Spatio-temporal representations during eye movements and their neuronal correlates

Raum-zeitliche Repräsentation während Augenbewegungen und ihre neuronalen Grundlagen

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Zusammenfassung

Unsere visuelle Wahrnehmung durchläuft während rascher Augenbewegungen, sogenannter Sakkaden, eine Reihe spezifischer Veränderungen. Die Fähigkeit, Helligkeitskontraste wahrzunehmen (Luminanzkontrastsensitivität), ist während Sakkaden reduziert (sakkadische Suppression) und kurz eingeblendete Reize können in Richtung der Sakkade oder zum Ziel der Sakkade hin verschoben wahrgenommen werden. Auch die Wahrnehmung der Zeit ist beeinflusst. Die zeitliche Reihenfolge zweier Reize kann invertiert und die Zeit zwischen den Reizen als verkürzt wahrgenommen werden. Die Dauer einer visuellen Reizänderung in der Nähe des Sakkadenziels kann hingegen als länger wahrgenommen werden (Chronostasis), wenn diese Änderung während einer Sakkade beginnt. In dieser Arbeit habe ich die raum-zeitlichen Profile von Änderungen der menschlichen perisakkadischen Wahrnehmung und mögliche damit zusammenhängende Veränderungen der neuronalen Aktivität im medio temporalen Areal (MT) des Rhesusaffen während Sakkaden untersucht. Ich habe herausgefunden, dass die sakkadische Suppression in einem augenzentrierten Koordinatensystem auftritt und ich konnte zeigen, dass die gefundenen Variationen der sakkadischen Suppression mit dem Stimulusort das raum-zeitliche Profil einer weiteren Wahrnehmungsänderung zu beeinflussen scheinen: Chronostasis. Die Daten widerlegen frühere Annahmen, dass Chronostasis nur eine lokal begrenzte Verzerrung der Zeitwahrnehmung ist. Sie zeigen vielmehr, dass sie im gesamten visuellen Feld auftritt. Zudem ergaben meine Messungen, dass Chronostasis nicht von der Augenbewegung selbst abhängt, sondern eine Konsequenz der sakkadenbedingten Änderung des Abbildes der visuellen Reize ist. In dieser Hinsicht unterscheidet sich Chronostasis klar von anderen perisakkadischen Wahrnehmungsänderungen wie der sakkadischen Suppression und der Kompression der Reizlokali-

sierung um das Sakkadenziel herum. Auf der Suche nach einer neuronalen Basis dieser Ergebnisse bezüglich der sakkadischen Suppression und der Zeitwahrnehmung habe ich Einzelzellsignale in MT eines wachen, sich verhaltenden Makaken gemessen. Die Ergebnisse meiner Untersuchungen liefern relevante Erkenntnisse über die Verarbeitung stationärer visueller Reize und Paare solcher Reize während Fixation und Sakkaden in MT. Die neuronalen Antworten auf den zweiten von zwei Reizen waren stark reduziert und die Antwortlatenzen erhöht; selbst bei einem zeitlichen Abstand der beiden Reize von ungefähr 100ms. Diese erhöhte Latenz ist ein wichtiger Unterschied zu den Berichten der zeitlichen Dynamik in anderen Hirnarealen des Makaken wie dem frontalen Augenfeld im Frontalcortex und dem Colliculus superior im Mittelhirn. Während Sakkaden blieben die Latenzen für Antworten auf einzelne helle Reize jedoch unverändert, während die Antwortstärke für Reize, die in der zweiten Hälfte der Sakkade gezeigt wurden, reduziert war. Der Vergleich mit Antworten auf Reize unterschiedlicher Helligkeit während Fixation zeigte, dass die perisakkadische Reduktion der Antworten in MT quantitativ zu bekannten, psychophysikalisch gemessenen, perisakkadischen Reduktionen der Kontrastsensitivität passt. Durch einen vorhergehenden Reiz bereits reduzierte Antworten schienen während Sakkaden nicht zusätzlich reduziert. Dies könnte auf eine Interaktion der beiden zugrundeliegenden Mechanismen hindeuten. Die sakkadische Suppression tritt also in einem augenzentrierten Koordinatensystem auf, wobei die Reduktion der Kontrastsensitivität vergleichbar mit der Reduktion der neuronalen Aktivität in Area MT eines Makaken war. Die perisakkadische Überschätzung von Reizdauern wird durch die sakkadische Suppression und die sakkadeninduzierten visuellen Änderungen beeinflusst, ist jedoch selbst nicht abhängig von Augenbewegungssignalen.

Summary

During fast ballistic eye movements, so-called saccades, our visual perception undergoes a range of distinct changes. Sensitivity to luminance contrasts is reduced (saccadic suppression) and the localization of stimuli can be shifted in the direction of a saccade or is compressed around the saccade target. The temporal order of two stimuli can be perceived as inverted and the duration in between can be underestimated. The duration of a target change close to the saccade target can be overestimated, when the change occurs during the saccade (chronostasis). In my thesis I investigated the spatial and temporal profiles of peri-saccadic changes in human visual perception and explored how these might result from changes in neural activity of the macaque middle temporal area (MT). I found that peri-saccadic contrast sensitivity was only reduced by a constant factor across space when the data was analyzed in retinal coordinates (as opposed to screen coordinates), indicating that saccadic suppression occurs in an eye-centered frame of reference. I demonstrated that the found variations of saccadic suppression with the location of the stimulus appear to cause variations in the spatio-temporal pattern of another peri-saccadic misperception: chronostasis. I was able to show that, unlike previously assumed, the saccadic overestimation of time is not a spatially localized disturbance of time perception but instead spans across the whole visual field. I further determined that chronostasis is not dependent on the eye movement itself, but is rather a consequence of the visual stimulation induced by it. This result clearly segregates chronostasis from other peri-saccadic perceptual changes like saccadic suppression and the compression of space. To relate these findings to a potential neuronal basis of saccadic suppression and time perception, I measured neuronal responses of single cells in MT of an awake behaving macaque. The results provide relevant insight into the processing of sta-

tionary stimuli and pairs of stimuli during fixation and saccades in MT. Responses to the second of a pair of stimuli were strongly suppressed and response latencies increased even at onset asynchronies of about 100ms. The increase in latency is an important difference to the temporal dynamics previously reported in other brain areas as the frontal eye field in the frontal cortex and the superior colliculus in the midbrain. During saccades, response latencies to single high luminance stimuli remained unchanged. For stimuli shown during the second half of the saccade, the average responses were reduced. By comparison with responses to single stimuli at different luminance levels during fixation, I was able to show that the peri-saccadic response reduction found in MT quantitatively fit to what could be expected from known psychophysical measurements of peri-saccadic contrast sensitivity. Responses that were already reduced due to a preceding stimulus were however not subject to further reductions, indicating a possible interaction of these two response modulations. Saccadic suppression occurs in an eye-centered frame of reference with changes in perception compatible to changes in single cell activity in the macaque monkey MT. The peri-saccadic overestimation of time is influenced by saccadic suppression and the saccade-induced visual changes, but is not dependent on eye-movement related signals.

Contents

Abbreviations

7a	Area **7a**
fMRI	**F**unctional **m**agnet **r**esonance **i**maging
FP	**F**ixation **p**oint
LGN	**L**ateral **g**eniculate **n**ucleus
LIP	**L**ateral **i**ntra**p**arietal area
M	**M**agnocellular
MST	**M**edial **s**uperior **t**emporal area
MT	**M**iddle **t**emporal area
P	**P**arvocellular
PSE	**P**oint of **s**ubjective **e**quivalence
RF	**R**eceptive **f**ield
SOA	**S**timulus **o**nset **a**synchrony
ST	**S**accade **t**arget
TMS	**T**ranscranial **m**agnetic **s**timulation
V1	Primary **v**isual cortex
VIP	**V**entral **i**ntra**p**arietal area
SC	**S**uperior **c**olliculus
SEM	**S**tandard **e**rror of the **m**ean

General Introduction

In the retina of most primates a small region, the fovea, with very high spatial acuity has developed. With increasing distance from the fovea, acuity decreases rapidly (Green, 1970). As a consequence we have to move our eyes in order to analyze different objects of interest at high resolution or to keep the image of a moving object in the fovea. One type of eye movements that serve this purpose are saccades. They are fast ballistic eye movements reaching speeds of up to 1000°/s. Yet, we do not perceive the saccade-induced fast motion of the image of the outside world projected onto the retina. The details of the mechanisms that help to maintain a stable percept of the world and prevent the eye movements from disrupting perception are subject of ongoing neuroscientific research.

Pathways of visual Information

In the retina the image of the visual world is transformed into electrical and chemical signals. These signals are processed and different aspects of information are separated. At the end of the retinal processing, axons of several types of so called ganglion cells leave the eye and connect to other parts of the brain. In the human retina about 90% of ganglion cells are the parvocellular (P) ganglion cells (Dacey & Petersen, 1992), which have comparably small dendritic trees. Another 5% of the cells are magnocellular (M); they have larger dendritic trees than P cells at identical distance from the fovea. The remaining 5% are made up of different types. The size of the dendritic trees increases with the distance from the fovea for M as well as P cells (Figure 1A). The different cell types have different response characteristics. While M cells quickly adapt to a stationary stimulus, responses of P cells are more sustained. Also, P cells are sensitive to higher spatial frequencies and are typically selective to chromatic con-

trast. M cells receive input from a larger number of photoreceptors and it is mostly the difference in luminance that determines their activity (Benardete & Kaplan, 1999).

