that secondary saccades in the same direction as the primary saccade are on average faster than secondary saccades in opposite direction to the primary saccade (Deubel, Wolf, & Hauske, 1982; Henson, 1978). Following a functional explanation first put forward by Robinson (1973), programming of secondary saccades is facilitated after undershooting as the undershot target object is kept in the same hemisphere of the cortex. Nevertheless, longer latencies in correcting overshoots could be also due to the in general smaller error for over- compared to undershoots (Henson, 1978; Kapoula & Robinson, 1986). Considering the relationship of amplitude and latency for corrective saccades (Becker, 1972; Kapoula & Robinson, 1986; Weber & Daroff, 1972), hence longer latencies for overshoots could be explained by the smaller amplitude of the subsequent corrective saccades. Given the conflicting results of previous research, the question whether undershooting and overshooting imply different consequences for postsaccadic processing needs further clarification. In the present study we provide new insights into this issue by determining the functional relationship between the magnitude of the saccadic under-/overshoot and the dependent variables latency, amplitude and orientation of secondary saccade.

3.1.3 Target eccentricity

The aim of our study is to examine the influence of primary saccades on small eye movements during postsaccadic fixation. In natural vision, fixations are preceded by saccades of different amplitudes. Recently, Wang et al. (2011) introduced a dynamic neural field model of the superior colliculus that explores the effect of primary saccades on the latency of subsequent saccades. Simulations predict a strong influence of primary saccade amplitude on the postsaccadic neuronal activity within their dynamic neural field model of the superior colliculus. Assuming that primary saccade amplitude significantly modulates the distribution of postsaccadic activity in the superior colliculus we would also expect a significant influence of target eccentricity on the characteristics of secondary saccades in our study.

Increasing target eccentricity is typically associated with an increasing number of secondary saccades (Frost & Pöppel, 1976; Lemij & Collewijn, 1989). Lemij and Collewijn (1989) also examined the latency of secondary saccades with respect to target eccentricity and found shorter secondary saccade latencies for larger target eccentricities which they thought to be independent of the magnitude of the saccadic error. Here we test the influence of target eccentricity on secondary saccade latency (amplitude and orientation) after statistical control of the magnitude of the saccadic error. In summary, the goal of the present study is to investigate how the process of postsaccadic fixation depends on the previously executed primary saccade. Therefore we examine the influence of (1) saccadic error, (2) under-/overshoot, and (3) target eccentricity on the latency, amplitude and orientation of secondary saccades.

3.2 Materials and methods

3.2.1 Participants

Ten undergraduate students from the University of Potsdam were paid seven Euros or received study credit for their participation. They were 19-28 years old (*M* = 23.4) and had normal or corrected-to-normal vision. A session involved 300 test and 8 training trials and lasted for approximately 45 minutes. This experiment was performed in accordance with the declaration of Helsinki (1964). Written informed consent was obtained from all participants.

3.2.2 Experimental setup and eye movement recordings

Participants were seated in a silent and dark room with the head positioned on a chin rest, 50 cm in front of the computer screen. Eye movements were recorded with the Eyelink-II system (SR Research, Osgoode, Ontario, Canada) with a high spatial resolution of less than 0.01°and a sampling rate of 500 Hz. Stimuli were displayed on a 19-in. EYE-Q 650 CRT monitor at a resolution of 1024 x 768 and a refresh rate of 100 Hz. The experiment was controlled by an Apple Power Macintosh G4 computer. The experimental software controlling stimulus display and response collection was implemented in Matlab (Mathworks, Natick, MA, USA) using the Psychophysics (Brainard 1997, Pelli 1997) and Eyelink (Cornelissen, Peters, & Palmer, 2002) toolboxes.

3.2.3 Procedure

A nine-point calibration was conducted to align eye and screen coordinate systems. At the beginning of a trial subjects were fixating a white point (0.67°diameter of visual angle) on a grey background at the center of the computer screen. After a uniform random interval of 1 to 1.5 seconds, the fixation point was removed and a white circle target (0.67 diameter of visual angle) appeared in the periphery at one of four possible positions (6°to the left or right of the fixation point, 14°to the left or right of the fixation point, respectively). Each of the four target locations occurred with the same probability in a randomized order. Subjects were instructed to move their eyes as quickly as possible to the target and keep their eyes on the target until the end of a trial. A trial was terminated 1500 ms after target presentation. After an inter-trial interval of 500 ms the next trial started. Before the first and every 30 trials, the eye tracker was calibrated and the calibration was validated. Before a new trial started, fixation was checked and the stimuli were only presented when the gaze of the subjects was in the specified fixation area. A drift correction was carried out when fixation check failed. If fixation check failed after drift correction a new calibration and validation was initialized.

3.2.4 Data preparation

Trials with eye blinks and saccadic reaction times (SRT) of primary saccades faster than 80 ms or longer than 400 ms were discarded. Primary saccades had to end within a distance of 2.5°around the center of the target in order to be included for further analysis. (Micro-)saccades were determined using an improved version (Engbert & Mergenthaler, 2006) of the algorithm reported by Engbert and Kliegl (2003). (Micro-)saccades were detected in 2D velocity space using thresholds for peak velocity (6 SD) and a minimum duration of 8 ms (4 data samples). Similar to Mergenthaler and Engbert (2010) we defined a 30 ms interval as minimum duration which separated two (micro-)saccades from each other. For further analysis we considered the first secondary (micro-)saccade occurring in an interval of 350 ms after the end of the primary saccade. Subjects contributed 137-251 (M = 178) secondary saccades, resulting in a total of 1778 secondary saccades within this interval. For analysis of secondary saccade orientation we assigned each postsaccadic eye movement to one of four categories according to their angular orientation. Secondary saccades with an angular orientation between 45°and 135°(upward) or between $-45°$and $-135°$(downward) were removed for analysis of secondary saccade orientation. Leftward and rightward secondary saccades were classified as either following the direction of the primary saccade or as in opposite direction to the primary saccade. Analysis of secondary saccade orientation comprises 1611 of the initial 1778 (91%) secondary saccades.

3.2.4.1 Data analysis

For statistical analyses of secondary saccade latency and amplitude we use linear mixed models (LMM) with subjects as random factor. Effects with a t-value larger than ± 2 are considered as significant (i.e. there is a strong correspondence between the t-statistic and the z-statistic given that the number of subjects and observations per subject is sufficiently large). We carefully checked that reported significant results are also significant (95% confidence interval) when drawing samples (n = 1000) from the posterior distribution of parameters of the given LMM. Secondary saccade orientation is analyzed as bivariate dependent variable (coding: 0 = same direction as primary saccade; 1 = opposite direction to primary saccade). Therefore we use a generalized linear mixed model (GLMM) for binomial data, again including subjects as random factor. Functions for LMMs (and GLMMs) are provided by the lme4 package (Bates & Maechler, 2010) in the R environment (R Development Core Team, 2010). Graphics were obtained with the ggplot2 package (Wickham, 2009).

3.2.4.2 Covariates

The following variables are used to setup our statistical models. To quantify the magnitude of the saccadic error we determined the absolute horizontal distance from the end point of the primary saccade to the center of the target in degree of visual angle. This magnitude of the saccadic error is coded contingent on whether the primary saccade under- or

Table 3.1: Mean (SD) are shown for saccadic reaction times (SRT), saccadic error (accuracy) and the percentage of secondary saccades. Negative accuracy indicates undershooting.

	Close target	Distant target
SRT (in ms)	159.9 (16.3)	168.6 (16.2)
Accuracy (in °)	0.198 (0.26)	−0.177 (0.36)
Secondary saccades (in %)	55 (19)	82.7 (8)

overshot the target. Specifically, the covariate undershoot contains the absolute size of the saccadic error in case of undershoot and zero for overshoots. Respectively, the covariate overshoot contains the size of the saccadic error when the target is overshot and zero for undershoots. Further we use the square for both variables (undershoot2, overshoot2). Target eccentricity (eccentricity) is coded as 0 (close target at 6°) and 1 (distant target at 14°).

3.3 Results

In Table 3.1 the overall characteristics (saccadic reaction time, accuracy) of the primary saccades and the proportion of secondary saccades depending on target eccentricity are shown. In general, primary saccades are very precise in our study. Interestingly, the number of secondary saccades is much higher after saccades to distant as opposed to close targets.

In Figure 3.1 the distribution for latency (left panel) and amplitude (right panel) of secondary saccades occurring within the first 350 ms is displayed. The peak of the latency distribution is reached 135 ms after the end of the primary saccade. The distribution of secondary saccade amplitude shows a large proportion of secondary saccades with an amplitude smaller than one degree of visual angle, hence meeting the criterion for microsaccades. In Figure 3.2 the distribution of saccadic error (for the 1778 events which are followed by a secondary saccade) is shown for close (left panel) and distant (right panel) targets. Note, we observe under- and overshoots for both target eccentricities.

3.3.1 Secondary saccade latency

Figure 3.3 shows two important results. First, secondary saccade latency is longer for close (red solid line) than distant (blue solid line) targets. This difference decreases with the magnitude of the saccadic error. Second, secondary saccade latency is longer for small than large saccadic error. The peak of secondary saccade latency is shifted to small overshoot with an even further shift in the distant target condition.

The LMM specified in order to model secondary saccade latency includes the covariates undershoot, overshoot, target eccentricity as well as the interactions undershoot x eccentricity and overshoot x eccentricity. Furthermore, we include the square of undershoot (undershoot2) and the square of overshoot (overshoot2) along with their interactions with target eccentricity (see Table 3.2). We find a significant main effect of target eccentricity ($t = -7.52$). Thus in the case of minimal saccadic error we still observe a significant decrease of secondary saccade latency in the distant target condition. Moreover, the linear

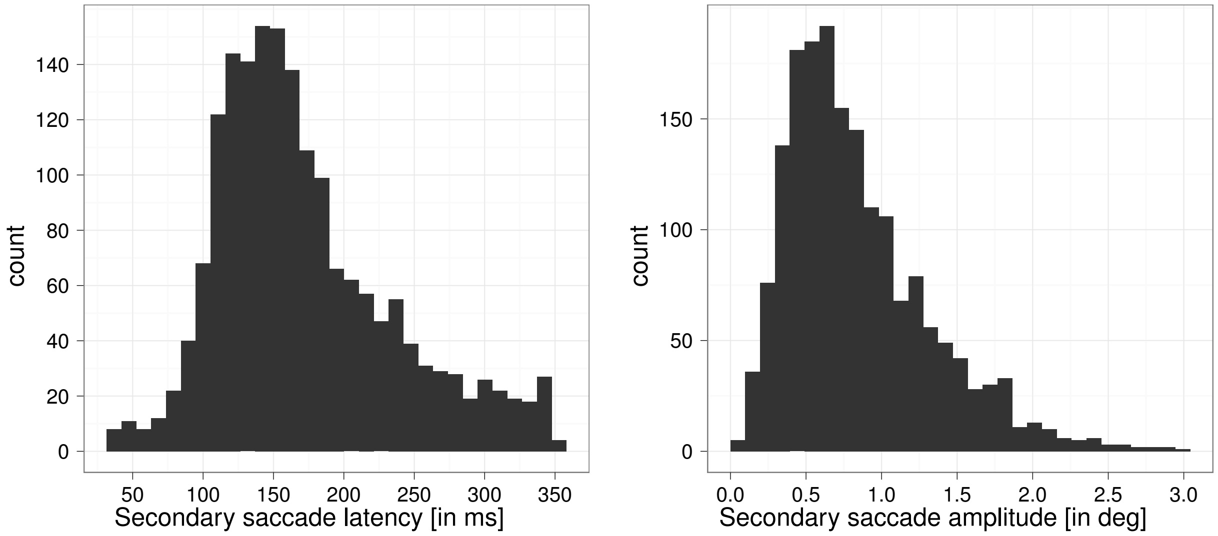

Figure 3.1: Distribution of secondary saccade latency (upper panel, binwidth = 10 ms) and secondary saccade amplitude (lower panel, binwidth = 0.1°).

and quadratic saccadic error is necessary to account for secondary saccade latency after under- and overshooting a close target. The prediction of the LMM (Figure 3.3, dashed lines) is in line with the notion of shorter latencies for a large saccadic error. The only significant interaction is overshoot2 x eccentricity (t = 2.04); meaning that overshoot2 is the only covariate associated with saccadic error which is modulated by target eccentricity.

3.3.2 Secondary saccade amplitude

Figure 3.4 illustrates the relationship of secondary saccade amplitude and saccadic error depending on target eccentricity. Four important characteristics are evident. First, increasing undershoot is associated with increasing secondary saccade amplitude. Second, irrespective of the size of saccadic undershoot amplitudes are larger in the distant target condition. Third, the minimum of secondary saccade amplitudes is shifted towards a small overshoot. Alike for latencies, the shift is more pronounced in the distant target condition. Fourth, for both target eccentricities a further increase in saccadic overshoot is associated with increasing amplitudes.

In order to model secondary saccade amplitude we use the same main factors and interactions as described in the LMM to account for secondary saccade latency (see Table 3.3). Again we find a significant main effect of target eccentricity (t = 5.84). Thus, even in the case of minimal saccadic error, secondary saccade amplitude is significantly larger in the distant than in the close target condition. Furthermore, we find a significant main effect of undershoot (t = 5.83). Importantly, neither quadratic undershoot (undershoot2; t = 1.2) nor the interactions undershoot x eccentricity ($t = -0.61$) and undershoot2 x eccentricity (t = 0.91) significantly influence secondary saccade amplitude. These results confirm our observations from Figure 3.4 and we can conclude that increasing saccadic undershoot is associated with increasing secondary saccade amplitude. Secondary saccade amplitude is significantly larger after undershooting distant than close targets as

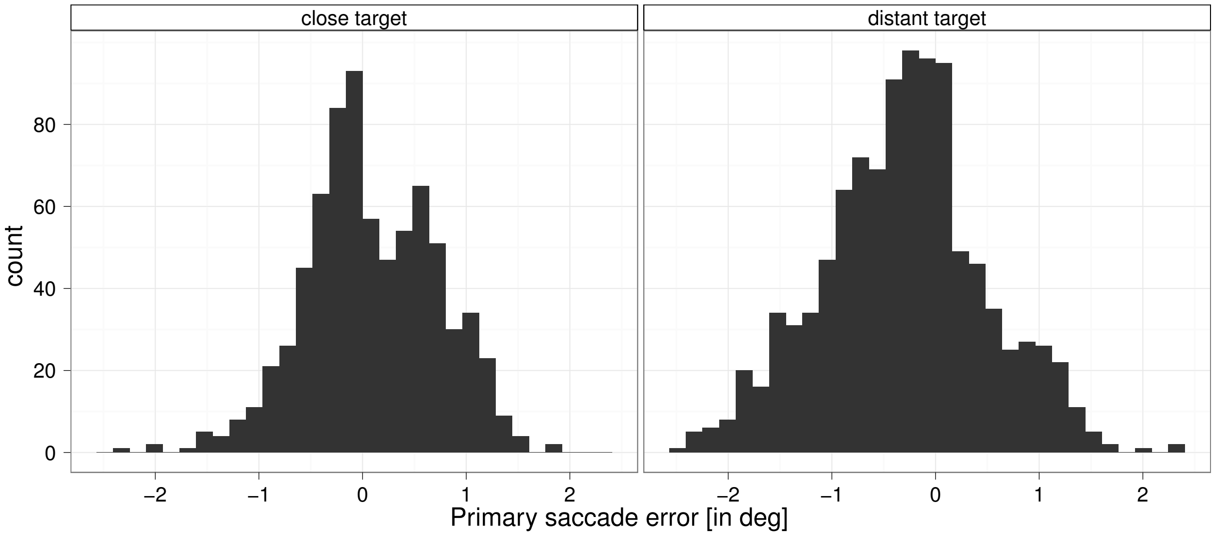

Figure 3.2: Distribution of saccadic error (binwidth = 0.15°) by target eccentricity. Negative values indicate undershooting, positive values indicate overshooting.

the significant main effects of undershoot and eccentricity are not further influenced by a significant interaction.

The pattern is more complex when studying the consequences of overshooting. Both, the linear (t = -2.19) and quadratic (t = 3.39) magnitude of saccadic overshoot significantly influence secondary saccade amplitude. Further, the effect of overshoot is modulated by target eccentricity ($t = -2.47$). These effects lead to the prediction of the LMM (Figure 3.4, dashed lines) that larger overshoot is associated with increasing amplitudes. An amplitude modulation by target eccentricity is not present anymore.

3.3.3 Secondary saccade orientation

In Figure 3.5, we show whether the orientation of secondary saccades follows the same (dots scattered around 0) or opposite (dots scattered around 1) direction of the primary saccade depending on the size of the saccadic error and target eccentricity. Moreover, the prediction of the GLMM is included for both target eccentricities (solid lines). Note, the GLMM models the probability that secondary saccade direction is opposite to the primary saccade's direction. Importantly, when a target is undershot almost all secondary saccades are in the same direction as the primary saccade. In contrast, after an overshoot secondary saccades in both directions are observed. With increasing overshoot secondary saccades are more likely to be executed in opposite direction to the primary saccade. This effect is modulated by target eccentricity. In the distant target condition an even larger saccadic overshoot is necessary in order to be followed by secondary saccades to the opposite direction.

The GLMM includes the same main factors and interactions like the above introduced LMMs but spares main effects and interactions including a quadratic term (see Table 3.4). The significant negative influence of the intercept ($p < 0.001$) tells us that secondary saccades are significantly more often executed into the direction of the primary saccade

Table 3.2: LMM statistics for secondary saccade latency.

	Estimate	SE	*t*-Value
Intercept	222.8	5.8	38.50
Eccentricity (ecc)	−42.1	5.6	−7.52
Undershoot	−132.6	16.0	−8.31
Undershoot2	43.4	10.5	4.15
Overshoot	55.6	16.6	3.35
Overshoot2	−81.1	13.0	−6.26
ecc x undershoot	31.6	18.7	1.69
ecc x undershoot2	−9.5	11.7	−0.81
ecc x overshoot	10.3	21.2	0.48
ecc x overshoot2	32.3	15.9	2.04
Variance components		SD	
Subjects		12.02	
Residuals		51.28	

Table 3.3: LMM statistics for secondary saccade amplitude.

	Estimate	SE	*t*-Value
Intercept	0.549	0.055	10.00
Eccentricity (ecc)	0.177	0.030	5.83
Undershoot	0.513	0.087	5.93
Undershoot2	0.068	0.057	1.20
Overshoot	−0.197	0.090	−2.19
Overshoot2	0.239	0.070	3.39
ecc x undershoot	−0.062	0.101	−0.61
ecc x undershoot2	0.058	0.063	0.91
ecc x overshoot	−0.285	0.115	−2.47
ecc x overshoot2	0.147	0.086	1.71
Variance components		SD	
Subjects		0.157	
Residuals		0.278	

when landing in the center of a close target. While undershooting ($p < 0.001$) significantly increases the probability to follow primary saccade direction, overshooting ($p < 0.001$) significantly increases the probability to go into the opposite direction of the primary saccade. Again, we find a significant main effect of target eccentricity ($p < 0.001$). At minimal saccadic error, significantly more secondary saccades at the distant than close target location follow primary saccade's direction.

3.3.4 Control analyses

In control analyses we carefully ruled out the possibility that the observed results are due to expectation drift during the initial period of fixation.

Following a reviewer's suggestion we checked for the possibility that the relationship between saccadic error and secondary saccade latency may arise from a correlation

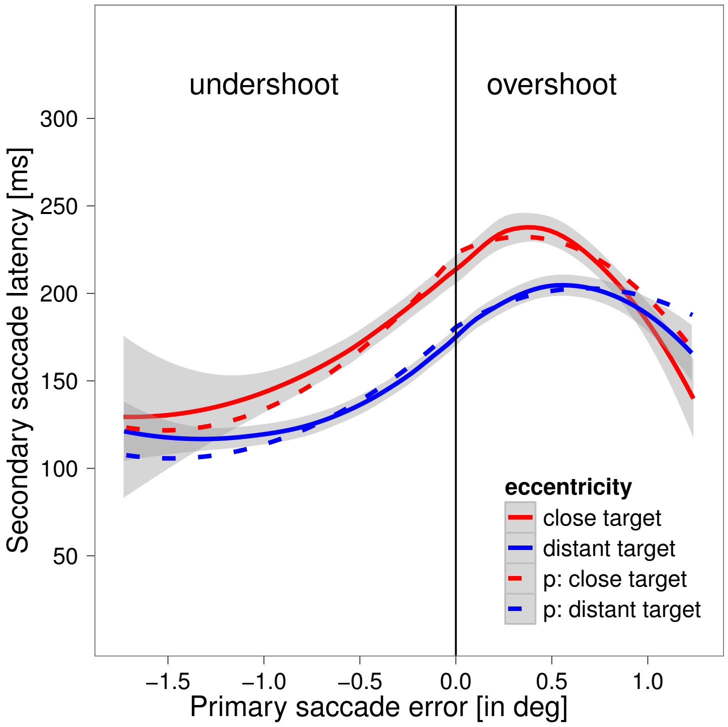

Figure 3.3: Secondary saccade latency as function of saccadic error by target eccentricity. Smoothing (solid line) is based on loess method (degree = 2). For smoothing we used saccadic error values ranging from the 2.5 to 97.5 percentile. Grey bands represent the 95% confidence interval. Prediction (p: close target, p: distant target) of the LMM (dashed line) after removal of between-subject variance.

between primary and secondary saccade latency as observed in tasks examining sequences of saccades (Zingale & Kowler, 1987). Given that fast primary saccades are less precise, the inverse relationship between saccadic error and secondary saccade latency could indirectly result from the saccadic system's tendency to execute saccades in a specific rhythm (e.g. fast saccades are also followed by saccades with short latency). Indeed, in control analyses (see Appendix) we find a small decrease of the absolute saccadic error with increasing primary saccade latency. Furthermore, we find a small increase of secondary saccade latency with increasing primary saccade latency. Nevertheless, this explanation can neither account for the overall differences between close and distant targets nor for the shift of minimum amplitude and maximum latency to a small overshoot. Furthermore, it cannot account for the bias to follow primary saccade direction.

3.4 Discussion

The main objective of the present study is to extend the focus on fixational eye movements by studying the fixational process following saccade execution. To this end we tested how (1) the magnitude of the saccadic error, (2) saccadic under-/overshoot, and (3) target eccentricity influence the latency, amplitude and orientation of secondary saccades. All factors contribute to motor programs of secondary saccades. Previous studies concerning the programming of secondary saccades were focusing on the magnitude of the saccadic error and a potentially different influence between under- and overshooting on secondary

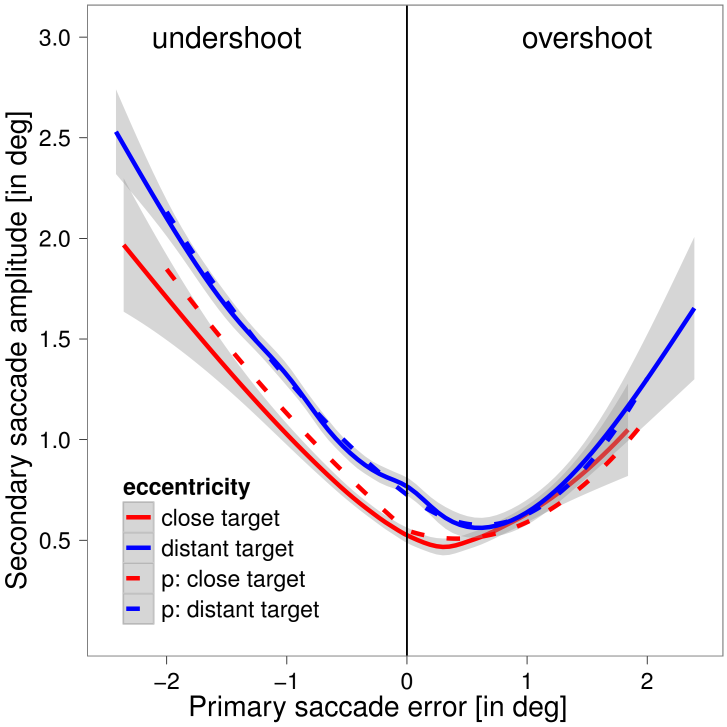

Figure 3.4: Secondary saccade amplitude as function of saccadic error by target eccentricity. Smoothing (solid line) is based on loess method (degree = 2). Grey bands represent the 95% confidence interval. Prediction (p: close target, p: distant target) of the LMM (dashed line) after removal of between-subject variance.

saccade latency. Our results demonstrate that these influences are strongly modulated by target eccentricity, which sheds new light on the postsaccadic oculomotor activity triggering secondary saccades.

When the primary saccade ends in the center of a target, we observed significant effects of target eccentricity with respect to the latency, amplitude and orientation of secondary saccades. Secondary saccades following precise primary saccades to distant targets are on average faster, have a larger amplitude and are more likely to follow primary saccade direction. This result is very surprising if one assumes the saccadic error to be the driving source to perform a secondary saccade. Our results support the initial observation by Lemij and Collewijn (1989) who reported also shorter secondary saccade latencies for increasing target eccentricities; a result they already thought to be independent of the saccadic error.

Increasing saccadic undershoot is associated with decreasing secondary saccade latency and increasing amplitude. Examining the orientation of secondary saccades after undershooting reveals that almost all secondary saccades follow the direction of an undershooting primary saccade. Importantly, maximum latency and minimum amplitude of secondary saccade are shifted to a small overshoot (with an even further shift in the distant target condition). This supports the idea of different consequences for the programming of secondary saccades depending on whether the target is under- or overshot. A further increase in saccadic overshoot is associated with shorter saccade latency and larger amplitude.

Table 3.4: LMM statistics for secondary saccade orientation.

	Estimate	SE	p-Value
Intercept	-1.97	0.274	< 0.001
Eccentricity (ecc)	-1.51	0.434	< 0.001
Undershoot	-7.09	2.094	< 0.001
Overshoot	2.60	0.349	< 0.001
ecc x undershoot	-0.04	4.145	0.992
ecc x overshoot	1.11	0.549	0.044
Variance components		SD	
Subjects		0.37	

Examining the orientation of secondary saccades reveals an additional important difference between under- and overshooting. As reported above, almost all secondary saccades after undershooting follow the direction of the primary saccade (they are corrective secondary saccades). Increasing saccadic overshoot also results in a higher probability of corrective secondary saccades (they are in opposite direction to primary saccade direction). Nevertheless, after overshooting it is still very likely that secondary saccades follow the direction of the primary saccade; therefore increasing the saccadic overshoot. We have shown a significant effect of target eccentricity on the orientation of secondary saccades following an overshoot. As illustrated in Figure 3.5, in the distant target condition a larger saccadic overshoot is necessary in order to elicit a corrective secondary saccade.

3.4.1 Secondary (micro-)saccades during postsaccadic fixation

A large proportion of the observed secondary saccades were in the range of microsaccades. Recently, two models of microsaccade generation have been put forward. Rolfs, Kliegl and Engbert (2008) introduced a conceptual model which assumes the rostral pole of the superior colliculus to be directly involved in microsaccade generation. Hafed, Goffart and Krauzlis (2009) demonstrated a causal involvement of the superior colliculus in the control of microsaccades. As the model of Rolfs and colleagues successfully accounted for various effects concerning small saccades we will use this framework to discuss the observed results of our study. In the model of Rolfs and colleagues a motor map is assumed coding eye movements of very small amplitudes (microsaccades) in the center of the map (around the amplitude of 0°) and increasing amplitudes in increasingly peripheral sites of the map (topographical organization). Rightward eye movements are programmed in the left hemisphere and leftward eye movements in the right hemisphere of the motor map. Activation in one site of the map will also cause excitation of neighboring locations (local excitation) while at the same time distant locations will be inhibited (global inhibition). When activation crosses a certain threshold in this model a saccadic eye movement of the corresponding amplitude and orientation will be executed.

Postsaccadic visual information is processed and will cause activity to build up within the motor map. Considering this model to account for secondary saccade characteristics we claim (1) the visually evoked postsaccadic activation within the hemisphere in which

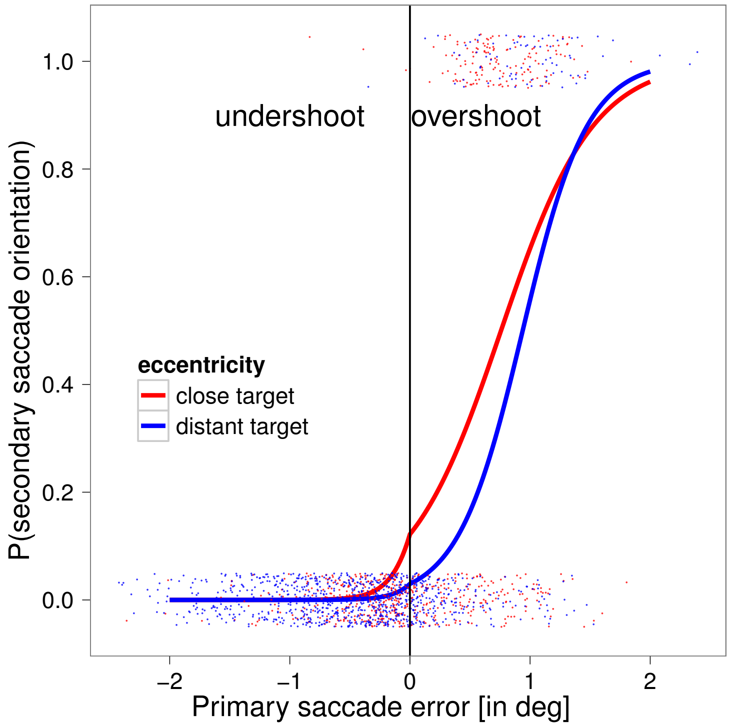

Figure 3.5: Probability of secondary saccade orientation as function of saccadic error by target eccentricity. Individual data points are jittered (vertical jitter = 0.05) around 1 for secondary saccades in opposite direction of the primary saccade. Secondary saccades following primary saccade direction are jittered around 0. Depending on target eccentricity data points are red (close target) and blue (distant target). Predictions of the GLMM (solid line) are displayed after removal of between-subject variance.

the primary saccade has been programmed is enhanced and (2) the enhancement is stronger for distant as opposed to close targets.

Strong support for these claims is given by studying the orientation of secondary saccades. When a target is undershot almost all secondary saccades follow the direction of the primary saccade. Note, even at minimal saccadic error secondary saccades still follow almost exclusively the direction of the primary saccade. Under the assumption of equal postsaccadic activation in both hemispheres of the motor map one would expect secondary saccades after minimal error to be executed in both directions with same probability. As shown, this is clearly not the case. The second claim is supported by the finding that primary saccades to distant in contrast to close targets have to overshoot the target even more in order to be followed by a corrective secondary saccade. Thus, the activation in the hemisphere which programmed the primary saccade is even more enhanced in the distant target condition as there is a stronger tendency of secondary saccades to follow primary saccade direction.

Interestingly, the assumption of different levels of postsaccadic activation in the two hemispheres of the motor map predicts a shift of maximum latency and minimum amplitude from zero saccadic error to a small overshoot. After exact primary saccades the incoming visual signal of the fixated target will cause activation to build up around the center of the motor map. This visually evoked response will be stronger in the hemisphere which programmed the initial primary saccade, causing the building up activation to be biased towards this hemisphere. Further, this bias will be stronger after saccades to distant as opposed to close targets. Consequently after saccades to distant targets maximum latency and minimum amplitude is even further shifted in the direction of overshooting. Revealing the sources for different activations in the two hemispheres of the motor map and their modulation by target eccentricity is beyond the scope of this study. We argue that at least three possible sources have to be considered in future investigations. First, execution of saccades with an amplitude of 6°(close target) and 14°(distant target) might result in a different distribution of activation within the oculomotor network (e.g. superior colliculus; Wang et al., 2011). Second, the strength of the incoming visual information might be modulated by the previous saccade amplitude. It has been shown that saccade execution causes a postsaccadic enhancement of neural firing in the lateral geniculate nucleus (Reppas, Usrey, & Reid, 2002; Royal et al., 2006). Nevertheless, examining the influence of saccade amplitude on postsaccadic enhancement of neural firing in the lateral geniculate nucleus, Reppas, Usrey and Reid (2002) conclude that the effect of saccade amplitude is rather small. Third, the generation of microsaccades is known to be influenced by the allocation of attention (Engbert & Kliegl, 2003; Hafed & Clark, 2002). Importantly, directly after the end of a saccade attention resides in retinotopic coordinates for some time, which is referred to as the retinotopic attentional trace (Golomb, Chun, & Mazer, 2008; Golomb et al., 2010). With increasing time attentional facilitation decays at the retinotopic and increases at the spatiotopic location. Given the close link of microsaccades and attention we suspect the retinotopic attentional trace to be an important phenomenon when studying secondary saccades and the process of postsaccadic fixation.

So far we have not addressed how our model accounts for the inverse relationship between the magnitude of saccadic error and secondary saccade latency. There are two possible mechanisms. First, an error signal is computed which impinges on the saccadic motor map. A larger saccadic error would be associated with a stronger error signal and consequently, cause faster secondary saccades. Second, it has been shown that primary saccade latency decreases with decreasing eccentricity but increases again for very small eccentricities (Kalesnykas & Hallett, 1994). The same latency-eccentricity function could be present for secondary saccades in our study. Thus, the inverse relationship between saccadic error and subsequent latency could simply result from the fact that very small eye movements in general need more time to be executed. Importantly, this explanation would not need an additional computation of an (extra-retinal) error signal.

3.5 Conclusion

The present study demonstrates that secondary saccades are strongly influenced by characteristics of the previous goal-directed saccade. We replicate that subsequent, postsaccadic eye movements are largely determined by the initial landing error. Importantly, we provide new evidence for a different postsaccadic fixational state depending on whether the target has been under- or overshot. We extend these results with an additional modulation by target eccentricity. All of them constitute important influences on the generation of secondary saccades during postsaccadic fixation. Our experimental task and the proposed mechanisms for a model of postsaccadic fixation strongly rely on postsaccadic visual processing of the target. Future experiments will examine how postsaccadic fixation depends on the quality of incoming visual information and a possible role of extra-retinal error correction.

Microsaccades constitute a large proportion of postsaccadic eye movements in our experiment. Much current research is devoted to understand how microsaccades contribute to perception: that is to their role in functions such as counteracting visual fading, maintaining visual stability, and as an index of covert attention. The present study constitutes an important step to examine small eye movements in a more natural situation which is examining (micro-)saccades being embedded in a sequence of saccades and fixations.

Chapter 4

Quantifying time-dependent influences on secondary saccade rate

4.1 Introduction

In the previous chapter we demonstrated that the process of fixation strongly depends on the preceding large saccadic eye movement (Ohl, Brandt, & Kliegl, 2011). More specifically, we observed a strong bias for secondary (micro-)saccades to follow the direction of the primary saccade. Secondary (micro-)saccades following a primary saccade to a more distant target, showed a stronger bias to follow primary saccade direction, they had shorter latencies and larger amplitudes. This shows that in addition to the classical influence of primary saccade error (see Becker & Fuchs, 1969), multiple factors influence the programming of small secondary saccades. Noteworthy, results on small eye movements during postsaccadic fixation may also constitute possible new constraints in order to falsify models of (micro-)saccade generation in general. In the remainder of the present study, we will refer to secondary (micro-)saccades as secondary saccades (which also include eye movements as small as microsaccades).

