

IN-BETWEEN FIXATION AND MOVEMENT:

ON THE GENERATION OF MICROSACCADES AND WHAT THEY CONVEY ABOUT SACCADE PREPARATION

by

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Introduction

When open, our eyes continuously move such that virtually at no point in time they will rest in the same place as in the moment before. Different kinds of movements contribute to this behavior. On the one hand, eye movements accomplish the task of keeping the image relatively stable on the retina; for instance, we keep track of a moving object by following its path of motion with *smooth pursuit eye movements*. On the other hand, we move our eyes in order to acquire new information in different parts of the scenery. To change the depth of the plane, for instance, the eyes *converge* or *diverge*, moving gaze to a closer or, respectively, to a further away object. The most noticeable behavior that our eyes exhibit, however, is the scanning of visual scenes in sequences of *saccades* and *fixations*. By definition, saccades and fixations are alternating states. Saccades are rapid eye jumps that bring the line of sight to areas of interest. During fixation, the eyes rest relatively stable at a point in the scene; in this state, we are able to acquire visual information. The term “fixation”, however, is somewhat misleading. Even while gazing at a certain location in space, the eyes will never be perfectly motionless. Instead, they will drift slowly with respect to the scene and once or twice per second, small involuntary so called microsaccades will intrude. Usually we are not able to become aware of these *fixational eye movements*, but as you will see below, they are indispensable for perception.

Thus, there is a whole toolbox of eye movements available to the visual system enabling it to flexibly acquire visual information. While each type of eye movement has been the subject of intense study, their interplay is often poorly understood. The thesis at hand is concerned mainly with fixational eye movements, especially microsaccades, and their relation to large-scale saccades. Only a handful of published studies relate to the interactions of these movements, possibly, since most of the time they amounted to null results. To get a broader understanding of potential interactions between large-scale saccades and microsaccades, a variety of behavioral

experimental paradigms and analytical techniques were employed here. The outcomes of these analyses give several new insights: First, we demonstrate strong relations between oculomotor processes during fixation and the latency of saccades. Second, we reduce the number of possible mechanisms underlying the interactions of microsaccades and saccades and infer implications on what microsaccade statistics convey about saccade preparation. Third, we discuss probable neurophysiological implementations of the generation of microsaccades within the circuitry of oculomotor control.

In the first chapter we introduce the three basic types of eye movements usually observed while the eyes fixate on a target in the visual scene. Chapter 2 is devoted to the literature concerning their relevance to visual information processing and oculomotor control. In Chapter 3, we present an overview of the neurophysiology of the oculomotor system along with the literature on potential neural substrates of microsaccade generation. We introduce a qualitative model incorporating our view as to how microsaccades are implemented and derive predictions for empirical work examining the relationship between microsaccade and saccade generation. In Chapters 4 to 6, we present the outcomes of a series of experimental examinations concerning the statistics of microsaccades and their impact on saccade characteristics in a variety of paradigms, thereby evaluating and constraining the model presented in Chapter 3. Finally, we summarize the outcomes of the present thesis and draw its conclusions in Chapter 7.

Chapter 1

Three types of fixational eye movements

It is not clear, when the first observation of fixational eye movements (FEyeM) was reported (cf., Wade & Tatler, 2005). Various authors noted very early that the eyes are never at rest. For instance, when discussing the anatomy of the eye, du Laurens reported that “the eye standeth not still but moveth incessantly” (1599, pp. 28-29). In a letter exchange with Perault, treating the question where visual perception begins—in the choroide or in the retina, Mariotte (1683) stated that “the *Eyes* are always in motion and very hard to be fixt in one place, tho it were desired” (1683, p. 266, original italics). We do not know, however, whether these notions really implied eye movements during fixation. Jurin (1738) was the first who definitely proposed that eye movements persist even during attempted fixation (see Kästner, 1755, for a German translation of J. Jurin’s essay). He made the following observation: To distinguish two separate marks they must have a distance greater than that needed to perceive a single mark in one location or another. Jurin suggested that the eyes “tremble”, causing two separate marks to merge into one percept. The first empirical evidence for the fact that the eyes move even during fixation was afforded by Robert Darwin, the father of Charles Darwin, who studied afterimages of colored stimuli (see Figure 1.1, for a demonstration). Darwin (1786) noticed that while one is trying to fixate a colored circle, a lucid edge is seen to liberate to the white-paper background. He concluded that “as by the unsteadiness of the eye a part of the fatigued retina falls on the white paper” (p. 341). Eighty

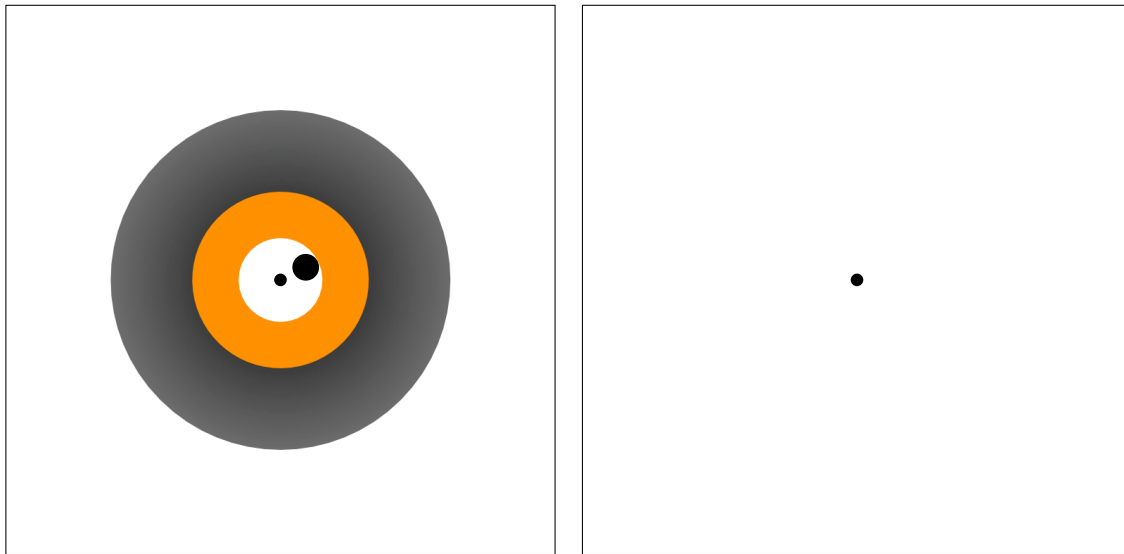


Figure 1.1: One's own fixational eye movements can be observed by inducing after images. Put the image at a distance of 20 cm in front of you. Fixate the spot at the center of the left image for about 30 sec. Soon you will notice lucid edges liberating from the colored circle. If you now look at the spot in the white field to the right, you will perceive an after image of the picture, which (for evident reasons) displays an eye ball. The after image constantly follows your eye movements. Note that the eye is never perfectly motionless, even when you try to hold it as still as possible. You will also see the after image fade spontaneously, especially if you try to omit eye movements.

years later, Hermann von Helmholtz (1924, German original published in 1866) remarked that “it requires extraordinary effort and attention to focus the gaze perfectly sharply on a definite point of the visual field even for 10 or 20 seconds.” (p. 266). He called this phenomenon the “wandering of the gaze” and proposed that the function of this motion would be to prevent retinal fatigue. As will be seen below, this proposal has been strongly substantiated since then. At those times, however, the accurate measurement of these miniature eye movements was technically impossible.

Objective measurements of FEyeM were made from the end of the 19th century on. For instance, Huey (1900) analyzed fixation durations in reading. He reported that the eyes moved even during “steady fixation”. Further early studies confirmed the existence of FEyeM (Dodge, 1907; McAllister, 1905), soon providing more detailed descriptions of this behavior (Adler & Fliegelman, 1934; Lord & Wright, 1948; Marx & Trendelenburg, 1911). Today it is generally accepted, that FEyeM are composed of three different types of movement: *tremor*, *drift*, and *microsaccades* (see Martinez-Conde, Macknik, & Hubel, 2004, for a recent review). Researchers in the field agreed on this typology in the early 1950s (Barlow, 1952; Ditchburn & Ginsborg, 1953;

Ratliff & Riggs, 1950), when the technical equipment of eye-movement recording was improved so far as to accurately visualize tremor, the smallest component of FEyeM.¹ In the following subsections, we will shortly describe the individual characteristics of these three types of FEyeM. A more exhaustive review of this topic was achieved by Martinez-Conde et al. (2004).

1.1 Drift

A characteristic trajectory of FEyeM is erratic and coined mainly by a low-velocity component, the so called drift movements (dark parts of the eye-movement trace in Figure 1.2). By definition, drifts occur during the inter-saccadic intervals and can be described as a random walk (Engbert & Kliegl, 2004; Matin, Matin, & Pearce, 1970). In these intervals, they carry the retinal image by about 1 to 8 min-arc at a speed (mostly well) below 30 min-arc per second.

It was subject to discussion whether or not drift movements are correlated between the eyes. Krauskopf, Cornsweet, and Riggs (1960) and Yarbus (1967) found no correlation of drift movements in the two eyes. In contrast, Riggs and Ratliff (1951) described that in their data drift movements were in general closely synchronized. Ditchburn and Ginsborg (1953) reported conjugacy for both the vertical and horizontal components of drift, the direction of these movements changing at random times. Moreover, the authors found intervals of convergence and divergence, respectively, for the horizontal components. According to Ditchburn and Ginsborg (1953), however, this effect could be attributed to accommodation. More recently, binocular coherence of drift (and microtremor) was also proposed by Spauschuss, Marsden, Halliday, Rosenberg, and Brown (1999, see description of microtremor). Finally, Thiel, Romano, Kurths, Rolfs, and Kliegl (submitted) used a sophisticated surrogate data method (see also Thiel, Romano, Kurths, Rolfs, & Kliegl, 2006) to demonstrate significant phase synchronization of FEyeM, mainly consisting of drift, between the two eyes but not between vertical and horizontal components of one eye. The authors proposed that motor neurons as the final common pathway of neural control of eye movements are candidates for the synchronization of fixational movements of both eyes. Note, that coherence and synchronization do not distinguish between conjugacy, convergence, and divergence. Rather, these terms refer to a coordinated behavior of drift in both eyes including velocity, acceleration, and, thus, also changes in movement direction.

¹The existence of tremor was first reported by Adler and Fliegelman (1934), but then doubted by some authors (e.g., Hartridge & Thomson, 1948; Lord & Wright, 1948).

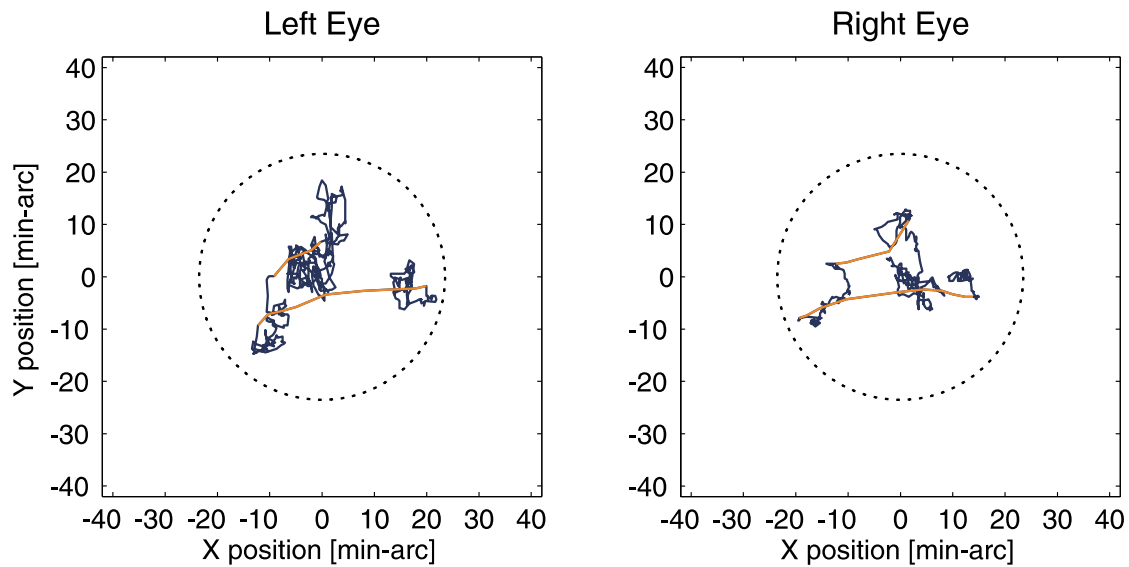


Figure 1.2: Trajectory of the two eyes during 1 s of fixation on a spot (dotted circle). Slow drift movements are displayed in dark color while rapid jerk-like microsaccades are highlighted in orange.

1.2 Microtremor

Microtremor (also tremor or physiological nystagmus) is an irregular, wave-like motion superimposed on the drift movements described before. Tremor has a high frequency and a small extent. The reported frequency and amplitude values, however, deviate between various studies; this can be attributed to individual differences (Barlow, 1952; Ratliff & Riggs, 1950) and different recording methods used in different laboratories (Ditchburn & Ginsborg, 1953; Simon, Schulz, Rassow, & Haase, 1984). Mostly, average frequencies of 90 Hz are reported with movement cycles having amplitudes of 0.1 to 0.5 min-arc (e.g., Adler & Fliegelman, 1934; Ratliff & Riggs, 1950; Higgins & Stultz, 1953).²

As with drift, binocular studies of FEyeM revealed different patterns of results. Early binocular examinations did not find a correlation of microtremor between the eyes (Ditchburn & Ginsborg, 1953; Riggs & Ratliff, 1951). More recently, however, evidence for a coherence of tremor movements in the two eyes emerged. Spauschuss et al. (1999) examined frequency components of ocular drift movements and microtremor in both eyes simultaneously. After a correction for head movements,

²Special high-resolution equipment is needed to record motion of this type (Bengi & Thomas, 1968; Yarbus, 1967). In the experiments reported in this thesis, we used a video-based eye-tracking system (see experimental sections), which is not capable of measuring microtremor. Therefore, this component of FEyeM is not part of the trajectory in Figure 1.2.

the authors found coherence of the eyes' accelerations in both low- (up to 25 Hz) and high- (60 to 90 Hz) frequency ranges. The authors suggested that these FEyeM might be related to the patterning of low-level but central drives to the extra-ocular muscle motor units. Concerning the high-frequency movements, this is in agreement with clinical studies showing a reduction or abolition of microtremor in disease of the brain system (Michalik, 1987) and in comatose patients (Shakhnovich & Thomas, 1977), respectively.

1.3 Microsaccades

At a typical rate of once or twice per second, the slow drift movements of the eyes during fixation are interrupted by small rapid shifts in eye position (orange parts of the eye-movement trace in Figure 1.2). This was first discovered by Dodge (1907). In most of the recent studies (including the thesis at hand) these jerk-like movements were called microsaccades, a term introduced by Zuber, Stark, and Cook (1965). It should be noted, however, that many terms can be found in the literature, including small, miniature, or fixational saccades, jerks, flicks, jumps, and so forth.³ Microsaccades clearly differ from drift movements by their high velocity, a fact used to detect these events in a stream of FEyeM data (e.g., Engbert & Kliegl, 2003b). Though monocular ones can be observed (Engbert & Kliegl, 2003a), microsaccades are binocular movements in most cases. Moreover, they show a clear preference for horizontal and vertical directions, the latter being clearly less frequent. Oblique directions are observed rather exceptionally (Engbert, 2006b).

All types of saccades (including most microsaccades) share the most important characteristics: They are conjugated binocular high-velocity movements with a distinct correlation of peak velocity and movement amplitude (the so-called main sequence; Zuber et al., 1965). It is a matter of definition how to tell apart microsaccades from other saccades. Since, by definition, microsaccades are movements *during* fixation, the overlap of the retinal image on the fovea before and after the shift should be substantial. Thus, a maximum of 1° appears to be an appropriate amplitude criterion, given the fovea covers an angle of 2°. Empirically, microsaccade amplitudes are rarely found to exceed 30 min-arc. However, microsaccades cannot be defined solely on the basis of

³Another labeling confusion comes from a recent study that aimed to subdivide several saccade-saccade and saccade-drift combinations during fixation (Abadi & Gowen, 2004). To sum up these different fixation behaviors, these authors used the generic term *saccadic intrusions*. Originally, this term comes from the clinical literature and describes non-repetitive saccadic interruptions of fixation in patients with ocular instabilities. It is often used synonymously with its most frequent sub-category, *square-wave jerks*, paired saccades away from and back to a target separated by some 200 ms (Ciuffreda & Tannen, 1995). In terms of the definition of microsaccades used in the present study, such instances would be treated as two separate movements.

amplitude, since saccades with amplitudes of less than 5.7 min-arc can be performed voluntarily in the presence of a stationary fixation target (Haddad & Steinman, 1973). In agreement with the recent literature (cf., Martinez-Conde et al., 2004) and the conventions in our group (Engbert, 2006b; Engbert & Kliegl, 2003a, 2003b, 2004; Laubrock, Engbert, & Kliegl, 2005; Laubrock, Engbert, Rolfs, & Kliegl, in press; Rolfs, Engbert, & Kliegl, 2004, 2005; Rolfs, Laubrock, & Kliegl, 2006), we will use the term microsaccades when referring to spontaneous, involuntary saccades of small amplitude ($\leq 1^\circ$) that occur during intended fixation. See Engbert and Kliegl (2003b), Engbert (2006b), and the methods sections, respectively, for the operational definition of microsaccades used for all analyses reported here.

Chapter 2

Is there a purpose to microsaccades and other fixational eye movements?

As we have seen above, it is a long known fact that our eyes continually move even during fixation. Are oculomotor mechanisms just not sufficiently accurate to realize perfect retinal stabilization or does this continuous variation in eye position have an important purpose? Do the different types of fixational eye movements (FEyeM) serve different functions? In this section we will review the various attempts that have been made (successively or not) to answer these questions. We will put some emphasis on the efforts that aimed to illuminate the specific functions of microsaccades.

2.1 Prevention of perceptual fading

The first approach to test the significance of FEyeM for visual perception was quite straight forward—FEyeM were effectively switched off. In a series of studies, the retinal image was artificially stabilized in human participants. Already in the early 50s of the last century, scientists developed techniques to largely counteract image motion with respect to the eye. At that time, the main method to stabilize the retinal image was to attach an optical system to the eye by using a contact lens. In that way, target stimuli presented through this system would constantly move with the eye. The results of these studies were striking: When the visual environment falls completely static on our retinae (as it nearly does under conditions of retinal stabilization), it fades from visual perception within a few seconds (Ditchburn & Ginsborg, 1952; Pritchard, 1961;

Pritchard, Heron, & Hebb, 1960; Riggs & Ratliff, 1952; Riggs, Ratliff, Cornsweet, & Cornsweet, 1953; Yarbus, 1957a, 1957b; Heckenmueller, 1965, carefully reviews these early findings).

The disappearance times reported in these early studies, however, have to be interpreted with caution, since the stabilization methods used may have been faulty to some extent (cf., Barlow, 1963), e.g., due to slippage of the contact lens.¹ Later studies used retinal stabilization techniques that make entoptic structures visible to the viewer (Campbell & Robson, 1961; Sharpe, 1971). By nature, these structures are virtually perfectly stabilized with respect to the retina since they are part of the eye itself. Ratliff (1958) proposed to take advantage of the phenomenon of "Haidinger's Brushes", entoptic images that can be seen by simply looking through a polarizer at a field of blue light. Interestingly, the perception of these brushes is transient as it would be expected from retinal stabilization studies. The same is true for Maxwell's spot and a visual phenomenon reported by Shurcliff (1959), which he called the "greenish-yellow blotch". Shurcliff (1959) had 110 observers viewing a uniformly colored field. Ninety-five of them reported that the perception of the field suddenly becomes broken up into blotchy areas of two very different colors—mostly in the range between yellow and green. After a few seconds, this pattern disappears, supposedly, since its position is stable with respect to the perceptual system (Alpern, 1972). Other authors used the shadow of blood vessels of the retina as visual stimuli, replicating the phenomenon of visual fading (Campbell & Robson, 1961; Drysdale, 1975; Sharpe, 1972). Again, the image faded over intervals of several seconds. Finally, using a very sophisticated illumination technique, Coppola and Purves (1996) demonstrated more recently that entoptic images of very fine blood capillaries disappeared in less than 80 ms on average, indicating that an active mechanism of image erasure and creation might be the basis of normal visual processing. Similarly, Rucci and Desbordes (2003) reported significant decreases in visibility of images presented for half a second. These retinal stabilization studies reinforce the idea that our nervous system has evolved to optimally detect changes in our environment. As a consequence, unchanging aspects of the visual field fade from

¹Barlow (1963) originally raised the debate about the quality of retinal stabilization using different techniques mainly because some authors reported that faded images reappear again after a few seconds, then to fade repeatedly (Barlow, 1963; Ditchburn, 1955; Ditchburn & Fender, 1955; Ditchburn, Fender, & Mayne, 1959; Ditchburn & Ginsborg, 1952; Ditchburn & Pritchard, 1956; Pritchard, 1961; Pritchard et al., 1960; Riggs & Ratliff, 1952; Riggs et al., 1953). Barlow (1963) argued that this effect may be caused by slippage of the contact lens attached to the eye. The author showed that under thorough control reappearance effects were much weaker and images did never reappear in all detail. The debate has not been settled (e.g., Arend & Timberlake, 1986; Ditchburn, 1987; Evans, 1965), first of all, because authors using suction caps to secure a strong attachment of the contact lens to the eye did not report reappearance of disappeared images (Gerrits, de Haan, & Vendrik, 1966; Yarbus, 1957a, 1957b), while after images of light flashes (which are most certainly fixed with respect to the retina) were observed to reappear after disappearance (e.g., Bennet-Clark & Evans, 1963).

view. To counteract this, our retinae have to move with respect to the visual surrounding. This strongly suggests that eye movements are essential to sustain visual perception during fixation.

All of the studies reported above, however, took place under typical, rather artificial laboratory conditions. Movements of the head and the torso were minimized by chin rests, bite boards, cheek pads, and so forth. This raised doubts in the significance of their results in understanding the maintenance of visual perception in natural situations (Kowler & Steinman, 1980; Steinman, 2003; Steinman, Haddad, Skavenski, & Wyman, 1973). Therefore, Skavenski, Hansen, Steinman, and Winterson (1979) examined retinal image motion under conditions of small natural and artificial body rotations. The authors reported substantial motion of the retinal image. A good deal of the body and head rotations was compensated for by eye movements (up to 90%), however, considerable retinal image motion remained—clearly more than under conditions of head fixation. Skavenski et al. (1979) argued that these additional movements could be sufficient to prevent any retinal fatigue. Indeed, Riggs et al. (1953) had shown before that if FEyeM were amplified by a factor of two, virtually no perceptual fading could be observed. The results by Skavenski et al. (1979) were confirmed under even more natural conditions—during active head rotations (Steinman & Collewijn, 1980; see also Steinman & Collewijn, 1978). Clearly, these papers were of outstanding interest to the field, since they first examined image visibility under more realistic conditions. The reported results, however, are not entirely conclusive with regard to the purpose of FEyeM, since these still took place in these studies. To date, as far as we know, there has been no study that eliminated FEyeM but provided an image to the retina that followed the motions of the head, which (to our mind) is the missing condition to disentangle the importance of both eye and head movements in the maintenance of visual perception. An important conclusion, however, could be drawn on the basis of the studies by Skavenski et al. (1979) and Steinman and Collewijn (1980): Oculomotor compensation for body and head rotations might not aim to achieve retinal image stabilization; rather it might adjust retinal image motion so as to be optimal for continuous visual processing under the range of natural body movements.

Assuming that FEyeM help to counteract retinal fatigue, it remains to be known which part of the motion contributes to this function. Ratliff and Riggs (1950) computed the amount of image motion FEyeM produce with respect to the photoreceptors on the retina. Drift movements cover up to a dozen photoreceptors. Equally, microsaccades can carry the retinal image over a dozen or more receptor cells, depending on the movement amplitude. Microtremor, in contrast, rarely exceeds

amplitudes that correspond to one retinal photoreceptor. Thus, while drift and microsaccades produce retinal image motion that might have a sensible effect on visibility, the importance of tremor in this regard appeared unlikely (Ditchburn, 1955; Krauskopf, 1957; Sharpe, 1972; Tulunay-Keesey & Riggs, 1962), unless it exceeds amplitudes of 0.3 min-arc and there is a summation of eye movements over the whole frequency spectrum (Ditchburn et al., 1959). In addition to these spatial aspects, tremor's temporal characteristics—its frequency is far above the flicker-fusion frequency of the human visual system—have raised further doubts in the significance of this motion for visibility (Ditchburn, 1955; Gerrits & Vendrik, 1970; Yarbus, 1967). Recently, however, Greschner, Bongard, Rujan, and Ammermüller (2002) reported that ganglion cells in the turtle retina were most effectively synchronized with imposed high-frequency low-amplitude motion similar to microtremor. Moreover, synchronization of cell-firing was shown to improve the estimation of the spatial frequency of stimuli. The authors concluded that tremor could advance stimulus feature estimation by the brain. In monkeys, Martinez-Conde, Macknik, and Hubel (2002) incidentally noticed that the activity of cells in the lateral geniculate nucleus, an early stage in the visual processing pathway of the brain, followed the refresh frequency of the monitor used for stimulus presentation (74 Hz). Thus, at early stages in human visual processing even microtremor might play a role in perception (cf., Martinez-Conde et al., 2004). To this date, however, this hypothesis has not been tested explicitly. In what follows, we will therefore focus on the role of drift and microsaccades in the maintenance of perception.

One method to examine the importance of different types of FEyeM for visibility is to eliminate FEyeM by means of retinal stabilization and then impose controlled movements with certain characteristics. To our knowledge, the first manuscript describing this approach was published by Krauskopf in 1957.² From this time on, however, this method became commonly used in many studies, revealing partially controversial findings.

Under conditions of retinal stabilization, Krauskopf (1957) introduced vibratory sinusoidal movements with amplitudes of 0.5 to 4 min-arc to the retinal image and varied their temporal frequencies. He found that movement frequencies of 1, 2, and 5 Hz decreased contrast thresholds when the movement amplitude was at least 1 min-arc; thus, these movements were beneficial for vision. In contrast, higher frequencies (10, 20, and 50 Hz) were detrimental for perception when

²Krauskopf (1957) acknowledges an earlier paper delivered by T.N. Cornsweet and L.A. Riggs at the Eastern Psychological Association Meeting in 1954. According to Krauskopf (1957), the results of these authors were in good agreement with his own.

compared to an image stabilized on the retina. He speculated that both drift and microsaccades could contribute to image visibility, but conceded that the motion induced in his study was largely artificial.

Subsequent studies corroborated the findings of Krauskopf (1957). Tulunay-Keesey and Riggs (1962) imposed oscillatory motion of different frequencies and amplitudes to an image otherwise stabilized with respect to the retina. Imposed motion was effective in increasing visibility times of their Mach-band stimuli, but only if it had a small frequency (about 3 cycles per second) and a large amplitude (greater than 1 min-arc per second). In a setup by Yarbus (1959), a glass capillary was mounted to a suction cap attached to the eye ball. Opaque fluids carrying an air bubble were moved through this capillary at different speeds. Yarbus (1959) showed that velocities of down to 3-5 min-arc per second prevented stimuli from disappearing, which under conditions of retinal stabilization had faded from perception. Similar conclusions could be drawn in a study by Fiorentini and Ercoles (1957), who imposed oscillatory motion to Mach band stimuli without making use of retinal stabilization, but controlling for FEyeM.

Ditchburn et al. (1959) studied image visibility while imposing motion imitating drift, microsaccades, and tremor, respectively, on an otherwise stabilized image. Visibility of faded images was not improved when imposing drift motion or microtremor by themselves. In contrast, simulated microsaccades strongly regenerated faded images to a very sharp percept which then faded again. It was concluded that microsaccades play a role in the maintenance of vision, but that the system cannot rely on these movements alone. The finding that microsaccades rate is lower when no stimulus is presented as compared to the presence of a fixation target (Nachmias, 1961), was in line with this proposal.

In a later study, Ditchburn, Drysdale, and Drysdale (1977b) investigated effects of step and pulse movements on the visibility of stimuli stabilized with respect to the retina. Step movements displaced the target abruptly to a new position, where it then remained. These movements clearly resulted in a sudden reappearance of the stimulus. The gain in visibility was a function of movement amplitude, with largest movements (24 min-arc) increasing visibility of the target most effectively and over long periods of time (up to 70 s). Pulse movements, also displaced the retinal image to a new position, however, returned it to the initial position after a short amount of time. Pulses did not notably enhance perception when return times were below 8 ms. Ditchburn et al. (1977b) argued that at such short times, the signal must be completely temporally integrated on

the level of photochemical processes in the receptors. The integration time appeared to be directly determined by neural processes accumulating just sufficient information to extract a signal from a background of photon noise. In a second paper, the consequences of oscillatory movements imposing sinusoidal, square-wave and triangular-wave motion of different frequencies and magnitudes were examined (Ditchburn, Drysdale, & Drysdale, 1977a). Imposed movement had two contradicting effects on the visibility of stimuli. First, visibility was increased due to fluctuations caused by the movement. Second, it was decreased by blurring stimulus boundaries. The parameters of the movement that optimally enhance visibility depended on the characteristics of the stimulus, in detail, its contrast and the sharpness of its boundaries. Appreciable perception was achieved with movement amplitudes of about 20 min-arc and frequencies of around 5 Hz. Movement of a small extent and with temporal frequencies below 0.5 Hz was found to be detrimental to perception, as were frequencies well above the flicker-fusion frequency. The authors concluded that all three components of FEyeM microsaccades, drift, and tremor probably contribute to the maintenance of perception during fixation. In a natural visual scene which includes contrasts of all levels and different types of gradings at boundaries, however, drift and tremor might not suffice to provide a basis for good vision. Microsaccades might, first of all, ensure that stimuli with low contrast or graded boundaries are continually restored.

The findings of Ditchburn and his colleagues, however, were challenged by the work of Gerrits and Vendrik (1970, 1974). In their experiments, an object was mounted in the rotor of a small electric motor, directly attached to the eye by means of a cap sucked onto the cornea. In this way, participants could be presented with stimuli following different movement paths. The first of these two studies used regular rotational movements to simulate fixational drift, microsaccades, and high-frequency tremor. This study revealed that only continuous, drift-like motion effectively restored vision of a faded object (Gerrits & Vendrik, 1970). Gerrits and Vendrik (1974) extended these findings using a more flexible stimulation method. Now, stimuli could be controlled to an extent that imposed movements were very similar to real microsaccades and drifts, which are neither regular nor rotational. Moreover, not only foveal but also parafoveal vision could be examined. Normal continuous vision was found only when the stimulus moved continuously and irregularly, i.e., if movement direction continuously changed. Since only drift of the eye possesses both these characteristics, it was concluded that these movements account for most of the effectiveness of FEyeM in the maintenance of visual perception. If at all, microsaccadic

movements would help to improve perception of stimulus features in parafoveal and peripheral areas (Gerrits & Vendrik, 1974). The results by Gerrits and Vendrik (1970, 1974), in turn, were corroborated by a series of experiments by Kelly and colleagues (Kelly, 1979b, 1979a, 1981; Kelly & Burbeck, 1980), carefully studying the effects of FEyeM on contrast thresholds. The authors achieved contrast sensitivity comparable to what is found in normal viewing when imposing continuous retinal image motion in the velocity range of fixation drift movements (Kelly, 1979a). Thus, drift appeared to play the significant role in the continuous maintenance of visual perception during fixation. As a consequence of these accumulated data, the results of Ditchburn et al. (1959) and Ditchburn et al. (1977b, 1977a), emphasizing that microsaccades can restore visibility in case of perceptual fading, were eclipsed.

Additional doubts in the significance of microsaccades for visual perception emerged at least for three reasons. First, microsaccades (as all saccades) cause saccadic suppression, i.e., they are accompanied by a strong elevation of the perceptual threshold. This was first noted by Ditchburn (1955) and later examined in more detail (Beeler, 1967; Zuber, Crider, & Stark, 1964; Zuber & Stark, 1966).³ Second, foveal vision of stimuli did not deteriorate notably in situations where microsaccades were effectively inhibited (Steinman, Cunitz, Timberlake, & Herman, 1967). Rather, microsaccades were suppressed if high foveal acuity was required to perform in observational (Bridgeman & Palca, 1980) and finely guided visuomotor tasks (Winterson & Collewijn, 1976). Third and finally, there was evidence that the occurrence of a microsaccade is not locked to the disappearance of a stabilized image (Cornsweet, 1956).

In 1986, however, two under-recognized papers by Deubel and Elsner (1986) and Elsner and Deubel (1986) aimed to improve the reputation of microsaccades and argued in favor of their significance for vision. In the task of Deubel and Elsner (1986), participants had to detect low-contrast sine gratings under unconstrained viewing conditions. The results were in support of the view that microsaccades are an important tool for the visual system to enhance performance in near-threshold detection tasks. First, Deubel and Elsner (1986) agreed with Steinman et al. (1973) that microsaccades indeed can be suppressed in observational tasks without deterioration of the visual capacities. However, if viewers are not explicitly required to do so, microsaccades form

³Krauskopf, Graf, and Gaarder (1966) found no difference comparing detection thresholds during and 50 ms after microsaccades, however, according to the experiments by Zuber and Stark (1966) and Beeler (1967) saccadic suppression may still be strong at that time. In one subject, Krauskopf et al. (1966) compared thresholds directly after the microsaccade and 200 ms later. The numerical difference between thresholds in the two time windows was not statistically reliable, revealing the general problem of the study—to argue the null-hypothesis. Thus, non of these analyses were informative with regard to saccadic suppression during microsaccades.

a considerable part of their oculomotor activity in such tasks. Second, microsaccade amplitudes adapt to the visual task demands. For instance, in the case of a 0.5 cycles per degree sine-grating stimulus, microsaccade amplitudes formed a peak at 1 degree of visual angle. Finally, the detection of the grating was often preceded by microsaccades indicating the dependence of detection thresholds on the occurrence of involuntary saccades. Deubel and Elsner (1986) argued that in the study by Kelly (1979a) the role of microsaccades might have been obscured, since observation took place under constraint viewing conditions. This consideration might also account for very different contrast threshold functions that were determined in similar tasks by Kelly (1979a) and Koenderink and Doorn (1979), respectively, the latter study using an unstabilized viewing condition.

To examine the plausibility of their empirical findings, Elsner and Deubel (1986) created a filter model implementing well-known properties of the visual sensory system. In this model, the effect of saccades on perception was simulated by sharp offsets and—after the duration of the saccade—replaced onsets of the visual scene, reproducing saccadic suppression. The model predicted that saccadic eye movements enhance detection performance of near-threshold patterns. Moreover, the detection performance predicted by the model depended on the congruency of the pattern to be detected and the amplitude of the saccade. Thus, the model's predictions were completely in line with the empirical results by Deubel and Elsner (1986). It is noteworthy that the effect of saccadic suppression—which was previously thought to be detrimental for vision—predicts better perception in this framework. These results were consistent with earlier proposals (Ditchburn et al., 1959; Nachmias, 1961) and other empirical findings in detection tasks. For instance, King-Smith and Riggs (1978) reported facilitation effects of a saccade-like square-wave motion (10-100 min-arc) in the detection of low-contrast stimuli stabilized with respect to the retina. Moreover, contrast-sensitivity was increased if exaggerated eye movements occurred in detection tasks using high-frequency gratings flickering with high temporal frequencies (Kulikowski, 1971).

Various authors raised the proposal that microsaccades might, first of all, enhance perception of stimuli in the visual periphery (Ditchburn, 1980; Gerrits & Vendrik, 1974; Martinez-Conde, Macknik, & Hubel, 2000; Martinez-Conde et al., 2004; Snodderly, 1987). This idea would be in agreement with the finding that simulated saccadic displacements greater than 10 min-arc reliably enhance the probability of stimulus reappearance in the Troxler illusion (Clarke & Belcher, 1962), i.e., the disappearance of peripheral low-contrast stimuli during normal fixation (Troxler, 1804). To

finally test this idea directly, Martinez-Conde, Macknik, Troncoso, and Dyar (2006) now studied the relationship between microsaccades and reappearance in the Troxler illusion (see also the preview by Engbert, 2006a). They suggested a causal role of microsaccadic activity in the fading and reappearance of Troxler stimuli. In detail, a higher probability of microsaccades preceded perceptual flips to visibility. Moreover, the average microsaccade rate prior to an intensifying percept was higher and microsaccade amplitudes exceeded their average magnitude. Opposite trends, i.e., a lower rate, probability and mean amplitude of microsaccades, were observed before perceptual disappearance. The demonstration of a potential perceptual function of microsaccades could be demonstrated for various eccentricities, ranging from 3 to 9°, the magnitude of the effects increasing with eccentricity. In addition, it was replicated under conditions where the head was free to move (not restrained by a chin rest) (Martinez-Conde et al., 2006, experiment 3), contradicting earlier objections that microsaccades do not aid vision in more natural conditions (Kowler & Steinman, 1980; Malinov, Epelboim, Herst, & Steinman, 2000; Skavenski et al., 1979; Steinman et al., 1973; Steinman & Collewijn, 1980; Steinman, 2003; Steinman, Pizlo, Forofonova, & Epelboim, 2003). The key results of Martinez-Conde et al. (2006) have since been replicated by Hsieh, Caplovitz, and Tse (submitted), showing that the microsaccade rate increases before a dot subjectively reappears from Troxler fading. These authors found no such relation for the reappearance of stimuli in the paradigm of motion-induced blindness, indicating that this phenomenon is central in origin.

The idea that microsaccades might play an important role in perception and the maintenance of visibility found support in the recent neurophysiological literature. The erratic motion of the retinal image that is caused by FEyeM contributes to the variability of cortical activity (Gur, Beylin, & Snodderly, 1997; Livingstone, Freeman, & Hubel, 1996), which could be attributed to the movement of cortical receptive fields with respect to the visual world (Gur & Snodderly, 1997). Especially, microsaccades raise visual responsiveness in many brain areas involved in visual information processing, as revealed by single-cell recordings in monkeys. The affected areas include the lateral geniculate nucleus (Martinez-Conde et al., 2002), V1 (Martinez-Conde et al., 2000, 2002; Snodderly, Kagan, & Gur, 2001; but see Leopold & Logothetis, 1998), V2 (Leopold & Logothetis, 1998), V4 (Leopold & Logothetis, 1998), and MT (Bair & O'Keefe, 1998).⁴

⁴Monkeys may serve as a model for investigating the impact of FEyeM in the human brain, since—after training in fixation tasks—their fixation behavior is very similar to that of human subjects (Skavenski, Robinson, Steinman, & Timberlake, 1975; Snodderly, 1987; Snodderly & Kurtz, 1985).

In neurons in the LGN and the primary visual cortex Martinez-Conde et al. (2000, 2002) found that microsaccades were better correlated with bursts of spikes than with single spikes or the instantaneous firing rate of neurons. Thus, assuming that microsaccades correlate with visibility (as later confirmed by Martinez-Conde et al., 2006), the authors suggested that bursts of spikes might be most reliable as neural signals for visibility (Martinez-Conde et al., 2000, 2002). This argument is in line with earlier proposals derived from V1 single-cell studies in freely viewing monkeys (Livingstone et al., 1996).

In addition to microsaccades, neural activity was also related to the slow components of FEyeM. In their study, Snodderly et al. (2001) monitored firing rates in response to the drift of the eye during fixation; other studies used these intervals simply as a baseline reference. Snodderly et al. (2001) proposed the subdivision of V1 neurons into cells that are driven by either saccades or drift movements or both. The authors argued that these distinct firing behaviors of cells could implement different functional purposes of the visual system, such as the coding of spatial details and saccadic suppression. Thus, there might be a continuum of neural activity in the visual cortex associated with FEyeM. At large, the new insights from studies of neural correlates of FEyeM strongly contributed to a reconsideration of the relevance of FEyeM in the field of Vision Science in the new century.

Altogether, human visual perception appears to rely upon constantly changing input. Preventing such changes results in rapid fading of the image falling onto the retina. The absence of these changes in static parts of a visual scene, hence, must be compensated for by eye movements taking place during fixation as well as head and body movements. Evidence was reviewed that both drift and microsaccades are necessary to achieve continual perception during fixation. The efficacy of different types of FEyeM in the preservation of a stable percept clearly depends on the location and the characteristics of the stimulus content in a visual scenery. In the visual periphery, receptive fields may be considerably larger than in the fovea. Therefore, only microsaccades are able to sustain perception in these regions, whereas drift movements might generate sufficient image motion to restore vision in the fovea. Recently, it has been shown that insufficient drift movements causing low retinal image slip predict a higher rate of microsaccades (Engbert & Mergenthaler, 2006), clearly increasing variance in the visual input signal. Thus, the dynamic interactions of drift movements and microsaccades might constitute the basis for sustained visual perception during fixation. This could also account for the strong interindividual variability found

in fixational eye-movement patterns. Future studies will have to examine whether changes in the visual input result in less effort of the eyes to move during fixation, which would be predicted if the need for a changing retinal image drives FEyeM.

2.2 Control of fixation position

If eye movements are inevitable during fixation, then the question arises, why the line of sight does not completely lose track of a target. Obviously, a built-in control mechanism must correct for emerging position errors during prolonged fixations. As some of the first, Ditchburn and Ginsborg (1953) recognized this problem. They found that drift movements carry the image randomly across the retina and suggested that microsaccades correct for the produced fixation errors if the image reaches the edge of a central region.

A cornerstone study by Cornsweet (1956) directly examined which conditions evoked drift and microsaccades during fixation. At that time, various proposals have been around what might be the stimuli that trigger FEyeM. First, it had been suggested that FEyeM are stimulated directly by retinal fading (Ditchburn & Ginsborg, 1952; Ginsborg, 1953). Second, various authors had argued that displacement of the fixation target from the optimal location could result in corrective drift or microsaccades or both (Ditchburn, 1955; Ditchburn & Ginsborg, 1953; Ginsborg, 1953; Ratliff & Riggs, 1950; ten Doesschate, 1954). Third, it had been proposed that oculomotor instability might be generated independent of visual signals (Ratliff & Riggs, 1950). Cornsweet (1956) manipulated temporal aspects of stimuli by presenting them at different flickering rates. Moreover, the author varied spatial displacement by using a retinal stabilization technique. The results of this study were surprisingly clear and straight-forward: Disappearance did not stimulate changes in the frequency of drift movements. However, drift rates were lower for normal viewing than they were in the stabilized condition. Thus, drift did not correct for displacement of the fixation target that must have resulted from normal viewing conditions. In addition, the author found that fixation errors increased over intervals of drift. Finally, it was concluded that drift is not under direct visual control. Microsaccade occurrences were not found to be correlated with disappearance times either. However, microsaccade rates could be attributed to fixational displacement. First, fewer microsaccades were observed in the retinal-stabilization condition. Second, the probability for a microsaccade to occur was increasing with the distance of the viewing point from the mean eye

position over a 45 sec trial. Third, this microsaccade then went most probably in the direction of the fixation stimulus—the probability being highly correlated with the amount of displacement. Finally, the amplitude of microsaccades depended on the magnitude of displacement. Larger displacements were associated with larger microsaccades. However, since the minimum saccade amplitude was found to be 3 min-arc, microsaccades tended to overshoot the point of minimum error after small displacements. After all, Cornsweet (1956) concluded that while the instability of the oculomotor system results in drift, taking the eye farther and farther away from some optimal locus, microsaccades serve the role of returning the eyes on a fixated target. A similar view was held by Yarbus (1967), who, however, did not substantiate this hypothesis with data.

At their time, the observations by Cornsweet (1956) triggered much dispute on the role of microsaccades in fixation control and not all of the subsequent studies could replicate his findings. In one of the first studies employing binocular eye-movement measurements, Krauskopf et al. (1960) found no correlation between the drift movements of the two eyes, however, a strong correlation of microsaccade direction and amplitude. This finding challenged the model proposed by Cornsweet (1956), since microsaccades should produce fixation errors in one eye at least. Thus, Krauskopf et al. (1960) modified the model of Cornsweet (1956), suggesting that both eyes independently correct for their individual fixation errors when a certain amount of error had accumulated. In addition, a central mechanism was assumed, triggering a microsaccade in both eyes. However, while microsaccade direction is highly correlated between the two eyes, the amplitude in the “passive” eye was proposed to be smaller, resulting in a correction of fixation errors in both eyes on average. Indeed, in Krauskopf et al.’s subjects, microsaccade amplitudes were reliably smaller in one eye than in the other. A subsequent study by (St. Cur & Fender, 1969) lend further support to this conclusion, showing that microsaccades significantly reduce disparity between the two eyes.

A stronger challenge to Cornsweet’s conclusions was a study by Nachmias (1959), who examined FEyeM along eight meridians. He discovered that microsaccade direction has an idiosyncratic component to it, i.e., it varies between subjects. In Nachmias’s subjects, microsaccades indeed compensated for fixation errors, however, the time passed since the last microsaccade was a better predictor for the occurrence of a microsaccade than the amount of displacement. Moreover, the author demonstrated that some compensation was achieved by drift, especially along those meridians where compensation by microsaccades was poor. Two years later, Nachmias (1961)

published another study, on the determiners of drift movements during fixation. He replicated the most important findings of his earlier study. First, microsaccade occurrence did not depend on the drift rate. Second, like microsaccades, drift could also correct for errors produced by other drift along certain meridians. Third, as compared to fixation error, again, the time since the last microsaccade was a better predictor for the observation of another microsaccade. In addition, the author demonstrated that drift could—at least indirectly—be influenced by visual factors, showing that drift rates and directions depended on the viewing distance of fixation targets. In detail, drift was more pronounced when subjects fixated a stimulus at a distance of 30 cm instead of at optical infinity.

The finding that drift might contribute to the maintenance of fixation on a target has been replicated by other authors, though correction by drift was not necessarily as effective as correction by microsaccades (Fiorentini & Ercoles, 1966; St. Cur & Fender, 1969). Boyce (1967) as well as Beeler (1965, as cited in St. Cur & Fender, 1969) found that only 30 percent, or less, of the microsaccades observed in their subject were correcting for previous drift. Also Proskuryakova and Shakhnovich (1967) and Glezer (1959) found no evidence for an inverse relationship between drift and microsaccade direction. It is important to note that due to the technical complexity of eye-movement recording decades ago, most of the early studies used highly trained observers (the authors, in most cases). However, using naïve participants, Møller, Laursen, and Sjølie (2006) recently reported similar findings. From these notions, a case was made that both microsaccades and drift may be error-correcting and -producing as well (Nachmias, 1961; Steinman et al., 1973).

These findings fueled the idea that microsaccades do not serve a purpose, but rather represent “busy work” of the oculomotor system while it is forced to fixate over unnaturally long periods of time (Kowler & Steinman, 1980; Steinman et al., 1973). In addition, it was discovered at that time that just by a simple change in the instruction (from “fixate” to “hold the eyes still”), subjects were capable of nearly completely avoiding microsaccades. This important finding was first noticed by Fiorentini and Ercoles (1966) and afterwards documented in some more detail by Steinman et al. (1967). Voluntary inhibition of microsaccades has been replicated frequently, in normal humans (Gowen, Abadi, & Poliakoff, 2005; Haddad & Steinman, 1973; Haddad & Winterson, 1975; Kowler & Steinman, 1977, 1979; Murphy, Haddad, & Steinman, 1974; Puckett & Steinman, 1969; Steinman, Skavenski, & Sansbury, 1969; Winterson & Collewijn, 1976) as well as in amblyopic patients (Ciuffreda, Kenyon, & Stark, 1979; Schor & Hallmark, 1978), a disorder of

the eye associated with high rates of drift movements. It was repeatedly emphasized in some of these studies, that the variability of fixation position was not enhanced when microsaccades were suppressed (Murphy et al., 1974; Puckett & Steinman, 1969; Steinman et al., 1967, 1973; Winterson & Collewijn, 1976).

As a consequence, these authors stressed the argument that microsaccades are not necessary for the control of fixation position. Rather, the process of *slow control*, i.e., drift movements that keep the eyes on a target, was proposed to serve that purpose. Winterson and Collewijn (1976) as well as Steinman et al. (1973) found that the slow control system is not equally effective in all observers. The latter authors, however, argued that in their study even the observer with the least effective slow control of fixation position, exceeded natural fixation durations by the factor of ten successively maintaining a fixation target within the foveal area. Slow-control mechanisms have since been observed in preschool children (Kowler & Martins, 1982) and a variety of species, including cats (Winterson & Robinson, 1975), rabbits (Collewijn & van der Mark, 1972), and monkeys (Skavenski et al., 1975; Snodderly, 1987). Kowler and Steinman (1980) summarized the literature, finding that 85% of the human subjects in all studies showed effective slow control in the absence of microsaccades. They conclude that microsaccades could not have evolved to control fixation position in subset of normal humans as small as 15%. Further research, primarily driven by the group around Robert M. Steinman, focussed on the origin of the signal used to establish slow control mechanisms (Matin et al., 1970; Murphy et al., 1974; Sansbury, Skavenski, Haddad, & Steinman, 1973; Skavenski, 1971, 1972; Skavenski & Steinman, 1970; Steinman, 1965, 1976).

In a nutshell, the findings concerning the role of microsaccades and drift movements in the control of fixation position are inconsistent and partially conflicting. Both microsaccades and drifts may enhance or decrease the deviation between the desired gaze position and the line of sight. Moreover, if microsaccades are suppressed, drift alone slowly controls for emerging fixation errors. Differences between observers, stimuli, analyses, and even in the definition of a “correcting” movement may account for a part of these inconsistencies (St. Cur & Fender, 1969). In addition, there is the chance that new analytic tools shed light on these controversies. In a recent paper, Engbert and Kliegl (2004) described FEyeM in terms of the statistics of a random walk. The authors showed that FEyeM, recorded during the fixation of a small dot, systematically deviate from Brownian motion, i.e., purely random movement; FEyeM were best described by a subdivision in two different time scales. Over short periods of time (2 to 20 ms), the eyes appeared

to actively increase the variance in spatial displacement, possibly to enhance the amount of retinal image motion. On longer time scales (100 ms, or more), in contrast, a control mechanism set in, ensuring that fixation errors were reduced. A considerable part of both these tendencies could be attributed to microsaccades. The different behavior on two temporal scales could explain the controversial results of earlier studies.

2.3 Visual acuity and scanning of small regions

Very early on, it was recognized that motion of the retinal image necessarily interferes with visual acuity (e.g., Jurin, 1738; von Helmholtz, 1924), at least if the motion is greater in extent than the anatomically determined spatial resolution of the retina. In fact, however, humans exhibit extraordinarily performance in visual-acuity tasks; they show hyperacuity. That is, visual acuity is far better than it could be obtained when resulting from a static mosaic of retinal cones. As a consequence, theories of hyperacuity in the first half of the 20th century often assumed that eye movements during fixation aid visual performance (e.g., Averill & Weymouth, 1925; Adler & Fliegelman, 1934; Marshall & Talbot, 1942). Inspired by these theories, Riggs, Armington, and Ratliff (1954) determined the amount of retinal image motion during fixation. For exposure durations of up to 10 ms the image was found to be virtually stable with respect to the photoreceptors of the retina (carriage very rarely exceeded 10 sec-arc). A 100 ms were needed to shift the image over 25 sec-arc, i.e., the diameter of a foveal cone. In 1 sec, the eye carried the image by some 3 min-arc. In an overview of the early findings on the relation between FEyeM and visual acuity, Riggs (1965, p. 341-345) argued that since the critical duration for visual acuity is 100 ms or less, visual acuity should not be affected by FEyeM.

One method used to test the effect of FEyeM on visual acuity was to vary exposure times of stimuli in high acuity tasks while measuring the extent of retinal motion taking place in these intervals. Ratliff (1952) presented his subjects for 0.075 sec with a test stimulus consisting of parallel lines. The tilt of this grating varied across trials; the task was to determine its orientation. Drift and tremor were shown to hinder the judgement. However, Ratliff (1952) admitted that FEyeM could still aid other perceptual acuity tasks, such as the evaluation of straightness of lines, the recognition of simple borders, or the detection of grainy structures. Moreover, the author

argued that FEyeM could come into play when longer stimulus exposure durations are needed for perceptual judgments.

A second approach was to counteract and exaggerate retinal motion, respectively. Riggs et al. (1953) showed that the counteraction of FEyeM in a retinal stabilization paradigm did not affect visual acuity with exposure durations of up to 0.11 sec. Rather, there was a slight tendency for better performance in the no-motion condition. As reported above, however, FEyeM were necessary to prevent the image from fading when longer presentation times were applied. These results were followed up in a study by Tulunay-Keesey (1960). Detection, resolution, and localization tasks (using fine lines, gratings, and vernier offsets, respectively) were performed with different exposure times. In none of these tasks visual acuity was affected by the exclusion of retinal image motion. Moreover, exposure times greater than 0.2 sec did not increase acuity rates. Thus, retinal motion was neither detrimental nor beneficial for visual-acuity performance in this study. Further, it was concluded that visual fading was too slow to impair visual acuity. Even in stereoscopic vision, Shortess and Krauskopf (1961) confirmed that FEyeM did not affect visual acuity for exposure durations between 20 ms and 1 sec. It appeared that in the visual system FEyeM are implemented such that visual acuity is good while retinal fatigue is properly prevented.

The general impact of FEyeM on visual acuity was continually discussed over several decades and modern theories again emphasize the role of FEyeM in visual information processing, hooking up with the early dynamic approaches (e.g., Ahissar & Arieli, 2001). It is beyond the scope of the present work to review the whole body of literature on that topic and thoughtful resumés have been drawn elsewhere (e.g., Steinman & Levinson, 1990). Rather, we would like to highlight those studies that directly assessed the relation between microsaccades and visual performance.