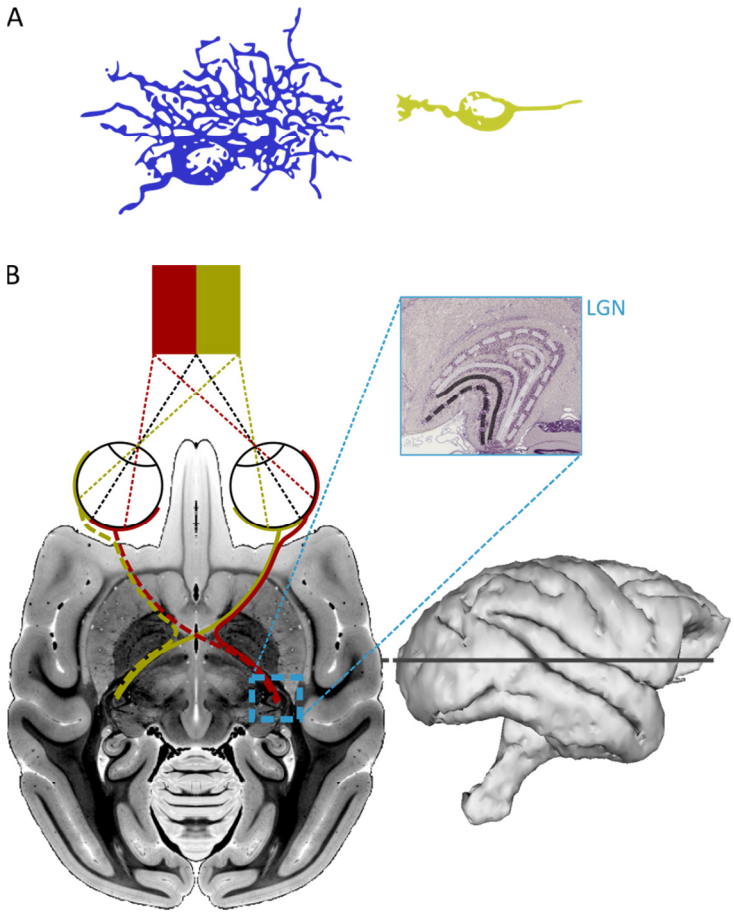

Figure 1. (A) A magnocellular (left) and parvocellular (right) ganglion cell at a distance to the fovea of 1mm of a macaque. Colors match those used in the schematic diagram in Figure 2A. Adapted from (Watanabe & Rodieck, 1989). (B) The path of visual information from a stimulus to the LGN shown in an axial section of an MRI scan of a macaque brain. The approximate location of the section is shown in the 3D brain surface on the right. The image is projected onto the retina. After retinal processing of the image, nerve fibers of the ganglion cells propagate to the optical chiasm where fibers from the nasal part of the retina (dashed red and solid yellow lines) cross the hemifield. Fibers from both eyes covering the same hemifield terminate in the contralateral LGN. The enlarged view shows a coronal section of a nissl stained LGN. The lower two layers receive input from M ganglion cells, the upper four from P cells. The dashed lines show the layers receiving input from the left (contralateral) eye. The Axial section is adapted from BrainInfo (1991), coronal section of the LGN and 3D brain surface taken from Brainmaps (Mikula, Stone, & Jones, 2008; Mikula, Trotts, Stone, & Jones, 2007; Trotts, Mikula, & Jones, 2007).

The vast majority of ganglion cells project to the lateral geniculate nucleus (LGN), located in the dorsal part of the thalamus (Perry, Oehler, & Cowey, 1984). In the LGN, M and P ganglion cells provide input to the eponymous magno- and parvocellular LGN neurons in strictly separate layers. The names were given for their distinct difference in the size of the cell somata; being larger for cells in the M than in P layers of the LGN. In between these layers are the koniocellular layers that receive input from cells that are neither parvo- nor magnocellular. Some 10% of the ganglion cells' nerve fibers project into other regions, most notably and

predominantly into the superior colliculus (Perry & Cowey, 1984). See Figure 1B for the path of visual information from the retina to the LGN.

The LGN projects almost exclusively to the primary visual cortex (V1), where fibers from M and P LGN neurons terminate in separate sub-layers. Starting from V1, the separation of M and P input becomes less strict. Due to functional deficits observed in lesion studies, Ungerleider and Mishkin (1982) proposed two streams of visual processing originating in V1, each responsible for the encoding of different stimulus properties. Lesions at the proposed end of the *ventral stream* ('what pathway'), the inferotemporal cortex, resulted in deficiencies of pattern discrimination but not in localization of objects. The opposite was found for lesions in the *dorsal stream* ('where or how pathway') which reaches to the posterior parietal region (see Goodale & Milner (1992) for a review). The *ventral stream* receives both parvo- and magnocellular input, while the *dorsal stream* is dominated by magnocellular input (Ferrera, Nealey, & Maunsell, 1994). These streams are however not strictly separate, with many known connections between regions attributed to either of the streams (DeYoe & Van Essen, 1988; Van Essen, Anderson, & Felleman, 1992; van Essen & Maunsell, 1983). A schematic diagram of the streams and a subset of the involved areas as well as some of their connections is shown in Figure 2.

For this thesis, the dorsal stream and its functional properties are of particular importance, as the middle temporal area (MT), which was the target of the electrophysiological recordings reported in chapter 3, is attributed to this stream. In the dorsal stream, information propagates on a major route from V1 to the thick stripes of V2. MT receives input from a number of cortical areas including this part of V2, but the most important input comes directly from V1 (Born & Bradley, 2005). In addition, MT also receives input from the superior colliculus via

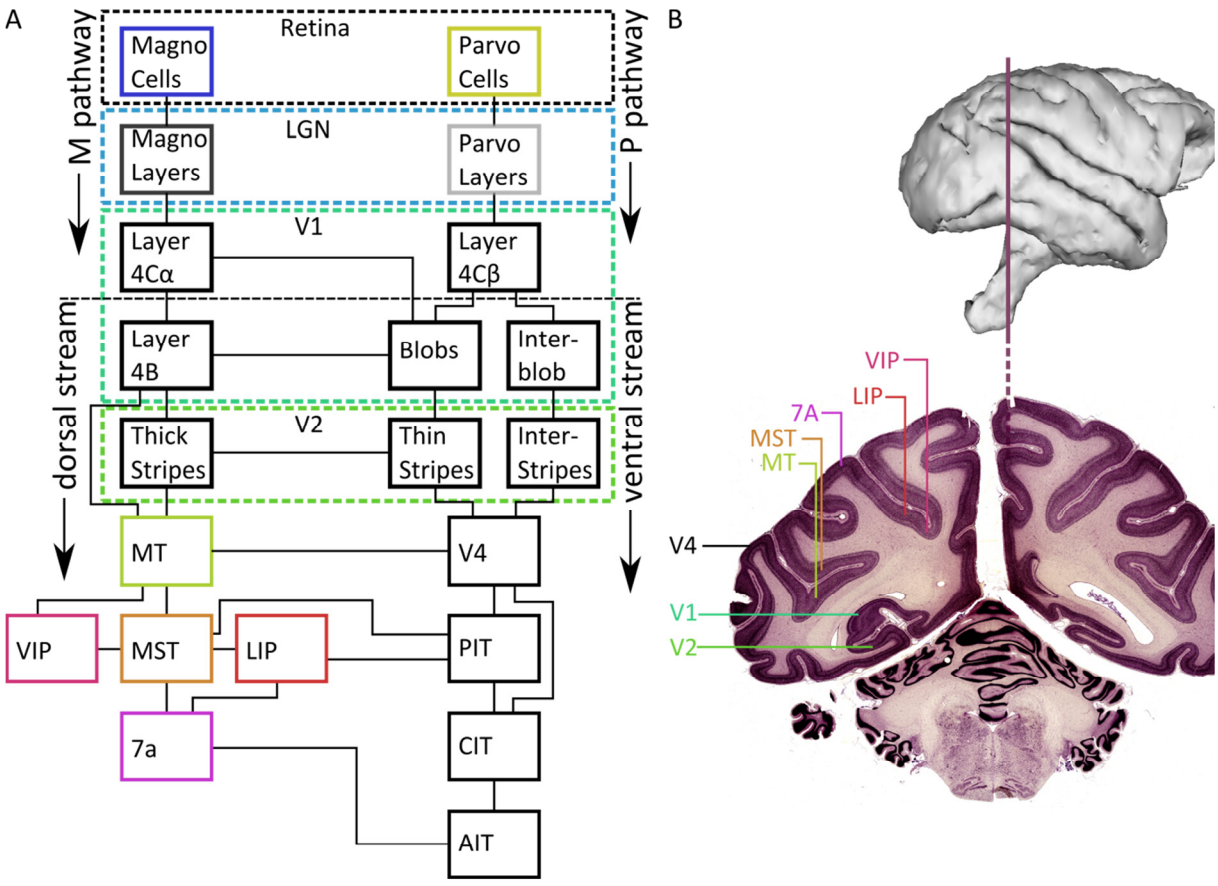

Figure 2. (A) Schematic diagram of connections of the M and P pathway as well as selected areas in the macaque monkey from the dorsal and ventral stream and some connections between these areas. Adapted from (Merigan & Maunsell, 1993). (B) A coronal nissl stained section of a macaque brain (bottom) at the approximate location indicated in the 3D brain surface (top). Parts of a number of visual areas are visible in this view. They are marked with the same colors as used in panel A. Both images taken from Brainmaps (Mikula et al., 2008, 2007; Trotts et al., 2007).

the pulvinar (Berman & Wurtz, 2010; Stepniewska, Qi, & Kaas, 1999). A small number of direct connections to MT have also been reported from the koniocellular layers of the LGN (Sincich, Park, Wohlgemuth, & Horton, 2004; Stepniewska et al., 1999), but it is unknown whether these LGN neurons received direct visual input (Born & Bradley, 2005; Stepniewska et al., 1999). Functionally, MT neurons encode motion direction (e.g. Albright, Desimone, & Gross, 1984), motion speed and binocular disparity (e.g. DeAngelis & Newsome, 1999). It has been shown that, while responses of MT are dominated by the cortical route via V1, input from either the superior colliculus (SC) or V1 is sufficient for the direction selectivity of MT neurons (Rodman, Gross, & Albright, 1989, 1990). MT provides strong input to other areas of

the dorsal stream. Among them are the medial superior temporal (MST) and the ventral intraparietal (VIP) areas, both of which are sensitive to visual stimulation induced by self motion, and the lateral intraparietal area (LIP), playing a role in the generation of eye movements (Born & Bradley, 2005). See Born & Bradley (2005) for a recent review about area MT.

Frames of reference

A neuron's (visual) *receptive field* (RF) is defined as the region of the visual field in which (visual) stimulation is able to cause changes to a neurons' activity. In early stages of visual processing (such as the LGN, SC and V1) neurons are organized in a *retinotopic* map. The RFs of such retinotopically organized neurons are determined by the location relative to the fovea (*retinocentric*) and neighboring neurons will encode for neighboring locations of the image projected onto the retina. At later stages of the cortical processing this functional organization may change. In order to determine the location of a stimulus in the world and, more importantly, to interact with objects in the world, the information of the retinal position and information of the eyes' position have to be combined and put in relation to the orientation of other body parts. For example, in order to grasp a glass of tea, the retinal information of its location has to be translated into a motor command of the arm and hand. Since we are able to grasp the glass even with our eyes closed, this process cannot simply rely on an iterative visual feedback loop. Three main mechanisms that might help solving this problem have been demonstrated.