To account for the characteristics of secondary saccades we extended a model introduced by Rolfs, Kliegl and Engbert (2008). The originally described model represents a topographically organized saccadic motor map (see Figure 4.1, upper panel). The center of the map is coding for the smallest possible saccadic eye movements (e.g., microsaccades). Increasingly distant locations to the left (right) are coding for increasing saccade amplitudes to the right (left). In order to generate a (micro-)saccade with a specific amplitude, activation at that specific location in the saccadic motor map has to cross a threshold for saccade triggering. In addition, the different locations in the map are connected following the principle that neighboring sites excite and distant areas inhibit each other.

In order to account for the generation of small eye movements following large primary saccades we extended this model by assuming (1) a hemispheric bias in the saccadic motor map, meaning a higher level of activation in the hemisphere of the saccadic motor map which also programmed the primary saccade (see Figure 4.1, middle panel) and (2) an even stronger hemispheric bias for larger target eccentricities.

In our recent study we did not examine the influences of primary saccade error and target eccentricity on the number of secondary saccades. Importantly, the model we proposed also makes strong predictions concerning the rate of secondary saccades which we are going to test in the present study.

First, our model assumes increased activation in the hemisphere of the saccadic motor map which also programmed the primary saccade. Following saccadic undershoot the target stimulus will elicit a neuronal response within this hemisphere. This neuronal response then adds to the baseline activation. Based on this reasoning it follows the prediction that the activation should be more likely to cross the threshold for saccade triggering after primary saccade undershoot. Consequently, we hypothesize an increased rate after primary saccade undershoot as compared to primary saccade overshoot.

Second, we proposed that the difference in activation between the two hemispheres increases with increasing target eccentricity. Therefore, we hypothesize an increase in secondary saccade rate after primary saccade to distant as compared to close targets (Frost & Pöppel, 1976, Lemij & Collewijn, 1989).

Finally, we hypothesize an increasing rate with increasing absolute saccadic error. This hypothesis is based on the observation that increasing primary saccade error often elicits corrective secondary saccades (Becker & Fuchs, 1969; Hallet, 1978; Prablanc & Jeannerod, 1975; Weber & Daroff, 1972) and may be associated with increasing signal strength. Thus increasing primary saccade error should also increase the urge to correct for it.

We are going to test these hypotheses in the present study. Prior to that we need to address several methodolical challenges associated with testing these hypotheses.

4.1.1 Methodological challenges

In our experiment subjects executed a saccade to target at an eccentricity of 6°or 14°to the left or right of a central fixation point. Furthermore, subjects were required to hold fixation at the target position for at least 1100 ms. Although the experimental design is straightforward, the statistical analyses of such data must address various challenges that have been largely ignored so far. First, given the time window under consideration we are likely to encounter trials in which no secondary saccade occurs. Usually such trials are excluded from the analysis. This procedure inevitably leads to biased estimates of experimental effects. Second, even when the above mentioned variables (target eccentricity, primary saccade error) exert significant effects on the average secondary saccade latency, the strength of this effect may vary as a function of time. Traditional analyses assume that the effect is of equal strength at all times.

A widely used statistical tool to analyze the dynamics of an event after some time, which also takes data into account without an event, is survival analysis. Survival models or, generally speaking, time-to-event models have a broad range of applications. They are useful, for example, in quality control (e.g., time until failure of a machine) or social sciences (e.g., time until marriage). However, the most important area of application is medical research. Examples here are death caused by a special illness, death after surgery, or time until relapse of a tumor. Death is, after healing, one of the most important and drastic outcomes in medicine. Hence, time until death is often used as a measure for therapeutic progress or for determination of risk factors for patients.

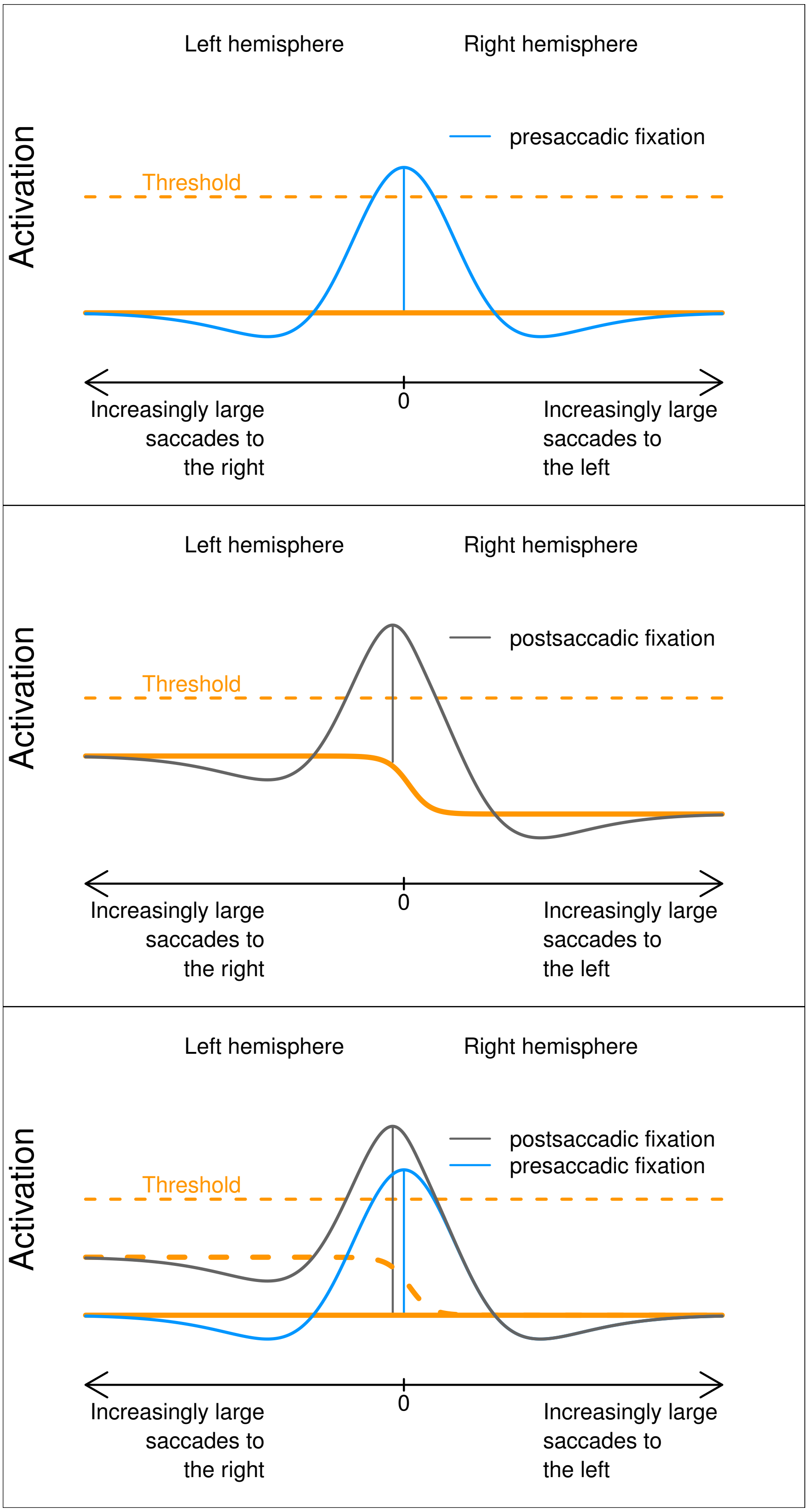

Figure 4.1: Illustration of activity distribution in a motor map for (micro-)saccade generation. **Top panel**, distribution of activation during presaccadic fixation (adapted from Rolfs, Kliegl, & Engbert, 2008). **Middle panel**, distribution of activation during fixation following a saccadic eye movement to the right. Activation is increased in the left hemisphere which programmed the primary saccade to the right. This causes a bias of the overall distribution towards the hemisphere that programmed the primary saccade. The bias increases with increasing target eccentricity. **Bottom panel**, comparison of pre- and postsaccadic fixation.

Concerning our second aim, we will use one type of survival model, the additive hazards model (Aalen, 1980). This type of model is particularly well suited for estimating time-varying covariate effects. The hazard rate is defined as the instantaneous risk for an event to occur at a specific point in time. The additive hazards model is a nonparametric model which yields estimates how a baseline hazard rate changes conditional on the included covariates in the model. Importantly, the covariates included as regression coefficients in the model are allowed to depend on time, thus allowing us to assess when during postsaccadic fixation covariates exert a significant influence on the rate. This is particularly interesting as some covariates may be very influential at some point in time and less influential at another (hence such a variable would be a time-varying covariate).

In summary, the goal of the present study is to examine the influence of primary saccade error, target eccentricity and whether the goal-directed saccade was either hypometric or hypermetric on the rate of secondary saccades. Such rate analysis will allow us to test hypotheses derived from our previously presented qualitative model of (micro-)saccade generation during postsaccadic fixation (Ohl, Brandt, & Kliegl, 2011).

4.2 Materials and methods

The present study is a re-analysis of data from a previously published experiment (Ohl, Brandt, & Kliegl, 2011). For detailed information on the experimental setup, eye movement recordings and procedure, the reader is referred to our previous publication (see chapter 3).

4.2.1 Participants and procedure

Ten undergraduate students (19-28 years old) participated in our study. For participation, they received study credit or were paid seven Euros. In an experimental trial, subjects had to fixate a central fixation point. After a random interval between 1000-1500 ms the fixation point was removed and a target appeared at an eccentricity of 6°or 14°to the left or right of the fixation point. Subjects had to move their eyes immediately to the target and hold fixation at the target location for at least 1100 ms. Each subject performed eight training and 300 test trials. We obtained written informed consent from all subjectes before beginning of the experiment. Our study was in line with the declaration of Helsinki from 1964.

4.2.2 Data preparation and analysis

Eye movements were recorded using an EyeLink-II system (SR, Research, Ontario, Canada) with a sampling rate of 500 Hz. For detection of microsaccades and saccades we used a previously published detection algorithm (Engbert & Kliegl, 2003; Engbert & Mergenthaler, 2006). In a first step, the eye trajectory was transformed in 2D velocity space and afterwards, thresholds for peak velocity (6 SD) and minimum duration (8 ms) were applied.

In order to be included for further analysis a trial had to meet three criteria: (1) primary saccadic reaction times were slower than 80 ms and faster than 400 ms, (2) the distance between primary saccade end point and center of the target was smaller than 2.5°; (3) no eye blinks occured during the trial.

For data analysis we considered an interval of 800 ms after execution of the primary saccade. In total, we observed 2134 trials including at least one secondary (micro-)saccade and 474 trials without any secondary saccade within an interval of 800 ms. Statistical analyses were performed using the *timereg* package (Martinussen & Scheike, 2006) in the R environment (R Development Core Team, 2011). For graphics we mainly used the *ggplot2* package (Wickham, 2009).

Our statistical model included the following covariates. First, saccadic error is the absolute horizontal distance from saccade end point to the center of the target in degree of visual angle. Second, the variable under/ overshoot indicates whether the primary saccade was either hypometric or hypermetric. Each saccade landing in between the fixation point and the center of the target has been regarded as undershoot while all other saccades were classified as overshoots. The final variable is the eccentricity which denotes whether the target was presented at a distance of 6°(close target) or 14°(distant target).

4.3 Results

Our model includes a baseline rate (i.e., intercept) and the following main effects: eccentricity (coding: -1 = close target, 1 = distant target), the absolute saccadic error in degrees of visual angle and under/ overshoot, indicating whether the target has been undershot (coding: -1) or overshot (coding: 1). In addition we included the interaction eccentricity x under/ overshoot.

All three main effects and the interaction significantly influenced the rate of secondary saccades (see Table 4.1; test for non-significant effects). Moreover, all of the observed effects were time dependent, meaning that their influence was significantly different at different time intervals during postsaccadic fixation (see Table 4.1, test for time-invariant effects).

The rate effects need to be interpreted with respect to the baseline rate. In Figure 4.2, the rate for the baseline and each covariate is displayed (the displayed rates are the first derivative of the obtained cumulative coefficients which allows easier interpretation). The rates now tell us whether a covariate increases or decreases the baseline rate at a given point in time. In the baseline rate (Figure 4.2, top left panel), a steep increase until 174 ms was followed by a flat decrease until the end of the observed time interval at 800 ms. The time dependency of the baseline rate was significant ($p = 0.002$).

4.3.1 Target eccentricity

We hypothesized a higher rate of secondary saccades with increasing target eccentricity. This prediction was based on an increasing hemispheric bias of activation with increasing target eccentricity. Indeed, we observed a significant influence of target eccentricity on the secondary saccade rate ($p < 0.001$) in the predicted direction. The rate was significantly higher in the distant target condition, reaching its peak influence around 148 ms (see Figure 4.2).

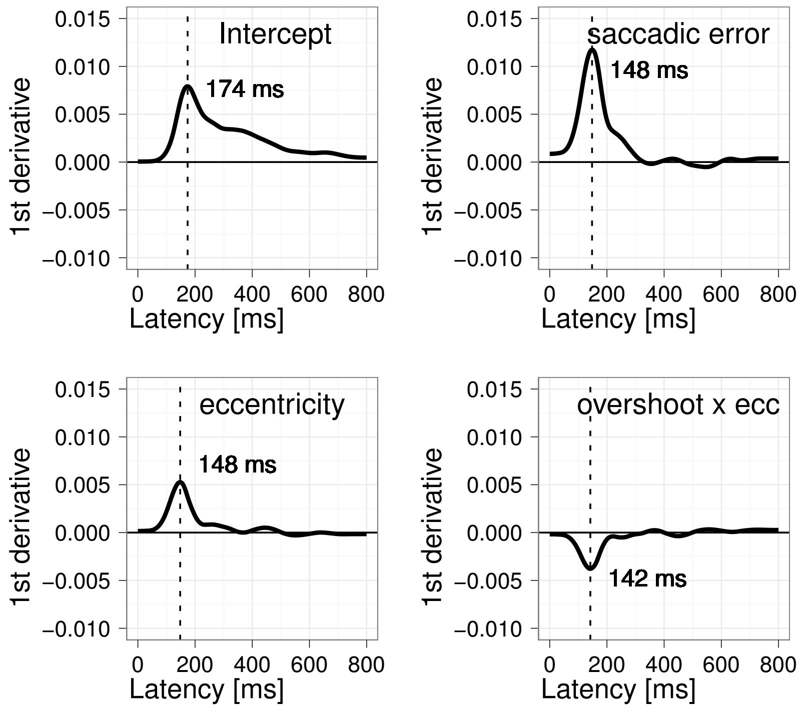

Figure 4.2: Displayed rates represent the estimated first derivative of the obtained cumulative coefficients for each term in our survival analysis.

4.3.2 Saccadic error

We predicted that increasing absolute error should be associated with a higher risk to observe a secondary saccade. This was confirmed by a significant effect of the absolute saccadic error ($p < 0.001$). In Figure 4.2, we see that the effect was strongest after 148 ms (the rate evolvement is shown for an absolute error of one degree of visual angle).

Saccadic error is a continuous covariate. Thus, we have to test whether it violates the null-hypothesis of being consistent with the model, meaning the assumption of additivity. Analysis of the residuals for the covariate saccadic error were not significant ($p = 0.436$), therefore the result concerning saccadic errror turned out to be consistent with the model assumption of additivity (see Figure 4.3).

4.3.3 Undershoot vs. overshoot

In our model we proposed an increased activation in the hemisphere of a saccadic motor map which programmed the primary saccade. Consequently, we hypothesized an increased rate of secondary saccades following primary saccade undershoot (and the opposite effect for primary saccade overshoot). We reasoned that the incoming visually evoked activation can add to the already increased level of activation in the one hemisphere of the saccadic motor map and is therefore more likely to cross the threshold for saccade execution. In Table 4.1 we see a very strong effect of the covariate under/ overshoot ($p < 0.001$). In Figure 4.2, we see a negative influence of this variable on secondary saccades which is in line with our hypothesis. Indeed, we predicted that the rate should be significantly lower for primary saccade overshoot as compared to primary

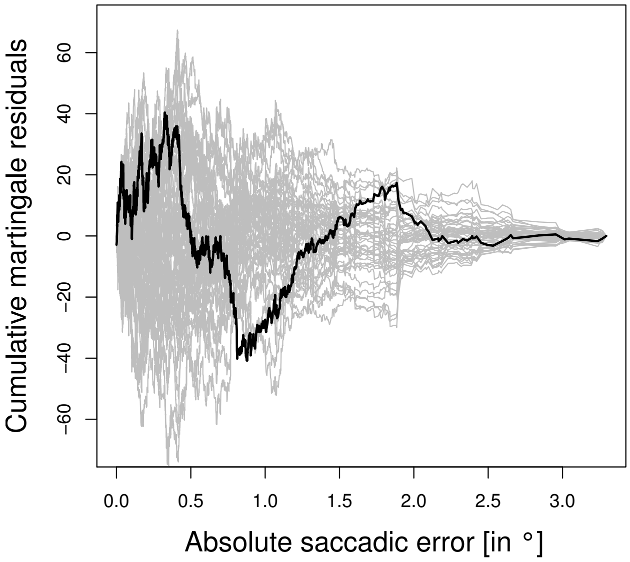

Figure 4.3: Observed cumulative residuals along with 95% confidence interval (dotted lines) for the covariate saccadic error.

Table 4.1: Results of Aalen's additive hazard model.

	Test for non-significant effects		Test for time-invariant effects	
	Test statistic	p-Value	Test statistic	p-Value
(Intercept)	12.4	<0.001	0.46	0.002
saccadic error	7.59	<0.001	0.8	<0.001
Under/Overshoot	22.3	<0.001	0.55	<0.001
Eccentricity (ecc)	10.7	<0.001	0.32	<0.001
Ecc x underover	10.8	<0.001	0.25	<0.001

saccade undershoot. This influence is strongest at 158 ms after beginning of postsaccadic fixation.

Finally, we observed a significant interaction of target eccentricity x under/ overshoot ($p < 0.001$). Undershooting a distant target (in contrast to a close target) further increased secondary saccade rate. The difference in secondary saccade rate between overshooting a close vs. distant target was reduced. The interaction reached its maximum influence after 142 ms.

4.4 Discussion

The main goal of the present study was to test predictions for secondary saccades rate derived from our recently introduced qualitative model. We used an additive hazard model in order to examine the influence of primary saccade error (quantified by the absolute saccadic error and whether the target was under- or overshot) and target eccentricity on secondary saccade rate.

We observed a higher rate following saccadic undershoot as compared to overshoot. Robinson (1973) already suggested an advantage in programming secondary saccades following saccadic undershoot. This explanation is based on the reasoning that the undershot target can be kept in the same hemisphere; thus avoiding the extra-cost of inter-hemispheric transfer. So far, a benefit in programming of secondary saccades following saccadic undershoot has been observed in terms of shorter secondary saccade latencies (Deubel, Wolf, & Hauske, 1982; Henson, 1978). The result of an increased secondary saccade rate following saccadic undershoot provides further support for Robinson's initial reasoning.

Furthermore, we found a significant influence of target eccentricity. Secondary saccade rate was higher for distant than close targets, therefore replicating results from previous studies (Frost & Pöppel, 1976; Lemij & Collewijn, 1989; Ohl, Brandt, & Kliegl, 2011). Finally, we observed an increasing number of secondary saccades with increasing primary saccade error (Lemij & Collewijn, 1989) which replicates a classical corrective influence on secondary saccades (Becker & Fuchs, 1969). These results are in line with hypotheses derived from our model of postsaccadic fixation which is based on two principles: (1) increased activation in the hemisphere of a saccadic motor map which programmed the primary saccade and (2) an even stronger increase with increasing target eccentricity.

A second important goal of our study was to introduce Aalen's additive hazard model as a promising tool for eye movement research. This allowed us to include also the information from trials in which we did not observe any secondary saccade. Importantly, our analysis did not only show the overall influence of a covariate on secondary saccade rate, but we also demonstrated the time-dependency of these influences. Thus, the results from the present survival analysis go beyond our recent observation concerning secondary saccades in a paradigm with visual feedback (Ohl, Brandt, & Kliegl, 2011).

Finally, our study provides new insights into mechanisms underlying the generation of small eye movements. Recently, an increasing number of mechanisms and models for microsaccade generation have been published (see chapter 2; e.g., Engbert et al., 2011; Hafed, Goffart, & Krauzlis, 2009; Inagaki, Hirata, & Usui, 2011; Otero-Millan et al., 2011; Rolfs, Kliegl, & Engbert, 2008). Consequently, there is a need to agree upon tests how to falsify these models (Otero-Millan et al., 2011). We think that studying secondary saccades, which are often as small as microsaccades, offers a potential new test to compare models that aim to account for generation of small-amplitude eye movements during fixation.

Chapter 5

The generation of secondary saccades without postsaccadic visual feedback

5.1 Introduction

Humans move their eyes 2-4 times per second in order to bring visual areas of interest onto the fovea and allow visual processing with highest resolution. Saccades are frequently followed by secondary saccades whose exact triggering mechanisms are not fully understood. In general, secondary saccades are thought to reduce the distance between saccade landing site and target location (Becker & Fuchs, 1969). Consequently, secondary saccades are often referred to as corrective saccades. Despite the seemingly obvious corrective function of secondary saccades, several lines of evidence suggest that an error-correcting signal constitutes only one source of activation that influences the programming of secondary saccades.

First, even very precise primary saccades can be followed by secondary saccades, which might then increase the distance between eye and target position (Lemij & Collewijn, 1989). Second, there is a bias of secondary saccades to follow the direction of the primary saccade (Ohl, Brandt, & Kliegl, 2011). Third, models have been proposed, assuming that target eccentricity significantly modulates the postsaccadic activity distribution in a saccadic motor map and accordingly the characteristics (e.g., latency, amplitude, and orientation) of subsequent eye movements (Ohl, Brandt, & Kliegl, 2011; Wang et al., 2011). Thus, multiple factors contribute to the programing of secondary saccades, a fact that requires further elaboration of the exact mechanisms producing secondary saccades. Focusing only on the process of error correction (e.g., minimizing the distance between eye and target position) falls short of providing a comprehensive view on secondary saccades.

In the present study our aim was to explore the factors that influence secondary saccade programs in the absence of postsaccadic visual feedback. Participants were asked to move their eyes to an upcoming target. During saccade flight the target was removed, therefore preventing postsaccadic visual feedback. In such a situation, it is typically observed that the number of secondary saccades is strongly reduced as

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compared to a situation with available postsaccadic visual information (Becker & Fuchs, 1969; Bonnetblanc & Baraduc, 2007; Deubel, Wolf, & Hauske, 1982; Prablanc & Jeannerod, 1975; Shebilske, 1976). This is, of course, strong evidence emphasizing the importance of postsaccadic visual information for the programming of secondary saccades.

The examination of secondary saccade orientation in a paradigm that omits postsaccadic visual information allows us to determine whether an extra-retinal error signal influences programming of secondary saccades. The idea that a copy of the saccade motor command, the efference copy, is used as source of information for various brain processes is old (Sperry, 1950; von Helmholtz, 1925; von Holst & Mittelstaedt, 1950) and also very successful, both on a conceptual (Wurtz, 2008) and a neurophysiological level (Sommer & Wurtz, 2008). Nevertheless, whether the copy of the primary saccade motor command also influences the generation of secondary saccades is strongly disputed.

A recent study suggests that the oculomotor error of the primary saccade (e.g., the distance between saccade landing position and target) might already be included in the efference copy of the saccade (Collins et al., 2009). Thus, subtraction of the vector of the efference copy from the target vector could easily determine the postsaccadic target position and consequently influence the programming of secondary saccades. It is uncertain whether such type of extra-retinal error signal directly influences the programming of secondary saccades. Interestingly, early studies that prevented postsaccadic visual processing by removing the target during the saccade resulted in opposing conclusions concerning the role of an extra-retinal error signal for secondary saccade programs (Becker & Fuchs, 1969; Morel, Deneve, & Baraduc, 2011; Prablanc & Jeannerod, 1975; Shebilske, 1976; Weber & Daroff, 1972).

A study by Deubel, Wolf and Hauske (1982) demonstrated convincingly that visual feedback is necessary to generate secondary saccades. It should be noted, however, that in their study, the role of visual feedback for the generation of secondary saccades was examined by blanking the target for some time during postsaccadic fixation. The target was lit again subsequently, thus providing visual feedback at a later point of the trial. Given a situation without visual feedback or very large saccadic error, Deubel et al. did not want to rule out that an extra-retinal error signal might come into play. Therefore, whether or not an extra-retinal error signal directly influences secondary saccades is not clear and needs further elaboration.

Studies supporting the notion that an extra-retinal error signal contributes to corrective secondary saccades mainly examined secondary saccades following primary saccades to very distant targets (e.g., larger than 20°; Becker & Fuchs, 1969; Shebilske, 1976). Since saccades to very distant targets are typically undershot, this is a necessary but nevertheless insufficient condition for demonstrating a direct influence of an extra-retinal error signal on secondary saccade programs. The subsequent secondary saccade then follows the same direction as the undershooting primary saccade and consequently reduces the target undershoot. This reaction can also be explained by usage of a simple strategy in such a way that secondary saccades follow the direction of the primary saccade. Indeed, we recently demonstrated that secondary (micro-)saccades are biased to follow the direction of the primary saccade (Ohl, Brandt, & Kliegl, 2011); therefore secondary saccades that follow the direction of the primary saccade to a very distant target (which is typically undershot by about 10 percent) are not necessarily triggered by an extra-retinal error signal but could simply be due to this bias. Strong support for the influence of an extra-retinal error signal requires a condition where the probability of secondary saccades in direction opposite to the primary saccade increases with increasing overshoot in the absence of postsaccadic visual information.

In the present study, subjects were asked to move their eyes from a central fixation point to a peripheral target (located at 6 or 14 degrees of visual angle on the horizontal meridian) and hold fixation at the new target location despite the target being removed immediately after saccade onset. Our experimental paradigm allows us to determine the functional relationship between primary saccade landing site and the characteristics of secondary saccades (e.g., latency, amplitude and orientation) for two different target eccentricities. Assuming that the latency, amplitude or orientation of secondary saccades depends on saccade landing site would strongly support the notion that an extra-retinal error signal can indeed influence secondary saccade programming. We also explore whether the bias of secondary saccades to follow the direction of the primary saccade might also be observed in a condition without postsaccadic visual information. Moreover, we test whether these relationships between primary saccade error and secondary saccade latency, primary saccade error and secondary saccade amplitude, as well as between primary saccade error and secondary saccade orientation vary as a function of target eccentricity.

In summary, we examine the influence of saccadic error on subsequent secondary saccade latency, amplitude, and orientation. This should add valuable information for the solution of the debate on whether an extra-retinal error signal influences secondary saccade programs. Furthermore, we test whether an orientation bias and a modulation by target eccentricity are also observed when no postsaccadic visual information is available.

5.2 Materials and methods

5.2.1 Participants

24 subjects, 19-44 years old (M = 24.4), participated in the study. The experimental session involved eight practice trials and 300 test trials. Participants were paid seven Euros or received study credit. The experiment was conducted in accordance with the declaration of Helsinki (1964) and written informed consent was obtained from all participants prior to the experiment.

5.2.2 Materials and procedure

Participants sat in a dark, silent room with their head positioned on a chin rest. Stimuli were presented at a viewing distance of 50 cm on a 19-in. EYE-Q 650 CRT monitor at a spatial resolution of 1024 x 768 pixels and a refresh rate of 100 Hz. Stimulus presentation was controlled by an Apple Power Macintosh G4 computer. Eye movements were recorded with the EyeLink-II system (SR Research, Osgoode, Ontario, Canada) with a sampling rate of 500 Hz. The experiment was implemented in MATLAB (MathWorks, Natick, Massachusetts, USA) using the EyeLink (Cornelissen, Peters, & Palmer, 2002) and Psychophysics (Brainard, 1997; Pelli, 1997) toolboxes.

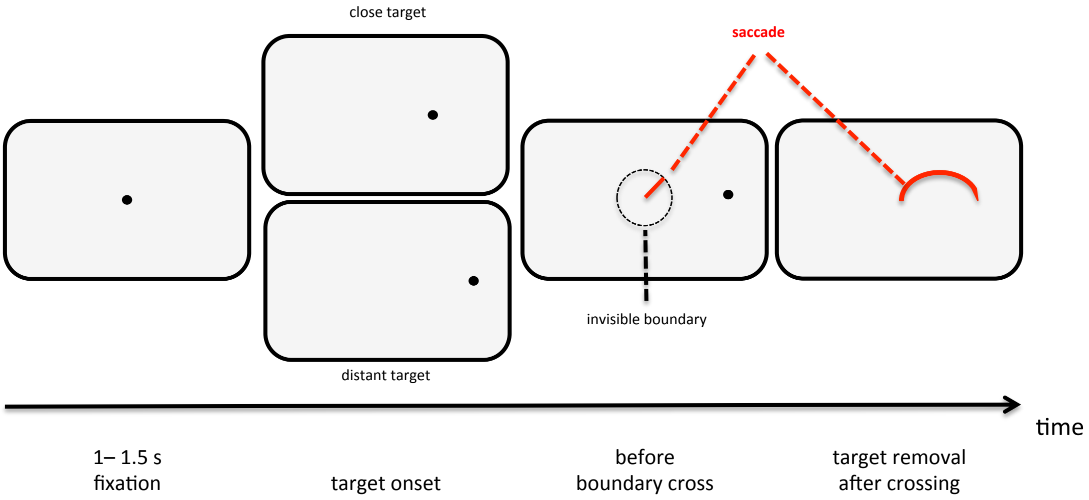

Figure 5.1: After an initial fixation of 1000-1500 ms, a target was presented on the horizontal meridian at close or distant eccentricity. The central fixation point was simultaneously removed. Subjects were required to move their eyes to the target. When the eyes crossed an invisible boundary, the target was also removed. After the end of the response saccade subjects had to keep fixating until the end of the trial.

Each trial started with the subject fixating a white point (0.67°diameter), presented on a grey background in the center of the monitor (see Figure 5.1). After a uniformly distributed random interval (1000-1500 ms), the fixation point was removed and a white circle target (0.67°diameter) was presented at a distance of 6°or 14°to the left or right of the fixation point. The order of target locations was randomly chosen and each target had the same probability to be presented. The subject's task was to move the eyes immediately to the target. After the subject's eyes crossed an invisible boundary located at a distance of 2°from the initial fixation point, the target was removed, resulting in a completely grey background. Subjects were instructed to hold fixation after the end of the goal-directed saccade (for at least 1100 ms). The next trial started after an interval of 500 ms, given that the eyes were within a defined area around the fixation point. When fixation failed, a drift correction was carried out (e.g., presentation of a fixation symbol and correction of small drifts in the computation of eye position). At the beginning of the experiment and after blocks of 30 trials eye-tracker calibration and validation procedures were run. Blinks occurring during a trial were detected online and lead to trial abort. These trials were presented again before the end of the experiment in randomized order.

5.2.3 Data preparation and analysis

For detection of (micro-)saccades we used an improved version (Engbert & Mergenthaler, 2006) of the original algorithm developed by Engbert and Kliegl (2003). For (micro-)saccade detection, eye positions were transformed in 2-D velocity space and thresholds for peak velocity (6 SD) and minimum duration (8 ms) were applied. Primary saccades were defined as the first saccadic eye movement landing within a distance of 2.5°around the target location. Primary saccadic reaction times (SRT) faster than 80 ms, slower than 500 ms and trials with eye movements larger than 1.5°prior to target onset were discarded

Table 5.1: Characteristics of primary saccades. Mean (SD) are displayed for primary saccadic reaction times (SRT), saccadic error (accuracy) and the percentage of secondary saccades. A negative (positive) accuracy indicates undershooting (overshooting).

	Close target	Distant target
SRT in ms	167.3 (25.6)	183.5 (31.1)
Accuracy in °	0.057 (0.374)	−0.419 (0.406)
Secondary saccades in %	42 (25.2)	50 (26.8)

from further analysis. We analyzed the first secondary (micro-)saccade occurring within an interval of 600 ms after the end of the primary saccade. Secondary saccade latency was defined as the interval between the end of the primary saccade and the beginning of the secondary saccade. Trials including a secondary saccade with an amplitude larger than 3.5°were also removed in order to exclude trials in which subjects wrongly directed their gaze back to the center of the screen in anticipation of the next trial. Overall, 2880 secondary (micro-)saccades were detected (M = 120 events per subject). In a second step, based on the angular orientation, we determined whether secondary (micro-)saccades (1) followed the direction of the primary saccade, (2) followed the opposite direction of the primary saccade or (3) were vertically oriented. For analysis of secondary saccade orientation, we excluded vertically oriented secondary (micro-)saccades. This resulted in 2619 events (of the original 2880 events; 91%).

For data analysis we used the R Environment (R Development Core, Team 2010) and more specifically the lme4 package (Bates & Maechler, 2010). The lme4 package allows specification of a linear mixed model (LMM) and also generalized LMMs (GLMM) for binary responses. The statistical advantages of LMMs over the classical ANOVA have been repeatedly addressed. They include the specification of random effects, tests of covariates, and the robustness of LMMs in not fully balanced designs as a result of missing data (Kliegl et al., 2010). Typically, t-values are reported when using LMMs. We considered absolute t-values larger than 2 as significant. Note, negative t-values indicate a coefficient of a model predictor with a negative sign. Graphics were created using the ggplot2 package (Wickham, 2009).

5.3 Results

5.3.1 General characteristics

The characteristics of primary saccades are displayed in Table 5.1. Primary saccades to close targets were very precise on average (Kowler & Blaser, 1995), while there was a tendency to undershoot distant targets (Becker & Fuchs, 1969; Henson, 1978; Kapoula, 1985). Within a time interval of 600 ms, secondary saccades occurred slightly more often in the distant target condition in comparison to those in close target condition. In Figure 5.2, distributions of saccadic error, secondary saccade latency, and amplitude are shown separately for the close and distant target condition. Importantly, under- and overshoots were observed for both target eccentricities. Moreover, many of the observed secondary saccades were smaller than one degree of visual angle, and thus met the criterion for microsaccades.