Rattle and Foley-Fisher (1968) were the first to report a correlation between microsaccades and visual function. They showed that inter-microsaccade-intervals were directly correlated to performance in a vernier-acuity task. The lower a participant's rate of microsaccades was, the higher was his or her performance. The authors suggested that microsaccades put an end to integration periods necessary to resolve fine spatial detail. Obviously then, microsaccades were not beneficial in Rattle and Foley-Fisher's paradigm.

Winterson and Collewijn (1976) directly assessed the role of microsaccades in finely guided visuomotor tasks. Their participants aimed and shot a rifle or threaded a sewing needle while eye

movements were recorded. It was observed, that the rate of microsaccades strongly decreased while performing the task as compared to a normal fixation condition. In a purely observational high-acuity localization task, Bridgeman and Palca (1980) confirmed the decrease in the rate of microsaccades before the judgment. It was concluded that microsaccades were neither important nor essential in high-acuity tasks. These findings fitted well the picture of the assumed uselessness of microsaccades, that built up when it was shown that these FEyeM can be suppressed voluntarily without noticeable deterioration of visual performance in observational tasks (see above).

The question whether microsaccades play a role in visual acuity is strongly related to the proposal that this type of miniature eye movements might resemble attentional shifts scanning confined regions near the target being fixated (Cunitz & Steinman, 1969; Steinman et al., 1973). That is, microsaccades might serve the same function as large saccades, namely visual search. For instance, as shown by Kowler and Anton (1987), observers strongly decreased their mean saccade amplitudes when they had to read twisted text. The hypothesis that microsaccades serve the purpose of visual search could also account for fixation errors produced by microsaccades; remember that microsaccades did not always correct for previous drift movements (Boyce, 1967) and if they did, the correction was frequently associated with large errors (Boyce, 1967; Cornsweet, 1956; Nachmias, 1961).

Taking a first step to examine the possibility that microsaccades generally obey the same principles as large saccades, Cunitz and Steinman (1969) examined frequencies of microsaccades in simple fixation tasks and in reading. Inter-saccade-interval distributions of microsaccades during fixation of a T-shaped stimulus were very similar to those found for reading saccades. Moreover, microsaccades occurred very rarely during reading fixations (in 2-5% only), in these cases doubling fixation durations. That is, microsaccades occurred after and were followed by intervals of a typical reading-fixation duration, implicating that they were observed only in very long fixations. Cunitz and Steinman (1969) speculated that both microsaccades and large saccades are controlled by a single system. In addition, the authors suggested that both types of movements serve to scan the visual scene, though on different spatial scales.

Timberlake, Wyman, Skavenski, and Steinman (1972) demonstrated that position errors, which were produced by target steps as small as 3.4 min-arc, can be reduced by small voluntary saccades, suggesting once more that both microsaccades and normal scanning saccades differ only in amplitude, but not in their purpose. Also Haddad and Steinman (1973) emphasized that saccades as

small as fixational microsaccades can be triggered voluntarily. In their study, involuntary saccades still occurred, most of the time correcting for preceding drift movements of the eyes. However, observers were aware of having made these microsaccades. It was argued, that tiny saccades serve visual search.

Kowler and Steinman (1977) examined the role of small saccades in counting. To this end, a number of bright bars was presented in a small display. The number of bars ranged from 10 to 19; they were presented for 7.6 seconds and reports were given immediately after the offset of the display. The authors themselves served as subjects and were instructed either to suppress saccades or to use saccades to their convenience during the task. Counting of the repetitive bar patterns did not require saccades. The number of correct reports did not differ as a function of instruction. In a second experiment, the authors showed that saccades may indeed improve counting accuracy, if perceptual confusion is reduced. This time, the items in a display had odd shapes and were haphazardly arranged. Under the instruction to suppress all saccades, counting accuracy was fair (somewhat more than 60% correct), however, if small saccades (mainly below 30 min-arc in amplitude) were used to scan the display, accuracy nearly reached the ceiling. Kowler and Steinman (1977) tried to determine why saccades yielded a benefit in counting accuracy, however, a clear answer could not be given in this study. In a follow-up experiment, however, Kowler and Steinman (1979) showed that saccades were not beneficial if the counting display spanned half a degree of diameter only. In this case, their counting accuracy with and without saccades could not be distinguished. The authors argued that saccades might only be generated as a consequence of attention shifts within the display; this is more likely to occur if the display spans a larger area (see also Kowler & Steinman, 1977). It was concluded that very small saccades (in the range of 15 min-arc, or less) do not serve a purpose in a counting task.

Another explanation why saccades may be beneficial in a counting task is that they repeatedly generate new onsets in the visual system. Kowler and Sperling (1980) rejected this hypothesis as unlikely. In their visual search task, participants searched a numeral contained in an array of letters. Two basic conditions were tested. In the one-onset condition, the search display set on and was then presented continuously throughout a trial. In the two-onsets condition, the display was shortly presented at the beginning of a trial and flashed again, resulting in an additional onset, in the midpoint of a dark interval. Multiple onsets did not aid search performance. On the contrary, search performance was better if the array was shown continuously across a trial, and

no second onset was induced. Unfortunately, however, Kowler and Sperling did not keep the total presentation times of the search arrays constant across conditions, neither in this study, nor in a follow up (Kowler & Sperling, 1983). Therefore, their results may not be interpreted offhand with regard to the role of onsets in visual-search performance.

There is no doubt that large saccades are used to scan a visual scene, bringing potentially interesting regions onto the foveae. Brockmann and Geisel (2000) confirmed that saccades freely inspecting a static visual scene show statistics of an optimal random-search process, minimizing the time needed to scan a whole scene. Microsaccades during continual fixation of a small dot, however, do not show such properties, as examined by Engbert (2006b). Thus, it could not be supported by these analyses that microsaccades serve the purpose of (optimal) visual search on a smaller scale. We have seen, however, that some studies reported strong benefits associated with the generation of small saccades. One possibility is that these are a consequence of attentional shifts in the foveated region. A link between microsaccades and covert attention is now well established as we will see in the next section.

2.4 Covert shifts of attention

Receptor shifts (i.e., movements of gaze) may also be referred to as *overt attention* shifts. In contrast, we use the term *covert attention* when attention is allocated to the visual periphery without moving the eyes to that location. Recently, much progress has been made in understanding the behavior of microsaccades during shifts of covert attention. Engbert and Kliegl (2003b) examined microsaccade statistics in an attentional cuing paradigm comparable to that originally introduced by Posner (1980). The authors analyzed the evolution of microsaccade statistics in response to endogenous spatial cues, in detail, centrally presented arrow or color cues. Microsaccade rate (mean frequency per second) evolved in a characteristic fashion. Initially, microsaccades occurred with normal frequency (about 1 microsaccade a second). After cue presentation, very few microsaccades were observed; this inhibition reached a minimum at about 150 ms after cue onset and was then followed by a strong enhancement in microsaccade rate. The microsaccade rate reached a maximum at 350 ms after cue onset, before it finally resettled at the baseline rate (500 ms after cue onset). The shape of this microsaccade-rate evolution was found to be very stereotypical in response to visual (Galfano, Betta, & Turatto, 2004; Laubrock et al., 2005, in press;

Rolfs et al., 2004, 2005, 2006) and auditory (Rolfs et al., 2005) stimuli, while the time course varied across the different paradigms and conditions employed (see Engbert, 2006b, for an overview). In the remainder of this thesis we will frequently refer to this pattern as the *rate signature*. Moreover, Engbert and Kliegl (2003b) showed that the direction of microsaccades clearly depended on the direction of the attentional shift induced by spatially informative cues. Before cue presentation, microsaccades were nearly equally likely to go in one direction or the other in the horizontal plane. During the enhancement epoch, that is, some time after cue presentation, distributions of microsaccade directions were clearly shifted towards the cued side. The authors concluded, that microsaccades may be used as an indicator of spatial shifts of covert attention. A similar finding was reported by Hafed and Clark (2002).

In a study by Rolfs et al. (2004), participants were presented with peripheral attentional cues, informative for the location of subsequently appearing targets of a discrimination task. In addition to the expected rate signature, it was observed that microsaccades strongly preferred to go in the direction opposite the cued location. Combining the evidence from a recent study by Tse, Sheinberg, and Logothetis (2003) on the distribution of attention in response to peripheral flashes with the knowledge from previous studies on the correspondence of microsaccade direction and spatial attention (Engbert & Kliegl, 2003b; Hafed & Clark, 2002), it was concluded that microsaccades indicated the direction of covert attention shifts in both central and peripheral cuing tasks. It was argued that the attentional shift was in the direction opposite the cue to inhibit automatic saccadic responses to the salient stimulus. Tse et al. exploited a change-blindness paradigm and found that attention (in terms of close-to-perfect change detection) extended along the cue-fixation axis in both directions—to the cued location and, in particular, to the opposite side. In their paradigm, however, a relation between FEyeM and shifts of attention was not evident (Tse, Sheinberg, & Logothetis, 2002, 2004). Rolfs et al. (2004) argued that the large amount of display changes taking place in the experiments of (Tse et al., 2002, 2003) must have resulted in a pronounced inhibition of microsaccades. Thus, direction effects could not become evident.

The evolution of microsaccade statistics was generalized along several lines. First, Galfano et al. (2004) studied the influence of uninformative cues on microsaccade statistics in a simple detection task. Uninformative exogenous attentional cues usually result in inhibition of return. That is, for some hundred milliseconds the cued spatial location receives less attention (see Klein, 2000, for a review and explanations of this effect). Galfano et al. (2004) replicated both the rate

signature and the cue opposing microsaccade-direction effect observed by Rolfs et al. (2004). The authors concluded that inhibition of return is reflected in the patterns of microsaccade directions.

Second, starting out from the literature on multisensory interactions in the control of attention and saccades, Rolfs et al. (2005) examined the impact of informative cues in auditory, visual, and intermodal spatial attention task. In all cuing conditions, i.e., also in the absence of any changes of the visual display, the microsaccade rate signature was observed. Primarily during the enhancement epoch of the rate signature, microsaccades were clearly biased towards the direction opposite the cue as soon as visual attention got involved in the task (i.e., with visual cues or targets or both). In the purely auditory cuing condition, microsaccades rather directed to the cued location. However, this effect was only found for cues to the left side. Rolfs et al. (2005) concluded that microsaccades might serve as a tool to study multisensory integration and the time course of saccade preparation during shifts of covert attention in different sensory modalities.

Finally, Laubrock et al. (2005) mapped the time course of microsaccade dynamics, comparing endogenous and exogenous cuing in spatial attention tasks. A paradigm by Müller and Rabbitt (1989) was employed to separate the attention shift from the preparation of the response saccade. Endogenous cues triggered only weak cue-opposing effects in microsaccade direction emerging late in the cue-target interval. Cue-directed microsaccade effects as reported by Engbert and Kliegl (2003b) could not be identified within this paradigm, possibly, since the authors used color cues as indicators of the response-target locations. However, a cue-congruent microsaccade-direction effect would neither be expected in this paradigm, if microsaccades indicate the process of motor preparation rather than the direction of the attentional shift. Exogenous flash cues in the study by Laubrock et al. (2005), rapidly evoked a microsaccade direction bias to the cued location. Short time later, this effect was replaced by a strong cue-opposing bias in microsaccade directions, which fits into the pattern of results reported by Rolfs et al. (2004). Laubrock et al. (2005), however, offer a different explanation for the cue-opposing bias in microsaccade directions: Frequently shifts of covert attention are perceived as actual eye movements (Deubel, Irwin, & Schneider, 1999). Thus, participants might have perceived their covert attention shift to the peripheral flash as an overt movement, which they correct for by microsaccades in the opposite direction.

In the studies reviewed above, microsaccades were shown to be an indicator of spatial attention. That is, their direction correlated with covert attention. A causal connection between microsaccades and attention seems unlikely a priori. Nevertheless, Horowitz, Fine, Fencsik, Yurgenson,

and Wolfe (in press) tested whether microsaccades or other types of FEyeM create attention shifts. The cuing paradigm employed by Engbert and Kliegl (2003b) was reused to this end. The authors reasoned that if microsaccade direction is a nearly perfect indicator of covert attention, it should more reliably predict response times than the location of the cue. As expected, no evidence for this causal hypothesis was found. In fact, Horowitz et al. (in press) concluded that microsaccades are not an index of covert attention. As Laubrock et al. (in press) argue, this is by far too much of a conclusion, since microsaccade direction strongly correlates with the direction of a spatial cue and, thus, with shifts of covert attention. In addition, these authors showed that microsaccade directions explain some of the residual variance in response times, when the effect of cuing is statistically controlled, however, the effect is one order of magnitude smaller than the classical cuing benefit. Thus, microsaccades clearly may not serve the function of shifting attention within the visual field. Still it is possible that microsaccades serve the stabilization of a created "hot spot" of attention at a given location, a hypothesis that has not been tested so far.

2.5 Further potential functions of fixational eye movements

So far we have discussed potential functions of microsaccades and other types of FEyeM that have yet been the subject of intense study. Further potential purposes of FEyeM were proposed in the body of existing literature, which (as yet) received less attention. Some of these proposals yielded fruitful results, others turned out disappointing. Here, we will shortly review these efforts.

Binocular rivalry. It has been discussed earlier that there is now evidence that microsaccades are related to alternations in bistable perceptual phenomena like the Troxler illusion (Martinez-Conde et al., 2006). A similar proposal was raised by Levelt (1967) with respect to binocular-rivalry phenomena. Binocular rivalry occurs when two dissimilar stimuli are dichoptically presented to an observer. Participants usually report seeing either one or the other object, but rarely both at a time. Levelt (1967) found that dominance and suppression intervals of either eye can be described by a gamma distribution. This distribution might be the result of a discrete process. He argued that microsaccades might constitute this stream of discrete events gradually reducing the visual threshold of the suppressed eye, which appears necessary to achieve a flip in perceptual dominance. Sabrin and Kertesz (1980, 1983) investigated the microsaccade statistics in the binocular rivalry phenomenon. Their data corroborated Levelt's proposal: In a rivalry situation,

microsaccade rates increased by 50% as compared to normal viewing (Sabrin & Kertesz, 1980). In addition, the rate of microsaccades declined at the end of a suppression interval, indicating that the depth of suppression is not constant over a rivalry interval. In a follow up study, the stimulus presented to one eye was stabilized with respect to the retina, and simulated microsaccades were artificially superimposed on it (Sabrin & Kertesz, 1983). Imposed movements were most effective in increasing the visibility of the suppressed stimulus if their rate and amplitude were similar to that of naturally occurring microsaccades. In this study, rivalry could also be observed when no movement was imposed to the stabilized retinal image. Sabrin and Kertesz (1983) concluded that FEyeM were not mandatory for the occurrence of the binocular rivalry phenomenon, however, microsaccades most efficiently provoke the phenomenon. Very recently, van Dam and van Ee (2006) extended these results by showing that alterations in bistable phenomena are due to the retinal image shifts induced by microsaccades, not by the eye movements per se.

Image reconstruction. In the field of computational vision, Kadyrov and Petrou (2004) presented a method for the reconstruction of missing parts of an image that integrates over a group of transformations of a spline grid used for interpolation. Applying this method, the authors achieved significantly better reconstructions of natural images and geometric structures than with any other simple interpolation approach; shape and accuracy were better preserved. Kadyrov and Petrou (2004) proposed that integration over a group of transformations may also be what the visual system does to transform the non-uniform input from the photoreceptors into clear and un-aliased images. Microsaccades may serve the purpose to provide the visual system with a number of simple image acquisitions to achieve an improved intake. Successive intakes may then be spatially aligned and averaged to obtain a clearer percept.

Mental imagery. Kowler and Steinman (1977) required their subjects to fixate on a small spot while answering questions like "How many windows are there on the second floor of your parents' home?" The specific questions were unbeknownst to them before the test. The authors reasoned that answering such questions requires the scanning of a mental image using the mind's eye. If saccades were necessary to correctly answer the questions, this could offer a hint at the purpose of small saccades during fixation. However, visual imagery did not require saccades. Saccades were rarely made during the task and their number was not different from a control condition, in which much less demanding questions had to be answered.

Development of neural wiring. One of the most promising ideas concerning the function of FEyeM was devised by Michele Rucci and his coworkers. These authors promote the idea that FEyeM serve a critical role in the refinement and stabilization of cell-response characteristics during the development of early visual-information processing. Before eye opening, the wiring of neurons on the way from the LGN to the primary visual cortex relies on spontaneous activity that is correlated on a very small spatial scale. After eye opening, however, the visual input to the system is typically highly correlated over broad spatial ranges. In a series of modeling studies of thalamic and thalamocortical activity, it was recently shown that this statistical dependency may be strongly reduced in the presence of simulated FEyeM during the acquisition of visual information (Casile & Rucci, 2006; Rucci & Casile, 2004, 2005; Rucci, Edelman, & Wray, 2000). Consequently, a regime of neural activity is established after eye opening that is similar to that before visual input may affect the neural wiring between the early visual processing stages. The refinement of receptive field organization in the primary cortex may, thus, rely on the same principles before and after eye opening. Moreover, the results of these studies suggest that FEyeM are crucial for structuring the visual input for further analysis in the course of visual information processing and, hence, for establishing efficient visual representations.

2.6 Summary of the functions of microsaccades

The function of microsaccades has a long tradition of being a research problem. Here, we presented an exhaustive review of the literature concerned with that issue. Using a number of methodological approaches, a number of hypotheses were tested over more than 50 years of research. Frequently, the findings concerning the purpose of microsaccades were not clear, or even contradictory. However, we have presented evidence that microsaccades (1) are involved in the preservation of peripheral vision during fixation, (2) embank position errors when the eye is fixated within a visual scene, (3) do not appear to aid visual search and visual acuity, (4) carry information about the allocation of spatial attention, and (5) may evoke perceptual switches in binocular rivalry situations, (6) are not necessary in mental imagery, and (7) may be involved in the development of neural wiring in the developing human.

Referring to the elevation of the perceptual threshold (saccadic suppression) associated with microsaccades, Bruce Bridgeman once stated: "A phenomenon which triples visual thresholds in

normal humans for over an hour each day requires explanation.” (Bridgeman & Palca, 1980, p. 813), which justifies the patience with which researchers pursued this issue. Concurrently, this statement pinpointed at the lack of success of researchers in finding a definite purpose specific to microsaccades. By the beginning of the nineteen-eighties, an emotional letter exchange between Kowler and Steinman on the one side (Kowler & Steinman, 1979, 1980) and Ditchburn on the other side (Ditchburn, 1980), put a temporary end to the debate, leaving open space for researches to come up 20 years later, equipped with new methods and ideas to finally reconsider the purpose of microsaccades. The results presented above demonstrate that there has been quite some progress over the last years in research studying the purpose of microsaccades. A handful of laboratories bore this renaissance and have yielded important new insights into perceptual and oculomotor functions associated with these movements. However, it is important to bear in mind that there is a long history of research on the issue, which has to be considered when drawing conclusions about the relevance of FEyeM in general, and microsaccades, in particular. The review presented here may help finding a way through the labyrinth of that literature.

Chapter 3

On the relation between microsaccades and saccades

Despite their very different amplitudes and traditional subdivision into different categories of eye movements, microsaccades and large-scale saccades share a wide range of characteristics. First, all saccades are defined as binocular eye movements with almost identical amplitudes and directions in both eyes (Ditchburn & Ginsborg, 1953; Krauskopf et al., 1960; Lord, 1951). Second, both microsaccades and large-scale saccades fall on the main sequence (Zuber et al., 1965), i.e., the relationship between peak velocity and amplitude in these movements follows a power law. Third, all kinds of saccades result in a strong elevation of the visual perceptual threshold covering some time around the movement (Beeler, 1967; Latour, 1962; Volkman, 1962; Volkman, Schick, & Riggs, 1968; Zuber et al., 1964; Zuber & Stark, 1966). Fourth, it was argued that inter-saccadic intervals in reading are equally distributed as inter-microsaccadic intervals during simple fixation of a letter (Cunitz & Steinman, 1969). Finally, there is a strong relationship between spatial attention and the generation of saccades. Saccades are virtually always preceded by shifts of covert attention (e.g., Deubel & Schneider, 1996; Kowler, Anderson, Doshier, & Blaser, 1995). Moreover, both covert attention and saccades appear to share the same neurophysiological basis (e.g., Corbetta et al., 1998; Kustov & Robinson, 1996). As has been outlined in Section 2.4, a pronounced correlation was also found for covert attention and microsaccades (Engbert & Kliegl,

2003b; Galfano et al., 2004; Hafed & Clark, 2002; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005).

The accumulation of these results strongly suggests that microsaccades and other saccades are the product of the same machinery implementing the manufacture of high-velocity eye movements. However, although repeatedly proposed (Engbert, 2006b; Gandhi & Keller, 1999b; Munoz, Dorris, Paré, & Everling, 2000), this hypothesis has rarely been tested explicitly by the means of neurophysiology, which might have at least two reasons. First, there might have been a lack of interest in neural correlates associated with the generation of microsaccades among neurophysiologists studying the oculomotor system. The first single-cell recordings of oculomotor neurons in monkeys were carried out during the early 1970s (Fuchs & Luschei, 1970; D. A. Robinson, 1970). By that time, however, the relevance of microsaccades was already largely doubted by many scientists in the field (cf., Steinman et al., 1973). Second, it could be a methodologically difficult task to record neural activity preceding involuntary and unpredictable eye movements, especially when they have amplitudes of well below 1° of visual angle. Have in mind that a big share of the neural machinery encodes saccades in neural motor fields topographically reflecting different amplitudes and directions (see below). For instance, activating a site in the motor field of the superior colliculus (SC) by applying suprathreshold stimulation results in a corresponding fixed-vector saccade (e.g., D. A. Robinson, 1972) and single-cell recordings from these neurons revealed enhanced activity prior to a saccadic eye movement associated with the cell (e.g., Sparks, Holland, & Guthrie, 1976). Thus, while recording from a cell within a motor field one would not only have to wait for the occurrence of a microsaccade during fixation, but also this microsaccade would have to possess the specific amplitude and direction associated with the cell's location in the neural field. Except for one study that is known to the author (van Gisbergen, Robinson, & Gielen, 1981; see also van Gisbergen & Robinson, 1977), only recently, oculomotor neurophysiologists have begun to study the generation of fixational eye movements (FEyeM), e.g., by lesioning cells potentially involved in microsaccade generation (U. J. Ilg, personal communication).

The present work aims to investigate the machinery underlying microsaccade generation on the basis of behavioral data—first of all, by studying their impact on large voluntary saccades. To psychologists these interactions should be of special interest, since knowing them will help understand the significance of microsaccades not only for perception, but also for action. Possibly, oculomotor behavior during fixation can account for some of the variance in behavioral measures

such as response latencies or error rates. As a desirable consequence, microsaccades would provide us with a (non-invasive) tool to study the time course of oculomotor preparation.

As will become clear in this and the following chapters, the neurophysiological implementation of normal saccades is comparably well understood. From this rich knowledge base, predictions will be derived about the neural correlates of microsaccades in the oculomotor control system. It will be inferred, which interactions between involuntary microsaccades and voluntary saccades might be observed in behavioral data. In the first place, however, we will give an overview of the neurophysiology behind the generation of saccadic eye movements.

3.1 Overview of the neural basis of saccade generation

The brain circuitry implementing the control of saccadic eye movements as well as the sub-systems involved have been described extensively in a great number of recent review articles (e.g., Hikosaka, Takikawa, & Kawagoe, 2000; Moschovakis, Scudder, & Highstein, 1996; Munoz, 2002; Munoz et al., 2000; Munoz & Everling, 2004; Munoz & Fecteau, 2002; Munoz & Schall, 2003; Pierrot-Deseilligny, Milea, & Müri, 2004; Schall, 1997; Scudder, Kaneko, & Fuchs, 2002; Sparks, 2002). Faced with the large amount of publications in this field, we cannot review in full detail what is known about this complex circuitry and the specific functions of its components. Instead, we will give an idea of the main brain structures involved, their most important connections, and the principles underlying saccade control in humans and non-human mammals (which were used as animal models in most of the pertinent literature), since these will be highly relevant to the research described here.

Figure 3.1 depicts a simplified outline of the connections of brain areas involved in saccade generation. Basically, two important processing streams can be distinguished, a reflexive one and one implementing voluntary control. The reflexive pathway permits fast and direct access of visual input to the subcortical saccade machinery through two major routes. First, visually evoked signals may be propagated to the SC directly from the retina, i.e., via the retinotectal pathway. Second, they may be propagated along the retino-geniculo-cortical pathway, i.e., first pass the lateral geniculate nucleus before impinging striate and extrastriate visual-cortex areas and possibly the lateral intraparietal area (LIP). The voluntary pathway separates from the reflexive routes at the LIP, from where neural activity is propagated to the frontal cortex, including the frontal eye

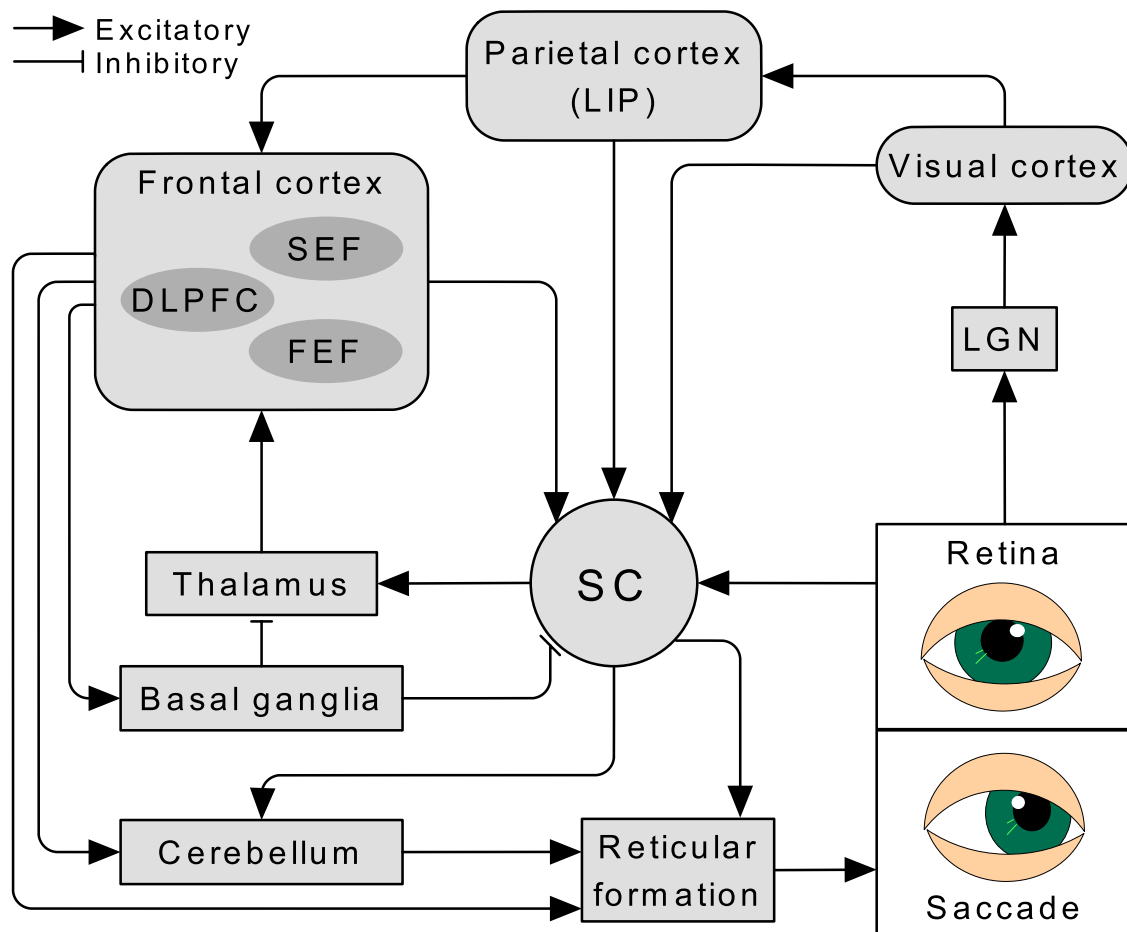


Figure 3.1: Circuitry of brain areas involved in saccade generation (modified after Munoz & Everling, 2004). DLPFC, dorsolateral prefrontal cortex; FEF, frontal eye fields; LGN, lateral geniculate nucleus; LIP, lateral intraparietal area; SC, superior colliculus; SEF, supplementary eye fields.

fields (FEF), the supplementary eye fields (SEF), and the dorsolateral prefrontal cortex (DLPFC), all of which are areas known to be crucially involved in the control of voluntary oculomotor behavior. The voluntary pathway further involves the basal ganglia, which receive input from the frontocortical network of oculomotor areas and are able to inhibit saccade programs evolving within the SC. Finally, a feedback loop relays signals from the SC to the FEF via the mediodorsal thalamus. These signals likely reflect corollary discharge information (Sommer & Wurtz, 2002, 2004a, 2004b).

Both the reflexive and the voluntary pathways converge to the SC, the key structure involved in the programming and execution of saccadic eye movements at the subcortical level (see Munoz et

al., 2000; Scudder et al., 2002; Sparks, 2002, for reviews). The SC is a layered structure in the dorsal mesencephalon. Its superficial layers contain stimulus-driven cells that receive input directly from the retina as well as from other visual brain areas (D. L. Robinson & McClurkin, 1989). These cells have well defined visual receptive fields and are organized into a topographical map coding the contralateral visual hemifield. The intermediate and deeper layers of the SC, in contrast, contain motor-related cells, that are correlated with the generation of saccadic eye movements and visual fixation. Again, these cells constitute a retinotopically organized motor map coding for saccades to the contralateral visual field. Saccade amplitudes are continuously represented, decreasing from the caudal to the rostral SC (D. A. Robinson, 1972). Neurons distributed throughout this map exhibit an increasing discharge rate prior to and during saccades directed into their response field (Munoz & Wurtz, 1995a; Sparks et al., 1976; Wurtz & Goldberg, 1972). These cells are often referred to as saccade-related neurons (SN). Many cells in the rostral pole tonically discharge during fixation and pause or decrease firing during most saccades (Munoz & Guitton, 1991; Munoz & Wurtz, 1993a). Consequently, these cells were labeled fixation neurons (FN), first of all in the work of Douglas Munoz and his colleagues (e.g., Munoz et al., 2000; Munoz & Istvan, 1998; Munoz & Wurtz, 1993a, 1993b, 1995a, 1995b).

Thus, FN and SN are active in an antagonistic fashion (see Munoz & Fecteau, 2002, for a review). These reciprocal patterns of activation may be shaped by local inhibitory connections between neurons in the SC motor map. Anatomical studies revealed that horizontal interconnections within the SC are so frequent that "neurons located in any one region in the colliculus could potentially influence any other region" (Behan & Kime, 1996, p. 1031). As a consequence, physiological investigations examined the intrinsic impact of applied stimulation in the intermediate layers of the SC. It was shown in alert behaving monkeys that a neuron's discharge in the motor map is inhibited shortly after electrical microstimulation of remote collicular regions (Munoz & Istvan, 1998). The opposite was observed for neighboring stimulation sites. That is, activating the contralateral FN, resulted in enhanced discharge rates of ipsilateral FN. Similar findings were reported in anesthetized animals (McIlwain, 1982) and reduced preparations of the ferret SC (Meredith & Ramoa, 1998).

More recently, it was argued that the distinction between FN and SN is misleading for at least three reasons. First, the interactions between FN and SN are the same as between SN and SN elsewhere in the motor map of the SC (Basso & Wurtz, 1997; Munoz & Istvan, 1998). Second, de-

sired gaze position rather than a fixed saccade vector appears to be represented in the motor map of the SC (Bergeron & Guitton, 2000, 2002; Bergeron, Matsuo, & Guitton, 2003; Choi & Guitton, 2006), indicating that the rostral end of the SC codes for small gaze-position errors. Finally and in agreement with this notion, FN and SN create a continuum with similar discharge characteristics for gaze-position errors of different amplitudes rather than representing two distinct types of neurons (Bergeron & Guitton, 2002; Krauzlis, Basso, & Wurtz, 1997). Therefore, Krauzlis and his coworkers concluded that "there are no fundamental differences between 'buildup cells' in the caudal SC and 'fixation cells' in the rostral SC; both are tuned for particular, albeit different, amplitudes of motor error" (Krauzlis et al., 1997, p. 1695). Gandhi and Keller (1999b) also discussed the ambiguous role (saccade prevention vs. saccade generation) of neurons in the rostral SC. They perturbed saccades by stimulation of the rostral pole of the SC and compared their trajectories to saccades perturbed by stimulation of omnipause neurons (OPN) in the premotor brainstem region, cells that were clearly associated with visual fixation (see below). Trajectories differed significantly between both conditions showing that cells in the rostral pole are not functionally equivalent to OPN. Consequently, the authors argue against the fixation zone model and favor the saccade zone model as it was already proposed by D. A. Robinson (1972).

Oculomotor plans generated within the SC motor map are directly propagated to the reticular formation where the burst generator circuit is located. This circuit is the final stage of signal processing and drives the oculomotor neurons controlling the eye muscles. It also receives signals from the cerebellum, which probably contain information related to moment-to-moment control of saccades (F. R. Robinson & Fuchs, 2001), e.g., the termination signal that stops the eyes when arrived at a desired location (Lefèvre, Quaia, & Optican, 1998). The details of the processes in the premotor brainstem region (burst generator circuit) were reviewed previously (Fuchs, Kaneko, & Scudder, 1985; Moschovakis et al., 1996; Scudder et al., 2002; Sparks, 2002). It is presumed that SN have excitatory connections to long-lead burst neurons (LLBN) in the reticular formation, while FN are thought to drive OPN (Büttner-Ennever, Horn, Henn, & Cohen, 1999; Gandhi & Keller, 1999a; Paré & Guitton, 1994; Raybourn & Keller, 1977). Accordingly, OPN fire tonically during fixation, but are silent during any saccade. In contrast, LLBN discharge saccade-related bursts of activity which is preceded by low-frequency activity (Everling, Paré, Dorris, & Munoz, 1998; Keller, 1974; Luschei & Fuchs, 1972; Raybourn & Keller, 1977; Strassman, Evinger, McCrea, Baker, & Highstein, 1987). OPN and LLBN relay signals to inhibitory and

excitatory medium-lead burst neurons (MLBN), respectively. It is important to note that these signals are spatiotemporally transformed on their way from the SC to the reticular formation. Different populations of MLBN code for upward, downward, leftward, and rightward saccades, respectively, while saccade amplitudes are coded temporally (Cullen & Guitton, 1997; Fuchs et al., 1985; van Gisbergen et al., 1981). Finally, a saccade is generated by a pulse of activity increase in agonistic oculomotor neurons while antagonistic oculomotor neurons sharply decrease firing rates. These push-and-pull patterns of activity are most probably produced by excitatory and inhibitory input from MLBN (van Gisbergen et al., 1981).

3.2 A neurophysiological perspective on microsaccade generation

Only a few publications address the origin of FEyeM. In particular, there are only a handful of studies that relate to or comment on the generation of microsaccades. In this section, we will shortly review these publications, introducing their results specifically relevant to the generation of microsaccades.

As described above, microsaccades parallel the characteristics and behavioral patterns of large-scale saccades in many ways. Thus, although little is known about the neurophysiological origin of microsaccades, it is reasonable to assume a common neural circuitry for the generation of microsaccades and saccades (see also Engbert, 2006b). Beyond that, microsaccades occur involuntarily (Ditchburn & Ginsborg, 1953; Ratliff & Riggs, 1950), suggesting that subcortical processes might be most relevant in their production. The SC is the most central subcortical institution in the production of saccades (see Figure 3.1), especially of those that are generated involuntarily. Therefore, we hypothesize that this subcortical structure carries neural correlates of microsaccade generation. This hypothesis has been formulated previously (see below), however, direct neurophysiological data concerning its veracity is not yet available. Still there are hints at an involvement of the SC in microsaccade generation.

The first question to ask is whether saccades of a small amplitude are represented in the motor map of the intermediate layers of the SC. In his landmark study, D. A. Robinson (1972) electrically stimulated sites in the intermediate layers of the SC in alert, behaving monkeys. Small saccades with amplitudes down to 0.42° were elicited by stimulation of the very rostral pole of the SC, while

larger saccades were encoded in more caudal areas. Comparable findings were reported by Gandhi and Keller (1999b) and Basso, Krauzlis, and Wurtz (2000), who reliably evoked saccades smaller than 1° by microstimulation of rostral SC neurons. Munoz and Wurtz (1993a, 1995b) reported that cells in the rostral pole of the monkey SC that were postulated to provide a signal related to active visual fixation, did not decrease their discharge rate for small-amplitude contraversive saccades. This finding was replicated by Krauzlis and his colleagues (Krauzlis, 2003; Krauzlis et al., 1997; Krauzlis, Basso, & Wurtz, 2000). Similarly, Anderson, Keller, Gandhi, and Das (1998) documented evidence for neurons discharging before and during small saccades in the rostral SC, however, in this study cells representing saccades smaller than 1° were described only anecdotally. As a consequence of these findings, many authors argued in favor of a continuous representation of gaze-position errors in the saccadic motor map of the SC (Anderson et al., 1998; Basso et al., 2000; Bergeron & Guitton, 2000; Bergeron et al., 2003; Gandhi & Keller, 1999b; Krauzlis, 2003; Krauzlis et al., 1997, 2000; Krauzlis & Dill, 2002). In one of the first of these studies, for instance, Krauzlis et al. (1997) quantified firing rates of neurons situated in the rostral pole of the SC after small displacements of a foveal fixation target. Increased firing rates indicated that even the most rostral cells in the SC motor map code a gaze-position error, though small errors were not always corrected by saccadic eye movements. To put it in the authors' words, "neurons in the rostral SC [...] encode small motor errors that can be either left unresolved or corrected with small saccades or pursuit" (Krauzlis et al., 1997, p. 1695).

It appears that the continuum of saccade amplitudes that find correlates in the SC extends even to very small saccades. These are coded at very rostral sites in the intermediate layers of the SC underneath the superficial, visually driven SC cells representing the fovea. Apparently, though these cells are often referred to as fixation cells, they still possess a movement field. However, knowing that cells in the rostral SC exhibit activity before and during very small contraversive saccades does not necessarily mean that microsaccades are encoded at these sites. Thus, the second question arises: Are microsaccades that occur involuntarily during attempted fixation preceded by enhanced activity in the rostral SC? Such proposals were made by some authors (Gandhi & Keller, 1999b; Munoz et al., 2000), however, they lack physiological examination.

What is known, however, is that the signals measured downstream of the SC, i.e., in the saccadic burst generator, are equivalent for both large saccades and microsaccades. van Gisbergen et al. (1981) recorded activity of MLBN and oculomotor neurons in the mesencephalic reticular

formation of the behaving monkey (see also van Gisbergen & Robinson, 1977). MLBN fired vigorously during microsaccades in the direction a given cell was coding for. Similarly, the firing rate of oculomotor neurons during microsaccades was modulated as expected from the study of large saccades. Compatible data were obtained by Yamazaki (1968) combining eye-movement and electromyogram recordings in humans.

Additional evidence for the role of burst neurons in the generation of microsaccades comes from the clinical literature on fixation instabilities. First, Doslak, Dell'Osso, and Daroff (1983) discussed that square-wave jerks (combinations of two oppositely directed small saccades) might result from spurious error signals and corresponding activity of the saccadic burst generator circuit in the brainstem. Various authors argued that square-wave jerks in patients and in normal observers might be equivalent to enlarged microsaccades (Feldon & Langston, 1977; Ohtsuka, Mukuno, Ukai, & Ishikawa, 1986; Vedel-Jensen, 1966). In this sense, the coupling of two subsequent saccades making up a square-wave jerk is hardly surprising, since a fixation error is created which must be corrected for by a second movement. Yet Ditchburn and Ginsborg (1953) reported couplings of microsaccades if they had amplitudes larger than 10 min-arc. Finally, Ashe, Hain, Zee, and Schatz (1991) reported five cases of patients suffering from microsaccadic flutter. These patients showed small-amplitude (3 to 60 min-arc) back-to-back saccadic oscillations (15 to 30 Hz). Model simulations of the saccadic burst generator revealed that lowering the tonic firing rate of the OPN results in saccadic oscillations like those observed in their population of patients. The authors concluded that malfunction of OPN or modified input to these cells might account for microsaccadic flutter (see also Zee & Robinson, 1979).

With the body of evidence provided, we would like to make a case for the hypothesis that microsaccades are generated by activity in the rostral pole of the SC. First, saccade amplitudes are continuously encoded in a motor map along the rostral-caudal dimension of the SC. The saccadic burst generator receives the bulk of its input from the SC motor map. Microsaccades and large saccades are generated by equivalent signals in the saccadic burst generator. We are aware that a common coding of microsaccades and saccades at the final stage of signal processing (in the saccadic burst generator) does not warrant common coding upstream the brain circuitry of eye movement control (i.e., in the SC). The combination of the findings reported here, however, is highly compatible with a correlation of activity in the rostral pole of the SC and the generation of microsaccades.

3.3 A field model of microsaccade generation

Conceptually, the interplay of saccades and fixations may be viewed as the observable results of continuous integration of motor plans competing for expression. Many models of saccade generation hold this view (e.g., Godijn & Theeuwes, 2002; Kopecz, 1995; Kopecz & Schöner, 1995; Munoz & Fecteau, 2002; Trappenberg, Dorris, Munoz, & Klein, 2001; Girard & Berthoz, 2005, for a review of computational models of saccade generation) and it is in good agreement with the rich knowledge base on the neurophysiology of saccade generation in the SC. These models propose that saccades of different amplitudes are coded in a motor field. In this field, the plan to hold fixation is represented at the very central location. Non-central locations in the field encode saccades, their amplitudes increasing with eccentricity. Thus, suprathreshold activity at a certain site in that field generates either a saccade or fixation, depending on the location activated. Endogenous and exogenous inputs reaching the field will modify the distribution of activity. Local excitation and global inhibition shape the internal dynamics in this motor field. This type of model is able to account for many different behavioral anomalies observed, for instance the decrease of saccade latencies in the gap task including the phenomenon of express saccades (e.g., Munoz & Fecteau, 2002). However, none of these models was ever used to account for microsaccade statistics in simple oculomotor tasks, nor did their authors propose a mechanism for the generation of spontaneous saccades during attempted fixation.

The generation of microsaccades and saccades might be implemented independent of each other. Given the overwhelming body of evidence for the similarity of these movements reviewed above, however, this option appears unlikely. Rather, microsaccade and saccade generation seem to follow the same principles. Thus, microsaccades and large saccades may also be conceived as different motor programs competing for expression. Based on the rich knowledge base on the neurophysiology of saccade generation in the SC and the few indications where microsaccades might find a neural substrate, we propose a common-field model of microsaccade and saccade generation. Following previous models of saccade generation, local excitation and global inhibition govern the dynamics of the field. In detail, neighboring locations in the field activate each other while distant sites reciprocally interact. During fixation, excitation focusses around the central part of the motor map (see Figure 3.2), which receives input from the foveal region of the retina. Assuming a threshold above which activity may trigger a saccadic eye movement (dashed

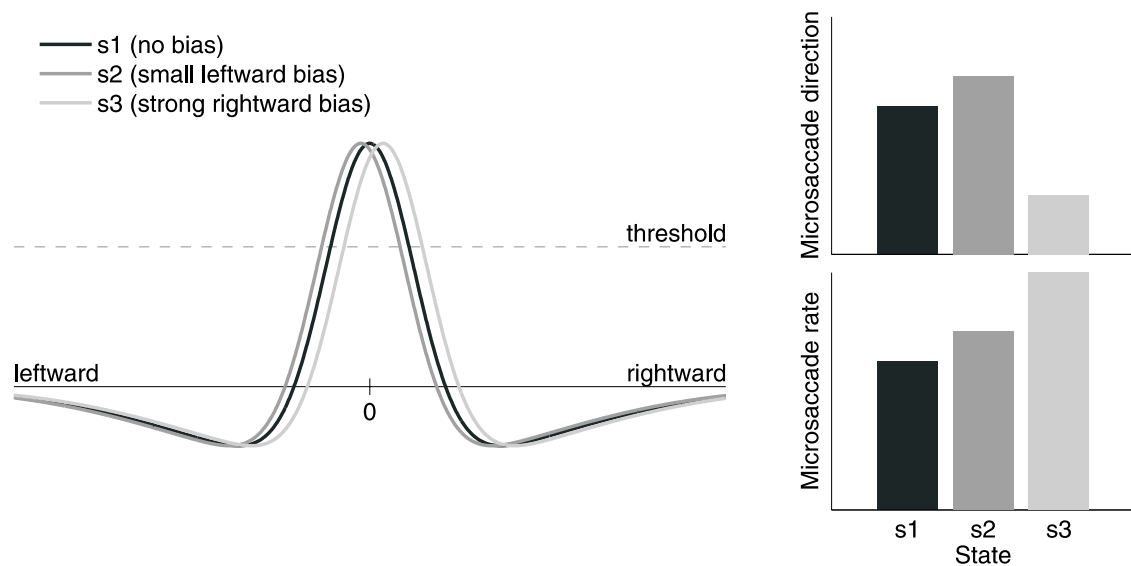


Figure 3.2: Outline of an activation-field model of microsaccade generation. Three different states of activity distributions in the movement field are shown: no bias, small leftward bias, and strong rightward bias. Activity distributions are hypothesized to translate into microsaccadic behavior. Microsaccade direction (here fraction of leftward microsaccades) is a direct function of the location of the “hill” of activity. Since microsaccades may not be triggered by activation near zero (Krauzlis et al., 1997), directional biases would implicate enhancements in microsaccade rate.

line in Figure 3.2), small-amplitude saccades could occur. Thus, microsaccades intrude while the system is in the state of steady fixation.

In general, any saccade amplitude may be generated, if the associated activity is above threshold. Two different mechanisms may bias the distribution of microsaccade directions. First, noise inherent to the field might modulate activity and, hence, bias the direction of microsaccades. Second, both endogenous and exogenous input to the field (e.g., a peripheral onset) will slightly modulate the distribution of activity at the central region (see Figure 3.2), since a mutually inhibitory network shapes activity in the field. This might account for direction-specific enhancements of microsaccade direction observed in spatial-cuing experiments (Engbert & Kliegl, 2003b; Galfano et al., 2004; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005). In addition and in line with the results in these studies, directional biases would implicate enhancements in microsaccade rate, since the probability of saccade generation increases with eccentricity in the rostral pole of the SC motor map (Krauzlis et al., 1997).

Since during fixation, activity may be above threshold at any time, a temporal trigger mechanism is needed to account for the rate of microsaccades typically observed during fixation. The

core of our model does not depend on how this timing process is implemented. However, various approaches to this issue could be considered. For instance, Engbert and Mergenthaler (2006) suggested that a trigger signal might depend on the variability of visual input signals. If slow drift movements of the eyes do not suffice, microsaccades might occur to enhance retinal image slip. Moreover, one might assume an autonomous timing mechanism for the generation of saccades (Richter, Engbert, & Kliegl, submitted). The dynamics of this mechanism might be subject to random variation and depend on current oculomotor needs. Thus, after the passage of some time following a saccadic eye movement, a new movement will be initiated. Alternatively or in addition to that, microsaccades might only be observed if the current gaze fixation error exceeds a critical value. Note that very small microsaccades may not be observed, since the fixation error created might not reach a critical level (cf., Krauzlis et al., 1997). Whatever the timing process might be like, the metrics of a triggered microsaccade depend on the current distribution of activity in the motor field; higher activity would be associated with a higher likelihood of motor expression.

3.4 Behavioral predictions of the common-field model

Microsaccades are observable behavior, for which we can derive predictions from the model proposed. Assuming that our common-field model is representative of the system underlying microsaccade and saccade generation, microsaccade occurrence is indicative of the state this system is in at a given point in time. As a consequence, microsaccade statistics would provide us with a measure of the activity at the central part of the movement field in which saccade generation is accomplished. In detail, we suggest that, first, microsaccade rate is an index of the amount of this activity. Second, microsaccade amplitude distributions should be dictated by the spread of the “hill” of activity in the motor field. Finally, microsaccade direction biases would be related to the mean center of the activated site in the field.

Distributions and rates are global measures, since they depend on pooling over many trials. As a consequence, they will inform about the general field dynamics in a given paradigm and may only be related to aggregated performance measures such as mean saccade latencies. In addition to global analyses of microsaccade statistics, the impact of single microsaccades on performance in single trials of saccade tasks may be informative with regard to the implementation

of microsaccades. One prediction would be that in trials in which a microsaccade is observed at the time at which a saccade is required, response latencies should be longer as compared to trials in which no microsaccades are observed. The rationale behind this prediction is that given a microsaccade was observed, it must have originated from activity at a central site in the movement field. Central sites are in competition with other saccade-related sites in the field. Thus, to generate a (large) saccade, competitive fixation-related activity must be overcome, before the saccade may be executed.

To our knowledge, microsaccade statistics were rarely related to performance in oculomotor tasks, however, the handful of publications deserve a short review. In one study, Kingstone, Fendrich, Wessinger, and Reuter-Lorenz (1995) tested whether microsaccades are responsible for the gap effect. When Saslow (1967) first reported the gap effect (a sharp decrease in response latencies when a fixation stimulus disappears shortly before a saccade target is presented), he proposed that the rate of microsaccades might decrease during the gap period, thereby reducing the likelihood of time-consuming refractory periods. Kingstone et al. (1995) tested this hypothesis by removing trials in which a microsaccade was observed during the gap period. Only a few trials contained microsaccades in this 200 ms time window; saccadic reaction times did not change after removal of these trials. Consequently, the authors were able to conclude that microsaccades did not *cause* the gap effect in their study. For one subject, Kingstone et al. (1995) also contrasted trials with trials without microsaccades during the gap period. On average, this subject was indeed faster when no microsaccade occurred (as predicted by the model proposed here), however, the numerical difference was not statistically reliable.

Gowen and Abadi (2005) also related microsaccades to the performance in standard oculomotor paradigms (gap, step, overlap, and antisaccade tasks). In ten subjects, the frequency of saccadic intrusions was correlated with mean saccade latency, the percentage of express saccades, and direction errors. By this, the authors aimed at testing the hypothesis that saccadic intrusions might be the result of imbalance in the saccadic system, favoring saccade initiation as opposed to saccadic inhibition. No significant correlations were found in this study. Unfortunately, the authors remained on a very global level of data analysis. Thus, no far-reaching conclusions can be drawn from that study concerning the implementation of microsaccades in the oculomotor system.

As we pointed out in this section, our common-field model of microsaccade and saccade generation generates a number of predictions, which can be evaluated empirically. Only a few studies generated data which could be of interest with regard to the questions raised here. In addition, these papers pursued different research issues. The following chapters aim at testing a number of implications associated with the model proposed here. Empirical analyses will include both the detailed analysis of microsaccade dynamics and the interactions of microsaccades with large reflexive and voluntary saccades.

Chapter 4

On the implementation of microsaccadic inhibition

As described in Section 2.4, microsaccade statistics were clearly modulated in spatial cuing experiments (Engbert & Kliegl, 2003b; Galfano et al., 2004; Hafed & Clark, 2002; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005). The finding common to these studies is the microsaccade-rate signature: After the presentation of an attentional cue, microsaccade rate drops down to a minimum (henceforth inhibition) before showing a rebound period (henceforth enhancement) and a final resettlement at the initial baseline level (Engbert & Kliegl, 2003b; Galfano et al., 2004; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005). In addition, microsaccade-direction statistics appeared to be indicative for the dynamic allocation of covert attention in such tasks (Engbert & Kliegl, 2003b; Galfano et al., 2004; Hafed & Clark, 2002; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005). It is important to note that, in contrast to the direction effect, the microsaccade-rate signature was observed for any display change, allocating spatial attention or not (Engbert & Kliegl, 2003b), and even in the absence of visual changes using auditory spatial cues (Rolfs et al., 2005).

Most previous studies that reported the microsaccade-rate signature remained on a descriptive level. Only recently, Engbert (2006b) examined the timeline of this effect, thereby constraining the number of physiological systems involved in its generation. Compiling a number of studies that reported the microsaccade-rate signature, the author concluded that the very robust inhibitory part of the signature can only be explained in terms of a very fast subcortical processing circuit,

probably involving the retinotectal pathway. In contrast, the enhancement phase, which is more variable across experiments, appears to be modulated by higher cognitive processes. Thus, for microsaccadic inhibition, Engbert (2006b) proposed, like other authors before (Laubrock et al., 2005; Rolfs et al., 2005), that the superior colliculus (SC) likely assists its implementation. Inhibition in response to irrelevant stimuli has also been observed for large saccades (saccadic inhibition) in a broad range of eye-movement tasks including simple saccade paradigms (Reingold & Stampe, 2002), reading (Reingold & Stampe, 1999, 2000, 2003, 2004; Stampe & Reingold, 2002), visual search (Reingold & Stampe, 1999, 2000, 2004; Stampe & Reingold, 2002), and picture viewing (Pannasch, Dornhöfer, Unema, & Velichkovsky, 2001). Thus, the mechanisms underlying microsaccadic inhibition might also apply to saccadic inhibition.