The first involves a transformation of coordinate systems in which visual information is encoded, e.g. from retinocentric to *craniocentric* receptive fields. In craniocentric neurons, the location of the RF is not constant relative to the fovea for different gaze directions but rather moves to remain at a constant location relative to the position of the head. For example

Duhamel, Bremmer, Ben Hamed, & Graf (1997) have reported neurons in VIP to express such properties. The RF of one of their recorded cells is shown in Figure 3. There is currently a dispute on whether the human MT might also encode information in a craniocentric frame of reference (Crespi et al., 2011; Gardner, Merriam, Movshon, & Heeger, 2008; d' Avossa et al., 2007). However, electrophysiological recordings so far do not provide evidence for this in the macaque MT (Hartmann, Bremmer, Albright, & Krekelberg, 2011; Ong & Bisley, 2011).

The second mechanism is a remapping/transfer of visual information to neurons that would encode for this information after a saccade in otherwise retinocentric areas (Duhamel, Colby, & Goldberg, 1992). The authors reported neurons that would be responsive to visual stimulation around the time of saccades at the location the RF would be at only after the execution of the saccade, even though the stimulus had disappeared before the neurons' RF matched the location of the stimulus. That is, the retinocentric information about the location of the stimulus was somehow transferred to the neuron that would encode it afterwards. In their study it was also shown that some neurons would respond to the stimuli shown in the future RF even shortly before the saccade actually occurred. This has been termed *predictive remapping*. Such remapping has now also been shown to occur in many other visual areas (see Wurtz (2008) for a review), yet, to a lesser extent than in area LIP.

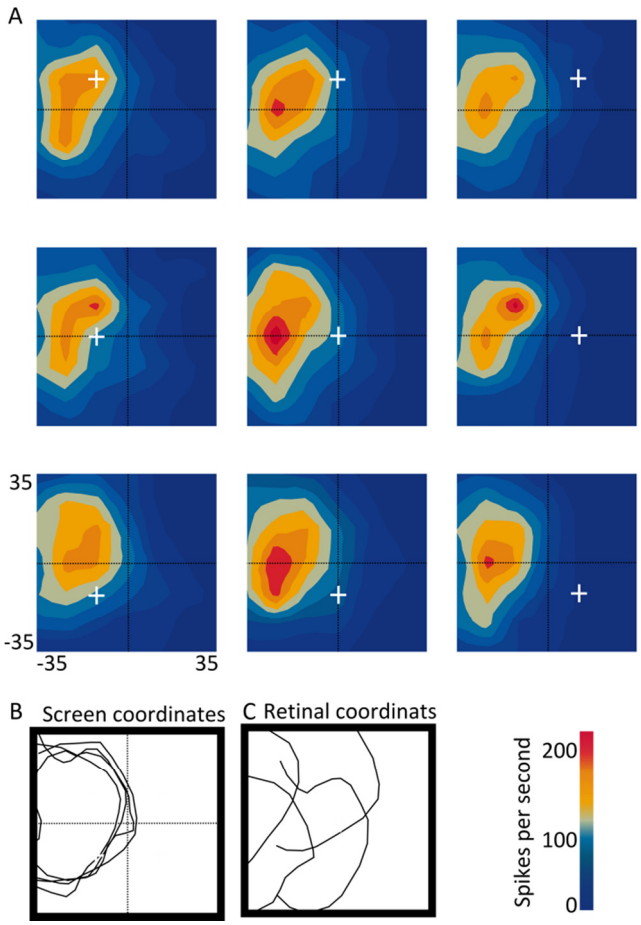

Figure 3. (A) Receptive field of one cell for different eye positions (indicated by the white cross). (B) Outline of the RFs either in screen coordinates (left) or retinal coordinates (right). The RF is clearly stable in screen but not in retinal coordinates. Adapted from Duhamel et al. (1997).

Another mechanism that allows craniocentric decoding of stimuli and might serve towards craniocentric receptive fields is the implicit encoding of eye-position information in retinocentric neurons. In a number of visual areas responses have been shown to be modulated by the eye-position. Even though the visual stimulus was encoded in retinocentric coordinates, the gain of the response depended on the current eye position (also visible in Figure 3A). The areas in which neurons have been shown to have such eye position dependent *gain fields* include MT and MST (Bremmer, Ilg, Thiele, Distler, & Hoffmann, 1997), LIP and 7a (Bremmer, Distler, & Hoffmann, 1997), VIP (Bremmer, Graf, Ben Hamed, & Duhamel, 1999), V4 (Bremmer, 2000), and FEF (Cassanello & Ferrera, 2007a). It has been shown that the eye position can be extracted from these gain modulated responses (Boussaoud & Bremmer, 1999; Bremmer, Pouget, & Hoffmann, 1998; Cassanello & Ferrera, 2007b) and that decoding the

spatial location of a stimulus using this information around the time of saccades would result in a pattern of mislocalization similar to that observed in behavioral studies (Morris, Kubischik, Hoffmann, Krekelberg, & Bremmer, 2012).

Peri-saccadic perception

While the mechanisms that help towards a stable percept work well during everyday life, changes in perception can be unveiled by limiting the information available to the visual system. This is typically done by using transient stimuli that are presented just around the time of a saccade. The specific details and the pattern of these changes can help to differentiate between different possible underlying neural mechanisms. Of the many perceptual changes during saccades, those to the sensitivity of detecting a stimulus and to the perception of time are most important for the scope of this thesis.

Saccadic suppression

During saccades the ability to detect stimuli presented only for a short duration is largely reduced (e.g. Holt, 1903; Matin, Clymer, & Matin, 1972). This *saccadic suppression* of visual processing is maximal just around the onset of a saccade. When a saccade is simulated by a fast rotating mirror, contrast sensitivity remains unchanged unless the transient stimuli are shown on a structured background that is visible from well before to well after the saccade (Diamond, Ross, & Morrone, 2000). Saccadic suppression thus appears to be linked to the eye-movement itself, not just to the fast retinal image motion that it induces. Saccadic suppression is however highly specific to the parameters of visual stimulation (Burr, Morrone, & Ross, 1994). Sensitivity to luminance contrasts with low spatial frequency is reduced by a factor of up to ten while sensitivity to color contrasts or to contrasts with high spatial frequency remains unchanged or is even enhanced after the saccade (Figure 4). The stimulus parame-

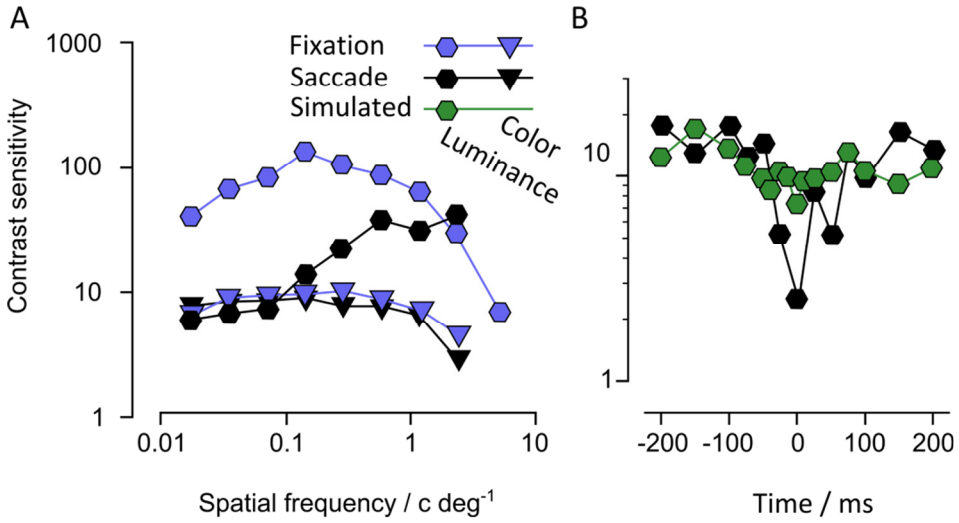

Figure 4. (A) Contrast sensitivity during saccades (black symbols) is reduced compared to fixation (blue symbols), but only when the stimulus is modulated in luminance (hexagons), not when modulated in color (triangles) and only for low spatial frequencies. Adapted from Burr et al. (1994). (B) Contrast sensitivity is reduced during saccades (black symbols) but not when the image is moved at saccadic speeds through a mirror (green symbols). Adapted from Diamond et al. (2000).

ters needed for saccadic suppression to occur closely match those known to be encoded by neurons in the M pathway. The results led to the assumption that saccadic suppression occurs at a stage of visual processing where M and P input is still strictly segregated; namely the LGN (e.g. Diamond et al., 2000). Additionally, detection thresholds of phosphenes generated in the retina (by electrical stimulation of the orbit), but not in V1 (by transcranial magnetic stimulation (TMS)) have been shown to increase during saccades, supporting a precortical source of saccadic suppression (Thilo, Santoro, Walsh, & Blakemore, 2004).

Electrophysiological recordings in the macaque brain however have failed to clearly support this theory. Modulations of activation in the LGN around the time of saccades have mainly shown an increase of action potentials that was not specific to the magnocellular layers of the LGN (Fischer, Schmidt, Stuphorn, & Hoffmann, 1996; Reppas, Usrey, & Reid, 2002). Because the dorsal stream is dominated by input from the M pathway, saccadic-suppression related changes in neuronal activity could also be expected in these areas. Such a peri-saccadic reduction of responses to stimuli has been reported for areas MST (Bremmer, Kubischik,

Hoffmann, & Krekelberg, 2009; Ibbotson, Crowder, Cloherty, Price, & Mustari, 2008; Thiele, Henning, Kubischik, & Hoffmann, 2002), MT (Bremmer et al., 2009; Thiele et al., 2002) and VIP and LIP (Bremmer et al., 2009). The spatio-temporal details of these reductions however differed between the areas, which led Bremmer et al. to propose that saccadic suppression might not act in the LGN, but differently in different higher visual areas.

Perception of time

Perception of time is also subject to a number of large distortions around the time of saccades. One such misperception is the stopped-clock illusion, also called chronostasis, which has been demonstrated in an experiment by Yarrow, Haggard, Heal, Brown, & Rothwell (2001). In their experiment, subjects made a saccade to a counter which initially displayed '0'. Triggered by the saccade, the '0' changed to a '1' for a variable duration and counted up to 4 in one-second intervals afterwards (Figure 5A). They found that the duration of the '1' had to be shorter than one second to be perceived lasting as long as the following one-second intervals, i.e. the duration was overestimated for a stimulus that started during the saccade (Figure 5B). The occurrence of this overestimation seemed to be dependent on certain boundary conditions. It did not occur when the counter was notably moved during the saccade. Additionally Georg and Lappe (2007) reported a lack of chronostasis when the counter was placed in the center of the saccade trajectory rather than near the saccade target itself. The authors concluded that chronostasis only occurs locally at the saccade target when another stimulus ('0') was at the location when the saccade started.