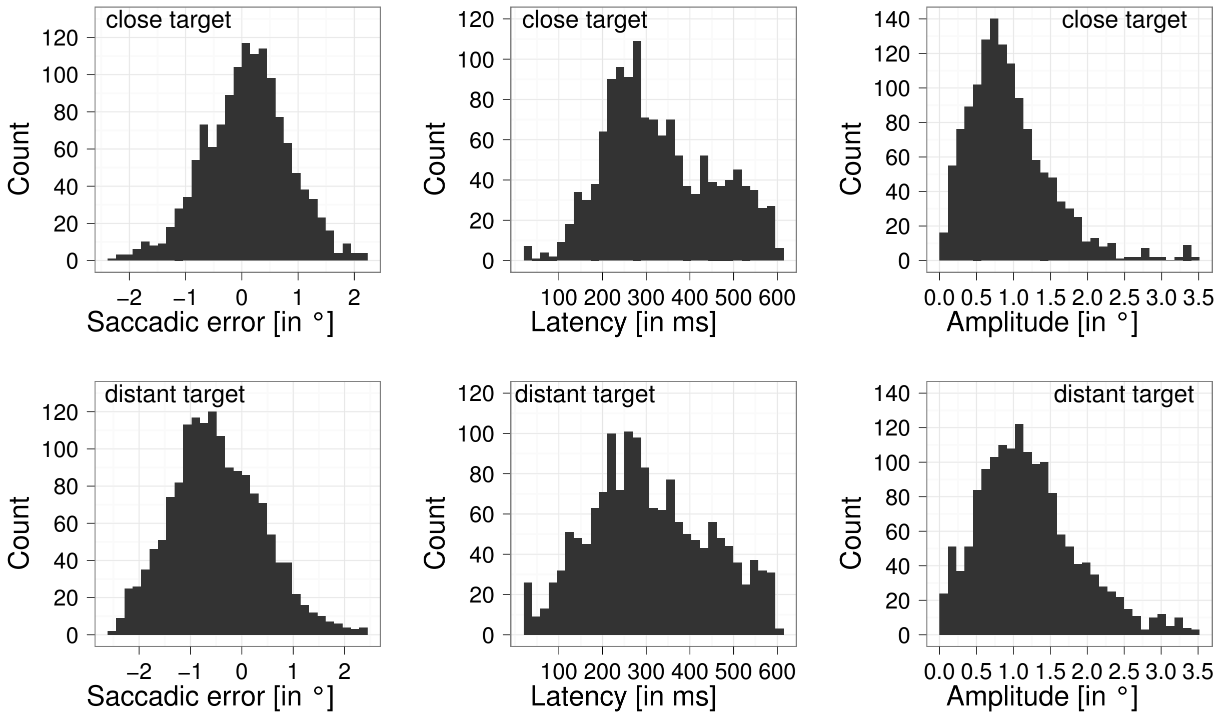

Figure 5.2: Distribution of primary saccade error, secondary saccade latency and secondary saccade amplitude for close and distant target conditions.

5.3.2 Secondary saccade latency

For tasks with postsaccadic visual information available it is typically reported that larger primary saccade errors are associated with larger secondary saccade amplitudes and shorter secondary saccade latencies (Kapoula & Robinson, 1986). This result can be explained by different mechanisms. First, an error signal (either retinal, extra-retinal, or both) is computed which is present as additional activity in a saccadic motor map. This additional activation increases with increasing magnitude of primary saccade error and consequently speeds up the generation of a secondary saccade. Second, the latency of saccadic eye movements is known to be influenced by target eccentricity (Kalesnykas & Hallett, 1994); increasing eccentricity is associated with increasing latency, while for very small eccentricities the latency is increased, too. Thus, decreasing secondary saccade latency with increasing error could simply reflect the general eccentricity-dependent modulation of saccade latency.

In the present study, postsaccadic visual feedback was not available. In this situation, secondary saccade latency can only vary with primary saccade error when the oculomotor system is informed about primary saccade error by an extra-retinal signal. Consequently, a significant modulation of secondary saccade latency by primary saccade error is a first hint of an extra-retinal influence on the generation of secondary saccades.

In Figure 5.3, secondary saccade latency is shown as a function of saccadic error and target eccentricity. Overall, secondary saccade latency is not strongly modulated by primary saccade landing site. Nevertheless, increasing undershoot as well as increasing overshoot (even if only slightly) appear to result in shorter secondary saccade latency,

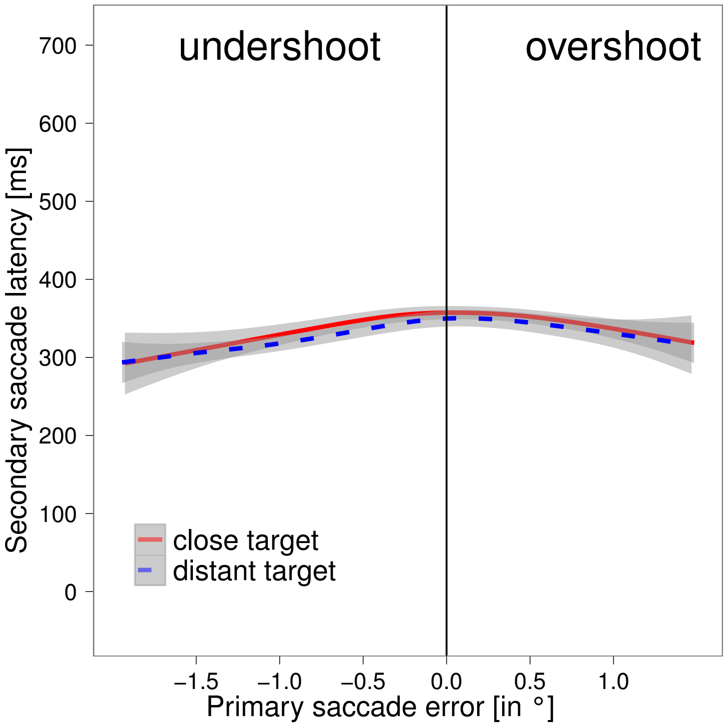

Figure 5.3: Secondary saccade latency as a function of primary saccade error and target eccentricity (red solid = close target; blue dashed = distant target) after removing between-subject variance. Grey bands show 95% confidence intervals.

while target eccentricity does not affect the functional relationship between saccadic error and latency.

We tested the influence of linear and square saccadic error, target eccentricity as well as interactions between saccadic error and target eccentricity on the latency of secondary saccades with a linear mixed model (LMM). In addition to these fixed effects, we included the intercept, saccadic error (linear and square) and target eccentricity as random effects in the LMM (see Table 5.2). Thus, besides the analysis of fixed effects we estimate variance components associated with the fixed effects as well as correlation parameters between them (Kliegl et al., 2010).

Indeed, in agreement with Figure 5.3, square saccadic error was the only fixed effect that significantly influenced secondary saccade latency ($t = -3.72$). The modulation of secondary saccade latency by primary saccade error supports the notion of an extra-retinal error signal influencing secondary saccade programming. The observed influence was in the expected direction with increasing error being associated with shorter secondary saccade latency. Target eccentricity had no significant influence on the latency of secondary saccades ($t = -1.34$).

5.3.3 Secondary saccade orientation

The main goal of our present study was to determine the functional relationship between primary saccade landing position and the orientation of secondary saccades. In the absence of any information about primary saccade error for programs of secondary

Table 5.2: LMM statistics for secondary saccade latency.

	Estimate	SE	t-Value
Intercept	356	12.3	28.89
Eccentricity (ecc)	−11.8	8.8	−1.34
error	6	6.3	0.96
square error	−21	5.6	−3.72
ecc x error	−7.7	7.1	−1.08
ecc x square error	1.4	5.8	0.24

Variance components	SD	Cor.		
Intercept	56.3			
eccentricity	30.9	0.54		
error	14.4	0.34	0.51	
square error	14.4	−0.32	−0.16	0.54

saccades, the orientation of secondary saccades should be independent of primary saccade error. In contrast, a significant influence of primary saccade error on secondary saccade orientation in the absence of postsaccadic visual feedback (i.e., secondary saccades in direction of an undershooting primary saccade and opposite to the direction of an overshooting primary saccade), strongly supports the hypothesis that an extra-retinal error signal influences the generation of secondary saccades.

In Figure 5.4, secondary saccade orientation is shown as a function of primary saccade error and target eccentricity. Saccadic undershoot was associated with secondary saccades in the same direction as the primary saccade. Moving from undershoot to overshoot resulted in an increasing probability to observe secondary saccades in direction opposite to the primary saccade. Furthermore, secondary saccade orientation was influenced by target eccentricity.

Secondary saccade orientation was modeled with a generalized linear mixed model (GLMM) for binomial data. The following covariates were included in the model: (1) magnitude of saccadic error, (2) target eccentricity, and (3) the interaction between saccadic error and target eccentricity (see Table 5.3). Again, we also estimated variance components and correlation parameters for intercept, saccadic error and eccentricity with this model.

The GLMM confirms our inspection of Figure 5.4, showing a significant effect of saccadic error ($p < 0.001$) on secondary saccade orientation. The more primary saccade landing site shifted from undershoot to overshoot, the higher was the probability of secondary saccades to be oriented in direction opposite to the primary saccade. This result strongly supports the hypothesis that secondary saccades are directly influenced by an extra-retinal error signal. The strength of this effect was significantly smaller in the distant target condition ($p < 0.001$). Also, we observed a significant orientation bias after primary saccades to distant targets ($p < 0.001$). Secondary saccades in the distant target condition mainly followed the direction of the primary saccade for precise saccades, therefore providing additional support for the hypothesis that both an extra-retinal error signal and target eccentricity contribute to the programming of secondary saccades.

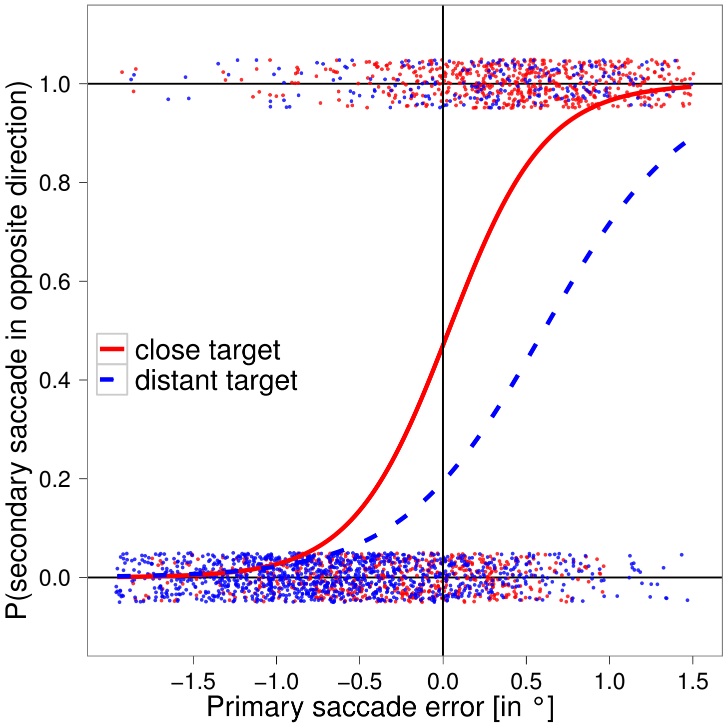

Figure 5.4: Probability of secondary saccades in the direction opposite to primary saccade direction as a function of saccadic error and target eccentricity. Data points are jittered (vertical jitter = 0.05) around p = 1 for secondary saccades in direction opposite to the primary saccade. When secondary saccades follow the direction of the primary saccade, they are jittered around p = 0. Data points are colored in red (close target) or blue (distant target). Predictions derived from the GLMM are shown for the close (red, solid) and distant (blue, dashed) target condition.

5.3.4 Secondary saccade amplitude

In the previous section we demonstrated that the direction of secondary saccades depends on primary saccade error even in the absence of postsaccadic visual feedback. Most notably, increasing the magnitude of primary saccade overshoot increased the probability that secondary saccades were in direction opposite to the primary saccade. In the next step, we were interested to examine secondary saccade amplitude as a function of primary saccade landing position and target eccentricity. We hypothesized that increasing primary saccade error should elicit secondary saccades with increasing absolute amplitude.

In Figure 5.5, secondary saccade amplitude is displayed as a function of primary saccade error and target eccentricity. Again, saccadic error appears to influence the amplitude of secondary saccades. This time we also observed a large difference between close and distant targets. In the LMM for modeling secondary saccade amplitude (see Table 5.4), we used primary saccade error and target eccentricity as predictors to account for signed secondary saccade amplitude (Collins, 2010; Joiner, Fitzgibbon, & Wurtz, 2010). Primary saccade error in the close target condition significantly influenced secondary saccade amplitude ($t = -14.42$), thus providing additional support for the idea that an extra-retinal error signal contributes to secondary saccade motor programs. As expected from such a perspective, increasing undershoot was associated with an increase in amplitude of secondary saccades that followed primary saccade direction.

Table 5.3: GLMM statistics for secondary saccade orientation.

	Estimate	SE	*t*-Value
Intercept	-0.118	0.403	0.77
Eccentricity (ecc)	-1.305	0.331	< 0.001
error	3.463	0.342	< 0.001
ecc x error	-1.109	0.275	< 0.001
Variance components	SD	Cor.	
Intercept	1.83		
eccentricity	1.33	-0.72	
error	1.11	-0.21	-0.31

Table 5.4: LMM statistics for secondary saccade amplitude.

		Estimate	SE	*t*-Value
Intercept		-0.009	0.165	-0.06
Eccentricity (ecc)		0.442	0.107	4.12
error		-0.765	0.053	-14.42
ecc x error		0.094	0.044	2.14
Variance components	SD	Cor.		
Intercept	0.793			
eccentricity	0.482	-0.14		
error	0.178	0.23	-0.44	

Also increasing overshoot resulted in an increase of absolute amplitude of secondary saccades in direction opposite to the primary saccade. The relationship between primary saccade error and secondary saccade amplitude was significantly weaker in the distant as compared to the close target condition (t = 2.14). In addition, we observed a significant effect of target eccentricity (t = 4.12) with a shift away from optimal corrective behavior in the distant target condition. Following precise primary saccades, secondary saccade amplitude was significantly increased in the distant as compared to the close target condition. This result implies that, in addition to an extra-retinal error signal, there is also an effect of target eccentricity. Obviously, more than one factor influence the programming of secondary saccades.

In additional analyses we determined the correlation between our dependent variables in order to examine the degree of overlap in information represented in our analyses of secondary saccade latency, amplitude and orientation. It is noteworthy that the correlation between secondary saccade amplitude and latency was not significantly different from zero (r = -0.01, p = 0.61). This holds for both secondary saccades in the same direction as the primary saccade (r = -0.02, p = 0.47) and for secondary saccades in direction opposite to the primary saccade (r = 0.04, p = 0.27). Thus, the reported results represent complementary rather than redundant information on secondary saccade programming.

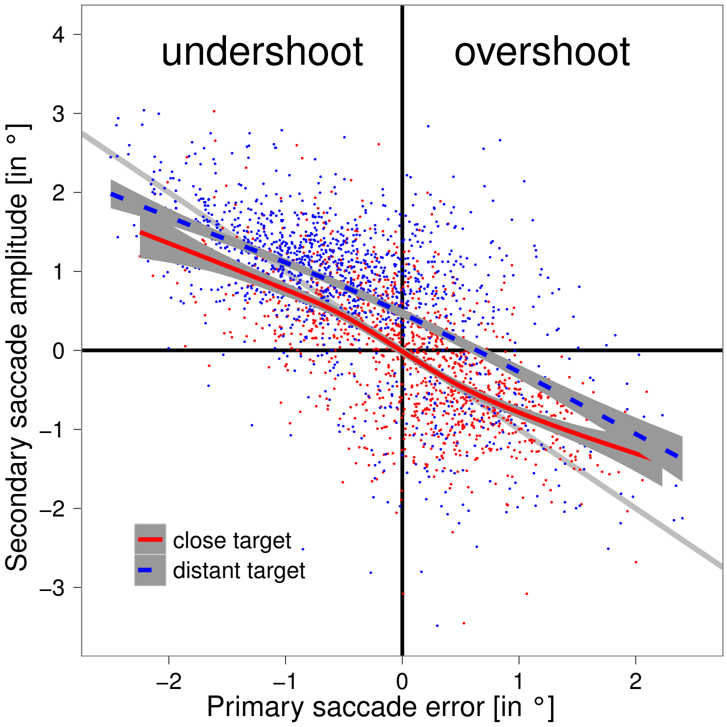

Figure 5.5: Secondary saccade amplitude as a function of primary saccade error and target eccentricity (red solid = close target; blue dashed = distant target) after removing between-subject variance. Negative values of primary saccade error indicate an undershooting primary saccade; positive values are overshoots. Negative values of secondary saccade amplitude indicate secondary saccades in the direction opposite to the primary saccade. Data points include data within a 95% interval of primary saccade error after removing between-subject variance. Grey bands show 95% confidence intervals. The light-grey diagonal indicates saccade amplitude that correct perfectly for primary saccade error.

5.3.5 Secondary saccade error

In the previous sections we found support for the hypothesis that an extra-retinal error signal and target eccentricity significantly influence the generation of secondary saccades. Next we examined whether secondary saccades decrease primary saccade error. Two important aspects need to be kept in mind about secondary saccade error.

First, the observation of secondary saccades that do not decrease primary saccade error is not an argument against the existence of the usage of an extra-retinal error signal. Indeed, a secondary saccade in direction opposite to an overshooting primary saccade is evidence for an extra-retinal signal, but an undershooting secondary saccade may have a larger absolute error than the initial primary saccade overshoot. Second, a large proportion of primary saccades landed precisely on the target. Obviously, secondary saccades following precise primary saccades are likely to increase the distance between eye- and target position.

Figure 5.6 displays secondary saccade error as a function of primary saccade error. Obviously, large distances between eye- and target location persist even after secondary saccades. This result can also be visualized by distributions of primary and secondary saccade error next to each other (see Figure 5.7). In the close target condition primary and secondary saccade error have similar distributions. In the distant target condition, the

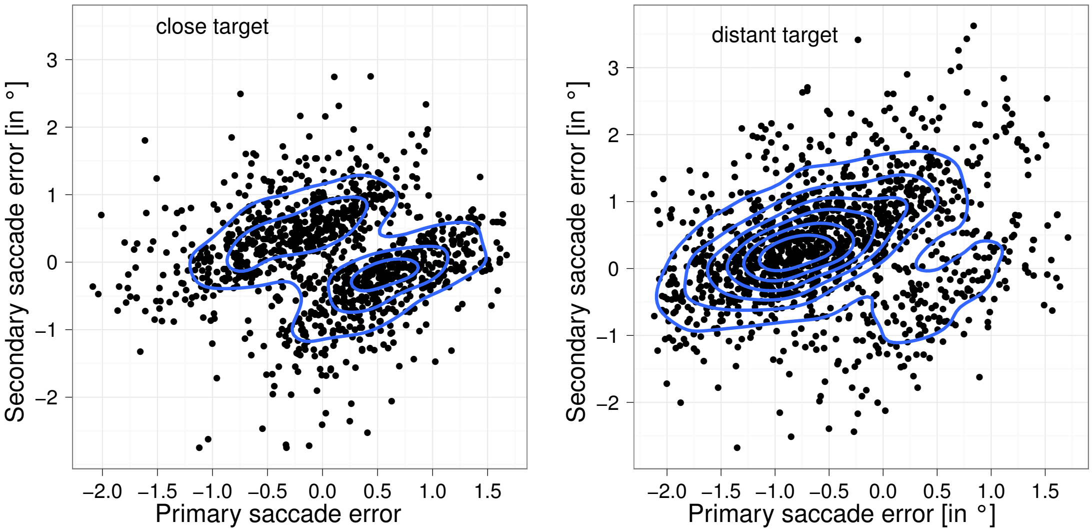

Figure 5.6: Secondary saccade error as a function of primary saccade error and target eccentricity. Individual data points are from within a 99% interval of primary and secondary saccade error. Contour lines (blue, solid) reflect increments of 10%.

Table 5.5: LMM statistics for error after primary vs. secondary saccades.

	Estimate	SE	*t*-Value
Intercept	1.34	0.044	30.6
eccentricity (ecc)	0.09	0.034	3.69
secondary saccade	−0.91	0.038	−23.68
ecc x secondary saccade	0.07	0.046	1.45
Variance components	SD		
Intercept	0.156		
Residual	0.387		

peaks differ for the two distributions: the peak of the primary saccade error distribution indicates many undershooting primary saccades; the distribution of secondary saccade error peaks around 0.

As mentioned above, we did not expect to observe a decreasing error after each secondary saccade, simply because many primary saccades were already quite precise. In Figure 5.8, we plot distributions of primary and secondary saccade error after removal of precise primary saccades; only primary saccades were included with an absolute error larger than 1 degree. Evidently, secondary saccades strongly reduced the error resulting from an imprecise primary saccade.

As a statistical test of this result, we specified an LMM for the effect of primary vs. secondary saccade and close vs. distant target condition on the absolute error, including only trials with imprecise primary saccades with an absolute error larger than 1 degree (see Table 5.5). As expected, absolute secondary saccade error was significantly smaller than absolute primary saccade error (t = −23.68). Initial absolute primary saccade error was larger in the distant as compared to the close target condition (t = 2.69). The interaction was not significant (t = 1.45).

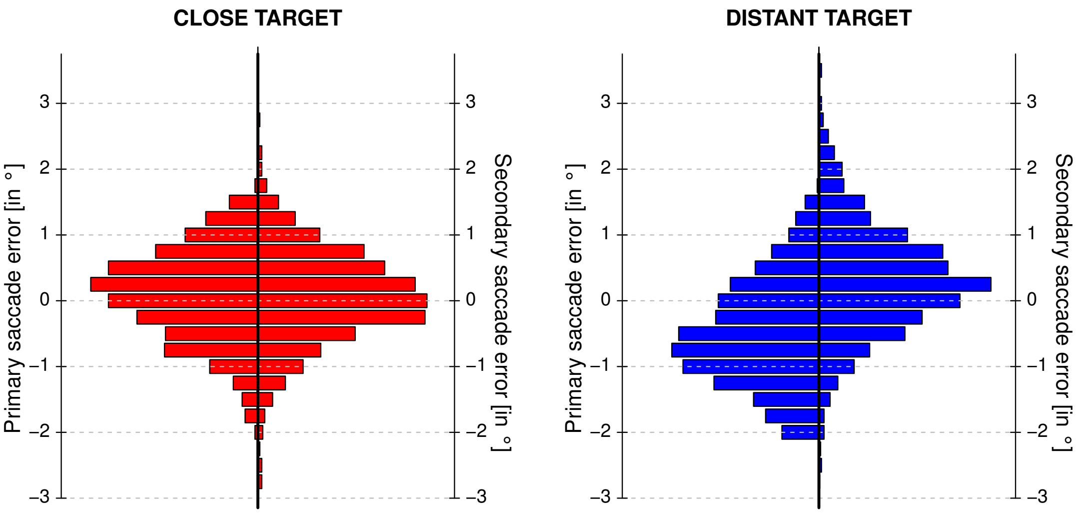

Figure 5.7: Distributions of primary and secondary saccade error for close (red) and distant target (blue) conditions.

5.4 Discussion

In the present study we demonstrated two important influences on the generation of secondary saccades when postsaccadic visual feedback is not available: (1) an extra-retinal error signal and (2) target eccentricity.

Our analyses show that the latency, amplitude and orientation of secondary saccades vary with primary saccade landing position. Increasing error results in faster and larger secondary saccades. The strongest evidence for an extra-retinal influence is found for secondary saccade orientation. A shift in primary saccade landing site from undershoot to overshoot increases the probability that a secondary saccade is generated in direction opposite to the primary saccade. Previous studies that argued for an extra-retinal influence on secondary saccade programming mainly studied very large eccentricities that were accompanied by a large undershoot. Thus, it was not possible to exclude the possibility that a secondary saccade in the same direction as the primary saccade was only due to a bias, in such a way that secondary saccades always follow primary saccade direction. We can now show that increasing overshoot results in secondary saccades of opposite direction.

Although we demonstrated that an extra-retinal signal influences the orientation of secondary saccades, this does not necessarily imply that secondary saccades always reduce absolute primary saccade error. Indeed we show that secondary saccades mainly correct for large primary saccade error. This also means that secondary saccades following rather precise primary saccades can also increase the distance between eye and target position. Nevertheless, the orientation of these eye movements is still influenced by an extra-retinal signal. These results further support the idea that secondary saccades are the result of an activity distribution in a saccadic motor map that can be influenced by multiple factors (e.g., extra-retinal signal, target eccentricity, noise, attention).

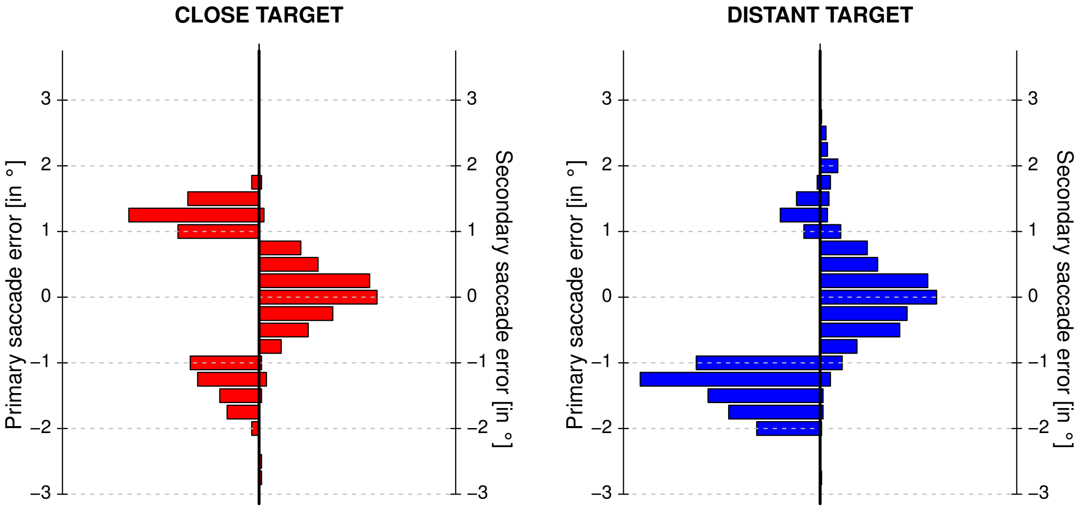

Figure 5.8: Distributions of primary and secondary saccade error after removal of precise primary saccades are shown for close (red) and distant target (blue) conditions.

So far, we strongly argued for an extra-retinal influence on the generation of secondary saccades. Nevertheless, it could also be argued that secondary saccades are pre-programmed along with the primary saccade. Thus, an extra-retinal error signal would not be necessary in order to explain the results in our experiment. But, there exist several findings in the literature that are difficult to reconcile with a pre-programming account. Importantly, fewer secondary saccades are observed when postsaccadic visual feedback is not available (Bonnetblanc & Baraduc, 2007; Prablanc & Jeannerod, 1975; Prablanc, Massé, & Echallier, 1978), highlighting the importance of a visual signal for secondary saccade programming. A strong pre-programming account would predict that the generation of secondary saccades should be independent of postsaccadic visual feedback. Also, a brief presentation of a postsaccadic visual stimulus can easily elicit secondary saccades (Deubel, Wolf, & Hauske, 1982), which again demonstrates the importance of the postsaccadic visual stimulus. In addition, a strong pre-programming account would predict that secondary saccades latency should exhibit small variability. In the present experiment we observed a broad distribution of secondary saccade latency, which provides further support against a strong pre-programming account. Nevertheless, we cannot completely rule out the possibility that a subset of secondary saccades in our experiment was pre-programmed, but in general we think that pre-programming of secondary saccades in a single target step experiment is unlikely.

Apart from the extra-retinal signal we found evidence for an additional influence on the programming of secondary saccades, namely target eccentricity. Secondary saccade amplitude and orientation were significantly different in the close and distant target condition. Secondary saccades to distant targets were more likely to follow the direction of the primary saccade than secondary saccades in the close target condition. This finding was also present in our earlier study with postsaccadic visual feedback (Ohl, Brandt, & Kliegl, 2011). In contrast to our previous study, target eccentricity did not influence secondary saccade latency.

We recently introduced a modified version of the model by Rolfs, Kliegl and Engbert (2008) to account for the generation of secondary (micro-)saccades. This model represents a one-dimensional topographically organized motor map. Very small saccades are generated when activation crosses a certain threshold in the center of the motor map. Increasingly distant locations from the center code for increasingly large saccade amplitude. The left hemisphere programs saccades to the right while the right hemisphere codes for saccades to the left. We proposed that activation in the hemisphere programing the primary saccade is enhanced compared to the opposite hemisphere. Moreover, this hemispheric bias in activation increases with increasing target eccentricity. This model was able to account for our findings that secondary saccades to distant targets were faster, larger, and more likely to follow primary saccade direction. With this mechanism we provided an explanation for eccentricity specific modulations of the latency, amplitude and orientation of secondary saccades.

In the present study, we observed a less coherent picture concerning the results on latency, amplitude, and orientation. Still, the eccentricity-dependent hemispheric bias in activation after primary saccade execution can account for larger secondary saccade amplitude and the bias in secondary saccade orientation with increasing target eccentricity. We proposed that the hemispheric bias could be the consequence of the retinotopic attentional trace (Golomb, Chun, & Mazer, 2008). The retinotopic attentional trace refers to the phenomenon that immediately after a saccade, attention persists at retinotopic coordinates. Attentional facilitation at the retinotopic location might pull secondary saccades in direction of the primary saccade, resulting in a bias of secondary saccades to follow primary saccade direction.

So far we discussed that both an extra-retinal error signal and target eccentricity have a direct influence on the generation of secondary saccades. Nevertheless, the influence of target eccentricity may still be rather indirect and the accuracy of the extra-retinal signal may decrease with increasing target eccentricity (e.g., a systematic underestimation of the extra-retinal signal). Consequently, after a precise primary saccade to a distant target, the underestimated extra-retinal signal would inform the system about a saccadic undershoot and favor secondary saccades in the same direction as the primary saccade. Such a mechanism is in line with our results concerning secondary saccade amplitude and orientation. Secondary saccades in the same direction as the undershooting primary saccade had larger amplitudes in the distant as compared to the close target condition. In addition, we observed an orientation bias for secondary saccades following precise primary saccades to distant targets. Finally, another possible indirect influence of target eccentricity concerns the usage of an extra-retinal signal. Increasing eccentricity could result in a decreasing influence of the extra-retinal signal. Consequently, a general tendency of secondary saccades to follow primary saccade direction would become increasingly dominant with increasing eccentricity.

Obviously, the relationship between saccade landing position and characteristics of secondary saccades as a function of target eccentricity in the present experiment is weaker than previously reported in an experiment with visual feedback (Ohl, Brandt, & Kliegl, 2011). This can be explained by the missing postsaccadic visual stimulation. The lack of visual stimuli during postsaccadic fixation results in a reduced activity in a saccadic motor map and consequently the activity often remained below the critical threshold for saccade triggering. With fewer observations of secondary saccades, it is of course more difficult to detect additional factors that contribute to secondary saccade motor programs.

Now, what might be the role of the extra-retinal signal in our experiment? Generally, in response to target onset a saccade is launched. A copy of the saccade command (efference copy) is available in the oculomotor system (Wurtz, 2008) that includes the information about the oculomotor error (Collins et al., 2009). This copy can now be used to predict the location where the target should be found during postsaccadic fixation. We propose that activation is increased at the predicted target location in a retinotopic motor map for saccades (e.g., superior colliculus). In a paradigm with postsaccadic visual feedback, the visually evoked response in this saccadic motor map can then add to this pre-built activation, causing a rapid triggering of the secondary saccade. This could explain the existence of very short-latency secondary saccades as reported earlier (Bahill, Clark, & Stark, 1975; Becker & Fuchs, 1969). These authors already reasoned that the short-latency secondary saccades were too fast to be triggered by postsaccadic retinal feedback alone.

We want to highlight the potential use of our experimental paradigm for studying non-retinal signals around saccadic eye movements. Visual feedback is not available during postsaccadic fixation therefore systematic influences on the generation of secondary saccades cannot be traced back to postsaccadic visual processing. In future studies secondary saccades may serve as dependent variable to study the influence of non-retinal signals on a postsaccadic motor map. Thus, studying secondary saccades can go beyond a pure interest in their underlying generating mechanisms.

A large proportion of secondary saccades in our study were smaller than one degree of visual angle, therefore meeting the criterion for microsaccades (for recent reviews on microsaccades see Hafed, 2011; Martinez-Conde et al., 2009; Rolfs, 2009). We recently argued that studying microsaccades requires also studying them in a more natural situation, that is, the fixation following a large saccade. We suggest that both microsaccades and the classical secondary saccades share a similar implementation in the oculomotor system which produces small-scale saccades.

The study of microsaccades and secondary saccades has been tackled from very different perspectives. Microsaccades have been shown to counteract visual fading (Engbert & Kliegl, 2004; Martinez-Conde et al., 2006), they are an index of covert attention shifts (Hafed & Clark, 2002; Hafed, Lovejoy, & Krauzlis, 2011; Engbert & Kliegl, 2003; Laubrock, Engbert, & Kliegl, 2005), visual suppression occurs in the superior colliculus around the time of microsaccades (Hafed & Krauzlis, 2010; see also Rolfs & Ohl, 2011). In contrast, secondary saccades are classically thought to exclusively fulfill a corrective function. We have shown that referring to secondary saccades as corrective saccades is only one part of the story as these small postsaccadic eye movements are the result of error-correcting and non-error-correcting factors (e.g., target eccentricity) that influence their generation. It is critical to identify the factors (e.g., saccadic error, target eccentricity) that contribute to the overall activity distribution in a motor map where saccadic eye movements are generated. Studying both microsaccades (during prolonged fixation) and secondary saccades (during postsaccadic fixation) can help to understand how small-amplitude eye movements are implemented in the oculomotor system.

5.5 Conclusion

We provide strong support for the idea that an extra-retinal signal influences motor programs of secondary saccades. Moreover, we demonstrate that target eccentricity can significantly influence the generation of secondary saccades. For future research it will be important to address whether and how these systematic influences in the oculomotor system affect postsaccadic visual perception.

Chapter 6

Postsaccadic location judgments after presentation of multiple target-like objects

6.1 Introduction

Despite constantly moving eyes, and a corresponding shift of the image across the retina, the world is perceived as stable and humans can easily keep track of objects across saccadic eye movements. The perception of a stable visual world is thought to be mediated by an in-built assumption in the visual system that the world does not change during saccadic eye movements; therefore preventing the visual system to notice disturbances of stability.

Interestingly, when a target is shifted in position during the saccade, subjects are typically not aware in which direction the target shift occurred. This effect is labeled saccadic suppression of image displacement (SSID) and has been examined in many studies (Bridgeman, Hendry, & Stark, 1975; Deubel, Schneider, & Bridgeman, 1996). In SSID, a small target shift, which goes unnoticed, is treated as oculomotor error (McLaughlin, 1967) and not as shift of target location.

While SSID suggests that information about the presaccadic target position is lost during the course of the saccadic eye movement, several studies have shown that the information is still available on the motor level, as demonstrated by accurate pointing behavior (Bridgeman et al., 1979; Prablanc & Martin, 1992). Thus, information about the real target position is still available but cannot be accessed for a conscious perceptual decision.

In a series of experiments Deubel et al. (1996) have shown that the information about the presaccadic target location can be restored by inserting a blank between onset of the saccadic eye movement and the appearance of the postsaccadic visual target. Inserting a blank strongly enhances the identification in which direction the target has been shifted during the saccade (blanking effect). Consequently, blanking allowed the information about the real target position to become available again for perception.