So far, however, nothing is known about the characteristics of microsaccadic inhibition, despite the fact that microsaccade rate drops off dramatically. The question remains how microsaccadic inhibition is implemented. In particular, can we derive predictions about what happens at the level of the SC during microsaccadic inhibition? Engbert (2006b) suggested that a stimulus-related signal may result in a transient increase of the mean-field activation in the motor map of the SC. As a result of global inhibition within this map, activity at the rostral pole would decrease. Thus, according to the hypothesis that microsaccades are the result of activity in the rostral pole of the SC (see Chapter 3.2), microsaccade rate drops off. This mechanism is in agreement with the model of microsaccadic inhibition put forward here. Enhanced activity triggered by a transient stimulus at some location in a saccadic motor map competes with activity at a fixation-related site. Figure 4.1 shows how activity at the central pole of a saccade map decreases with time. Assuming that above-threshold activation is a necessary condition for microsaccade generation, fewer microsaccades may be generated. Moreover, this model predicts that inhibition affects microsaccades with large amplitudes first, since corresponding activity sooner falls short of threshold. This hypothesis is in line with current models of oculomotor control on the level of the SC, in which fixation-related activity is part of the saccade motor-map (e.g., Godijn & Theeuwes, 2002; Kopecz, 1995; Kopecz & Schöner, 1995; Munoz & Fecteau, 2002; Trappenberg et al., 2001). Thus, an understanding of the implementation of microsaccadic inhibition would benefit from an analysis of the temporal evolution of microsaccade amplitude.

In addition to closer analyses of microsaccadic inhibition, experimental manipulations of the effect may evaluate the proposed model of microsaccade generation. If microsaccadic inhibition

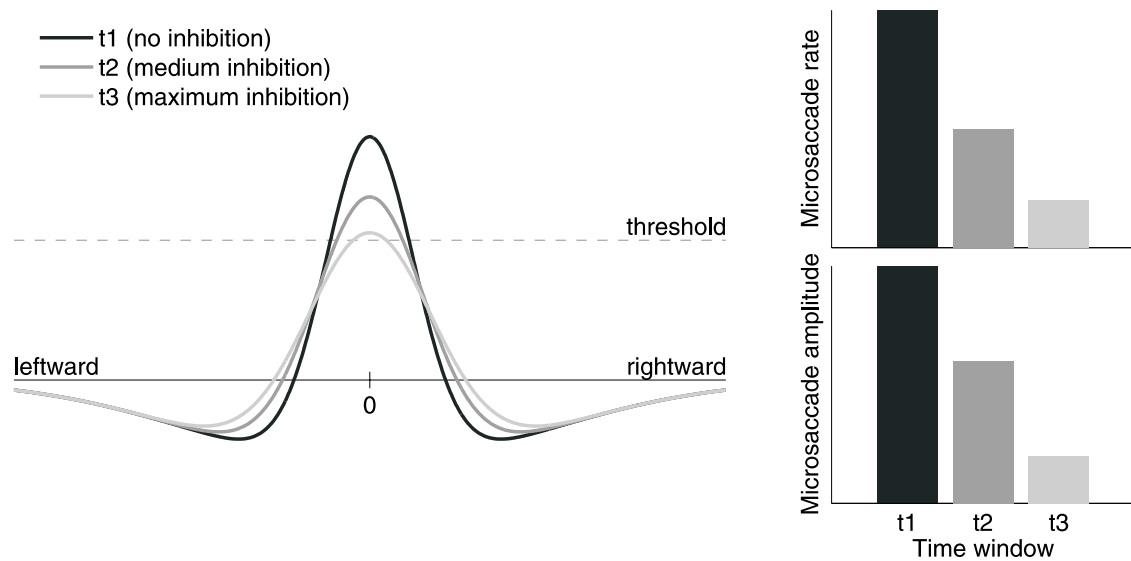


Figure 4.1: Process causing microsaccadic inhibition as explained by a field model of saccade generation. As a result of enhanced activation somewhere in the peripheral motor map (not shown), activity at the fixate center decreases. Consequently, fewer microsaccades may be generated. This model predicts that inhibition affects microsaccades with large amplitudes at first, since corresponding activity falls below threshold.

relates to subcortical processes in the oculomotor system, its time course should be sensitive to the properties of the irrelevant stimulus. First, as revealed by single-cell studies in monkeys and cats, the latency with which auditory input can modulate activity in the SC motor map is lower than that of visual stimuli, even when visual input directly impinges on the SC via the retinotectal pathway (Jay & Sparks, 1984, 1987; Stein & Meredith, 1993). Thus, if the SC constitutes a major neural substrate of microsaccadic inhibition, auditory stimuli should elicit faster inhibition than visual ones. A re-inspection of data from a previous study, where microsaccadic inhibition was observed for both visual and auditory stimuli, suggests that this might be the case (Rolfs et al., 2005, compare Figures 2 and 3). Second, in response to isoluminant visual stimuli, microsaccadic inhibition should be delayed, since the retinotectal pathway is “blind” to stimuli that are isoluminant to the background (Schiller & Malpeli, 1977). Thus, isoluminant input must be relayed via the corticotectal pathway before impacting ongoing processes in the SC. It is known that the latency of express saccades (saccades with extremely short latencies) is much longer in response to low contrast stimuli as compared to high-contrast targets. In fact, the distribution of saccade latencies was simply shifted towards longer latencies when lower target contrasts were used in simple

saccade tasks (Boch, Fischer, & Ramsperger, 1984; Kingstone & Klein, 1993; McPeck & Schiller, 1994; Reuter-Lorenz, Hughes, & Fendrich, 1991). Like microsaccadic inhibition, express saccades are thought to be generated by a low-level oculomotor circuit, involving the SC (e.g., Dorris, Paré, & Munoz, 1997; Munoz & Wurtz, 1992; Schiller, Sandell, & Maunsell, 1987). Therefore, longer onset latencies of the inhibition of microsaccades can be expected if luminance contrast of the stimulus is minimized.

In the present experiment, uninformative stimuli were used to trigger microsaccadic inhibition in the course of a demanding visual discrimination task. Two experimental factors were manipulated, the modality of the irrelevant stimulus (auditory or visual) and, for visual stimuli, their luminance (luminant or isoluminant). Thus, both analytical and experimental approaches were used to shed new light on the processes underlying microsaccadic inhibition and, hence, on the system implementing microsaccade generation. We suggest that a detailed analysis of microsaccadic inhibition is basal for understanding the interaction of microsaccades and saccades, which is the key topic of this dissertation.

4.1 Methods

4.1.1 Participants

Data were collected in two epochs. In a first epoch, 40 undergraduate and high-school students performed one session each. In a second epoch, 20 undergraduate and high-school students completed two sessions each (within 1 to 13 days, 4.6 days on average). Subjects were paid 6€ per session or received study credit for their participation. They were 16 to 27 years old (18.9 years on average), had normal or corrected-to-normal vision, reported normal hearing, and were in good health. This experiment as all the experiments reported in this thesis was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and individuals gave their informed consent prior to their participation in the study.

4.1.2 Experimental setup and eye-movement recording

Participants were seated in a silent and dimly lit room with the head positioned on a chin rest, 50 cm in front of a computer screen. Eye-movement data were recorded using an EyeLink-II

system (SR Research, Osgoode, Ontario, Canada) with a sampling rate of 500 Hz and a noise-limited spatial resolution better than 0.01° . Visual stimuli were presented on a 19-inch EYE-Q 650 Monitor (1024 x 768 resolution or 40° by 30° of visual angle; frame rate 120 Hz) using a gray background color. Auditory stimuli were played via Sennheiser HD 520 II headphones. The experiment was controlled by an Apple Power Macintosh G4 computer. Manual responses were recorded via the standard keyboard connected to the computer. The experimental software controlling stimulus display and response collection was implemented in Matlab (MathWorks, Natick, Massachusetts, USA), using the Psychophysics (Brainard, 1997; Pelli, 1997) and EyeLink (Cornelissen, Peters, & Palmer, 2002) toolboxes.

4.1.3 Procedure

Participants were divided into two groups, each consisting of 30 individuals (20 from the first and 10 from the second data-collection epoch). Both groups performed the same visual discrimination task, but were presented with either visual or auditory irrelevant stimuli. Aside from the modality of the irrelevant stimuli, the procedure was identical for both the visual and the auditory group.

After a key training, linking "red" to the up- and "green" to the down-arrow key, participants performed eight randomly ordered practice trials, introducing the task, and 240 test trials. The practice trials were comparable to the test trials in all respects except that there was no fixation check at the beginning of a trial. Before the first and after every 15th test trial, the eye tracker was calibrated (standard 9-point grid) and calibration was validated. Before every fifth trial, a drift correction was carried out. Before each trial, the fixation spot was displayed at the center of the computer screen. Participants began fixating and correct fixation was checked. If gaze position was not detected in a region four times as large as the fixation spot, the experimenter carried out a drift correction and started again. If the eyes were still not detected within the critical area, the calibration was repeated.

Figure 4.2 illustrates the sequences of stimulation used in the visual and auditory conditions. Participants were required to look at the fixation spot during the whole trial while they performed a visual discrimination task. That is, after a variable period, the dark gray fixation spot changed its color shortly to green or red (isoluminant to the previous gray) and disappeared subsequently. Participants made speeded responses to this target stimulus, indicating which color was displayed. In 120 of 240 trials, task-irrelevant stimuli (a yellow ring surrounding the fixation spot in the visual

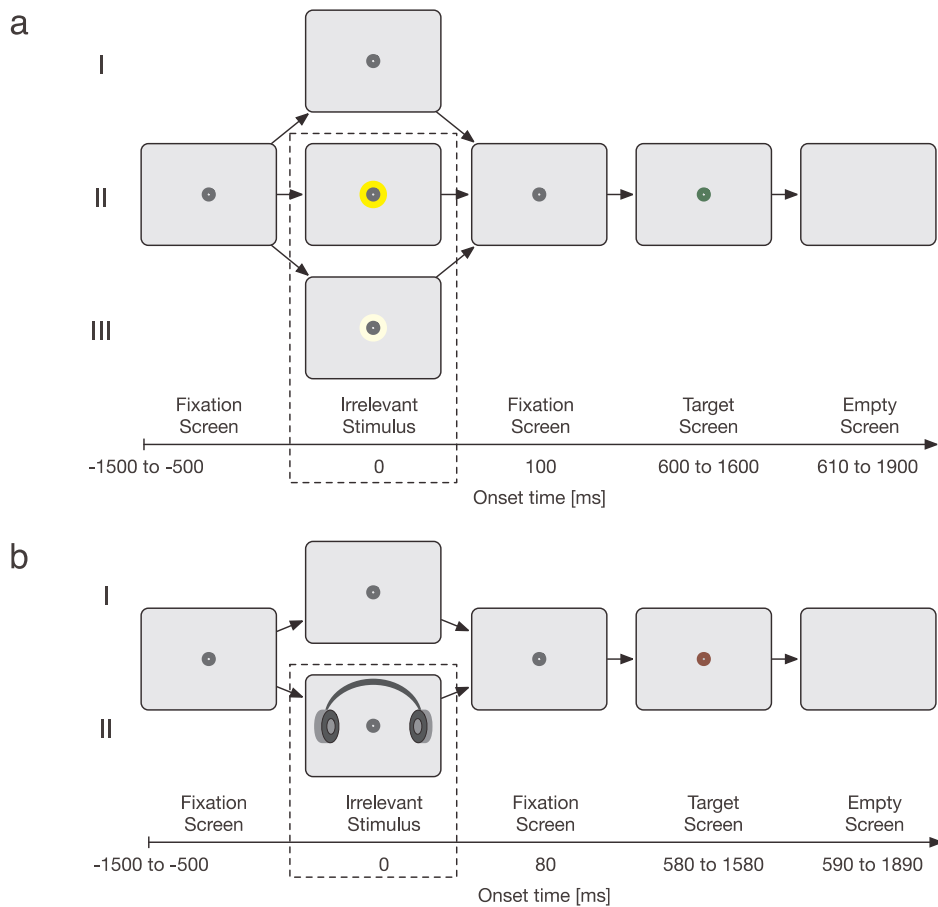


Figure 4.2: Illustration of sequences of stimulation in two experimental groups performing (a) the visual and (b) the auditory conditions. In half of the trials (sequences II and III in panel a and sequence II in panel b), an irrelevant stimulus was presented while participants waited for a visual discrimination target replacing the central fixation spot. Stimuli in the visual condition were either isoluminant (II) or had a luminance contrast (III) to the background.

condition; a noisy sound in the auditory condition) were interspersed during the fixation period, and participants were instructed to simply ignore them. A within-subject factor luminance was nested in the visual condition; in 60 trials the yellow ring was isoluminant to the background and in 60 trials non-isoluminant. For the sake of convenience, we will refer to the latter condition as luminant. The two types of target stimuli (red or green) replacing the fixation stimulus after that period had equal probability over the 240 trials and within each condition. All kinds of trials were presented in a random order.

The fixation times preceding and following irrelevant stimuli, respectively, ranged from 500 to 1500 ms of fixation. Irrelevant visual stimuli were presented for 100 ms, irrelevant tones had

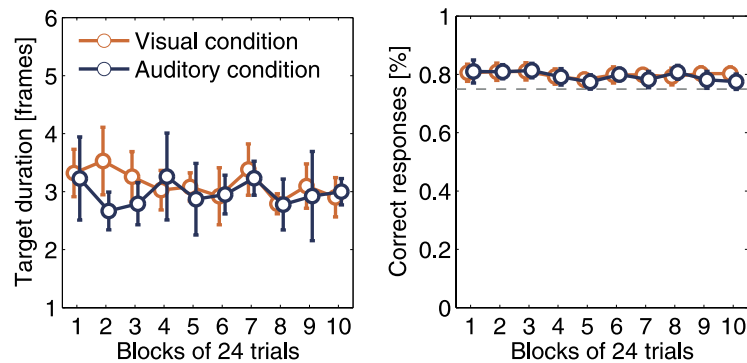


Figure 4.3: Median of target presentation times and mean of percentage of correct responses. Target presentation times adapted using the weighted up-down method proposed by Kärnbach (1991). The envisaged performance levels of 75% correct responses (dashed line in right panel) were slightly exceeded for reasons of ceiling performance (see text). Error bars are bootstrapped 95% confidence intervals.

a duration of 82 ms. Thus, altogether, fixation periods had a duration of 1100–3100 ms in the visual condition and 1082–3082 ms in the auditory condition, respectively. Target presentation times adapted to the participants' performance, such that a relatively constant performance level of about 75% correct responses was maintained over the whole experiment. To achieve this, the weighted up-down method proposed by Kärnbach (1991) was used. In the first trial of a session, the target stimulus was presented for 12 monitor frames (100 ms). After a correct response (e.g., up-arrow key after a red target) presentation time was reduced by 1 frame. After incorrect responses, it was increased by 3 frames. Figure 4.3 shows the median of target presentation times and the mean of percentages of correct responses as a function of blocks of 24 trials (each accompanied by bootstrapped 95% confidence intervals; see below). An average performance of 80% correct responses was produced; the median of target presentation times was about 3 frames in both the visual and the auditory condition. The deviation from the expected performance level of 75% is due to the limitation of stimulus presentation times to units of frames. The minimum presentation time was 8.3 ms (1 refresh frame of the screen). Once a participant performed correctly on a trial with a one-frame target display, presentation time could not be decreased any further, resulting in what could be called a ceiling effect of performance. The maximum target presentation time was set to 250 ms (30 frames). Hence, an incorrect response in a trial with a target duration of 27, 28, 29, or 30 frames was followed by a trial with a 30 frames target-presentation time.

Incorrect responses released an error feedback; correct responses directly initiated the next fixation check. After every eighth trial, participants were presented with a performance feedback, included to induce a strong focus on the visual discrimination task. This feedback screen showed the participant's evolution of mean latencies of correct responses averaged over blocks of eight trials.

4.1.4 Stimuli

The fixation spot was a ring with a diameter of 0.8° of visual angle in dark gray color and an inset with a diameter of 0.1° . Target stimuli were identical to the fixation spot with respect to form and luminance, i.e., they only differed in color (red or green). Irrelevant stimuli were visual for one group and auditory for another group of participants. Visual irrelevant stimuli were yellow rings transiently surrounding the fixation spot (duration: 100 ms). A 70 dbA approximated white noise sound (duration: 82 ms) was employed as irrelevant stimuli in the auditory condition. Response errors triggered feedback composed of a central white circle with a diameter of 2.4° and a binaural 660 Hz tone at 70 dbA for 82 ms.

Before an experimental session, isoluminant colors were determined on an individual basis using a flicker fusion method. Participants were instructed to minimize the flickering of two colored spots alternating with 24 Hz by adjusting the luminance of one color. This procedure was performed for the different pairs of colors employed: In both experiments, the colors of the target stimuli (green and red) were adjusted to the dark gray of the fixation spot. In addition, in the visual condition, the color of the irrelevant isoluminant stimulus was adjusted to the light gray of the background.

4.1.5 Data preparation

Microsaccades were detected using an improved version of an algorithm proposed by Engbert and Kliegl (2003b). The algorithm was described in detail by Engbert (2006b). Velocities were computed from successive eye positions recorded in a trial. Microsaccades were detected in 2D velocity space using thresholds for peak velocity and minimum duration. A relative threshold of 6 SDs of the velocity was used and a minimal duration of 6 ms (or four data samples) was mandatory. We considered only binocular microsaccades, that is, microsaccades detected in both eyes with temporal overlap.

Trials including saccades larger than 1° of visual angle were discarded, as were trials with incorrect responses and response times (RTs) shorter than 70 ms or longer than 1000 ms. Some trials had to be excluded due to data loss during eye-movement recording. To be included in the analyses, a participant had to contribute at least 60 trials with and 60 trials without an irrelevant stimulus. In the visual condition at least 30 trials with isoluminant and 30 trials with luminant irrelevant stimuli had to meet the given criteria. In the visual condition, 28 participants contributed 138 to 463 trials to the final data analyses, resulting in a total of 6560 trials (out of 9120 or 71.9%) in which 24901 microsaccades were detected. In the auditory condition, 23 participants contributed 140 to 453 trials to the final data analyses, resulting in a total of 4882 trials (out of 7440 or 65.6%) in which 21057 microsaccades were detected.

4.1.6 Data analysis

To compute continual microsaccade-rate evolutions, we applied a causal filter similar to that described in Engbert (2006b), which has been adopted from analyses of neural firing rates (Dayan & Abbott, 2001). For each participant, microsaccadic events were collapsed across all trials of a certain condition (pooled from all sessions performed). Microsaccade onsets t_{MS} were aligned relative to a critical event, e.g., the onset of the irrelevant stimulus. The series of microsaccadic events of $i = 1, 2, 3, \dots, N$ at times t_i is formalized by

$$\rho(t) = \sum_{i=1}^N \delta(t - t_i), \quad (4.1)$$

where δ denotes Dirac's δ -function. Microsaccade rate $r(t)$ was then determined by temporal averaging, i.e.,

$$r(t) = \int_0^\infty \omega(\tau) \rho(t - \tau) d\tau, \quad (4.2)$$

applying the smoothing kernel

$$\omega(\tau) = \alpha^2 \tau \exp(-\alpha\tau). \quad (4.3)$$

The form of the function $\omega(\tau)$ combined with the fact that $r(t)$ is integrated from 0 to infinity results in a very conservative computation of the temporal evolution of microsaccade rate. That is, microsaccades occurring at time t do not have any impact on $r(t)$. Rather, $r(t)$ is only influenced by past events. In addition, the extent of this conservativeness in computing $r(t)$ strongly depends

on the choice of the decay parameter α . To eliminate these problems, the whole function $r(t)$ will be shifted to finally get

$$\hat{r}(t) = r\left(t + \frac{1}{\alpha}\right) \quad (4.4)$$

ensuring that the maximum impact on microsaccade rate $r(t)$ comes from microsaccades observed at time t . In our analyses, t had a temporal resolution of 1 ms. To transform the rate to Hz, thus, $r(t)$ was multiplied by 1000 and divided by the number of contributing trials. Finally, to generate mean microsaccade-rate evolutions, individual rates were averaged across participants. The decay parameter was set to $\alpha = 1/20$.

Wherever confidence intervals are provided, these were computed using a simple bootstrapping technique (Efron & Tibshirani, 1993). From an original sample of N values, 1000 bootstrap samples were generated, each by selecting (with replacement) N values of the original sample. The 1.96-fold of the standard deviation of the means of these 1000 bootstrap samples was computed to generate 95% confidence intervals ($CI_{95\%}$) of the mean of the original sample. In graphical illustrations, confidence intervals will often allow the reader to compare different conditions by so-called “rules of eye” (Cumming & Finch, 2005). For within-subject comparisons, uninformative between-subject variance was therefore removed (Cousineau, 2005); these confidence intervals will be labeled $CI_{95\%ws}$.

4.2 Results

4.2.1 Performance in the task

Figure 4.4 depicts response latencies and error rates in the different conditions. The histograms plot distributions of latencies for correct responses upwards and incorrect responses downwards. The two panels on the right display mean response latencies and error rates as a function of condition. Apparently, irrelevant stimuli in the visual condition were accompanied by a reduction in response latencies as compared to the no-stimulus trials. In the auditory condition, the irrelevant stimulus did not influence the manual response latencies. Accordingly, a mixed-model ANOVA with stimulus presence (yes or no) and modality group (visual or auditory) as independent variables revealed an interaction of these two factors; $F(1, 49) = 23.84, p < 0.001$. In addition, a main effect of stimulus presence was evident; $F(1, 49) = 15.51, p < 0.001$. Despite the shorter mean target-

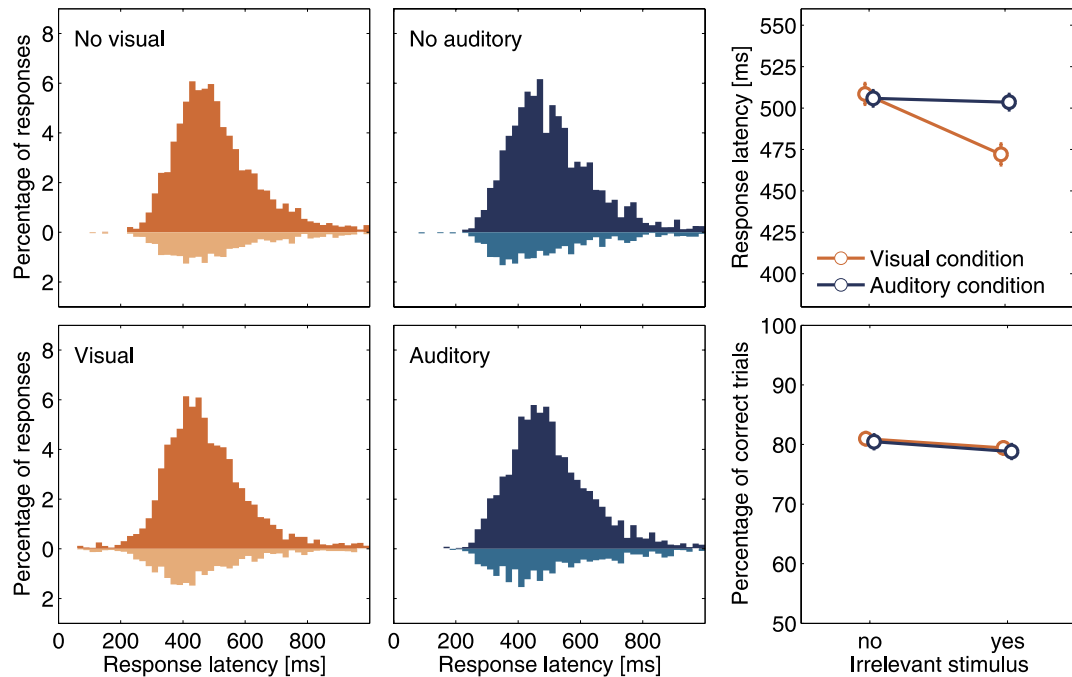


Figure 4.4: Performance as a function of the presence of an irrelevant stimulus. The histograms show response latency distributions for both the visual and the auditory conditions with correct responses plotted upwards and incorrect responses plotted downwards. The two panels to the right show an aggregation of these data. The upper-right panel depicts mean latencies of all responses in the visual (blue) and auditory (brown) conditions. The lower-right panel shows the percentage of correct responses as a function of stimulus condition. Error bars are $CI_{95\%}$ s.

presentation times (see Figure 4.3), no main effect of modality was observed; $F < 1$. Post-hoc contrasts revealed that stimulus presence affected response times in the visual modality only (visual: $t[27] = 5.43; p < 0.001$; auditory: $t[22] = 0.47; p = 0.64$). On average, participants were 36 ms faster if an irrelevant visual stimulus was presented while waiting for the discrimination target.

No differences were observed in the error rates, neither between groups (visual or auditory) nor as a function of the presence of an irrelevant stimulus. These results were corroborated statistically in a mixed-model ANOVA with stimulus presence (yes or no) and modality group (visual or auditory) as independent variables. There was a slight but insignificant difference contingent on stimulus presence; $F(1, 49) = 3.95, p = 0.052$. Moreover, there was no effect of modality, nor an interaction of the two factors; $F_s < 1$.

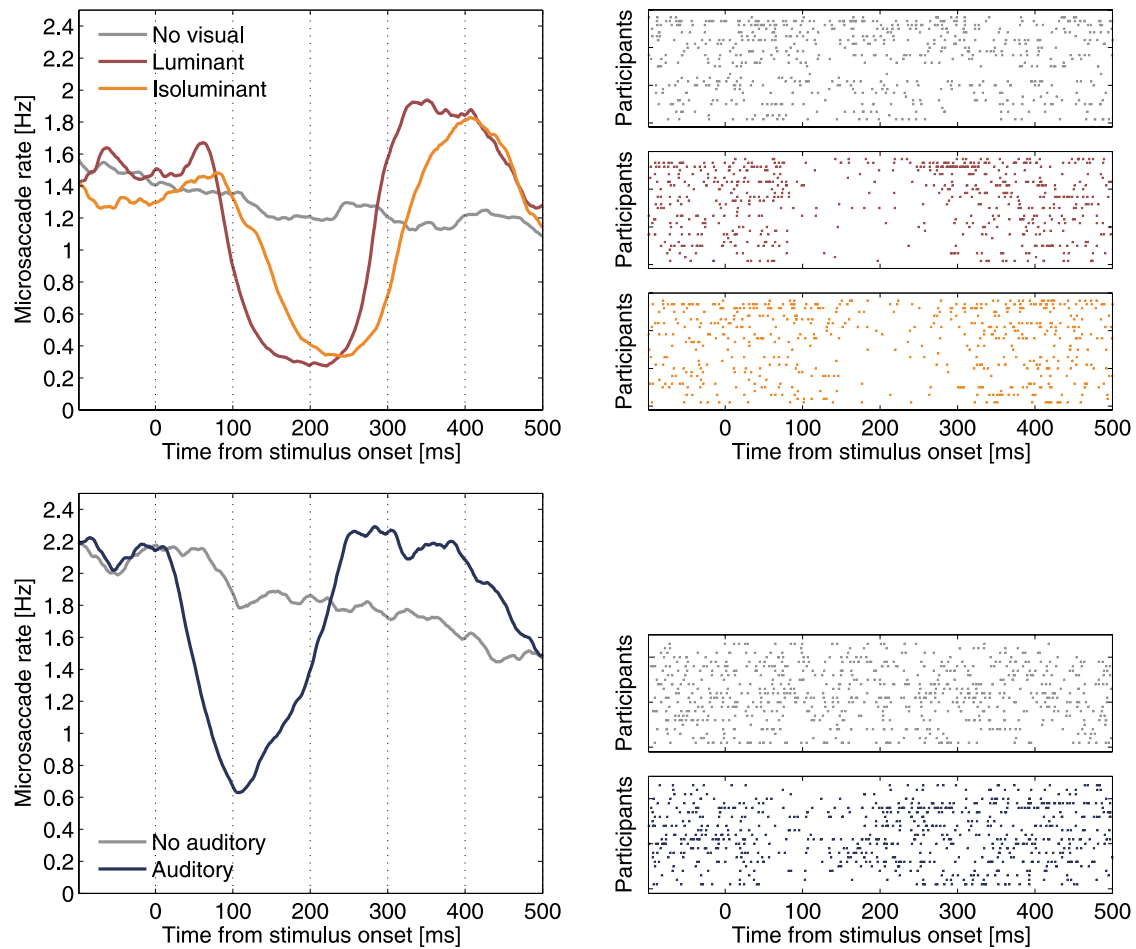


Figure 4.5: Microsaccade rate in the visual and auditory conditions. The left panels display microsaccade-rate evolutions computed using an asymmetric filter and averaged across participants. The raster plots on the right show the corresponding individual microsaccade data from 30 randomly chosen trials for each participant. Each line represents one participant, each dot corresponds to a microsaccade observed at the corresponding point in time.

4.2.2 Microsaccade rate

The dynamics of microsaccade rate $r(t)$ in response to the irrelevant stimuli were determined along the lines described in the methods section. The results are plotted in Figure 4.5. Both the visual and the auditory stimuli produced a strong inhibition in the microsaccade rate. Figure 4.5 also displays raster plots, in which each line shows a participant's microsaccade data from 30 randomly chosen trials. Eyeballing these plots gives the strong impression that the inhibition effect is stable across observers.

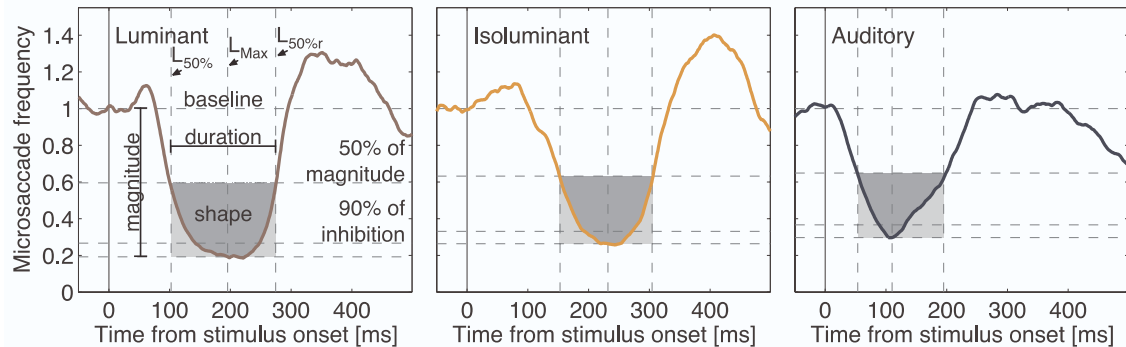


Figure 4.6: Microsaccadic inhibition in response to irrelevant stimuli. Microsaccade frequency is a standardized representation of the microsaccade rate. Latencies ($L_{50\%}$ = Latency to 50% of maximum inhibition; L_{Max} = Latency to maximum inhibition; $L_{50\%r}$ = Latency to return to 50% of maximum inhibition), duration, magnitude, and shape of microsaccadic inhibition are illustrated in the first panel.

To describe the microsaccade-rate evolutions in more detail and to examine its differences between conditions, microsaccade rates were standardized in a first step. To this end, a baseline rate was determined by averaging the rate across the last 50 ms before the onset of the irrelevant stimulus. The microsaccade-rate evolution was then normalized to a baseline of 1 Hz by dividing the rate at each point in time by the baseline rate (expected microsaccade rate). The resulting curves of microsaccade-frequency $f(t)$ are depicted in Figure 4.6.

Subsequently, various measures of inhibition were computed, most of which were introduced in the work of Reingold and Stampe on saccadic inhibition (e.g., Reingold & Stampe, 2004). First, we located the *minimum saccadic frequency* in a time window from 0 to 400 ms. Second, we defined the *bottom of the dip* as the period at which microsaccade frequency was below the minimum plus 10% of the difference between 1 (the baseline) and the minimum frequency. The center of the bottom of the dip will be referred to as *latency to maximum saccadic inhibition* (L_{Max}). Third, the *magnitude of inhibition* μ (henceforth, *magnitude*) is 1 minus the frequency at L_{Max} . Thus, higher values indicate stronger inhibition. Fourth, we computed the *latency to 50% of maximum saccadic inhibition* ($L_{50\%}$). i.e., the time at which inhibition reached 50% of its magnitude. To this end, a period before L_{Max} was determined during which inhibition was in between $\frac{1}{3}$ and $\frac{2}{3}$ of its magnitude. The center of this period was taken as $L_{50\%}$. The same was done for the period after L_{Max} to determine the point of return to 50% of magnitude ($L_{50\%r}$). Finally, the *duration of inhibition* δ (henceforth, *duration*) was the interval while the inhibition remained above 50% of its magnitude.

In addition, we propose a new measure—the *shape of inhibition* σ (henceforth, *shape*). This measure is sensitive to the form of the dip of the frequency curve. A visualization of the shape (and all other measures of inhibition) is displayed in Figure 4.6 (the dark share of the shaded area). The shape is computed as the ratio of areas

$$\sigma = \frac{1}{0.5\mu\delta} \int_{t=L_{50\%}}^{L_{50\%r}} (1 - 0.5\mu - f(t))dt, \quad (4.5)$$

which translates to

$$\sigma = \frac{2}{\mu} - 1 - \int_{t=L_{50\%}}^{L_{50\%r}} f(t)dt, \quad (4.6)$$

since $\delta = L_{50\%r} - L_{50\%}$. Thus, the shape has a range of $0 < \sigma \leq 1$ with higher values describing more bellied curves and lower values covering a range of rather pointed curves.

These measures of inhibition could not be reliably estimated on the level of participants, since, most of the time, individual microsaccade-frequency evolutions are very noisy. Therefore, we employed a bootstrap method to create a sample of more stable frequency curves that is representative of the assumed underlying frequency-evolution population. This algorithm can be used to reliably estimate standard errors in this type of data (Efron & Tibshirani, 1993). One hundred independent bootstrap samples were created, each consisting of N individual frequency evolutions drawn with replacement from the pool of N observed individual microsaccade-frequency evolutions. That is, in a given bootstrap sample, participants could be included 0 to N times, but over the whole set of replications, a participants' data were included approximately equally often. For each replication, a mean frequency evolution was computed, resulting in 100 bootstrap frequency evolutions and corresponding measures of inhibition. Standard deviations of the various measures of inhibition were computed across the 100 replications. These approximate the standard errors of means (Efron & Tibshirani, 1993), which, in turn, were used to compute corresponding $CI_{95\%}$. This procedure was conducted for all conditions under investigation (luminant, isoluminant, and auditory). It is important to note that for the two visual conditions, the same 100 bootstrap samples were used, effectively controlling for between-subject variability.

Table 4.1 displays means with associated $CI_{95\%}$ of the various measures of inhibition that were estimated using this procedure; mean differences between conditions ($\pm CI_{95\%}$) will be given in the text. Let us first consider the effects of luminance on microsaccadic inhibition. The latency of inhibition was strongly modulated by the luminance of the irrelevant stimulus with luminant

stimuli resulting in faster inhibition than isoluminant ones. This difference in latencies between the two conditions was 51 ± 18 ms as measured by $L_{50\%}$ and 35 ± 20 ms as measured by L_{Max} . In contrast, the strength of inhibition as measured by its duration, its magnitude, and its shape was not reliably affected by the luminance manipulation; confidence intervals of these differences included zero.

In the auditory condition, a very short latency of inhibition was observed as measured by $L_{50\%}$ (53 ms) and L_{Max} (110 ms). Overall, the latency of inhibition was shorter in the auditory than in the visual conditions. $L_{50\%}$ differed by 50 ± 14 ms and L_{Max} differed by 89 ± 16 ms comparing the auditory with the luminant condition; latency measures in the auditory condition differed even stronger from the isoluminant condition ($L_{50\%}$: 101 ± 19 ms; L_{Max} : 124 ± 23 ms). In contrast, the strength of inhibition as measured by its duration and its magnitude did not differ between the visual and auditory conditions. However, the shape was more pointed in the auditory as compared to both the luminant (0.19 ± 0.10) and the isoluminant condition (0.12 ± 0.09). This finding suggests a higher sensitivity of this measure to the total amount of inhibition as compared to the magnitude and duration of inhibition.

Table 4.1: Means ($\pm CI_{95\%ws}$) of measures of inhibition in the irrelevant-onset paradigm, estimated from 100 bootstrap samples (see text for details).

Condition	$L_{50\%}$ [ms]	L_{Max} [ms]	Duration δ [ms]	Magnitude μ [proportion]	Shape σ [proportion]
luminant	102 (± 9)	199 (± 14)	176 (± 21)	0.81 (± 0.11)	0.78 (± 0.08)
isoluminant	153 (± 17)	234 (± 22)	153 (± 25)	0.75 (± 0.09)	0.71 (± 0.07)
auditory	53 (± 9)	110 (± 8)	142 (± 27)	0.71 (± 0.09)	0.59 (± 0.05)

Note. $L_{50\%}$ = Latency to 50% of maximum inhibition; L_{Max} = Latency to maximum inhibition.

4.2.3 Microsaccade amplitude

Strong microsaccadic inhibition was triggered by the irrelevant stimuli used in this experiment. As a next step, we determined how this inhibition affected microsaccades of different amplitudes. Figure 4.7 plots the temporal evolution of mean microsaccade amplitudes (and $CI_{95\%ws}$), locked to the onset of the irrelevant stimulus. Mean amplitudes were determined using a 50 ms moving boxcar window centered at t . A strong decrease in the mean amplitude is evident for the two visual conditions. A similar effect, though weaker, was observed after auditory distractors. In each condition, the decrease in microsaccade amplitude closely followed the time course of

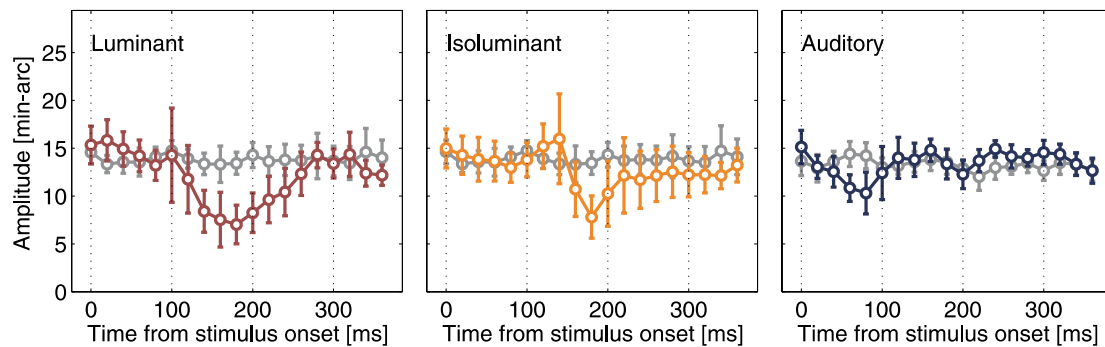


Figure 4.7: Microsaccade amplitude in the visual and auditory conditions is plotted as a function of time. Gray lines represent amplitude evolution in trials with no irrelevant stimulus. Error bars are $CI_{95\%}$.

microsaccadic inhibition. Significant deviations, as measured by non-overlapping confidence intervals of the amplitude evolutions in the stimulus as compared to the no-stimulus conditions, were evident from about 140 ± 25 to 220 ± 25 ms in the luminant, at 180 ± 25 ms in the isoluminant, and from 60 ± 25 to 80 ± 25 ms in the auditory condition.

To examine the process of microsaccadic inhibition in more detail, amplitude distributions were computed for each of the distractor conditions by collapsing amplitude data from all participants at a given time window. Figure 4.8 presents amplitude distributions at a baseline time window (last 50 ms before stimulus onset; dark colored) and in a time window when the lowest mean amplitude was observed (width: 50 ms around minimum; light colored). Gamma distributions were fitted to the data. For all three conditions, this figure nicely depicts how the frequency of microsaccades drops off across all amplitudes. However, very small microsaccades are inhibited to a lesser extent, consistently shifting the mode of the distributions towards smaller amplitudes.

4.2.4 Microsaccades and performance measures

Although this chapter is primarily dedicated to microsaccade-rate signature, we will report some intriguing results concerning the relationship between fixational oculomotor activity and the performance in the task. This excursion is justified since it pursues the origin of the following effect: In the visual condition, participants responded faster on average if an irrelevant stimulus was presented in the course of a trial (displayed in Figure 4.4). It is unlikely that this result was caused by the little temporal information that the stimuli still might have carried. Otherwise it

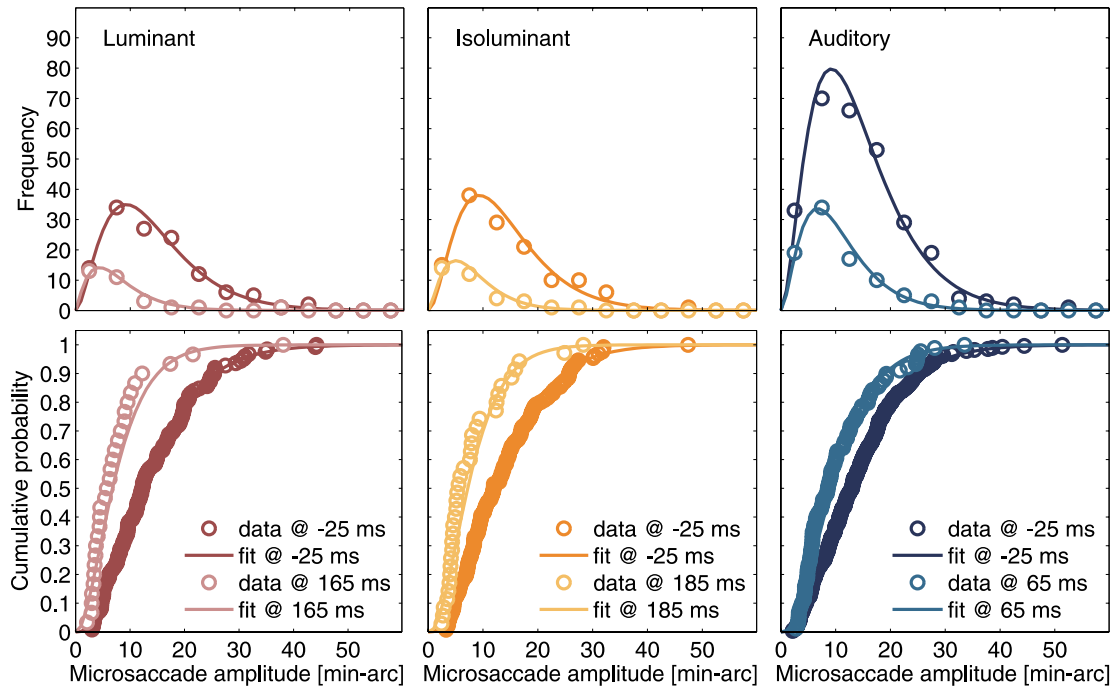


Figure 4.8: Microsaccade-amplitude distributions for luminant (brown), isoluminant (orange), and auditory (blue) conditions in two time windows (each 50 ms wide). Dark colors plot distributions at a baseline time window; light colors show distributions at the time window where the smallest mean amplitude was observed. Gamma distributions were fitted to the data.

should have been observed in the auditory condition as well. Rather, this effect might be due to an activation of the visual system induced by the incoming information. If this were to be the case, response latencies should be a function of the duration between stimulus presentation and the onset of the discrimination target. With increasing time intervals, response latencies would increase. This relationship should not exist in trials where no irrelevant stimulus was presented, taking similar pre-target durations as a reference.

The two upper-left panels of Figure 4.9 display mean response latencies with $CI_{95\%}$ in the tested conditions as a function of the duration of the stimulus-to-target interval (STI). STIs ranged between 500 and 1500 ms and were cut into quartiles for this analysis, resulting in 250 ms wide bins. A clear dissociation is captured by that graph, which, however, is completely opposite to what was hypothesized before: In the visual condition, participants benefitted from the presentation of an irrelevant stimulus only if 700 ms (difference in second STI quartile: 34 ± 16 ms), or more,

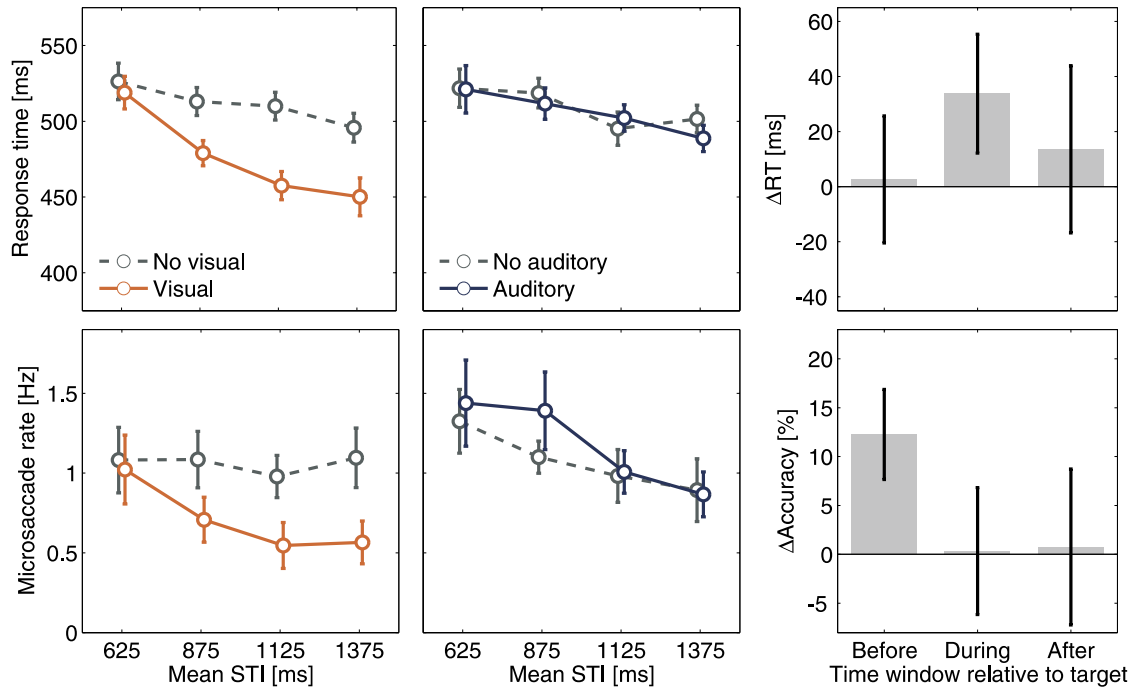


Figure 4.9: Correlation between microsaccade statistics and performance. The four panels on the left side show manual response times and microsaccade rate as a function of the delay between the irrelevant stimulus and the discrimination target (STI; cut into quartiles) for the visual (left panels) and auditory (right panels) conditions. Dashed lines show data from no-stimulus trials, matched for pre-target duration. Error bars are $CI_{95\%ws}$. The two panels on the right show benefits and costs in manual response latencies (ΔRT) and accuracy ($\Delta Accuracy$) after microsaccades observed in three time windows: just before, during, and directly after target presentation (see text for details). Positive values indicate an increase in response time and accuracy, respectively, given a microsaccade was observed in a time window. Error bars are $CI_{95\%}$.

passed by before the discrimination target was presented. In the auditory condition, this kind of dependence was not observed.

Next we sought to determine whether these findings can be related to oculomotor activity. For each STI bin, we computed mean microsaccade rates in a 250 ms time window before target onset. The results are plotted in the two lower-left panels of Figure 4.9. Microsaccade rates quite closely mimicked the relation between response times and STIs. For STIs of 700 ms, or longer, microsaccade rate was lower if a visual distractor was presented (difference in second STI quartile and corresponding $CI_{95\%}$: 0.38 ± 0.26 Hz). If microsaccades were associated with worse performance in the visual discrimination task, we would have a clue why participants performed better in the visual distractor conditions. To examine this possibility, performance was computed as a function of microsaccade presence in three different time windows: before, during,

and after target presentation. The time windows were matched such that for every trial, all three time windows had the width of a given target presentation time. For instance, if the target was presented for 25 ms, the before-target time window covered the last 25 ms before target onset, while the first 25 ms after target offset served as the after-target time window. In that way, we sought to account for the variable target-presentation times, hence, omitting imbalances due to different widths for different time windows. Only a subset of 10 participants (collapsed across conditions) had a minimum of 5 microsaccade observations per time window and were included in this analysis. The upper-right panel of Figure 4.9 displays the costs imposed on response latencies when a microsaccade was observed in one of the three time windows. On average, these participants' response times were increased by 34 ms if a microsaccade was observed during target presentation; $t(9) = 2.99, p = 0.015$. In contrast, no significant costs were associated with microsaccades occurring before or after target presentation; $ts < 1$. Rather, as the lower-right panel of Figure 4.9 shows, participants responded more accurately if a microsaccade was observed just before target onset; $t(9) = 5.05, p < 0.001$.

4.3 Discussion

In the present experiment, we examined the effect of irrelevant stimuli on microsaccade statistics. Both irrelevant auditory and irrelevant visual stimuli elicited strong drops in microsaccade rate, i.e., microsaccadic-inhibition. The effect had a similar magnitude and duration, but a shorter latency for luminant as compared to isoluminant flashes. Auditory stimuli triggered extremely fast inhibition of microsaccades, again of similar magnitude but the rate evolution had a different shape. In addition, microsaccade amplitude strongly decreased in the course of microsaccadic inhibition. These findings parallel and extend previous work on oculomotor control and are in agreement with the idea that microsaccades are a behavioral substrate of the activity at fixation-related parts of a saccadic motor map. Here, we will discuss the implications of the present study for understanding the mechanisms of microsaccade and saccade generation. In addition, we will offer a tentative explanation for the interesting side finding that irrelevant visual stimuli enhanced performance in a visual discrimination task.

4.3.1 On the processes underlying microsaccadic inhibition

Microsaccadic inhibition is thought to be related to a fast oculomotor circuitry involving the SC (Engbert, 2006b; Laubrock et al., 2005; Rolfs et al., 2005). Accordingly, we have argued that the temporal evolution of microsaccadic inhibition should be sensitive to the properties of the triggering stimulus. While luminance-contrast stimuli may exert influence on the SC motor-map via direct retinotectal connections, this pathway is blind to pure color-contrast (Schiller & Malpeli, 1977), i.e., stimuli that are isoluminant to the background. This detour of a stimulus-related neural signal was hypothesized to express itself in a delayed microsaccadic-inhibition effect. Our data confirm this prediction. As compared to the luminant stimuli, isoluminant input delayed microsaccadic inhibition by 39 to 48 ms on average (depending on the measure).

In addition, we found much shorter latencies of microsaccadic inhibition after auditory as compared to visual stimuli. This is in agreement with physiological data showing that signals may reach the SC motor map earlier, when they originate from auditory rather than visual stimulation (Jay & Sparks, 1984, 1987; Stein & Meredith, 1993). However, the intensity of the auditory stimuli was not systematically varied with respect to the flashes in the present experiment and no attempt was made to obtain subjectively matched stimulus intensities for the two modalities. Instead, the used stimuli were clearly above threshold and held constant across the experiment with respect to their physical properties. Therefore, a direct comparison of microsaccadic inhibition evoked by auditory and visual irrelevant stimuli is not ultimately informative. The argument we would like to bring forward here, however, is even stronger: Auditory stimuli evoked microsaccadic inhibition after extremely short latencies, which never could have been produced by visual input. The reasoning for this claim is that the lower physiological limit for an impact of visual input on oculomotor behavior is in the range of 60 to 70 ms (cf., Reingold & Stampe, 2002). This estimation is derived from a series of single-cell-recording studies in monkeys: First, for bright flashed stimuli, Rizzolatti, Buchtel, Camarda, and Scandolara (1980) reported visual latencies in the superficial layers of the SC of 35 to 47 ms. Second, transmission delays to the intermediate layers of the SC are in the range of 5 to 10 ms (Lee, Helms, Augustine, & Hall, 1997). Finally, the time after which suprathreshold stimulation of the intermediate SC evokes a saccadic response is at least 20 ms (Munoz & Wurtz, 1993b). While the final motor delay of 20 ms should be independent of stimulus modality, input delays are not. In contrast to visual input, sound-induced signals often reach the intermediate SC within 30 ms or less (Jay & Sparks, 1984, 1987). Thus, a latency of

50 ms (as observed in the present study) for microsaccadic inhibition is physiologically plausible in response to auditory stimuli, however, probably impossible after visual input. We conclude that the SC is a likely candidate for the implementation of microsaccadic inhibition.

We proposed a model of microsaccadic inhibition in which the drop off in microsaccade rate is explained in terms of a decreased activity in fixation-related sites of the saccade motor map (see also Engbert, 2006b). The decrease of microsaccade amplitudes during microsaccadic inhibition is in line with this proposal. Activity at the fixation-related site of an oculomotor map coding saccades of different amplitudes decreases as the result of stimulus-related input to the map. With decreasing activity, only the very central parts of the saccade map remain activated above threshold. As a consequence, fewer microsaccades and smaller mean amplitudes are produced.

Two things deserve mention in this connection. First, a decrease in mean amplitude is not necessarily predicted if the microsaccade-rate modulation is triggered by a spatial stimulus, e.g., a cue. In this case, mean microsaccade direction is biased (Engbert & Kliegl, 2003b; Galfano et al., 2004; Hafed & Clark, 2002; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005), which might indicate a shift of the activity distribution in the saccadic motor map towards larger amplitudes. Second, in general, a concurrent decrease in microsaccade rate and amplitude might also be explained in terms of enhanced fixation of gaze and a corresponding decrease of saccade-related activity (cf., Rolfs et al., 2005). In this case, microsaccade amplitude would decrease because activity collapses around the fixation center. On the basis of the data presented here, we cannot distinguish between the alternative mechanisms and it is possible that both are at work in different situations (e.g., after foveal vs. peripheral stimulation). Importantly, however, both these hypotheses rely on a representation of microsaccades in the rostral pole of the SC.