But also the opposite effect, an underestimation of time during saccades, has been reported (Morrone, Ross, & Burr, 2005). When two large stimuli are presented shortly at different locations with a temporal delay between their appearances, the temporal interval between these two stimuli was underestimated compared to an interval defined by a second pair of stimuli shown well after the saccade (Figure 5C). In a critical time window, just prior to the onset of the saccade, another distortion of temporal perception was observed. The temporal order of appearance of the two stimuli was systematically perceived inverted to the correct presenta-

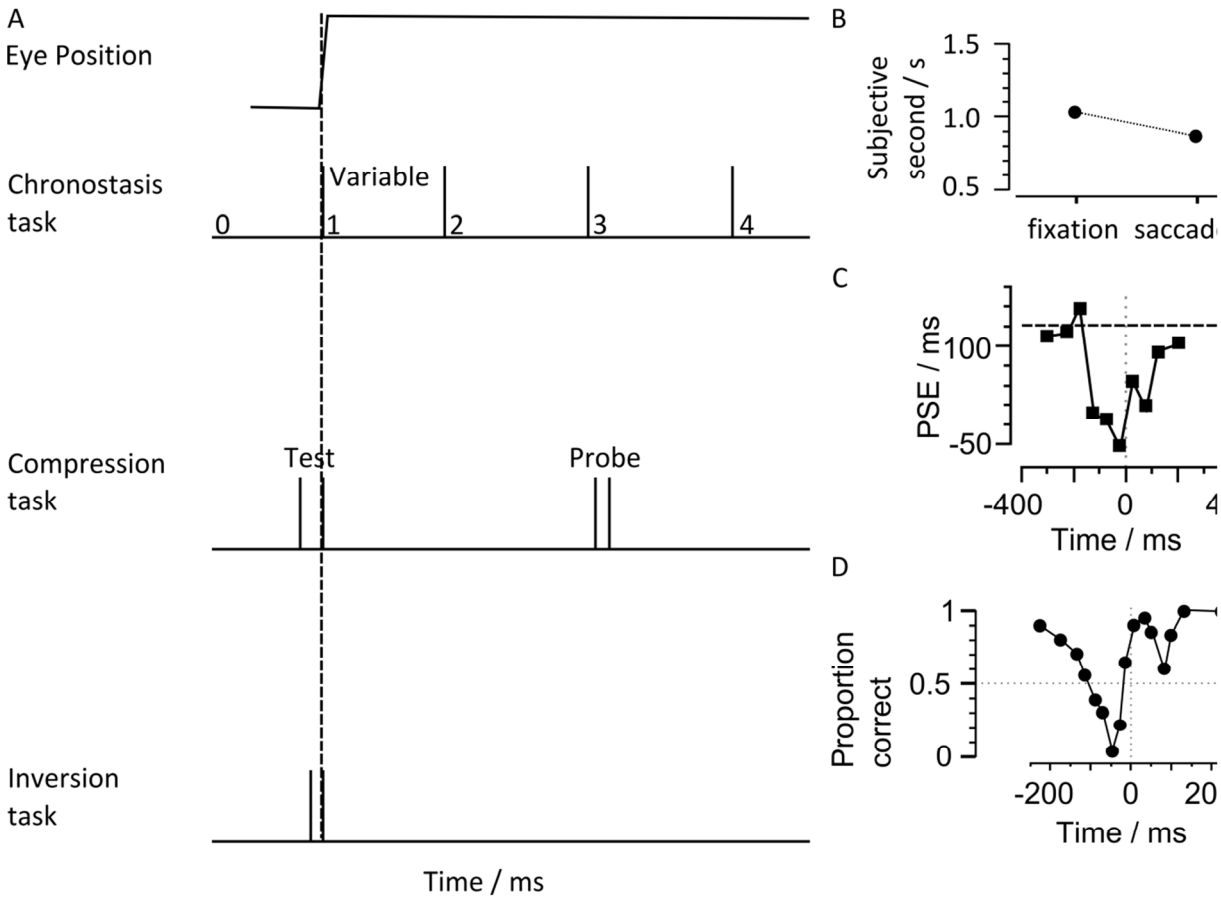

Figure 5. (A) Experimental paradigm used for the chronostasis task by Yarrow et al. (2001) and the task to investigate the compression and inversion of perceived time by Morrone et al. (2005). (B) Results from the experiment by Yarrow et al. (2001), demonstrating chronostasis. During saccades, the '1' had to be presented for less than the comparisons intervals to be perceived as lasting equally long. (C) Results from the time compression experiment by Morrone et al. (2005). The time is relative to the onset of a saccade. The dashed horizontal line marks the PSE during fixation. Perceived duration is peri-saccadically reduced. (D) Results from the temporal inversion experiment (again by Morrone et al. (2005)). The proportion of correctly stating the temporal order of the two stimuli falls below change level indicating an inversion of perceived temporal order. Adapted from Yarrow et al. (2001) and Morrone et al. (2005).

tion sequence (Figure 5D).

Perception dramatically changes during saccades. Due to current findings (e.g. Bremmer et al., 2009) there is a renewed debate about the stage of visual processing that some of these changes in perception (like saccadic suppression) occur at. The neuronal correlates of many of these peri-saccadic effects and possible relations and interactions to one another are largely unknown.

Aim and Scope of this thesis

Chapter 1

Bremmer et al. (2009) proposed that a significant part of saccadic suppression might arise in higher visual areas. Neurons in VIP of the macaque brain are known to have craniocentric receptive fields (Bremmer, Duhamel, Ben Hamed, & Graf, 2002). A potential functional equivalent to this area in humans has been reported in an fMRI study (Bremmer et al., 2001). Additionally there is currently a dispute, whether activity in human MT might also be organized in a non-retinal frame of reference (Crespi et al., 2011; Gardner et al., 2008; d' Avossa et al., 2007). From these results the question arises whether changes of contrast sensitivity due to saccadic suppression might also occur in a craniocentric reference frame. Thus the experiment reported in the first chapter was aimed to determine the frame of reference of saccadic suppression in a psychophysical experiment by measuring the temporal profile of contrast sensitivity around the time of saccades at different locations. The comparison of the spatio-temporal profile of peri-saccadic contrast sensitivity analyzed either in eye-centered or screen-centered coordinates aimed to determine the frame of reference of saccadic suppression.

Chapter 2

The results gathered in my first study (chapter 1) led to a hypothesis about saccade induced chronostasis, the misperception that leads to an overestimated duration of stimuli appearing during a saccade: chronostasis might be influenced by or functionally related to saccadic suppression. If this was the case, the spatio-temporal profile reported in chapter 1 should be reflect-

ed in the spatio-temporal profile of chronostasis. The aim of the experiments described in this chapter was thus to test this hypothesis by measuring the spatio-temporal profile of chronostasis and by measuring the perisaccadic duration perception in different conditions under which stimuli are not subject to saccadic suppression (isoluminant stimuli and simulated eye-movements).

Chapter 3

Two aspects of the neuronal mechanisms of visual perception were set to explore in the third chapter. The first again directly relates to the findings of Bremmer et al. (2009), proposing higher visual areas such as MT as a possible acting point of saccadic suppression. I therefore aimed to first characterize MT responses to stationary stimuli at different luminance levels and durations during fixation and to next compare these responses to responses during saccades. To achieve this, I recorded from single cells in MT of an awake behaving macaque monkey while presenting said stimuli during fixation or around the time of saccades. Saccadic suppression causes contrast sensitivity to increase by a factor of up to ten. It is likely that a number of visual areas contribute to the detection of luminance contrast stimuli. If MT was to play a major role in the perisaccadic suppression of perception of these stimuli, one could thus expect the peri-saccadic response to a bright stimulus to be similar to the response to a stimulus in the range of one tenth (or less) of the contrast. The second aim was to investigate a potential role of MT in time perception, by presenting pairs of stimuli with different stimulus onset asynchronies (SOAs).

Declaration of the author's contributions

Chapter 1: The content of chapter 1 has been published in a peer reviewed journal in identical wording and with identical graphics (Knöll, Binda, Morrone, & Bremmer, 2011). Experiment 1 was planned and conducted in the scope of a diploma thesis by JB (Beyer, 2008), extending results from the diploma thesis of JK (Knöll, 2007). Experiment 2 was planned and conducted by PB. JK analyzed the data of experiment 1. Data from experiment 2 was analyzed by PB. The manuscript was prepared by JK (50%), PB (50%), MCM (corrections) and FB (corrections).

Chapter 2: The content of chapter 2 has been submitted to a peer reviewed journal (Vision Research; Manuscript number: VR-12-240) in identical wording and with identical graphics. JK planned and programmed all experiments, including contributions to the software Neurostim (http://neurostim.sourceforge.net). Experiments 1 and 5 were conducted only by JK. Experiments two through 4 were conducted by JK (50%) and student research assistant JV (50%) after instruction and training by JK. All experiments were analyzed by JK. The manuscript was prepared by JK (100%), MCM (corrections) and FB (corrections).

Chapter 3: The content of chapter 3 has not been previously submitted to a journal. The experiment was planned by JK (90%), FB, BK and TH. Programming (including contributions to the software Neurostim), conduction and analysis of the experiment were done by JK. The chapter was written by JK (100%).

Initials:
BK: Prof. Dr. Bart Krekelberg, FB: Prof. Dr. Frank Bremmer, JB: Jens Beyer, JK: Jonas Knöll, JV: Judith Vornwald, MCM: Prof. Dr. Maria Concetta Morrone, PB: Dr. Paola Binda, TH: Dr. Till Hartmann

Chapter 1: Spatiotemporal profile of peri-saccadic contrast sensitivity

Abstract

Sensitivity to luminance contrast is reduced just before and during saccades (saccadic suppression), whereas sensitivity to color contrast is unimpaired peri-saccadically and enhanced post-saccadically. The exact spatiotemporal map of these perceptual effects is as yet unknown. Here, we measured detection thresholds for briefly flashed Gaussian blobs modulated in either luminance or chromatic contrast, displayed at a range of eccentricities. Sensitivity to luminance contrast was reduced peri-saccadically by a scaling factor, which was almost constant across retinal space. Saccadic suppression followed a similar time course across all tested eccentricities and was maximal shortly after the saccade onset. Sensitivity to chromatic contrast was enhanced post-saccadically at all tested locations. The enhancement was not specifically linked to the execution of saccades, as it was also observed following a displacement of retinal images comparable to that caused by a saccade. We conclude that luminance and chromatic contrast sensitivities are subject to distinct modulations at the time of saccades, resulting from independent neural processes.

Introduction

With each saccade (rapid ballistic eye movement), the image of the visual scene sweeps across the retina at high speed; yet, this dramatic change of the visual input completely escapes our notice. In natural viewing conditions, many factors contribute to this temporary blindness, including retinal smear (stimuli displayed throughout the eye movement result in blurred retinal images) and masking by the high-contrast images acquired before and after the saccade (Matin et

al., 1972). However, even in experimental conditions where these factors are controlled for (with stimuli flashed briefly in an otherwise empty visual field), peri-saccadic sensitivity is found to be strongly and selectively modulated.