What are the assumed mechanisms underlying visual stability around saccadic eye movements? Deubel (2004) presented a modified version of the reference object theory which is based on previous work from that group (Deubel et al., 1996, 1998). This theory assumes a number of sequential processing steps, beginning with target selection during presaccadic fixation. Features of the saccade target are extracted and stored in trans-saccadic memory. Afterwards, the saccade is executed and brings the eyes close to the target location. The incoming visual information at the new fixation location is used for postsaccadic localization judgment. When no visual information is present during early postsaccadic fixation (blanking), postsaccadic localization is veridical. In contrast, when an object is present in the vicinity of the saccade landing site at the beginning of fixation, the object is used as landmark object for space recalibration. Such a landmark object is assumed to be stable across saccades.

These in-built assumptions can cause the percept of a stable landmark and a shifted target which reappears after a blank period although the opposite is true; the landmark object was shifted and the target location was identical between pre- and postsaccadic fixation. Inserting a blanking during early postsaccadic fixation allows recovery of the correct presaccadic location information for perception.

In a recent study, Collins et al. (2009) examined in more detail the necessary information in order to compute a correct prediction of the postsaccadic target location. The authors showed that postsaccadic location judgments did not vary with saccade landing position in a blanking paradigm. Location judgments were dependent on the comparison of the correct presaccadic target with the postsaccadic probe location. Thus, the trial-by-trial variability of saccade landing site had no influence on the location judgments which allowed the authors to conclude that information about the oculomotor error was already included in the efference copy of the saccade. A prediction of the target location is then computed, based on the efference copy and compared to the position of the postsaccadic visual probe. Differences in position between prediction and probe are consequently interpreted as a shift in target position.

Research on postsaccadic location judgments typically study situations by (1) providing a single target without blank which typically results in SSID, or (2) an inserted blank between saccade onset and reappearance of the target, which typically restores the percept of a target shift. In the first case, a target is present during the critical time window during postsaccadic fixation, while in the second case, no information is provided at all.

In the present study, we were interested to examine whether the correct presaccadic target location can be restored by other means than inserting a blanking period. To this end we examine the influence of ambiguous information presented during the early critical time period of postsaccadic fixation. At postsaccadic fixation onset we present a screen filled with multiple target-like objects side-by-side (see Figure 6.1). The participants were asked to identify the postsaccadic probe that matched the correct presaccadic target location. This experiment differs in two major aspects from the classical paradigm that studies postsaccadic location judgments. First, immediate postsaccadic information consists of a screen with multiple target-like objects (ambiguous information), and second, in contrast to a 2-AFC (whether the target shift occurred to the left or right), subjects could decide between many possible target locations.

What information may be used in order to identify the correct target in our task? Subjects made a response saccade to the target. This response saccade is associated with some oculomotor error. Nevertheless, the information about the oculomotor error will be already included in the efference copy of the saccade. This allows to derive a precise prediction of where the target should be located right after the saccade. Presentation of the multiple target-like object screen may now have different consequences.

First, similar to the classic effect of SSID, the multiple target-like object screen may overwrite the precise prediction of the target location. Right after the saccade, a target-like object in the vicinity of the saccade end point is accepted as spatial landmark and then used for postsaccadic space recalibration. Importantly, this target-like object has identical features as the presaccadic target and may thus be judged as the target. Consequently, location judgments should primarily depend on the initial saccade landing position.

Second, the presentation of multiple target-like objects and the presentation of a blanking period have potentially one thing in common: the presaccadic target cannot be identified immediately during postsaccadic fixation and may thus disturb the process of choosing a landmark. Now assuming that the presentation of a multiple target-like object screen during a critical time period after saccade end acts similar to a blanking period, in such a way that choosing a landmark is disturbed and the information about presaccadic target location is restored, we expect very precise postsaccadic location judgments.

Perception of a stable visual world is not exclusively determined by the inability to detect a shift in target position across saccades. Of course, it is also essential that subjects are very confident that they made a correct response. In our present study, we were further interested in determining what factors influence a subject's confidence associated with the location judgment. To this end, subjects had to indicate after each location judgment how confident they were in identifying the correct target location. Thus, our paradigm allows us to study the influence of saccade landing position, location judgment, and postsaccadic oculomotor behavior (e.g., secondary saccades) on the confidence associated with the location judgment.

In summary, the present study seeks to identify the information that is used to judge the location of a presaccadic target during postsaccadic fixation when faced with multiple target-like objects. Moreover, we focus on the confidence associated with each location judgment.

6.2 Experiment 1

6.2.1 Materials and methods

6.2.1.1 Participants

Twelve undergraduate students (9 female, 3 male) from the University of Potsdam participated in this experiment. They were paid 7 Euros or received study credit for participation. Their age ranged from 21 to 44 years (M = 25). All participants had normal or corrected-to-normal vision. The experiment conformed to the Declaration of Helsinki and we obtained written informed consent from all participants prior to the experiment.

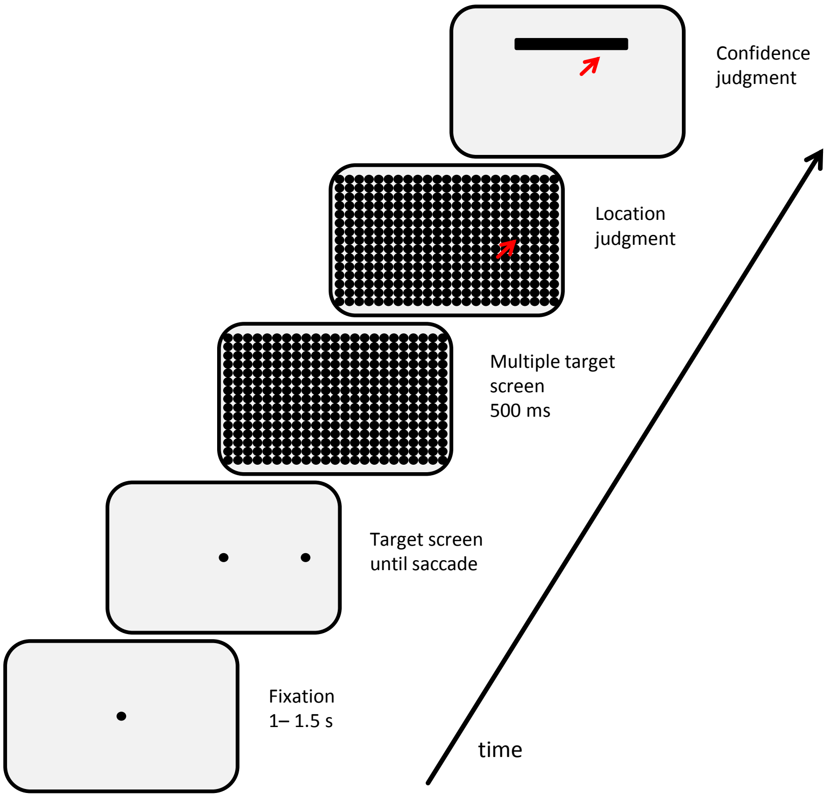

Figure 6.1: Event sequence in experimental trial After initial fixation, a target was presented on the horizontal meridian. Subjects were required to move their eyes immediately to the new target location. After the eyes crossed an invisible boundary, a screen filled with multiple target-like objects was presented. Subjects had to indicate the position of the presaccadic target by locating the mouse over one of the target-like objects. The location judgment was followed by a subsequent confidence judgment.

6.2.1.2 Materials and procedure

Participants were seated 50 cm in front of a computer screen, with their head positioned on a chin rest. The experiment was conducted in a silent, dark room. Visual stimuli were displayed on a 19-in. EYE-Q 650 CRT monitor at a resolution of 1024 x 768 pixels and a refresh rate of 100 Hz. The experiment ran on an Apple Power Macintosh G4 computer, using Matlab (Mathworks, Natick, MA, USA) and the Psychophysics toolbox (Brainard, 1997; Pelli, 1997) for control of the stimulus display and response collection. Eye movements were recorded using the EyeLink-II system (SR Research, Osgoode, Ontario, Canada) with a sampling rate of 500 Hz. For control of the EyeLink II system we used the Eyelink toolbox (Cornelissen, Peters, & Palmer, 2002).

Before the first and every 30 trials we conducted a nine-point calibration in order to align eye and screen coordinate systems. At the beginning of each trial, subjects fixated a white point (0.58°diameter of visual angle) in the center of the screen, presented on a grey background. After a random interval of 1000-1500 ms, the central fixation point was removed and a white target point (0.58°diameter of visual angle) was simultaneously presented on the horizontal meridian at a distance of 7.16°, 7.88°or 8.59°of visual angle to the left or right of fixation. Subjects were instructed to move their eyes immediately to the new target location and fixate the target.

When the eyes crossed the border of an invisible square (center-to-border distance of 2°) located around the fixation point, a screen was presented, filled with multiple target-like objects. This multiple target-like object screen consisted of 2961 disks (63 horizontal x 47 vertical) that looked identical to the presaccadic target. 1000 ms after target onset, a mouse cursor was presented at a random location within a distance of 2°around the current eye position. The task was now to identify the presaccadic target location by pressing the mouse button when the cursor was located over a target-like object. The chosen target was enlarged for 200 ms, which ensured feedback that subjects carefully pressed the mouse button.

Following the location judgment, a white rectangle (width: 13.43°, height 2.24°) was presented 6.71°above the position of the initial fixation point. Subjects were asked to report how confident they were with their location judgment; meaning how confident they were that they identified the correct presaccadic target position. A mouse click close to the left border of the rectangle indicated *not confident* and close to the right border *very confident*. After an inter-trial interval of 500 ms a new trial was initiated.

Before trial beginning, fixation position was checked and the trial only continued when the eyes were within a distance of 1.5°around the fixation point. When fixation check failed, a drift correction was applied. When fixation check failed after a drift correction had been applied, new calibration and subsequent validation procedures were run. Blinks during the interval between presentation of the fixation point and presentation of the mouse cursor resulted in trial abort. Those trials were presented again in random order at the end of the experiment. Subjects were allowed to blink after presentation of the mouse cursor. Each subject was tested on 8 training trials and 220 test trials.

6.2.1.3 Data preparation and analysis

Response saccades were defined as the first saccadic eye movement within a distance smaller than 3.5°between center of the target and saccade end point. When an eye movement larger than 2°occurred prior to this response saccade, the trial was not included in the analysis. Furthermore, we included only trials with response saccades having a latency between the minimum of 80 ms and a maximum of 500 ms after target presentation.

For detection of (micro-)saccades we used an improved version (Engbert & Mergenthaler, 2006) of the algorithm reported by Engbert and Kliegl (2003). In this algorithm eye movement trajectories are transformed into 2D velocity space and a threshold for peak velocity (6 SD) and a minimum duration of 4 samples (8 ms) were applied to identify (micro-)saccades. In total we identified 2375 response saccades (135-220 per subject, median = 204.5) with subsequent localization and confidence judgment. Thus, 90% of all trials were included after applying our detection criteria.

Primary saccades are frequently followed by secondary saccades (Ohl, Brandt, & Kliegl, 2011, 2013). In our study, secondary saccades were only analyzed when they occurred in the interval between the end of the response saccade and appearance of the mouse cursor (for the localization judgment). This resulted in a time window covering the first 500 ms after the end of the saccade. In total we observed 1339 secondary saccades (11-203 per subject, median = 129). Based on the angular orientation, secondary saccade direction was categorized as either following the direction of the response saccade or in opposite direction to the response saccade.

Screen coordinates of the confidence judgments were collected. The horizontal screen coordinates were mapped onto continuous values ranging from 0 (not confident) to 1 (very confident). Linear mixed models (LMM) were used for statistical analyses. We used the R environment (R Development Core Team, 2011) with the additional *lme4* (Bates and Maechler, 2010) and *ggplot2* (Wickham, 2009) packages.

6.2.2 Results

6.2.2.1 Oculomotor behavior

Response saccades had a mean latency of 178 ms (SD = 52.44). The mean saccade landing position indicated a slight undershoot ($M = -0.29$; $SD = 0.66$). In Figure 6.2, secondary saccade statistics are shown. First, the distribution of secondary saccade latency is bimodal (see Figure 6.2, top left), where the early peak of the latency distribution can be solely attributed to secondary saccades from one single subject. Second, most of secondary saccades were indeed smaller than one degree of visual angle, thus meeting the criterion of microsaccades (see Figure 6.2, top right).

We analyzed the influence of primary saccade error on the latency, amplitude and orientation of secondary (micro-)saccades (see Figure 6.2). In our LMM we included the linear and square saccadic error in order to assess the influence of primary saccade landing position on secondary saccade latency. Indeed, we found a statistically significant influence of linear saccadic error (beta = 21.87, t = 4.99), but not square saccadic error (beta = -3.97, t = -0.95) on secondary saccade latency. Thus, shifting the landing position from undershoot to increasing overshoot increased the latency of secondary saccades (see Figure 6.2, bottom left).

Alike for secondary saccade latency, we included the linear and square saccadic error in our LMM to account for secondary saccade amplitude. The linear saccadic error (beta = -0.08, t = -1.67) did not significantly influence the amplitude of secondary saccades. In contrast, the amplitude was significantly influenced by square saccadic error (beta = 0.18, t = 5.86; see Figure 6.2, bottom center). We ran a GLMM to determine the influence of saccadic error on the direction of secondary saccades. Primary saccade error (beta = 2.39, $p < 0.001$) significantly influenced the direction of secondary saccades. While undershooting was associated with secondary saccades in the same direction as the primary saccade, increasing overshoot resulted in small eye movements in direction opposite to the primary saccade (see Figure 6.2, bottom right).

To sum up, we have some evidence that the generation of secondary saccades was influenced by primary saccade error, which suggests that an extra-retinal error signal influenced secondary saccades in this experiment. Although the orientation and amplitude of secondary saccades were significantly dependent on primary saccade landing position, there was no clear effect on secondary saccade latency.

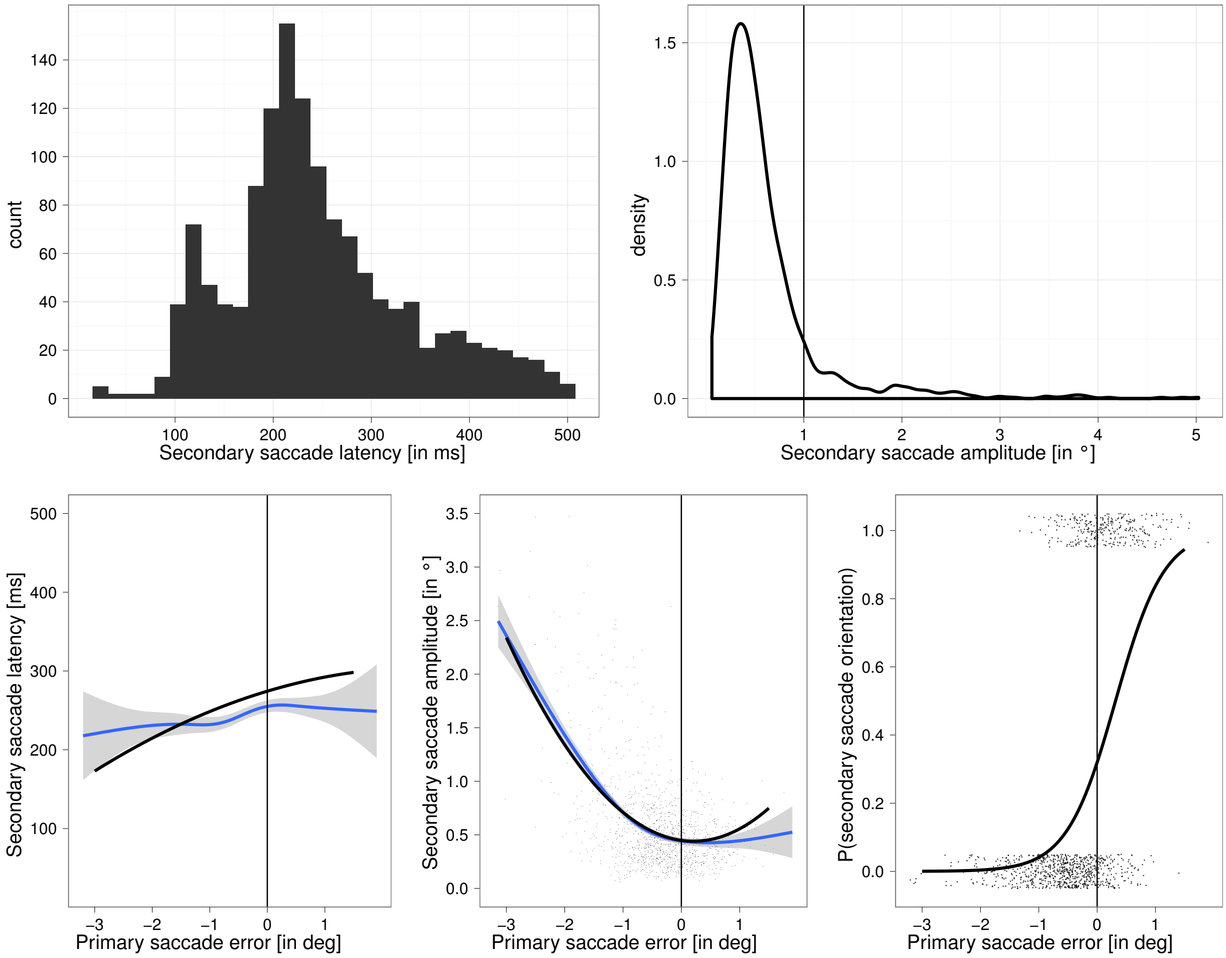

Figure 6.2: Secondary saccade characteristics. **Top left**, distribution of secondary saccade latency. **Top right**, distribution of secondary saccade amplitude. **Bottom left**, secondary saccade latency as function of primary saccade error. **Bottom center**, secondary saccade amplitude as function of primary saccade error. **Bottom right**, secondary saccade orientation as function of primary saccade error.

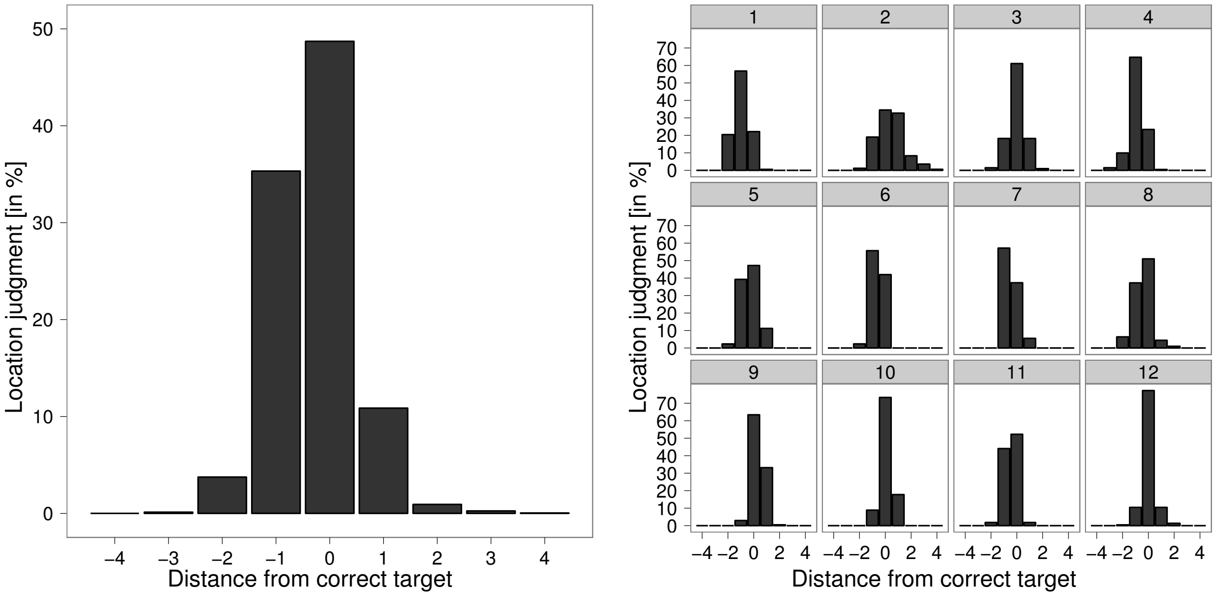

Figure 6.3: Left, postsaccadic location judgments depicted as distance from the correct target postion. Negative values indicate location judgements between the center of the screen and the correct target location. **Right**, distribution of postsaccadic location judgments for each individual subject.

6.2.2.2 Location judgments

In Figure 6.3, we see the overall distribution of location judgments along with the distribution of location judgments for each individual subject. While for the majority of location judgments the correct target was identified, we see that in a large proportion of judgments also the object next to the correct target location was chosen. In general, 51% of the judgments did not identify the correct target.

Figure 6.4 (top left) shows the influence of primary saccade landing site on the subsequent location judgment for each individual subject. We see some dependency of location judgment on primary saccade error. Importantly, the distribution of location judgments consists of two subpopulations. Location judgments can be either preceded by a secondary saccade or not. Figure 6.4 (top right) shows the dependency of location judgments on saccadic error for saccades that are not followed by a secondary saccade. In contrast, Figure 6.4 (bottom left) shows the influence of saccadic error on location judgments for saccades that were followed by a secondary saccade. A first inspection of Figure 6.4 (top right vs. bottom left) suggests a stronger influence of primary saccade error on location judgments when no secondary saccade was observed.

We were interested to quantify this result with a LMM including the covariates of saccadic error, whether a secondary saccade occurred (coding: 0 = without secondary saccade, 1 = with secondary saccade) and the interaction between these two covariates (model 1, see Table 6.1). In model 1 we found a significant effect of primary saccade error ($t = 12.07$). Thus, we see that location judgments depend on the landing site of the primary saccade which speaks against the idea that location judgments might be independent of oculomotor error in our paradigm. The effect of primary saccade error was significantly weaker when a secondary saccade occurred (indicated by the significant interaction, $t = -8.56$). This highlights the importance of small eye movements during

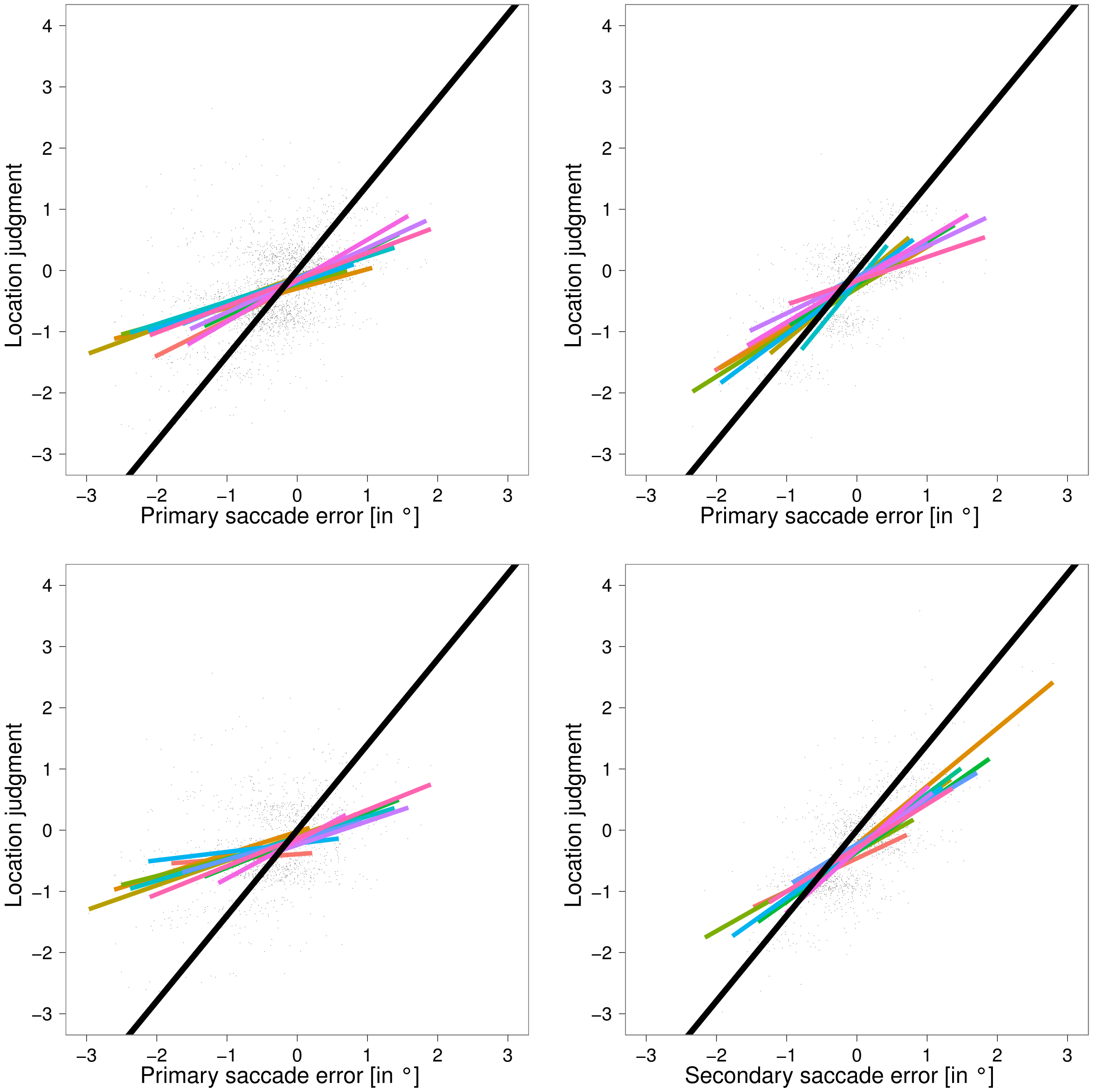

Figure 6.4: Postsaccadic location judgment as function of primary saccade error. The thick black line depicts the prediction when postsaccadic location judgments can be predicted by saccade landing position. Colored lines depict results of individual subjects. **Top left**, shows results for the comlete population of postsaccadic location judgments. **Top right**, shows results for postsaccadic location judgments when no secondary saccade was observed. **Bottom left**, shows results for postsaccadic location judgments when secondary saccades were observed. **Bottom right**, postsaccadic location judgments as function of secondary saccade error in trials which included secondary saccades.

Table 6.1: LMM statistics for postsaccadic location judgments.

	Estimate	SE	*t*-Value
Intercept	−0.206	0.065	−3.17
saccadic error (sac.error)	0.703	0.058	12.07
secondary saccade (sec.sac)	0.071	0.08	0.9
Sac.error x sec.sac	−0.373	0.044	−8.56
Variance components		SD	
Subject:			
Intercept		0.21	
Saccadic error		0.16	
Secondary saccade		0.25	
Residuals		0.58	

Table 6.2: LMM statistics postsaccadic location judgments for the subset when secondary saccades were observed.

	Estimate	SE	*t*-Value
Intercept	−0.327	0.08	−4.09
saccadic error (sac.error)	−0.11	0.055	−2
secondary saccade error (sec.sac.error)	0.857	0.078	11
sac.error x sec.sac.error	−0.014	0.035	−0.4
Variance components		SD	
Subject:			
Intercept		0.26	
saccadic error		0.14	
secondary saccade		0.21	
Residuals		0.53	

postsaccadic fixation for the present location judgment task. Whether or not a secondary saccade occurred did not further influence the location judgment (t = 0.9).

Location judgments that were preceded by a secondary saccade can be influenced by both primary and secondary saccade error (i.e., the error remaining after primary or secondary saccade). A first, comparison of Figure 6.4 (bottom left and bottom right) suggests that location judgments varied strongly with secondary saccade error. In model 2 (see Table 6.2), we ran a LMM including the error related to the primary saccade, the error related to the secondary saccade, and their interaction. We observed a small effect of primary saccade error (t = −2) which was in opposite to the expected direction. Moreover, there was a strong effect of secondary saccade error (t = 11), showing that location judgments varied strongly with the landing position of the secondary saccade. The interaction between the two main factors did not reach significance (t = −0.4). Overall, primary saccade landing site had a strong influence on location judgment when no secondary saccade was observed. This results contradicts our hypothesis that location judgments may depend on a strong extra-retinal mechanism in our paradigm.

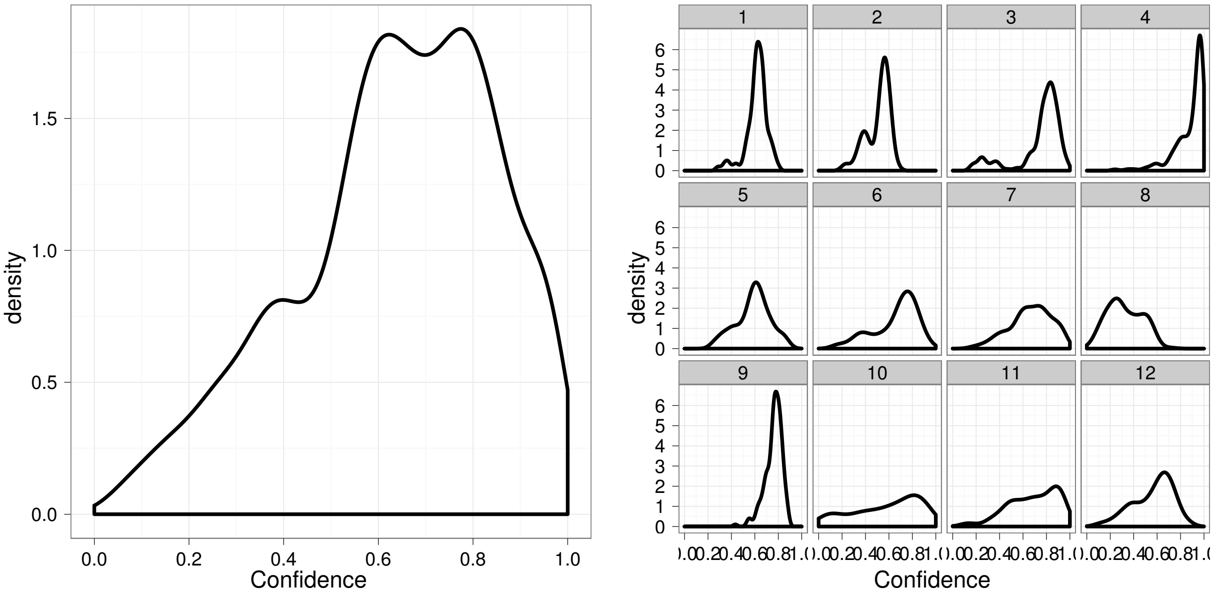

Figure 6.5: Left, confidence density associated with location judgments. Values to the right (close to 1) indicate very confident judgments while values close to 0 indicate judgments that were *not confident*. **Right**, confidence density for each individual subject.

6.2.2.3 Confidence

The distribution of confidence judgments is displayed in Figure 6.5. Inspection of the distribution indicated that confidence judgments varied over the whole range of possible values ranging from 0 to 1. Moreover, we observed that the density had a peak close to the right border, indicating that subjects were in general rather confident in their decisions. Examining individual distributions showed that confidence judgments varied strongly between subjects. On the one hand side, there were subjects showing high confidence (e.g., subject 4 and 9), while other subjects had a very broad distribution (e.g., subject 10).

In our LMM (model 3) we examined the influence of primary saccade error (linear and square), location judgment in terms of distance from chosen minus correct target position (linear and square) and the influence of occurrence of a secondary saccade on the confidence judgment (see Table 6.3). The only variable that exerted a significant influence on the confidence was the square location judgment ($t = -2.8$). Thus, increasing distance between real and chosen target resulted in decreasing confidence. This result is very interesting as it suggests that confidence is influenced by an extra-retinal prediction of the correct target location which, at the same time, does not influence the location judgment.

6.2.3 Discussion

In this experiment we observed that a large proportion of location judgments were wrong. This speaks against the idea that a screen with multiple target-like objects may, similar to a blank screen, allow access to the information of the presaccadic target location. Furthermore, we observed that location judgments were dependent on the landing position of the primary saccade, but only under the condition when no secondary saccade was generated. In contrast, when also a secondary saccade was observed, location

Table 6.3: LMM statistics of confidence associated with postsaccadic location judgments.

	Estimate	SE	*t*-Value
Intercept	0.658	0.041	15.93
sac.error linear	0.011	0.012	0.89
sac.error square	−0.016	0.01	−1.64
judgment error linear	−0.011	0.011	−0.94
judgment error square	−0.019	0.007	−2.8
secondary saccade	−0.01	0.013	−0.74
Variance components		SD	
Subject:			
Intercept		0.14	
sac.error linear		0.03	
sac.error square		0.03	
judgment error linear		0.03	
judgment error square		0.02	
secondary saccade		0.03	
Residuals		0.16	

judgments varied strongly with the landing position of the secondary saccade. Analyses of secondary saccade characteristics indicated that the information of primary saccade error was available to the oculomotor system.

Interestingly, we also observed an influence of presaccadic target location on confidence judgments, which indicates that subjects had at least some access to the correct presaccadic target location. In the next step, we are interested in examining whether inserting a blank, which is followed by a multiple target-like object screen, can cause improved location judgments as compared to a no-blank condition (see Figure 6.6). In the first experiment, we observed that for a few subjects even the vertical location judgment turned out to be erroneous in some cases. Therefore we decided to use a simpler target array in experiment 2, which allows us to exclude the possibility of vertical judgment errors. In experiment 2, multiple target-like objects were arranged in one horizontal line. Moreover, we tested location judgments by comparing a blank vs. no-blank condition.

6.3 Experiment 2

6.3.1 Materials and methods

6.3.1.1 Participants

Twelve subjects participated in two session of experiment 2. All of them reported normal or corrected-to-normal vision. Participants were paid 7 Euros per session or they received study credit. The experiment was in accordance with the declaration of Helsinki and all participants gave written informed consent to the experiment prior to participation.