Can the model put forward here account for the differences between microsaccade statistics observed in the present set of conditions? A major difference in the dynamics of microsaccade rate across conditions was the latency of inhibition. We propose that, in general, the same mechanisms underly microsaccadic inhibition in all tested conditions (luminant, isoluminant, and auditory). However, as input arrives at different point in time at the saccadic motor-map, microsaccadic inhibition varies in latency.

Another difference in the dynamics of inhibition was that the modulation of microsaccade amplitude was less pronounced and had a shorter duration in the auditory condition than in the visual conditions. This effect may be associated with the higher baseline rate of microsaccades in

this condition. The latter finding suggests that activity at the rostral pole of the SC was higher, though not necessarily more broadly distributed. Since the magnitude of inhibition was comparable across conditions, a decrease of activity in the auditory condition would, thus, not reduce mean microsaccade amplitude as strongly. This may explain why amplitude modulations differed across conditions. It fits the picture that the shape forming the dip of microsaccade rate differed between visual and auditory conditions. As captured by the shape measure, the time course of microsaccadic inhibition followed a rather pointed trajectory in the auditory condition, but was quite bellied in response to visual stimulation. Pointed curves may indicate that saccadic inhibition did not reach its full extent. Rather, in the auditory condition examined here, microsaccade rate suddenly increases again after about 100 ms. A more bellied curve in the visual conditions may be explained by combining our finding of a lower baseline rate of microsaccades in this condition with the assumption that there is a minimum level of microsaccade rate. Once inhibition drives the rate to this minimum, it remains on this level until inhibition is released. In addition to the data published here, the microsaccade-rate signatures reported by Rolfs et al. (2005, Figures 2 and 3) for auditory and visual spatial cues appear to be in line with these proposals. Finally, the stimuli employed in the present study differed not only with respect to their modality, but also in their spatial alignment. In contrast to the clear spatial location of the colored flashes surrounding the point of fixation, auditory stimuli were played to both ears via headphones, thus, not containing any spatial information. As a consequence, auditory stimuli might have induced an enhanced, but unspecifically distributed mean-field activity in the collicular motor map, less efficiently driving competitive processing in the map. Put differently, the near-fovea presentation of irrelevant visual distractors may have favored a strong modulation of microsaccade rate and amplitude.

4.3.2 Relation to saccadic inhibition

As already noted in the literature (Engbert, 2006b; Engbert & Kliegl, 2003a, 2003b; Laubrock et al., 2005; Rolfs et al., 2005), the appearance of the microsaccade-rate signature is evocative of a phenomenon commonly referred to as saccadic inhibition. Saccadic inhibition describes a strong effect of irrelevant transients on the frequency of saccades that was observed in a broad range of eye-movement tasks including simple saccade paradigms (Reingold & Stampe, 2002), reading (Reingold & Stampe, 1999, 2000, 2003, 2004; Stampe & Reingold, 2002), visual search (Reingold & Stampe, 1999, 2000, 2004; Stampe & Reingold, 2002), and picture viewing (Pannasch et al.,

2001). In a typical saccadic-inhibition paradigm, short flashes are presented in the course of an oculomotor task. The main effect that arises from this manipulation is a strong, knee-jerk decrease in the frequency of (large) saccades forming a dip, time-locked to the flash. This saccadic inhibition occurs as early as 60-70 ms after flash onset. Therefore, it was thought to be an oculomotor reflex related to inhibitory processes in low-level oculomotor structures, i.e., the SC.

Thus, the phenomena of saccadic and microsaccadic inhibition as well as their proposed locus of implementation are very similar, suggesting that both effects are probably essentially the same, but originate in different fields of research. However, one major difference between the studies on saccadic inhibition and those investigations reporting microsaccade-rate evolutions, is that in the latter case the examined stimuli were always task-relevant, i.e., they carried information relevant to the task at hand. In the present study, much effort was spent to engage performers in the visual discrimination task, thus, to have them ignoring the irrelevant stimuli. First, the task itself was very demanding; low contrast target stimuli were presented for extremely short times. Second, irrelevant stimuli were not indicative of the current choice of the discrimination target. Third, they were little predictive for the time of target occurrence. Given an irrelevant stimulus was presented, the discrimination target could appear at any time in a range of 500 to 1500 ms. Finally, continual feedback was given to the participant concerning the performance in the visual discrimination task. Despite all this, strong microsaccadic inhibition was observed in response to the irrelevant stimulus. Thus, the present results bring microsaccadic and saccadic inhibition closer together.

Moreover, the present paper demonstrated differential effects of stimulus properties on the time course of microsaccadic inhibition. Similar findings can be found in the literature on saccadic inhibition. As has been documented by Stampe and Reingold (2002), saccadic inhibition is sensitive to luminance and spatial frequency content of the transient event. In detail, strong changes in the luminance of the visual display result in short-latency inhibition. Transient images of low spatial frequency elicit faster and stronger saccadic inhibition than changes in the range of high spatial frequency, e.g., the sharpening of an image. In the present study, the same pattern of results was evident for the impact of luminance on the inhibition of microsaccades.

But what about auditory input? Reingold and Stampe (2004) argued that saccadic inhibition is an optomotor reflex of the oculomotor system, which is sensitive to visual input only. In their paradigm, auditory input (2000 Hz beeps presented for 33 ms) did not affect the rate of saccades

during reading. Unfortunately, the authors did not report the volume of the auditory stimuli, however, frequency and duration of the beeps used suggest that stimulus intensity might not have been sufficient to trigger saccadic inhibition. Using (possibly more salient) auditory stimuli, Pannasch et al. (2001) also found saccadic inhibition for this input modality. During fixations in a free-picture-viewing task, participants were presented with either of two types of distractors: a small black spot in the visual periphery or a 1000 Hz tone played through loudspeakers. As compared to the effect elicited by visual distractors, saccadic inhibition was weaker in the auditory condition. And, more crucially, it had a shorter latency. Specifically, transient visual events were associated with a drop in the probability of saccades at a latency of 100 ms after distractor onset, whereas auditory distractors reduced the rate of saccades 80 ms after stimulus onset. These results are consistent with the time courses of microsaccadic inhibition reported here. Pannasch et al. (2001) argued that any new event entering a sensory channel will produce an orienting reflex which expresses itself in a decreased saccade rate. Thus, saccadic inhibition is not an optomotor reflex but instead arises from a more general process.

The extremely short response time of the oculomotor system in saccadic inhibition in combination with its sensitivity to low-level properties of the stimulus pushed investigators to propose that the SC is the primary candidate for mediating the effect. It was subject to discussion, however, how saccadic inhibition is implemented within that structure (see Reingold & Stampe, 2002). First, it might result from increased activity of saccade-related sites in the SC motor map, thereby decreasing the activity at other locations. Second, the stimulus-induced activity might enhance fixation-related activity, which, in turn, decreases the likelihood of saccadic activity to reach a necessary threshold. Here, we proposed that microsaccades are a behavioral substrate of activity in fixation-related sites in saccadic motor maps, i.e., the rostral pole of the SC. The study of microsaccade amplitudes in tasks exploring saccadic inhibition might help clarifying which mechanisms constitute this effect.

4.3.3 Enhanced performance after irrelevant visual stimuli

Besides the findings concerning microsaccadic inhibition, the present experiment yielded potentially interesting results in participants' performance in the visual discrimination task and its relation to microsaccade statistics. If an irrelevant visual stimulus was presented while performers waited for the target to come, microsaccade rate decreased as compared to trials where no

stimulus occurred or when a sound was presented. At the same time, response times improved as a function of the aging STI; the amount of benefit mimicked the rate of microsaccades just before target presentation. Thus, the decrease in response times was strongest in the visual-stimulus conditions. But why should the inhibition of microsaccades come along with a benefit in response times? The discrimination targets were presented for about 30 ms on average. Thus the occurrence of a microsaccade and the corresponding saccadic suppression (Beeler, 1967; Zuber et al., 1964; Zuber & Stark, 1966) might have hindered the perception of the stimulus, thus, resulting in worse performance. Indeed, microsaccade occurrence during target presentation significantly affected response times (see Figure 4.9). In contrast, microsaccades occurring just before target presentation could have enhanced perception by replacing the retinal image (see Section 2.1). Here, such benefit was evident in a higher accuracy for these cases.

There is no reason to believe that this inhibition of microsaccades in a late time window was consciously produced. Why should it not be observed in the auditory condition as well, then? At the moment, however, we can only speculate on the origin of this effect. Visual stimuli in the present study were located in the direct surrounding of the discrimination target. Possibly, this automatically drew attention to the location of the discrimination target. Focused attention by itself can account for an increase in performance, but not for the dependence of this increase on the interval between the two stimuli. Rather, the time course of exogenous attention is fast and would predict a beneficial effect for short inter-stimulus intervals. The opposite was observed here. We propose that microsaccade inhibition in a late time window was a result of focused attention in an earlier time window. Thus, the response-time benefit in the visual discrimination task would be a result of attention, but mediated by the rate of microsaccades potentially imposing costs on performance in the task.

Admittedly, this account is speculative and based on an analysis of a subset of participants only. Thus, the origin of late microsaccadic inhibition as observed in the present experiment may be subject to dispute. Nevertheless, this finding points to one potential purpose of (micro)saccadic inhibition for visual perception, the omission of saccadic suppression when new input arrives. Further evolutionary mechanisms might have given rise to the phenomenon of saccadic inhibition, e.g., an enhancement in the ability to detect motion by fixing the eye in its place, the cancellation of all movement programs to be ready to grasp newly arriving stimuli, or even the preparation of a saccadic movement to the new stimulus. It has been established here and in a series of earlier

studies that (micro)saccadic inhibition is a very stable and strong effect found in response to nearly everything, including perceptual input and even amodal shifts of attention (e.g., task switching; Pannasch et al., 2001). One goal for future research will be to determine what ecological necessities are inherent to this phenomenon.

Chapter 5

Approaching the interactions of microsaccades and saccades

Investigating the effect of microsaccades on the characteristics of subsequent saccades might offer an important window into the processes underlying microsaccade generation. In addition, it may help explain some of the variability that is observed in saccade latencies during simple oculomotor tasks. Here, we will have a first look at the potential interactions of microsaccades and saccades.

The effect of microsaccades on the latency of subsequent saccades may be twofold. First, as we have seen in the chapter on the functions of FEyeM, microsaccades were associated with an enhancement of visual perception. A concert of studies using psychophysical experiments, neurophysiological approaches, and modeling techniques corroborated this important purpose of microsaccades (for reviews see Section 2.1 and Martinez-Conde et al., 2004). Microsaccades modulate spiking activity in the visual cortex (Bair & O'Keefe, 1998; Leopold & Logothetis, 1998; Martinez-Conde et al., 2000, 2002; Snodderly et al., 2001) and may increase stimulus visibility (Deubel & Elsner, 1986; Elsner & Deubel, 1986; Gerrits & Vendrik, 1974; Martinez-Conde et al., 2006). Such perceptual enhancement should facilitate visual processing of a saccade target and, consequently, shorten the latency of a saccadic response towards it.

Second and contrary to that prediction, the physiological mechanisms involved in the generation of microsaccades and saccades, respectively, might interfere. It is the nature of this interference that is being focussed in the thesis at hand. Potential interactions between microsaccades and

subsequent saccades were described in Chapter 3. Therefore, we will only shortly recapitulate the potential impact of microsaccades on saccade generation here. Microsaccades' binocularity (Ditchburn & Ginsborg, 1953; Krauskopf et al., 1960; Lord, 1951), involuntary occurrence (Ditchburn & Ginsborg, 1953; Ratliff & Riggs, 1950), and shared kinematic characteristics with large-scale saccades (Zuber et al., 1965) hint at common subcortical mechanisms for the generation of both types of movements. The similarity between saccadic inhibition and the inhibition of microsaccades in response to task-irrelevant stimuli is highly compatible with this view, as has been pointed out in Chapter 4. We proposed that the rostral pole of the superior colliculus (SC), a brainstem structure critically involved in the control of saccades and fixations (see Section 3.1 and Munoz et al., 2000; Scudder et al., 2002; Sparks, 2002, for reviews), is a likely neural correlate of microsaccade generation. The rationale is that saccade amplitudes are coded along the SC rostral-caudal dimension; small amplitudes like those associated with microsaccades being related to activity in the rostral pole (D. A. Robinson, 1972). Neurons in the rostral pole, however, are associated with active fixation (Munoz & Guitton, 1991; Munoz & Wurtz, 1993a, 1993b). Consequently, SC activation that causes a microsaccade generates also a longer latency for a subsequent saccade because neurons coding the next saccade target must overcome the activity of these fixation-related neurons in the SC before a saccade may be executed (Munoz & Istvan, 1998; Munoz & Wurtz, 1993b, 1995b). Thus, if microsaccades result from activity in fixation-related neurons in the rostral pole of the SC, they should delay the latency of subsequent saccades.

To our knowledge, neither the proposal that microsaccades originate from fixation-related neurons nor the predictions for the relationship between microsaccades and subsequent saccade latencies have been tested. Here, we show in a single delayed-saccade task that microsaccades are indeed associated with opposite effects in saccadic response latencies. The direction of this effect depends on two factors: (1) when a microsaccade occurs, during the perception of the target or during preparation of a saccade, and (2) whether a visually or memory-guided saccade is required.

In addition, the present study relates to a paper by Supèr, van der Togt, Spekreijse, and Lamme (2004). These authors showed that neural activity in V1 increased significantly from 100 ms prior to visually and memory-guided saccades. Presaccadic visual activity was strongest at saccade target locations. Moreover, Supèr et al. (2004) reported a strong correlation between the strength of presaccadic activity in V1 and latencies of saccades to memorized targets. Based on earlier research from our group (Engbert & Kliegl, 2003b; Laubrock et al., 2005; Rolfs et al., 2004, 2005),

we were interested in how microsaccade statistics relate to Supèr et al.'s results and modeled the delayed-saccade task closely on theirs to facilitate comparison. Some of the results reported here have already been published elsewhere (Rolfs et al., 2006).

5.1 Methods

5.1.1 Participants

Thirty-one students of the University of Potsdam were paid 7€ or received study credit for their participation. They were 19 to 40 years old (24.3 years on average), had normal or corrected-to-normal vision, and were in good health.

5.1.2 Experimental setup and eye-movement recording

Participants were seated in a silent and darkened room with the head positioned on a chin rest, 50 cm in front of a computer screen. Stimuli were presented on a 19-inch EYE-Q 650 CRT (1024 by 768 resolution or 40° by 30° of visual angle; refresh rate 100 Hz). The experiment was controlled by an Apple Power Macintosh G4 computer. Eye-position data were recorded and available on-line using an EyeLink-II system (SR Research, Osgoode, ON, Canada) with a sampling rate of 500 Hz and a noise-limited spatial resolution better than 0.01°. The experimental software controlling stimulus display and response collection was implemented in Matlab (MathWorks, Natick, Massachusetts, USA), using the Psychophysics (Brainard, 1997; Pelli, 1997) and EyeLink (Cornelissen et al., 2002) toolboxes.

5.1.3 Procedure

Participants performed 306 test trials of a delayed response task similar to that used by Supèr et al. (2004). Before the first and after every 50 trials the eye tracker was calibrated (standard 9-point grid) and calibration was validated. To start a trial, participants had to fixate a red spot at the center of a random-noise screen (each pixel was set to black or white). Correct fixation was checked, and the stimulus screen appeared if gaze position was detected in the fixation region. Otherwise, a drift correction was carried out and the trial was started over. If the eyes were still not detected within the critical area, the calibration was repeated.

Figure 5.1 depicts the sequences of visual stimulations used in the present experiment. Participants fixated a point at the center of the computer screen. After 1500 ms of fixation, a square target appeared at one of three possible positions in the periphery (top: 90° , bottom-left: 210° , or bottom-right: 330°). Participants maintained fixation for an additional 1000 ms until a go signal (fixation point offset) commanded a saccadic response to the target. Response saccades (eye position shift to either of the three target square regions) were detected on-line. If either a response saccade was detected or a response interval of 500 ms was exceeded, the next trial was started after an inter-trial interval of 500 ms. The sequence of visual stimulation was varied according to three different experimental conditions: (1) The target remained on the screen during the whole fixation period (visual-static condition), (2) it was replaced by a different figure of the same size 280 ms after target onset (visual-change condition), or (3) it disappeared after 280 ms of presentation (memory condition). Trials were presented in randomized order, with 102 trials per condition. In addition to stimulus condition (visual-static, visual-change, memory), the factorial design included target position (top, bottom-right, or bottom-left) and target orientation (45° or 135°).

If gaze position left a fixation square (2° side length, centered on the fixation spot) during the 2500 ms fixation period, the trial was aborted. Aborted trials were repeated in random order after the 306 regular trials.

5.1.4 Stimuli

The background of the stimulus screen consisted of a texture of randomly distributed white-on-black line segments of a single orientation. In each trial, a square target (side length 3°) was presented at one of three possible locations (top, bottom-left, or bottom-right, with 4.4° eccentricity of the squares center from the central red fixation spot of 0.2° diameter). Target squares set on abruptly and consisted of a random texture of line segments with an orientation orthogonal to the background pattern (see 5.1b for example screens). In two of three conditions, this texture was replaced after 280 ms by either a background-homogeneous texture (memory trials) or another figure texture (visual-change trials). Line segments were 16×1 pixels ($0.62^\circ \times 0.039^\circ$) and had an orientation of 135° or 45° . Both orientations were used for both figure and background, resulting in complementary stimulus pairs. On average, 40% of the screen were covered by lines.

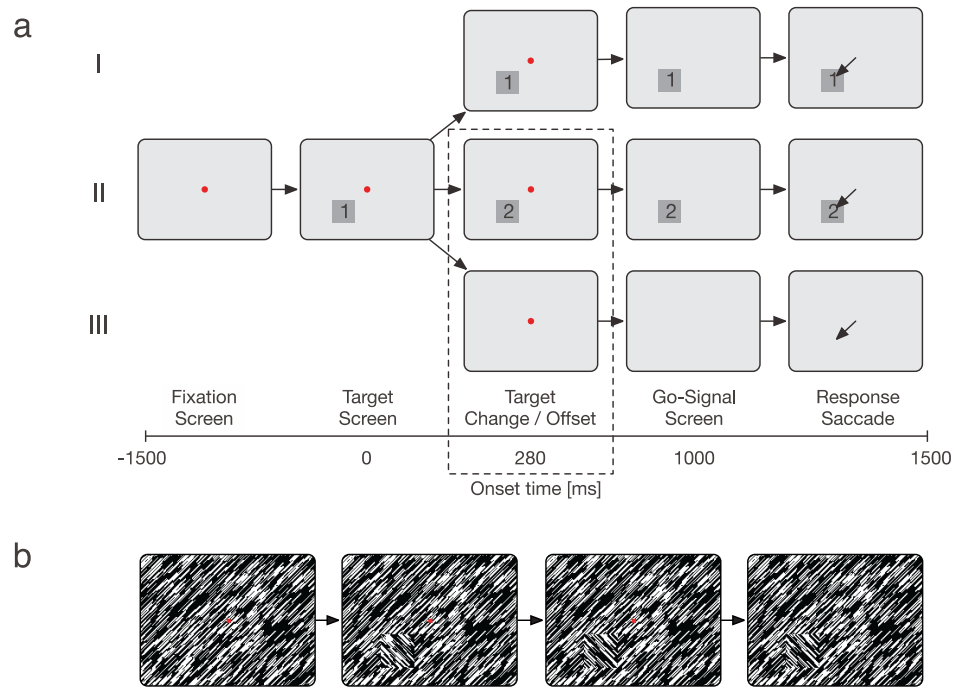


Figure 5.1: Illustration of the experimental procedure in the delayed-saccade task. (a) Sequences of visual stimulation in the three conditions (I = visual static; II = visual change; III = memory). (b) Example of a display sequence in the visual change condition. Note, that the target square changes its appearance from the second to the third screen, while the background remains the same.

5.1.5 Data preparation

For data analysis, a post-hoc saccade detection was performed using a new version (Engbert, 2006b) of the algorithm by Engbert and Kliegl (2003b). Velocities were computed from subsequent samples in the series of eye positions in the response time window 500 ms on from the go signal. Saccades were detected in 2D velocity space using thresholds for peak velocity and minimum duration. We used a relative threshold of 6 SDs of the velocity and a minimal duration of 6 ms (or three data samples). The first saccade that shifted gaze across one of the three target areas was taken as a response saccade. Saccadic reaction time (SRT) was defined as the latency between go signal and saccade onset.

Subsequently, we used the same algorithm to detect microsaccades (amplitude $< 1^\circ$) in the interval from fixation onset to the response saccade. We considered only binocular microsaccades, that is, microsaccades detected in both eyes with temporal overlap.

Trials including saccades larger than 1° prior to the response saccade were discarded, as were trials with incorrect responses and SRTs shorter than 70 ms. Some trials had to be excluded due to data loss during eye-movement recording. Thirty-one participants contributed 238 to 304 trials to the final data analyses, resulting in a total of 8603 trials (out of 9486 or 90.7%; 2853 visual-static, 2887 visual change, and 2863 memory trials) in which 35953 microsaccades were detected.

5.1.6 Data analysis

Wherever confidence intervals are provided, these were computed using a simple bootstrapping technique (Efron & Tibshirani, 1993). From an original sample of N values, 1000 bootstrap samples were generated, each by selecting (with replacement) N values of the original sample. The 1.96-fold of the standard deviation of the means of these 1000 bootstrap samples was computed to generate 95% confidence intervals ($CI_{95\%}$) of the mean of the original sample. In graphical illustrations, confidence intervals will often allow the reader to compare different conditions by so-called “rules of eye” (Cumming & Finch, 2005). For within-subject comparisons, uninformative between-subject variance was therefore removed (Cousineau, 2005); these confidence intervals will be labeled $CI_{95\%ws}$.

5.2 Results

5.2.1 Saccadic reaction time

Mean SRTs ($\pm CI_{95\%}$) were 235 (± 10) ms for visual-static, 237 (± 12) ms for visual-change, and 238 (± 12) ms for memory trials. There was no evidence for SRT differences between conditions, as supported by a repeated-measures ANOVA; $F < 1$.

5.2.2 Microsaccade rate

Since microsaccade rate is of fundamental importance for the study of microsaccadic influences on SRTs, we first examined this evolution. Figure 5.2 shows microsaccade rates across the time course of trials aligned to target onset. Rate evolutions represent averages over participants. Individual rates were computed as described in Section 4.1.6. When the target remained unchanged on the screen over the entire trial (visual-static), microsaccade rate declined from a relatively stable

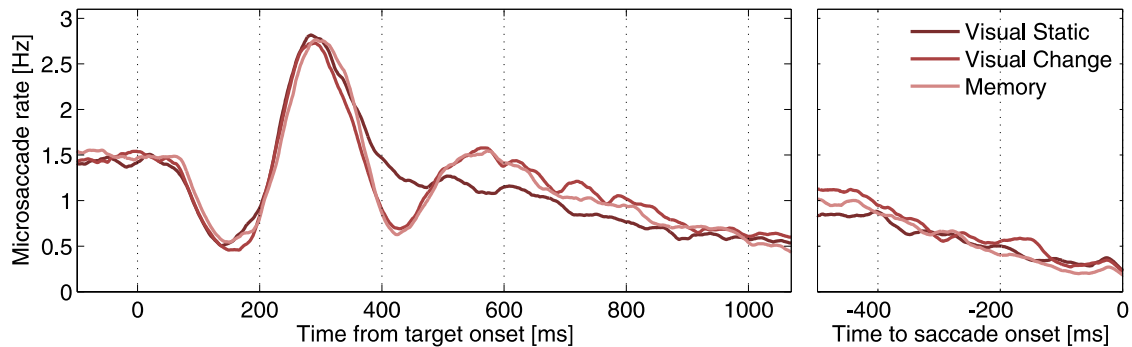


Figure 5.2: Temporal microsaccade-rate evolution for the three experimental conditions aligned on target onset (left panel) and saccade onset (right panel). Rates were computed using an asymmetric filter and averaged across participants.

baseline level of 1.5 s^{-1} towards a minimum of 0.5 s^{-1} (inhibition) at about 140 ms. Subsequently, an enhancement of microsaccade rate led to a maximum (2.8 s^{-1}) about 290 ms after target onset. Finally, rate resettled at a level of 1.2 s^{-1} (at 430 ms), before slightly decreasing, reaching a level of 0.5 s^{-1} at the time of the go signal (1000 ms). This result replicates the signature of previous research within a new experimental paradigm. During the first 280 ms visual stimulation was identical and, hence, the rate pattern was not different between visual and memory conditions. However, when a change took place, a second inhibition phase superseded the enhancement phase, leading to a second minimum (0.6 s^{-1}) at about 420 ms. After an additional small enhancement epoch forming a peak of 1.5 s^{-1} at 570 ms, rates join the slight decrease that was found in the visual-static condition. The second inhibition-enhancement epoch represents a new result in agreement with expectations. Note, however, that there was no difference between memory and visual-change conditions in the time course of microsaccade rate, which was thus unaffected by a figure vs. ground interpretation of the change.

To study the decrease in microsaccade rate prior to the response saccade, we computed microsaccade onsets relative to saccade onsets. As can be seen in Figure 5.2, rate drops to a value around 0.2 s^{-1} just before the saccade.

5.2.3 Microsaccade amplitude

Microsaccade rate strongly decreased prior to the response saccade. To explore whether this effect was accompanied by a reduction in mean microsaccade amplitude, we defined three time

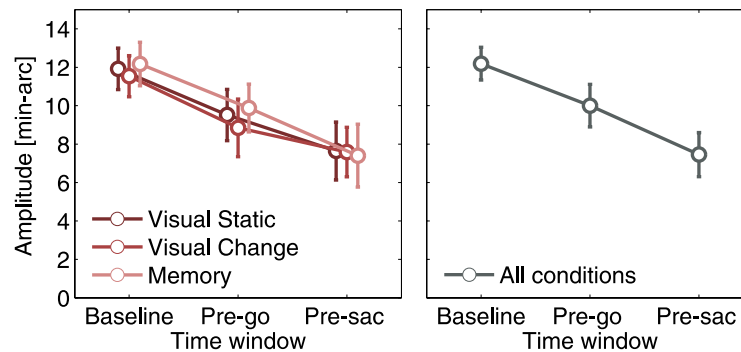


Figure 5.3: Mean microsaccade amplitude in three time windows: baseline (last 100 ms before target onset), go signal (100 ms centered at fixation point offset), and pre-sac (last 100 ms before the response saccade) is plotted for each condition separately (left panel) and collapsed across all conditions (right panel), respectively. Error bars are $CI_{95\%ws}$.

windows, a baseline time window (-100 to 0 ms, before target onset), a pre-go time window (900 to 1000 ms after target onset), and a pre-sac time window (last 100 ms before the saccade). Figure 5.3 displays means and $CI_{95\%ws}$ of microsaccade amplitudes. The left panel plots amplitude data for each condition separately. This plot suggests that mean microsaccade amplitude decreased significantly from about 12 min-arc before target onset to 10 min-arc around the time of the go signal. Microsaccade occurring just before the response saccade had the lowest amplitude (about 8 min-arc). This pattern of results was corroborated by a repeated-measures ANOVA with time window as an independent variable, for which (for the sake of power) amplitude data was collapsed across conditions as shown in the right panel of Figure 5.3; $F(2, 58) = 13.75, p < 0.001$.

5.2.4 Microsaccades and saccadic response latencies

To examine the relation between microsaccades and SRTs, we focus on the difference in SRTs when microsaccades were either present or absent in one of three time windows. First, the baseline time window (-100 to 0 ms) again served as a reference. Second, a target time window was chosen to test whether microsaccades during target presentation decreased saccade latencies. By setting this time window to 150 to 250 ms, we ensured a 30 ms interval between detected microsaccades and target removal in memory trials, allowing for good visual perception of the target after the microsaccade. Third, the pre-go time window was used again (900 to 1000 ms). Mean SRTs are presented in Figure 5.4. For each time window, we conducted a 3x2 repeated-measures ANOVA, including the factors condition and microsaccade presence. In the baseline time window, no significant

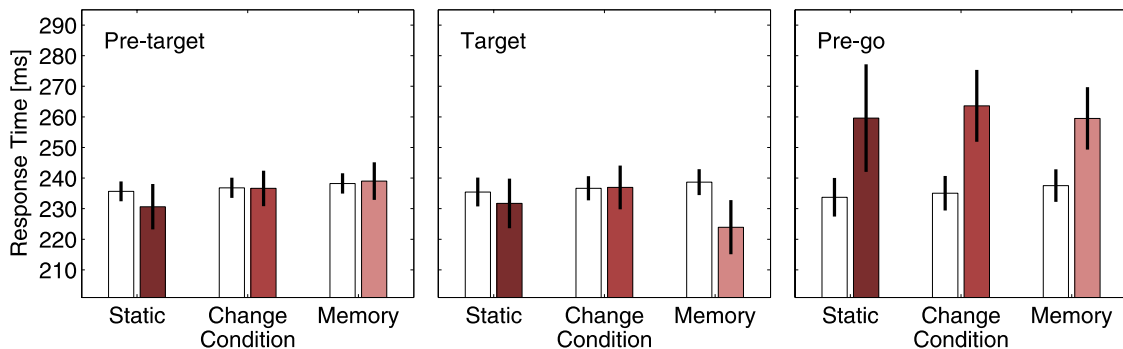


Figure 5.4: Response times in visual-static, visual-change, and memory trials conditional on microsaccade occurrence (white bars no microsaccade, colored bars microsaccade present) plotted for three different time windows: baseline (-100 to 0 ms), target (150 to 250 ms), and pre-go (900 to 1000 ms). Error bars are $CI_{S95\%ws}$.

differences were found; all F s < 1.2. In the target time window, again no significant main effects were found (all F s < 2.2, p s > 0.15) and the interaction was not reliable; $F(2, 56) = 2.8, p = 0.07$. However, planned orthogonal contrasts revealed an interaction of microsaccade presence with the contrast comparing memory with the average of the visual conditions ($F[1, 28] = 4.61, p = 0.041$), whereas no interaction was obtained for the contrast comparing SRT in the two visual conditions ($F[1, 28] = 0.55, p = 0.46$). For the memory condition, a post-hoc t -test for paired samples confirmed that SRTs were shorter if a microsaccade was detected in the target time window (238 vs. 225 ms; $t[28] = 2.5, p = 0.019$). In the pre-go time window, there was a main effect of microsaccade presence ($F[1, 25] = 20.94, p < 0.001$), i.e., if microsaccades occurred, SRTs were longer than when no microsaccade was observed (262 vs. 240 ms). In this time window, there was no interaction of microsaccade presence with condition, $F(2, 50) = 0.03, p = 0.97$.

Next, we determined the extension of these effects by computing microsaccade-induced modulations in SRTs across the whole temporal range of target presentation. For each 100 ms time window, we first computed individual mean SRTs given that either a microsaccade occurred or no microsaccade occurred, and subtracted the latter value from the first. Mean benefits (negative values) and costs (positive values) in saccade latencies (Δ SRT) embedded in $CI_{S95\%}$ are displayed in Figure 5.5. Based on this analysis, a significant speed-up effect in the memory condition was found continuously in the time windows from 130–230 ms to 170–270 ms. The pre-saccadic slowing effect extended back to the time windows 650–750 ms (visual-static), 530–630 ms (visual-change), and 510–610 ms (memory), respectively, after target presentation. Though on average

microsaccades always induced a cost in response times when they occurred in the time windows between the most early and the latest reliable slowing effects, the effect did not always reach a significant level. However, the upmost panel of Figure 5.5 shows that when power is enhanced by collapsing trials from all conditions, significant costs were observed continuously back to a time window of 490–590 ms. This means that SRTs were increased more than 500 ms after the generation of a microsaccade.

Using repeated-measures multiple regression analyses (rmMRA) as described by Lorch and Myers (1990, method 3), we aimed to predict SRT by the onset times and the log-transformed amplitudes of microsaccades preceding the go signal. The rmMRA has the advantage that predictors enter the analysis as continuous values such that the whole variability in the raw data is used. However, dispensing with data aggregation results in a higher level of noise and, thus, the SRT variance explained by our predictors will be necessarily very small. All microsaccades observed during the last 500 ms before the go signal in any of the conditions entered the analyses. The results of the rmMRA are shown in Table 5.1. The table contains the unstandardized regression coefficients for the two predictors. These coefficients are the means from the rmMRAs estimated separately for each individual (Lorch & Myers, 1990, method 3, individual regression equations). An analysis of medians yielded the same pattern of results. For each regression coefficient, Table 5.1 also provides the associated standard errors and statistics for testing it against zero as well as the decrease in R^2 for its removal from the complete model (i.e., the unique variance explained by the predictor) along with test statistics for the R^2 decrement.

The effects of microsaccade onset and amplitude are visualized in Figure 5.6. For this figure, predictors were binned to quantiles of the overall distribution of microsaccade onset and amplitude, respectively, ensuring a similar number of data points for each aggregation. Microsaccade onset yielded a strong effect on SRT; later microsaccades were associated with longer SRTs ($r = 0.123$). In addition, microsaccade amplitude reliably affected SRT; larger microsaccades imposed higher costs on performance ($r = 0.147$). In a second run, we included the additional multiplicative interaction term of onset and amplitude in the regression equation. As shown in Table 5.1, the interaction term is a reliable predictor for SRTs. In this model, however, microsaccade onset cannot account for any additional variance. Thus, as Figure 5.6 (right panel) indicates, onset time does not play a role for small microsaccades, but is important for large microsaccades.

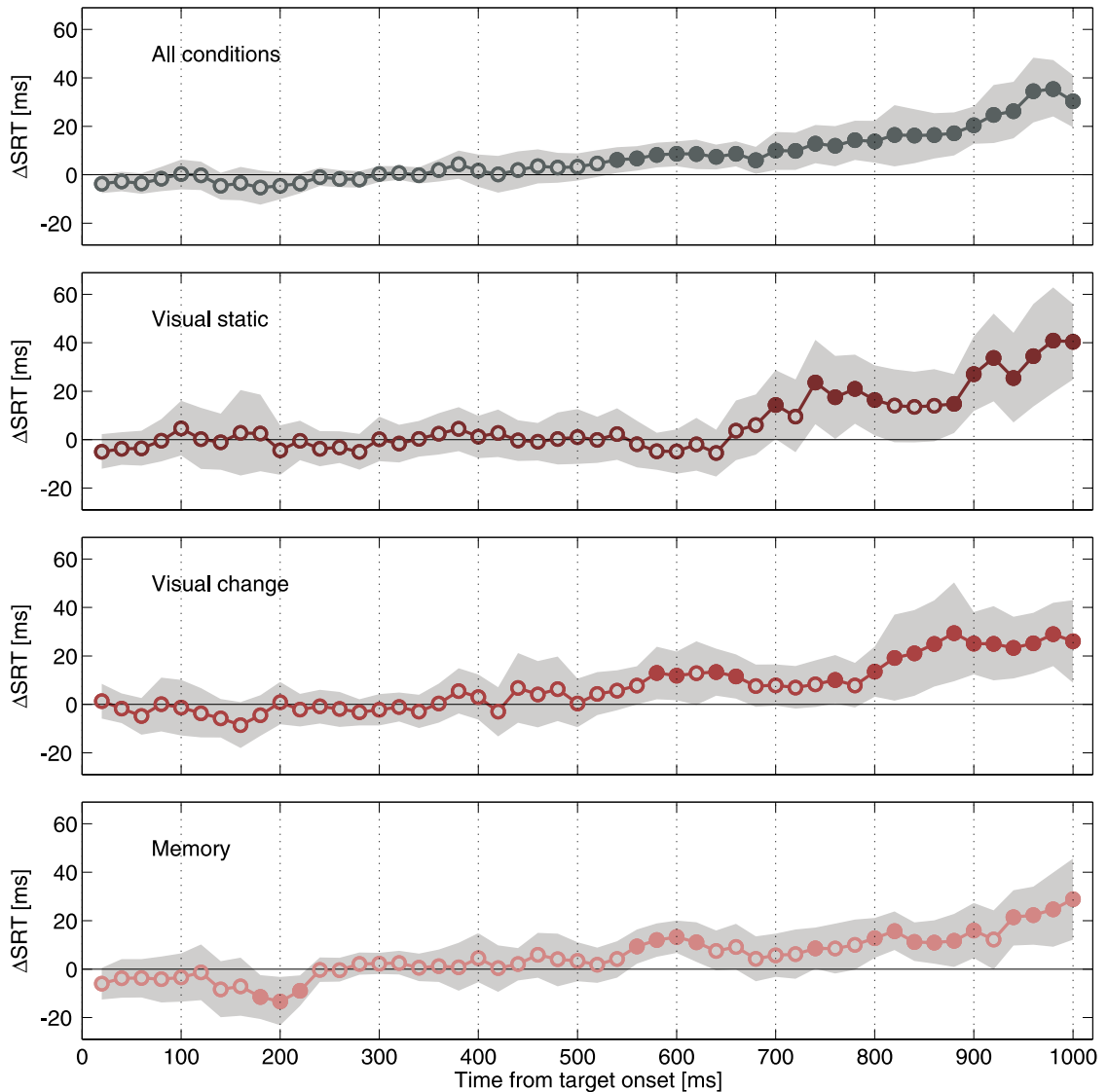


Figure 5.5: Temporal extension of benefits (negative values) and costs (positive values) in saccade latencies (ΔSRT) after microsaccades in different time windows (width: ± 50 , centered on the abscissae), plotted for each condition and after collapsing trials from all conditions. At 280 ms the target changed in the visual-change condition and disappeared in the memory condition; at 1000 ms the fixation spot disappeared (go signal). Shaded areas are $CI_{95\%}$; filled markers highlight deviations.

5.3 Discussion

The present study shows for the first time the relationship between microsaccades and latencies of subsequent saccades. First, saccade latencies to memorized target locations were shorter when a

Table 5.1: Mean and standard errors of means (SEM) of regression coefficients of the rmMRA for SRTs after microsaccades occurring within the last 500 ms before the go signal

Predictor	Mean	SEM	t	p_t	$-\Delta R^2$	$F_{-\Delta R^2}$	$p_{-\Delta R^2}$
<i>Regression model for main effects</i> ($R^2_{Predictors} = 0.039$; $R^2_{Subjects} = 0.259$; $R^2_{Model} = 0.275$)							
Constant	251.522	6.203	40.55	< 0.001			
Onset	0.058	0.010	5.92	< 0.001	0.0118	66.885	< 0.001
Amplitude	11.096	2.400	4.62	< 0.001	0.0053	29.827	< 0.001
<i>Regression model including interaction term</i> ($R^2_{Predictors} = 0.043$; $R^2_{Subjects} = 0.259$; $R^2_{Model} = 0.278$)							
Constant	251.720	6.199	40.61	< 0.001			
Onset	-0.062	0.040	-1.57	0.063	0.0001	0.781	0.377
Amplitude	26.908	5.487	4.90	< 0.001	0.0059	33.718	< 0.001
Onset \times Amplitude	0.054	0.016	3.49	< 0.001	0.0022	12.710	< 0.001

Note. Means, SEM and t statistics for predictors (based on 31 participants, i.e., 30 degrees of freedom for t statistics). $-\Delta R^2$ is the drop of variance of the full model after removal of the predictor; corresponding $F_{-\Delta R^2}$ and $p_{-\Delta R^2}$ values for the significance test of the decrement are reported. $R^2_{Predictors}$, $R^2_{Subjects}$, and R^2_{Model} show variance explained by predictors alone, by subjects alone, and by the full model, respectively.

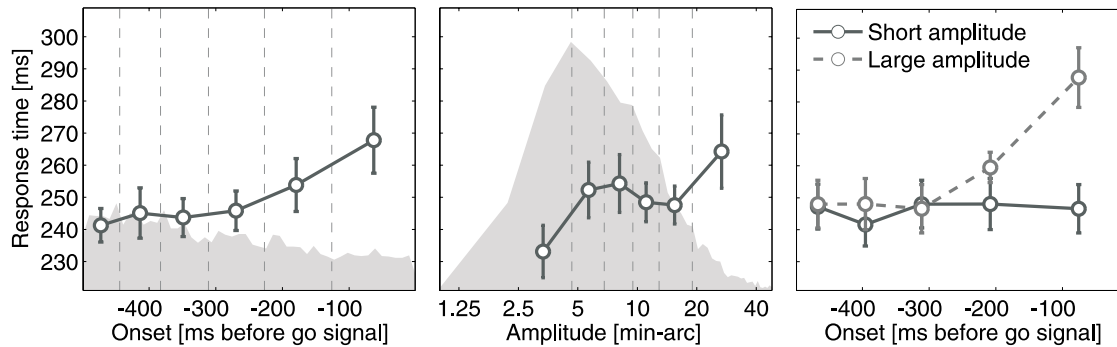


Figure 5.6: Microsaccade onset and amplitude effects on SRT. Error bars are $CI_{95\%}$ s. Shaded areas show distributions of the criterion, with binnings set to 1 min-arc for amplitudes and 10 ms for onset times. Dashed lines indicate the borders of the quantiles used for data aggregation.

microsaccade occurred while the target was transiently presented. This was true despite a delay of roughly one second between target removal and saccade. Second, microsaccades were associated with a pronounced slowing of SRTs if they occurred up to 500 ms before a saccade had to be executed. The costs that microsaccades imposed on performance were a function of microsaccade

onset and amplitude, later and greater microsaccades exhibiting a stronger impact. Moreover, we obtained the characteristic signature of microsaccade rate evolution in response to display changes in a new experimental paradigm. This is of relevance because, in a recent paper, Supèr et al. (2004) studied pre-saccadic activity in the primary visual cortex of monkeys trained on a delayed saccade task with this paradigm. They showed that V1 activity relating to saccade-target locations increased strongly and continuously from 100 ms prior to saccade execution. The authors carefully checked for the presence of a higher rate of microsaccades in this time window to rule out fixational movements as a cause of this V1 effect, but found no modulation of microsaccade rate. Based on our earlier research (Engbert & Kliegl, 2003b; Laubrock et al., 2005; Rolfs et al., 2004, 2005), we expected a substantial microsaccade-rate modulation to show up in a time window different from the ones analyzed by Supèr et al. (2004). Obviously, our results yielded the expected modulation and we think that the same microsaccade rate evolution could also be found in Supèr et al.'s data.

5.3.1 Shortening of saccade latencies following microsaccades

We provide three speculative explanations for the SRT shortening effect after microsaccades during target presentation in memory trials.

Perceptual-enhancement account. Single-cell recordings in monkeys have shown that microsaccades raise visual responsiveness of neurons in a number of brain areas involved in processing of visual information, including the Lateral Geniculate Nucleus (Martinez-Conde et al., 2000, 2002), V1 (Martinez-Conde et al., 2000, 2002; Snodderly et al., 2001, but see Leopold & Logothetis, 1998), V2 (Leopold & Logothetis, 1998), V4 (Leopold & Logothetis, 1998), and MT (Bair & O'Keefe, 1998). Since microsaccades relocate the retinal image, these effects have been associated with a refresh of visual information and it was hypothesized that microsaccades might enhance perception of stimuli in the visual periphery (Ditchburn, 1980; Gerrits & Vendrik, 1974; Martinez-Conde et al., 2000, 2004, 2006). If the SRT benefits following microsaccades during target presentation are caused by perceptual enhancements, the question arises why they are restricted to memory-guided saccades. Interestingly, Supèr et al. (2004) also reported a dissociation of visual and memory trials with respect to V1 activity and SRT; they found a strong negative correlation between strength of V1 activity and SRT for memory but not for visual trials. Obviously, a correlation between V1 activity prior to the saccade and the presence of microsaccades during the earlier target presentation

in memory but not visual trials would constitute strong evidence for a perceptual-enhancement interpretation restricted to the memory condition. However, given their focus on presaccadic activity in V1, this analysis was not provided by Supèr et al..

Motor-preparation account. An oculomotor area that is highly relevant to the production of memory-guided saccades is the frontal eye fields (FEF). This area receives retinotopically organized input from the visual cortex (Schall, Morel, King, & Bullier, 1995) and contains cells that show tonic discharge activity during the delay periods in memory-saccade tasks. This activity is spatially specific for the target location and available to the target saccade (Bruce, Friedman, Kraus, & Stanton, 2003; Friedman et al., 1997). In our experiment, conditions were presented in randomized order; the memory condition was certainly the most attention demanding one. Assume that subjects prepared for the disappearance of the target on a fraction of the time and that the occurrence of microsaccades reflects a spillover of enhanced activity in FEF, associated with the preparation of memory-guided saccades during target presentation. In this case, microsaccades are the consequence of task-set related activity. A faster SRT in the memory than the visual conditions would be a consequence of subjects' correct anticipation of a memory trial.

Attentional account. Finally, there is also an attentional interpretation of shorter SRTs after microsaccades during target presentation in memory trials. In previous work, we linked microsaccades to shifts of covert attention (Engbert & Kliegl, 2003b; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005). It could be that in those trials where a microsaccade was present this might indicate endogenous shifts of attention to the target incurring an SRT benefit. As the target disappears, there may be more incentive to hold endogenous attention at the remembered target location in the memory condition, whereas in the visual conditions attention may move back to the fixation point. The fact that the microsaccade-related SRT benefit is limited to the memory condition is also compatible with this account. This approach, for the time being, states a pure correlation between microsaccades during target presentation and faster SRTs.

Note that also a combination of the accounts given above might explain the SRT shortening effect. Consider, for instance, that microsaccades during target presentation caused a perceptual enhancement. As a consequence, motor-preparation signals in oculomotor areas might be enhanced. Due to a spillover effect, these might, in turn, result in microsaccades.

5.3.2 Prolongation of saccade latencies following microsaccades

Microsaccades share important characteristics with large-scale saccades. First, microsaccades are defined as binocular eye movements with almost identical amplitudes and directions in both eyes (Ditchburn & Ginsborg, 1953; Krauskopf et al., 1960; Lord, 1951), pointing at a central rather than a peripheral nervous origin. Second, both microsaccades and large-scale saccades fall on the main sequence (Zuber et al., 1965), i.e., the relationship between peak velocity and amplitude in these movements follows a power law. Thus, although the neurophysiological origin of microsaccades is still unknown, it is reasonable to assume a common neural circuitry for the generation of microsaccades and saccades (see also Engbert, 2006b). Beyond that, microsaccades occur involuntarily (Ditchburn & Ginsborg, 1953; Ratliff & Riggs, 1950), suggesting that subcortical processes might be most relevant in their production.

A number of voluntary and reflexive pathways exist to generate saccades, most of which converge to the SC, the key structure involved in the programming and execution of saccadic eye movements at the subcortical level (see Munoz et al., 2000; Scudder et al., 2002; Sparks, 2002, for reviews). Indeed, there are indications that microsaccades might have a neural correlate in the SC. In detail, the intermediate layers of the SC constitute a retinotopically organized motor map coding for saccades to the contralateral visual field. Here, saccade amplitudes are continuously represented, decreasing from caudal to rostral SC (D. A. Robinson, 1972). Cells in the rostral pole tonically discharge during fixation and pause or decrease firing during most saccades (Munoz & Guitton, 1991; Munoz & Wurtz, 1993a). Consequently, they were referred to as fixation neurons (FN, e.g., Munoz et al., 2000; Munoz & Fecteau, 2002; Munoz & Guitton, 1991; Munoz & Istvan, 1998; Munoz & Wurtz, 1993a, 1993b, 1995a, 1995b). Saccade-related neurons (SN) are located in the more caudal parts of the SC. SN pause firing during fixations while producing bursts of spikes prior to and during saccades directed to their response field (Munoz & Wurtz, 1995a; Sparks et al., 1976; Wurtz & Goldberg, 1972). Converging data from microstimulation studies (Gandhi & Keller, 1999b; D. A. Robinson, 1972) and single cell recordings (Krauzlis et al., 1997; Munoz & Wurtz, 1993a, 1995b) reveal that FN like SN still possess a movement field, i.e., activity of FN is associated with small contraversive saccades. It is not known whether microsaccades that occur involuntarily during fixation originate in the rostral pole of the SC as proposed by Gandhi and Keller (1999b), Munoz et al. (2000), and earlier in this thesis. Our findings that there is a drop of microsaccade rate prior to saccades and an increase of SRTs in both memory and visual trials when microsaccades

occurred around the time of the go signal are consistent with this explanation. As the process of saccade generation requires reciprocal activation of the FN and SN (Munoz & Istvan, 1998; Munoz & Wurtz, 1993b, 1995b), a decrease in microsaccade rate would be necessary for saccade generation. Concurrently, a decrease in mean microsaccade amplitude would be predicted, since a deactivation of the rostral pole of the SC is proposed to affect microsaccades with large amplitudes at first (see Chapter 4). Indeed, the amplitude of the few remaining microsaccades observed just before the saccade was very short on average as compared to the amplitude of microsaccades occurring before target presentation. In addition, large microsaccades imposed greater costs to the latency of participants' response, especially when occurring just before a response saccade was instructed by the go signal. Thus, the findings reported here are in good agreement with our model.

The present chapter demonstrates on a behavioral basis the differential role of microsaccades in saccade generation. To clarify whether microsaccades are a cause, a correlate, or a consequence of altered saccade dynamics, it will be important to determine their neurophysiological correlates. In agreement with our knowledge about the saccadic system, we propose that the SC is a likely candidate at which attentional and perceptual benefits of microsaccades and their inhibiting impact on the generation of large saccades converge.

Chapter 6

Microsaccades in the course of saccade preparation

In Section 3.3 we proposed a model of microsaccade generation according to which microsaccades originate from activity in the central part of a saccadic movement field, the site that is primarily active during attempted fixation. This model holds that microsaccades and saccades result from activity of competing motor programs. In the set of experiments presented here, we further pursued these ideas by experimentally manipulating the activity of the saccadic motor map. This was achieved by using oculomotor paradigms, which are known to encounter specific effects on the processes of saccade generation: the gap task and the anti-saccade task.

In three experiments, we will address several questions. First, it is at issue how microsaccade generation is implemented. Comparing microsaccade statistics to neurophysiological results from single-cell recordings of the monkey's superior colliculus (SC) motor map, we will evaluate our view on the physiological origin of microsaccades. According to the model that we proposed, microsaccades originate in the fixation-related part of a movement field that finds its physiological equivalent in the rostral pole of the SC motor map. We further ask, what microsaccades reveal about the current plans to generate a saccade. The basic reasoning behind this question is that if activity in the central part of the saccadic motor map translates to microsaccadic behavior, then the characteristics of this behavior might carry information about the current state of the motor map. In the previous chapters, we have presented evidence corroborating this hypothesis. Most

importantly, we have shown that the occurrence of a microsaccade implicates remarkable costs for the oculomotor system trying to generate a saccade (see Chapter 5). We suggested that microsaccades provide insight into saccade preparation, indicating that, at the time of their occurrence, fixation-related activity is still high and saccade preparation presumably less advanced. Here, we aimed to replicate these findings in different experimental settings and ask whether there is more to the relation between microsaccades and saccade preparation than this rather global and purely temporal information. To this end, we again build a link between physiological processes of saccade preparation in the SC motor map and microsaccade statistics in saccade tasks for which these processes have been well described.

6.1 Microsaccades and reflexive saccades: The gap task

In the gap paradigm, a fixated target is removed before a peripheral saccade target sets on (Saslow, 1967). As compared to a condition where there is no asynchrony between the two events (step condition) or the fixation target remains visible (overlap condition), this manipulation results in a shortening of saccade latencies. Commonly, we refer to this phenomenon as the *gap effect*. Despite the simple nature of the stimulus arrangement in the gap task, saccadic reaction times vary appreciably. In fact, bimodal distributions of saccade latencies are often observed, consisting of express saccades (latencies of 80 to 130 ms) and saccades with regular latencies (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). Note that the gap effect and the occurrence of express saccades are related but separate phenomena, since even if no express saccades can be observed, saccade latencies are strongly reduced in the gap as compared to the overlap condition (e.g., Kingstone & Klein, 1993; Paré & Munoz, 1996; Reuter-Lorenz et al., 1991; Wenban-Smith & Findlay, 1991).

6.1.0.1 Temporal preparation by fixational disengagement in the gap task

Since Saslow's introduction of the gap paradigm, much progress has been made in understanding both the processes involved in the generation of the gap effect as well as the variability in response times. One major component thought to generate the gap effect relates to activity in the fixate system, which is assumed to be reduced after the removal of the fixation stimulus. As a consequence, the generation of saccades will be facilitated as compared to a situation where the eyes stick to an enduring fixation stimulus. There is general agreement that this kind of temporal

preparation by fixational disengagement may account for one part of the gap effect, even though the hypothesis was formulated in several ways by different authors (e.g., Dorris & Munoz, 1995; Findlay & Walker, 1999; Fischer & Weber, 1993; Kingstone & Klein, 1993; Mayfrank, Mobashery, Kimmig, & Fischer, 1986; Pratt, Bekkering, & Leung, 2000; Tam & Stelmach, 1993).

We hypothesized that microsaccades are a behavioral correlate of activity at fixation-related sites in the saccadic motor map of the SC, i.e., at its rostral pole. Thus, fixational disengagement implicates predictions for the evolution of microsaccade statistics in the gap task and will shed new light on the relationship between microsaccades and saccades.