The sensitivity to flashed stimuli modulated in luminance contrast and with low spatial frequency is reduced by 0.5–1 log unit, whereas high spatial frequencies and stimuli modulated in chromatic contrast are detected with the same sensitivity peri-saccadically and during steady fixation (Burr, Holt, Johnstone, & Ross, 1982; Burr et al., 1994; Diamond et al., 2000; Uchikawa & Sato, 1995; Volkmann, 1986). The suppression of low-frequency luminance-defined stimuli is contingent on the preparation and execution of a saccade; it is not observed when the displacement of retinal images is simulated (by sweeping the stimulus display at saccadic speeds) while the observer maintains steady fixation. This constitutes strong evidence that contrast sensitivity is actively suppressed during saccades, possibly via extraretinal "efference copy" or "corollary discharge" signals generated by the oculomotor system (Diamond et al., 2000).

Burr et al. (1994) and Diamond et al. (2000) proposed that saccadic suppression may occur as early as in the lateral geniculate nucleus (LGN), which encompasses three segregated populations of neurons: the parvocellular (P), koniocellular (K), and magnocellular (M) pathways (Hendry & Reid, 2000; Merigan & Maunsell, 1993). A selective suppression of activity in the M pathway would account for the suppression of low spatial frequency luminance modulations (and of motion signals), preferentially processed by M neurons, while sparing the sensitivity to chromatic contrast and high-frequency modulations of luminance contrast, preferentially processed by the K and P pathways. Forward and backward masking experiments suggest that suppression is achieved by a gain reduction of the M neurons' response (Burr, Morgan, &

Morrone, 1999; Burr et al., 1994), which Diamond et al. (2000) modeled as the result of the interaction between the retinal input and an extraretinal corollary discharge signal. This hypothesis—that saccadic suppression is achieved with a gain reduction of visual responses under the control of extraretinal signals—predicts suppression to be homogeneous across the retinal space, reducing contrast sensitivity by a constant divisive factor. The conclusions from two psychophysical studies, however, challenge this prediction. Mitrani, Mateeff, and Yakimoff (1970) and Osaka (1987) argued that the magnitude and time course of suppression is different for small luminance-modulated stimuli flashed in the proximity of the fovea, being stronger perisaccadically (Osaka, 1987)and recovering more quickly after the saccade (Mitrani et al., 1970) than for peripheral flashes. However, because both studies measured detection performance (percent correct responses for stimuli set to be near threshold during steady fixation), their results would be equally compatible with a constant suppression factor across the retinal space, producing a larger and quicker drop of correct detection responses in the most sensitive retinal regions.

Our first experiment addressed these issues by measuring contrast sensitivity for small luminance-modulated stimuli, flashed at various times relative to saccade onset and at various spatial locations. We quantified suppression by comparing sensitivity at matching locations during steady fixation and peri-saccadically, and we characterized the time course of the sensitivity change across the range of stimulus locations. Results were analyzed after encoding stimulus locations in both their screen coordinates as well as in retinal coordinates, computed by taking into account the position of the eyes at the time of stimulus presentation. In this way, the com-

parison of the dynamics in both coordinate systems allowed to identify the frame of reference in which saccadic suppression occurs.

Peri-saccadic suppression is selective for luminance contrast, but the sensitivity to chromatic contrast varies around the time of saccades too. The variation has the opposite sign and different dynamics relative to saccadic suppression: An enhancement of color contrast sensitivity is observed post-saccadically, starting about 100–200 ms after the completion of the saccade. Interestingly, a similar pattern of suppression/enhancement is observed in coincidence with another class of eye movements: smooth pursuit (Schütz, Braun, Kerzel, & Gegenfurtner, 2008), during which sensitivity to low-frequency luminance modulation is decreased and sensitivities to high-frequency modulations and to chromatic contrast are enhanced.

The co-occurrence of luminance contrast suppression and chromatic contrast enhancement is suggestive of a link between the two phenomena. The same extraretinal signal proposed to trigger the suppression of M responses may be responsible for the enhancement of the P pathway, as suggested for the case of smooth pursuit (Schütz et al., 2008). Another hypothesis proposes that suppression and enhancement both result from the effect of saccades on luminance signals. P cells probably carry both chromatic information and an achromatic signal; a saccade might destroy the notional equiluminance of the chromatic stimuli, thereby making the target more visible (Morgan, 1994).

To investigate the relationship between peri-saccadic suppression and post-saccadic enhancement, our second experiment measured sensitivity to stimuli similar to those in our first experiment but equiluminant to the background and modulated in chromatic contrast only. As in our

first experiment, we varied stimulus position to ask whether the modulations of contrast sensitivity depend on stimulus position, both during a saccade and during a 300-ms post-saccadic epoch. In addition, we measured chromatic contrast sensitivity in a condition where saccadic retinal motion was simulated while observers maintained steady fixation. This approach allowed us to ask whether the post-saccadic enhancement is tied to the execution of a saccade, as peri-saccadic suppression is (Diamond et al., 2000), testing the hypothesis that both phenomena can be explained by an active extraretinal modulation of visual sensitivity at the time of eye movements.

While the stimuli used in the two experiments presented here were similar (small and brief modulations of luminance or chromatic contrast), the methodological approach of each experiment was optimized to its specific aims. Experiment 2 was designed to measure potentially small effects: the post-saccadic enhancement of chromatic contrast sensitivity, previously reported to be in the order of a factor of 2, and the absence of peri-saccadic suppression of chromatic contrast sensitivity (Burr et al., 1994; Diamond et al., 2000). To maximize the precision of the method, we adopted a 2AFC color identification task combined with an adaptive method to sample the psychometric curve (QUEST; Watson & Pelli, 1983) and we performed all analyses at the single-subject level. Experiment 1 measured the large peri-saccadic suppression of sensitivity to luminance contrast and aimed at estimating its variations across a wide and densely sampled range of stimulus positions and timings. For this experiment, we favored efficiency over precision and used a seen/not seen task with analyses performed on data pooled across subjects. Two previous studies (Burr et al., 1994; Diamond et al., 2000) measured saccadic suppression with

both a forced choice identification task and a seen/not seen task and reported comparable estimates of the effects, demonstrating the validity of this approach for peri-saccadic stimuli.

Methods

Experiments were performed in part at the Philipps-Universität Marburg (Germany) and in part at the Neuroscience Institute of the CNR in Pisa (Italy). Experimental procedures, approved by the local ethics committees, were in line with the declaration of Helsinki. Care was taken to produce comparable experimental conditions with the different equipment of the two laboratories. A total of nine observers participated in the experiments (age range: 22–46, four naives and one subject familiar with the goals of the study for Experiment 1 and two authors and two naives for Experiment 2), all with normal or corrected-to-normal vision.

Visual stimuli were produced by CRT devices, driven at 100-Hz refresh rate and covering at least the central 60 deg × 50 deg of the visual field. Subjects had their head stabilized with a chin rest and eye movements were monitored. Contrast sensitivity was measured for 2D Gaussian blobs (standard deviation: 1 deg in both spatial dimensions) flashed for one monitor frame on a uniform background. Either the stimulus was modulated in luminance (Experiment 1) or it was equiluminant to the background and modulated in chromaticity (Experiment 2). The contrast of the stimulus was varied from trial to trial to determine psychometric functions. For statistical analysis, we used the Psignifit Matlab package (Wichmann & Hill, 2001a, 2001b), which fits the data set with integral-of-Gaussian functions and provides estimates of the perceptual threshold and its standard error (based on 1999 Monte Carlo simulations). Sensitivity was defined as the inverse of the threshold.

In both Experiments 1 and 2, we tested two main conditions. In the "saccade condition," trials began with subjects gazing at a fixation spot (FP, a black spot of 0.4-deg diameter, located 7.5 deg left of the screen center). After a variable delay (randomly chosen between 700 and 1100 ms), the fixation target was extinguished; an identical target (the saccade target, ST) was presented 7.5 deg to the right of the screen center eliciting a 15-deg rightward saccade. In the "steady fixation condition," no saccade target was presented and subjects maintained their gaze on a fixation point that remained visible throughout the duration of a trial. An additional condition ("simulated saccades") was tested only in Experiment 2 (see below).

Experiment 1: Sensitivity to luminance contrast

Apparatus

Stimuli were generated on a PC using C++ and OpenGL routines and displayed on a 1.6 m × 1.2 m screen (located at 1.14 m from the observer) by a CRT projector (Electrohome Marquee 8000, resolution: 1152 × 864 pixels). Eye movements were recorded with an infrared eye tracker (SR Research Eyelink II running at 500 Hz). Saccades were detected with a velocity criterion (200 deg/s). The start and end of a saccade were defined as the first and last samples with a velocity above 20 deg/s, respectively. Trials were discarded (i) if the start point or the end point of the saccade differed by more than 2 deg from the target position, (ii) if the saccade latency was negative or larger than 300 ms, and/or (iii) if the stimulus presentation occurred more than 100 ms before or 150 ms after saccade onset. Based on these criteria, about 15% of all trials were excluded from further analysis.