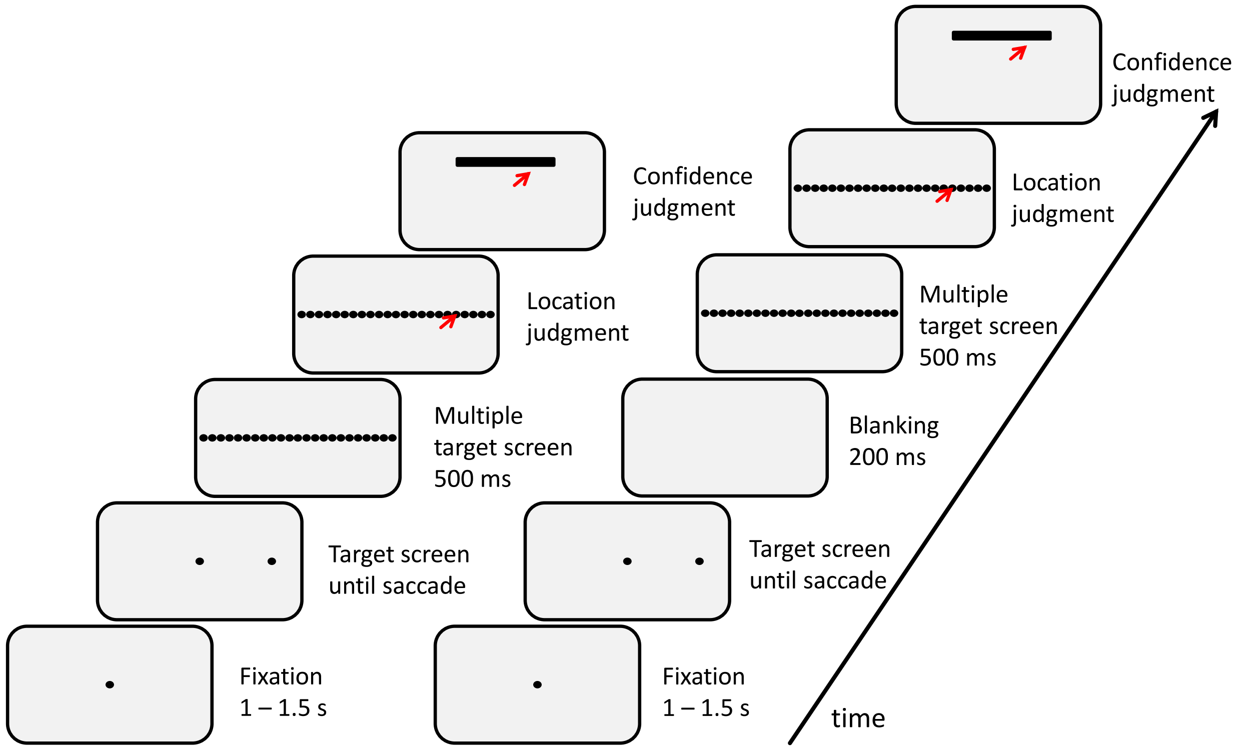

Figure 6.6: Event sequence in experimental trial After an initial period of fixation, a target was presented on the horizontal meridian. Subjects were required to move their eyes immediately to the new target location. After the eyes crossed an invisible boundary, visible input was changed. Depending on condition, either a screen filled with one horizontal line of multiple target-like objects was presented or an additional blank for 200 ms was inserted before presentation of multiple target-like objects. Subjects had to indicate the position of the presaccadic target by locating the mouse over one of the target-like objects. The location judgment was followed by a subsequent confidence judgment.

6.3.1.2 Materials and procedure

Apart from two aspects, experiment 2 is identical to the previous experiment. First, the multiple target-like object screen was modified. In experiment 1, a screen filled with white disks was presented. In experiment 2, a screen with one horizontal line full of target-like objects is presented. Thus, the vertical component of the location judgments can no longer be wrong. Second, in experiment 2 we examined postsaccadic location judgments also in a blanking condition. In this condition, a blank screen was presented for 200 ms following right after fixation onset. Right after the blank, the multiple target-like objects screen was presented. Each subject was tested in the blank and no-blank condition in two separate sessions; each session comprising 8 training and 220 test trials.

6.3.2 Results

6.3.2.1 Oculomotor behavior

Mean saccade latency in the blank condition was 201 ms (SD = 68.9) and 209 ms (SD = 78.8) in the no-blank condition. Furthermore, saccadic accuracy was $-0.21°$(SD = 0.62) in the blank, and $-0.18°$(SD = 0.61) in the no-blank condition. Inspection of Figure 6.7 shows large differences in the distribution of secondary saccade latency between the blank and no-blank condition. Indeed, the number of secondary saccades was strongly decreased during the blank interval, which is line with results from studies that examined secondary saccades without postsaccadic visual feedback (Ohl, Brandt, & Kliegl, 2013).

Again, we ran LMMs in order to establish a possible relationship between primary saccade error and secondary saccade characteristics (e.g., latency, amplitude and orientation) as a function of the experimental condition (blank vs. no-blank). In Figure 6.7, we see the influence of primary saccade error on secondary saccade latency. A LMM revealed that secondary saccade latency was significantly increased by inserting a blank (beta = 70.37, t = 6.43). Neither linear (beta = -5.07, t = -1.6) nor square saccadic error (beta = -0.76, t = -0.17) significantly influenced secondary saccade latency. In addition, a significant interaction of the experimental condition (blank vs no-blank) x square saccadic error had a significant influence (beta = -12.17, t = -3.21) on secondary saccade latency. Increasing primary saccade error was associated with a decrease in secondary saccade latency in the blank condition.

We used the same predictors as in the previous analysis of secondary saccade amplitude (see Figure 6.7). Square saccadic error was the only variable exerting a significant influence on secondary saccade amplitude (beta = 0.097, t = 2.81). Increasing error was associated with larger secondary saccades. This influence was not significantly different for the blank and no-blank condition (beta = 0.026, t = 1.01). Thus, we replicate the findings of our first experiment that increasing error was associated with increasingly large secondary saccades.

Finally, we determined the influence of primary saccade error and blank vs. no blank on the direction of secondary saccades. We observed a significant effect of saccadic error (beta = 1.27, p = 0.001). Increasing overshoot resulted in an increasing number of secondary saccades that were directed in direction opposite to the primary saccade. Again, we can replicate the finding from experiment 1 which supports the view that information about the correct target location, in form of an extra-retinal error signal, was available to the oculomotor system. In addition, we observed a significant main effect of the blanking (beta = -0.759, p = 0.034). There was a significant bias to follow the direction of the primary saccade in the blank condition. This result is suprising because in the blanking condition we expected the correct target location to become available for the perceptual decision. Consequently, we predicted a stronger error-correcting influence in the blanking condition as compared to the no-blank condition.

6.3.2.2 Location judgments

In Figure 6.8, the distribution of postsaccadic location judgments for both the blank and no-blank condition is presented. Both distributions peak at a distance of 0 which represents the correct location judgment. Nevertheless, in the no-blank condition 62% and in the blank condition 58% of judgments were wrong. The number of correct location judgments was not significantly different between the blank and no-blank condition as revealed by a paired t-test (t(10) = 1.32; p = 0.216), although a decreasing number of errors in the blank condition was in the expected direction.

In Figure 6.9, we display the dependency of postsaccadic location judgments on error in landing position, the occurence of secondary saccades, and the blank vs. no-blank

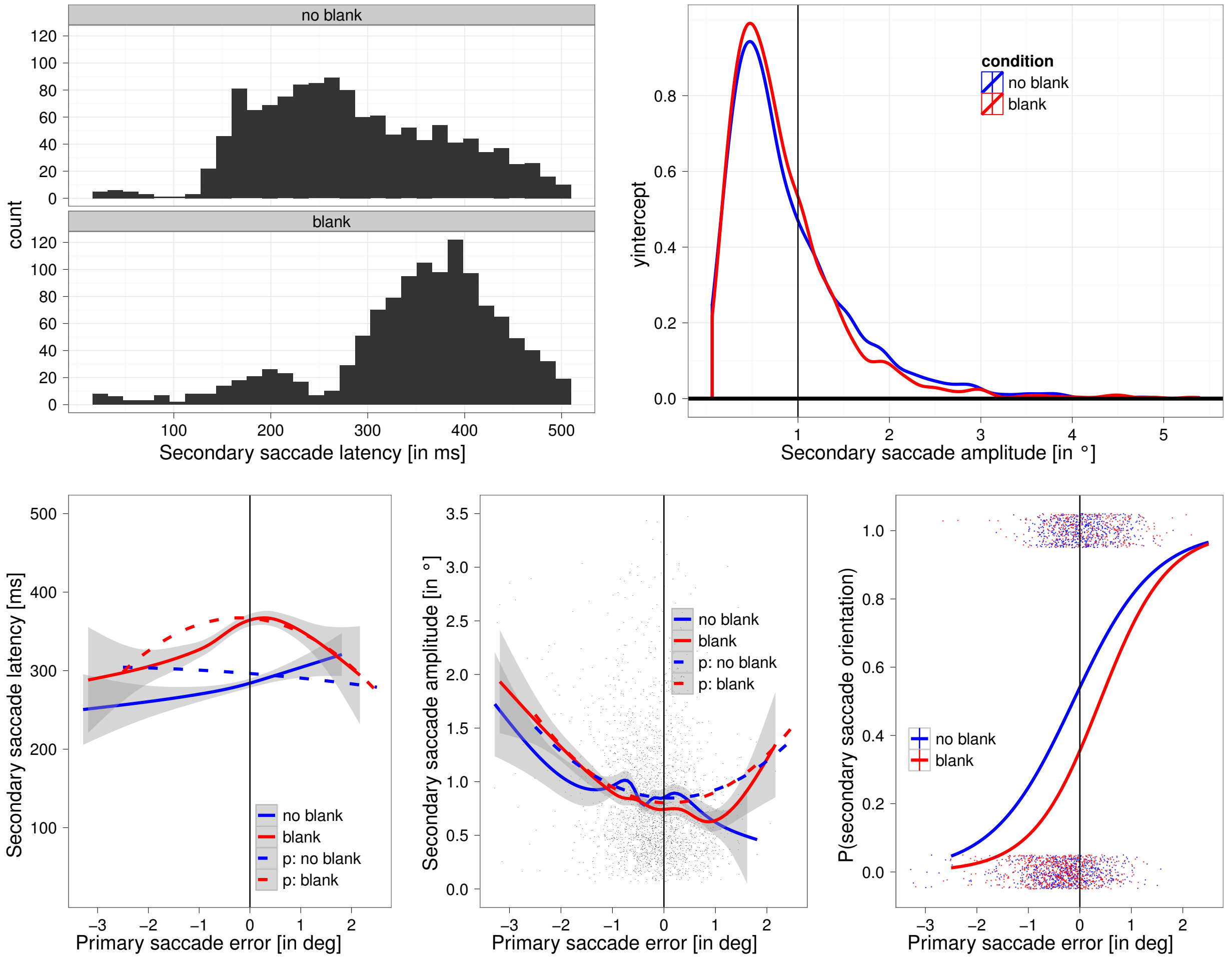

Figure 6.7: Secondary saccade characteristics. **Top left**, distribution of secondary saccade latency as function of blank vs. no blank. **Top right**, distribution of secondary saccade amplitude as function of blank (blue) vs no blank (red). **Bottom left**, secondary saccade latency as function of primary saccade error and as function of blank (blue) vs no blank (red). **Bottom center**, secondary saccade amplitude as function of primary saccade error and as function of blank (blue) vs no blank (red). **Bottom right**, secondary saccade orientation as function of primary saccade error and as function of blank (blue) vs no blank (red).

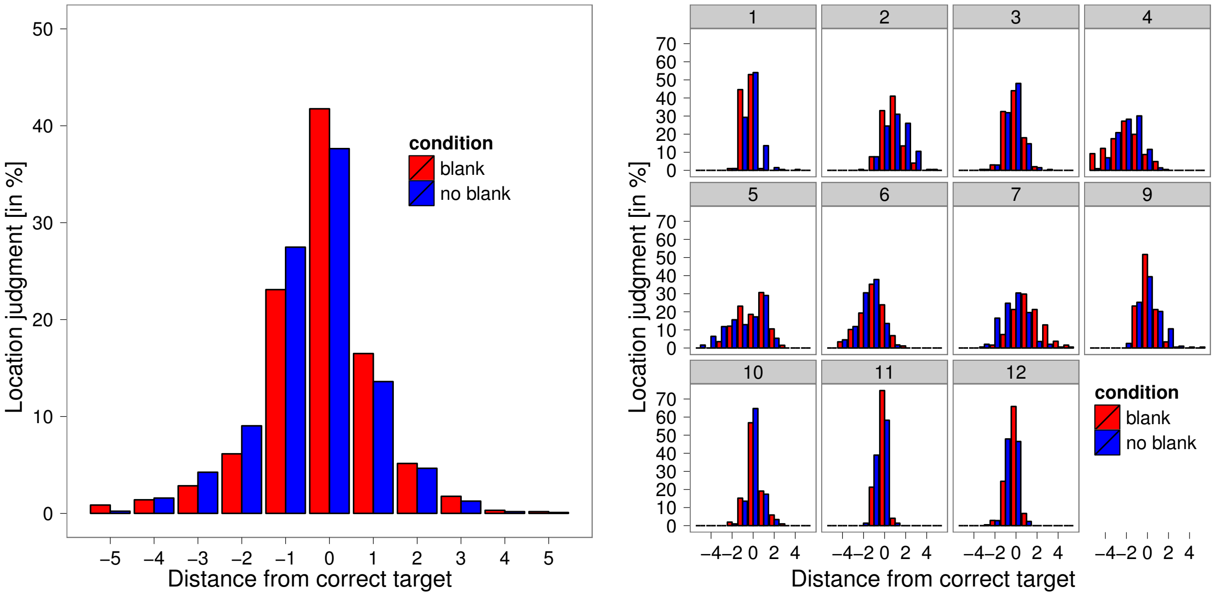

Figure 6.8: **Left**, postsaccadic location judgments as function of blank (red) vs. no blank (red), depicted as distance from the correct target postion. Negative values indicate location judgements between the center of the screen and the correct target location. **Right**, distribution of postsaccadic location judgments for each individual subject as function of blank (red) vs. no blank (blue).

condition. In our LMM (model 4, see Table 6.4), we used the same predictors as in model 1 of experiment 1, and included the additional main factor of the experimental condition (blank vs no-blank), as well as the interactions associated with the experimental condition. We observed a significant effect of primary saccade error on location judgments (t = 5.22). While the main effects of inserting a blank (t = 0.64), and the generation of a secondary saccade (t = −0.11) did not significantly influence location judgments, we observed a significant interaction between the two variables (t = 2.41). Location judgments were significantly better when secondary saccades were observed in the blank condition. One additional significant interaction was observed between saccadic error and the presence of a secondary saccade (t = −5.08). Alike in experiment 1, the influence of primary saccade error on location judgments was significantly reduced in the presence of a secondary saccade.

In model 5 (see Table 6.5), we examined the influence of error related to the primary saccade, error related to the secondary saccade, both in the blank and no-blank condition on location judgments for the subpopulation of trials with secondary saccades. Alike in experiment 1, we found a weak effect of primary saccade error in the no-blank condition which was in opposite to the expected direction (t = −2.55). In the blank condition, this effect was significantly reduced (t = 2.38). Also, as in experiment 1, there was a strong correspondence between secondary saccade error and location judgments in the no-blank condition (t = 9.55). This effect was not significantly different in the blank condition (t = −1.67). Thus, when secondary saccades were observed, location judgments were in line with the landing site of the secondary saccade. In trials without secondary saccades, primary saccade error largely predicted the subsequent location judgment. Surprisinlgy,

Table 6.4: LMM statistics for postsaccadic location judgments.

	Estimate	SE	*t*-Value
Intercept	−0.359	0.134	−2.67
saccadic error (sac.error)	0.752	0.144	5.22
secondary saccade (sec.sac)	−0.02	0.177	−0.11
blank	0.106	0.165	0.64
sac.error x sec.sac	−0.353	0.07	−5.08
sac.error x blank	0.029	0.079	0.37
sec.sac x blank	0.148	0.061	2.41
sac.error x sec.sac x blank	−0.076	0.097	−0.79
Variance components		SD	
Subject:			
Intercept		0.43	
saccadic error		0.44	
secondary saccade		0.57	
blank		0.53	
Residuals		0.94	

in our paradigm the blanking did not exert a strong influence on location judgments (e.g., only a small trend was observed).

6.3.2.3 Confidence

In Figure 6.10 (left panel), we show the distribution of confidence for the blank and no-blank condition. Alike in experiment 1, there was an overall tendency of confident judgments. Individual confidence distributions (Figure 6.10, right panel) were very similar between the blank and no-blank condition. Nevertheless, there were large individual differences, ranging from very confident subjects (i.e., subject 7) to subjects that were not confident at all (i.e., subject 1).

In our LMM (model 6, see Table 6.6) we included primary saccade error (linear and square), the distance from the chosen target position minus correct target position (linear and square) and the occurrence of a secondary saccade in order to account for the confidence associated with a location judgment. In addition, we included the experimental condition (blank vs. no-blank), as well as the interactions with the above mentioned terms as predictors in our model. Alike in experiment 1, the only variable that exerted a significant influence on the confidence was the square location judgment (t = 3.27). Increasing distance between location judgment and real target position was associated with a decrease in confidence.

6.3.3 Discussion

In experiment 2, we replicated that a large proportion of location judgments did not indicate the correct target location. Moreover, we have shown a strong relationship between location judgments and saccade landing position in trials without secondary

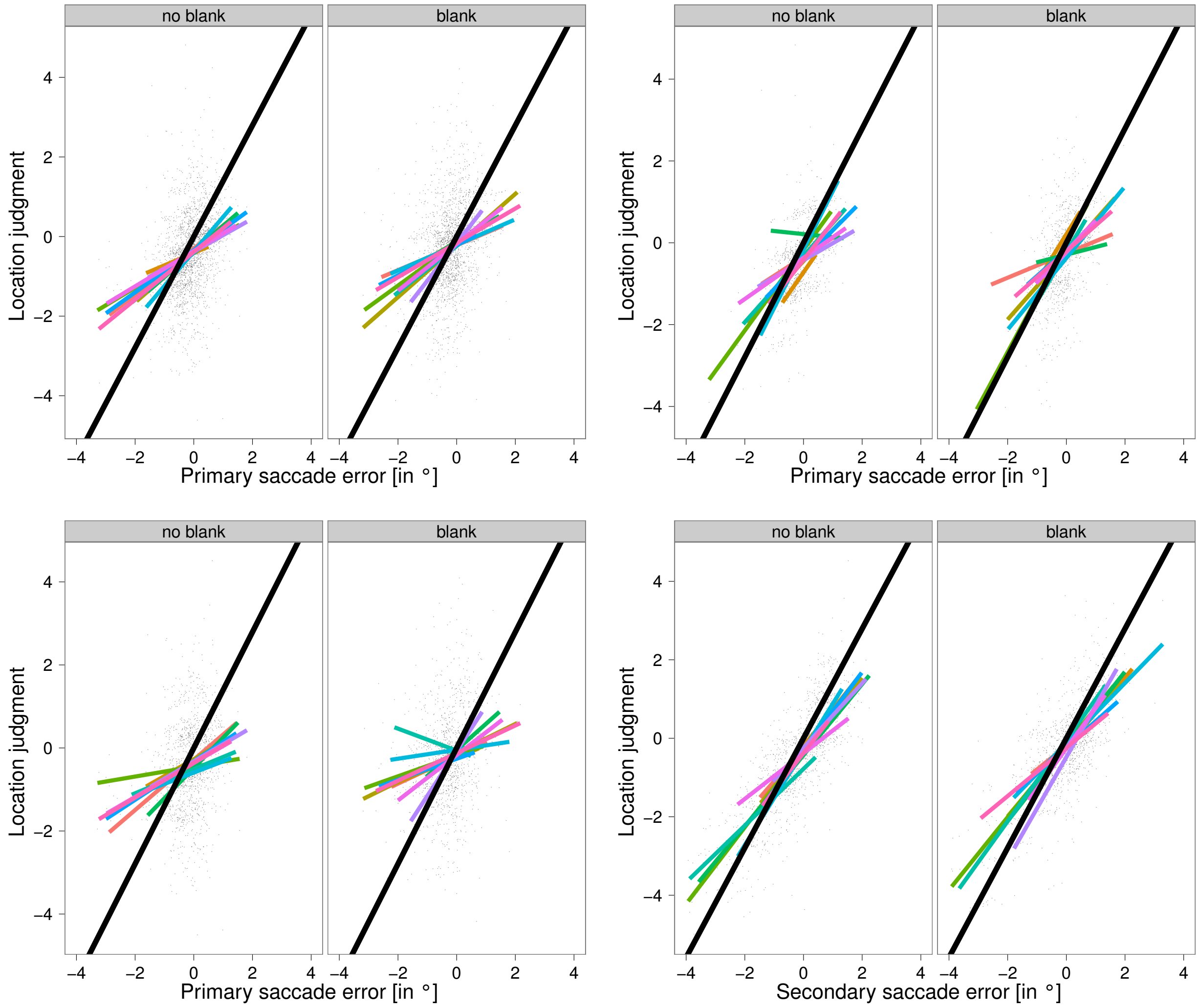

Figure 6.9: Postsaccadic location judgment as function of primary saccade error. The thick black line depicts the prediction when postsaccadic location judgments can be predicted by saccade landing position. Colored lines depict results of individual subjects. Left column represents blocks with no blank while the right columns depicts results for blocks with a blank at the beginning of postsaccadic fixation. **Top left**, shows results for the comlete population of postsaccadic location judgments. **Top right**, shows results for postsaccadic location judgments when no secondary saccade was observed. **Bottom left**, shows results for postsaccadic location judgments when secondary saccades were observed. **Bottom right**, postsaccadic location judgments as function of secondary saccade error in trials which included secondary saccades.

Table 6.5: LMM statistics of postsaccadic location judgments for the subset when secondary saccades were observed.

	Estimate	SE	*t*-Value
Intercept	−0.342	0.101	−3.4
saccadic error (sac.error)	−0.229	0.102	−2.25
sec.sac.error	0.975	0.102	9.55
blank	0.079	0.109	0.727
sac.error x sec.sac.error	−0.017	0.034	−0.5
sac.error x blank	0.134	0.057	2.38
sec.sac.error x blank	−0.066	0.04	−1.67
sac.error x sec.sac.error x blank	0.138	0.041	3.34
Variance components		SD	
Subject:			
Intercept		0.32	
saccadic error		0.32	
secondary saccade		0.32	
blank		0.34	
Residuals		0.76	

saccades. In contrast, trials with secondary saccades indicated a strong relationship between location judgments and the remaining secondary saccade error.

In general, inserting a blank in experiment 2 did not significantly improve postsaccadic location judgments. The only clear evidence of a blanking effect was observed for the generation of secondary saccades. The latency distribution of secondary saccades was strongly reduced in the blank condition during an early time window.

In experiment 1, we reported the surprising result that increasing location judgment error was associated with less confident judgments. This suggested that in experiment 1, the information of the correct target location was able to influence the confidence judgment, but not the location judgment. Importantly, we replicated the same finding in our second experiment. Again, increasing distance between location judgment and correct target location was associated with a decrease in confidence.

6.4 General discussion

What information is used to judge a postsaccadic location in the face of a multiple target-like object screen? We hypothesized that location judgments in the presence of a multiple target-like object screen are either based on the information of the correctly predicted target location, or on the initial landing position of the response saccade.

A strong dependency of location judgments on the initial landing position of the saccade would indicate that disturbing the recalibration of postsaccadic space by showing multiple target-like objects is different to the presentation of a blank screen. On the other hand, if the presentation of a multiple target-like object screen results in precise postsaccadic location judgments, we would conclude that information of the correct target

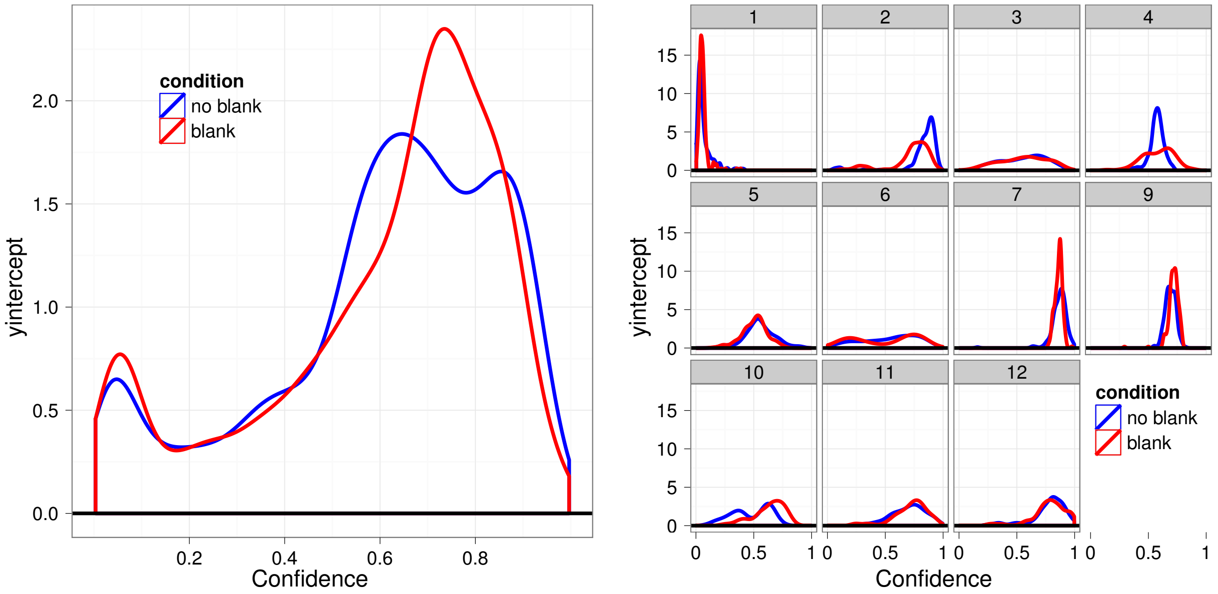

Figure 6.10: Left, confidence density associated with location judgments as function of blank (red) vs. no blank (blue). Values to the right (close to 1) indicate very confident judgments while values close to 0 indicate judgments that were *not confident*. **Right**, confidence density for each individual subject as function of blank (red) vs. no blank (blue).

location can be restored by means of disturbing the process of recalibration during an early time window of postsaccadic fixation.

Examination of location judgments showed that in both experiments participants were not able to correctly identify the target in a large proportion of judgments (experiment 1: 51%; experiment 2 blank condition: 58%; experiment 2 no-blank condition: 62%). This result strongly contradicts our hypothesis that a multiple target-like object screen may act similar to a blank screen. Now we turn to the question whether the saccade landing position influences the postsaccadic location judgment.

Indeed, we observed in both experiments a significant influence of primary saccade error on postsaccadic location judgment. Importantly, we identified that the influence of the primary saccade landing position was strongly dependent on whether or not a secondary saccade was generated. When no secondary saccade was observed, primary saccade error predicted postsaccadic location judgments. In contrast, when a secondary saccade was generated, the influence of the initial landing position was significantly reduced. Noteworthy, overall we observed a center-bias in location judgments: participants' judgments were slightly shifted into the direction of the screen center.

Surprisingly, in experiment 2 the presentation of an additional blank screen only weakly influenced postsaccadic location judgments. We observed that a secondary saccade in the blank condition was associated with a weaker center-bias. In additional analyses we have shown that location judgments in trials with secondary saccades varied strongly with the remaining error after the secondary saccade. Again, this finding was shown in both experiments.

In previous research it has been shown that information of the correct target location can be available on the motor level (Prablanc & Martin, 1992), while it is not accessible

Table 6.6: LMM statistics of confidence associated with postsaccadic location judgments.

	Estimate	SE	*t*-Value
Intercept	0.622	0.065	9.56
sac.error linear	0.001	0.008	0.14
sac.error square	−0.004	0.003	−1.25
judgment error linear	−0.005	0.007	−0.73
judgment error square	−0.006	0.002	−3.27
secondary saccade	−0.015	0.008	−1.97
blank	−0.0003	0.021	−0.02
Variance components		SD	
Subject:			
Intercept		0.215	
sac.error linear		0.023	
sac.error square		0.001	
judgment error linear		0.022	
judgment error square		0.005	
secondary saccade		0.02	
blank		0.07	
Residuals		0.14	

for a perceptual decision. Thus, we examined the dependency of secondary saccade characteristics on saccadic error in order to test whether location information is available to the oculomotor system. In experiment 1, we saw weak but significant effects of either linear or square saccadic error on the latency, amplitude and orientation of secondary saccades. In experiment 2, our experimental condition (blank vs. no-blank) strongly modulated the influence of saccadic error on secondary saccade characteristics. The most obvious result was a strong decrease in the number of secondary saccades during an early time window of postsaccadic fixation in the blank condition.

Moreover, primary saccade error influenced secondary saccade latency in the blank condition. Also, increasing saccadic error caused increasing secondary saccade amplitude. This effect was not dependent on the blank vs no-blank condition. Finally, primary saccade error influenced the orientation of secondary saccades. Increasing overshoot was associated with an increasing probability to generate a secondary saccade in direction opposite to the primary saccade. In the blank condition we observed a significant bias to produce more secondary saccades in the same direction as the primary a saccade.

Overall, in both experiments the characteristics of secondary saccades revealed weak but significant influences of primary saccade error on the generation of small eye movements during postsaccadic fixation. So far, the results of the present two experiments do not provide evidence that a multiple target-like object screen can restore location information for perception. Nevertheless, we found that saccadic error weakly influenced characteristics of secondary saccades, which would be predicted under the assumption that the location information is still available to the oculomotor system.

What is the possible role of small eye movements for location judgments in our task? We have shown that in trials without a secondary saccade, there was a strong relationship between location judgment and initial saccade landing position. But when a secondary saccade occured, the location judgment could be predicted by the remaining error after the secondary saccade.

Surprisingly, inserting a blank did not significantly improve postsaccadic location judgments. The major consequence of the blank was a strong reduction of secondary saccades during an early time window during postsaccadic fixation. Why did the blanking fail to improve postsaccadic location judgments? We speculate that the strong transient associated with the onset of the multiple target-like object screen may have disturbed the usually observed blank effect. Although surprising, this result offers a new and important perspective on the blank effect. Typically, inserting a blank results in recovered location information of the presaccadic target. In our experiment, it seems that the strong visual transient overwrote the recovered information. Thus, even after a critical time period of 200 ms, visual information can still affect postsaccadic location judgment in such a way that the correct location information cannot be used for the perceptual decision.

The presentation of the multiple target like objects constitutes an important difference between the current experiments and the classical paradigm studying postsaccadic location judgments. Another approach examining the influence of multiple target-like objects could make use of a 2-AFC task. In such an experiment, a screen with multiple target-like objects would be presented at the beginning of fixation, followed by the presentation of single target-like object. The subject would have to decide whether or not this object is identical to the initial target. Thus, the experiment would be identical to the classical paradigm, just replacing the blank by a screen with multiple target-like objects. Consequently, the results could be directly compared to the time course of the classical blanking effect.

In our experiments we observed strong inter-individual differences in both location judgments and the confidence associated with the location judgment. These inter-individual differences also persisted after inserting a blank. Typically, the variance in location judgments between subjects is strongly reduced in the classical blank effect. The strong inter-individual differences observed in our experiments are additional support for the claim that a multiple target-like object screen did not result in restored location information for the perceptual decision.

The degree of confidence associated with a decision constitutes an important cognitive function that evaluates past outcomes and might help to plan future actions (Vickers, 1979). Typically, confidence correlates both with decision accuracy and reaction time (Petrusic & Baranski, 2003). Recently, a study on the neural mechanisms underlying choice certainty identified neurons in the parietal cortex to carry information about confidence (Kiani & Shadlen, 2009). Thus, the degree of confidence can be a valuable source of information to further characterize a perceptual decision process like postsaccadic location judgments.

In our experiments, the location information (weakly) influenced the confidence associated with each location judgment. Participants were less confident in situations with wrong location judgments as compared to correct location judgments. Importantly, we have shown this surprising effect in two separate experiments. Thus, apart from the motor (and oculomotor) system, we identified an additional behavior, namely the computation of confidence, which was affected by the information about the correct presaccadic target location.

Chapter 7

Influences of the preparatory set on microsaccades before endogenously vs. exogenously defined targets

7.1 Introduction

The majority of recent studies in the field of fixational eye movements were conducted in order to examine a possible relationship between microsaccades and attention. Although some authors doubted a relationship between microsaccades and attention (Horowitz et al., 2007; Tse, Sheinberg, & Logothetis, 2002), there is much evidence by now that the orientation of microsaccades as well as the time of occurrence of these effects are influenced by covert attention and task characteristics (Engbert & Kliegl, 2003; Engbert, 2006, 2012; Hafed & Clark, 2002; Hafed, Lovejoy, & Krauzlis, 2011; Laubrock, Engbert, & Kliegl, 2005; Laubrock et al., 2007, 2010; Pastukhov & Braun, 2010; Pastukhov et al., in press).

In previous research, the relation between covert shifts of attention and microsaccade rate and orientation was almost always examined in the cue-target interval. Shifting attention to a specific location in the visual field, however, is not exclusively bound to occur after the presentation of an attentional cue or a target. Of course, attention can be shifted to future informative locations already prior to a cue/ target onset, in order to optimize the upcoming response. In general, preparatory processes that take place before the presentation of a critical stimulus are referred to as preparatory set (Evarts, Shinoda, & Wise, 1986; Hebb, 1972). A detailed understanding of these preparatory processes is of relevance for models of (micro-)saccade generation because the preparatory set has been shown to directly influence neuronal firing in important nodes of the oculomotor system (Everling et al., 1999; Everling & Munoz, 2000).

In the present study we ask whether processes in the preparatory set (that is before cue/ target onset) influence microsaccade statistics. There is only one recent study that addressed this issue. Hermens, Zanker and Walker (2010) examined microsaccade rate in separate blocks of pro- and antisaccade trials. They also analyzed the influence of an immediate vs. delayed response and of a central endogenous cue vs. a peripheral exogenous cue on microsaccade rate. Interestingly, microsaccade rate was increased in prosaccade as compared to antisaccade trials already prior to target onset. This effect was only observed when the target was presented in the periphery and subjects were instructed to give delayed responses. Thus, differences in microsaccade rate between prosaccades and antisaccades were observed neither for endogenous cues nor in the condition of an immediate response after presentation of an exogenous cue.

There remain open questions whether top-down preparatory processes affect microsaccade statistics. Hermens, Zanker and Walker (2010) did not examine the differences in microsaccade rate in the preparatory interval between exogenous and endogenous target onset. Importantly, comparing microsaccade rate between endogenous and exogenous target onsets informs about an important aspect of the preparatory set: the allocation of covert attention. Given that subjects know where endogenous and exogenous cues are presented during the experiment, differences between centrally vs. peripherally located attention during a preparatory interval are likely to have an impact on microsaccade rate.

Indeed, we can derive a prediction about these effects based on a qualitative model of microsaccade generation (Rolfs, Kliegl, & Engbert, 2008). This model assumes that saccades result from activity crossing a triggering-threshold in a topographically organized saccadic motor map. Eye movements to the right are generated in the left hemisphere and leftward saccades in the right hemisphere of the saccade motor map. Importantly, the center of the motor map is coding for microsaccades. Increasing fixation-related activation in the center of this map increases the number of microsaccades. In contrast, increasingly distant sites are coding for increasingly large saccadic eye movements. Such a model is supported by the finding that the rostral pole of the superior colliculus (SC) is causally involved in the generation of microsaccades (Hafed, Goffart, & Krauzlis, 2009).

Now, we assume that attending to a specific location in a visual scene will increase the baseline activation at the corresponding location in the saccadic motor map. This assumption is supported by the finding that covert attention increases baseline activation in the superior colliculus at the attended location (Ignashchenkova et al., 2004; Kustov & Robinson, 1996).

Against this theoretical background, we examined microsaccades in a simple experiment (divided into two blocks) in which subjects where required to saccade to a square target. With trial onset, two square placeholders were presented on the horizontal meridian of the computer screen with equal center-target distance, indicating the two potential target locations. A cue was presented after a variable duration in order to indicate the target. Subjects were asked to move their eyes immediately to the target. Depending on the block, the cue was either a centrally presented arrow directing to one of the placeholders (endogenous condition) or the placeholder was blinking (exogenous condition) in order to highlight the target location.