A first set of hypotheses aims to directly assess the relationship of the activity of fixation-related neurons (FN) in the SC to statistics of microsaccades. We propose that the rate and amplitude of microsaccades is a function of activity at the central part of the movement field (the rostral site of the SC). As reported by Dorris and Munoz (1995), the activity of FN decreases after fixation offset. In their monkey subjects, FN discharge rates before target onset were minimal for 200 to 300 ms gap durations, before increasing again for longer gap durations and settling at a level well below what is observed during visual fixation. According to the model proposed in Section 3.3, this pattern of activity could translate to microsaccadic behavior in at least two ways, depending on the exact spatial distribution of activity and how it evolves with increasing gap durations. First, decreased neurophysiological activity could result from a narrowing of that part of the movement field that exhibits supra-threshold activity. As a consequence, mean microsaccade amplitude would decline with decreasing neural activity, without necessarily affecting microsaccade rate. Second, neural activity could show a general decrease, however, letting the width of the area exhibiting supra-threshold activity remaining unchanged. In this case, microsaccade rate would be subject to this change in activation, while the distribution of microsaccade amplitudes remained virtually the same. Third, a combination of the two mechanisms is possible. To the best of our knowledge, the exact spatial distribution of rostral SC activity in the gap task is not known so far. Consequently, the interaction of microsaccade rate and amplitude cannot be predicted in great detail. We propose that the combination of microsaccade rate and amplitude should most closely resemble the temporal evolution of rostral SC activity in the gap task; both higher microsaccades rates and larger amplitudes would correspond to higher levels of activity in the rostral SC.

A second set of hypotheses concerns the relationship between the occurrence of microsaccades and the latencies of subsequent saccades. First, trials with long saccade latencies should be

associated with higher pre-target microsaccade rates, while short saccade latencies should be observed after lower pre-target microsaccade rates. Second, the occurrence of a microsaccade around the time of target appearance is predicted to result in saccades of longer latency, replicating the findings reported in Chapter 5. These predictions originate in the idea that microsaccades represent supra-threshold activity that is in competition with the generation of large saccades. A new saccade may only be generated if this activity is overcome.

6.1.0.2 Spatial preparation by localized readiness in the gap task

As a consequence of fixational disengagement, motor preparation may be favored by the gap condition, therefore, accounting for further reduction of response latencies in the gap task. In agreement with this hypothesis, the occurrence of express saccades often requires extensive training and these training effects were shown to be spatially selective; i.e., once trained on specific targets, monkeys made express saccades only to targets at or close to the trained location (Boch & Fischer, 1986; Fischer, Boch, & Ramsperger, 1984; Paré & Munoz, 1996). To account for spatial selectivity in oculomotor preparation, many authors proposed localized readiness of the saccadic system (Becker, 1989; Dorris & Munoz, 1998, 1999; Dorris et al., 1997; Kowler, 1990; Munoz et al., 2000; Munoz & Fecteau, 2002; Paré & Munoz, 1996; Rolfs & Vitu, submitted; Trappenberg et al., 2001; West & Harris, 1993). This motor-preparation hypothesis assumes that topographically organized oculomotor programs coding saccade metrics can be partially prepared before the target comes on or the signal to launch a saccade is given. Paré and Munoz (1996) followed up the motor-preparation hypothesis with monkeys trained on the gap task. In addition to the established spatially circumscribed training effects on express-saccade occurrence, the authors found that the latency of regular saccades was lowest at the trained location, when a set of different locations was interleaved. Albeit, a gap effect was present for each location. Moreover, target uncertainty reduced the probability of express saccades.

The motor-preparation hypothesis is able to account for these results by postulating that preparatory processes are spatially confined to those locations where target appearance is probable (as known from prior experience with the task at hand). Single-cell recordings in monkeys provided strong support for the motor-preparation hypothesis. As pointed out before, recordings from FN in the rostral pole of the SC revealed that their activity is attenuated during the gap period, in a manner consistent with the idea of fixational disengagement (Dorris & Munoz, 1995).

More crucially, a good share of the variability in saccadic response times could be attributed to variability in pre-stimulus activity in buildup neurons in the SC motor map (Dorris et al., 1997), if the saccade-target location fell into their response field. Pre-stimulus activity in these neurons increases with target-location certainty (Basso & Wurtz, 1997, 1998) and saccadic probability (Dorris & Munoz, 1998), respectively. In further studies, this activity has been associated with motor-preparation since it is predictive of saccadic response times (Basso & Wurtz, 1998; Dorris & Munoz, 1998). Thus, these studies revealed spatially specific movement-preparation effects on the neurophysiological level, which strongly favor the motor-preparation account.

Like fixational disengagement, spatial preparation mechanisms might exert impact on microsaccade statistics in the gap paradigm. We will examine whether the direction of a microsaccade carries information about the spatial preparation of a saccade. The rationale is that the spatial distribution of activity of the central part of the saccadic motor map may be subject to preparatory activity elsewhere in the field. This idea relates to the finding that the spatial allocation of attention results in a bias in microsaccade directions (Engbert & Kliegl, 2003b; Galfano et al., 2004; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005). It has been proposed that these directional biases indicate the preparation of a motor response to a peripheral location (Betta, Galfano, & Turatto, in press; Galfano et al., 2004; Rolfs et al., 2004). Thus, in turn, activity associated with the preparation of a saccade to a specific location might shift the center of activation at fixation-related sites of the motor map towards more peripheral locations. If this was true, saccadic performance should depend on the congruency between the direction of an observed microsaccade and the location of a subsequent target presentation.

However, various factors dilute our confidence in finding a relationship of this kind. First, the preparation of a saccade to one certain location in the visual field is not instructed in the gap task. Here we used two target locations that were randomly interleaved. Thus, it is possible that two saccades are prepared concurrently, obliterating their effects on the distribution of activity at the central part of the hypothesized movement field. Second, the direction of microsaccades is probabilistic in nature. Thus, even if saccade preparation is restricted to one side only, not every microsaccade will go in the corresponding direction. Third, we do not know, how the preparation of a saccade would influence the spatial distribution of activity at the central part of the movement field. To the best of our knowledge, there is no data showing how, during fixation, activity is spatially distributed in the rostral pole of the SC, nor is there any information regarding the

dynamics of this distribution in the gap task. Consequently, we cannot derive detailed predictions about the relationship of microsaccade direction and the latency of subsequent saccades to either of the two potential target locations. In any case, this influence will be indirect, since we assume that it is not the preparatory activity itself that generates the microsaccade. Finally and most crucially, motor preparation inherently comes with a decrease of fixation-related activity due to mutual interactions intrinsic to the saccadic motor map. If microsaccade generation relies on this activity (as we propose), mutual inhibition unfortunately reduces the likelihood of finding a reliable relationship between target location and microsaccade direction. In face of all that, we tested for effects of microsaccade-target congruency.

The hypotheses described above will be tested in a classical gap task. The layout of the task was largely adopted from Dorris and Munoz (1995) and Dorris et al. (1997), respectively, to facilitate comparison to their physiological data. Seven gap durations were interleaved (0, 100, 200, 300, 400, and 600 ms). In that way, it was possible to measure saccadic performance while the activity of the saccadic motor map evolved in time.

6.1.1 Methods

6.1.1.1 Participants

Ten students of the University of Potsdam were paid 14€ or received study credit for their participation in this experiment. Each participant performed two sessions; inter-session intervals ranged from one to seven days (3.4 days on average). They were 19 to 27 years old (21.3 years on average), had normal or corrected-to-normal vision, and were in good health.

6.1.1.2 Experimental setup and eye-movement recording

Participants were seated in a silent and darkened room with the head positioned on a chin rest, 50 cm in front of a computer screen. Stimuli were presented on a 22-inch iiyama HM204DT CRT (1024 by 768 resolution or 46° by 34° of visual angle; refresh rate 100 Hz). The experiment was controlled by an Apple Power Macintosh G4 computer. Eye-position data were recorded and available online using an EyeLink-II system (SR Research, Osgoode, ON, Canada) with a sampling rate of 500 Hz and a noise-limited spatial resolution better than 0.01°. The experimental software controlling stimulus display and response collection was implemented in Matlab (MathWorks, Natick, MA,

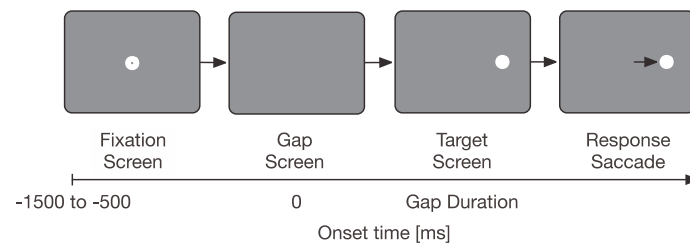


Figure 6.1: Sequence of visual stimulation in the gap experiment.

USA), using the Psychophysics (Brainard, 1997; Pelli, 1997) and Eyelink (Cornelissen et al., 2002) toolboxes.

6.1.1.3 Procedure

Participants performed 10 practice trials and 300 test trials in a variant of the classical gap task (Saslow, 1967). Practice trials were comparable to test trials in all respects. Before the first and after every 30 trials the eye tracker was calibrated (standard 9-point grid) and calibration was validated. Every fifth trial, a drift correction was carried out. Before each trial, the fixation spot was displayed at the center of the computer screen. To start a trial, correct fixation was required. Otherwise, a drift correction was carried out and the trial was started over. If the eyes were still not detected within the critical area (see below), the calibration was repeated.

Figure 6.1 illustrates a typical sequence of visual stimulation used in the gap task. At the beginning of each trial, a small ring, displayed centrally on a gray background, was fixated. After a random interval of 500 to 1500 ms, the fixation spot was removed. Subsequently, a target stimulus appeared in the visual periphery, 10° to the left or to the right. Between the offset of the fixation spot and the onset of the target (gap duration), a temporal gap of no visual stimulation could be introduced. Participants were asked to hold fixation centrally until the target was displayed. Then, a speeded response saccade was required. Response saccades (eye position shift to either of the two targets) were detected on-line and terminated the trial 200 ms after the eyes had passed a virtual border 2° away from the fixation spot. If no response was detected, the trial ended after 700 ms of target presentation. Inter-trial intervals of 500 ms with no visual stimulation were introduced to enable blinking. Gap durations were 0, 100, 200, 300, 400 or 600 ms, with each duration having equal probability across the 300 trials. Consequently, 50 trials of every gap duration (25 left and 25 right targets) were presented in a randomized order.

If gaze position left a fixation square (4° side length, centered on the fixation spot) while fixation was still required (i.e., before the target was presented), an error feedback was triggered and the trial was aborted. Aborted trials were repeated in random order after the 300 regular trials.

6.1.1.4 Stimuli

The fixation spot was a white ring with a diameter of 0.67° of visual angle and an inset with a diameter of 0.13° . The target stimulus was a filled white circle (diameter: 0.67°). The error feedback, triggered by blinks and anticipatory saccades, was a binaural 660 Hz tone at about 70 dbA, played for 82 ms via the internal speakers of the G4 computer.

6.1.1.5 Data preparation

For data analysis, a post-hoc saccade detection was performed using a new version (Engbert, 2006b) of the algorithm by Engbert and Kliegl (2003b). Velocities were computed from subsequent samples in the series of eye positions in the response time window 500 ms on from the go signal. Saccades were detected in 2D velocity space using thresholds for peak velocity and minimum duration. We used a relative threshold of 6 SDs of the velocity and a minimal duration of 6 ms (or three data samples). The first saccade that directed gaze to a location less than 8° away from a target location was taken as a response saccade.

Subsequently, we used the same algorithm to detect microsaccades (amplitude $< 1^\circ$) in the interval from fixation onset to the response saccade. We considered only binocular microsaccades.

Trials including saccades larger than 1° prior to the response saccade were discarded, as were trials with incorrect responses and SRTs shorter than 70 ms. Some trials had to be excluded due to data loss during eye-movement recording. By mistake, eye movements of one participant were sampled at 250 Hz instead of 500 Hz in one of the two sessions; his data were excluded from the analyses. Consequently, nine participants contributed 493 to 588 trials to the final data analyses, resulting in a total of 5084 trials (out of 5400 or 94.1%) in which 16257 microsaccades were detected.

6.1.1.6 Data analysis

Where provided, confidence intervals were computed using a simple bootstrapping technique (Efron & Tibshirani, 1993). From an original sample of N values, 1000 bootstrap samples were generated, each by selecting (with replacement) N values of the original sample. The 1.96-fold of

the standard deviation of the means of these 1000 bootstrap samples was computed to generate 95% confidence intervals ($CI_{95\%}$) of the mean of the original sample. In figures, confidence intervals will often allow the reader to compare different conditions by so-called “rules of eye” (Cumming & Finch, 2005). For within-subject comparisons, uninformative between-subject variance was therefore removed (Cousineau, 2005); these confidence intervals will be labeled $CI_{95\%ws}$.

6.1.2 Results

6.1.2.1 Performance in the task

Figure 6.2 shows average saccade-latency distributions for each gap duration tested. Dotted white lines indicate the border of 130 ms below which saccades are usually counted as express saccades (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). If at all, bimodal distributions consisting of separate modes of express saccades and regular saccades were obtained for gap durations longer than or equal to 200 ms. In the 0 ms gap condition, clearly less saccades were produced at express latency than in any other condition. For the 100 ms gap condition, a unimodal response-time distribution consisted of both express saccades and regular saccades.

Response-latency data were aggregated for Figure 6.3, displaying saccade latencies and the percentage of express saccades (latencies below 130 ms) as a function of gap duration. Means are shown with $CI_{95\%ws}$. The most prominent effect is that introducing a temporal gap between the offset of the fixation stimulus and the onset of the saccade target results in a strong decrease of saccade latencies and a concurrent increase in the number of saccades with extremely short latencies. This replicates the classical gap effect (Saslow, 1967). Only minor variations in response latencies and the number of express saccades were found between gap durations of 100 ms and more. Note that we present percentages of express saccades only for the sake of convention. In our subsequent analyses we do not hang on to a distinction of express and regular saccades, respectively, but rather contrast fast to slow responses more generally.

6.1.2.2 Microsaccade rate

Microsaccade rates were computed as described in Section 4.1.6. The left panel of Figure 6.4 shows microsaccade rates across the time course of trials, aligned on the offset of the fixation stimulus (gap onset). Rate evolutions represent averages over participants and are presented for each gap

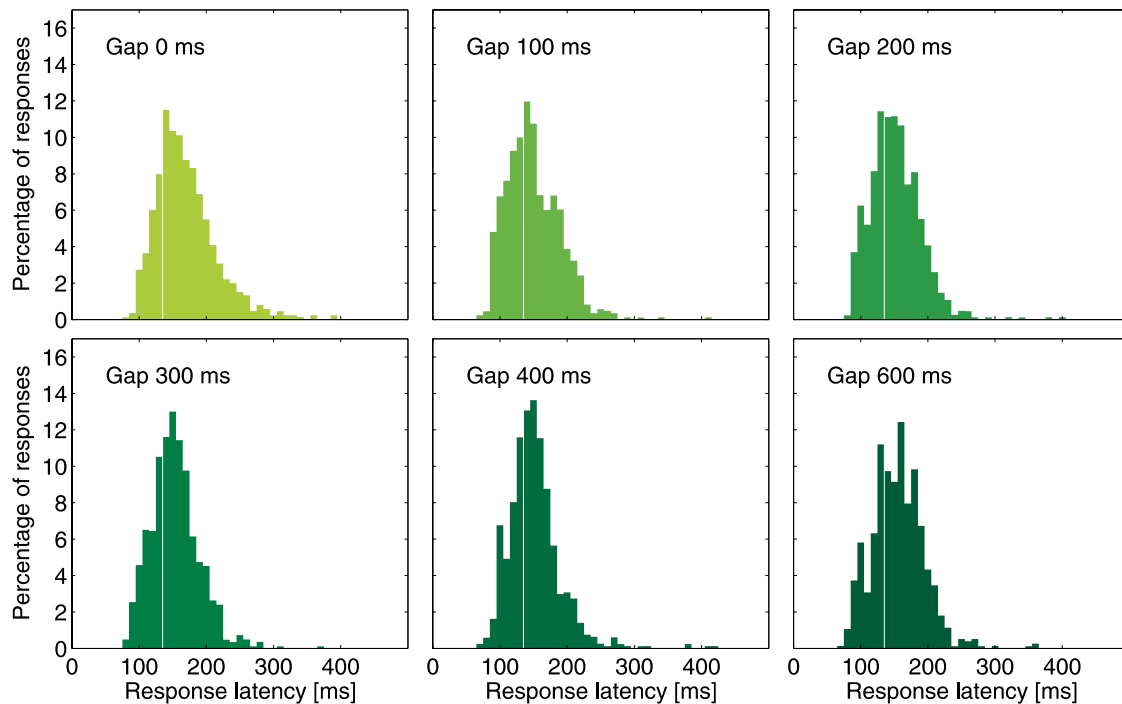


Figure 6.2: Average response-latency distributions plotted for each gap duration separately. Thin white lines indicate the border of 130 ms below which saccades are traditionally considered express saccades (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). Bin width was set to 10 ms.

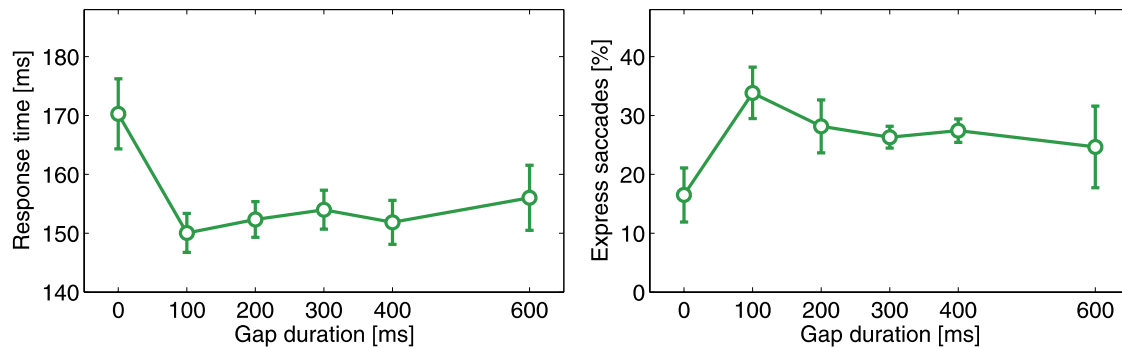


Figure 6.3: Aggregated response latencies and percentages of express saccades as a function of gap duration. Error bars are $CI_{95\%}$ s.

duration separately.¹ About 70 ms after the offset of the fixation target, microsaccade rate sharply decreased from a baseline level of 1.2 s^{-1} , reaching a minimum of 0.5 s^{-1} after about 130 ms. Subsequently, microsaccade rate increases again, peaking at 250 ms (1.1 s^{-1}), before continuously

¹Note, however, that gap durations were interleaved in our task and participants were not informed about the conditions in a certain trial. Thus, all deviations between the different microsaccade rates in Figure 6.4 must be random.

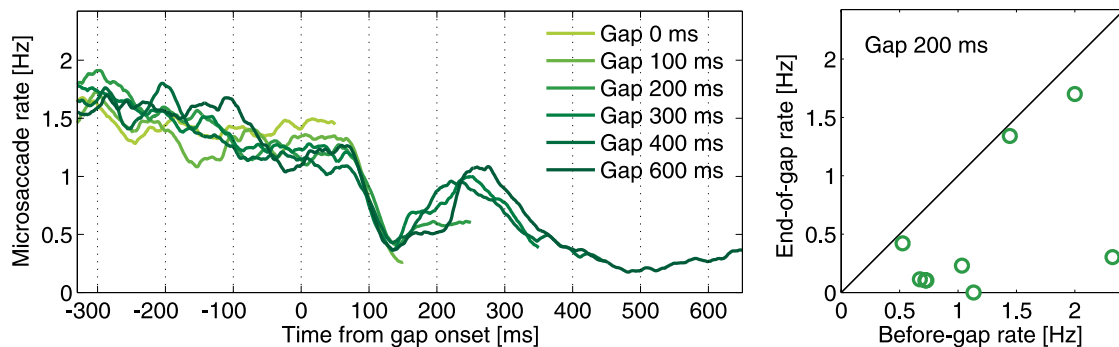


Figure 6.4: Microsaccade rate as a function of time. Rate evolutions are plotted for the six gap durations tested, aligned on gap onset (left panel). Rates were computed using an asymmetric filter and averaged across participants. In the right panel, a comparison of individual microsaccade rates before gap onset (-100 to 0 ms) and before target onset (100 to 200 ms) is presented for the 200 ms gap condition. Each dot represents one participant.

decreasing for longer gap durations. The right panel of Figure 6.4 depicts individual rates in two different time windows of 200 ms gap trials, before the gap (-100 to 0 ms) and at the end of the gap period (100 to 200 ms). The initial decline in microsaccade rate after the offset of the fixation stimulus is very stable across participants; all nine participants showed a reduction from the first to the second time window.

6.1.2.3 Microsaccade amplitude

Strong microsaccadic inhibition was triggered by fixation offset in the gap task. As a next step, we determined the time course of microsaccade amplitudes. For this purpose, we computed differences of microsaccade amplitudes in different time windows to a baseline time window. A baseline time window of 175 to 125 ms before gap onset was chosen to ensure that no microsaccade may be included in both the baseline time window and the test time windows for the saccade-locked analyses (see below), which could happen to be the case for short gap durations. The upper panels of Figure 6.5 plot the temporal evolution of these differences locked to the beginning of the gap period (left panel) and to saccade onset (right panel), respectively. To create this figure, mean amplitudes were determined using a 50 ms moving boxcar window centered at a time; then, we subtracted the baseline amplitude from these values. For the gap-onset-locked analyses, we collapsed data of all gap conditions and included only microsaccades that occurred 70 ms

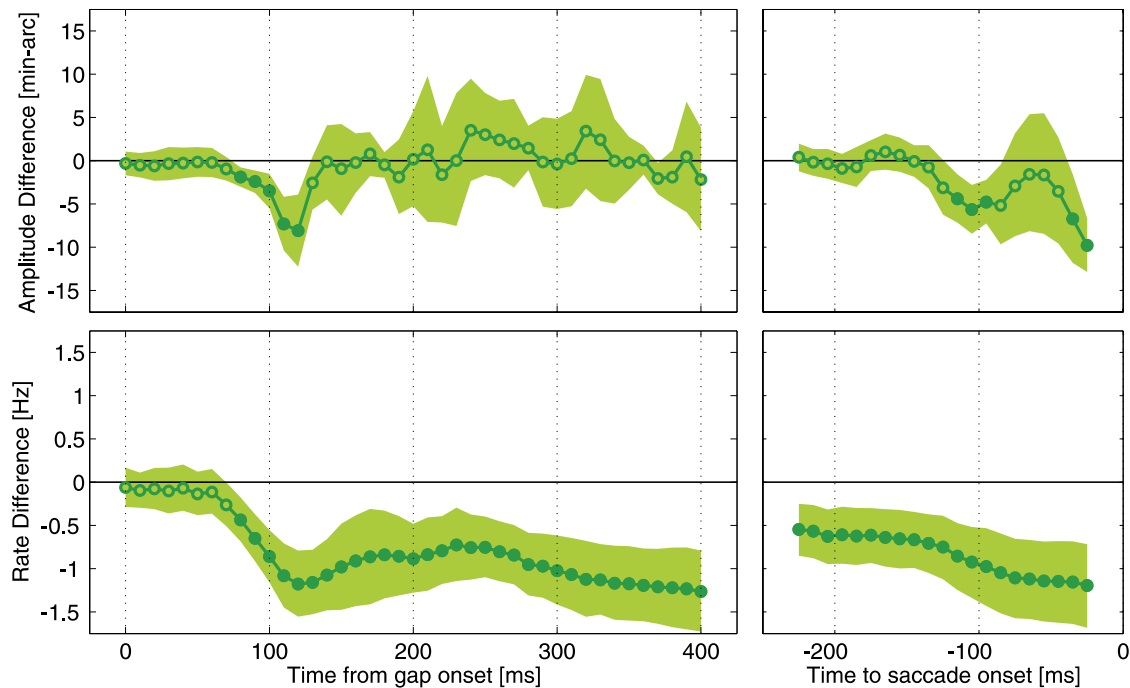


Figure 6.5: Differences of mean microsaccade amplitudes (upper panels) and rates (lower panels) to a baseline time window (-175 to -125 ms before gap onset) as a function of time to gap onset (left panels) and time to saccade onset (right panels), respectively. Data were collapsed across all gap-duration conditions. Shaded areas are $CI_{95\%}$ of the differences. Filled dots indicate significant effects after applying FDR (Benjamini & Hochberg, 1995).

after target presentation, or earlier.² Shaded areas represent $CI_{95\%}$; filled dots indicate significant effects after applying the false-discovery-rate procedure (FDR) by Benjamini and Hochberg (1995), effectively controlling the expected proportion of erroneously rejected null-hypotheses (less than 5%). We also recomputed microsaccade-rate evolutions using this type of analysis (lower panels of Figure 6.5) to warrant optimal comparability. Note that the number of contributing trials per participant decreases with increasing gap duration, since short gap durations include data from long gap durations, but not vice versa. Moreover, the lower rate of microsaccades in later time windows decreases the inner-subject reliability of the amplitude scores. As a consequence, confidence intervals will increase over time.

Starting from a baseline amplitude of 17.6 min-arc before fixation offset, an average decrease in microsaccade amplitude to 9.3 min-arc was found about 120 ms after gap onset. The decrease

²In an additional analysis we checked whether gap-onset locked effects were confounded with saccade-locked effects. To this end, we only included microsaccades that occurred 150 ms before target presentation, or earlier. None of the results reported here changed by applying this constraint.

closely followed the time course of microsaccadic inhibition as indicated by the rate-difference plot in the lower left panel of Figure 6.5. Subsequently amplitude increases again and cannot be distinguished from the baseline amplitude in any other time window. Microsaccade rate remains on a low level (as compared to the baseline rate) for all gap times longer than 120 ms, where the initial inhibition peaked. When locked to saccade onset, both microsaccade rate and amplitude strongly decline, reaching a minimum just before the onset of the response saccade.

6.1.2.4 Microsaccade rates and saccadic response latencies

One approach to identify the correlation of saccadic response latencies with microsaccade statistics was to split response-time data into quantiles, allowing for the computation of associated microsaccade rates prior to saccadic responses-latency classes. In that way, we aimed to determine the overall impact of microsaccade occurrence on saccade generation.

In a first run of this analysis, trials of all gap durations were collapsed. We centered saccade latencies for every gap duration and session by removing the mean from each latency on the level of individuals. By that, we omitted confounding the response-time related rate evolutions examined here with gap-induced and/or session-related saccade-latency modulations, respectively. We split each participant's saccade-latency distribution into quartiles and computed microsaccade rates over the trials assigned to these quartiles. Rates were determined for all microsaccades together as well as for target-congruent and target-incongruent microsaccades, separately. Microsaccades were considered target-congruent, if the associated movement angle deviated from the direct connection between fixations spot and target by no more than $\pm 45^\circ$; microsaccades were considered target-congruent, if the associated movement angle deviated no less than $\pm 135^\circ$ from the fixation-target axis. Figure 6.6 compiles the results from this analysis. The line plot in the leftmost panel shows average microsaccade rates (and $CI_{95\%wts}$) in a time window around target onset (before-target rates: -30 to 70 ms relative to target onset); the longer the response latencies, the higher the associated preceding microsaccade rates. The bar charts in the same panels present the analysis as a function of microsaccade-target congruency. These results were statistically assessed by a repeated-measures ANOVA using response time quantiles and microsaccade-target congruency as independent variables. There was a main effect of response time on microsaccade rate; $F(3, 24) = 8.60, p < 0.001$. Rates did not differ as a function of microsaccade-target congruency $F(1, 9) = 1.84, p = 0.21$. Finally, we obtained no interaction of saccade latency and congruency;

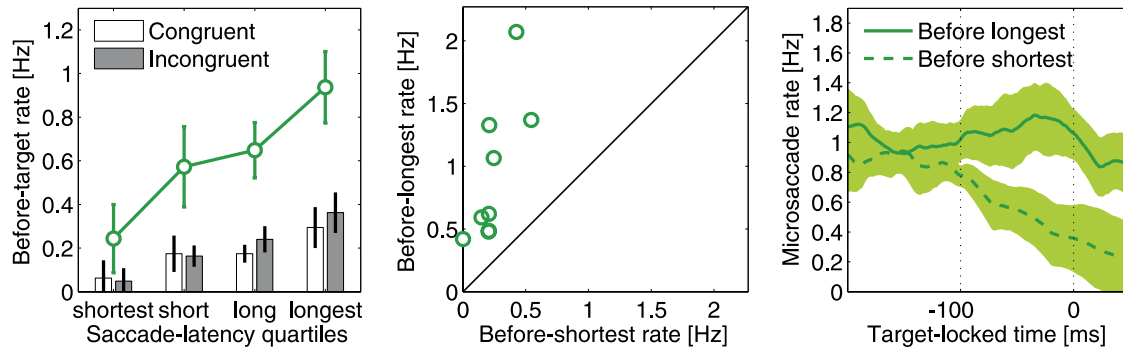


Figure 6.6: Pre-target microsaccade rate as a function of saccade latency. The leftmost panel shows microsaccade rate (overall and as a function of microsaccade-target congruency) before shortest, short, long, and longest saccade latencies (quartiles of individual response-time distributions), respectively. Error bars are $CI_{95\%ws}$. The middle panel compares microsaccade rates before the fastest (first quartile) and the slowest responses (fourth quartile) on the level of individuals. Each dot represents one participant. The rightmost panel displays the time course of microsaccade rates before the fastest and slowest responses. Shaded areas are $CI_{95\%ws}$.

$F(3, 24) = 1.17, p = 0.34$. Thus, microsaccades (independent of congruency) were more frequent prior to slow responses, but were rarely observed before short response latencies. As shown in the middle panel of Figure 6.6, all nine participants had a lower probability of microsaccades before their shortest responses (first quartile) than before their longest response latencies (fourth quartile). A significant separation of microsaccade rates before these two extreme classes of response times was yet to be observed about 100 ms before target onset (rightmost panel of Figure 6.6).

We also ran this analysis for each gap duration separately. For these analyses, we performed a median split of response latencies to counter the loss of data associated with a separation of gap durations. The results are displayed in Figure 6.7. As can readily be seen in Figure 6.4, however, pre-target microsaccade rate was fairly low if gap durations exceeded 0 ms. Therefore, we settled for reporting two repeated-measures ANOVAs, one for the 0 ms gap condition, in which the pre-target microsaccade rate was comparably high, and one collapsing all other trials (again controlling for the effects of gap duration and session on saccade latency; see above). For the gap 0 ms condition, the ANOVA yielded a significant main effect of response-latency class; $F(1, 9) = 7.29, p = 0.027$. There was no effect of microsaccade-target congruency; $F(1, 9) = 2.41, p = 0.16$. However, microsaccade-target congruency was a function of response-latency class as indicated by a significant interaction of both factors; $F(1, 9) = 6.58, p = 0.033$. A higher rate of target-incongruent microsaccades was associated with a lower response latency. For the gap ≥ 100 ms

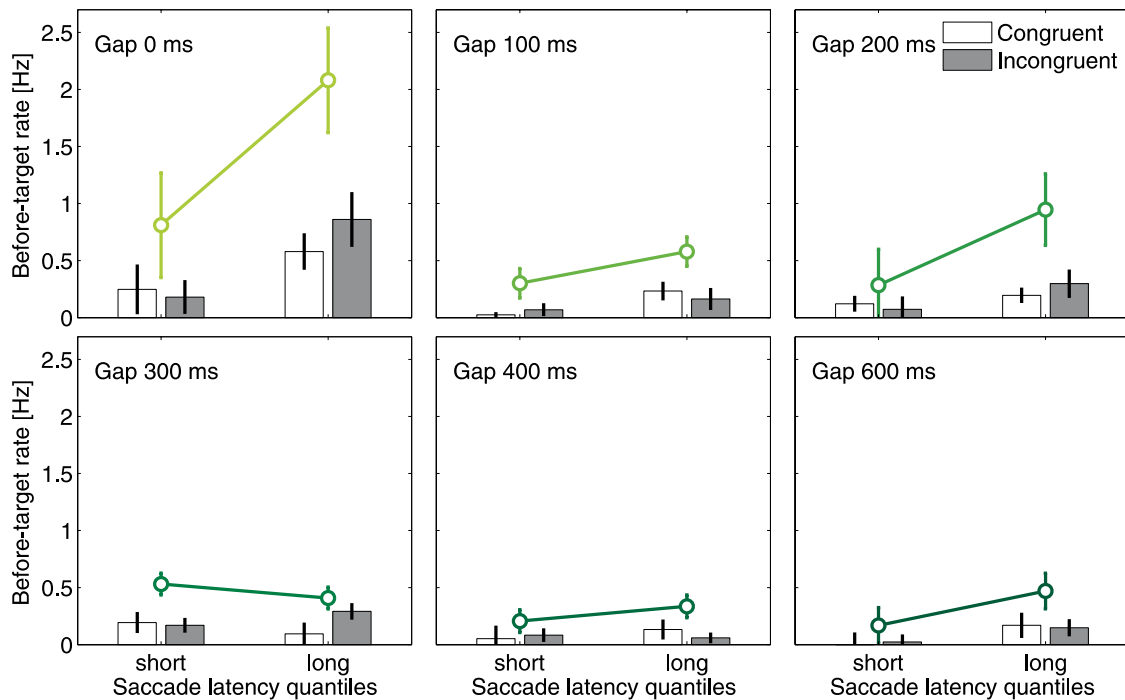


Figure 6.7: Pre-target microsaccade rate as a function of gap duration, response time, and microsaccade-target congruency. Error bars are $CI_{95\%}$.

conditions, we obtained only a main effect of saccade-latency class; $F(1, 9) = 10.00, p = 0.013$. No main effect of microsaccade-target congruency and no interaction with response-time quantile were evident; all $F_s < 1$, all $p_s > 0.58$.

Two aspects of the data might contribute to the weaker relationship between microsaccade rate and response time for longer gap durations. Both microsaccade rate and response time had smaller ranges, in which potential effects could have expressed themselves. This is illustrated in Figure 6.8, depicting mean rates of microsaccades (and $CI_{95\%}$) in the pre-target time window as a function of subject-centered saccade-latency quantiles and response type. The effect of session was controlled as in the previous analyses. Clearly, both microsaccade rate and saccade latency span greater ranges for the 0 ms gap duration than for all other gap durations.

6.1.2.5 Microsaccade-induced changes in saccadic response latencies

In a second set of analyses, we determined microsaccade-induced modulations in saccade latencies on a trial-by-trial basis. Again, we collapsed data from all trials after centering saccade latencies

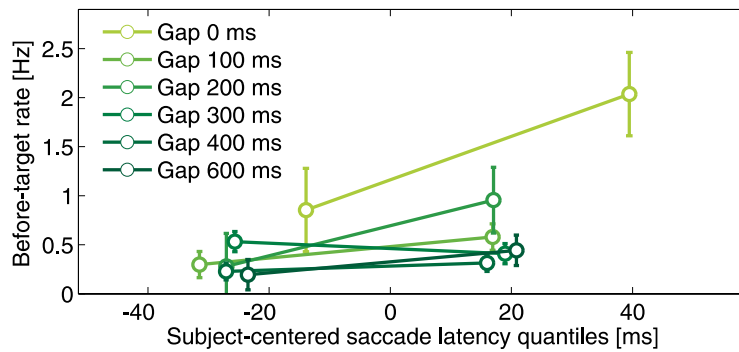


Figure 6.8: Pre-target microsaccade rate for the different gap durations as a function of subject-centered response time. Error bars are $CI_{95\%ws}$.

for every gap duration and session by removing the mean from each latency on the level of individuals. For time windows in a broad temporal range, reaching back from target onset, we computed individual mean SRTs given that either a microsaccade occurred or no microsaccade was observed and subtracted the latter value from the first for each of these time windows (width: 100 ms). Mean benefits (negative values) and costs (positive values) in saccade latencies (ΔSRT_{mic}) embedded in $CI_{95\%}$ are displayed in the upper panel of Figure 6.9. Filled markers highlight deviations from zero that were significant after applying FDR (Benjamini & Hochberg, 1995) to adjust the alpha level. Slowing extended back in time to about 160 ± 50 ms. Nine out of nine subjects showed slower response times if a microsaccade was observed in the pre-target time window (-30 to 70 ms relative to target onset). The magnitude of the individual effects are shown in the upper right panel of Figure 6.9.

Next, we examined whether the microsaccade-induced impact on saccade latencies was a function of microsaccade-target congruency. Like in our previous congruency analyses, microsaccades were considered target-congruent, if the associated movement angle deviated from the direct connection between fixations spot and target by no more than $\pm 45^\circ$; microsaccades were considered target-incongruent, if the associated movement angle deviated by no less than $\pm 135^\circ$ from the fixation-target axis. Response times of trials with incongruent microsaccades in a given time window were subtracted from those with congruent ones. The resulting ΔSRT_{con} is plotted as a function of time to target onset in the lower left panel of Figure 6.9. Negative values indicate that response latencies were shorter after congruent microsaccades as compared to response times after incongruent microsaccades in the same time window. Controlling the expected proportion

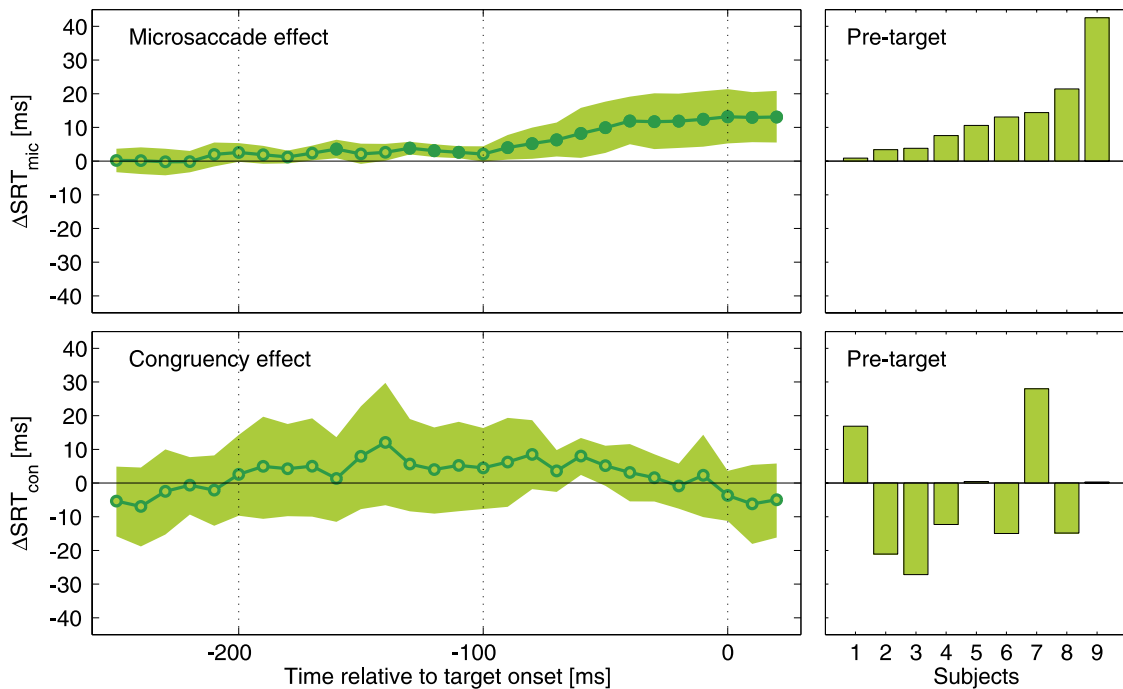


Figure 6.9: Microsaccade-induced modulations of saccade latencies in the gap task, collapsing all trials. In the upper left panel, mean modulations ΔSRT_{mic} are presented as a function of microsaccade (offset) events in different time windows (width: ± 50 , centered on the abscissa) relative to target onset. Positive ΔSRT_{mic} values depict costs induced by a microsaccade in a given time window. In the lower left panel, ΔSRT_{con} depict the influence of microsaccade-target congruency, by subtracting response times of trials with incongruent microsaccades in a given time window from those with congruent ones. Shaded areas are $CI_{95\%}$. Filled markers indicate significant deviations from zero after applying FDR (Benjamini & Hochberg, 1995). The panels on the right show individual ΔSRT_{mic} and ΔSRT_{con} , respectively, for the pre-target time window (-30 to 70 ms relative to target onset). Subjects were ordered by their ΔSRT_{mic} effect.

of erroneously rejected null-hypotheses with FDR (Benjamini & Hochberg, 1995), no significant deviations from zero were obtained. Note, however, that the dominant impression from that figure is that confidence intervals are way too large to interpret this data. The lower right panel of Figure 6.9 shows individual data for the pre-target time window. There was no systematic effect of microsaccade-target congruency on saccadic response latencies.

6.1.3 Discussion

We examined microsaccade statistics and their relation to saccadic performance in the gap task. First, we replicated classical patterns of saccadic performance. The introduction of a temporal gap between the offset of a fixation point and the onset of a peripheral target resulted in a strong

decrease of saccade latency, i.e., the gap effect, and a corresponding increase of express saccades. In addition, we found strong support for an interaction of microsaccades and saccades, advocating the idea that both types of movements are generated by mutually dependent processes. We would like to put forward the idea that microsaccades carry information about the current state of the saccadic motor map in which saccades are prepared during the gap period and executed in response to the onset of a target. In what follows, we will discuss what microsaccades convey about temporal and spatial aspects of saccade preparation. Further, we will derive potential mechanisms that, in the framework of our model, may trigger a microsaccade. Finally, we will link our results to earlier proposals concerning the relation between microsaccades and the gap effect.

6.1.3.1 Fixational disengagement and microsaccades

We showed that saccade latencies depend on the probability of preceding microsaccade occurrences. These results replicate and extend what has been shown in Chapter 5. First, we found a lower frequency of microsaccades before fast than before slow response times. Second, microsaccades occurring around the time of target onset, notably prolonged saccade latencies. These findings can well be explained by the model of microsaccade implementation proposed in Section 3.3. Generally speaking, high rates of microsaccades indicate a lower level of disengagement of activity at fixation-related sites of the saccadic motor map. As a consequence of mutual inhibition of remote locations within this motor map, saccades may only be generated when fixation-related activity has reached a sufficiently low level. The time courses of our effects suggest that the fixation system needs to be released 100 ms, or more, before the target comes on in order to generate a fast saccadic response. This is in line with several physiological and behavioral findings. First, the gap-related reduction of activity in FN in the rostral pole of the SC is achieved within 100 ms (Dorris et al., 1997). Second, the probability of a saccade evoked by electrical stimulation of FEF cells depends on the gap duration; a high probability was obtained for gap durations of 100 ms, or more, but not for shorter ones (Opris, Barborica, & Ferrera, 2001). Finally, as revealed by former studies (e.g., Krauzlis & Miles, 1996; Mayfrank et al., 1986) and, more crucially, by the present data, performers show an increased proportion of express saccades for gap durations of 100 ms, or longer. Based on these results, we suggest that microsaccade statistics carry temporal information about fixational disengagement and, thus, the potential level of saccade preparation.

6.1.3.2 Localized motor preparation and microsaccades

During the gap period, the preparation of a saccade is often accompanied by increased pre-target activity in saccade-related SC buildup neurons representing the potential target location (Dorris & Munoz, 1998; Dorris et al., 1997). We speculated that this activity, which would be located in rather peripheral parts of the saccadic motor map, could influence the spatial distribution of fixation-related activity in the central part of the motor map and, thus, influence the direction of microsaccades. However, we also emphasized that such an effect would not necessarily show up in microsaccade statistics in the gap task, first of all, since saccade preparation in the gap task comes at the cost of fixational disengagement (see Section 6.1.0.2, in the introduction of this experiment). The gap task strongly favors both motor preparation and fixational disengagement, reducing the potential to find an effect of motor preparation on microsaccade direction. Albeit, we found one indication of a systematic effect of microsaccade-target congruency on saccade latencies. We obtained a higher rate of target-incongruent microsaccades before slow responses in the 0 ms gap condition. In this condition, however, motor-preparation was assumed to be lowest. Of course, motor-preparation is also observed in the presence of a fixation target and biases in microsaccade direction in response to attentional cues (Engbert & Kliegl, 2003b; Galfano et al., 2004; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005) suggest that mechanisms involving motor preparation indeed have the chance to influence the distribution of microsaccade orientation. Our data indicate that this influence may in fact rely on a low level of fixational disengagement, even if motor-preparation is weaker at the same time.

6.1.3.3 Translation of fixation-related activity into microsaccades

There is strong evidence for a disengagement of fixation at the level of neuronal activity in the SC motor map. Dorris et al. (1997) reported that FN in the SC sharply decrease their firing rate about 100 ms after fixation offset while buildup neurons associated with the saccade-target location increase their activity antidromically. The minimum of FN activity was reached about 200 to 300 ms after fixation offset. Subsequently, as gap durations increased to 400 ms, or more, the activity level increased again, before abating to a plateau somewhat lower than the level that preceded the gap. But how might this neuronal activity translate into microsaccadic behavior? To be able to tackle this question on the basis of the present data, we used the same spatiotemporal layout of the gap paradigm as Dorris et al. After the offset of the fixation stimulus, microsaccade

rate decreased dramatically from a stable baseline level, reaching a minimum 130 ms into the gap. During a subsequent small enhancement period peaking at 280 ms, microsaccade rate remained below the baseline, before settling at a low rate for gap durations longer than 300 ms. Microsaccade amplitude also showed an initial decrease, time-locked to the inhibition in microsaccade rate. However, this decrease was short-lived. As soon as 130 ms after gap onset, mean microsaccade amplitude increased to its initial level.

Neither microsaccade rate, nor mean amplitude recover the exact time course of activity of FN in the monkey SC. In the face of the quite different sources of the two data sets, however, the qualitative agreement between the time course of FN activity and microsaccade statistics in the gap task is notable. Both consist of a baseline, a subsequent inhibition period, an enhancement epoch, and a final settlement at a stable level. So, if microsaccades are generated in the fixation-related part of a saccadic motor map, the question remains, what really triggers a microsaccade? Such a trigger mechanism could either be located within the saccadic motor map, e.g., in terms of a threshold mechanism, or outside the motor map, e.g., in the sense of an autonomous timer. In the latter case, microsaccade rate is independent of the temporal evolution of FN activity in the SC. However, we find a qualitative analogy between FN activity and microsaccade rate. This opens up the possibility that microsaccade frequency is dependent on activity in the motor map. We would like to discuss a mechanism triggering microsaccades conditional on supra-threshold activity in the saccadic motor map. A threshold model implies that if FN activity falls below a certain level, no more microsaccades will be produced. Indeed, the decrease of FN activity after gap onset may account for the break-down in microsaccade generation in the inhibition phase. Following this argumentation, the subsequent small enhancement period in microsaccade rate would result from increased neural activity found in FN cells in the SC. This activity then decreases again and may fall short of a critical threshold for microsaccade generation, compatible with a very low microsaccade rate following the enhancement period.

In addition to these considerations, an examination of microsaccade amplitude might help in understanding the processes involved in microsaccade generation. Our model holds that mean microsaccade amplitude is a function of the width of the fixation-related distribution of activity in the saccadic motor map. Moreover, we have argued that stimulus-elicited microsaccadic inhibition will be accompanied by a decrease in microsaccade amplitude, since more central parts in the movement field will be the last to be affected by a decrease of activity at fixation-related sites (see

Chapter 4). Indeed, we found a decrease in microsaccade amplitude, time-locked to microsaccadic inhibition. We have no evidence, however, for a decrease in microsaccade amplitude in a later time window, when microsaccade rate settles from a small enhancement epoch to a very low level. Assuming that the lack of this effect is not due to the low power in that time window, this may be interpreted in at least two ways in the framework of our model. First, the rate decrease might be related to top-down response preparation that was not represented in the SC recordings of Dorris et al. (1997). Indeed, there is evidence that the pure preparation of a response decreases the rate of microsaccades in human subjects, even in manual tasks (Betta & Turatto, 2006). On the one hand, this inhibition could be related to cortical inhibitory processes that are not reflected in SC activity, thus, directly impinging on saccade-related signals downstream the SC, e.g., via corticoreticular connections. On the other hand, human subjects could simply differ from monkeys with respect to the amount of top-down control in this task, slightly hampering the comparison of our microsaccade data and SC activity measured in the monkey brain, at least in this time window. Second, fixation-related activity may in fact be decreased at the level of the SC, but the remaining activity, aiming to prevent any premature saccades, is as broadly distributed as during visual fixation. In that case fewer microsaccades would be produced but the distribution of amplitudes would be the same.

Thus, in general, this data is compatible with the idea of a translation of FN activity into microsaccadic behavior. In fact, this hypothesis generates predictions for the dynamics of the spatial distribution of rostral activity during the gap task, which can be evaluated using physiological approaches.

6.1.3.4 Relationship between microsaccades and the gap effect

Yet in 1967, when Saslow published his original work on the gap effect, he speculated that the gap effect might be the consequence of microsaccade incidences during fixation. At that time, it was known from a study by Cornsweet (1956) that the absence of a fixation stimulus reduces the frequency of microsaccades. Saslow reasoned that microsaccades might incur refractory periods to saccade generation. A lower rate of microsaccades during the gap period could, thus, account for faster saccadic responses. However, Cornsweet's observations were made on a completely different time scale than that of the gap task; the author compared microsaccade rate with fixations stimulus present or absent over intervals of 45 seconds.

Indeed, we find microsaccades to be far less frequent in the gap condition (as compared to a 0 ms gap condition). However, a direct causal relationship between microsaccades and the gap effect was ruled out previously: Kingstone et al. (1995) examined the influence of microsaccades on saccade latencies in the gap task and showed that microsaccades are observed way too rarely to account for the very robust gap effect. We agree with this view. We propose, however, that microsaccades are correlates of fixation-related activity. In a way, this proposal parallels Saslow's original idea. The necessity to shut down fixation-related activity delays the generation of a saccade. Thus, according to our view, microsaccades do not impose a refractory period on saccade generation, but may only be observed when motor-preparation is less advanced.

6.2 Microsaccades and voluntary saccades: The anti-saccade task

Saccades to an appearing stimulus (pro-saccades) recover a phenomenon called the *visual grasp reflex* (Hess, Burgi, & Bucher, 1946; Ingle, 1973). In the anti-saccade task (Hallett, 1978), in contrast, participants are asked to inhibit the automatic response elicited by a suddenly appearing stimulus (imperative stimulus) and to generate a voluntary saccade to its mirror location instead (see Everling & Fischer, 1998, and Munoz & Everling, 2004, for reviews of the anti-saccade literature). Saccade latencies are usually much longer in the anti-saccade task as compared to pro-saccade performance and participants often fail to inhibit the reflexive pro-saccade. The number of pro-saccade errors in the anti-saccade task is even higher if the fixation stimulus is removed shortly before the appearance of the peripheral stimulus (gap condition).

As has been pointed out before, the SC figures prominently in the generation of reflexive pro-saccades. However, to generate an anti-saccade, movement-related signals from other brain areas such as the FEF are probably required (e.g., Everling, Dorris, Klein, & Munoz, 1999; Everling & Munoz, 2000). Nevertheless, the SC plays a role in the generation of anti-saccades and the physiological processes in the course of preparing and generating anti-saccades and pro-saccades, respectively, are well described at the level of the SC. We introduced two factors relevant to pro-saccade preparation in the gap task, fixational disengagement and localized motor preparation. These processes may also be distinguished in the course of the preparation of anti-saccades.

6.2.1 Fixational-related activity differs between pro- and anti-saccade tasks

Everling and colleagues (Everling et al., 1999; Everling, Dorris, & Munoz, 1998) recorded neural activity from cells in the intermediate layers of the SC while having monkeys perform randomly interleaved pro-saccade and anti-saccade trials. The color of the initial fixation marker instructed the current type of trial (instruction period). The neural activity recorded in both pro- and anti-saccade tasks with 200 ms gaps replicated the general pattern reported by Dorris et al. (1997) in the gap task, but involved several differences in firing rates of FN and buildup neurons, respectively, when pro- and anti-saccade tasks were compared. In the instruction period, FN had a higher activity during anti-saccade trials as compared to pro-saccade trials. In the gap period, this difference could no longer be shown. Evidently then, FN exhibited a stronger decrease in discharge rates during the gap period for anti-saccade trials than for pro-saccade trials. Buildup neurons, in turn, generally produced reciprocal firing patterns. During the instruction period, their activity was lower for anti-saccade than for pro-saccade trials. After fixation offset, buildup neurons continuously increased their low-frequency firing rate, but this increase was stronger in pro-saccade trials than in anti-saccade trials, indicating advanced response-preparation in the pro-saccade task.

Thus, fixation-related activity was higher in the anti-saccade task than in the pro-saccade task. Assuming a more or less direct translation of this activity to microsaccade rate, one would expect a higher rate of microsaccades in anti-saccade trials as compared to pro-saccade trials. This mechanism has received some support in the gap experiment reported earlier in this chapter, and examining microsaccade amplitude, was of additional help in understanding this relationship. In the anti-saccade task, however, a direct relationship between fixation activity and microsaccade rate might not be the whole story. The rationale is that the SC indeed plays a role in the generation of anti-saccades, but cortical areas must act in addition, first of all to inhibit reflexive pro-saccades (Everling et al., 1999; Everling & Munoz, 2000). These inhibitory processes need not necessarily show up in the activity of the SC motor map and could potentially also hinder microsaccade generation. Exploring the differences between microsaccade statistics in pro-saccade and anti-saccade tasks will shed further light on the processes relevant to the generation of microsaccades.