Stimuli

A 2D Gaussian blob (standard deviation: 1 deg in both spatial dimensions) was displayed against a gray background (CIE coordinates: x = 0.324; y = 0.329; luminance: 12 cd/m2) and it appeared along the horizontal meridian, at a random location between ±30 deg relative to the screen center (white symbols in Figure ; stimuli were never presented at ±1.5 deg around the fix-

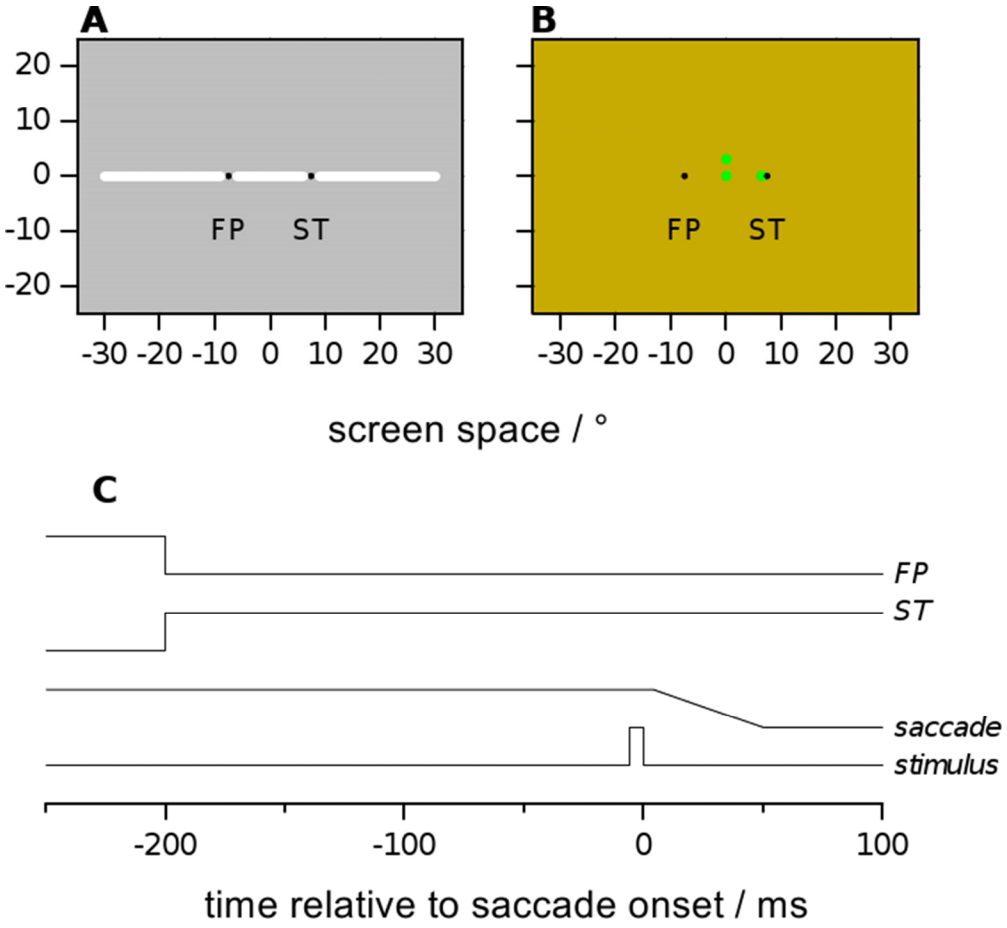

Figure 1. Spatial arrangement of the stimuli in (A) Experiments 1 and (B) 2, respectively, and (C) time course of presentations. Stimuli were 2D Gaussian blobs modulated in luminance contrast (Experiment 1) or they were modulated in chromatic contrast (along the red–green axis) and equiluminant to the yellow background (Experiment 2). Stimuli were presented for 1 monitor frame at variable delays from the onset of a 15-deg saccade. The white line in (A) represents the possible stimulus locations in Experiment 1, varied at random in the range ±30 deg at gaze level, except in the ±1.5 deg surrounding the fixation point (FP) and the saccadic target (ST). The green dots in (B) represent the tested locations in Experiment 2: at screen center and at two additional locations at or above gaze level.

ation and saccade targets). The visible screen (70 deg × 50 deg) was surrounded by very low ambient light (<0.1 cd/m2). The stimulus was brighter than the background, with incremental contrasts of 6, 12, 18, 24, and 46%, which varied from trial to trial according to the method of constant stimuli. Three additional contrast levels (4, 8, and 20%) were tested in the steady fixation condition. Subjects reported detection of the stimulus by pressing a key on the computer keyboard (seen/not seen task). This task has been successfully used in two previous saccadic suppression studies (Burr et al., 1994; Diamond et al., 2000) yielding similar sensitivity estimates as a 2AFC procedure.

Data analysis

For each subject, a minimum of 1400 and a maximum of 3500 trials were collected, with a grand total of 13,521 trials. Analyses were performed on data pooled across the five subjects: Trials were sorted according to the stimulus location and stimulus time relative to saccade onset, then divided into bins of at least 30 samples using a sliding spatiotemporal window (for some spatiotemporal bins, this pooling method resulted in an uneven distribution of data from the different subjects). In a separate analysis, we confirmed that this unevenness did not systematically affect the estimates of threshold values. The width of the window in space and time and the step size by which it moved was variable for different analyses (see figure legends). Behavioral data were analyzed after coding the spatial location of the stimuli in either screen coordinates or retinal coordinates; the latter were determined by subtracting the position of the eyes at the time of stimulus presentation from the position of the stimulus on the screen. In each spatiotemporal bin, detection rate (i.e., the proportion of trials where the stimulus was reported as "seen") was plotted against stimulus contrast yielding psychometric curves. A representative sample curve is

shown in Figure 2. The contrast level yielding a detection probability of 0.5 was considered the perceptual threshold (T). Sensitivity (S) was defined as the inverse of threshold (S = 1/T). For fitting psychometric functions, we imposed a constraint on the slope parameter, such that the fitted curve could not grow from 0 to 1 in an interval smaller than the distance between two consecutive tested contrast values. In a small percentage of instances (3%), removing this constraint led to unrealistically small estimates of the standard error of the estimated thresholds while not significantly affecting the threshold values themselves. Error bars in Figure 3 and Figure 4 report the larger standard error as estimated by the two fitting methods (unconstrained fit and fit with the slope constraint). Only data points for which both methods yielded an estimate of the SE are shown.

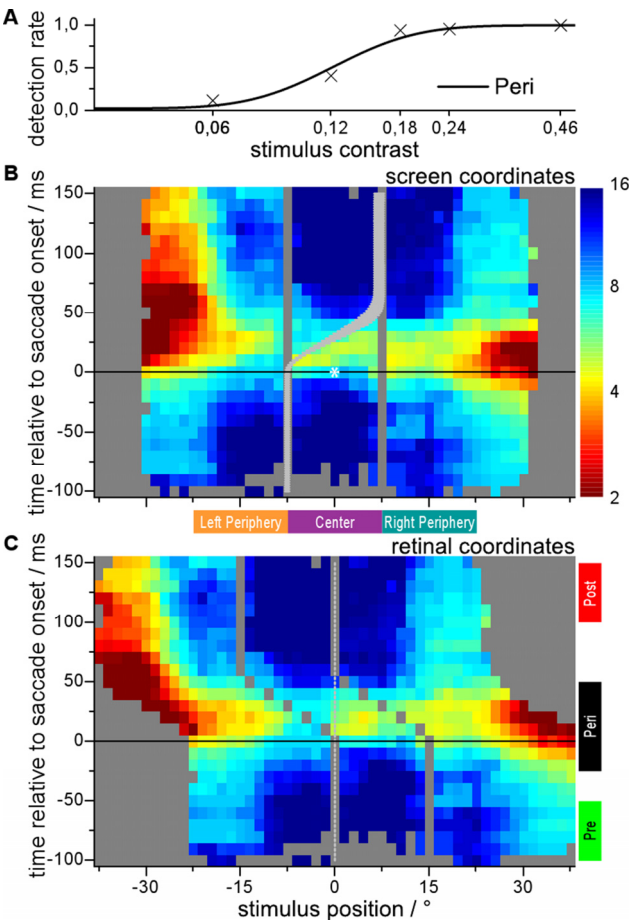

Figure 2. Luminance contrast sensitivity during fixation and saccades. (A) Sample psychometric curve for stimuli presented peri-saccadically at screen center (in the central 7.5-deg area). The threshold determined from this curve represents one data point in (B) (marked by white star).Threshold is defined as the contrast value allowing stimulus detection in 50% of trials (sensitivity = 1/threshold); for example, the curve in (A) estimated a threshold of about 0.15 corresponding to a sensitivity of about 6.7. Sensitivity values as a function of the time of stimulus presentation relative to the saccade onset (y-axis) and stimulus location (x-axis), coded in (B) spatial or (C) retinal coordinates. Each sensitivity value (color-coded in the maps) was computed in a 20 ms × 7.5 deg spatiotemporal window (including an average of 70 trials), which was shifted in steps of 10 ms and 1.5 deg. Colored boxes to the right of (C) show the pre-, peri-, and post-saccadic temporal windows used for data analysis shown in Figure 3. Colored boxes between (B) and (C) illustrate the left, center, and right spatial windows used for data analysis shown in Figure 4. The horizontal line at time = 0 ms marks the saccade onset.

Experiment 2: Sensitivity to chromatic contrast

Apparatus

Experiment 2 employed a 35 × 27.5 cm CRT color monitor (Barco Calibrator, resolution: 464 × 645 pixels) viewed from 30-cm distance. Stimuli were generated using a specialized graphics board (Cambridge Research Systems VSG2/5) housed in a PC and controlled by customized Matlab (Mathworks) programs. Eye movements were monitored by an infrared limbus eye tracker (ASL 310). The PC sampled the raw data at 1000 Hz and stored the eye trace for offline quality checks: As in previous studies (Binda, Morrone, Ross, & Burr, 2011), the saccade onset was determined online by fitting the eye trace with a three-line-segment function. Here, the three segments correspond to the pre-saccadic, saccadic, and post-saccadic epochs; the point of intercept between the first and second segments then yields an estimate of the saccadic onset. This procedure is more complex than the standard velocity threshold. However, it is more appropriate for the ASL 310 eye tracker (which requires calibrations every few trials) given that a velocity threshold is more sensitive to changes of spatial gain. In a later offline analysis, the experimenter checked the quality of saccades and, when necessary, discarded the trial (this happened in about 5% of trials, due to a corrective saccade or unsteady fixation).

Stimuli

The 2D Gaussian blob (standard deviation: 1 deg in both spatial dimensions) was equiluminant to the yellow background (Commission Internationale de l'Eclairage (CIE) coordinates: x = 0.48, y = 0.44; luminance: 19.6 cd/m2) and its chromatic contrast was modulated along the red–green axis. Note that the chromaticity of background was different from that in Experiment 1 (where the background was gray). The yellow background was chosen to maximize the chromatic con-

trast along the red–green axis attainable within the monitor gamut, while minimizing the stimulation of S cones. Equiluminance was established for each individual subject, by the minimum flicker technique (Boynton, 1979), adjusting the ratio of the red to green gun output to produce minimal flicker of the stimulus when modulated at 20 Hz. The color of the stimulus (red or green) was randomly chosen on each trial; at maximum contrast, the stimulus had CIE coordinates of x = 0.62, y = 0.64 for red and x = 0.28, y = 0.59 for green and produced a root-mean-squared (RMS) cone contrast of 0.31 relative to the background. RMS cone contrast was defined as: $\sqrt{[(\Delta L/L)^2 + (\Delta M/M)^2]/2}$, where L and M denote the excitation of L and M cones induced by the background and ΔL and ΔM denote the difference in cone excitation between the stimulus and the background. Cone excitation levels were computed using the CIE 1931 observer modified by Judd and Smith and Pokorny's copunctal points (following the procedure detailed in Appendix III of Kaiser & Boynton, 1996). Stimulus contrast was varied from trial to trial, using the adaptive QUEST procedure (A. B. Watson & Pelli, 1983). Subjects reported, in a 2-alternative forced-choice