Endogenous and exogenous conditions in our experiment differ strongly in cue location. While in the exogenous condition the cue location is identical to one of the two already present placeholders in the periphery, the cue (arrow) in the endogenous condition will be presented in the center of the screen (see Figure 7.1). Subjects can direct their attention to the future location of the informative cue, which we predict to have a direct influence on the rate of microsaccades. Attending the center of the screen (endogenous condition) should increase activation in the center of the saccade motor map (e.g., rostral pole of the SC). Increasing fixation-related activation in the center of

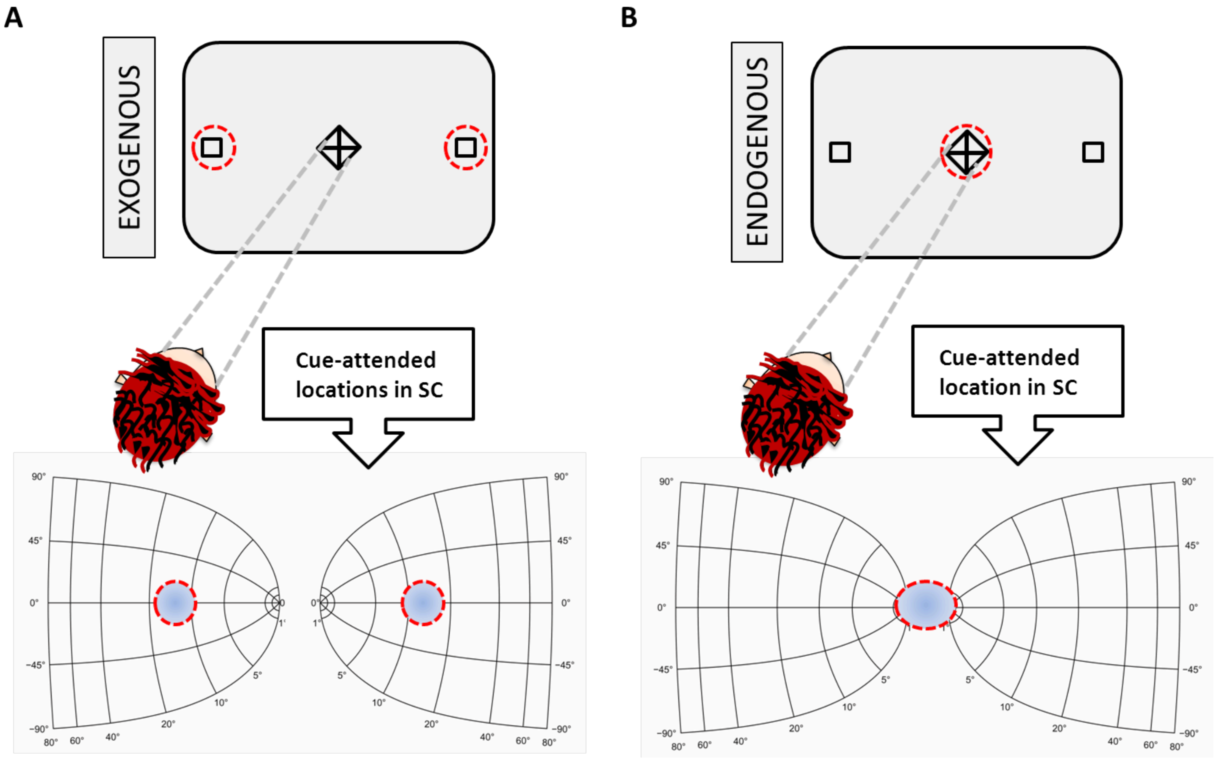

Figure 7.1: Attention during the preparatory interval is assumed to be directed towards future relevant locations. **A**, In the exogenous condition the target will be presented at one of the two placeholders in the periphery. This is hypothesized to result in increased baseline firing of neurons in the two caudal regions of the SC indicated by the dashed circle. **B**, In the endogenous condition the informative cue will be presented in the center of the screen. Attending to this foveal location is assumed to result in increasing firing rates in the rostral pole of the SC.

the saccade motor map results in a higher number of microsaccades (Rolfs, Kliegl, & Engbert, 2008): therefore we hypothesize a higher microsaccade rate in the endogenous as compared to the exogenous condition during the preparatory interval before cue onset. Thus, in contrast to previous studies on the relationship between attention and the orientation of microsaccades, we are specifically interested in the influence of attention on the number of microsaccades (see also Pastukhov & Braun, 2010).

In addition to cue type, we randomly varied target eccentricity (close vs. distant target) within each block. Oculomotor behavior can be strongly influenced by target eccentricity; e.g. the latency of saccades, and therefore their programming, varies for different target eccentricities (Kalesnykas & Hallett, 1994; Ohl, Brandt, & Kliegl, 2011). The interacting structure within the SC is shaped by excitatory and inhibitory connections which are assumed to result in local excitation and global inhibition. Consequently, different eccentricities are characterized by a different ratio of excitation and inhibition. This implies that preparatory processes may also vary as a function of target eccentricity.

The present experiment may also give us further insights into the interaction between microsaccade and subsequent saccade generation. It has been observed repeatedly, that microsaccades around target presentation are associated with prolonged saccade latencies (Hafed & Krauzlis, 2010; Kliegl et al., 2009; Rolfs, Laubrock, & Kliegl, 2006, 2008; Sinn & Engbert, 2011). With the present experiment we can examine whether the initially reported result of prolonged latencies for saccades in response to exogenous stimuli (Rolfs, Laubrock, & Kliegl, 2006) can be replicated. Moreover, we can test whether microsaccade-induced prolongation of saccadic reaction times is also observed for endogenously triggered saccades and whether the prolongation of latencies varies for different target eccentricities.

Obviously, in reaction time paradigms the influence of microsaccades on the reaction time can be reduced my minimizing the chance of microsaccade occurrence (e.g., by employing short cue-target intervals). Nevertheless, as soon as microsaccade rate is significantly influenced by the preparatory set, there is a condition-specific contribution of microsaccades to the overall reaction time. Thus, identifying the conditions which produce differences in the number of microsaccades and delineating the mechanisms underlying the prolongation of saccadic reaction times by microsaccades is relevant for reaction time experiments in general.

To sum up, the present study examines the sensitivity of microsaccade rate to the preparatory set, in terms of differences in microsaccade rate before endogenously vs. exogenously triggered saccades. Moreover, we test whether microsaccade induce prolongation of saccadic reaction times of endogenous saccades.

7.2 Materials and methods

7.2.1 Participants

Thirty (21 female) subjects participated in the experiment. They received study credit for participation or were paid seven Euros. The participants were 17-44 years old (M = 24.97) and had normal or corrected-to-normal vision. The experiment is in accordance with the declaration of Helsinki (1964) and we obtained written informed consent from all participants prior to the experiment.

7.2.2 Materials and procedure

Participants were sitting 50 cm in front of a computer screen, with their head positioned on a chin rest. Visual stimuli were presented on a 19-inch EYE-Q 650 CRT monitor at a resolution of 1024 x 768 and a monitor frame rate of 100 Hz. For eye movement recording we used the EyeLink-II system (SR Research, Osgoode, Ontario, Canada) with a sampling rate of 500 Hz. The experiment was running on an Apple Power Macintosh G4 computer and was implemented in Matlab (Mathworks, Natick, MA, USA) using the Psychophysics toolbox (Brainard, 1997; Pelli, 1997) and the Eyelink toolbox (Cornelissen, Peters, & Palmer, 2002).

Each subject participated in two experimental blocks, which were collected in a single session that lasted approximately one hour. The two experimental blocks differed with regard to whether the saccade target was indicated by a centrally presented arrow (endogenous condition) or by blinking of a white square at the target location (exogenous condition). In detail, after eye position was detected within an invisible square, the trial was initialized by the presentation of a central, white fixation symbol (see Figure 7.2 and 7.3), and two white, square placeholders (diameter: one degree of visual angle) on a

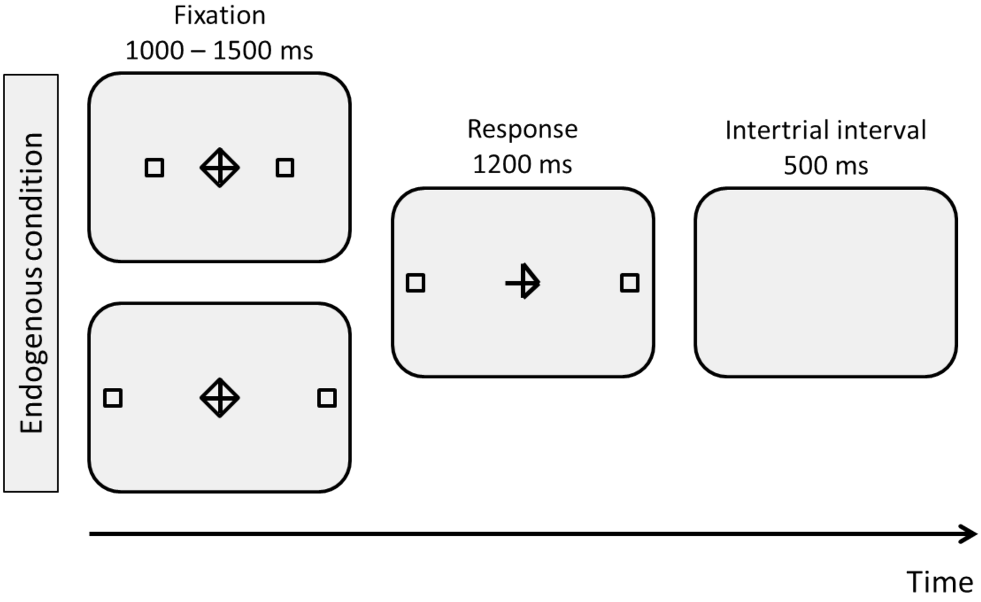

Figure 7.2: Outline of event sequence in experimental trial during endogenous block. Bottom (top) row indicates event sequence for distant (close) target eccentricity.

grey background. The inner of the two placeholders was filled with background color (grey). Placeholders were presented on the horizontal meridian to the left and right of the fixation symbol with equal distance to the center of the screen. Placeholders were presented either at an eccentricity of 6 degree of visual angle (close target) or 14°of visual angle (distant target). After 1000-1500 ms a cue indicated the target and instructed the subjects to move their eyes to the target location. Depending on the current block, the cue was either a change of color from background color (grey) to white and back to grey (blink duration of 80 ms) at the position of one of the two placeholders (exogenous condition). Alternatively, two lines from the fixation symbol were removed, thus resulting in an arrow pointing to the target (endogenous condition). Subjects were instructed to move their eyes immediately to the target. Block order was counterbalanced across subjects.

A new trial began after an inter-trial interval of 500 ms. After failing of the fixation check at trial onset, a drift correction was initialized. A failed fixation check after drift correction resulted in a new calibration and subsequent validation procedure. In addition, every 30 trials the eyetracking system was calibrated and validated. Blinks were detected online during the experimental trial and caused immediate trial abort. Those trials were presented again at the end of the experiment in randomized order.

7.2.3 Data preparation and analysis

Eye movement recordings of one participant were removed from the analysis due to a corrupted data file. This resulted in a total of 29 participants completing successfully the experiment. For detection of microsaccades we used a modified version (Engbert & Mergenthaler, 2006) of the algorithm developed by Engbert and Kliegl (2003). Eye movement trajectories were transformed into 2D velocity space and microsaccades were

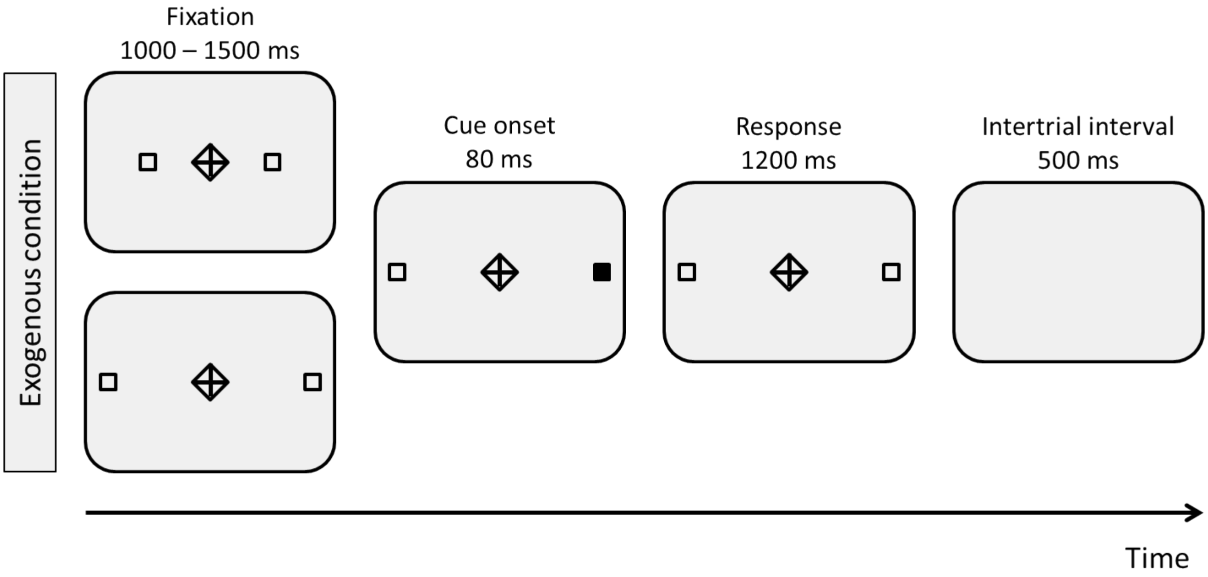

Figure 7.3: Outline of event sequence in experimental trial during exogenous block. Bottom (top) row indicates event sequence for distant (close) target eccentricity.

detected using a threshold for peak velocity (6 SD) and a minimum duration of 8 ms (4 samples). For our analyses we considered only binocular microsaccades, meaning that microsaccades had to be detected in both eyes with a temporal overlap.

The response saccade was defined as the first eye movement that landed within a distance of 3°of visual angle around the target. Trials in which the response saccade was preceded by an eye movement larger than 1.5°were removed from further analyses. Furthermore, saccadic reaction time (SRT) had to be slower than 80 ms and faster than 600 ms.

Overall, 8376 trials (259-316 trials per subject, M = 288.83) of the initial 9280 experimental trials (90%) passed the criteria to be included for further analyses. In the interval from trial onset to cue onset, we detected a total of 13,254 microsaccades.

The microsaccade rate was computed for each subject individually by moving a window (window size of 150 ms) in steps of 1 ms across time, counting the number of microsaccades inside the window and divide it by the number of trials for a given experimental condition. Such smoothed versions of individual histograms were then normalized to the number of microsaccades per second.

When we were interested to quantify the effect of a continuous variable on the saccade latency we used linear mixed models (LMM) for statistical analysis, including subjects as random factor. Effects were considered as significant when (1) magnitude of t-value was larger than 2, and (2) the effect was also significant after drawing 1000 samples from the posterior distribution of parameters of the LMM. For data analysis we used the R environment (Development Core Team, 2011) including the additional lme4 (Bates & Maechler, 2011) and ggplot2 (Wickham, 2009) packages.

To allow comparison of different LMMs we provide goodness of fit measures; the Akaike Information Criterion (AIC), the Bayesian Information Criterion (BIC) and the log likelihood (logLik). Moreover, we report the chi-square-distributed likelihood ratio.

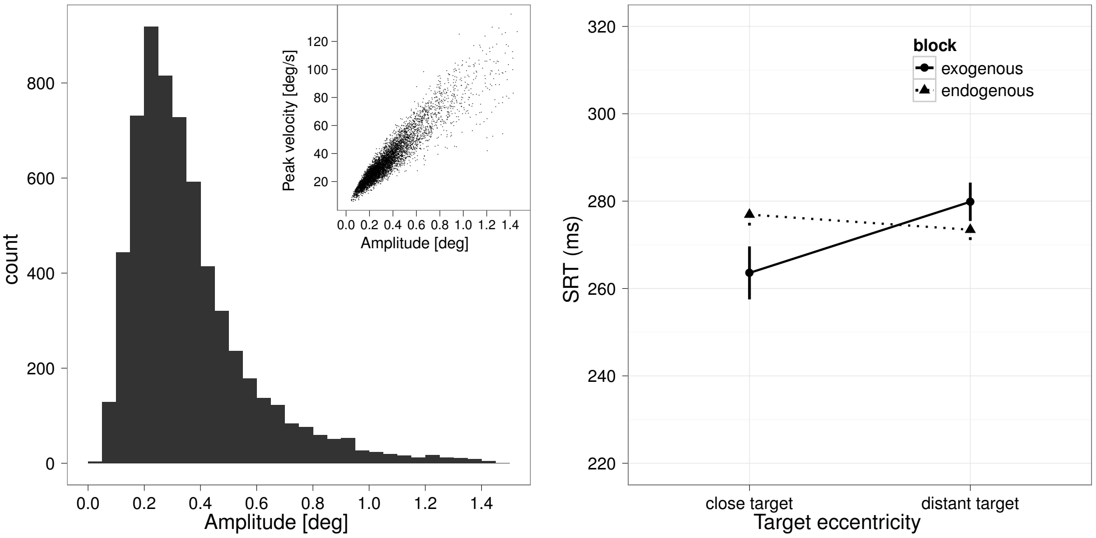

Figure 7.4: Left panel, amplitude distribution of eye movements during fixation. In the upper right of the left panel, the main sequence relationship between eye movement amplitude and peak velocity is shown. **Right panel**, SRT as a function of block (endogenous block is shown as dashed line; exogenous block as solid line) and target eccentricity.

Finally, the present experiment allows us to study also secondary saccades during early postsaccadic fixation. We included only secondary saccades following primary saccades that were faster than 600 ms and landed within a range of 2.5°around the center of the target. Secondary saccades were only considered for further analysis when they occured within the first 350 ms during postsaccadic fixation with an amplitude smaller than 3.5°. Moreover, secondary saccades were classified as either following the direction of the primary saccade or in opposite direction to the primary saccade. Secondary saccades in vertical direction (up or down) were removed. Overall, we included a total of 5651 secondary saccades for further analyses.

7.3 Results

In the present study, we were interested to examine microsaccades during a preparatory interval before cue signal presentation. In the left panel of Figure 7.4, the distribution of microsaccade amplitude and the main sequence is shown.

In right panel of Figure 7.4, SRTs are shown as a function of target eccentricity in both, the exogenous and endogenous block. A 2 x 2 ANOVA revealed significant main effects of block [F(1,28) = 4.78; p = 0.037] and eccentricity [F(1,28) = 4.57; p = 0.041]. SRTs were significantly faster to close than to distant targets and slower in endogenous as compared to exogenous trials. The main effects were further substantiated by a significant interaction [F(1,28) = 15.87; p < 0.001]. Post-hoc t-tests for paired samples revealed [t(28) = 3.15; p = 0.004] significantly faster SRTs for close as compared to distant targets in the exogenous block. In contrast, SRTs were marginally slower for close as compared to distant targets in the endogenous condition [t(28) = 1.84; p = 0.077].

Table 7.1: LMM statistics for saccade latency

	Estimate	SE	*t*-Value
Intercept	279.77	6.37	43.95
exogenous vs. endogenous (block)	1.74	0.61	2.83
eccentricity (ecc)	3.03	0.61	4.94
block x ecc	−4.93	0.61	−8.04
foreperiod (fp)	−0.02	0.004	−5.59
Variance components		SD	
Subjects		33.59	
Residuals		56.08	

Table 7.2: Models including additional interactions are compared to model 1.

Model comparison	AIC	BIC	logLik	p-value
(compare with model 1)	91374	91423	−45680	
model 1 + (fp x block)	91375	91431	−45680	0.37
model 1 + (fp x ecc)	91376	91432	−45680	0.56
model 1 + (fp x ecc x block)	91377	91440	−45679	0.56

Cue presentation in our study was uniformly distributed in an interval from 1000-1500 ms after trial start. In an experiment with equally distributed foreperiods, increasing foreperiod is associated with an increasing hazard rate and typically decreasing reaction times are observed (Luce, 1986; Niemi & Näätänen, 1981; Nobre, Correa, & Coull, 2007). We included foreperiod as covariate in a LMM along with the main factors block (coding: exogenous = -1; endogenous = 1), eccentricity (coding: close target = -1; distant target = 1) and the interaction block x eccentricity. In line with the results from the above reported 2 x 2 ANOVA, our LMM (see Table 7.1) yielded a significant main effect of block (t = 2.83) and eccentricity (t = 4.94). Moreover, we found a significant interaction between block and eccentricity (t = -8.04) in the direction already observed in the ANOVA. Interestingly, we observed a significant main effect of foreperiod (t = -5.59). Increasing foreperiod was associated with a decreasing saccadic reaction time, which is an effect in the expected direction. We computed additional models including also the interaction of foreperiod x block, foreperiod x eccentricity, or both interactions and compared those models to model 7.1. None of the additional terms significantly improved model 7.1.

7.3.1 Microsaccade rate during the preparatory interval

The main goal of the current study is to test the influence of exogenously vs. endogenously defined targets and target eccentricity on the microsaccade rate during a preparatory interval. Figure 7.5 shows the microsaccade rate locked to trial start as a function of target eccentricity and block (exogenous vs. endogenous condition). Overall, we see a decreasing microsaccade rate over time, replicating earlier findings on the interaction of microsaccades and response preparation (Betta & Turatto, 2006). Moreover, we observed a large difference in microsaccade rate between the endogenous and exogenous condition. Microsaccades rate was higher during the preparatory interval before presentation of

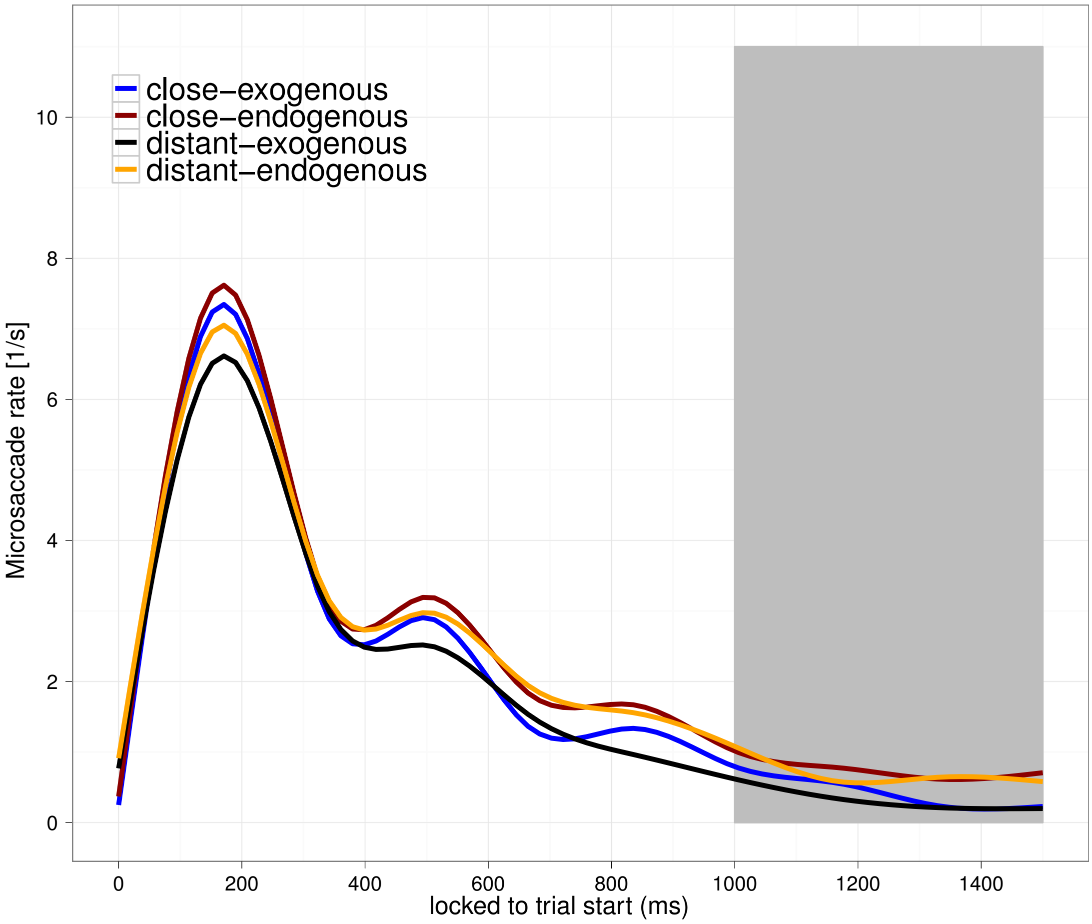

Figure 7.5: Microsaccade rate as a function of block and target eccentricity locked to trial onset. The experimental conditions are coded by color; close-exogenous (blue), distant-exogenous (black), close-endogenous (red), and distant-endogenous (orange). The displayed rates are based on smoothing (loess method) over the 29 individual (moving window) rates in each of the four experimental conditions.

the endogenous as opposed to the exogenous cue. Furthermore, Figure 7.5 shows differences in microsaccade rate depending on target eccentricity in the exogenous condition. Starting around 750 ms after trial onset, microsaccade rate is increased for close targets as compared to distant targets. We ran a 2 (eccentricity: close vs. distant) x 2 (block: exogenous vs. endogenous) ANOVA in an interval from 400 ms to 1500 ms after trial onset. We observed a significant main effect of block [F(1,28) = 21.07, $p < 0.001$], confirming our hypothesis of a significantly higher rate in the endogenous condition. Moreover, the influence of eccentricity was significant [F(1,28) = 6.62, p = 0.016], indicating a higher rate for close as compared to distant targets. The interaction turned out to be marginally significant [F(1,28) = 3.48, p = 0.072]. Post-hoc t-tests for paired samples showed a significantly higher rate for close as compared to distant targets in the exogenous block [t(28) = 2.90; p = 0.007]. In the endogenous condition, we did not observe a significantly different result for the two eccentricities [t(28) = 1.57; p = 0.127].

As cue presentation was uniformly distributed within an interval between 1000-1500 ms after trial onset we also examined the microsaccade rate locked to cue onset (see Figure 7.6). Again we observed an increased microsaccade rate in the endogenous as

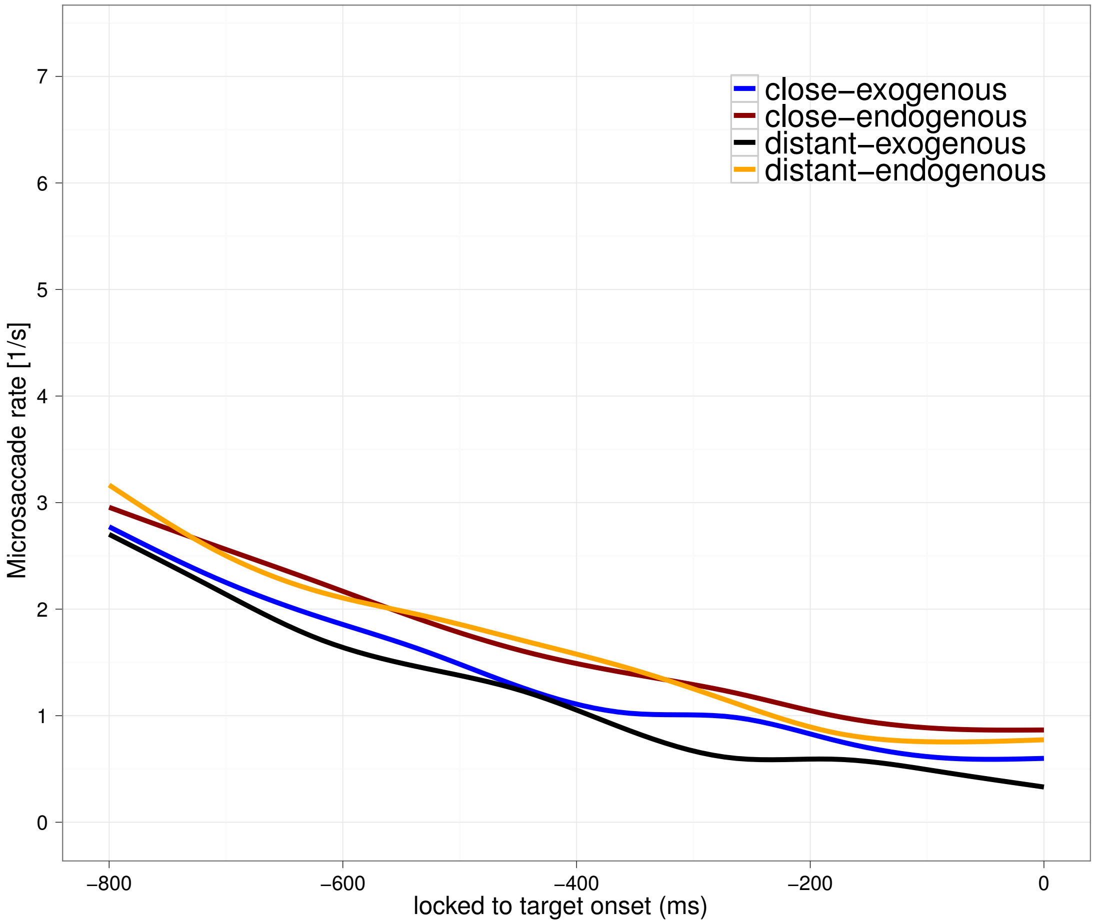

Figure 7.6: Microsaccade rate as a function of block and target eccentricity locked to target onset. The experimental conditions are coded by color; close-exogenous (blue), distant-exogenous (black), close-endogenous (red), and distant-endogenous (orange).

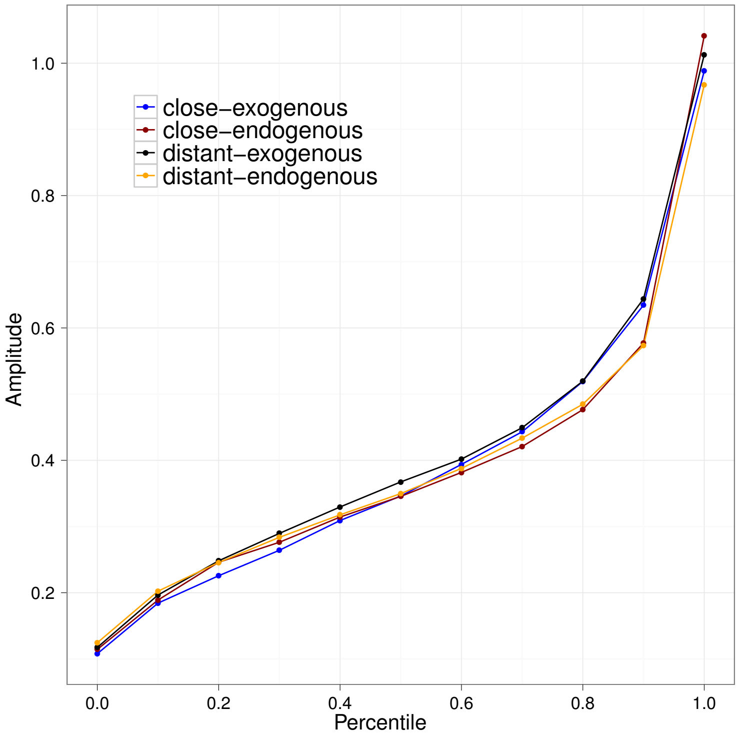

Figure 7.7: Cumulative amplitude distribution as a function of block and eccentricity. The experimental conditions are coded by color; close-exogenous (blue), distant-exogenous (black), close-endogenous (red), and distant-endogenous (orange).

compared to exogenous condition. Also, in the exogenous condition microsaccade rate was increased for close as compared to distant targets. Now, there was also a trend in the endogenous condition that close targets were associated with a higher microsaccade rate starting around 300 ms before cue presentation. We ran a 2 x 2 ANOVA in the interval 800 ms to 0 ms before cue onset including the same main effects and interaction as above. Similar to the results when microsaccades were locked to trial onset, we observed a strong effect of block [$F(1,28) = 14.20$, $p < 0.001$] and this time just a marginally significant effect of target eccentricity [$F(1,28) = 3.81$, $p = 0.061$]. The interaction did not reach significance [$F(1,28) = 2.47$, $p = 0.13$].

So far, we have seen that microsaccade rate during a preparatory interval varied as a function of block (exogenous vs. endogenous) and target eccentricity. Finally, we tested whether microsaccade amplitude varied for the different experimental conditions. In Figure 7.7 we see the cumulative distribution of microsaccade amplitude as a function of block and target eccentricity. We computed 2 x 2 ANOVAs (block x eccentricity) at 10 different percentiles of the cumulative amplitude distribution. Our analyses revealed a significant influence of block on the cumulative amplitude distribution at the 80th and 90th percentile. At both percentile positions amplitude was larger in the exogenous as compared to the endogenous condition.

In the present study, our major goal was to examine the influence of endogenously vs. exogenously triggered saccades and target eccentricity on the microsaccade rate during a preparatory interval before cue onset. We hypothesized differences in microsaccade rate as the location of the cue differed between a foveal (endogenous block) and peripheral (exogenous block) presentation. Indeed, as predicted we found an increased microsaccade rate in the endogenous in contrast to the exogenous condition. Thus, microsaccade rate was very sensitive to the preparatory set in our blocked design. Moreover, we observed an effect of target eccentricity on the microsaccade rate. The rate was higher for close as compared to distant targets, which was mainly the case for the exogenous condition.

7.3.2 Microsaccades prolong subsequent saccade latency

Recently, several studies reported prolonged saccadic reaction times induced by microsaccades occurring around go signal presentation (Hafed & Krauzlis, 2010; Kliegl et al., 2009; Rolfs, Laubrock, & Kliegl, 2006; Sinn & Engbert, 2011). We were interested to examine the effect of microsaccades on the latency of subsequent saccades as a function of target eccentricity and endogenously vs. exogenously triggered saccades.

Two different types of analyses can be considered in order to quantify the influence of microsaccades on the subsequent saccade latency. First, saccadic reaction times following microsaccades in a specific time interval can be compared to all other saccadic reaction times. Second, regression analyses can be used to infer the influence of cue-locked microsaccades on the saccade latency. The second analysis obviously neglects trials in which no microsaccade occurred. In the following section, we will report both analyses.

In Figure 7.8, the costs and benefits associated with a microsaccade occurring in a specific time window before target presentation are displayed. Positive values indicate an increase in saccadic reaction time (costs), while negative values mean faster saccadic reaction times (benefits). For all four conditions we see a similar pattern of results. Saccadic reaction times seem not to be affected by the presence of microsaccades that occur long before target onset. Importantly, the closer the temporal distance between microsaccades and target onset, the higher were the costs for saccadic reaction times. This replicates the previously reported finding in exogenously triggered saccades of microsaccade-induced saccadic reaction time prolongation (Rolfs, Laubrock, & Kliegl, 2006). The analysis shows that the effect is also true for endogenously triggered saccades and irrespective of target eccentricity.