Some general predictions may be derived from what is known about the generation of pro- and anti-saccades, respectively. First, we expect to replicate our findings reported earlier in this chapter for pro-saccades in the gap task. Short-latency pro-saccades were associated with a lower rate of

preceding microsaccades than long-latency pro-saccades. As revealed by a trial-by-trial analysis, saccadic response latencies were prolonged when microsaccades are observed at the time of the onset of the imperative stimulus. In the anti-saccade task, in contrast, high levels of FN activity helped preventing erroneous pro-saccades (Everling, Dorris, & Munoz, 1998). Consequently, microsaccades, that are hypothesized to result from that activity, would play a distinct role in the generation of anti-saccades. First, anti-saccade trials with microsaccades occurring around the time of stimulus onset are thought to have lower rates of pro-saccade errors when compared with other anti-saccade trials. Second, given a correct anti-saccade is produced, its latency should depend on fixational disengagement in the same way as in pro-saccade generation. The sooner fixation is released, the faster the anti-saccade will be generated.

6.2.2 Motor-preparation activity in the pro- and the anti-saccade tasks

Performance in the anti-saccade task strongly depends on pre-stimulus activity in SC buildup neurons which represent either the stimulus location, or the saccade-target location. Specifically, correct anti-saccades are preceded by an enhanced level of pre-stimulus activity of neurons associated with the saccade-target location (Everling et al., 1999; Everling, Dorris, & Munoz, 1998). In contrast, the generation of erroneous pro-saccades has been associated with exceptionally high levels of pre-stimulus activity in buildup neurons at sites in the SC motor map where the visual stimulus was represented (Everling, Dorris, & Munoz, 1998). The authors of these studies used a combined gap-anti-saccade task (the fixation marker is removed prior to the onset of the peripheral stimulus) to increase the percentage of pro-saccade errors in anti-saccade trials. In this task, higher pre-stimulus activity before pro-saccade errors in the anti-saccade task were traced back to about 130–140 ms before stimulus onset (Everling, Dorris, & Munoz, 1998). Thus, in the combined gap-anti-saccade task assumptions concerning the spatial preparation of specific saccades can be made on the basis of the behavioral result, i.e., a correct anti-saccade or an erroneous pro-saccade. Following this idea, we propose that this task may be suited to reveal motor-preparation effects on microsaccade direction. Pro-saccade errors would be associated with higher rates of stimulus-congruent microsaccades than correct anti-saccades. The latter would rather be preceded by saccade-target-congruent microsaccades, indicating response preparation at the mirror location of the upcoming peripheral stimulus. Note that comparable predictions in the simple pro-saccade task could be substantiated only partially by the previous experiment. Potential reasons for this

have been discussed at length in Section 6.1.0.2. First of all, microsaccades may be much to rare at the time of stimulus onset, since saccade preparation is highly advanced in this period of a trial. Again, we have to let the data decide whether effects of microsaccade-target congruency on response latencies become evident.

In the present experiment we compared microsaccade statistics in randomly interleaved pro- and anti-saccade tasks. A temporal gap of 200 ms was introduced between the offset of the fixation stimulus and the onset of the imperative stimulus, to increase the likelihood of pro-saccade errors in the anti-saccade task. We adopted the spatial and temporal layout of the combined pro- and anti-saccade task used by Everling et al. (1999), to facilitate comparison of the two data sets.

6.2.3 Methods

6.2.3.1 Participants

Thirty students of the University of Potsdam were paid 7€ or received study credit for their participation. They were 19 to 45 years old (25.1 years on average), had normal or corrected-to-normal vision, and were in good health.

6.2.3.2 Experimental setup and eye-movement recording

Participants were seated in a silent and darkened room with the head positioned on a chin rest, 50 cm in front of a computer screen. Stimuli were presented on a 22-inch iiyama HM204DT CRT (1024 by 768 resolution or 46° by 34° of visual angle; refresh rate 100 Hz). The experiment was controlled by an Apple Power Macintosh G4 computer. Eye-position data were recorded and available on-line using an EyeLink-II system (SR Research, Osgoode, ON, Canada) with a sampling rate of 500 Hz and a noise-limited spatial resolution better than 0.01°. The experimental software controlling stimulus display and response collection was implemented in Matlab (MathWorks, Natick, Massachusetts, USA), using the Psychophysics (Brainard, 1997; Pelli, 1997) and Eyelink (Cornelissen et al., 2002) toolboxes.

6.2.3.3 Procedure

Participants performed 10 practice trials and 300 test trials of a simple saccade task. Half of the trials were pro-saccade trials, i.e., participants were instructed to make a saccade to an appearing

stimulus. In the other half of the trials, participants were required to make a saccade to the mirror location of the appearing stimulus, i.e., to perform an anti-saccade task (Hallett, 1978). Practice trials were comparable to test trials in all respects. Before the first and after every 30 trials the eye tracker was calibrated (standard 9-point grid) and calibration was validated. Every fifth trial, a drift correction was carried out. Before each trial, the fixation spot was displayed at the center of the computer screen. To start a trial, correct fixation was required. Otherwise, a drift correction was carried out and the trial was started over. If the eyes were still not detected within the critical area, the calibration was repeated.

Figure 6.10 illustrates a typical stimulation sequence in trials of the pro-/anti-saccade task. Participants fixated a small ring displayed centrally on a gray background; the color of the ring (red or green) instructed the type of response that had to be given. After a random interval of 500 to 1500 ms, the fixation spot was removed. Subsequently, an imperative stimulus appeared, 10° to the left or to the right in the visual periphery. Between the offset of the fixation spot and the onset of the stimulus, a 200 ms gap of no visual stimulation was introduced. Participants were required to hold fixation centrally until the imperative stimulus was displayed. Then, a speeded response saccade was required. Response saccades (eye position shift to either of the two target locations) were detected on-line and terminated the trial 200 ms after the eyes had passed a virtual border 2° away from the fixation spot. A trial ended after 800 ms of presentation of the imperative stimulus. Inter-trial intervals of 500 ms with no visual stimulation were introduced to enable blinking. If gaze position left a fixation square (4° side length, centered on the fixation spot) while fixation was still required (i.e., before the onset of the imperative stimulus), an error feedback was triggered and the trial was aborted. Aborted trials were repeated in random order after the 300 regular trials.

Target locations (left or right) and trial types (pro-saccade or anti-saccade) were randomly interleaved across the 300 trials. Participants were randomly assigned to one of two groups. In one group a red fixation spot was associated with an anti-saccade task and green with a pro-saccade task. In the other group the instructed association was vice versa.

6.2.3.4 Stimuli

The fixation spot was a ring with a diameter of 0.67° of visual angle in dark red or green color and an inset with a diameter of 0.13° . Red and green fixation spots were identical in form and adjusted

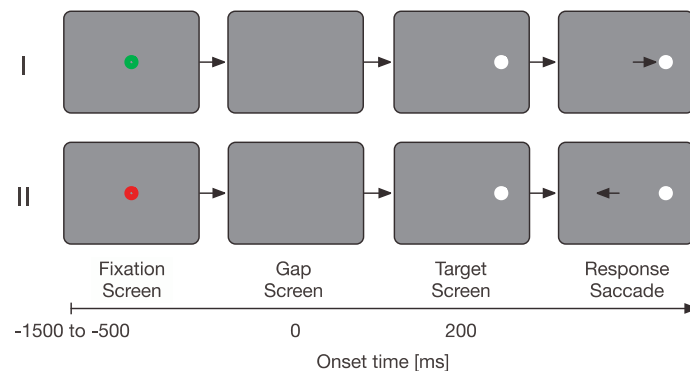


Figure 6.10: Sequence of visual stimulation in the pro-/anti-saccade paradigm. The color of the initial fixation spot instructed the required response. The assignment of red and green to the pro- and anti-saccade tasks was counterbalanced across participants. In this example, a green spot indicated a pro-saccade trial (I) and a red spot indicated an anti-saccade trial (II).

in luminance using a flicker-fusion method. That is, before an experimental session, participants were instructed to minimize the flickering of two colored spots (red and green) alternating with 20 Hz by adjusting the luminance of the green spot.

The imperative stimulus was a white circle (diameter: 0.67°). The error feedback, triggered by blinks and anticipatory saccades, was a binaural 660 Hz tone at about 70 dbA, played for 82 ms via the internal speakers of the G4 computer.

6.2.3.5 Data preparation

For data analysis, a post-hoc saccade detection was performed using a new version (Engbert, 2006b) of the algorithm developed by Engbert and Kliegl (2003b). Velocities were computed from subsequent samples in the series of eye positions in the response time window 500 ms on from the go signal. Saccades were detected in 2D velocity space using thresholds for peak velocity and minimum duration. We used a relative threshold of 6 SDs of the velocity and a minimal duration of 6 ms (or three data samples). The first saccade that shifted gaze to the center of a target location $\pm 8^\circ$ was taken as a response saccade. Saccadic reaction time was defined as the latency between stimulus and saccade onsets.

Subsequently, we used the same algorithm to detect microsaccades (amplitude $< 1^\circ$) in the interval from fixation onset to the response saccade. As in the previous experiments, we considered only binocular microsaccades.

Trials including saccades larger than 1° prior to the response saccade were discarded, as were trials with SRTs shorter than 70 ms. Some trials had to be excluded due to data loss during eye-movement recording. Thirty participants contributed 151 to 297 trials to the final data analyses, resulting in a total of 8044 trials (out of 9000 or 89.4%) in which 18329 microsaccades were detected.

6.2.3.6 Data analysis

Where provided, confidence intervals were computed using a simple bootstrapping technique (Efron & Tibshirani, 1993). From an original sample of N values, 1000 bootstrap samples were generated, each by selecting (with replacement) N values of the original sample. The 1.96-fold of the standard deviation of the means of these 1000 bootstrap samples was computed to generate 95% confidence intervals ($CI_{95\%}$) of the mean of the original sample. To allow the reader to compare different conditions, we removed between-subject variance in figures containing within-subject comparisons; these confidence intervals will be labeled $CI_{95\%ws}$.

6.2.4 Results

6.2.4.1 Performance in the task

Figure 6.11 shows the average saccadic response-latency distributions for both pro-saccade and anti-saccade trials. Distributions for correctly directed saccade (targeting the instructed location) are plotted upwards, distributions for direction errors plotted downwards. Dashed white lines indicate the border of 130 ms below which saccades are traditionally counted as express saccades (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). Two important observations can be made. First, correct anti-saccades are initiated later than correct pro-saccades; $t(29) = 16.58, p < 0.001$ (paired t -test). Second, direction errors are confined to the anti-saccade task and the latency of these erroneous pro-saccades is much shorter than the latency of correct anti-saccades; $t(29) = 10.79, p < 0.001$ (paired t -test). In fact, these direction errors were generated about as fast as correct pro-saccades; $t(29) = 1.53, p = 0.14$ (paired t -test).

6.2.4.2 Microsaccade rate

Microsaccade rates were computed as described in section 4.1.6. The upper panels of Figure 6.12 show the evolution of the microsaccade rate averaged over participants and time-locked to gap

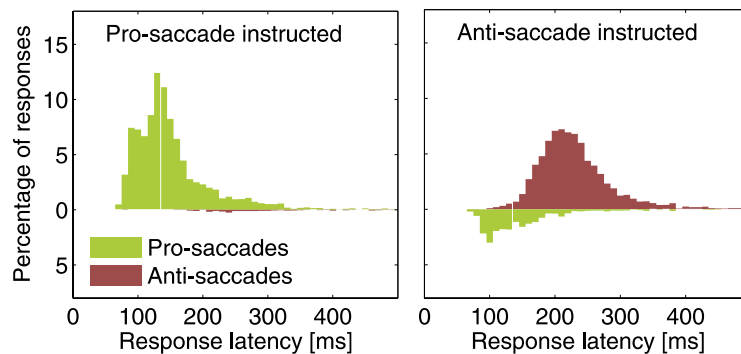


Figure 6.11: Average response-latency distributions for correctly (plotted upwards) and incorrectly (plotted downwards) directed saccades in the pro- and anti-saccade conditions. Dotted white lines indicate the border of 130 ms below which saccades are usually counted as express saccades (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). Bin width was set to 10 ms.

onset (left panel) and saccade onset (right panel), respectively, for three different types of responses: correct pro-saccades, correct anti-saccades, and pro-saccade errors in the anti-saccade task. In general, a sharp decrease in microsaccade rate followed the offset of the fixation spot, regardless of type of trial. Rates remained on a very low level throughout the remainder of all types of trials. The upper two panels in Figure 6.13 show an analysis testing for changes in microsaccade rate, contingent on gap onset. In time windows of 50 ms width centered on the x-axis, the mean deviation of microsaccade rate from a baseline time window (-50 to 0 ms) are shown with associated error bars representing $CI_{95\%}$ for these multiple comparisons. Filled dots indicate significant differences after applying FDR (Benjamini & Hochberg, 1995). Clearly, microsaccade rate decreases about 100 ms after the offset of the fixation spot. This effect is independent of whether correct pro-saccade trials or correct anti-saccade trials were considered.

Differences in rates for the three trial types were compared by computing their confidence intervals in 100 ms time windows. Mean rate differences and corresponding $CI_{95\%}$ for tests against zero are shown in the six lower panels of Figure 6.12. Alpha inflation was prevented by using the FDR procedure (Benjamini & Hochberg, 1995); significant deviations from zero are highlighted by filled dots. Microsaccade rate before correct anti-saccades differed from rate before correct pro-saccades. In the pro-saccade task, participants exhibited a higher microsaccade rate than in correct anti-saccade trials. This effect was observed over a long period. It was more pronounced for the saccade-locked analysis, where significant deviations were observed from -570 ± 50 ms to -230 ± 50 ms. In the stimulus-locked analysis, this effect surrounded the time of

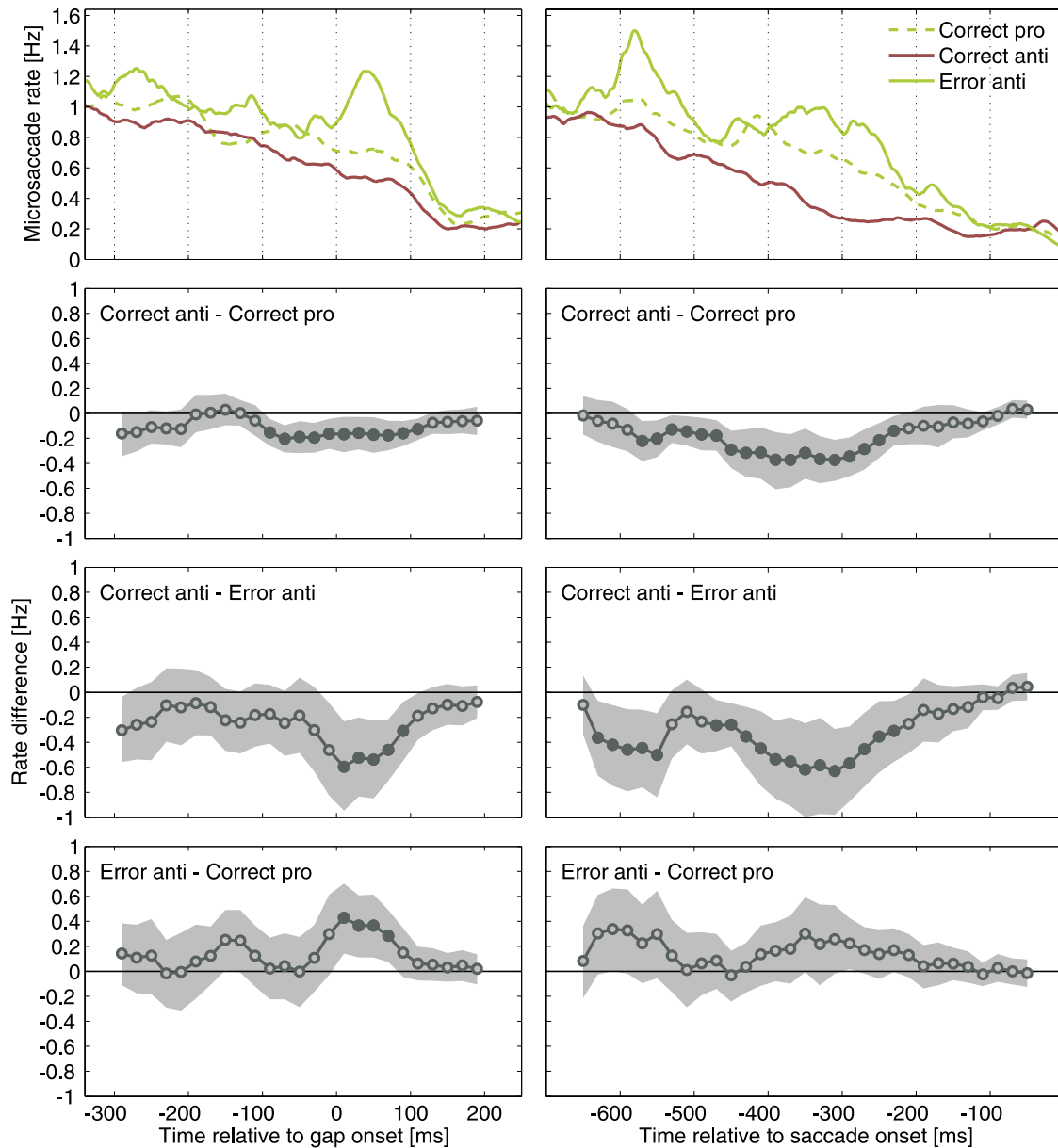


Figure 6.12: Microsaccade rate as a function of time from gap onset (left panels) and time to saccade onset (right panels) for three types of responses: correct pro-saccades, correct anti-saccades, and erroneous pro-saccades in the anti-saccade task. The lower six panels display tests for differences between rates in the different response conditions. Time windows of ± 50 ms were used. Error bands are $CI_{95\%}$. Filled dots indicate time windows in which deviations were significant after controlling the alpha level using the FDR procedure (Benjamini & Hochberg, 1995).

fixation offset (about -100 ± 50 to 100 ± 50 ms, relative to gap onset). The same pattern of results was obtained for the comparison of correct anti-saccades with pro-saccade errors in the anti-saccade

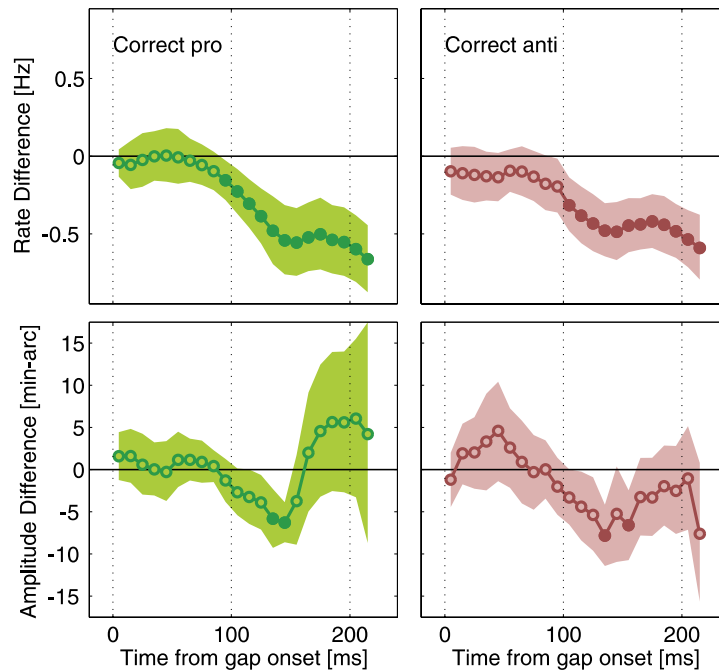


Figure 6.13: Gap-contingent differences of mean microsaccade rates (upper panels) and amplitudes (lower panels) for correct pro- and anti-saccades. Error bands are $CI_{95\%}$ for the differences of rates and amplitudes, respectively, to a baseline time window (-50 to 0 ms). Filled dots indicate significant differences after applying FDR (Benjamini & Hochberg, 1995).

task (third line of panels in Figure 6.12). Here, rates differed reliably in time windows between -610 ± 50 ms to -230 ± 50 ms before saccade onset and from about 0 ± 50 ms to 100 ± 50 ms, relative to gap onset. In the saccade-locked analyses, microsaccade rates before erroneous pro-saccades in anti-saccade trials could not be distinguished from those before correct pro-saccades (lowest two panels of Figure 6.12); in the stimulus-locked analyses, rates were higher before erroneous than before correct pro-saccades; significant deviations were found in time windows of 0 ± 50 ms to 80 ± 50 ms, relative to gap. All rates continuously declined and were nearly zero just before the response saccade.

6.2.4.3 Microsaccade amplitude

The upper panels of Figure 6.14 show the evolution of microsaccade amplitude time-locked to gap onset (left panel) and saccade onset (right panel), respectively, for correct pro- and anti-saccades as well as prosaccade errors in the anti-saccade task. Mean amplitudes were determined using a 100 ms moving boxcar window centered on the x-axis. The six panels below test for significant

deviations between amplitudes in the different conditions. These analyses were analogous to those applied to microsaccade rates (see above). Amplitude evolutions for the three types of responses could not be distinguished in the stimulus-locked analyses. Before gap onset, mean microsaccade amplitude was at a stable level. About 100 ms after gap onset, mean amplitude declined slightly. A strong decrease in mean microsaccade amplitude, however, could be observed in the saccade-locked analyses. This was true for all types of responses, but the decrease started earliest in correct anti-saccade trials. In fact, the mean microsaccade amplitude was significantly shorter in correct anti-saccade trials than in correct pro-saccade trials in all time windows from -330 ± 50 ms to -290 ± 50 ms aligned on the initiation of the saccade. Microsaccade-amplitude evolution in erroneous pro-saccades appeared to follow the curve for correct pro-saccades; both curves could not be distinguished (see lowest panels of Figure 6.14). Mean microsaccade amplitudes were slightly higher before correct anti-saccades than before erroneous pro-saccades in an early time window (-130 ± 50 to 90 ± 50 ms before gap onset).

Gap-contingent changes in microsaccade amplitudes were determined for correct pro-saccades and correct anti-saccades, respectively. The lower row of panels in Figure 6.13 display this analysis. In time windows of 50 ms width centered on the x-axis, the mean deviation of microsaccade rate from a baseline time window (-50 to 0 ms) are shown with associated error bands, representing $CI_{95\%}$ for these comparisons. Filled dots indicate significant differences after controlling for Type-I-error inflation using FDR (Benjamini & Hochberg, 1995). Mean microsaccade amplitude decreased in response to gap onset; in both conditions, the deviation was significant about 140 \pm 50 ms after fixation offset.

6.2.4.4 Relationship between microsaccades and saccadic responses

In the gap experiment (first experiment in this chapter) we showed that microsaccades, when occurring shortly before a pro-saccade is required, will delay the generation of this subsequent response saccade. We expect to replicate this finding in the pro-saccade task employed here. Given, an anti-saccade was generated successfully, similar costs for the generation of a response were hypothesized. To test these hypotheses, we conducted three analyses, comparable to those applied in the gap experiment reported earlier.

First, we conducted a saccade-latency based microsaccade-rate analysis. Individual response latencies were centered for each subject by removing the average overall response time from the

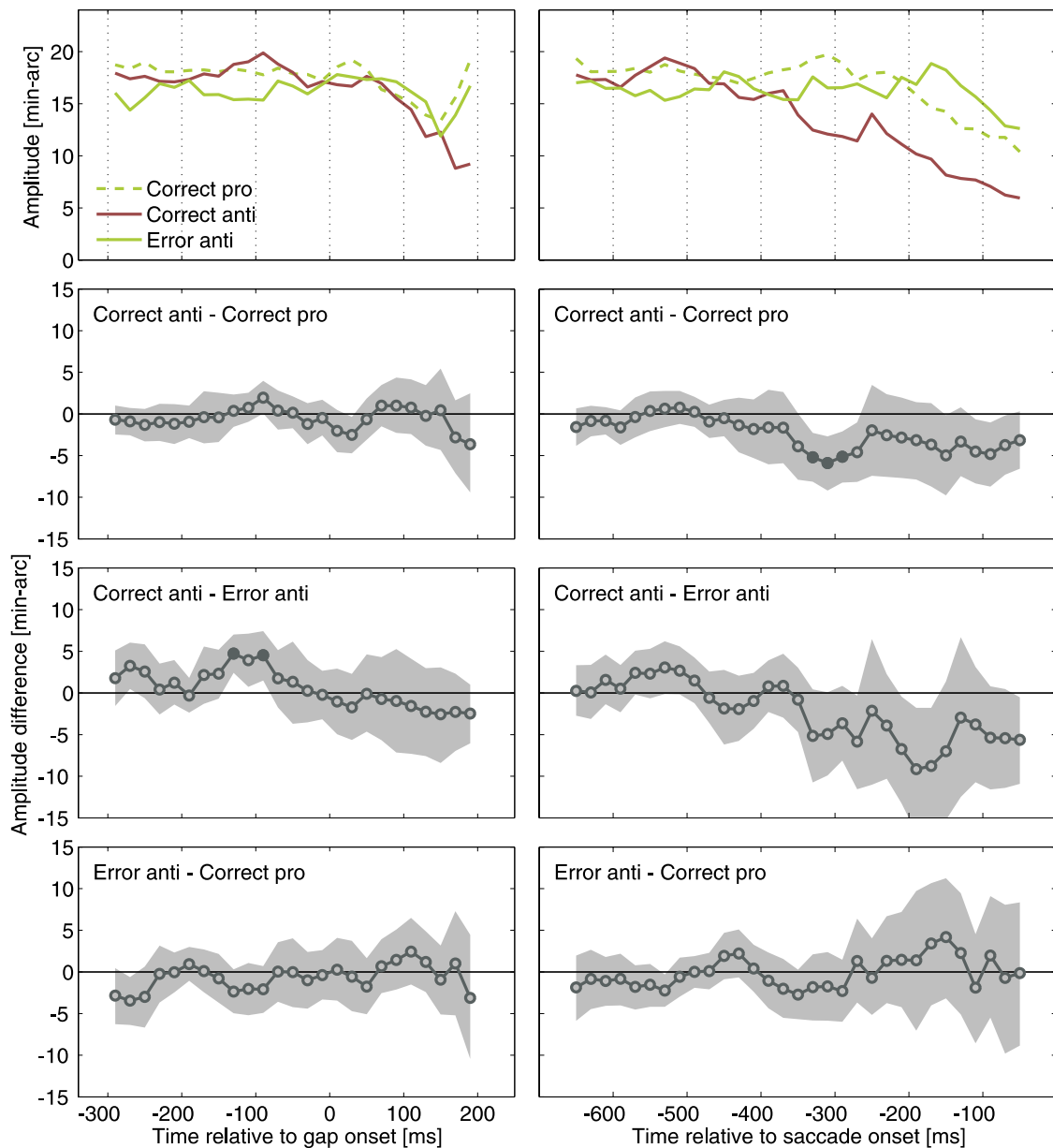


Figure 6.14: Microsaccade amplitude as a function of time to stimulus onset (left panels) and time to saccade onset (right panels) for three types of responses: correct pro-saccades, correct anti-saccades, and erroneous pro-saccades in the anti-saccade task. The lower six panels display tests for differences between amplitudes in the different response conditions. Time windows of ± 50 ms were used. Error bands are $CI_{95\%}$. Filled dots indicate time windows in which deviations were significant after controlling the alpha level using the FDR procedure (Benjamini & Hochberg, 1995).

latency of single trials. Subsequently, we determined quantiles of response latencies for both correct pro-saccades and correct anti-saccades on the basis of all a participant's responses. Finally,

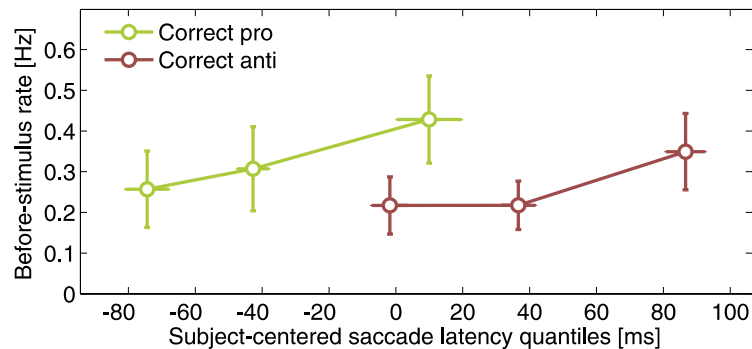


Figure 6.15: Before-stimulus microsaccade rate for correct pro-saccades and correct anti-saccades as a function of subject-centered response-time quantiles (means are displayed). Error bars are $CI_{95\%}$.

we computed individual microsaccade rates in the pre-stimulus time window for trials assigned to each of these quantiles. Figure 6.15 depicts mean rates of microsaccades (and $CI_{95\%}$) in the pre-stimulus time window (-130 to 70 ms relative to stimulus onset)³ as a function of saccade latency and response type. The response-time quantiles are displayed as their mean latency averaged across subjects (plus $CI_{95\%}$). For both types of responses, microsaccade rates were higher before long than before short saccade latencies. This finding is also represented in the result of an ANOVA using saccade-latency quantile and response type as independent variables. A main effect of response-time quantile ($F[2, 58] = 4.15, p = 0.021$) came in the absence of an interaction with response type; $F < 1$. Moreover, the analyses yielded no main effect of response type on microsaccade rate; $F(1, 29) = 2.24, p = 0.15$.

Second, we computed the costs that microsaccades imposed on saccade latencies on a trial-by-trial basis. We based our analysis on those reported earlier in the gap task. In a first step, we computed individual mean SRTs given that either a microsaccade occurred or no microsaccade was observed in a pre-target time window (-30 to 70 ms relative to stimulus onset). For the resulting response latencies, we conducted a two-by-two repeated-measures ANOVA with microsaccade presence (present or absent) and response condition (correct pro- or correct anti-saccades) as independent variables. Only 14 out of 30 participants had an entry in each cell of the design. For these subjects, we replicated that pro-saccades had a shorter latency than anti-saccades, i.e., a main effect of condition; $F(1, 13) = 122.92, p < 0.001$. Surprisingly, there was no evidence for a

³In previous experiments we used pre-target time windows with a width of 100 ms. Here, we chose a width of 200 ms, since some of the most relevant analyses by Everling et al. (1999) were conducted in this time window.

pre-saccadic slowing, i.e., there was no main effect of microsaccade presence; $F(1, 13) = 0.23, p = 0.64$. Finally, the interaction term crossing microsaccade presence with condition did not yield significant effects; $F(1, 13) = 2.92, p = 0.11$. To gain statistical power (by decreasing the number of empty cells), additional t -tests were conducted for each response type separately. These tests revealed that microsaccade presence affected saccade latencies for correct pro-saccade responses ($t[21] = 2.45, p = 0.023$), but not for correct anti-saccades; $t(16) = -0.38, p = 0.71$.

To examine the temporal extension of the pre-saccadic slowing in the pro-saccade task and to determine whether there was an effect of microsaccade presence in other time windows before correct anti-saccades, we subtracted individual mean response latencies given a microsaccade occurred in a certain time window (width of 100 ms) from individual mean response latencies given no microsaccade was observed in that time window, resulting in ΔSRT_{mic} . This was done across a broad temporal range reaching back from 300 ms before gap onset to stimulus onset. Mean benefits (negative values) and costs (positive values) in saccade latencies, embedded in $CI_{95\%}$, are displayed in Figure 6.16. Filled markers indicate that zero was not included in the confidence interval. For both correct pro-saccade and correct anti-saccade trials, we observed pre-saccadic slowing effects over a long period before stimulus onset. However, while costs in pro-saccade latencies were largest for microsaccades occurring in the pre-target time window, similar costs vanished in the time window -80 ± 50 ms before stimulus onset, in the correct anti-saccade condition.

6.2.4.5 Microsaccade-target congruency

Everling, Dorris, and Munoz (1998) showed that preparatory saccade-related activity was particularly strong in SC buildup neurons before erroneous pro-saccades in the anti-saccade task. Before these errors, buildup neurons with a response field representing the location of the visual stimulus exhibited significantly stronger activity (than in correct trials) from 130 ms before the onset of the stimulus. To test the hypothesis that microsaccade direction is an indicator of these motor-preparation signals in the saccadic motor map, we computed the proportion of target-congruent microsaccades in two time windows, a baseline period (last 200 ms before the gap) and a pre-target period (-130 to 70 ms relative to stimulus onset). A surplus of target-incongruent microsaccades before pro-saccade errors as compared to correct anti-saccades would substantiate the idea of a positive correlation between saccade preparation and microsaccade direction. In our

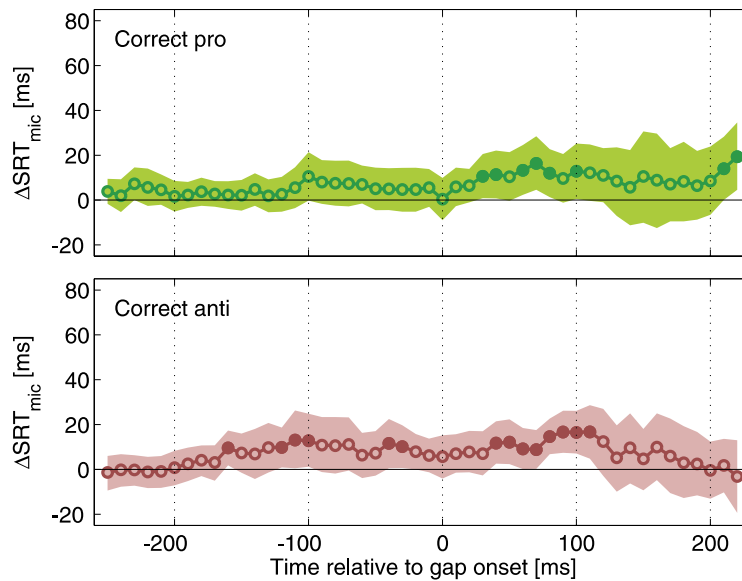


Figure 6.16: Microsaccade-induced modulations of saccade latencies in the interleaved pro-/anti-saccade task. Results are shown for correct pro-saccades (upper panel) and correct anti-saccades (lower panel). Mean modulations ΔSRT_{mic} are presented as a function of microsaccade (offset) events in different time windows (width: ± 50 , centered on the abscissa) relative to gap onset. Positive ΔSRT_{mic} values depict costs induced by a microsaccade in a given time window. The imperative stimulus set on at 200 ms. Shaded areas represent $CI_{95\%}$; filled markers indicate that zero was not included in the confidence interval.

analyses, microsaccades were considered target-congruent, if their direction deviated from the direct connection between the fixation spot and the instructed saccade-target location by no more than $\pm 45^\circ$; the angle of incongruent microsaccades deviated by no less than $\pm 135^\circ$ from this axis.

The left panel of Figure 6.17 displays the mean fraction (and $CI_{95\%tw}$) of target-congruent microsaccades before both correct anti-saccades and erroneous pro-saccades in the two time windows. Proportions greater than 0.5 indicate that more target-congruent microsaccades were observed; proportions below 0.5 indicate that more target-incongruent microsaccades were observed. Note that if for a subject no microsaccades were detected in a time window, the corresponding proportion was set to 0.5. Out of the 30 participants in this experiment, this was the case for 1 subject (correct anti-saccade condition) and 9 subjects (erroneous pro-saccade condition) in the baseline time window and, respectively, for 8 subjects (correct anti-saccade condition) and 16 subjects (erroneous pro-saccade condition) in the pre-stimulus time window. There appeared to be a small bias in favor of target-incongruent microsaccades before erroneous pro-saccades. In an ANOVA using time window and response type as independent variables, no significant

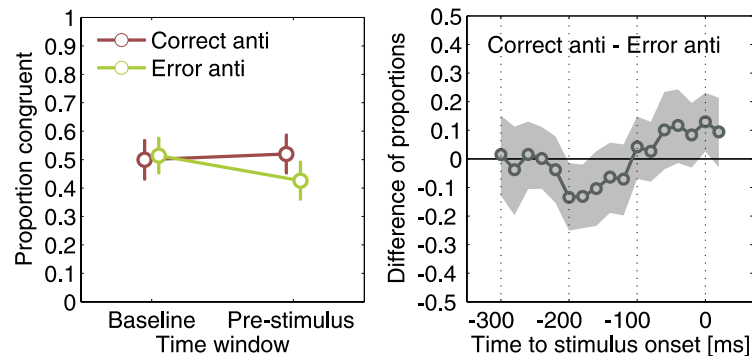


Figure 6.17: Proportion of microsaccades congruent with the instructed saccade target, computed for correct responses and errors in the antisaccade task, respectively. In the left panel, two time windows are compared, a baseline time window (last 200 ms before the gap) and a pre-stimulus time window (-130 to 70 ms relative to stimulus onset). Error bars are $CI_{95\%ws}$. In the right panel, the difference of proportions between correct anti-saccades and erroneous pro-saccades are presented across time, each dot displayed at the center of a 100 ms time window. Positive differences indicate a stronger microsaccade-target congruency before correct anti-saccades. The shaded area represents $CI_{95\%}$.

interaction was obtained; $F(1, 29) = 2.04, p = 0.16$. Merely a one-sided paired t -test, comparing the congruency proportions for the two response types in the pre-stimulus time window was significant; $t(29) = 1.70, p = 0.0496$. Finally, a one-sided paired t -test comparing the proportion of congruent microsaccades before pro-saccade errors to 0.5, was marginally significant ($t[29] = -1.69, p = 0.0505$), while the proportion of congruent microsaccades before correct anti-saccades was probably not different from 0.5; $t(29) = 0.48, p = 0.64$.

For the right panel of Figure 6.17, the difference between congruency proportions for the two response types was computed as a function of time to stimulus onset. Time windows of 100 ms centered at the x-axis were used to this end. Negative differences indicate higher microsaccade-target congruency before pro-saccade errors than before correct anti-saccades; positive values indicate weaker microsaccade-target-congruency before pro-saccade errors than before correct anti-saccades. Indeed, the $CI_{95\%}$ (shaded area) do not include zero in all time windows tested. At times around the onset of the imperative stimulus, stronger incongruency was observed for erroneous pro-saccade responses as compared to correct anti-saccades. The reverse pattern was observed at times around gap onset. None of these differences, however, survived the control of the alpha-level using the FDR procedure (Benjamini & Hochberg, 1995). We also checked whether the early deviation, which was strongest in the time window of 180 ± 50 ms before stimulus

onset, was due to differences between the two proportions, or whether it could be traced back to one response type only. Congruency proportions before neither response type could reliably distinguished from 0.5 in this time window. If anything, there was a bias towards more target-incongruent microsaccades before erroneous pro-saccades; $t(29) = 1.59, p = 0.12$ (two-sided t -test for paired samples). There was no evidence that the proportion of target-congruent microsaccades was different from 0.5 for the correct anti-saccade trials; $t(29) = 1.06, p = 0.30$.

6.2.5 Discussion

In a saccade task composed of interleaved pro- and anti-saccade trials, we aimed to manipulate the activity in the saccadic motor map that we proposed to be involved in the generation of both microsaccades and saccades. We obtained the classical pattern of saccadic performance; correct anti-saccades had a shorter latency than pro-saccades, and pro-saccade errors in the anti-saccade task were observed frequently. More crucially, the experimental manipulation differentially affected microsaccadic behavior in the two tasks. In part, these results contradicted our expectations. In what follows we will discuss our findings in detail and try to integrate them into our understanding of microsaccade and saccade generation. Again, temporal and spatial aspects of these processes will be considered separately.

6.2.5.1 Microsaccade statistics during fixational disengagement before pro- and anti-saccades

A primarily temporal aspect of saccade preparation is the process of fixational disengagement, i.e., the release of activity in the fixate system, thereby allowing for a saccade to be generated. The gap period preceding the onset of the imperative stimulus in the present experiment is known to strongly favor fixational disengagement. To determine behavioral correlates of fixational disengagement, we analyzed microsaccade rate and amplitude evolutions as a function of time from gap onset. From a typical baseline level of about one per second, microsaccade rate decreased sharply about 100 ms after the offset of the fixation spot, independent of the type of response to be generated. For the remainder of a trial, it settled on a minimal level. In a similar analysis of the evolution of the mean amplitude of microsaccades, a transient decrease in response to gap onset was detected. These findings closely resemble our results from the gap experiment and are in good agreement with the hypothesis that fixational disengagement is accompanied by a sharp reduction of the generation of microsaccades.

We hypothesized that microsaccades are the behavioral correlate of activity in fixation-related sites in the saccadic motor map. Using the same task as that employed here, Everling et al. (1999) indeed reported a strong decrease in the activity of neurons in the rostral pole of the SC motor map. These FN steadily fired during fixation, but firing dropped out about 100 ms after gap onset. We propose that as soon as this activity falls below a certain threshold, microsaccades will no longer be generated. The time course of microsaccadic inhibition in the present data is in good agreement with this hypothesis.

The proposed relationship between microsaccadic inhibition and fixational disengagement was further substantiated by showing that before both correct anti-saccades and correct pro-saccades, very low rates of microsaccades preceded the fastest responses. In turn, long saccade latencies were associated with high pre-stimulus microsaccade rates. We suggest that on the occasions of fast responses, fixational disengagement was exceptionally well accomplished by the time of the onset of the imperative stimulus. However, in a complementary analysis this line of argumentation was supported only for pro-saccade responses: Microsaccades occurring around the time of the go-signal, delayed the subsequent saccadic response. Microsaccades occurring at the time when the imperative stimulus was presented induced significantly fewer costs on anti-saccade latency. While there were costs relating to microsaccades occurring in earlier time windows, in fact, no costs could be observed for that condition just before stimulus onset. We have argued that high levels of fixational activity, as potentially reflected in microsaccade occurrences, could prevent pro-saccade errors in the anti-saccade task. However, given the oculomotor system has accomplished generating a correct anti-saccade response, the latency of this response should benefit from fixational disengagement. We may only speculate why this has not been the case for anti-saccade trials in the present set of data and offer two potential accounts. First, mean amplitudes of microsaccades were smaller before correct anti-saccades in the pre-target time window than before pro-saccade errors in anti-saccade trials. Though our confidence in this result must be limited (the difference was not significant), it may account for the lower impact of microsaccades on response latencies, since this impact is positively correlated with microsaccade amplitude (see Chapter 5). Alternatively, the lack of an influence of microsaccades may be related to the proposal that the signals relayed by the SC are not the most critical ones in the generation of anti-saccades (Everling et al., 1999). The latter account, however, may not conclusively explain why there were costs to response latencies for microsaccades observed in earlier time windows.

One suggestion would be that until an influence is exerted by high-level areas, microsaccades are an indicator fixation-related activity, indeed; with aging pre-stimulus periods, however, high-level influences will become stronger in order to prevent pro-saccade errors.

Due to the distinct mutual inhibitory network within the saccadic motor map, fixational disengagement will reach its maximum level when a saccade is about to be generated. Consequently, shortly before the onset of a saccade, virtually no microsaccades should be observed. Our results lend strong support to this hypothesis. In addition, the very few microsaccades that were observed just before the response saccade had very short mean amplitudes, suggesting that they were generated at a low level of fixation-related activity (see Chapter 4).

The main share of findings reported so far were common to all types of trials, regardless of whether pro-saccades or anti-saccades were generated. However, there were also clear differences in microsaccade statistics between the conditions. To understand these findings, please recall that we distinguished three types of responses: correct pro-saccades, correct anti-saccades, and pro-saccade errors in the anti-saccade task. In the latter case, participants first looked at the imperative peripheral stimulus, instead of moving to its mirror location. These erroneous pro-saccades had latencies that were comparable to those of correct pro-saccades. Our data indicate, in addition, that both microsaccade-rate and -amplitude evolutions evolve in a similar pattern for these two types of responses. That is, the time courses of microsaccade rate and amplitude were a function of the *generated* response, not of the *instructed* response. Microsaccade statistics before pro-saccade errors in the anti-saccade task were different from those associated with correct anti-saccades, but could not be distinguished from microsaccade statistics in the pro-saccade task. We suggest that the oculomotor system was actually prepared to produce a pro-saccade in these instances and, as a consequence, failed to generate a correct anti-saccade. This indicates that the generation of both correct pro-saccades and erroneous pro-saccades in the anti-saccade task is associated with a lower level of fixation-related activity. For the activity of FN in the SC, this has been shown for correct pro-saccades (Everling et al., 1999). For errors in the anti-saccade task, a decreased fixation activity may be inferred from the fact that saccade-related neurons exhibited high levels of activity when monkeys failed to inhibit reflexive pro-saccades (Everling, Dorris, & Munoz, 1998).

6.2.5.2 Differences in microsaccade statistics before pro- and anti-saccades

Our current model of microsaccade generation holds that both types of eye movements microsaccades and saccades are encoded in and generated by a common movement field. This model predicts that microsaccade generation relies on supra-threshold activity in the central part of the field, which is active during fixation. However, the exact nature of the trigger mechanism have not yet been determined.

A physiological counterpart of the saccadic movement field is the SC motor map; the processes therein are likely relevant as to how activity in the field translates to microsaccadic behavior. One reason to conduct the current study was to determine whether microsaccade rates replicate the differential patterns of FN activity in the SC motor map that were described by Everling et al. (1999). In their single-cell studies of the monkey SC, the authors reported lower activity of FN for pro-saccades than for anti-saccades, during the instruction period. Microsaccade rates in the present study, in contrast, were significantly higher before pro-saccades than before anti-saccades, in a comparable time window. Thus, microsaccade rates were indeed sensitive to the experimental manipulation, however, in a manner completely opposite to what would be expected assuming a direct translation of fixation-related activity to microsaccadic behavior. Notably, while there clearly was a difference in microsaccade rates during the instruction period, differential evolutions of mean microsaccade amplitudes contingent on the instructed response could not be observed.⁴ Consequently, microsaccade rate may be interpreted directly.

We assumed that microsaccades are the behavioral correlate of fixation-related activity of neurons in the rostral SC. But then, how can we explain the obvious difference between microsaccades generation and FN activity? Several accounts may be offered, first of all, depending on the trigger mechanism underlying the generation of a microsaccade. A first explanation relates to differences in the implementation of pro- and anti-saccade generation in the brain circuitry of oculomotor control. The SC is highly involved in the generation of pro-saccades, but its function in the generation of anti-saccades is limited, as noted by Everling et al. (1999). Rather, the frontocortical areas involved in oculomotor control figure prominently in the prevention of a reflexive pro-saccade and the generation of a correct response in the anti-saccade task (e.g., Everling & Munoz, 2000). Direct connections between the frontocortical network (e.g., the FEF) and the saccadic burst generator

⁴Remember that microsaccade amplitude was a second measure hypothesized to be sensitive to the distribution of fixation-related activity (see Chapter 4).

circuit in the brainstem may bypass the SC (see also Forbes & Klein, 1996) and directly inhibit saccadic eye movements (see Munoz & Everling, 2004, and Schall, 1997, for reviews). In the case of the anti-saccade task, this inhibition may prevent reflexive pro-saccades on the one side, but it will also inhibit the generation of microsaccades. Consequently, a lower microsaccade rate may result before correct anti-saccades as compared to pro-saccade errors in the anti-saccade task and correct pro-saccades, respectively. This argument is also compatible with the finding that in a comparison of correct anti-saccade trials to pro-saccade trials microsaccade rates (and amplitudes to some extent) began to diverge more than 500 ms ahead of the response saccade; voluntary control may have affected saccade generation as soon the type of trial was known. Thus, according to the explanation stressed here, activity in the rostral pole of the SC and thus the potential rate of microsaccades may well be greater while preparing for an anti-saccade task (as shown by Everling et al., 1999), however, inhibitory mechanisms impinging on the signal downstream the SC motor map would prevent microsaccades from being produced. Unfortunately, a direct test of this hypothesis may only be achieved by the means of physiology, and will, thus, not be approached in the current work.

An alternative to the mechanism proposed above, a lower microsaccade rate may result before anti-saccades, if an external timing mechanism rather than one depending on the current distribution of activity of the saccadic motor map controls the generation of saccades. Consider, for instance, an autonomous timer for the generation of saccades (e.g., Richter et al., submitted). The anti-saccade task could be associated with a slowed timer resulting in fewer microsaccades per unit of time as compared to the pro-saccade task. This explanation for our results is, however, very speculative; we are not aware of any data substantiating a systematic relationship between timing mechanisms and the generation of reflexive and voluntary saccades, respectively.

Third, it may be the case that microsaccade rate in the pro- and/or anti-saccade tasks is subject to training effects. Participants in the present study were not trained on saccade paradigms, nor were they familiar with the given task. This strongly contrasts with the monkey subjects used by Everling et al., who were prepared for chronic experiments and certainly trained amply to reliably perform in the combined pro-/anti-saccade task. Indeed, performance in both the pro- and anti-saccade task should benefit from practice. Faster responses rely on higher levels of fixational disengagement and may, thus, be associated with lower rates of microsaccades. Since high levels of fixational disengagement likely result in pro-saccade errors in the anti-saccade task,

a decrease in microsaccade rate with progressing practice should first of all apply to pro-saccade generation. The hypothesis that the difference in microsaccade rate between pro- and anti-saccade trials reverses with training will be at issue in the final experiment of this thesis.

Finally, there were slight differences between the design of the present experiment and that of Everling et al. (1999). In their study, Everling et al. included overlap versions of the pro- and anti-saccade trials. In order to obtain the maximum number of erroneous pro-saccades possible in the anti-saccade task, we left these conditions out in favor of more gap trials. However, in consideration of the performance data in our experiment, which closely resembles that reported by Everling et al. (1999), we do not think these procedural differences may account for an inverse relationship of fixation-related activity.

6.2.5.3 Microsaccade direction rather weakly revealed motor-preparation signals

In addition to the strong support for microsaccadic correlates of temporal aspects of saccade preparation, we aimed to find out whether localized motor-preparation activity is reflected in microsaccade direction. Physiological data revealed that erroneous pro-saccades are associated with high levels of preparatory pre-stimulus activity of neurons coding for saccades directed to the location of the peripheral stimulus in the anti-saccade task (Everling, Dorris, & Munoz, 1998). In contrast to that, enhanced preparatory activity of SC buildup-neurons coding for the target location of anti-saccades was reported for correct responses in the anti-saccade task. Accordingly, we hypothesized a lower congruency between microsaccade direction and the direction of a correct anti-saccade in trials in which a pro-saccade error was generated. In the data of Everling, Dorris, and Munoz (1998), the pre-stimulus-activity effect was evident from about 130 ms before the onset of the imperative stimulus. Indeed, we have some evidence that microsaccades in that time window show a stronger bias to stimulus-congruent directions in pro-saccade-error trials as compared to correct anti-saccade trials. It is in line with our hypothesis that this bias can be traced back primarily to stimulus-directed microsaccades before erroneous pro-saccades rather than to target-directed microsaccades before correct anti-saccades, since motor preparation was considered being particularly strong for direction errors.

Note, however, that the congruency effect was small, relied on little data, and was preceded by a direction effect, opposite to the one just reported. Both the expected congruency effect and the reverse one in an earlier time window primarily resulted from a bias in microsaccade direction in

those trials in which an erroneous pro-saccade was generated, suggesting a relationship between one and the other. This latter finding raises another interesting question, not discussed so far: What happens to the distribution of activity in the saccadic motor map, after the generation of a microsaccade? Is activity at a certain site in the map “consumed” by the generation of a microsaccade? Does activity at other sites in the motor map benefit from this consumption? Or does activity just shift to another location in the map, depending on the amplitude of the movement and the location of the fixation target? Based on the present set of data, these questions may not be answered conclusively. Clearly, a quantitative implementation of our model would help in understanding these processes.

6.3 Effects of practice in the pro- and anti-saccade tasks

In the previous experiments, we reported evidence for a systematic relationship between microsaccades and performance in the pro- and anti-saccade tasks. The majority of these findings were predicted based on our understanding of the generation of microsaccades as resulting from fixation-related activity in the saccadic motor map that is in competition with the generation of large saccades. In addition to these findings, we attempted to establish a relationship between microsaccades and activity in the rostral pole of the SC by comparing microsaccade rates and amplitudes to physiological data collected in single-cell recordings in monkeys. Indeed, we obtained systematic variations of microsaccade rate in the instruction period, depending on whether a pro-saccade or an anti-saccade was generated. However, this relationship of rates was opposite to what has been described for the activity of FN in the rostral SC in a similar time window. We have considered several explanations for this result. Most probably, high-level influences from cortical areas involved in the generation of voluntary saccades may exert their influence on the inhibition of reflexive pro-saccades in the anti-saccade task, thereby also preventing the generation of microsaccades. On the other hand, however, it is conceivable that the absence of a systematic relationship results from an extreme imbalance in the amount of practice in the tasks. In our experiments, naïve participants performed one session each. Monkey subjects like those used by Everling et al. (1999), in contrast, are highly trained, often performing thousands of trials a day, five days a week, for many months (cf., Fecteau & Munoz, 2003). Here, we tested how performance in pro- and anti-saccade tasks changes with practice and how microsaccade statistics

evolve at the same time, when participants complete five consecutive training sessions. We will focus on the change of performance and microsaccade rate over sessions, omitting the wealth of additional analyses that were reported in the previous experiment.

6.3.1 Methods

6.3.1.1 Participants

Ten students of the University of Potsdam were paid 35€ or received study credit for their participation in five experimental sessions (within 2 to 21 days, 12.2 days on average). They were 20 to 26 years old (23.1 years on average), had normal or corrected-to-normal vision, and were in good health. Four of our ten subjects had prior experience with the combined pro- and anti-saccade task, i.e., they had been participants in prior studies using this paradigm.