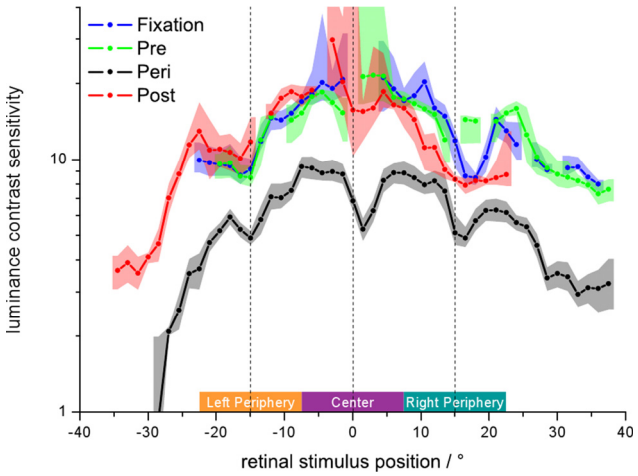

Figure 3. Sensitivity as a function of the stimulus retinal eccentricity. Colored lines represent contrast sensitivity as measured during saccades or during fixation. Each data point was computed in a 3-deg-wide spatial window, sliding across space in steps of 1.5 deg and including an average of 85 trials. The figure reports sensitivity values for steady fixation (where subjects maintained their gaze on a fixation spot located 7.5 deg left of screen center, blue line) and for three ranges of times relative to saccade onset (pre-saccadic (green): −100 to −50 ms, peri-saccadic (black): −25 to 50 ms, post-saccadic (red): 100 to 150 ms; see colored boxes in Figure 2 and Figure 4). Standard errors of individual sensitivity values are shown as shaded areas. Missing data points are those for which the SE could not be reliably estimated (see Methods section). Colored boxes define the spatial windows used for data analysis shown in Figure 4. Light gray indicates eye position.

task, whether the stimulus was red or green. The stimulus was presented at the screen center (i.e., midway along the real or simulated saccade path); for two subjects, sensitivity at two additional stimulus positions was tested in separate sessions, one at gaze level 6.5 deg right of the screen center (i.e., aside the saccade target) and another at screen center 3 deg above gaze level (see green blobs in Figure).

Simulated saccades condition

In addition to the "saccades" and "steady fixation conditions," we tested a condition where the displacement of retinal images produced by saccadic eye movements was simulated by viewing the monitor screen through a small (4 × 3 cm) mirror caused to rotate at saccadic speeds by a galvanometric engine controlled by the VSG. The mirror was placed 27 cm in front of the monitor. Subjects were seated laterally to the monitor, with their right eye about 3 cm from the mirror; a patch covered the left eye. Through the mirror, subjects had a clear monocular view of the central area of the display (20 × 20 deg). They maintained fixation on the fixation point (FP) throughout an experimental session. The rotation of the mirror produced a 15-deg leftward shift of the displayed image, therefore reproducing the displacement of retinal images caused by a 15-deg rightward saccade and bringing the saccadic target (ST) to the former retinal position of FP. The duration and velocity of the mirror rotation were monitored throughout the experiment. The typical duration for a 15-deg displacement was 45 ms (about the same as the duration of eye movements observed in the real saccades condition). During the experiment, we also monitored the subjects' eye movements (with a second eye tracker, model: HVS SP150) to control fixation.

Data analysis

One thousand to two thousands trials were collected for each subject and condition (two subjects were tested with one stimulus position only; the other two with 3 stimulus positions), yielding a grand total of 12,132 trials. Data were analyzed at the single-subject level. Trials from each of the four tested subjects were ranked according to the delay of the stimulus presentation from the onset of the real/simulated saccade and grouped in contiguous bins of variable width (each bin included at least data from 30 trials). For each bin, the proportion of correct responses was plotted as a function of the stimulus contrast. Performance varied from chance level (probability of correct response = 0.5) at low contrast to perfect behavior at high contrast. The contrast level allowing for a probability of correct responses of 0.75 was taken as threshold.

Results

We measured sensitivity to luminance and chromatic contrast with small 2D Gaussian blobs flashed for one monitor frame around the time of a 15-deg saccade (Figure). The choice of the stimulus represented a compromise between keeping the stimulus small enough to probe the spatial pattern of sensitivity, on the one hand, and to ensure a rich content of spatial frequencies that are peri-saccadically suppressed, on the other hand (Burr et al., 1982, 1994; Diamond et al., 2000; Uchikawa & Sato, 1995; Volkmann, 1986).

In Experiment 1, we tested the saccade-related spatiotemporal profile of sensitivity to luminance contrast with stimuli presented at gaze level. Figure 2 shows a sample psychometric function for stimuli flashed in the central region of the screen. Thresholds were defined as the contrast for which the stimulus was reported as “seen” in 50% of trials.

Figure 2 and Figure 2 shows the spatiotemporal map of contrast sensitivity (the inverse of threshold), with stimulus location encoded either in screen coordinates (panel B) or in retinal coordinates (panel Figure 2). For all positions, contrast sensitivity was strongly reduced from about 25 ms before saccade onset and throughout its duration, implying saccadic suppression. The peri-saccadic contrast sensitivity was not homogenous across the visual field, being higher in the more central regions compared to eccentric parts of the visual field.

In order to test whether the peri-saccadic topography of contrast sensitivity can be explained by a multiplicative modulation (gain control) of contrast sensitivity during fixation, we analyzed contrast sensitivity in three temporal windows as a function of the retinal location of the stimu-

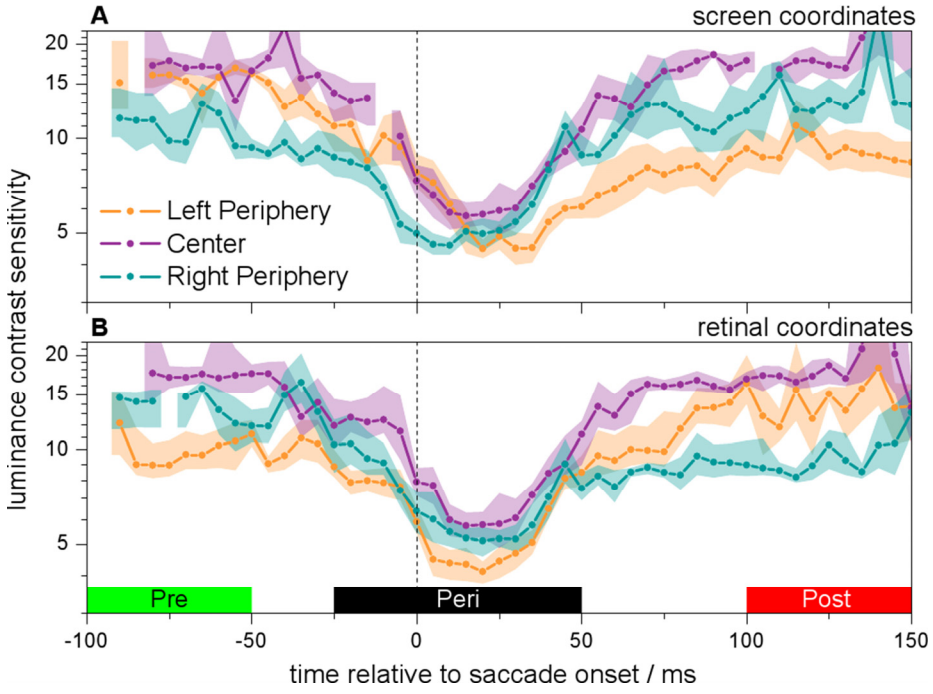

Figure 4. Time course of peri-saccadic suppression for three ranges of stimulus positions. The position ranges were: left periphery (orange line): −22.5 to −7.5 deg; center (magenta line): −7.5 to 7.5 deg; right periphery (green line): 7.5 to 22.5 deg, defined with respect to the screen center (screen coordinates, A) or in retinal coordinates (B). Each point was computed in a temporal window 10 ms wide, sliding across time in steps of 5 ms and including an average of 72 trials. Standard errors of individual sensitivity values are shown as shaded areas. Colored boxes illustrate the temporal windows used for data analysis shown in Figure 3.

lus (Figure 3). The black curve shows data for the detection of stimuli presented peri-saccadically, i.e., from 25 ms before to 50 ms after saccade onset. Detection data for stimuli presented pre-saccadically (between 100 and 50 ms before saccade onset) are shown in green, whereas detection data for stimuli shown post-saccadically (between 100 and 150 after saccade onset) are shown in red. Control data representing sensitivity for luminance contrast stimuli during steady fixation are shown in blue. The peri-saccadic curve lies below the others, indicating suppression. The shape of all curves is similar, implying that suppression is well described as a sensitivity reduction by a scaling factor that is constant across retinal space.

Sensitivity during steady fixation (blue curve) clearly shows two local minima at 15-deg eccentricity, roughly corresponding to the locations of the blind spot. The same drops of sensitivity are observed for peri- and post-saccadic stimuli (black and red curves, respectively), whereas the local minimum in the +15 deg region (the pre-saccadic retinal location of the saccade target, rightmost dashed line) is not evident for pre-saccadic presentations (green curve).

A small reduction of sensitivity in the blind spot regions was expected (in these regions, vision is monocular, predicting a reduction of sensitivity by a factor of about $\sqrt{2}$) and our success in detecting it indicates that the present seen/not seen technique is adequate for measuring contrast sensitivity, both peri-saccadically and in steady fixation conditions.

We note two additional features of the results in Figure 3. The curves tend to show a decline of sensitivity in the foveal region, which is consistent with the relatively low spatial frequencies of our stimuli; sensitivity tends to be lower in the far left retinal periphery than in the far right periphery during and after the saccade, possibly reflecting a different level of retinal adaptation

before and after the saccade. During fixation and before the saccade, locations with eccentricity <−27.5 deg lay outside the screen area and they are therefore dark adapted (after the saccade, the same happens to locations with eccentricity >27.5 deg). Thus, the saccade brings about a change in mean luminance for all positions with eccentricity larger than 27.5 deg. In particular, for retinal positions left of −27.5 deg, the saccade causes an abrupt increase of mean luminance, which can explain the observed decrement of contrast sensitivity.