We ran additional LMMs in order to establish the effect that decreasing temporal distance between microsaccade onset and target presentation causes prolonged saccadic reaction times. In our LMM, we included the same predictors as in model 7.1. Furthermore, we included the variable of microsaccade onset locked to the target presentation (msOnset) and the interaction of msOnset x foreperiod (see Table 7.3). We compared this model with models including the additional terms of msOnset x block, msOnset x eccentricity, and both interactions (see Table 7.4). Results were similar to the reported findings of model 7.1. Only the effect of block was not significant anymore in model 2 (t $= -1.27$). The most important finding of model 2 was the significant effect of msOnset (t $= -3.501$). Thus, a decreasing temporal distance between microsaccade onset and target presentation was associated with longer saccadic reaction times, therefore replicating the microsaccade induced prolongation of saccadic reaction times. Neither the interaction of msOnset x block nor the interaction of msOnset x eccentricity significantly improved the LMM.

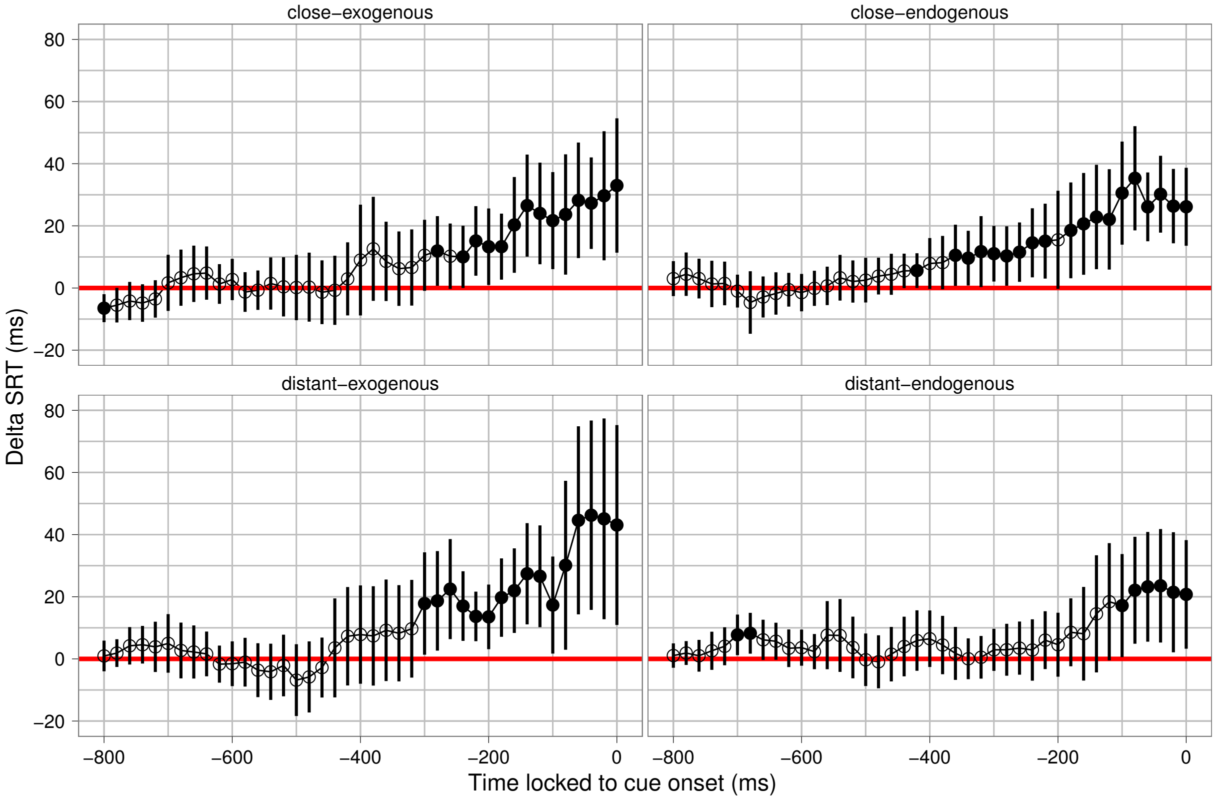

Figure 7.8: Costs and benefits for SRTs (in ms) when microsaccades occur in a specific time window before target onset. Filled disks indicate a value that is significantly different from zero.

Table 7.3: LMM statistics for microsaccade-induced prolongation of saccadic reaction times.

	Estimate	SE	t-Value
Intercept	355.9	16.43	21.66
exogenous vs. endogenous (block)	−1.14	0.9	−1.27
eccentricity (ecc)	3.03	0.887	3.41
block x ecc	−5.75	0.887	−6.49
foreperiod (fp)	−0.518	0.012	−4.15
msOnset	−0.108	0.031	−3.5
msOnset x fp	0.000058	0.00002	2.31
Variance components		SD	
Subjects		30.26	
Residuals		51.59	

Table 7.4: Models including addtional interactions are compared to model 2.

Model comparison	AIC	BIC	logLik	p-value
(compare with model 2)	36933	36988	−18458	
model 2 + (msOnset x block)	36934	36996	−18457	0.42
model 2 + (msOnset x ecc)	36935	36996	−18457	0.67
model 2 + (msOnset x block + msOnset x ecc)	36936	37004	−18457	0.67

7.3.3 Secondary saccades

In the present study, we also examined secondary saccades in more detail. Although there are considerable differences between the visual layout of the current experiment and our previous experiments concerning secondary saccades (Ohl, Brandt, & Kliegl, 2011, see chapter 3), there are also important similarities. In both experiments we tested the influence of target eccentricity on the oculomotor behavior. In our previous study, we reported a strong influence of target eccentricity on the latency, amplitude and orientation of secondary saccades. Although the target in our previous study was a filled disk and in the present study the outline of a square, we were still interested whether we could replicate our previous findings. Moreover, we can test whether oculomotor behavior during postsaccadic fixation depends on characteristics of primary saccade programming, namely the presentation of an endogenous vs. exogenous cue.

First of all, we examined whether primary saccade error differed between the exogenous vs. endogenous condition and whether target eccentricity had a significant effect on primary saccade landing position (see Figure 7.9). For analysis we used a LMM including target eccentricity and endogenous vs. exogenous condition, as well as their interaction as fixed effects (see Table 7.5). In addition, we included target eccentricity and exogenous vs. endogenous condition as random effects in our model. Target eccentricity was the only significant effect on primary saccade error (t = −3.42). As expected, we observed a significant primary saccade undershoot in the distant target condition. Importantly, neither exogenous vs. endogenous condition nor the interaction significantly influenced primary saccade error. Thus, differences between the exogenous and endogenous condition with respect to the functional relationships between primary saccade error and the

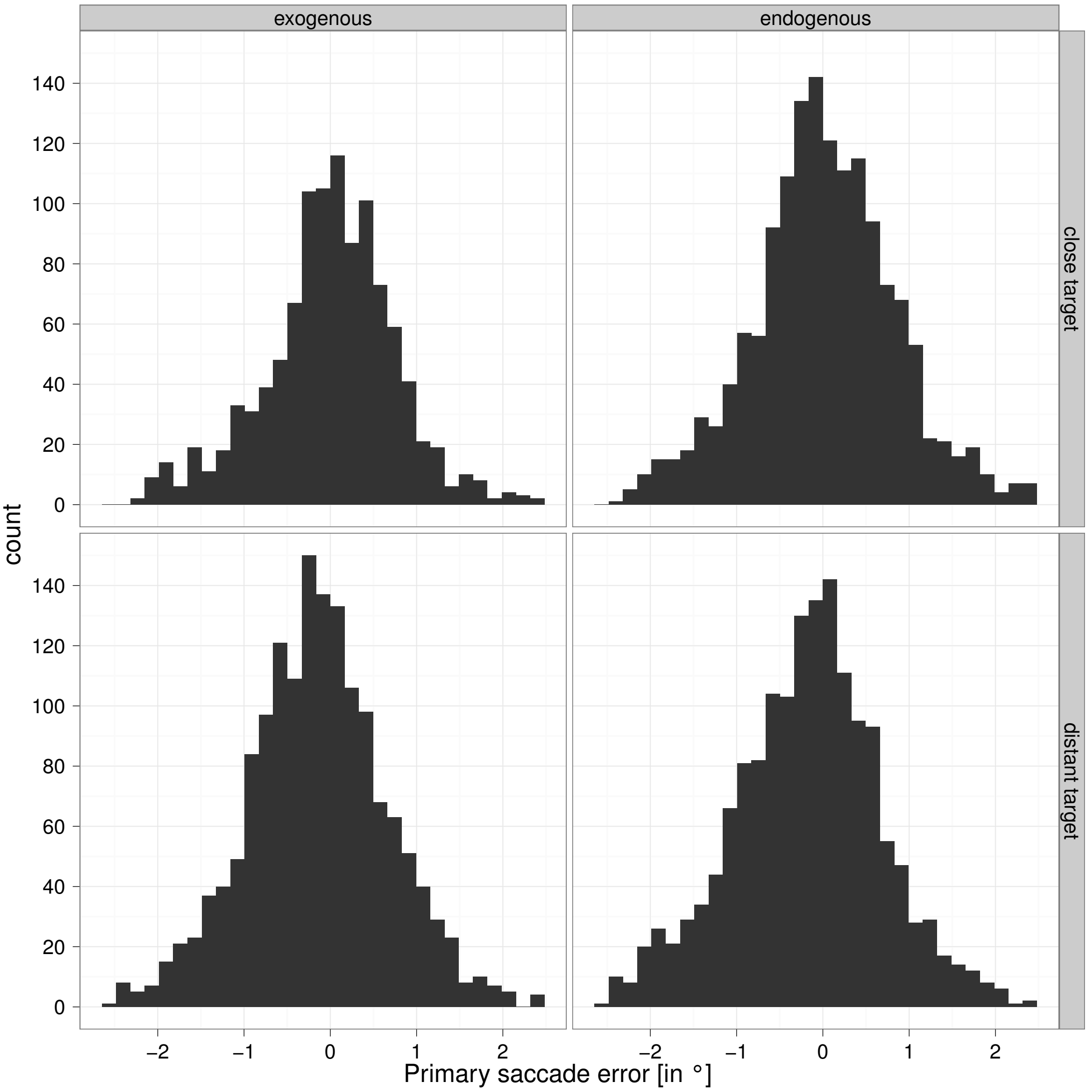

Figure 7.9: Distribution of primary saccade landing position as a function of experimental block (exogenous vs. endogenous) and target eccentricity (close vs. distant target). Distributions comprise only trials in which a secondary saccades was observed that was included for further analyses.

characteristics of secondary saccades (e.g., latency, amplitude and orientation) cannot be attributed to condition-dependent differences in primary saccade landing position.

In Figure 7.10 and 7.11 we show the distributions of secondary saccade latency and secondary saccade amplitude. In all conditions, secondary saccade latency clearly peaked within the first 350 ms of postsaccadic fixation. Moreover, distributions of secondary saccade amplitude highlight that a large proportion of secondary saccades was in the amplitude range of microsaccades.

7.3.3.1 Secondary saccade latency

Based on our modified model for the generation of secondary saccades (see chapter 3 and 4), we predicted a decrease in secondary saccade latency with increasing primary saccade error. In addition, we argued that the maximum latency is shifted to small primary saccade overshoot, which results from an hemispheric bias in activation within the saccade

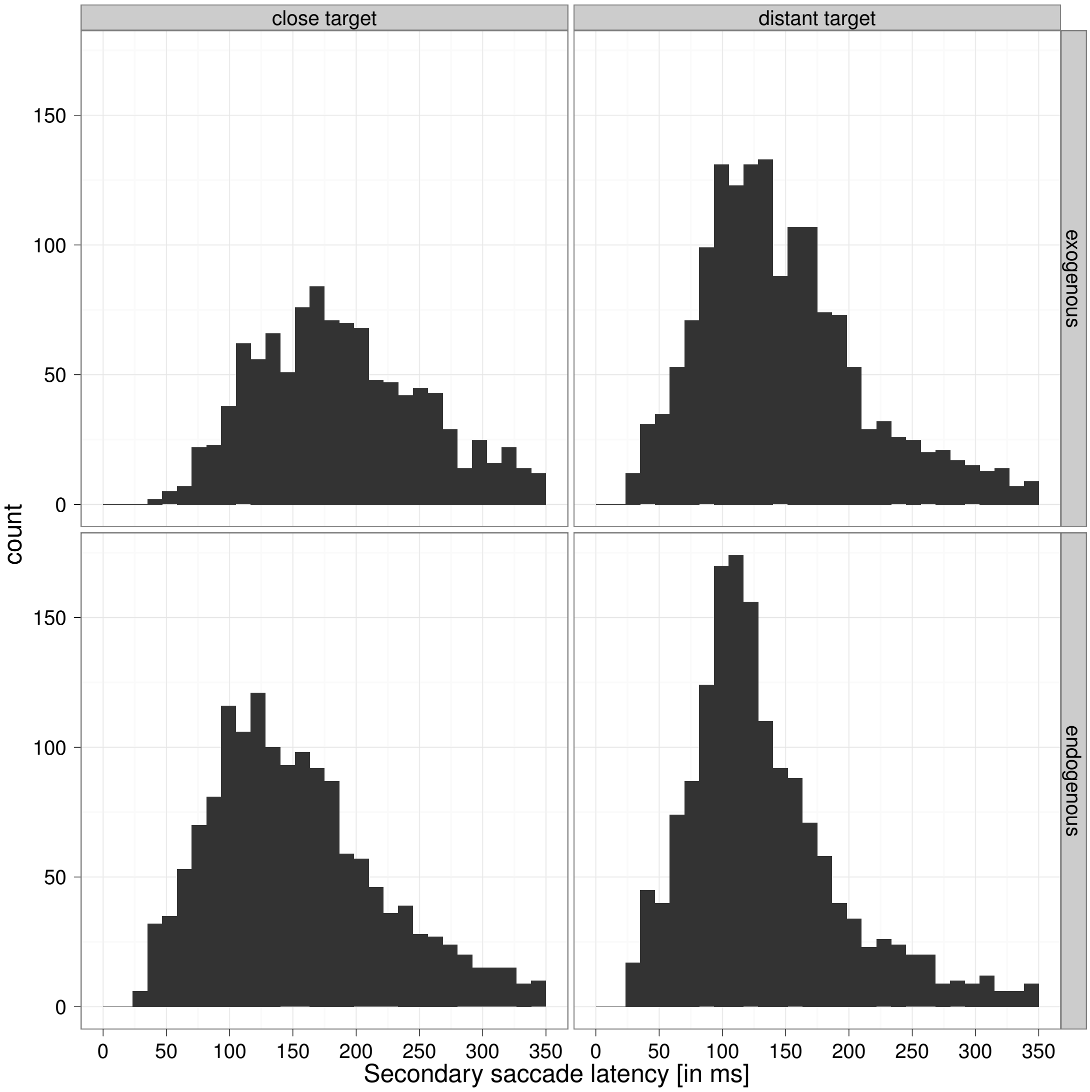

Figure 7.10: Distribution of secondary saccade latency as a function of experimental block (exogenous vs. endogenous) and target eccentricity (close vs. distant target).

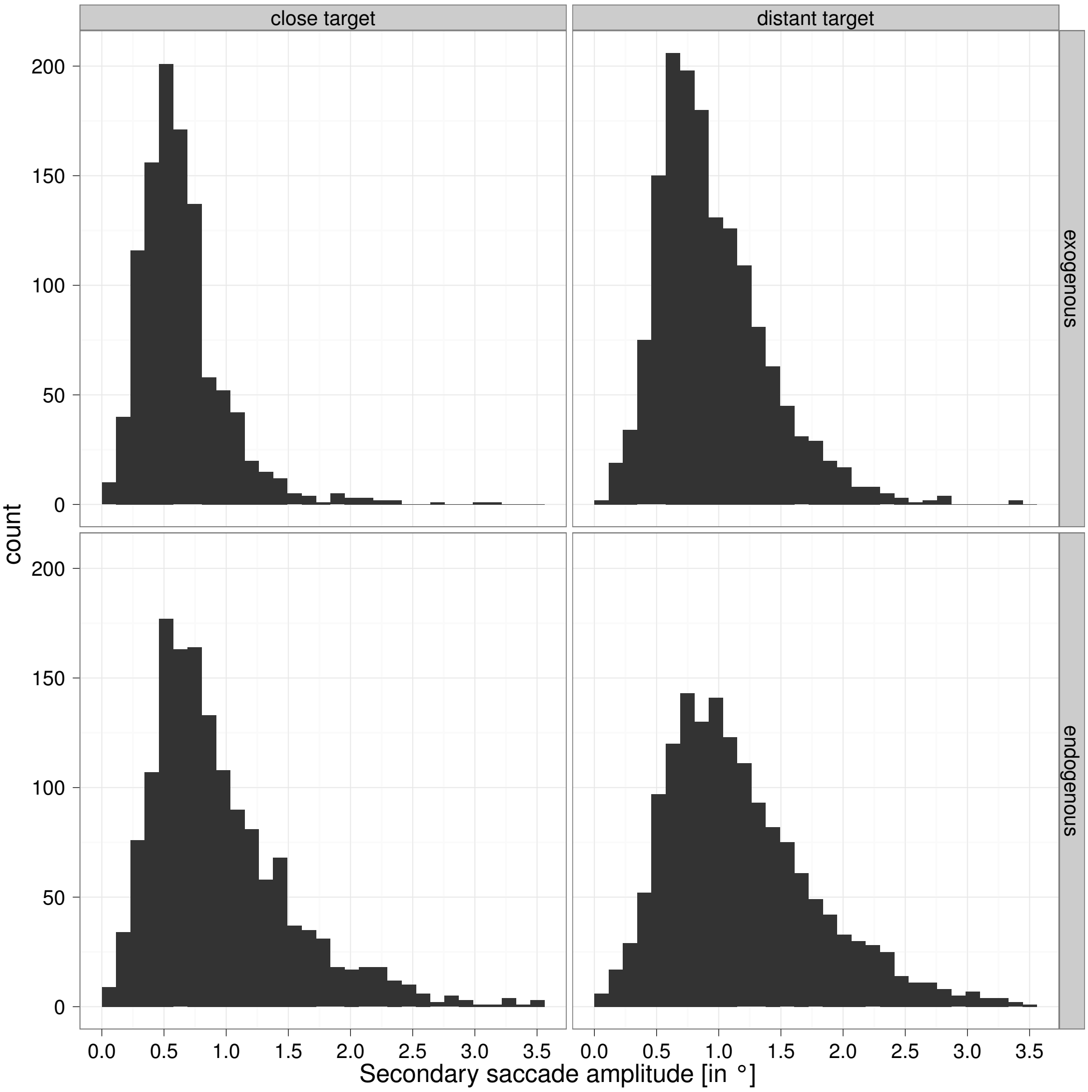

Figure 7.11: Distribution of secondary saccade amplitude as a function of experimental block (exogenous vs. endogenous) and target eccentricity (close vs. distant target).

Table 7.5: LMM statistics for primary saccade error.

	Estimate	SE	*t*-Value
Intercept	0.016	0.061	0.27
eccentricity (ecc)	−0.159	0.046	−3.42
exogen. vs. endogen. (exen)	−0.001	0.032	−0.02
ecc x exen	−0.034	0.040	−0.85
Variance components		SD	
Subject:			
Intercept		0.304	
eccentricity		0.191	
exogen. vs. endogen.		0.066	
Residuals		0.740	

motor map due to primary saccade programming. We assumed this bias to increase with increasing target eccentricity, therefore secondary saccades were hypothesized to be faster after precise primary saccades to distant as compared to close targets.

Importantly, despite some differences in the details of the visual layout between the present and our previous experiment, the results in Figure 7.12 are in line with the predictions from our model. First, there was a strong modulation of secondary saccade latency as a function of primary saccade error. Increasing error was associated with faster secondary saccades. Moreover, Figure 7.12 suggests large differences in secondary saccade latency depending on target eccentricity. This effect seems to be weaker in the endogenous condition. Also, the overall latency seems to be longer in the exogenous as compared to the endogenous condition.

We were interested to quantify these results with a LMM, including linear and quadratic primary saccade error, target eccentricity, exogenous vs. endogenous, along with all possible interactions between these variables (see Table 7.6). Moreover, we added the intercept, primary saccade error (linear and quadratic), target eccentricity and exogenous vs. endogenous condition as random effects in the LMM. In line with Figure 7.12, in the exogenous condition we were able to replicate a strong influence of primary saccade error on secondary saccade latency ($t = -4.49$). Increasing primary saccade error resulted in a decrease of secondary saccade latency. The peak was significantly shifted in the direction of a small overshoot ($t = 3.18$). Moreover, secondary saccades were faster following precise primary saccades to distant targets as compared to close targets ($t = -10.37$). An additional modulation of primary saccade error by target eccentricity in the exogenous condition was not significant.

We also observed significant differences between the exogenous and endogenous condition. In line with Figure 7.12, secondary saccade latency was overall decreased in the endogenous condition ($t = -7.85$) and the effect of target eccentricity was significantly reduced ($t = 5.11$). Also, we observed further modulations in interactions demonstrating a stronger influence of primary saccade error in the endogenous condition ($t = -4.68$). Noteworthy, inspection of the endogenous condition in Figure 7.12 suggests two peaks in secondary saccade latency. This can be explained by the square target, which may be decomposed into two possible targets (e.g. the left vs. right border of the square).

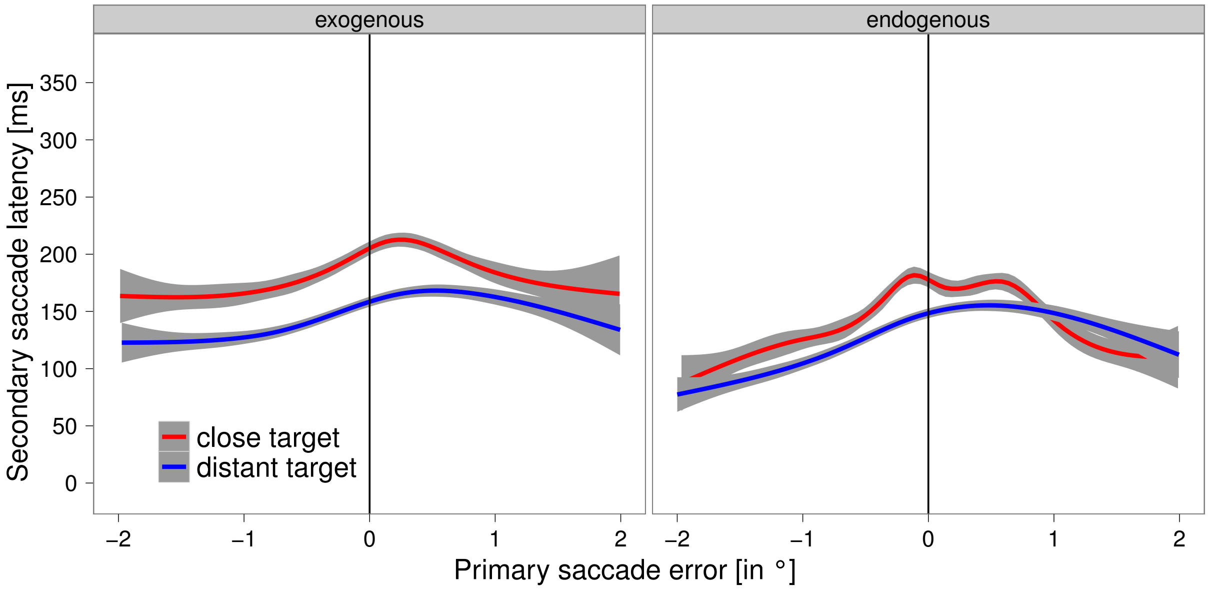

Figure 7.12: Secondary saccade latency as a function of primary saccade error, target eccentricity (red = close target, blue = distant target) and experimental block (exogenous vs. endogenous). We smoothed values of secondary saccade latency after removal of between-subject variance. Smoothing is based on loess method. Grey bands represent the 95% confidence interval.

7.3.3.2 Secondary saccade amplitude

Given the repeatedly demonstrated influence of error signals on secondary saccade characteristics, we tested the influence of primary saccade landing position on secondary saccade amplitude. In Figure 7.13 we depict the relationship between secondary saccade amplitude and primary saccade error as a a function of target eccentricity and exogenous vs. endogenous condition. A first inspection of Figure 7.13 is in line with the prediction that increasingly large primary saccade error should be associated with larger secondary saccades. Noteworthy, this relationship seems to be stronger in the endogenous as compared to the exogenous condition. In addition, a strong influence of target eccentricity becomes immediately evident. Again replicating previous findings, secondary saccade amplitude was increased in the distant target condition.

For quantification, we used a LMM including the same fixed and random effects as specified for the analysis of secondary saccade latency (see Table 7.7). Our analysis yielded a complex structure of relationships accounting for secondary saccade amplitude. In line with our model and previous findings, we observed that increasing error was associated with increasingly large secondary saccades in the exogenous condition ($t = 3.7$). Again, minimum amplitude did not occur at perfectly precise primary saccades. Indeed, the minimum amplitude was shifted to small primary saccade overshoot ($t = -3$).

Our analysis revealed a strong influence of target eccentricity. Secondary saccade amplitude in the exogenous condition was significantly increased after primary saccades to distant targets ($t = 9.63$). Also, we observed a significantly stronger shift of minimum secondary saccade amplitude in the direction of primary saccade overshoot ($t = -4.22$). There was also a significantly stronger increase in secondary saccade amplitude with primary saccade error for distant targets ($t = 2.17$).

Table 7.6: LMM statistics for secondary saccade latency.

	Estimate	SE	t-Value
Intercept	197.66	6.41	30.84
eccentricity (ecc)	−42.78	4.13	−10.37
error	8.83	2.78	3.18
square error	−10.15	2.26	−4.49
exogen. vs. endogen. (exen)	−29.58	3.77	−7.85
ecc x exen	18.26	3.58	5.11
ecc x error	4.99	3.02	1.65
ecc x square error	3.29	2.43	1.35
exen x error	0.05	2.96	0.02
exen x square error	−11.12	2.38	−4.68
ecc x exen x error	4.00	3.85	1.04
ecc x exen x square error	6.52	3.13	2.08
Variance components		SD	
Subject:			
Intercept		32.69	
eccentricity		16.95	
exogen. vs. endogen.		14.38	
error		7.45	
square error		6.19	
Residuals		54.51	

We observed additional error-unrelated influences on secondary saccade amplitude. Indeed, secondary saccade amplitude was significantly increased in the endogenous as compared to the exogenous condition (t = 4.58). The influence of quadratic primary saccade error was significantly more pronounced in the endogenous condition (t = 10.87) and we also observed a stronger shift of minimum amplitude into the direction of primary saccade overshoot (t = −4.96). In the endogenous condition, the effect of target eccentricity was reduced (t = −2.03). Finally, we observed a significant 3-way interaction. The functional relationship between secondary saccade amplitude and primary saccade landing position was reduced for distant targets in the endogenous condition (t = −2.43). To sum up, although the outline of a square constitutes a different target than a small filled disk, we observed very similar results to our previous studies. Most notably, we replicated a shift of minimum amplitude into the direction of small primary saccade overshoot. Moreover, we replicated our previous findings that larger eccentricities were associated with larger secondary saccade amplitude.

7.3.3.3 Secondary saccade orientation

In the present thesis, we demonstrated the importance of studying secondary saccade orientation in order to elucidate error-related (visual and extra-retinal) and error-unrelated (e.g. eccentricity) signals in a saccadic motor map during early postsaccadic fixation. So far, we replicated several basic influences on secondary saccade latency and secondary saccade amplitude. In Figure 7.14, we depict secondary saccade orientation as a function

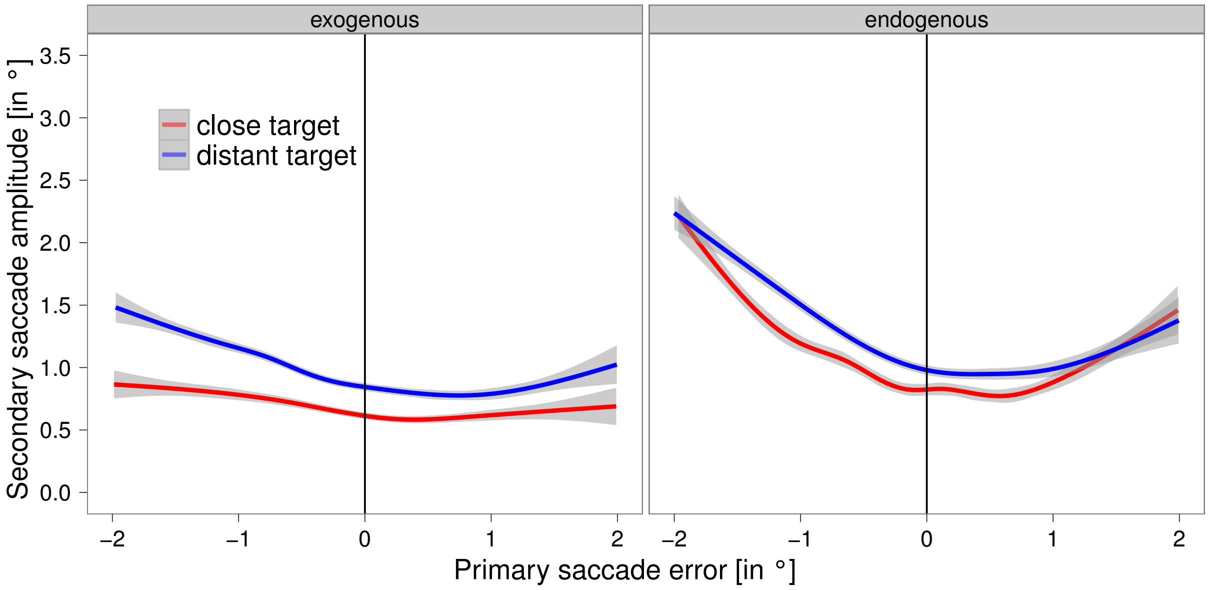

Figure 7.13: Secondary saccade amplitude as a function of primary saccade error, target eccentricity (red = close target, blue = distant target) and experimental block (exogenous vs. endogenous). For smoothing we used secondary saccade latency after removal of between-subject variance. Smoothing is based on loess method. Grey bands represent the 95% confidence interval.

of primary saccade error, target eccentricity, as well as exogenous vs. endogenous condition. Inspection of Figure 7.14 reveals that the results were in line with our previous findings. Moving primary saccade landing position from undershoot to overshoot resulted in an increasing probability to generate a secondary saccade in direction opposite to the primary saccade. Moreover, we observed a bias to follow the direction of precise primary saccades. This bias increased with increasing target eccentricity which is line with our previous findings. Noteworthy, in the exogenous condition we observed some secondary saccades that followed primary saccade direction despite considerable primary saccade overshoot. We need to highlight again the differences in the target stimulus between our different experiments. The larger outline of the square in the present study might directly affect the orientation of secondary saccades.

Again, we quantified our observations using a LMM (see Table 7.8). In line with our previous study, we observed a strong influence of primary saccade error ($p < 0.001$) supporting the role of a visual error signal on secondary saccade orientation. Again, we demonstrated a strong influence of target eccentricity ($p < 0.001$). Secondary saccades following primary saccades to distant targets had a stronger bias to follow the direction of the primary saccade. The difference between the close and distant target condition was significantly decreased in the endogenous condition ($p = 0.017$). Finally, the influence of primary saccade error on secondary saccade orientation was increased in the endogenous condition ($p < 0.001$).

The present experiment allowed us to analyze secondary saccades, too. Although we highlighted that the exact visual parameters between the present and previous experiments differed, we replicated our important findings from the previous studies. Most importantly, we demonstrated again that error-related and error-unrelated signals

Table 7.7: LMM statistics for secondary saccade amplitude.

	Estimate	SE	*t*-Value
Intercept	0.621	0.043	14.52
eccentricity (ecc)	0.238	0.025	9.63
error	−0.064	0.022	−3.00
square error	0.06	0.016	3.70
exogen. vs. endogen. (exen)	0.188	0.041	4.58
ecc x exen	−0.051	0.025	−2.03
ecc x error	−0.09	0.021	−4.22
ecc x square error	0.037	0.017	2.17
exen x error	−0.105	0.021	−4.96
exen x square error	0.185	0.017	10.87
ecc x exen x error	0.026	0.027	0.94
ecc x exen x square error	−0.054	0.023	−2.43
Variance components		SD	
Subject:			
Intercept		0.217	
eccentricity		0.086	
exogen. vs. endogen.		0.196	
error		0.067	
square error		0.043	
Residuals		0.385	

influence the programming of secondary saccades. Most importantly, we demonstrated a bias to follow primary saccade direction and an even stronger bias for the distant target condition. Thus, target eccentricity had a strong influence on the latency, amplitude, and orientation of secondary saccades. In line with previous findings, secondary saccades following primary saccades to distant targets were faster, larger and more likely to be oriented in the same direction as the primary saccade.

Moreover, we identified considerable differences between the exogenous and endogenous condition. Secondary saccades in the endogenous condition were faster and had larger amplitudes. Although the exact mechanisms underlying a difference between the exogenous and endogenous condition with respect to the programming of secondary saccades are not clear, our results demonstrate that signals during a presaccadic interval (endogenous vs. exogenous cue) influence the characteristics of small saccades during postsaccadic fixation.

7.4 Discussion

In the present study we investigated the influence of the preparatory set on microsaccades. To this end, we examined microsaccade statistics while participants were asked to generate response saccades in separate blocks of endogenously vs. exogenously presented targets.