6.3.1.2 Experimental setup and eye-movement recording

Participants were seated in a silent and darkened room with the head positioned on a chin rest, 50 cm in front of a computer screen. Stimuli were presented on a 22-inch iiyama HM204DT CRT (1024 by 768 resolution or 46° by 34° of visual angle; refresh rate 100 Hz). The experiment was controlled by an Apple Power Macintosh G4 computer. Eye-position data were recorded and available on-line using an EyeLink-II system (SR Research, Osgoode, ON, Canada) with a sampling rate of 500 Hz and a noise-limited spatial resolution better than 0.01°. The experimental software was implemented in Matlab (MathWorks, Natick, MA, USA), using the Psychophysics (Brainard, 1997; Pelli, 1997) and EyeLink (Cornelissen et al., 2002) toolboxes.

6.3.1.3 Procedure

In each of five sessions, participants performed 10 practice trials and 300 test trials of a simple saccade task. Half of the trials were pro-saccade trials, i.e., participants were instructed to make a saccade to an appearing imperative stimulus. In the other half of the trials, participants were required to make a saccade to the mirror location of the appearing stimulus, i.e., to perform an anti-saccade task (Hallett, 1978). Practice trials were comparable to test trials in all respects. Before the first and after every 30 trials the eye tracker was calibrated (standard 9-point grid) and calibration was validated. Every fifth trial, a drift correction was carried out. Before each trial, the

fixation spot was displayed at the center of the computer screen. To start a trial, correct fixation was required. Otherwise, a drift correction was carried out and the trial was started over. If the eyes were still not detected within the critical area, the calibration was repeated.

Except for small differences, sequences of visual stimulation were comparable to those illustrated in Figure 6.10. Participants fixated a small spot displayed centrally on a gray background; the color of the spot (red or green) instructed which kind of saccade had to be performed. After a random interval of 1500 to 2500 ms, the fixation spot was removed. Subsequently, an imperative stimulus appeared, 10° to the left or to the right in the visual periphery. Between the offset of the fixation spot and the onset of the stimulus, a 200 ms gap of no visual stimulation was introduced. Participants were required to hold fixation centrally until the imperative stimulus was displayed. Then, a speeded response saccade was required. Response saccades (eye position shift to either of the two target locations) were detected on-line. A trial ended after 800 ms of presentation of the imperative stimulus. Inter-trial intervals of 500 ms with no visual stimulation were introduced to enable blinking. If gaze position left a fixation square (3° side length, centered on the fixation spot) while fixation was still required (i.e., before the imperative stimulus was presented), an error feedback was triggered and the trial was aborted. Aborted trials were repeated in random order after the 300 regular trials.

Target locations (left or right) and trial types (pro-saccade or anti-saccade) were randomly interleaved across the 300 trials. Participants were randomly assigned to one of two groups. In one group, a red fixation spot was associated with an anti-saccade task and green with a pro-saccade task. In the other group, the learned association was vice versa.

6.3.1.4 Stimuli

The fixation spot was a red or green circle (depending on the task) with a diameter of 0.5° of visual angle. Red and green fixation spots were identical in form and adjusted in luminance using a flicker-fusion method. That is, before an experimental session, participants were instructed to minimize the flickering of two colored spots (red and green) alternating with 20 Hz by adjusting the luminance of the green spot.

The imperative stimulus was a white circle (diameter: 0.5°). The error feedback, triggered by blinks and anticipatory saccades, was a binaural 660 Hz tone at about 70 dbA, played for 82 ms via the internal speakers of the G4 computer.

6.3.1.5 Data preparation

For data analysis, a post-hoc saccade detection was performed using a new version (Engbert, 2006b) of the algorithm by Engbert and Kliegl (2003b). Velocities were computed from subsequent samples in the series of eye positions in the response time window 500 ms on from the go signal. Saccades were detected in 2D velocity space using thresholds for peak velocity and minimum duration. We used a relative threshold of 6 SDs of the velocity and a minimal duration of 6 ms (or three data samples). The first saccade that shifted gaze to the center of a target location $\pm 8^\circ$ was taken as a response saccade. Saccadic reaction time was defined as the latency between stimulus and saccade onsets.

Subsequently, we used the same algorithm to detect microsaccades (amplitude $< 1^\circ$) in the interval from fixation onset to the response saccade. As in the previous experiments, we considered only binocular microsaccades.

Trials including saccades larger than 1° prior to the response saccade were discarded, as were trials with SRTs shorter than 70 ms. Some trials had to be excluded due to data loss during eye-movement recording. Participants contributed 1408 to 1556 trials⁵ to the final data analyses, resulting in a total of 14812 trials (out of 15000 or 98.7%) in which 41488 microsaccades were detected.

6.3.1.6 Data analysis

Where provided, confidence intervals were computed using a simple bootstrapping technique (Efron & Tibshirani, 1993). From an original sample of N values, 1000 bootstrap samples were generated, each by selecting (with replacement) N values of the original sample. The 1.96-fold of the standard deviation of the means of these 1000 bootstrap samples was computed to generate 95% confidence intervals ($CI_{95\%}$) of the mean of the original sample. In graphical illustrations, confidence intervals will often allow the reader to compare different conditions by so-called “rules of eye” (Cumming & Finch, 2005). For within-subject comparisons, uninformative between-subject variance was therefore removed (Cousineau, 2005); these confidence intervals will be labeled $CI_{95\%ws}$.

⁵Indeed, some participants contributed more than 1500 trials to the data analyses. This is due to recycling of trials in the course of the experiment, which, however, were fully usable after post-hoc data processing.

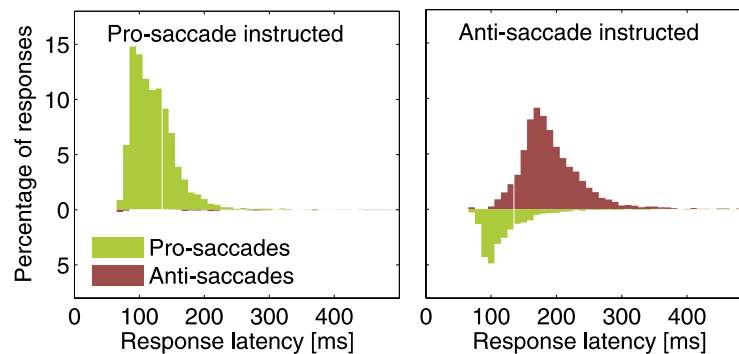


Figure 6.18: Average response-latency distributions for correctly (plotted upwards) and incorrectly (plotted downwards) directed saccades in the pro- and anti-saccade conditions. Responses were pooled over sessions. Dotted white lines indicate the border of 130 ms below which saccades are usually counted as express saccades (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). Bin width was set to 10 ms.

6.3.2 Results

6.3.2.1 Performance in the task

Figure 6.18 shows the average saccadic response-latency distributions for both pro-saccade and anti-saccade trials. Trials for all sessions were collapsed for this plot. Distributions for correctly directed saccades (targeting the instructed location) are plotted upwards, distributions for direction errors plotted downwards. Dashed white lines indicate the border of 130 ms below which saccades are traditionally counted as express saccades (Fischer & Boch, 1983; Fischer & Ramsperger, 1984). We replicated longer saccade latencies for correct responses in the anti-saccade task as compared to both correct pro-saccades ($t[9] = 12.67, p < 0.001$; paired t -test) and pro-saccade errors in the anti-saccade task ($t[9] = 12.57, p < 0.001$). Again, direction errors were mainly confined to the anti-saccade task. The latency of these direction errors cannot be distinguished from those of correct pro-saccades; $t(9) = 1.04, p = 0.33$ (paired t -test).

Figure 6.19 shows cumulative distributions of latencies of three types of responses, correct prosaccades, correct antisaccades, and pro-saccade errors in the anti-saccade task as a function of session. To generate this figure, responses were sorted by their latency; every dot represents one response. Clearly, performance increases over sessions. Participants responded faster in later sessions than in earlier ones. First of all, the tail of the distribution changes with time, i.e., the range of longer response latencies most prominently benefits from practice.

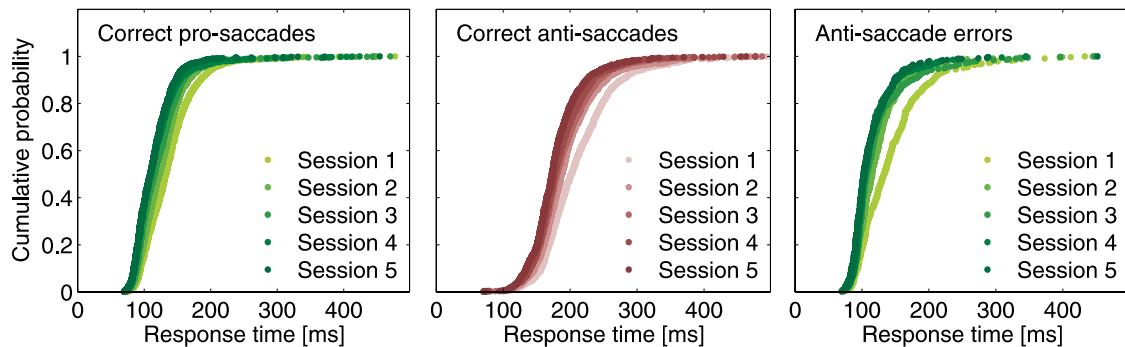


Figure 6.19: Cumulative distributions of saccade latencies as a function of session for three types of responses: correct pro-saccades, correct anti-saccades, and pro-saccade errors in the anti-saccade task. Responses of all participants were collapsed. Every dot represents one response; responses are sorted by their latency.

Figure 6.20 displays aggregations of these distributions. As a function of condition and session, means and $CI_{95\%}$ s are shown for the latencies of correct responses (left panel), the percentages of express saccades for correct responses (middle panel), and the percentage of direction errors (right panel). Repeated-measures ANOVAs using condition and session as independent variables were conducted on each of these three dependent variables. First, participants were faster in the pro-saccade task than in the anti-saccade task; $F(1, 9) = 146.81, p < 0.001$. Response times decreased with session ($F[4, 36] = 10.47, p < 0.001$), and this decrease was slightly stronger for anti-saccades than for pro-saccades; $F(4, 36) = 3.01, p = 0.030$. Second, the percentage of express saccades was much higher for pro-saccades than for anti-saccades; $F(1, 9) = 97.74, p < 0.001$. This percentage increased with sessions; $F(4, 36) = 7.05, p < 0.001$. The number of express saccades increased much faster in the pro-saccade task as compared to the anti-saccade task, resulting in a significant interaction of condition and session; $F(4, 36) = 6.92, p < 0.001$. Finally, the number of direction errors was higher in the anti-saccade task as compared to the pro-saccade task; $F(1, 9) = 63.34, p < 0.001$. Neither the number of errors (main effect of session) nor the difference between the two conditions (interaction) changed with sessions; $F_s < 1.15, p_s > 0.35$.

6.3.2.2 Overall microsaccade rate

As a first step, we repeated the analysis of microsaccade rate as conducted in our first pro-/anti-saccade experiment. The upper panels of Figure 6.21 show the evolution of microsaccade rate, time-locked to gap onset (left panel) and saccade onset (right panel), respectively, for correct pro-

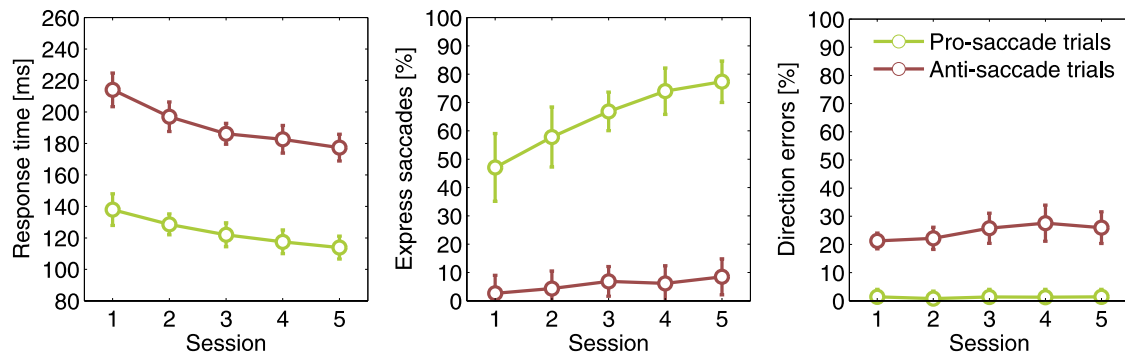


Figure 6.20: Average performance in the pro-/ anti-saccade task is plotted as a function of session. Response times and percentages of express saccades include correct responses only. Error bars are $CI_{s95\%ws}$.

and anti-saccades as well as pro-saccade errors in the anti-saccade task. The analysis is identical to that described in the previous experiment (see Section 6.2.4.2), except that data from five sessions were collapsed.

We replicate the results of our previous experiment (reported in Section 6.2.4.2) in many but not all their facets. Let us first consider the findings common to both experiments. First, a sharp decrease in microsaccade rate followed the offset of the fixation spot with a latency of about 100 ms, regardless of type of trial. Rates remained on a very low level throughout the remainder of all types of trials. Second, over a long period before the onset of a saccade, participants exhibited higher microsaccade rates in correct pro-saccade trials than in correct anti-saccade trials. Significant deviations were observed from -630 ± 50 ms to -150 ± 50 ms. Third, a similar pattern of results (though not significant when controlling for alpha inflation) was evident for the comparison of erroneous pro-saccades and correct anti-saccades. Finally, no deviations in microsaccade rate could be observed between correct pro-saccades and pro-saccade errors in anti-saccade trials.

However, the results obtained in the present experiment also differed from those described in Section 6.2.4.2. In contrast to the previous experiment, we did not obtain rate differences in the stimulus-locked analyses, neither between correct anti-saccade and correct pro-saccade trials, where the effect was small in the previous study, nor between correct anti-saccade and pro-saccade error trials, where a larger effect had been shown. Rather, as can be seen in the upper left panel of Figure 6.21, microsaccade rates for all response types lie on top of each other.

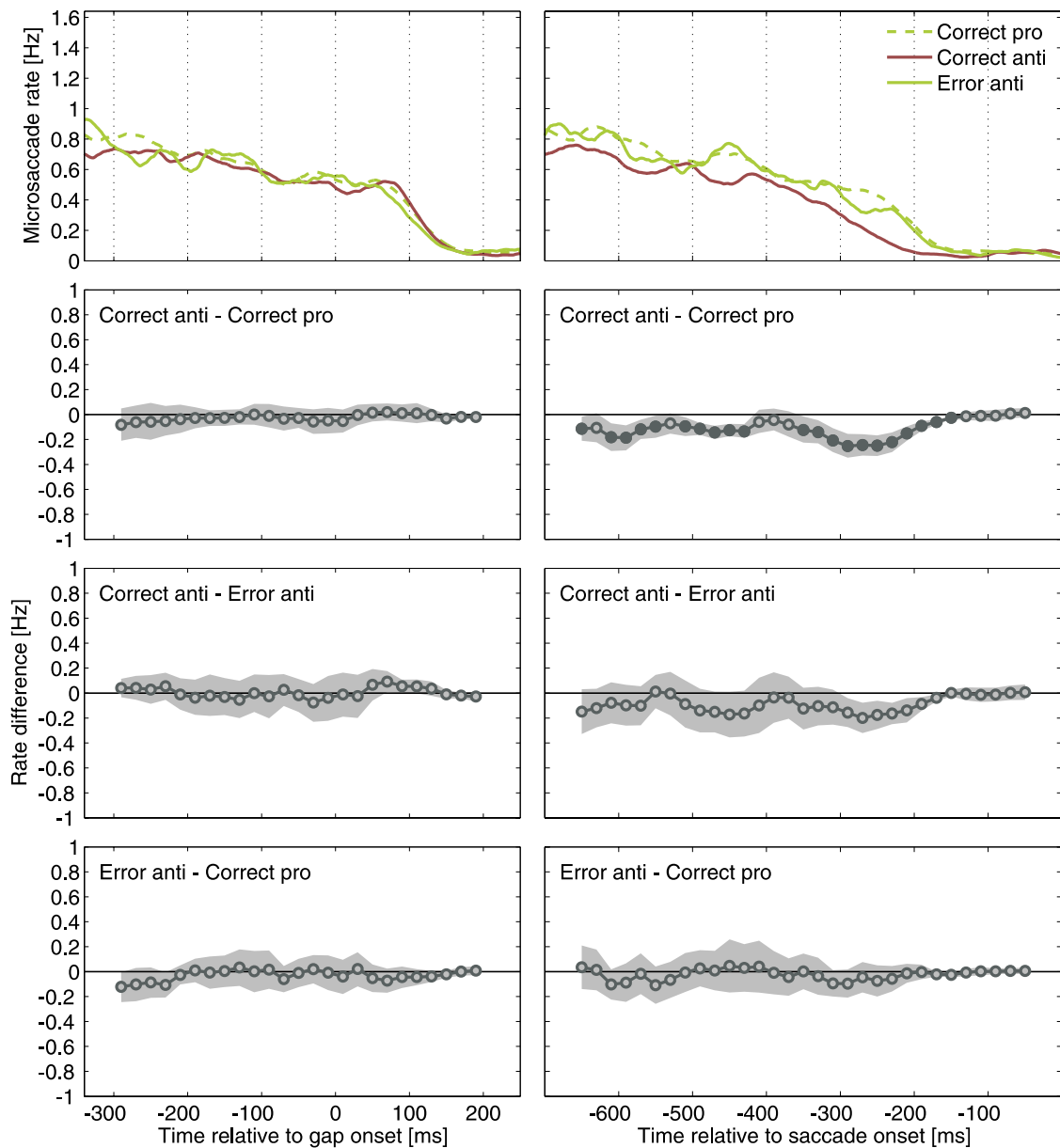


Figure 6.21: Microsaccade rate as a function of time from gap onset (left panels) and time to saccade onset (right panels) for three types of responses: correct pro-saccades, correct anti-saccades, and erroneous pro-saccades in the anti-saccade task. The lower six panels display tests for differences between rates in the different response conditions. Time windows of ± 50 ms were used. Error bands are $CI_{95\%}$. Filled dots indicate time windows in which deviations were significant after controlling the alpha level using FDR (Benjamini & Hochberg, 1995).

6.3.2.3 Effects of practice on microsaccade rates

The present data do not reveal differences in overall microsaccade rates between pro-saccade and anti-saccade responses, in the stimulus-locked analyses. To find out whether this was due to

practice, which was experimentally manipulated in the present but not in the previous study, we analyzed microsaccade rate as a function of session. Rates were computed in a time window of 200 ms centered on gap onset, where the strongest deviations were observed in the first experiment. The line plots in the two panels of Figure 6.22 show mean microsaccade rates as a function of session. Corresponding bar plots show rate differences per session, comparing correct anti-saccade trials to correct pro-saccade trials (left panel) and, respectively, to erroneous pro-saccades in the anti-saccade task (right panel). The basic pattern of mean rates is compatible with a practice effect on microsaccade rate: In the first session, we replicated higher rates before pro-saccades (instructed and erroneous) as compared to correct anti-saccade trials. With increasing practice (sessions), the ratio of rates switched for both comparisons. That is, in the last two of five sessions, participants exhibited a lower mean rate of microsaccades before pro-saccades as compared to correct anti-saccade trials. We performed two analyses to evaluate these rate differences statistically. First, two repeated-measures ANOVAs were conducted using session and response type as independent variables, seeking for an interaction of the two variables. No interaction was obtained, neither between correct anti-saccade trials and correct pro-saccade trials ($F[4, 36] = 0.66, p = 0.62$), nor between correct anti-saccade trials and pro-saccade errors in the anti-saccade task ($F[4, 36] = 1.95, p = 0.12$). Second, we tested the difference of rates for every session against zero (see bar plots in Figure 6.22). For the comparison of rates in correct pro-saccade trials to correct anti-saccade trials, all confidence intervals included zero; the comparison of correct to erroneous responses in the anti-saccade task revealed significant differences in the final session (0.23 ± 0.13 Hz; mean difference and corresponding $CI_{95\%}$).

Four participants reported that they had prior experience with the combined pro- and anti-saccade task; i.e., they had participated in prior studies using this task that were conducted in our lab. To check whether the inclusion of these participants in the above analyses obscured potential effects of practice on microsaccade rate, we repeated the reported analyses with the remaining six participants. Mean rates and differences per session for this sub-sample are shown in Figure 6.23. The same basic pattern of mean rates was observed as in the analysis of all subjects, however, the differences between response types were much larger. For the comparison of correct trials in the anti-saccade and the pro-saccade task, the interaction of response type and session was marginally significant in a repeated-measures ANOVA; $F(4, 20) = 2.53, p = 0.072$. For a similar analysis of rates in correct and incorrect anti-saccade trials, this interaction was in fact reliable;

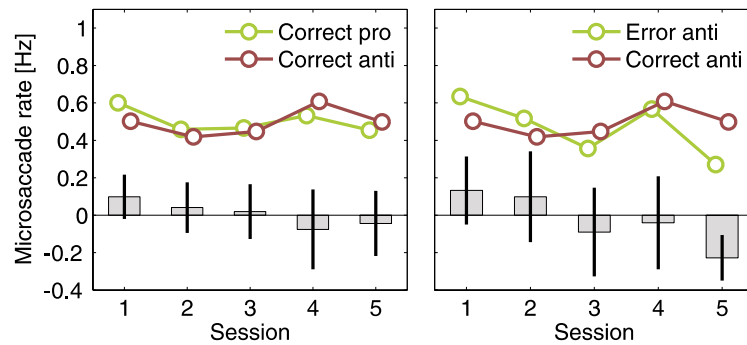


Figure 6.22: Microsaccade rate around the time of gap onset (± 100 ms) as a function of session and response type. Mean rates in correct anti-saccade trials are compared to mean rates in correct pro-saccade trials (left panel) and erroneous pro-saccades in the anti-saccade task (right panel). Bar plots show corresponding rate differences for each session. Error bars are $CI_{95\%}$.

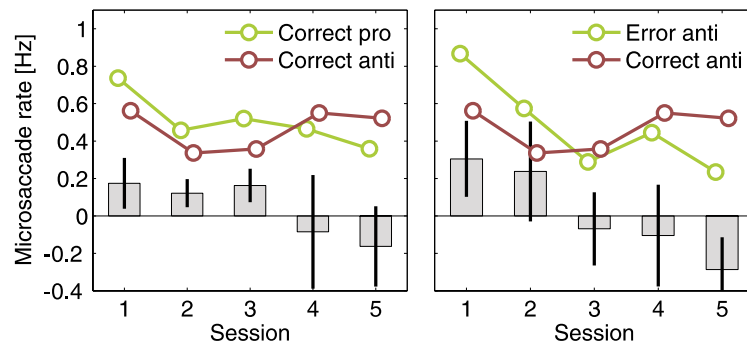


Figure 6.23: Same as in Figure 6.22, but for a reduced sample of six participants, who had no prior knowledge of the task.

$F(4, 20) = 4.42, p = 0.010$. To find the origin of these interactions, we determined for each response type whether microsaccade rates were different between the first and the last session. This was the case for correct pro-saccade trials (0.38 ± 0.33 Hz; mean difference and corresponding $CI_{95\%}$) and pro-saccade errors in anti-saccade trials (0.63 ± 0.35 Hz), but not for correct anti-saccade trials (0.04 ± 0.40 Hz). As in the analyses of the complete sample, we also computed rate differences between response types for each session separately. Mean differences and corresponding $CI_{95\%}$ are visualized as bar plots in Figure 6.23. In the first sessions, rates were higher before correct pro-saccades (sessions 1 to 3) and pro-saccade errors in the anti-saccade task (session 1) as compared to rates before correct anti-saccade responses. In the final session, rate was lower before errors than before correct responses in the anti-saccade task.

6.3.3 Discussion

In our previous experiment we found a higher rate of microsaccades in the instruction/gap period before correct pro-saccades as compared to correct anti-saccade responses. Similarly, erroneous pro-saccades in the anti-saccade task were preceded by higher microsaccade rates than were correct responses. Here, we tried to answer the question whether these differences in microsaccade rates reverse with practice. To this end, we replicated the interleaved pro-/anti-saccade paradigm in a second experiment, having participants perform five consecutive sessions. Practice greatly improved saccadic performance in both the pro-saccade and the anti-saccade task. Response times continuously decreased with sessions while mean error rates remained on a constant level.

In addition to the strong improvements in performance, we examined how microsaccade rates were affected by practice. In the overall analyses, collapsing the data from all sessions, differences in microsaccade rates between different response types were completely gone as compared to our first pro-/anti-saccade experiment, hinting at the possibility of a reversal effect. Indeed, in the first three sessions, mean microsaccade rate was higher in the correct pro-saccade condition as compared to the correct anti-saccade condition. In turn, in the final two sessions, the pattern of means switched; now lower mean rates of microsaccades were observed in the pro-saccade task. Though suggestive, this pattern was not reliable on the basis of the ten subjects tested in the present experiment. However, in a subsequent analysis we excluded four participants, who had prior experience with the task. For the resulting sub-sample of six participants, the ratio of microsaccade rates indeed switched over sessions, though this effect was only marginally significant in the comparison of correct pro-saccades to correct anti-saccades. The switch was even clearer when comparing pro-saccade errors to correct responses in the anti-saccade task. Thus, for the comparison of rates before pro-saccades to rates before anti-saccades, we have some evidence for a reversal of microsaccade rates during the instruction period. At the end of five sessions, participants appeared to produce fewer microsaccades before generating a pro-saccade than before successfully making an anti-saccade. This pattern of microsaccade rates matches the relative activation of FN in the SC motor map during the interleaved pro-/anti-saccade task that has been reported in trained monkeys (Everling et al., 1999).

How do these findings relate to those of the previous study and how may the results of the two experiments be explained in a common framework? First of all, we have to emphasize that the data presented here are not decisively supporting practice effects on microsaccade rate on

either pro- or anti-saccades. Power was limited in the present analyses, but in the face of that, the effects of practice on microsaccade rate must be considered notable. Nevertheless, two scenarios must be discussed. The first one is that microsaccade rate does not change critically with practice. In this case, the most favorable explanation for the discrepancy between rostral SC activity and microsaccade statistics is that the inhibition of microsaccades in the anti-saccade task is a byproduct of the effort to prevent reflexive pro-saccades. According to this hypothesis microsaccade rate will no longer be informative with regard to the amount of activity at fixation-related sites in the saccadic motor map, as soon as voluntary control is involved. Rather, microsaccade-related signals relayed by the motor map will be inhibited downstream in oculomotor processing, possibly by direct connection from cortical areas.

The second scenario is that microsaccadic activity is in fact attenuated with practice in the pro-saccade task, for which we have presented at least some evidence. In this case, differences in microsaccade statistics between the pro- and the anti-saccade task could indeed be proportional to fixation-related activity in the rostral SC. In the present data, the interactive influence of practice and response type on microsaccade rate was primarily due to changes associated with pro-saccade responses. Microsaccade rate decreased with sessions before pro-saccade responses, but there was no apparent change associated with correct anti-saccade trials. Thus, while the rate of microsaccades was not correlated to practice-related performance benefits in the anti-saccade task, these two measures may well be related in the pro-saccade task. Indeed, practice in the pro-saccade task has been associated with higher levels of motor-preparation (Paré & Munoz, 1996), which, in turn, result in higher levels of fixational disengagement (Dorris et al., 1997). Adopting this rationale would emphasize that microsaccade rate in the anti-saccade task is rather loosely coupled to fixation-related activity in the SC; rather it is determined by inhibitory processes on the generation of saccadic eye movements in general. In contrast, microsaccade statistics reflect quite a number of behavioral and physiological findings related to fixational disengagement in the pro-saccade task. From the account stressed here, one could infer that the differences in rostral SC activity between pro- and anti-saccades are subject to training effects.

Further research will have to decide which of the two scenarios described comes closest to what really makes up the mechanisms underlying the generation of microsaccades. Based on the results that have been presented here, a combination of both appears most probable. That is, the amount of top-down inhibition of saccades may be adjusted with practice in the anti-saccade

task, allowing more microsaccades to occur. At the same time, practice is proposed to favor fixational disengagement at the level of the SC in both pro- and anti-saccade trials. Thus, for anti-saccades the two effects may effectively cancel each other, while for pro-saccades microsaccade rate decreases with practice.

Chapter 7

General summary and conclusions

Microsaccades are one type of small eye movements generated by normal observers when they try to fixate. There have been vivid controversies in the literature broaching the issue of potential perceptual and oculomotor functions of this behavior. We elaborated the universe of these studies at length in Chapter 2. In contrast to the wealth of publications elaborating these issues, the implementation of microsaccades in the system controlling oculomotor behavior has not yet been discussed in great detail. In Chapter 3, we reviewed the few existing studies relating to this topic. By embedding their results in the context of the well-described neural circuitry of saccade generation, we build a case for a model according to which microsaccades and saccades result from mutually dependent motor plans, competing for expression.

The basis of our model is an activation field coding for the whole range of saccade amplitudes; the center of this field represents fixation. Activity in the field is modulated by both stimulus-related and task-related input. Excitation of neighboring sites and inhibition of remote locations in the field govern its dynamics. A saccade of a certain amplitude may only be generated if there is at least a certain amount of activity at the associated site in the field; the proportion of activity relative to the overall activity in the field determines the probability of a certain amplitude to be selected for the generation of a saccade. During fixation, activity is highest at the central part of the field. Spreading over to slightly peripheral locations in the field, this activity may result in the generation of microsaccades. Thus, according to our model, these saccades are a byproduct of fixation-related activity.

In five experiments, presented in Chapters 4, 5, and 6, we tested implications of the common-field model of microsaccade and saccade generation. Three dominant strategies were pursued to this end. First, we analyzed the temporal evolution of microsaccade statistics in response to certain stimulus-related and task-related inputs to the oculomotor system, hence, the assumed movement field. Second, we examined the impact of microsaccades on subsequent response saccades. Third, where possible, we related our findings to physiological processes at the level of the motor map in the superior colliculus (SC), a structure thought to represent a neurophysiological equivalent to the field model introduced here. We designed our paradigms in such a way that critical predictions could be derived in this regard.

In what follows, we will first sum up our evidence corroborating the proposed model. Further, we will highlight the increasing requirement of a quantitative model, if a more detailed understanding of the processes underlying microsaccade generation and their relation to saccade preparation is strived for. Finally, we will discuss alternative accounts for the generation of microsaccades.

7.1 Evidence for the common-field model of microsaccade and saccade generation

Our model turned out to be a reasonable framework for the study of microsaccade implementation and the consequences thereof for their interactions with other saccades. Our results were generally compatible with the view that microsaccades result from fixation-related activity in the central part of a movement field coding saccades of all amplitudes on different sites of this field. We proposed that the topographic motor map in the SC is a likely neuronal pendant of the movement field. In this section, we will shortly summarize how well these ideas were met by the results obtained in the empirical part of the present work.

Our model places microsaccade generation in the fixation-related part of the saccadic motor map. A first prediction, thus, was that the evolution of microsaccade rates indicates the level of fixation-related activity unfolding across time. In our first experiment, we examined the dynamics of microsaccade generation in response to the presentation of irrelevant stimuli (see Chapter 4). Strong microsaccadic inhibition, i.e., a transient drop in the rate of microsaccades, was induced by stimulus onsets. We hypothesized that microsaccadic inhibition is implemented as decreasing

activity at the central part of the motor map, resulting in the generation of fewer microsaccades. In the framework of our model, this hypothesis implies that the mode of the microsaccade-amplitude distribution is constantly shifted towards smaller amplitudes. We reasoned that larger microsaccades will be inhibited first, since activity at corresponding sites in the movement field will fall short of threshold. Our data are in good agreement with this hypothesis; time-locked to the strong decline in microsaccade rate, microsaccade amplitude decreased substantially. This was true for both visually and auditorily induced microsaccadic inhibition. We replicated decreases in mean microsaccade amplitudes in the gap experiments reported in Chapter 6; here, microsaccade rates and mean amplitudes decreased in response to the offset of foveal fixation stimuli. In these tasks, the evolution of microsaccade rate was compatible with the time course of fixational disengagement as measured by its physiological correlate at the level of fixation neurons (FN) in the SC.¹

It is an important feature of most of contemporary models of saccade generation that fixation-related activity is inhibited to allow for the initiation of a saccadic eye movement. One of the cornerstones of our common-field model is that microsaccades are generated by this kind of activity. Therefore, we hypothesized that microsaccades may not be generated shortly before a saccade is executed. Several results of our studies confirm this prediction. First, microsaccade rate dropped in the course of response preparation; this result nicely showed up for fixed target-to-go-signal intervals in the delayed-response task (see Chapter 5). In addition, microsaccade rates reached a minimum just prior to saccades in all of our experiments. Second, mean microsaccade amplitude decreased consistently as a saccade was about to be generated, a finding consistent with a drop of activity at the central part of the movement field (see above). These findings were evident across the whole range of saccade tasks tested here, in a delayed saccade task as well as in pro-saccade and anti-saccade tasks.

Complementary to these results, long response latencies were preceded by high rates of microsaccades while fast response times followed very low rates of microsaccades, as reported for pro- and anti-saccades in Chapter 6. In the gap task, significant deviations between microsaccade rates preceding either fast or slow responses were found yet about 100 ms prior to the presenta-

¹Considering these mechanisms, one must keep in mind carefully that the generation of a microsaccade can only be a snapshot of the distribution of activity in the SC motor-map and a potential reconstruction of this distribution necessarily requires examining microsaccade statistics over many trials.

tion of a target, which is in line with the time course of fixational disengagement documented in several other studies (see Section 6.1.3.1).

Another prediction of a model ascribing microsaccade generation to above-threshold fixation-related activity, is that single microsaccadic incidences are an expression of high levels of this activity. Consequently, response times in trials where microsaccades were observed shortly before a saccade needed to be initiated, should exhibit increased response latencies as compared to trials in which no microsaccades were observed in a critical time window before the go signal. Indeed, this is what we observed in the delayed-saccade paradigm presented in Chapter 5. The prediction of microsaccade-induced slowing of saccadic responses holds especially for larger microsaccades, since these, according to our model, indicate that fixational disengagement has not yet been initiated. The results of the delayed saccade task (Chapter 5) yielded strong support for this hypothesis. We replicated microsaccade-induced slowing of response times in Chapter 6, whenever pro-saccades had to be generated. However, we have no evidence for response-time increases after microsaccades occurring just before the onset of an imperative stimulus in the anti-saccade task.

The results described so far emphasized the inverse relationship between microsaccade occurrences and fixational disengagement, a rather temporal aspect of saccade preparation. In addition to that, we examined the correspondence between spatial aspects of saccade preparation and the direction of microsaccades. These analyses were inspired by the established finding that microsaccade direction is biased in response to visuospatial attentional cues (see Section 2.4), which has been attributed to saccade-preparation mechanisms by some authors (Betta et al., in press; Galfano et al., 2004; Rolfs et al., 2004). In Chapter 6 we reported how localized saccade preparation may affect the distribution of microsaccade directions in both pro- and anti-saccade tasks. We reasoned that activity in the peripheral parts of the movement field, associated with the preparation of a saccade to a certain location, may result in a spatial shift of the activity at the central part of the field. Hence, a bias in microsaccade direction would be predicted. We found some, but rather weak evidence that microsaccade direction is predictive of saccadic reaction times in our tasks, where target direction was random. First, we obtained a bias towards target-incongruent microsaccades preceding slow response times in the 0 ms gap condition. In fact, motor preparation was assumed to be lowest in this condition as compared to longer gap durations. We argued, however, that the chance of obtaining an effect of microsaccade-target congruence on

response times was highest in the 0 ms gap condition, since microsaccade generation relies on a low level of fixational disengagement. Second, the analysis of microsaccade directions in the anti-saccade task yielded some support to the idea that microsaccade direction is an indicator of localized aspects of saccade preparation. Neurophysiological studies have shown that errors in the anti-saccade task are associated with exceptionally high levels of preparation for saccades the stimulus location (Everling, Dorris, & Munoz, 1998). Just before the presentation of an imperative stimulus in the antisaccade task, indeed, more microsaccades were directed towards the stimulus when an erroneous pro-saccade was generated as compared to when a correct anti-saccade was produced. Note, however that these effects were weak and at the edge of significance. In part, this may have been caused by a high level of fixational disengagement, strongly favored in these tasks by the inclusion of a gap period of no visual stimulation just before target presentation. We summarize that microsaccade direction is not a good predictor of response-saccade behavior if fixational disengagement has reached an advanced level.

We have hypothesized that the motor map in the intermediate and deeper layers of the SC is a probable neural counterpart to the movement field forming the basis of our model. Several predictions of this hypothesis were met by the results of the present thesis, others found no direct support. We may shortly summarize the critical results. First, we discussed that microsaccadic inhibition has a time course that may only be achieved by a fast subcortical processing circuit, probably involving the direct pathway from the retina to the SC (see also Engbert, 2006b). In an irrelevant-onset paradigm we induced strong microsaccadic inhibition (Chapter 4). Its latency was subject to experimental manipulations such as stimulus luminance and modality. As predicted by the hypothesis that microsaccadic inhibition has its locus in the SC, the latency was longer for visual than for auditory stimuli. Moreover, luminance-contrast stimuli resulted in faster inhibition than stimuli that were subjectively isoluminant to the background.

In Chapter 6, we aimed to further substantiate our claim that microsaccades are a derivative of the activity at the rostral part of the SC by comparing the dynamics of microsaccade generation to the dynamics of neural activity as measured by Munoz and colleagues in a number of single-cell recording studies in monkeys. We examined the time courses of microsaccade rate in a gap task and a combined pro-/anti-saccade task and related these to the time courses of the activity of FN in the same tasks. Indeed, we found a qualitative agreement between the evolution of microsaccade rate and the time course of SC fixation-cell activity in the gap task; both dropped

after fixation offset, followed by a slight rebound period and a final settlement at a plateau at a level lower than during visual fixation. However, the results of a first combined pro-/anti-saccade experiment were not in agreement with this direct translation of FN activity to microsaccadic behavior. In trained monkeys, Everling et al. (1999) reported higher rates of FN activity in anti-saccade trials as compared to pro-saccade trials. Our human subjects, however, exhibited higher rates of microsaccades in the pro- as compared to the anti-saccades task, hence, the reverse pattern. A training study employing the combined pro-/anti-saccade task revealed some evidence for a switch of this pattern of microsaccade rates. Microsaccade rate before pro-saccades declined significantly from the first to the last session. In contrast, microsaccade rate before correct anti-saccades did not change as a function of session. Consequently, we argued that training may change the pattern of activity at the level of the SC. Alternatively, high-level influences may affect microsaccade generation downstream the SC in the saccade-generation circuitry, without necessarily being represented in the activity patterns of the SC. Both these accounts are compatible with the view that microsaccades originate from activity of FN in the rostral SC.

We introduced our model as a prolific framework for the study of how the generation of microsaccades and saccades may be related. We have presented ample evidence corroborating a common-field model of microsaccade and saccade generation. Moreover, the notion of a congruency of rostral SC activity and microsaccade generation turned out plausible. Finally, when introducing our model in Section 3.3, we have discussed potential mechanisms as to how fixation-related activity in the movement field could translate into microsaccadic behavior; triggering could be based on perceptual needs Engbert and Mergenthaler (2006), on an autonomous timing mechanism (Richter et al., submitted), or on the distribution of activity in the field itself. However, it has been beyond the scope of the present set of experiments to identify the processes underlying the initiation of microsaccades. Any of the above may be compatible with the results described in the studies reported here if additional assumptions would be made. First, these mechanisms may be evaluated on the basis of physiological data as soon as these are available. Second, a quantitative model of microsaccade generation may help in clarifying this issue. In any case, microsaccade statistics might offer a valuable source for predictions of the dynamics of physiological processes in the SC motor map. In fact, if our hypotheses hold true, microsaccade statistics in a variety of experimental paradigms would predict spatiotemporal characteristics of the distribution of activity in the SC motor map.

7.2 The need for a quantitative model

The number of findings concerning the behavior of microsaccades in a variety of tasks steadily increases. There has been a vivid interest of researchers in the relationship between microsaccades and attention as well as microsaccades and motor preparation. In a number of attentional cuing paradigms (Engbert & Kliegl, 2003b; Galfano et al., 2004; Gowen et al., 2005; Horowitz et al., in press; Laubrock et al., 2005, in press; Rolfs et al., 2004, 2005), observational (Horowitz & Albright, 2003; Valsecchi, Betta, & Turatto, in press) and simple manual-response tasks (Betta et al., in press; Betta & Turatto, 2006), rich data sets of the dynamics of microsaccade generation were produced over the last few years. Moreover, some progress has been made in the field of saccade generation and its relation to microsaccade statistics (Gowen & Abadi, 2005; Kingstone et al., 1995). The present work adds several new findings to both these topics, showing a wealth of quantitative data on microsaccade generation (Chapters 4 to 6) and their relation to large saccades (Chapter 5 and 6). In addition, this thesis goes beyond the exploratory description of microsaccadic behavior in a variety of task settings; it aimed to integrate these findings in a common framework of microsaccade generation. The qualitative model proposed has the potential to account for the data on microsaccade generation presented here and elsewhere, and may advance our knowledge of the oculomotor plant itself, in which both microsaccades and saccades are afforded.

However, the more findings there are, the more difficult the question may be answered, whether the various mechanisms proposed to account for those empirical findings, may be accomplished by the same machinery. In a qualitative model, empirical results may be explained by one mechanism or the other within a single framework. However, it will turn out difficult to understand whether one set of general principles may account for a series of results at the same time. Put more drastically, a qualitative model may hardly be falsified. Moreover, the predictions of a qualitative model will inescapably become soft, if more complex behavior is considered. We have encountered this problem in Chapter 6, when trying to derive hypotheses about the direction of microsaccades in response to enhanced activity in the periphery of the movement field. Similar difficulties will emerge for predictions concerning the influence of slow drift movements on activity in the movement field.

Thus, clearly, there is now the need for a formal model implementing microsaccade generation. The common-field model of microsaccade and saccade generation, that has been put forward here, is ideally suited for implementation in terms of a dynamic-field model (e.g., Erlhagen & Schöner,

2002). This type of a model has the advantage of being physiologically plausible with respect to processes in the SC saccadic motor map. There have been various approaches to model different aspects of the neurophysiological implementation of saccade generation in the SC (see Girard & Berthoz, 2005, for a review). These models were successfully implemented to explain the generation of large saccades in a variety of oculomotor tasks (Kopecz, 1995; Kopecz & Schöner, 1995; Trappenberg et al., 2001).

None of these models, however, aimed at explaining the involuntary production of small saccades. This is clearly deserved: With respect to understanding the generation of microsaccades, an implemented model has several advantages over a purely qualitative one. First, hypotheses with regards to microsaccade statistics as well as their relation to saccade generation, that were made here and in previous publications, may be tested for plausibility. Second, a qualitative model may be utilized to generate predictions for the behavior of microsaccades in new experimental situations. Third, detailed physiological processes may be proposed that underlie the generation of this behavior. Crucially, an implemented model is able to derive such predictions both in qualitative and in quantitative terms. Together, these characteristics of a quantitative model may provide a basis for understanding the implementation of microsaccade generation in a more thorough way. Clearly, the long-term goal is to provide an account for the interplay of saccades and fixations, including microsaccades, saccades, and possibly drift. The thesis at hand may provide a signpost as to how this may be accomplished.

7.3 Alternative accounts for microsaccade generation

Though the accumulation of results reported here and in previous work is consistent with a model that associates microsaccade generation with activation of the rostral pole of the SC, other accounts could, of course, be offered. Indeed, different physiological processes were proposed that could potentially result in the production of microsaccades. We will shortly discuss these alternative accounts in the light of the data collected so far.

According to one alternative explanation, microsaccades are spurious events caused by a transient lack of activation in omnipause neurons (OPN) in the saccadic burst generator circuit in the brainstem (Ashe et al., 1991; Zee & Robinson, 1979). OPN serve a gatekeeper function for the generation of saccades (e.g., Bergeron & Guitton, 2002). Consequently, short-term dropouts in

their tonic activation may indeed trigger small saccadic eye movements that would be interrupted as soon as OPN activity revives. We do not think, however, that OPN malfunction is the origin of microsaccades during normal fixation. A key difference between firing patterns of OPN and FN in the rostral SC is that OPN do not change their level of activity during the period of no foveal stimulation in the gap task (Everling, Paré, et al., 1998). Microsaccade rate, however, is strongly modulated by this experimental manipulation, as shown in the present work. The temporal evolution of microsaccade rate during the gap period qualitatively recovered the signature found for the activation of rostral cells in the SC (see Chapter 6). In addition, as has been pointed out earlier (Abadi & Gowen, 2004), interrupted saccades would likely exhibit an abnormal velocity profile, i.e., higher peak velocities. This is not the case for microsaccades. On the contrary, microsaccades were the first saccadic eye movements, for which the correspondence to the main sequence has been shown (Zuber et al., 1965). For these reasons, we may refuse OPN firing anomalies as the origin of microsaccades.

A further potential origin of microsaccades is sub-threshold activation of saccade-related neurons in the caudal SC, i.e., neurons that generate large saccades if activated above threshold. This proposal has been considered by Gowen and Abadi (2005), but could not be substantiated by their analyses (see also Rolfs et al., 2004). From a physiological perspective, there is only one hint (known to the authors) that this kind of activity could be related to microsaccade production. In a sidenote, Carello and Krauzlis (2004) remarked that after applying sub-threshold microstimulation to cells at caudal sites in the intermediate and deeper layers of the SC, small saccades (amplitudes of 0.5 to 1°) were evoked in 10% of the trials. Note, however, that this finding may also be explained by a model that puts microsaccade generation to the rostral site of the SC. We speculate that the distribution of rostral activity was shifted as a consequence of peripheral stimulation. Since the likelihood of generating a saccade increases with eccentricity in the rostral pole (Krauzlis et al., 1997), microsaccades could have been triggered at short latencies. The data reported in Chapter 6 of the present work may yield further support for the notion that the origin of microsaccades is more likely located in the rostral than in the caudal part of the SC. First, microsaccade statistics before pro- and anti-saccades were closely matching firing patterns of rostral SC neurons, but completely opposite to what has been shown for caudal SC neurons in the very same paradigms. Second, strong effects of microsaccade-target congruency on saccade latencies in both the gap and the anti-saccade task would have been expected if microsaccade

direction was directly related to preparatory activity in saccade-related neurons at the periphery of the motor map. If anything, these effects were weak in the present studies. We can not reject the idea that microsaccades are generated by sub-threshold activity at saccade-related sites in the caudal SC. However, physiological as well as behavioral data available so far rather corroborate the rostral-SC account for microsaccade generation (see Chapter 3).

As the schematic of the brain circuitry presented in Chapter 3 (Figure 3.1) suggests, a number of further neurophysiological origins of microsaccade production could be added, e.g., a precipitate termination of a saccade command by the cerebellum. However, the central role of the SC in the generation of involuntary saccades and the compatibility of this account with the wealth of data presented here and elsewhere strongly suggests that activation of the rostral SC is at least involved in the generation of microsaccades, if not their major determinant. We therefore suggest that this structure is the most probable physiological analogue of the movement-field model developed in this thesis work. We would like to emphasize, however, that this model of microsaccade generation is, in general, independent of an exact physiological equivalent. Rather, we would like this model to be considered as proposing the core principles that the regulation of microsaccade generation as well as the interactions of microsaccades and saccades.

7.4 A final remark

With the increasing interest in microsaccades and their function for perception and oculomotor control, it becomes more important to understand their implementation in the circuitry of saccade generation. Throughout this thesis, we have emphasized that microsaccades and saccades have a great deal in common and that it may be delusive to treat them as different types of motor acts (see the first paragraph of Chapter 3 for a list of similarities between microsaccades and saccades). However, the present work further suggests that there is a fundamental difference in the generation of microsaccades as compared to saccades. It is broadly accepted that for the generation of a saccade, fixation-related activity has to be reduced if not shut down completely. This is in clear contrast to what we propose for the generation of microsaccades. According to our model, these movements are a byproduct of fixation-related activity; in fact, their generation requires fixation-related activity! This is what makes microsaccades peculiar—they are caught between two stools, in-between fixation and movement.

References

- Abadi, R. V., & Gowen, E. (2004). Characteristics of saccadic intrusions. *Vision Research*, *44*, 2675-2690.
- Adler, F. H., & Fliegelman, M. (1934). Influence of fixation on the visual acuity. *Archives of Ophthalmology*, *12*, 475-483.
- Ahissar, E., & Arieli, A. (2001). Figuring space by time. *Neuron*, *32*, 185-201.
- Alpern, M. (1972). Eye movements. In D. Jameson & L. Hurvich (Eds.), *Handbook of sensory physiology* (Vol. VII/4, pp. 304-331). Berlin: Springer.
- Anderson, R. W., Keller, E. L., Gandhi, N. J., & Das, S. (1998). Two-dimensional saccade-related population activity in superior colliculus in monkey. *Journal of Neurophysiology*, *80*, 798-817.
- Arend, L. E., & Timberlake, G. T. (1986). What is psychophysically perfect image stabilization? Do perfectly stabilized images always disappear? *Journal of the Optical Society of America A*, *3*, 235-241.
- Ashe, J., Hain, T. C., Zee, D. S., & Schatz, N. J. (1991). Microsaccadic flutter. *Brain*, *114*, 461-472.
- Averill, H. L., & Weymouth, F. W. (1925). Visual perception and the retinal mosaic. II. The influence of eye movements on the displacement threshold. *Journal of Comparative Psychology*, *5*, 147-176.
- Bair, W., & O'Keefe, L. P. (1998). The influence of fixational eye movements on the response of neurons in area MT of the macaque. *Visual Neuroscience*, *15*, 779-786.
- Barlow, H. B. (1952). Eye movements during fixation. *Journal of Physiology*, *116*, 290-306.
- Barlow, H. B. (1963). Slippage of contact lenses and other artefacts in relation to fading and regeneration of supposedly stable retinal images. *Quarterly Journal of Experimental Psychology*, *15*, 36-51.
- Basso, M. A., Krauzlis, R. J., & Wurtz, R. H. (2000). Activation and inactivation of rostral superior colliculus neurons during smooth-pursuit eye movements in monkeys. *Journal of Neurophysiology*, *84*, 892-908.
- Basso, M. A., & Wurtz, R. H. (1997). Modulation of neuronal activity by target uncertainty. *Nature*, *389*, 66-69.
- Basso, M. A., & Wurtz, R. H. (1998). Modulation of neuronal activity in superior colliculus by changes in target probability. *Journal of Neuroscience*, *18*, 7519-7534.
- Becker, W. (1989). Metrics. In R. H. Wurtz & M. E. Goldberg (Eds.), *The neurobiology of saccadic eye movements* (Vol. 3, pp. 13-67). New York: Elsevier.
- Beeler, G. W. (1967). Visual threshold changes resulting from spontaneous saccadic eye movements. *Vision Research*, *7*, 769-775.
- Behan, M., & Kime, N. M. (1996). Intrinsic circuitry in the deep layers of the cat superior colliculus. *Visual Neuroscience*, *13*, 1031-1042.
- Bengi, H., & Thomas, J. G. (1968). Three electronic methods for recording ocular tremor. *Medical and Biological Engineering*, *6*, 171-179.
- Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society B*, *57*, 289-300.

- Bennet-Clark, H. C., & Evans, C. R. (1963). Fragmentation of patterned targets when viewed as prolonged after-images. *Nature*, *199*, 1215-1216.
- Bergeron, A., & Guitton, D. (2000). Fixation neurons in the superior colliculus encode distance between current and desired gaze positions. *Nature Neuroscience*, *3*, 932-939.
- Bergeron, A., & Guitton, D. (2002). In multiple-step gaze shifts: Omnipause (OPNs) and collicular fixation neurons encode gaze position error; OPNs gate saccades. *Journal of Neurophysiology*, *88*, 1726-1742.
- Bergeron, A., Matsuo, S., & Guitton, D. (2003). Superior colliculus encodes distance to target, not saccade amplitude, in multi-step gaze shifts. *Nature Neuroscience*, *6*, 404-413.
- Betta, E., Galfano, G., & Turatto, M. (in press). Microsaccadic response during inhibition of return in a target-target paradigm. *Vision Research*.
- Betta, E., & Turatto, M. (2006). Are you ready? I can tell by looking at your microsaccades. *Neuroreport*, *17*, 1001-1004.
- Boch, R., & Fischer, B. (1986). Further observations on the occurrence of express-saccades in the monkey. *Experimental Brain Research*, *63*, 487-494.
- Boch, R., Fischer, B., & Ramsperger, E. (1984). Express-saccades of the monkey: Reaction times versus intensity, size, duration, and eccentricity of their targets. *Experimental Brain Research*, *55*, 223-231.
- Boyce, P. R. (1967). Monocular fixation in human eye movement. *Proceedings of the Royal Society of London B*, *167*, 293-315.
- Brainard, D. H. (1997). The Psychophysics Toolbox. *Spatial Vision*, *10*, 433-436.
- Bridgeman, B., & Palca, J. (1980). The role of microsaccades in high acuity observational tasks. *Vision Research*, *20*, 813-817.
- Brockmann, D., & Geisel, T. (2000). The ecology of gaze shifts. *Neurocomputing*, *32-33*, 643-650.
- Bruce, C. J., Friedman, H. R., Kraus, M. S., & Stanton, G. B. (2003). The primate frontal eye field. In L. M. Chalupa & J. S. Werner (Eds.), *The visual neurosciences* (pp. 1428-1448). Cambridge: MIT Press.
- Büttner-Ennever, J. A., Horn, A. K. E., Henn, V., & Cohen, B. (1999). Projections from the superior colliculus motor map to omnipause neurons in monkey. *Journal of Comparative Neurology*, *413*, 55-67.
- Campbell, F. W., & Robson, J. G. (1961). A fresh approach to stabilized retinal images. *Journal of Physiology*, *158*, 11P-12P.
- Carello, C. D., & Krauzlis, R. J. (2004). Manipulating intent: evidence for a causal role of the superior colliculus in target selection. *Neuron*, *43*, 575-583.
- Casile, A., & Rucci, M. (2006). A theoretical analysis of the influence of fixational instability on the development of thalamocortical connectivity. *Neural Computation*, *18*, 569-590.
- Choi, W. Y., & Guitton, D. (2006). Responses of collicular fixation neurons to gaze shift perturbations in head-unrestrained monkey reveal gaze feedback control. *Neuron*, *50*, 491-505.
- Ciuffreda, K. J., Kenyon, R. V., & Stark, L. (1979). Suppression of fixational saccades in strabismic and anisometric amblyopia. *Ophthalmological Research*, *11*, 31-39.
- Ciuffreda, K. J., & Tannen, B. (1995). *Eye movement basics for the clinician*. St. Louis: Mosby.
- Clarke, F. J. J., & Belcher, S. J. (1962). On the localization of Troxler's effect in the visual pathway. *Vision Research*, *2*, 53-68.
- Collewijn, H., & van der Mark, F. (1972). Ocular stability in variable feedback conditions in the rabbit. *Brain Research*, *36*, 47-57.
- Coppola, D., & Purves, D. (1996). The extraordinarily rapid disappearance of entoptic images. *Proceedings of the National Academy of Sciences, USA*, *93*, 8001-8004.
- Corbetta, M., Akbudak, E., Conturo, T. E., Snyder, A. Z., Ollinger, J. M., Drury, H. A., et al. (1998). A common network of functional areas for attention and eye movements. *Neuron*, *21*, 761-773.
- Cornelissen, F. W., Peters, E. M., & Palmer, J. (2002). The Eyelink Toolbox: Eye tracking with MATLAB and the Psychophysics Toolbox. *Behavior Research Methods, Instruments, & Computers*,

- 34, 613-617.
- Cornsweet, T. N. (1956). Determination of the stimuli for involuntary drifts and saccadic eye movements. *Journal of the Optical Society of America*, 46, 987-995.
- Cousineau, D. (2005). Confidence intervals in within subject designs: A simpler solution to Loftus and Masson's method. *Tutorial in Quantitative Methods for Psychology*, 1, 42-45.
- Cullen, K. E., & Guitton, D. (1997). Analysis of primate IBN spike trains using system identification techniques. I. Relationship to eye movement dynamics during head-fixed saccades. *Journal of Neurophysiology*, 78, 3259-3282.
- Cumming, G., & Finch, S. (2005). Inference by eye: Confidence intervals and how to read pictures of data. *American Psychologist*, 60, 170-180.
- Cunitz, R. J., & Steinman, R. M. (1969). Comparison of saccadic eye movements during fixation and reading. *Vision Research*, 9, 683-693.
- Darwin, R. W. (1786). New experiments on the ocular spectra of light and colours. *Philosophical Transactions of the Royal Society*, 76, 313-348.
- Dayan, P., & Abbott, L. F. (2001). *Theoretical neuroscience: Computational and mathematical modeling of neural systems*. Cambridge, MA: MIT Press.
- Deubel, H., & Elsner, T. (1986). Threshold perception and saccadic eye movements. *Biological Cybernetics*, 54, 351-358.
- Deubel, H., Irwin, D. E., & Schneider, W. X. (1999). The subjective direction of gaze shifts long before the saccade. In W. Becker, H. Deubel, & T. Mergner (Eds.), *Current oculomotor research: Physiological and psychological aspects* (pp. 65-70). New York: Plenum Press.
- Deubel, H., & Schneider, W. X. (1996). Saccade target selection and object recognition: Evidence for a common attentional mechanism. *Vision Research*, 36, 1827-1837.
- Ditchburn, R. W. (1955). Eye-movements in relation to retinal action. *Optica Acta*, 1, 171-176.
- Ditchburn, R. W. (1980). The function of small saccades. *Vision Research*, 20, 271-272.
- Ditchburn, R. W. (1987). What is psychophysically perfect image stabilization? Do perfectly stabilized images always disappear?: Comment. *Journal of the Optical Society of America*, 4, 405-406.
- Ditchburn, R. W., Drysdale, F. R. S., & Drysdale, A. E. (1977a). The effect of retinal-image movements on vision. II. Oscillatory movements. *Proceedings of the Royal Society of London B*, 197, 385-406.
- Ditchburn, R. W., Drysdale, F. R. S., & Drysdale, A. E. (1977b). The effect of retinal-image movements on vision. I. Step movements and pulse movements. *Proceedings of the Royal Society of London B*, 197, 131-144.
- Ditchburn, R. W., & Fender, D. H. (1955). The stabilised retinal image. *Optica Acta*, 2, 128-133.
- Ditchburn, R. W., Fender, D. H., & Mayne, S. (1959). Vision with controlled movements of the retinal image. *Journal of Physiology*, 145, 98-107.
- Ditchburn, R. W., & Ginsborg, B. L. (1952). Vision with a stabilized retinal image. *Nature*, 170, 36-37.
- Ditchburn, R. W., & Ginsborg, B. L. (1953). Involuntary eye movements during fixation. *Journal of Physiology*, 119, 1-17.
- Ditchburn, R. W., & Pritchard, R. M. (1956). Stabilized interference fringes on the retina. *Nature*, 177, 434.
- Dodge, R. (1907). An experimental study of visual fixation. *Psychological Review Monograph Supplements*, 8, 1-95.
- Dorris, M. C., & Munoz, D. P. (1995). A neural correlate for the gap effect on saccadic reaction times in monkey. *Journal of Neurophysiology*, 73, 2558-2562.
- Dorris, M. C., & Munoz, D. P. (1998). Saccadic probability influences motor preparation signals and time to saccadic initiation. *Journal of Neuroscience*, 18, 7015-7026.
- Dorris, M. C., & Munoz, D. P. (1999). The underrated role of the "move system" in determining saccade latency. *Behavioral and Brain Sciences*, 22, 681-682.

- Dorris, M. C., Paré, M., & Munoz, D. P. (1997). Neuronal activity in monkey superior colliculus related to the initiation of saccadic eye movements. *Journal of Neuroscience*, *17*, 8566-8579.
- Doslak, M. J., Dell'Osso, L. F., & Daroff, R. B. (1983). Multiple double saccadic pulses occurring with other saccadic intrusions and oscillations. *Neuro-ophthalmology*, *3*, 109-116.
- Drysdale, A. E. (1975). The visibility of retinal blood vessels. *Vision Research*, *15*, 813-818.
- du Laurens, A. (1599). *A discourse of the preservation of the sight: Of melancholike disease; of rheumes, and of old age* (Translated by R. Surphlet ed.). London: Humphrey Milford / Oxford University Press.
- Efron, B., & Tibshirani, R. J. (1993). *An introduction to the bootstrap*. New York: Chapman & Hall.
- Elsner, T., & Deubel, H. (1986). The effect of saccades on threshold perception - A model study. *Biological Cybernetics*, *54*, 359-366.
- Engbert, R. (2006a). Flick-induced flips in perception. *Neuron*, *49*, 168-170.
- Engbert, R. (2006b). Microsaccades: A microcosm for research on oculomotor control, attention, and visual perception. *Progress in Brain Research*, *154*, 177-192.
- Engbert, R., & Kliegl, R. (2003a). Binocular coordination in microsaccades. In J. Hyönä, R. Radach, & H. Deubel (Eds.), *The mind's eye: Cognitive and applied aspects of eye movements* (pp. 103-117). Amsterdam: Elsevier.
- Engbert, R., & Kliegl, R. (2003b). Microsaccades uncover the orientation of covert attention. *Vision Research*, *43*, 1035-1045.
- Engbert, R., & Kliegl, R. (2004). Microsaccades keep the eyes' balance during fixation. *Psychological Science*, *15*, 431-436.
- Engbert, R., & Mergenthaler, K. (2006). Microsaccades are triggered by low retinal image slip. *Proceedings of the National Academy of Sciences, USA*, *103*, 7192-7197.
- Erlhagen, W., & Schönner, G. (2002). Dynamic field theory of movement preparation. *Psychological Review*, *109*, 545-572.
- Evans, C. R. (1965). Some studies of pattern perception using a stabilized retinal image. *British Journal of Psychology*, *56*, 121-133.
- Everling, S., Dorris, M. C., Klein, R. M., & Munoz, D. P. (1999). Role of primate superior colliculus in preparation and execution of anti-saccades and pro-saccades. *Journal of Neuroscience*, *19*, 2740-2754.
- Everling, S., Dorris, M. C., & Munoz, D. P. (1998). Reflex suppression in the anti-saccade task is dependent on prestimulus neural processes. *Journal of Neurophysiology*, *80*, 1584-1589.
- Everling, S., & Fischer, B. (1998). The antisaccade: A review of basic research and clinical studies. *Neuropsychologia*, *36*, 885-899.
- Everling, S., & Munoz, D. P. (2000). Neuronal correlates for preparatory set associated with pro-saccades and anti-saccades in the primate frontal eye field. *Journal of Neuroscience*, *20*, 387-400.
- Everling, S., Paré, M., Dorris, M. C., & Munoz, D. P. (1998). Comparison of the discharge characteristics of brain stem omnipause neurons and superior colliculus fixation neurons in monkey: Implications for control of fixation and saccade behavior. *Journal of Neurophysiology*, *79*, 511-528.
- Fecteau, J. H., & Munoz, D. P. (2003). Exploring the consequences of the previous trial. *Nature Reviews Neuroscience*, *4*, 1-9.
- Feldon, S. E., & Langston, J. W. (1977). Square-wave jerks: A disorder of microsaccades? *Neurology*, *27*, 278-281.
- Findlay, J. M., & Walker, R. (1999). A model of saccade generation based on parallel processing and competitive inhibition. *Behavioral and Brain Sciences*, *22*, 661-721.
- Fiorentini, A., & Ercoles, A. M. (1957). Vision of oscillating non-uniform fields. *Optica Acta*, *4*, 150-157.
- Fiorentini, A., & Ercoles, A. M. (1966). Involuntary eye movements during attempted monocular fixation. *Atti della Fondazione Giorgio Ronchi*, *21*, 199-217.

- Fischer, B., & Boch, R. (1983). Saccadic eye movements after extremely short reaction times in the monkey. *Brain Research*, 260, 21-26.
- Fischer, B., Boch, R., & Ramsperger, E. (1984). Express-saccades of the monkey: Effect of daily training on probability of occurrence and reaction time. *Experimental Brain Research*, 55, 232-242.
- Fischer, B., & Ramsperger, E. (1984). Human express saccades: Extremely short reaction times for goal directed eye movements. *Experimental Brain Research*, 57, 191-195.
- Fischer, B., & Weber, H. (1993). Express saccades and visual attention. *Behavioral and Brain Sciences*, 16, 553-610.
- Forbes, K., & Klein, R. M. (1996). The magnitude of the fixation offset effect with endogenously and exogenously controlled saccades. *Journal of Cognitive Neuroscience*, 8, 344-352.
- Friedman, H. R., Burman, D. D., Russo, E. C., Dias, E. C., Stanton, G. B., Shi, D., et al. (1997). Neuronal activity in primate frontal eye field during memory-guided saccades. *Society for Neuroscience Abstracts*, 23, 844.
- Fuchs, A. F., Kaneko, C. R. S., & Scudder, C. A. (1985). Brainstem control of saccadic eye movements. *Annual Reviews of Neuroscience*, 8, 307-337.
- Fuchs, A. F., & Luschei, E. S. (1970). Firing patterns of abducens neurons of alert monkeys in relationship to horizontal eye movement. *Journal of Neurophysiology*, 33, 382-392.
- Galfano, G., Betta, E., & Turatto, M. (2004). Inhibition of return in microsaccades. *Experimental Brain Research*, 159, 400-404.
- Gandhi, N. J., & Keller, E. L. (1999a). Activity of the brain stem omnipause neurons during saccades perturbed by stimulation of the primate superior colliculus. *Journal of Neurophysiology*, 82, 3254-3267.
- Gandhi, N. J., & Keller, E. L. (1999b). Comparison of saccades perturbed by stimulation of the rostral superior colliculus, the caudal superior colliculus, and the omnipause neuron region. *Journal of Neurophysiology*, 82, 3236-3253.
- Gerrits, H. J. M., de Haan, B., & Vendrik, A. J. H. (1966). Experiments with retinal stabilized images. Relations between the observations and neural data. *Vision Research*, 6, 427-440.
- Gerrits, H. J. M., & Vendrik, A. J. H. (1970). Artificial movements of a stabilized image. *Vision Research*, 10, 1443-1456.
- Gerrits, H. J. M., & Vendrik, A. J. H. (1974). The influence of stimulus movements on perception in parafoveal stabilized vision. *Vision Research*, 14, 175-180.
- Ginsborg, B. L. (1953). Small voluntary movements of the eye. *British Journal of Ophthalmology*, 37, 746-754.
- Girard, B., & Berthoz, A. (2005). From brainstem to cortex: Computational models of saccade generation circuitry. *Progress in Neurobiology*, 77, 215-251.
- Glezer, V. D. (1959). The eye as a scanning system. *The Journal of Physiology of USSR*, 45, 271-279.
- Godijn, R., & Theeuwes, J. (2002). Programming of endogenous and exogenous saccades: Evidence for a competitive integration model. *Journal of Experimental Psychology: Human Perception and Performance*, 28, 1039-1054.
- Gowen, E., & Abadi, R. V. (2005). Saccadic instabilities and voluntary saccadic behaviour. *Experimental Brain Research*, 164, 29-40.
- Gowen, E., Abadi, R. V., & Poliakoff, E. (2005). Paying attention to saccadic intrusions. *Cognitive Brain Research*, 25, 810-825.
- Greschner, M., Bongard, M., Rujan, P., & Ammermüller, J. (2002). Retinal ganglion cell synchronization by fixational eye movements improves feature estimation. *Nature Neuroscience*, 5, 441-447.
- Gur, M., Beylin, A., & Snodderly, D. M. (1997). Response variability of neurons in primary visual cortex (V1) of alert monkeys. *Journal of Neuroscience*, 17, 2914-2920.
- Gur, M., & Snodderly, D. M. (1997). Visual receptive fields of neurons in primary visual cortex (V1) move in space with the eye movements of fixation. *Vision Research*, 37, 257-265.

- Haddad, G. M., & Steinman, R. M. (1973). The smallest voluntary saccade: Implications for fixation. *Vision Research*, *13*, 1075-1086.
- Haddad, G. M., & Winterson, B. J. (1975). Effect of flicker on oculomotor performance. In G. Lennerstrand & P. Bach-y-Rita (Eds.), *Basic mechanisms of ocular motility and their clinical implications* (pp. 489-493). Oxford: Pergamon Press.
- Hafed, Z. M., & Clark, J. J. (2002). Microsaccades as an overt measure of covert attention shifts. *Vision Research*, *42*, 2533-2545.
- Hallett, P. E. (1978). Primary and secondary saccades to goals defined by instructions. *Vision Research*, *18*, 1279-1296.
- Hartridge, H., & Thomson, L. C. (1948). Methods of investigating eye movements. *British Journal of Ophthalmology*, *32*, 581-591.
- Heckenmueller, E. G. (1965). Stabilization of the retinal image: A review of method, effects, and theory. *Psychological Bulletin*, *63*, 157-169.
- Hess, W. R., Burgi, S., & Bucher, V. (1946). Motor function of tectal and tegmental area. *Monatsschrift für Psychiatrie und Neurologie*, *112*, 1-52.
- Higgins, G. C., & Stultz, K. F. (1953). Frequency and amplitude of ocular tremor. *Journal of the Optical Society of America*, *43*, 1136-1140.
- Hikosaka, O., Takikawa, Y., & Kawagoe, R. (2000). Role of the basal ganglia in the control of purposive saccadic eye movements. *Physiological Review*, *80*, 953-978.
- Horowitz, T. S., Fine, E. M., Fencsik, D. E., Yurgenson, S., & Wolfe, J. M. (in press). Fixational eye movements are not an index of covert attention. *Psychological Science*.
- Horwitz, G. D., & Albright, T. D. (2003). Short-latency fixational saccades induced by luminance increments. *Journal of Neurophysiology*, *90*, 1333-1339.
- Hsieh, P.-J., Caplovitz, G. P., & Tse, P. U. (submitted). Microsaccades reveal that motion-induced blindness is not due to sensory adaptation. *Neuroreport*.
- Huey, E. B. (1900). On the psychology and physiology of reading. I. *American Journal of Psychology*, *11*, 283-302.
- Ingle, D. (1973). Disinhibition of tectal neurons by pretectal lesions in the frog. *Science*, *180*, 422-424.
- Jay, M. F., & Sparks, D. L. (1984). Auditory receptive fields in primate superior colliculus shift with changes in eye position. *Nature*, *309*, 345-347.
- Jay, M. F., & Sparks, D. L. (1987). Sensorimotor integration in the primate superior colliculus. II. Coordinates of auditory signals. *Journal of Neurophysiology*, *57*, 35-55.
- Jurin, J. (1738). An essay on distinct and indistinct vision. In R. Smith (Ed.), *Complete system of opticks in four books* (Vol. 2, pp. 115-171). Cambridge: published by the author.
- Kadyrov, A., & Petrou, M. (2004). Reverse engineering the human vision system: A possible explanation for the role of microsaccades. In *Pattern recognition. Proceedings of the 17th International Conference on Pattern Recognition* (pp. 64-67).
- Kärnbach, C. (1991). Simple adaptive testing with the weighted up-down method. *Perception & Psychophysics*, *49*, 227-229.
- Kästner, A. G. (1755). Dr. Jurins Abhandlung zum deutlichen und undeutlichen Sehen. In *Vollständiger Lehrbegriff der Optik nach Robert Smith's Englischen mit Änderungen und Zusätzen ausgearbeitet von Abraham Gotthelf Kästner* (pp. 483-514). Altenburg: Richter.
- Keller, E. L. (1974). Participation of medial pontine reticular formation in eye movement generation in monkey. *Journal of Neurophysiology*, *37*, 316-332.
- Kelly, D. H. (1979a). Motion and vision. II. Stabilized spatio-temporal threshold surface. *Journal of the Optical Society of America*, *69*, 1340-1349.
- Kelly, D. H. (1979b). Motion and vision. I. Stabilized images of stationary gratings. *Journal of the Optical Society of America*, *69*, 1266-1274.
- Kelly, D. H. (1981). Disappearance of stabilized chromatic gratings. *Science*, *214*, 1257-1258.

- Kelly, D. H., & Burbeck, C. A. (1980). Motion and vision. III. Stabilized pattern adaptation. *Journal of the Optical Society of America*, 70, 1283-1289.
- King-Smith, P. E., & Riggs, L. A. (1978). Visual sensitivity to controlled motion of a line or edge. *Vision Research*, 18, 1509-1520.
- Kingstone, A., Fendrich, R., Wessinger, C. M., & Reuter-Lorenz, P. A. (1995). Are microsaccades responsible for the gap effect? *Perception & Psychophysics*, 57, 796-801.
- Kingstone, A., & Klein, R. M. (1993). What are human express saccades? *Perception & Psychophysics*, 54, 260-273.
- Klein, R. M. (2000). Inhibition of return. *Trends in Cognitive Sciences*, 4, 138-147.
- Koenderink, J. J., & Doorn, A. J. van. (1979). Spatiotemporal contrast detection threshold surface is bimodal. *Optics Letters*, 4, 32-34.
- Kopecz, K. (1995). Saccadic reaction times in gap/overlap paradigms: A model based on integration of intentional and visual information on neural, dynamic fields. *Vision Research*, 35, 2911-2925.
- Kopecz, K., & Schöner, G. (1995). Saccade motor planning by integrating visual information and pre-information on neural dynamic fields. *Biological Cybernetics*, 73, 49-60.
- Kowler, E. (1990). The role of visual and cognitive processes in the control of eye movements. In E. Kowler (Ed.), *Eye movements and their role in visual and cognitive processes* (pp. 1-70). New York: Elsevier.
- Kowler, E., Anderson, E., Doshier, B., & Blaser, E. (1995). The role of attention in the programming of saccades. *Vision Research*, 35, 1897-1916.
- Kowler, E., & Anton, S. (1987). Reading twisted text: Implications for the role of saccades. *Vision Research*, 27, 45-60.
- Kowler, E., & Martins, A. J. (1982). Eye movements in preschool children. *Science*, 215, 997-999.
- Kowler, E., & Sperling, G. (1980). Transient stimulation does not aid visual search: Implications for the role of saccades. *Perception & Psychophysics*, 27, 1-10.
- Kowler, E., & Sperling, G. (1983). Abrupt onsets do not aid visual search. *Perception & Psychophysics*, 34, 307-313.
- Kowler, E., & Steinman, R. M. (1977). The roll of small saccades in counting. *Vision Research*, 17, 141-146.
- Kowler, E., & Steinman, R. M. (1979). Miniature saccades: Eye movements that do not count. *Vision Research*, 19, 105-108.
- Kowler, E., & Steinman, R. M. (1980). Small saccades serve no useful purpose: Reply to a letter by R.W. Ditchburn. *Vision Research*, 20, 273-276.
- Krauskopf, J. (1957). Effect of retinal image motion on contrast thresholds for maintained vision. *Journal of the Optical Society of America*, 47, 740-744.
- Krauskopf, J., Cornsweet, T. N., & Riggs, L. A. (1960). Analysis of eye movements during monocular and binocular fixation. *Journal of the Optical Society of America*, 50, 572-578.
- Krauskopf, J., Graf, V., & Gaarder, K. (1966). Lack of inhibition during involuntary saccades. *American Journal of Psychology*, 79, 73-81.
- Krauzlis, R. J. (2003). Neuronal activity in the rostral superior colliculus related to the initiation of pursuit and saccadic eye movements. *Journal of Neuroscience*, 23, 4333-4344.
- Krauzlis, R. J., Basso, M. A., & Wurtz, R. H. (1997). Shared motor error for multiple eye movements. *Science*, 276, 1693-1695.
- Krauzlis, R. J., Basso, M. A., & Wurtz, R. H. (2000). Discharge properties of neurons in the rostral superior colliculus of the monkey during smooth-pursuit eye movements. *Journal of Neurophysiology*, 84, 876-891.
- Krauzlis, R. J., & Dill, N. (2002). Neural correlates of target choice for pursuit and saccades in the primate superior colliculus. *Neuron*, 35, 355-363.
- Krauzlis, R. J., & Miles, F. A. (1996). Release of fixation for pursuit and saccades in humans: Evidence for shared inputs acting on different neural substrates. *Journal of Neurophysiology*,

- 76, 2822-2833.
- Kulikowski, J. J. (1971). Effect of eye movements on the contrast sensitivity of spatio-temporal patterns. *Vision Research*, *11*, 261-273.
- Kustov, A. A., & Robinson, D. L. (1996). Shared neural control of attentional shifts and eye movements. *Nature*, *384*, 74-77.
- Latour, P. L. (1962). Visual threshold during eye movements. *Vision Research*, *2*, 261-262.
- Laubrock, J., Engbert, R., & Kliegl, R. (2005). Microsaccade dynamics during covert attention. *Vision Research*, *45*, 721-730.
- Laubrock, J., Engbert, R., Rolfs, M., & Kliegl, R. (in press). Microsaccades are an index of covert attention. *Psychological Science*.
- Lee, P. H., Helms, M. C., Augustine, G. J., & Hall, W. C. (1997). Role of intrinsic synaptic circuitry in collicular sensorimotor integration. *Proceedings of the National Academy of Sciences, USA*, *94*, 13299-13304.
- Lefèvre, P., Quaiia, C., & Optican, L. M. (1998). Distributed model of control of saccades by superior colliculus and cerebellum. *Neural Networks*, *11*, 1175-1190.
- Leopold, D. A., & Logothetis, N. K. (1998). Microsaccades differentially modulate neural activity in the striate and extrastriate visual cortex. *Experimental Brain Research*, *123*, 341-345.
- Levelt, W. J. M. (1967). Note on the distribution of dominance times in binocular rivalry. *British Journal of Psychology*, *58*, 143-145.
- Livingstone, M. S., Freeman, D. C., & Hubel, D. H. (1996). Visual responses in V1 of freely viewing monkeys. *Cold Spring Harbor Symposia On Quantitative Biology*, *61*, 27-37.
- Lorch, R. F., & Myers, J. L. (1990). Regression analyses of repeated measures data in cognitive research. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *16*, 149-157.
- Lord, M. P. (1951). Measurement of binocular eye movements of subjects in the sitting position. *British Journal of Ophthalmology*, *35*, 21-30.
- Lord, M. P., & Wright, W. D. (1948). Eye movements during monocular fixation. *Nature*, *162*, 25-26.
- Luschei, E. S., & Fuchs, A. F. (1972). Activity of brain stem neurons during eye movements of alert monkeys. *Journal of Neurophysiology*, *35*, 445-461.
- Malinov, I. V., Epelboim, J., Herst, A. N., & Steinman, R. M. (2000). Characteristics of saccades and vergence in two types of sequential looking tasks. *Vision Research*, *40*, 2083-2090.
- Mariotte, E. (1683). An account of two letters of Mr. Perault and Mr. Mariotte concerning vision. *Philosophical Transactions of the Royal Society*, *13*, 265-267.
- Marshall, W. H., & Talbot, S. A. (1942). Recent evidence for neural mechanisms in vision leading to a general theory of sensory acuity. *Biological Symposia*, *7*, 117-164.
- Martinez-Conde, S., Macknik, S. L., & Hubel, D. H. (2000). Microsaccadic eye movements and firing of single cells in the striate cortex of macaque monkeys. *Nature Neuroscience*, *3*, 251-258.
- Martinez-Conde, S., Macknik, S. L., & Hubel, D. H. (2002). The function of bursts of spikes during visual fixation in the awake primate lateral geniculate nucleus and primary visual cortex. *Proceedings of the National Academy of Sciences, USA*, *99*, 13920-13925.
- Martinez-Conde, S., Macknik, S. L., & Hubel, D. H. (2004). The role of fixational eye movements in visual perception. *Nature Reviews Neuroscience*, *5*, 229-240.
- Martinez-Conde, S., Macknik, S. L., Troncoso, X. G., & Dyar, T. A. (2006). Microsaccades counteract visual fading during fixation. *Neuron*, *49*, 297-305.
- Marx, E., & Trendelenburg, W. (1911). Über die Genauigkeit der Einstellung des Auges beim Fixieren. *Zeitschrift für Sinnesphysiologie*, *45*, 87-102.
- Matin, L., Matin, E., & Pearce, D. G. (1970). Eye movements in the dark during the attempt to maintain a prior fixation position. *Vision Research*, *10*, 837-857.
- Mayfrank, L., Mobashery, M., Kimmig, H., & Fischer, B. (1986). The role of fixation and visual attention in the occurrence of express saccades in man. *European Archives of Psychiatry and Neurological Sciences*, *235*, 269-275.

- McAllister, C. N. (1905). The fixation of points in the visual field. *Psychological Review Monograph Supplements*, 7, 17-53.
- McIlwain, J. T. (1982). Lateral spread of neural excitation during microstimulation in intermediate gray layer of cats superior colliculus. *Journal of Neurophysiology*, 47, 167-178.
- McPeck, R. M., & Schiller, P. H. (1994). The effects of visual scene composition on the latency of saccadic eye movements of the rhesus monkey. *Vision Research*, 34, 2293-2305.
- Meredith, M. A., & Ramoa, A. S. (1998). Intrinsic circuitry of the superior colliculus: Pharmacophysiological identification of horizontally oriented inhibitory interneurons. *Journal of Neurophysiology*, 79, 1597-1602.
- Michalik, M. (1987). Spektralanalysen des okulären Mikrotremors bei Hirnstammfunktionsstörungen. *Zeitschrift für Elektroenzephalographie und Elektromyographie*, 18, 20-26.
- Møller, F., Laursen, M. L., & Sjølie, A. K. (2006). The contribution of microsaccades and drifts in the maintenance of binocular steady fixation. *Graefes Archives for Clinical and Experimental Ophthalmology*, 244, 465-471.
- Moschovakis, A. K., Scudder, C. A., & Highstein, S. M. (1996). The microscopic anatomy and physiology of the mammalian saccadic system. *Progress in Neurobiology*, 50, 133-254.
- Müller, H. J., & Rabbitt, P. M. (1989). Reflexive and voluntary orienting of visual attention: Time course of activation and resistance to interruption. *Journal of Experimental Psychology: Human Perception and Performance*, 15, 315-330.
- Munoz, D. P. (2002). Saccadic eye movements: Overview of neural circuitry. In J. Hyönä, D. P. Munoz, W. Heide, & R. Radach (Eds.), *Progress in brain research* (Vol. 140, pp. 89-96). Amsterdam: Elsevier.
- Munoz, D. P., Dorris, M. C., Paré, M., & Everling, S. (2000). On your mark get set: Brainstem circuitry underlying saccadic initiation. *Canadian Journal of Physiology and Pharmacology*, 78, 934-944.
- Munoz, D. P., & Everling, S. (2004). Look away: The anti-saccade task and the voluntary control of eye movement. *Nature Reviews Neuroscience*, 5, 218-228.
- Munoz, D. P., & Fecteau, J. H. (2002). Vying for dominance: Dynamic interactions control visual fixation and saccadic initiation in the superior colliculus. In J. Hyönä, D. P. Munoz, W. Heide, & R. Radach (Eds.), *Progress in brain research* (Vol. 140, pp. 3-19). Amsterdam: Elsevier.
- Munoz, D. P., & Guitton, D. (1991). Control of orienting gaze shifts by the tectoreticulospinal system in the head-free cat. II Sustained discharges during motor preparation and fixation. *Journal of Neurophysiology*, 66, 1624-1641.
- Munoz, D. P., & Istvan, P. J. (1998). Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. *Journal of Neurophysiology*, 79, 1193-1209.
- Munoz, D. P., & Schall, J. D. (2003). Concurrent distributed control of saccades. In W. C. Hall & A. K. Moschovakis (Eds.), *The oculomotor system: New approaches for studying sensorimotor integration* (pp. 55-82). Boca Raton, FL: CRC Press.
- Munoz, D. P., & Wurtz, R. H. (1992). Role of the rostral superior colliculus in active visual fixation and execution of express saccades. *Journal of Neurophysiology*, 67, 1000-1002.
- Munoz, D. P., & Wurtz, R. H. (1993a). Fixation cells in monkey superior colliculus I. Characteristics of cell discharge. *Journal of Neurophysiology*, 70, 559-575.
- Munoz, D. P., & Wurtz, R. H. (1993b). Fixation cells in monkey superior colliculus II. Reversible activation and deactivation. *Journal of Neurophysiology*, 70, 576-589.
- Munoz, D. P., & Wurtz, R. H. (1995a). Saccade-related activity in monkey superior colliculus I. Characteristics of burst and buildup cells. *Journal of Neurophysiology*, 73, 2313-2333.
- Munoz, D. P., & Wurtz, R. H. (1995b). Saccade-related activity in monkey superior colliculus II. Spread of activity during saccades. *Journal of Neurophysiology*, 73, 2334-2348.
- Murphy, B. J., Haddad, G. M., & Steinman, R. M. (1974). Simple forms and fluctuations of the line of sight: Implications for motor theories of form processing. *Perception & Psychophysics*, 16, 557-563.

- Nachmias, J. (1959). Two-dimensional motion of the retinal image during monocular fixation. *Journal of the Optical Society of America*, *49*, 901-908.
- Nachmias, J. (1961). Determiners of the drift of the eye during monocular fixation. *Journal of the Optical Society of America*, *51*, 761-766.
- Ohtsuka, K., Mukuno, K., Ukai, K., & Ishikawa, S. (1986). The origin of square wave jerks: Conditions of fixation and microsaccades. *Japanese Journal of Ophthalmology*, *30*, 209-215.
- Opris, I., Barborica, A., & Ferrera, V. P. (2001). On the gap effect for saccades evoked by electrical microstimulation of frontal eye fields in monkeys. *Experimental Brain Research*, *138*, 1-7.
- Pannasch, S., Dornhöfer, S. M., Unema, P. J. A., & Velichkovsky, B. M. (2001). The omnipresent prolongation of visual fixations: Saccades are inhibited by changes in situation and in subject's activity. *Vision Research*, *41*, 3345-3351.
- Paré, M., & Guitton, D. (1994). The fixation area of the cat superior colliculus: Effects of electrical stimulation and direct connection with brainstem omnipause neurons. *Experimental Brain Research*, *101*, 109-122.
- Paré, M., & Munoz, D. P. (1996). Saccadic reaction time in the monkey: Advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. *Journal of Neurophysiology*, *76*, 3666-3681.
- Pelli, D. G. (1997). The VideoToolbox software for visual psychophysics: Transforming numbers into movies. *Spatial Vision*, *10*, 437-442.
- Pierrot-Deseilligny, C., Milea, D., & Müri, R. M. (2004). Eye movement control by the cerebral cortex. *Current Opinion in Neurology*, *17*, 17-25.
- Posner, M. I. (1980). Orientation of attention. The VIIIth Sir Frederic Bartlett lecture. *Quarterly Journal of Experimental Psychology*, *32A*, 3-25.
- Pratt, J., Bekkering, H., & Leung, M. (2000). Estimating the components of the gap effect. *Experimental Brain Research*, *130*, 258-263.
- Pritchard, R. M. (1961). Stabilized images on the retina. *Scientific American*, *204*, 72-78.
- Pritchard, R. M., Heron, H., & Hebb, D. O. (1960). Visual perception approached by the method of stabilized images. *Canadian Journal of Psychology*, *14*, 67-77.
- Proskuryakova, N. G., & Shakhnovich, A. R. (1967). Quantitative characteristics of fixation micromovements of the eye. *Biophysics*, *13*, 117-126.
- Puckett, J. W., & Steinman, R. M. (1969). Tracking eye movements with and without saccadic correction. *Vision Research*, *9*, 695-703.
- Ratliff, F. (1952). The role of physiological nystagmus in monocular acuity. *Journal of Experimental Psychology*, *43*, 163-172.
- Ratliff, F. (1958). Stationary retinal image requiring no attachments to the eye. *Journal of the Optical Society of America*, *48*, 274-275.
- Ratliff, F., & Riggs, L. A. (1950). Involuntary motions of the eye during monocular fixation. *Journal of Experimental Psychology*, *40*, 687-701.
- Rattle, J. D., & Foley-Fisher, J. A. (1968). A relationship between vernier acuity and intersaccadic interval. *Optica Acta*, *15*, 617-620.
- Raybourn, M. S., & Keller, E. L. (1977). Colliculoreticular organization in primate oculomotor system. *Journal of Neurophysiology*, *40*, 861-878.
- Reingold, E. M., & Stampe, D. M. (1999). Saccadic inhibition in complex visual tasks. In W. Becker, H. Deubel, & T. Mergner (Eds.), *Current oculomotor research: Physiological and psychological aspects* (pp. 249-255). London: Plenum.
- Reingold, E. M., & Stampe, D. M. (2000). Saccadic inhibition and gaze contingent research paradigms. In A. Kennedy, R. Radach, D. Heller, & J. Pynte (Eds.), *Reading as a perceptual process* (pp. 119-145). Amsterdam: Elsevier.
- Reingold, E. M., & Stampe, D. M. (2002). Saccadic inhibition in voluntary and reflexive saccades. *Journal of Cognitive Neuroscience*, *14*, 371-388.

- Reingold, E. M., & Stampe, D. M. (2003). Using the saccadic inhibition paradigm to investigate saccadic control in reading. In J. Hyönä, R. Radach, & H. Deubel (Eds.), *The mind's eye: Cognitive and applied aspects of eye movement research* (pp. 347-360). Amsterdam: Elsevier.
- Reingold, E. M., & Stampe, D. M. (2004). Saccadic inhibition in reading. *Journal of Experimental Psychology: Human Perception and Performance*, *30*, 194-211.
- Reuter-Lorenz, P. A., Hughes, H. C., & Fendrich, R. (1991). The reduction of saccadic latency by prior offset of the fixation point: An analysis of the gap effect. *Perception & Psychophysics*, *49*, 167-175.
- Richter, E. M., Engbert, R., & Kliegl, R. (submitted). The perpetual gap paradigm: Evidence for an autonomous timer for saccade generation.
- Riggs, L. A. (1965). Visual acuity. In *Vision and visual perception* (pp. 321-349). New York: Wiley.
- Riggs, L. A., Armington, J. C., & Ratliff, F. (1954). Motions of the retinal image during fixation. *Journal of the Optical Society of America*, *44*, 315-321.
- Riggs, L. A., & Ratliff, F. (1951). Visual acuity and the normal tremor of the eyes. *Science*, *114*, 17-18.
- Riggs, L. A., & Ratliff, F. (1952). The effects of counteracting the normal movements of the eye. *Journal of the Optical Society of America*, *42*, 872-873.
- Riggs, L. A., Ratliff, F., Cornsweet, J. C., & Cornsweet, T. N. (1953). The disappearance of steadily fixated visual test objects. *Journal of the Optical Society of America*, *43*, 495-501.
- Rizzolatti, G., Buchtel, H. A., Camarda, R., & Scandolara, C. (1980). Neurons with complex visual properties in the superior colliculus of the macaque monkey. *Experimental Brain Research*, *38*, 37-42.
- Robinson, D. A. (1970). Oculomotor unit behavior in the monkey. *Journal of Neurophysiology*, *33*, 393-404.
- Robinson, D. A. (1972). Eye movements evoked by collicular stimulation in the alert monkey. *Vision Research*, *12*, 1795-1808.
- Robinson, D. L., & McClurkin, J. W. (1989). The visual superior colliculus and pulvinar. In *The neurobiology of saccadic eye movements* (pp. 337-360). Amsterdam: Elsevier.
- Robinson, F. R., & Fuchs, A. F. (2001). The role of the cerebellum in voluntary eye movements. *Annual Reviews of Neuroscience*, *24*, 981-1004.
- Rolfs, M., Engbert, R., & Kliegl, R. (2004). Microsaccade orientation supports attentional enhancement opposite a peripheral cue. *Psychological Science*, *15*, 705-707.
- Rolfs, M., Engbert, R., & Kliegl, R. (2005). Crossmodal coupling of oculomotor control and spatial attention in vision and audition. *Experimental Brain Research*, *166*, 427-439.
- Rolfs, M., Laubrock, J., & Kliegl, R. (2006). Shortening and prolongation of saccade latencies following microsaccades. *Experimental Brain Research*, *169*, 369-376.
- Rolfs, M., & Vitu, F. (submitted). On the limited role of target onset in the gap task: Support for the motor-preparation hypothesis.
- Rucci, M., & Casile, A. (2004). Decorrelation of neural activity during fixational instability: Possible implications for the refinement of V1 receptive fields. *Visual Neuroscience*, *21*, 725-738.
- Rucci, M., & Casile, A. (2005). Fixational instability and natural image statistics: Implications for early visual representations. *Network: Computation in Neural Systems*, *16*, 121-138.
- Rucci, M., & Desbordes, G. (2003). Contributions of fixational eye movements to the discrimination of briefly presented stimuli. *Journal of Vision*, *3*, 852-864.
- Rucci, M., Edelman, G. M., & Wray, J. (2000). Modeling LGN responses during free-viewing: A possible role of microscopic eye movements in the refinement of cortical orientation selectivity. *Journal of Neuroscience*, *20*, 4708-4720.
- Sabrin, H. W., & Kertesz, A. E. (1980). Microsaccadic eye movements and binocular rivalry. *Perception & Psychophysics*, *28*, 150-154.
- Sabrin, H. W., & Kertesz, A. E. (1983). The effect of imposed fixational eye movements on binocular rivalry. *Perception & Psychophysics*, *34*, 155-157.

- Sansbury, R. V., Skavenski, A. A., Haddad, G. M., & Steinman, R. M. (1973). Normal fixation of eccentric targets. *Journal of the Optical Society of America*, *63*, 612-614.
- Saslow, M. G. (1967). Effects of components of displacement-step stimuli upon latency for saccadic eye movement. *Journal of the Optical Society of America*, *57*, 1024-1029.
- Schall, J. D. (1997). Visuomotor areas of the frontal lobe. In K. S. Rockland, A. Peters, & J. H. Kaas (Eds.), *Extrastriate cortex of primates* (Vol. 12, pp. 527-638). New York: Plenum Press.
- Schall, J. D., Morel, A., King, D. J., & Bullier, J. (1995). Topography of visual cortex connections with frontal eye field in macaque: Convergence and segregation of processing streams. *Journal of Neuroscience*, *15*, 4464-4487.
- Schiller, P. H., & Malpeli, J. G. (1977). Properties and tectal projections of monkey retinal ganglion cells. *Journal of Neurophysiology*, *40*, 428-445.
- Schiller, P. H., Sandell, J. H., & Maunsell, J. H. (1987). The effect of frontal eye field and superior colliculus lesions on saccadic latencies in the rhesus monkey. *Journal of Neurophysiology*, *57*, 1033-1049.
- Schor, C., & Hallmark, W. (1978). Slow control of eye position in strabismic amblyopia. *Investigative Ophthalmology*, *17*, 577-581.
- Scudder, C. A., Kaneko, C. R. S., & Fuchs, A. F. (2002). The brainstem burst generator for saccadic eye movements: A modern synthesis. *Experimental Brain Research*, *142*, 439-462.
- Shakhnovich, A. R., & Thomas, J. G. (1977). Micro-tremor of the eyes of comatose patients. *Electroencephalography and Clinical Neurophysiology*, *42*, 117-119.
- Sharpe, C. R. (1971). A fresh approach to stabilized retinal images. Part II. *Journal of Physiology*, *217*, 9P-10P.
- Sharpe, C. R. (1972). The visibility and fading of thin lines visualized by their controlled movement across the retina. *Journal of Physiology*, *222*, 113-134.
- Shortess, G. K., & Krauskopf, J. (1961). Role of involuntary eye movements in stereoscopic acuity. *Journal of the Optical Society of America*, *51*, 555-559.
- Shurcliff, W. A. (1959). New visual phenomenon: The greenish-yellow blotch. *Journal of the Optical Society of America*, *49*, 1041-1048.
- Simon, F., Schulz, E., Rassow, B., & Haase, W. (1984). Binocular micromovement recording of human eyes: – methods. *Graefe's Archives for Clinical and Experimental Ophthalmology*, *221*, 293-298.
- Skavenski, A. A. (1971). Extraretinal correction and memory for target position. *Vision Research*, *11*, 743-746.
- Skavenski, A. A. (1972). Inflow as a source of extraretinal eye position information. *Vision Research*, *12*, 221-229.
- Skavenski, A. A., Hansen, R. M., Steinman, R. M., & Winterson, B. J. (1979). Quality of retinal image stabilization during small natural and artificial body rotations in man. *Vision Research*, *19*, 675-683.
- Skavenski, A. A., Robinson, D. A., Steinman, R. M., & Timberlake, G. T. (1975). Miniature eye movements of fixation in rhesus monkeys. *Vision Research*, *15*, 1269-1273.
- Skavenski, A. A., & Steinman, R. M. (1970). Control of eye position in the dark. *Vision Research*, *10*, 193-203.
- Snodderly, D. M. (1987). Effects of light and dark environments on macaque and human fixational eye movements. *Vision Research*, *27*, 401-415.
- Snodderly, D. M., Kagan, I., & Gur, M. (2001). Selective activation of visual cortex neurons by fixational eye movements: Implications for neural coding. *Visual Neuroscience*, *18*, 259-277.
- Snodderly, D. M., & Kurtz, D. (1985). Eye position during fixation tasks: Comparison of macaque and human. *Vision Research*, *25*, 83-98.
- Sommer, M. A., & Wurtz, R. H. (2002). A pathway in primate brain for internal monitoring of movements. *Science*, *296*, 1480-1482.

- Sommer, M. A., & Wurtz, R. H. (2004a). What the brain stem tells the frontal cortex. I. Oculomotor signals sent from superior colliculus to frontal eye field via mediodorsal thalamus. *Journal of Neurophysiology*, *91*, 1381-1402.
- Sommer, M. A., & Wurtz, R. H. (2004b). What the brain stem tells the frontal cortex. II. Role of the SC-MD-FEF pathway in corollary discharge. *Journal of Neurophysiology*, *91*, 1403-1423.
- Sparks, D. L. (2002). The brainstem control of saccadic eye movements. *Nature Reviews Neuroscience*, *3*, 952-964.
- Sparks, D. L., Holland, R., & Guthrie, B. L. (1976). Size and distribution of movement fields in the monkey superior colliculus. *Brain Research*, *113*, 21-34.
- Spauschuss, A., Marsden, J., Halliday, D. M., Rosenberg, J. R., & Brown, P. (1999). The origin of ocular microtremor in man. *Experimental Brain Research*, *126*, 556-562.
- St. Cur, G. J., & Fender, D. H. (1969). The interplay of drifts and flicks in binocular fixation. *Vision Research*, *9*, 245-265.
- Stampe, D. M., & Reingold, E. M. (2002). Influence of stimulus characteristics on the latency of saccadic inhibition. In J. Hyönä, D. P. Munoz, W. Heide, & R. Radach (Eds.), *Progress in brain research* (Vol. 140, pp. 73-87). Amsterdam: Elsevier.
- Stein, B. E., & Meredith, M. A. (1993). *The merging of the senses*. Cambridge, MA: MIT Press.
- Steinman, R. M. (1965). Effect of target size, luminance, and color on monocular fixation. *Journal of the Optical Society of America*, *55*, 1158-1165.
- Steinman, R. M. (1976). Role of eye movements in maintaining a phenomenally clear and stable world. In R. A. Monty & J. W. Senders (Eds.), *Eye movements and psychological processes* (pp. 121-149). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Steinman, R. M. (2003). Gaze control under natural conditions. In L. M. Chalupa & J. S. Werner (Eds.), *The visual neurosciences* (Vol. 2, pp. 1339-1356). Cambridge: MIT Press.
- Steinman, R. M., & Collewijn, H. (1978). How our two eyes are held steady. *Journal of the Optical Society of America*, *68*, 1359.
- Steinman, R. M., & Collewijn, H. (1980). Binocular retinal image motion during active head rotation. *Vision Research*, *20*, 415-429.
- Steinman, R. M., Cunitz, R. J., Timberlake, G. T., & Herman, M. (1967). Voluntary control of microsaccades during maintained monocular fixation. *Science*, *155*, 1577-1579.
- Steinman, R. M., Haddad, G. M., Skavenski, A. A., & Wyman, D. (1973). Miniature eye movement. *Science*, *181*, 810-819.
- Steinman, R. M., & Levinson, J. Z. (1990). The role of eye movement in the detection of contrast and spatial detail. In E. Kowler (Ed.), *Eye movements and their role in visual and cognitive processes* (pp. 115-212). New York: Elsevier.
- Steinman, R. M., Pizlo, Z., Forofonova, T. I., & Epelboim, J. (2003). One fixates accurately in order to see clearly not because one sees clearly. *Spatial Vision*, *16*, 225-241.
- Steinman, R. M., Skavenski, A. A., & Sansbury, R. V. (1969). Effect of lens accommodation on holding the eye in place without saccades. *Vision Research*, *9*, 629-631.
- Strassman, A., Evinger, C., McCreary, R. A., Baker, R. G., & Highstein, S. M. (1987). Anatomy and physiology of intracellularly labelled omnipause neurons in the cat and squirrel monkey. *Experimental Brain Research*, *67*, 436-440.
- Supèr, H., van der Togt, C., Spekrijse, H., & Lamme, V. A. F. (2004). Correspondence of presaccadic activity in the monkey primary visual cortex with saccadic eye movements. *Proceedings of the National Academy of Sciences, USA*, *101*, 3230-3235.
- Tam, W. J., & Stelmach, L. B. (1993). Viewing behavior: Ocular and attentional disengagement. *Perception & Psychophysics*, *54*, 211-222.
- ten Doesschate, J. (1954). A new form of physiological nystagmus. *Ophthalmologica*, *127*, 65-73.
- Thiel, M., Romano, M. C., Kurths, J., Rolfs, M., & Kliegl, R. (2006). Twin surrogates to test for complex synchronisation. *Europhysics Letters*, *75*, 535-541.

- Thiel, M., Romano, M. C., Kurths, J., Rolfs, M., & Kliegl, R. (submitted). Recovering dynamics from recurrences.
- Timberlake, G. T., Wyman, D., Skavenski, A. A., & Steinman, R. M. (1972). The oculomotor error signal in the fovea. *Vision Research*, *12*, 1059-1064.
- Trappenberg, T. P., Dorris, M. C., Munoz, D. P., & Klein, R. M. (2001). A model of saccade initiation based on the competitive integration of exogenous and endogenous signals in the superior colliculus. *Journal of Cognitive Neuroscience*, *13*, 256-271.
- Troxler, I. P. V. (1804). Über das Verschwinden gegebner Gegenstände innerhalb unseres Gesichtskreises. *Ophthalmologische Bibliothek*, *2*, 1-53.
- Tse, P. U., Sheinberg, D. L., & Logothetis, N. K. (2002). Fixational eye movements are not affected by abrupt onsets that capture attention. *Vision Research*, *42*, 1663-1669.
- Tse, P. U., Sheinberg, D. L., & Logothetis, N. K. (2003). Attentional enhancement opposite a peripheral flash revealed using change blindness. *Psychological Science*, *14*, 91-99.
- Tse, P. U., Sheinberg, D. L., & Logothetis, N. K. (2004). The distribution of microsaccade directions need not reveal the location of attention. *Psychological Science*, *15*, 708-710.
- Tulunay-Keesey, U. (1960). Effects of involuntary eye movements on visual acuity. *Journal of the Optical Society of America*, *50*, 769-774.
- Tulunay-Keesey, U., & Riggs, L. A. (1962). Visibility of Mach bands with imposed motions of the retinal image. *Journal of the Optical Society of America*, *52*, 719-720.
- Valsecchi, M., Betta, E., & Turatto, M. (in press). Visual oddballs induce prolonged microsaccadic inhibition. *Experimental Brain Research*.
- van Dam, L. C. J., & van Ee, R. (2006). Retinal image shifts, but not eye movements per se, cause alternations in awareness during binocular rivalry. *Journal of Vision*, *6*, 1172-1179.
- van Gisbergen, J. A. M., & Robinson, D. A. (1977). Generation of micro- and macrosaccades by burst neurons in the monkey. In R. Baker & A. Berthoz (Eds.), *Control of gaze by brain stem neurons, developments in neuroscience* (Vol. 1, pp. 301-308). Amsterdam: Elsevier.
- van Gisbergen, J. A. M., Robinson, D. A., & Gielen, S. (1981). A quantitative analysis of generation of saccadic eye movements by burst neurons. *Journal of Neurophysiology*, *45*, 417-442.
- Vedel-Jensen, N. (1966). Pathological ocular fixation instability. *Acta Ophthalmologica*, *44*, 481-493.
- Volkman, F. C. (1962). Vision during voluntary saccadic eye movements. *Journal of the Optical Society of America*, *52*, 571-578.
- Volkman, F. C., Schick, A. M. L., & Riggs, L. A. (1968). Time course of visual inhibition during voluntary saccades. *Journal of the Optical Society of America*, *58*, 562-569.
- von Helmholtz, H. (1924). *Treatise on physiological optics - Translated from the third German edition* (Vol. 2; J. P. C. Southall, Ed.). Menasha, Wisconsin: The Optical Society of America. (originally published as *Handbuch der physiologischen Optik* [1866; Leipzig: Voss])
- Wade, N. J., & Tatler, B. W. (2005). *The moving tablet of the eye*. Oxford: Oxford University Press.
- Wenban-Smith, M. G., & Findlay, J. M. (1991). Express saccades: Is there a separate population in humans? *Experimental Brain Research*, *87*, 218-222.
- West, P., & Harris, C. M. (1993). Are express saccades anticipatory? *Behavioral and Brain Sciences*, *16*, 593-594.
- Winterson, B. J., & Collewijn, H. (1976). Microsaccades during finely guided visuomotor tasks. *Vision Research*, *16*, 1387-1390.
- Winterson, B. J., & Robinson, D. A. (1975). Fixation by the alert but solitary cat. *Vision Research*, *15*, 1349-1352.
- Wurtz, R. H., & Goldberg, M. E. (1972). Activity of superior colliculus in behaving monkey. III. Cells discharging before eye movements. *Journal of Neurophysiology*, *35*, 575-586.
- Yamazaki, A. (1968). Electrophysiological study on "flick" eye movements during fixation. *Acta Societatis Ophthalmologicae Japonicae*, *72*, 2446-2459.
- Yarbus, A. L. (1957a). A new method of studying the activity of various parts of the retina. *Biophysics*, *2*, 165-167.

- Yarbus, A. L. (1957b). The perception of an image fixed with respect to the retina. *Biophysics*, 2, 683-690.
- Yarbus, A. L. (1959). The perception of images moving across the retina at a fixed speed. *Biophysics*, 4, 70-80.
- Yarbus, A. L. (1967). *Eye movements and vision*. New York: Plenum Press.
- Zee, D. S., & Robinson, D. A. (1979). A hypothetical explanation of saccadic oscillations. *Annual Neurology*, 5, 405-414.
- Zuber, B. L., Crider, A., & Stark, L. (1964). Saccadic suppression associated with microsaccades. *Quarterly Progress Report*, 74, 244-249.
- Zuber, B. L., & Stark, L. (1966). Saccadic suppression: Elevation of visual threshold associated with saccadic eye movements. *Experimental Neurology*, 16, 65-79.
- Zuber, B. L., Stark, L., & Cook, G. (1965). Microsaccades and the velocity-amplitude relationship for saccadic eye movements. *Science*, 150, 1459-1460.