Importantly, microsaccade statistics in our experiment were significantly influenced by the preparatory set. Microsaccade rate was significantly higher in the endogenous as

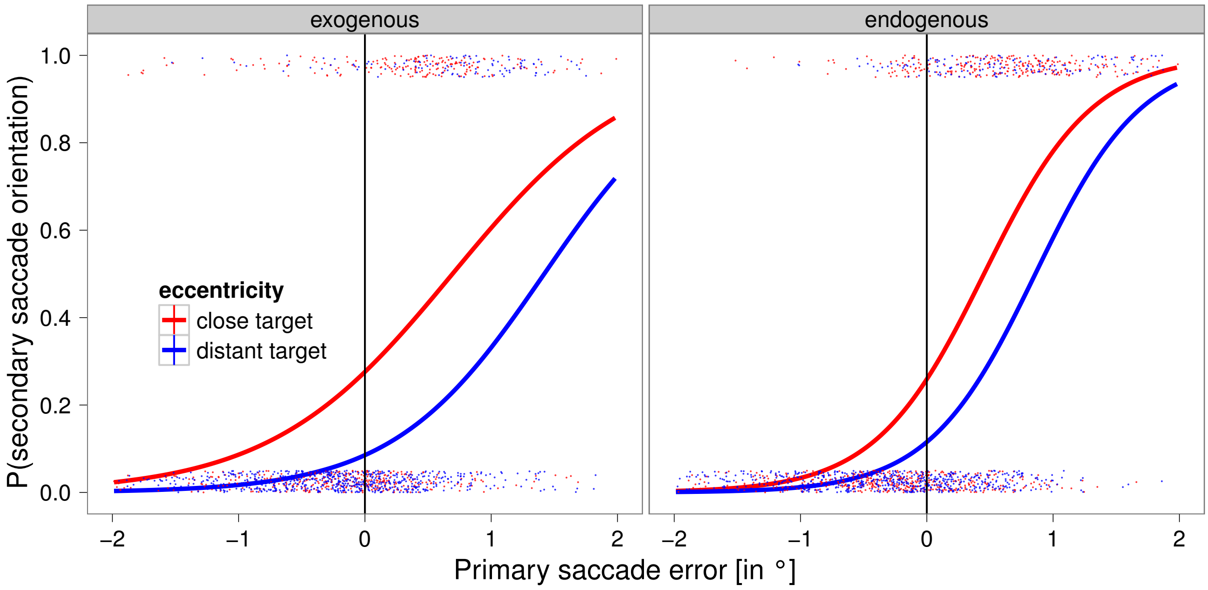

Figure 7.14: Secondary saccade orientation as a function of primary saccade error, target eccentricity (red = close target, blue = distant target) and experimental block (exogenous vs. endogenous). Individual data points are vertically jittered around 1 for secondary saccades in direction opposite to the primary saccade. Secondary saccades in the same direction as the primary saccade are jittered around 0. Solid lines represent predictions from the GLMM after removal of between-subject variance.

compared to the exogenous condition. Thus, in contrast to the study by Hermens, Zanker and Walker (2010), we find a significant influence of the preparatory set on microsaccade rate in a task that requires an immediate response after target onset.

The result is in line with our hypothesis, which was derived from a qualitative model of microsaccade generation (Rolfs, Kliegl, & Engbert, 2008) and further supported from neurophysiological findings (Hafed, Goffart, & Krauzlis, 2009). First, the rostral pole of the superior colliculus is known to be causally involved in microsaccade generation (Hafed, Goffart, & Krauzlis, 2009). Second, attending to a specific location in a visual scene will cause increased neuronal firing at a region in the superior colliculus representing the attended location (Ignashchenkova et al., 2004; Kustov & Robinson, 1996). Thus, we reasoned that attending to the center of the screen (endogenous condition), which is represented in center of the saccade motor map (e.g., the rostral pole of the superior colliculus), should result in a higher microsaccade frequency as compared to attending to peripheral locations that are represented more caudally.

Our study implies that the allocation of attention during a preparatory interval influences the microsaccade rate. Nevertheless, one should critically reflect that the endogenous vs. exogenous condition also differ in the number of the potentially attended locations. In the endogenous block, the cue will always be presented at the same location in the center of the screen. In contrast, there are two possible cue locations in the exogenous condition. Consequently, the exact mechanisms underlying preparatory processes in the oculomotor system will certainly continue to be an interesting topic for future research. The primary goal of the current study was to demonstrate that such preparatory processes indeed have an immediate influence on microsaccades.

Table 7.8: LMM statistics for secondary saccade orientation.

	Estimate	SE	p-Value
Intercept	−0.965	0.198	< 0.001
eccentricity (ecc)	−1.406	0.176	< 0.001
error	1.392	0.177	< 0.001
exogen. vs. endogen. (exen)	−0.086	0.198	0.666
ecc x exen	0.420	0.176	0.017
ecc x error	0.278	0.180	0.123
exen x error	0.928	0.178	< 0.001
ecc x exen x error	−0.228	0.258	0.377
Variance components		SD	
Subject:			
Intercept		0.960	
eccentricity		0.626	
exogen. vs. endogen.		0.873	
error		0.634	

In our experiment, we also examined whether microsaccade statistics in a preparatory interval are significantly influenced by target eccentricity. Indeed, we find a higher microsaccade rate in the close than distant target condition. More specifically, our results suggest, that an eccentricity effect is only observed in the exogenous condition. This is particularly interesting, as preparation in the exogenous condition requires sustained attention in the periphery (where the target will be presented), and an effect of target eccentricity implies that sustained attention at varying eccentricities differently modulates microsaccade rate.

Moreover, in our present study we replicate the finding that microsaccades occurring around the presentation of a go signal prolong saccadic reaction times (Rolfs, Laubrock, & Kliegl, 2006). In addition, our data do not support the view of benefits associated with microsaccades occuring long before the presentation of a go signal. This can be explained by the fact that the original finding of benefits due to microsaccades was only observed for memory-guided saccades (Rolfs, Laubrock, & Kliegl, 2006). We show, that prolongation of saccadic reaction times caused by microsaccades is observed for different target eccentricities, and for both endogenously and exogenously triggered saccades. Rolfs, Laubrock and Kliegl (2006) reasoned that the microsaccade-induced prolongation of saccade latencies might be due to inhibitory interactions in the intermediate layer of the superior colliculus: Increased activation in the rostral pole coding for microsaccades would directly inhibit more caudally located neurons which program for large saccades. This original interpretation has been challenged by the proposal that prolonged saccadic reaction times due to microsaccades can be explained by visual suppression around the onset of microsaccades (Hafed & Krauzlis, 2010). According to this idea, microsaccades occurring around target presentation will cause a reduced neural response in the intermediate layer of the superior colliculus. Consequently, it will take longer to initiate a saccadic eye movement to the target.

The proposal by Hafed and Krauzlis (Hafed & Krauzlis, 2010) is straightforward. Nevertheless, we think it is rather unlikely that visual suppression alone can account for microsaccade-induced prolongation of saccade latencies. The time course of visual suppression reported by Hafed and Krauzlis starts about 70 ms before target presentation and last 140 ms until 70 ms after target onset. Importantly, results from the present study (and also recent studies, see Rolfs, Laubrock, & Kliegl, 2006) imply that prolongation of saccade latencies occurs already in an earlier time window than the observed interval of visual suppression. Further experimental evidence is necessary to distinguish the influence of visual suppression and inhibitory interactions on subsequent saccade latencies caused by microsaccades (Rolfs & Ohl, 2011).

Studying response times also requires accounting for the influence of the foreperiod. In the present study foreperiod was uniformly distributed, therefore resulting in an increasing hazard rate with increasing foreperiod. Our results show that saccadic reaction times are significantly decreased with increasing foreperiod; thus replicating a classical finding (Luce, 1986). Consequently, we agree with Hermens, Zanker and Walker (2010) that the influence of foreperiod is a crucial variable that has to be considered when studying microsaccades and preparatory processes in the oculomotor system.

It has often been discussed whether microsaccades are functional relevant for perception (Collewijn & Kowler, 2008). Unfortunately, this debate often distracts from the well-established findings that demonstrate strong consequences of microsaccades for (1) neuronal firing (Martinez-Conde, Macknik, & Hubel, 2000), (2) EEG analysis (Dimigen et al., 2009; Yuval-Greenberg et al., 2008) as well as (3) action (e.g., prolongation of subsequent response times) and (4) perception (Beeler, 1967; Martinez-Conde et al., 2006; McCamy et al., 2012; Otero-Millan, Macknik, & Martinez-Conde, 2012). As we know that microsaccades systematically influence information processing, it is of course essential to understand how microsaccades affect the outcome of interest in a given task. The results of our present study show that the preparatory interval already influences microsaccade statistics. Thus, if one is interested in studying saccadic reaction times to endogenously vs. exogenously triggered targets in a blocked design, one has to be aware that the two blocks already differ in the process of fixation before target onset.

Although the primary interest in the present study focused on preparatory influences during presaccadic fixation, the experiment allowed us to examine also secondary saccades during postsaccadic fixation. In line with predictions from our model of (micro-)saccade generation during postsaccadic fixation (see chapter 3 and 4) and in line with our previous findings, we observed a strong influence of primary saccade error and target eccentricity on the generation of secondary saccades. Thus, again we demonstrated the need to consider error-related and error-unrelated influences in order to account for secondary saccade motor programs. Studying secondary saccades complements the analysis of microsaccades during postsaccadic fixation and demonstrates the usefulness of studying small eye movements during fixation as a tool to improve our understanding of cognition in general.

7.5 Conclusion

The preparatory set encompasses processes during a preparatory interval that aim to optimize the outcome in a given task. In line with previous publications (Hafed et al., 2011; Hermens, Zanker, & Walker, 2010) we show that microsaccades are sensitive to the preparatory set. An important question for future research is to investigate whether modulated microsaccade rates are just the by-product of attention that is allocated to future informative locations in the visual scene or whether the number of microsaccades itself is reduced in order to minimize the potential costs associated with visual suppression.

Chapter 8

General summary and conclusions

Studying eye movement behavior has been proven to be a fruitful approach in order to examine human cognition. The traditional view on eye movement behavior held the simplistic view that it can be described as an alternating sequence of saccades and fixation. Fixation is characterized by visual information uptake while saccades bring the eyes to a new location in the visual scene.

This antagonistic interplay between saccades and fixation has also been thought to underlie models of saccade generation and the implementation of a saccade motor map in the oculomotor system (e.g., with its neurophysiological implementation in the superior colliculus). So called saccade neurons were thought to form a topographically organized saccade motor map with a continuous representation of saccade amplitude. Saccades with a specific amplitude and orientation were the result of saccade neurons firing at the corresponding site in the saccade motor map. During fixation the counterpart of saccade neurons, so called fixation neurons, are active. Thus, saccade and fixation neurons were thought to underlie alternating periods of fixation interleaved with fast saccadic eye movements.

This approach turned out to be very successful in explaining a variety of observations regarding saccadic eye movement behavior (e.g., remote distractor influence on saccade curvature, global effect in landing position). Nevertheless, there were also a number of findings that did not fit into such a model frame. In particular, the existence of microsaccades, which by definition have amplitudes smaller than one degree of visual angle, turned out to be problematic for such a model because microsaccade amplitude is too small in order to be the result of saccade neuron activity.

This behavioral observation and additional strong evidence from neurophysiological studies (Hafed, 2011; Hafed, Goffart, & Krauzlis, 2009) caused a shift in paradigm concerning models of saccade generation. Now, a saccade motor map is not anymore thought to consist of topographically organized saccade neurons coding for saccades with specific vectors, and fixation neurons coding for periods of fixation. Instead, it is assumed that saccades are generated in a topographic motor map (e.g., saccade amplitudes increase with eccentricity) but without the existence of a specific fixation area (Hafed, Goffart, & Krauzlis, 2009; Rolfs, Kliegl, & Engbert, 2008). In this new model frame it is thought that microsaccades are generated in the saccade motor map where previously the fixation neurons were thought to be located.

Models that emerged from such a new perspective are very successful in explaining microsaccade-related findings in oculomotor research (see chapter 2.5), but there is still a need to revisit many findings that have been accounted by the old saccade-fixation-neuron perspective, and which have to be tested against the prediction of eye movement models that include also microsaccades. Previous findings that were explained by the level of fixation neuron activity can be translated in a first step into predictions on the number of microsaccades. Increased fixation-related activation is thought to result in more microsaccades (Rolfs, Kliegl, & Engbert, 2008) therefore findings that were previously explained by a stronger activity of fixation neurons should also elicit more microsaccades.

The present thesis was set out to examine two aspects concerning small-amplitude saccadic eye movements using the new model frame, namely the generation of small saccadic eye movements during postsaccadic fixation and top-down preparatory influences on microsaccade generation during presaccadic fixation. Following, the results obtained from experiments conducted towards these topics will be discussed.

8.1 The generation of small eye movements during postsaccadic fixation

Microsaccades are classically studied during prolonged presaccadic fixation (but see also Mergenthaler & Engbert, 2010; Otero-Millan et al., 2008). Models of microsaccade generation, which are based on findings concerning presaccadic fixation, typically neglect that fixations are preceded by large saccadic eye movements. This is surprising because postsaccadic fixation constitutes a far more natural environment for small-amplitude eye movements than prolonged presaccadic fixation.

Importantly, small-amplitude eye movements during postsaccadic fixation have been studied already for more than a century under another term, namely secondary saccades. These secondary saccades include also eye movements of a size that would normally be classified as microsaccade. I reviewed findings on secondary saccades (see chapter 2.6) and highlighted that they have been primarily regarded as small eye movements that correct for primary saccade error. This dominant perception as corrective secondary saccades implied an automatized computation of secondary saccades as a result of the primary saccade. Such an approach falls short of explaining several findings like the observation of secondary saccades following precise primary saccades. Moreover, viewing secondary saccades as purely corrective distracts from the alternative view that secondary saccades, alike presaccadic microsaccades, are the outcome of an activity distribution within a saccade motor map that crosses the triggering-threshold for an eye movement at a specific site in the motor map. Important questions arising from such an alternative perspective ask for the factors that influence the overall postsaccadic activity distribution in the saccade motor map.

In the first experiment (see chapter 3) we were interested to examine potential error-related and error-unrelated influences on the programming of secondary saccades during postsaccadic fixation when visual feedback is available. We replicated a strong influence of primary saccade error on the latency, amplitude and orientation of secondary saccades. This error-related influence can be explained by the presence of a postsaccadic visual target, which easily allows for the computation of an error signal. Importantly, we also identified non-error related influences on secondary saccade characteristics.

We observed different secondary saccade characteristics depending on whether the primary saccades undershot or overshot their target. Moreover, we demonstrated a strong bias of secondary saccades to follow the direction of the primary saccade. Target eccentricity significantly influenced the latency, amplitude, orientation and number of secondary saccades, which is not predicted by a pure error-correction account of secondary saccades. First analyses even imply that the generation of secondary saccades is even influenced by the type of cue (exogenous vs. endogenous) during presaccadic fixation.

These error-unrelated influences support the view that secondary saccades need to be explained by a model that integrates influences in addition to error-related signals. We extended a recently introduced model of microsaccade generation (Rolfs, Kliegl, & Engbert, 2008; see also chapter 2.5). In our model we proposed that baseline activation in the saccade motor map is increased in the hemisphere that programmed the primary saccade. After primary saccade undershoot the visual target will elicit activation within the hemisphere where the primary saccade has been programmed.

Consequently, secondary saccades should be generated more likely after primary saccade under- as compared to overshoot. Furthermore, we predicted that this difference between under- and overshooting should increase with increasing target eccentricity. Finally, larger primary saccade error was thought to increase the likelihood to observe a secondary saccade. Several statistical challenges had to be addressed in order to test our model predictions. These challenges included the possibility to assess the influence of continuous and categorical variables on a rate, possible underlying time-dependent influences, and the fact that in a subset of trials no event was observed.

We proposed a solution to these statistical constraints in form of Aalen's additive hazard model, which is a non-parametric (regression-like) model that belongs to the family of survival analyses. It allowed us to assess time-dependent influences of continuous and categorical variables on a rate, which provides a potential powerful technique for future research on eye movements in general. The results obtained with Aalen's additive hazard model confirmed the hypotheses and provide additional support for the assumptions underlying our proposed qualitative model. Future studies using Aalen's additive hazard model should also address individual differences which will further increase the usefullness of such analyses for research on eye movements.

In the second experiment (see chapter 5), we studied secondary saccades when no postsaccadic visual feedback was available. This study was designed in order to examine whether an extra-retinal error signal is computed and influences the generation of secondary saccades. Several studies addressed this topic over the last five decades (Becker & Fuchs, 1969; Deubel, Wolf, & Hauske, 1982; Morel, Deneve, & Baraduc, 2011; Prablanc & Jeannerod, 1975; Shebilske, 1976; Weber & Daroff, 1972) but the results so far were very inconsistent and contradictory which might be due to differences in the experimental methods but also due to intransparent pre-processing criteria. Also, the rare studies supporting the notion of an extra-retinal error influence on the generation of secondary saccades demonstrated only necessary but not sufficient evidence. Those studies showed that strongly undershooting primary saccades to very distant targets were followed by secondary saccades in the same direction as the primary saccade. Such a result can also be explained by a general bias of secondary saccades to follow primary saccade direction. Obviously, this explanation would make an extra-retinal error signal needless. We highlighted that convincing evidence for an extra-retinal error influence is only provided when undershooting primary saccades are followed by secondary saccades in the same direction and overshooting primary saccades are followed by secondary saccades in opposite direction.

In our experiment we observed that the probability of secondary saccades to be directed opposite to the primary saccade increased as primary saccade error shifted from under- to overshoot. This result strongly supports the notion of an extra-retinal error signal that influences the generation of secondary saccades. In addition, our results support a recent proposal by Collins and colleagues (2009) that an efference copy (of the saccade command) includes already information about the oculomotor error (e.g., the landing position error).

An extra-retinal error signal that impinges on a saccadic motor map can also account for the observation of short-latency secondary saccades when postsaccadic visual feedback is available. In such a situation the extra-retinal error signal can increase activation at the predicted location of the postsaccadic visual target. Activation elicited by the postsaccadic visual target can then add to the pre-built activation and cause fast crossing of the triggering-threshold. Again we observed a significant influence of target eccentricity, which provides additional evidence for an important error-unrelated influence. As previously described our model provides a possible explanation for an effect of target eccentricity but elucidating the exact underlying mechanism will certainly remain an important topic for future studies.

I also want to highlight a potential use of the presented paradigm for future research on influences around saccadic eye movements. Removing the influence of the postsaccadic visual stimulus may allow us to uncover the dynamics of extra-retinal signals around saccadic eye movements. In particular, the use of additive hazard models in combination with our paradigm can answer whether a given continuous or categorical variables exerts a significant influence on a (post-) saccadic motor map and whether such an influence is time-dependent or remains constant over time.

A future interesting study would be to examine the influence of postsaccadic visual white noise on characteristics of secondary saccades. We argued that activity in the motor map often remained under the triggering threshold despite the activity arising from an extra-retinal error signal. Following the principles of stochastic resonance, adding visual white noise might help activity to cross the threshold for secondary saccade triggering. Consequently, the number of corrective secondary saccades could be increased despite the presentation of a spatially uninformative visual signal. I want to highlight that this paradigm might be even interesting for cross-modal studies, using auditory white noise as postsaccadic stimulus.

In the analyses of our experiments we focused on the first postsaccadic eye movement that was observed. Importantly, additive hazard models provide the opportunity to go beyond the first postsaccadic eye movement by including also subsequent eye movements during postsaccadic fixation. Such an analysis of recurrent events is not only interesting for a time interval during postsaccadic fixation. Obviously, such type of analysis could also be applied to microsaccades during presaccadic fixation, which may be particularly interesting as it allows for quantification of the influence of continuous variables on a rate in a regression-like manner.

So far I highlighted the need to account for the characteristics of secondary saccades by a model that incorporates error-related and error-unrelated influences. I proposed to use models of microsaccade generation as secondary saccades are small-amplitude eye movements during postsaccadic fixation, which include a large proportion of microsaccades. I discussed the importance of the postsaccadic visual stimulus, an extra-retinal error signal, and target eccentricity for the latency, amplitude, orientation, and number of secondary saccades.

Over the last years, additional models of microsaccade generation have been introduced that go beyond the qualitative model by Rolfs, Kliegl and Engbert (2008) which we relied on in our studies (see chapter 2.5 and 3). Indeed, computational models have been introduced which allow precise quantitative predictions on fixational eye movements including microsaccades and drift (Engbert et al., 2011).

An important future step will be to predict the observed results on secondary saccades also in the framework of a quantitative model. In the model proposed by Engbert and colleagues (2011) the error in primary saccade landing position could be modeled by an additional potential that represents a valley in the activity distribution in which the walker would be situated at the beginning of postsaccadic fixation. The intended landing position of the primary saccade would be indicated by the center of the lattice. As a consequence of the additional potential, the walker would climb up within the additional potential when primary saccade error is sufficiently large. After crossing the threshold for triggering an eye movement the walker would move to the position with minimum activation which would be located closer to the center. Consequently, the additional potential could be used to account for error-related influences on secondary saccade characteristics. Also, such a quantitative model can generate very precise predictions concerning the dynamics of secondary saccades and the time-dependent influences of primary saccade landing position on the characteristics of secondary saccades.

Above I have shown that an extra-retinal error signal is computed which influences the generation of secondary saccades. In a next step, we were interested to examine how such an extra-retinal error signal and secondary saccades influence the performance in a postsaccadic location judgment task. Typically subjects are unaware of experimentally induced small shifts in target location that occur during saccadic eye movements. The shift is attributed to oculomotor error and not to a shift in target location, which consequently allows the perception of a stable visual world. Not very intuitively this stable perception can be broken by inserting a blank for a short duration at the beginning of postsaccadic fixation. In such a condition, subjects become aware of small shifts in target location that occurred during the saccade, demonstrating that precise information about postsaccadic target location is not lost across saccadic eye movements (Deubel, Schneider, & Bridgeman, 1996).

In a recent study it has been proposed that an efference copy is used to derive a prediction of the postsaccadic target location (Collins et al., 2009). Such an efference copy signal is thought to include already information about the oculomotor error associated with the primary saccade. Comparison of this efference copy with the initial target vector allows a precise prediction of the postsaccadic target location. Nevertheless, this information (which is equal to an extra-retinal error signal) becomes only accessible when a blank is inserted at the beginning of postsaccadic location; otherwise this information will be overwritten by the incoming postsaccadic visual stimulus and by the processes which enable a stable perception of the world across saccadic eye movements.

In two experiments we were interested to study the nature of the blanking effect in more detail (see chapter 6). More specifically, we asked whether the improved performance in location judgments results from a period without visual information or whether the same performance increase can be obtained by ambiguous visual information. We reasoned that identification of a postsaccadic landmark would be disturbed after the presentation of many target-like objects during postsaccadic fixation. Thus, presenting ambiguous postsaccadic visual information may be sufficient to prevent that the precise postsaccadic location information is overwritten. Such a result would open a new door to study the dependency of accessibility to precise postsaccadic location information on characteristics of the postsaccadic visual stimulus configuration.

Previous studies indicated that information about precise postsaccadic target location is available to the motor system. Our results provided additional support for that claim as the orientation of secondary saccades was significantly influenced by primary saccade landing site despite the presentation of multiple target-like objects. Secondary saccades followed the direction of primary saccades after target undershoot and reversed the direction after overshoot of the target. Also, the amplitude of secondary saccades increased with increasing absolute primary saccade error.

Nevertheless, the potential influence of an extra-retinal error signal in the oculomotor system does not necessarily imply that this information can be used for perceptual location judgments. Indeed, our results demonstrated that a large proportion of postsaccadic location judgments indicated a wrong target location. Moreover, we emphasized the importance to include secondary saccades as covariate when studying postsaccadic location judgments. When no secondary saccade was observed, location judgments had a positive relationship with primary saccade landing position. In contrast, after the generation of secondary saccades, location judgments displayed a positive relationship with secondary saccade landing position. Overall, our results did not support our hypothesis that the presentation of ambiguous postsaccadic visual stimuli may disturb the process of early postsaccadic space calibration. We simply observed too many wrong location judgments. We proposed that in our experiment, visual stimuli close to the saccade landing site were predominantly chosen as landmark for postsaccadic space recalibration and consequently as target location. Nevertheless, the extra-retinal error signal was able to elicit secondary saccades that significantly influenced the postsaccadic location judgment.

Finally, we argued that the computation of postsaccadic location judgments should also result in different degrees of confidence associated with each judgment. Reporting the confidence of judgments adds a new dimension to the field of trans-saccadic perception. We observed in both experiments that a large distance between correct and chosen target location resulted in less confident judgments. This is an interesting result as it implies that the computation of confidence may be influenced by the precise prediction of postsaccadic target location. Although this information influences the computation of confidence, it was not used in order to make a better postsaccadic location judgment in our study. Confidence judgments varied strongly between subjects and were often influenced by large individual response biases.

To sum up, the built-in assumption of a stable visual world should also comprise the confidence that the position of objects did not shift during the saccade. Future studies that examine confidence for postsaccadic location judgments promise to further increase our understanding of perceiving a stable visual world despite saccadic eye movements. A promising next experiment would be to examine confidence judgments in the classical paradigm studying postsaccadic location judgments. In one version of this paradigm the target is shifted by a constant size either to the left or to right during saccadic eye movements. Inserting a blank of varying durations at the beginning of fixation (or of a constant duration at different points during postsaccadic fixation) can determine the development of improved perceptual performance. Simultaneous recording of confidence associated with each location judgment can depict the development of confidence as a function of blank-duration (or time of blank presentation) and provide further insights into the question how confidence and performance evolve during postsaccadic fixation.

Although our experiments can be regarded as a new step to examine continuous-like location judgments instead of 2-AFC, there are also drawbacks related to the current paradigm. Foremost, in our paradigm it turned out to be difficult to determine whether performance was good or bad, which is important in order to derive conclusions on the influence of an extra-retinal prediction of the postsaccadic target location. Additionally, in our study we examined the relationship between secondary saccades and postsaccadic location judgments. Secondary saccades were not under experimental control, which consequently does not allow us to derive conclusions on the causal direction of such an influence. Finally, we did not observe significantly better location judgments after we included an additional blanking at the beginning of postsaccadic fixation. This might point to the problem that our multiple target-like object array may be a too strong visual transient, which effectively overwrites signals within the visual processing pathway.

In the present thesis I studied postsaccadic location judgments, which obviously reflect only one small facet of visual perception. The influence of small eye movements during postsaccadic fixation on vision may also encompass a similar function as supposed for microsaccades during prolonged presaccadic fixation, namely refreshing the retinal image. In any case, studying visual perception during postsaccadic fixation and the influence of secondary saccades constitutes a promising approach as visual information uptake typically occurs during postsaccadic fixation.

8.2 The influence of preparatory processes on microsaccades

In a second goal of this thesis I examined the interplay of microsaccades and preparatory processes. How are preparatory processes implemented in the oculomotor system? There seemed to be a convincing answer to this question, which focused on top-down control of fixation neuron activity. From this perspective saccadic responses can be delayed (e.g. in the antisaccade task) by increasing the level of activation of fixation neurons in the rostral pole of the superior colliculus. This increased fixation neuron activity then inhibits saccade neuron activity and consequently delays saccadic reaction times.

Following from the above discussed shift in paradigm, the rostral pole of the superior colliculus is now thought to code for small-amplitude saccades and microsaccades. It has been argued that the previously assumed increased activity of fixation neurons could be translated into increased fixation-related activity, which should consequently result in an increasing number of microsaccades

In the present thesis I examined the influence of the preparatory set on microsaccades during presaccadic fixation. More specifically, I was interested whether presaccadic microsaccade rate differs between blocks of endogenously vs. exogenously defined targets. The endogenous cue was presented in the center of the screen while exogenous cues were presented in the periphery. It was hypothesized that shifting attention to the future location of the cue would increase activity at the corresponding site in the saccadic motor map. We reckoned that shifting attention to the endogenous cue would increase activity in the center of the map (where microsaccades are programmed). Consequently, we hypothesized that microsaccades rate should be increased in blocks of endogenous as compared to exogenous cues.

In line with this prediction microsaccade rate was increased in blocks of endogenous as compared to exogenous cues. Most importantly, this result further supports the notion of preparatory influences in the oculomotor network which can be uncovered by microsaccades. Moreover, we tested whether preparatory influences depend on target eccentricity. We demonstrated that microsaccade rate was higher in the close target condition (particularly before exogenous cues). This result implies that effects of attention on microsaccades depend on the distance of the attentional shift.

Overall, these results were in line with predictions from the model proposed by Rolfs, Kliegl and Engbert (2008). An additional prediction of this model states that microsaccades occurring around the presentation of a go signal should increase saccade latencies. It is thought that microsaccades mirror an increased level of fixation-related activity which inhibits activation at distant locations in the saccade motor map where large saccades are programmed. Such microsaccade-induced prolongation of saccadic response times has been repeatedly observed (Hafed & Krauzlis, 2010; Kliegl et al., 2009; Rolfs, Laubrock, & Kliegl, 2006, 2008; Sinn & Engbert, 2011). We now demonstrated that this is also true in blocks of endogenously defined saccades irrespective of target eccentricity.

Nevertheless, we also highlighted that such a result is also predicted by microsaccade-induced visual suppression (Hafed & Krauzlis, 2010; see also Rolfs & Ohl, 2011). From that perspective, prolonged response latencies result from a weaker visual go signal (due to visual suppression). We discussed that a visual suppression account indeed is very likely to influence response latencies, but it may be insufficient to account for the long time-course of microsaccade-induced prolongation of reaction times. Indeed, prolongation starts much earlier than predicted by visual suppression alone.

A future interesting experiment could test the influence of microsaccades around go signal presentation on manual reaction times. A pure motor account as proposed by Rolfs, Laubrock and Kliegl (2006) would not predict a significant prolongation of manual response latencies while the visual suppression account, in contrast, would predict prolongations of reaction times only in a narrow time window around go signal presentation (about 70 ms). This experiment opens the interesting possibility that microsaccades may prolong manual responses, alike for saccadic responses, in a much earlier time window. Such a result could not be explained by the two proposed mechanisms and would further stimulate the discussion on how response latencies are influence by microsaccades.

8.3 Conclusions

I investigated small eye movements during pre- and postsaccadic fixation. Fixation is far from being a passive mode that only has the purpose of visual information uptake, and is often used as control condition in psychological experiments. Indeed eye movements during fixation can be very sensitive to experimental manipulations and consequently provide very useful information on very different topics in perception, action and cognition in general. In the present thesis I add new findings to the fast growing body of literature concerning small-amplitude saccades during fixation. In particular, I demonstrate that postsaccadic fixation is a promissing environment for studying fixational eye movements and falsifying current models of microsaccade generation. I reported evidence for multiple factors (error-related and error-unrelated signals) that influence the computation of small saccades and highlight that the classical field of secondary saccades needs to be addressed when models of microsaccade generation should also account for eye movements during postsaccadic fixation.

In recent years, many studies demonstrated a cognitive influence, in form of attention, on microsaccades. In the present thesis I uncovered an additional cognitive influence, in terms of preparatory signals, on microsaccades during presaccadic fixation. Such a finding is important as it demonstrates the need of models to account for both stimulus-driven and top-down influences on microsaccades. Future findings in combination with computational models will certainly increase our understanding how such signals unfold and influence small eye movements during fixation.

Appendix A

Supplementary material for chapter 3

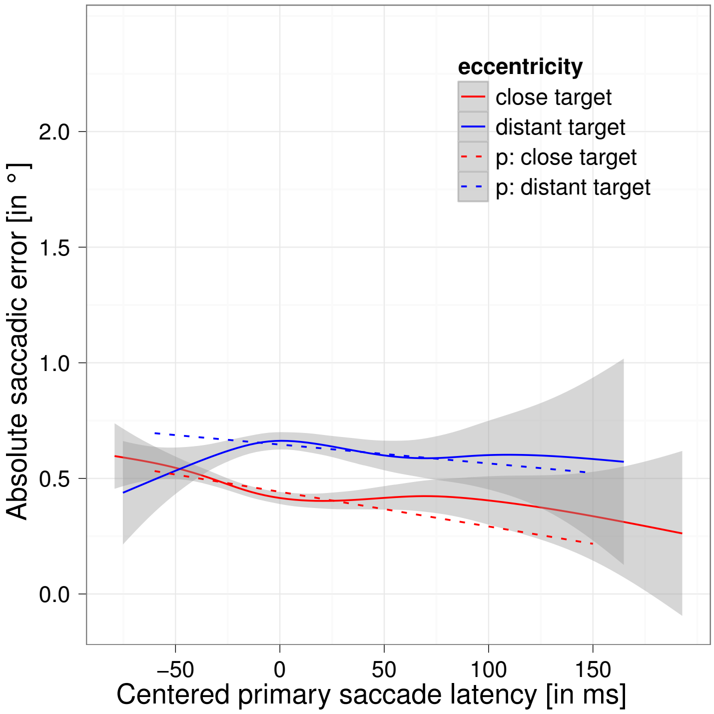

Figure A.1: Absolute primary saccade error as a function of (centered) primary saccade latency by target eccentricity. Smoothing (solid line) is based on loess method (degree = 2). Grey bands represent the 95% confidence interval. Prediction (p: close target, p: distant target) of the LMM (dashed line) after removal of between-subject variance.

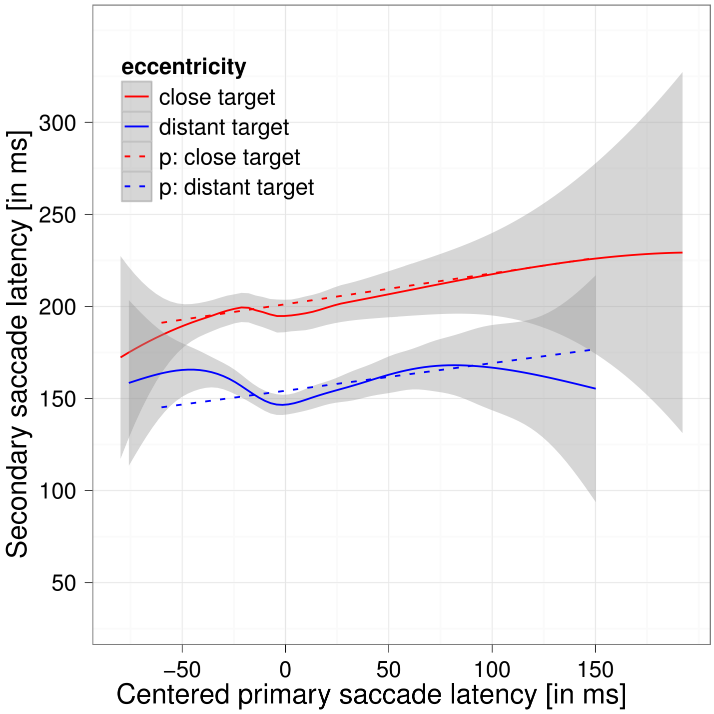

Figure A.2: Secondary saccade latency as a function of (centered) primary saccade latency by target eccentricity. Smoothing (solid line) is based on loess method (degree = 2). Grey bands represent the 95% confidence interval. Prediction (p: close target, p: distant target) of the LMM (dashed line) after removal of between-subject variance.

Table A.1: Absolute primary saccade error as function of primary saccade latency and target eccentricity. Eccentricity is coded as 0 (close targets) and 1 (distant targets). Saccadic reaction times of primary saccades are centered (SRTc). Subjects and eccentricity were included as random effects.

	Estimate	SE	*t*-Value
Intercept	0.442	0.027	16.2
Eccentricity (ecc)	0.205	0.044	4.7
SRTc	-0.001	0.0004	-3.9
SRTc x ecc	0.0007	0.0006	1.2
Variance components		SD	
Subjects		0.079	
Eccentricity		0.128	

Table A.2: Lmm statistics for saccadic rhythm account. Secondary saccade latency as a function of primary saccade latency and target eccentricity. Eccentricity is coded as 0 (close targets) and 1 (distant targets). Saccadic reaction times of primray saccades are centered (SRTc). Subjects and eccentricity were included as random effects.

	Estimate	SE	*t*-Value
Intercept	201.2	5.34	37.7
Eccentricity (ecc)	-46.97	9.14	-5.1
SRTc	0.17	0.08	2.2
SRTc x ecc	-0.02	0.16	-0.2
Variance components		SD	
Subjects		15.25	
Eccentricity		27.36	

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