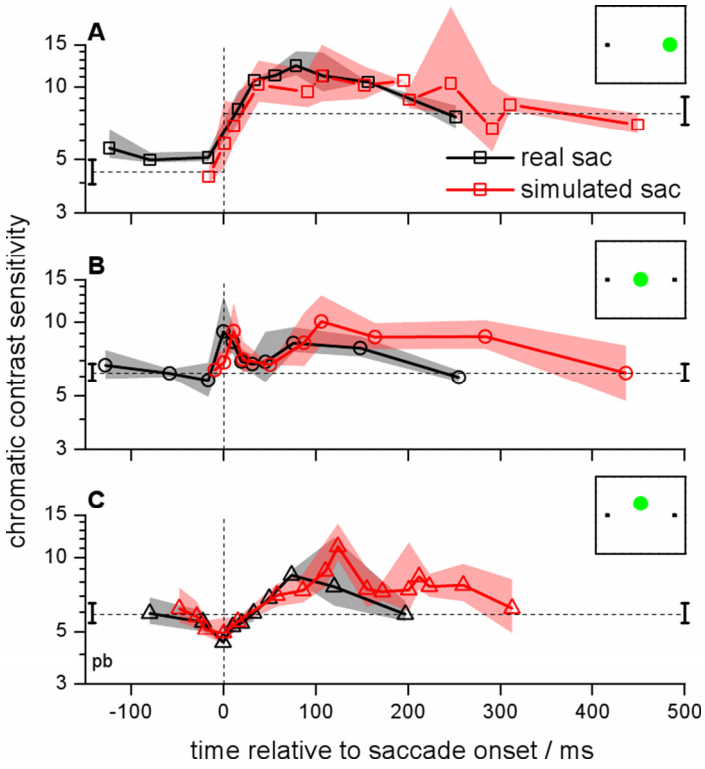

Figure 5. Sensitivity to chromatic contrast. Data from one subject (PB, an author) in Experiment 2. Sensitivity to chromatic contrast for three stimulus locations (A–C; see insets), measured while subjects maintained steady fixation at the fixation point (dotted lines for t < 0) or at the saccade target (dotted lines for t > 0 ms) and at various times from a saccade (black symbols) or from a simulated saccade (red), i.e., a displacement of the whole visual display simulating the retinal motion caused by a saccadic eye movement. Data points in each curve report sensitivity measures computed in contiguous bins of variable width, each including at least 30 trials. Standard errors of individual sensitivity values are shown as shaded areas. Sensitivity is the reciprocal of the root-mean-squared (RMS) cone contrast of the L and M cones at threshold ($\sqrt{[(\Delta L/L)^2 + (\Delta M/M)^2]}/2$; see Methods section for more details).

Figure 4 compares the time course of suppression for stimuli presented in the central region of the retina (eccentricity <7.5 deg) and for stimuli in the left or right periphery (average eccentricity: ±15 deg). The sensitivity in the left and right peripheries is not matched pre- and post-saccadically; it tends to be lower in the left retinal periphery than in the right periphery before the saccade, while the opposite trend is observed after the saccade. Because only positions with eccentricity <22.5 deg were considered for this analysis, differences of adaptation level (discussed above) cannot directly account for this result; possible contributing factors include a general attentive enhancement at the screen center or residual inhomogeneities of the display luminance at these outer positions.

Peri-saccadically, the three time courses run parallel and the maximum sensitivity reduction (0.4–0.5 log unit) is observed right after the saccade onset for all positions. On the contrary, if stimuli positions are coded in screen coordinates (Figure 4) rather than in retinal eccentricity, peak suppression occurs at different times for stimuli presented at the right, central, and left regions of the screen (respectively, at about 5, 15, and 30 ms after the saccade onset).

Thus, the peri-saccadic suppression of sensitivity to luminance contrast appears to be homogeneous across retinal space (and inhomogeneous in external space).

Next, we asked whether a peri-saccadic change of sensitivity to chromatic contrast can be observed. As we did for luminance contrast sensitivity, we investigated its dependency on retinal eccentricity. Experiment 2 measured sensitivity to stimuli similar in all respects to those employed in Experiment 1, except that they were equiluminant to the (yellow) background and modulated in chromatic contrast along the red–green axis. Sensitivity was measured with a

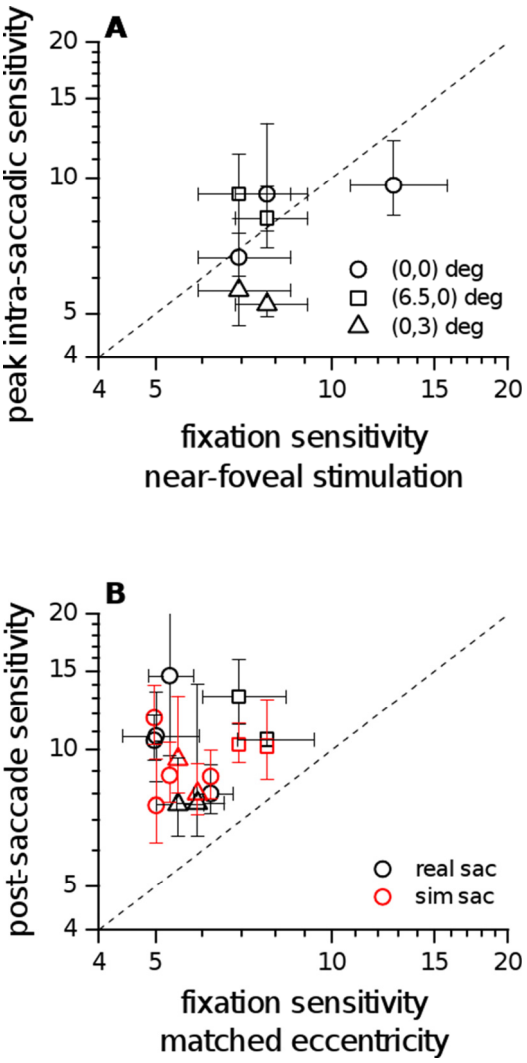

Figure 6. Chromatic contrast sensitivity during real and simulated saccades. (A) Peak chromatic contrast sensitivity observed for stimuli presented during a saccade plotted against the sensitivity for stimuli at 1-deg eccentricity, during steady fixation. The eccentricity of 1 deg was chosen to be close to the fovea, while producing small overlap between the stimulus and the fixation spot (always visible, like in all other experiments). (B) Peak sensitivity to chromatic contrast observed after a saccade or a simulated saccade against sensitivity at matching retinal eccentricities. Peak sensitivity was defined as the maximum sensitivity value in a time course like the ones in Figure 5, measured for each subject and condition; the average/standard deviation (across subjects and stimulus positions) of the delays from saccade onset where the peak sensitivity was observed were 153 ms/40 ms for the saccade condition and 149 ms/44 ms for the simulated saccade condition. Black symbols: real saccades. Red symbols: simulated saccades. Different symbol shapes refer to different stimulus locations. Data from four subjects were collected with the stimulus at position (0, 0) deg; two of the subjects were tested with the additional (6.5, 0) and (0, 3) deg positions. Error bars report standard errors of individual sensitivity values.

2AFC color discrimination task, given that the small expected size of the effects required a more sensitive technique than the yes/no task used in Experiment 1. Four subjects were tested with the stimulus presented at gaze level (as in Experiment 1), at a location midway between the fixation spot and the saccade target ([x, y] = [0 deg, 0 deg], see inset in Figure 5). Following the same logic of Experiment 1, we asked whether any peri-saccadic sensitivity modulation depends on the stimulus eccentricity. To this end, two of the four subjects were tested at a different, more peripheral location ([x, y] = [6.5 deg, 0 deg], i.e., next to the saccade target, see inset in Figure 5). Finally, because the saccade causes both these stimulus locations to become foveal at different

times during the movement of the eyes, the same two subjects were also tested with the stimulus presented above the line of sight ([x, y] = [0 deg, 3 deg], inset of Figure 5), which remains in a parafoveal region at all times.

Figure 5 (black symbols) reports the results from one subject tested with a ll three stimulus locations (see insets), plotting sensitivity as a function of the delay of stimulus presentation from the onset of a saccade. Sensitivity to chromatic contrast was not suppressed in the peri-saccadic interval, but it rather increased during the saccade for stimuli presented at gaze level (Figure 5 and Figure 5) and it remained approximately constant for stimuli presented above the line of sight (Figure 5; for this stimulus position, there is a tendency toward a peri-saccadic reduction of sensitivity; a bootstrap t-test with 2000 resamplings revealed that it is not statistically significant: $p > 0.1$ for both subjects tested with this stimulus position). During the saccade, the fovea sweeps over stimuli presented at gaze level and this reduction of stimulus eccentricity could explain the gradual (Figure 5) or transient (Figure 5) peri-saccadic sensitivity increase. To verify this hypothesis, we plot the peak sensitivity observed during the saccade against sensitivity at 1-deg eccentricity, for all tested subjects and positions (Figure 6). All points lie close to the identity line, implying a good match between peri-saccadic and fixation sensitivity at comparable retinal locations.

After the saccade, chromatic sensitivity was higher than that observed during fixation. This post-saccadic enhancement of chromatic contrast sensitivity peaked around 100 ms after saccade onset. Figure 6 (black symbols) plots peak post-saccadic sensitivity against sensitivity at matched retinal locations observed during normal fixation. For all tested locations, chromatic contrast sensitivity was enhanced by about 0.3 log units relative to normal fixation.

To test whether this post-saccadic enhancement of sensitivity to chromatic contrast is tied to the active execution of a saccade or rather emerges as a by-product of the changes of retinal stimulation caused by an eye movement, we tested an additional condition: simulated saccades. Here, we asked subjects to maintain their gaze on a fixation point, while we displaced the whole visual display so to mimic saccadic retinal motion (see Methods section). Like we did for real saccades, we measured chromatic sensitivity at various times from the onset of the simulated saccade. The results are reported by red symbols in Figure 5 and Figure 6. It is clear from inspection of these figures that real and simulated saccades caused a comparable enhancement of sensitivity to chromatic contrast, with very similar dynamics (compare black and red curves in Figure 5). A two-tailed paired t-test confirmed that peak sensitivity values observed after real and simulated saccades (black and red symbols in Figure 6, respectively) were not statistically different with $p > 0.3$.

Discussion

We studied visual sensitivity for small stimuli, briefly flashed around the time of a saccade, and we characterized the spatiotemporal topography of sensitivity modulations.

Luminance contrast sensitivity for peri-saccadic stimuli was clearly multiplicatively reduced as compared to sensitivity for stimuli presented before or after saccades, or during fixation. This result is in line with previous reports (Burr et al., 1982, 1994; Diamond et al., 2000; Uchikawa & Sato, 1995; Volkmann, 1986).

The topography of the contrast sensitivity function (Figure 3) was similar for stimuli presented peri-, pre-, and post-saccadically or during steady fixation. In all conditions, sensitivity de-

creased with eccentricity except for local minima of sensitivity observed at about the fovea and at around ±15 deg. The foveal decline of sensitivity is consistent with the relatively low spatial frequencies of our stimuli. The other two local minima occur at regions roughly corresponding to the blind spots; the drop of sensitivity in the +15 deg region, corresponding to the pre-saccadic retinal location of the saccade target, was less evident for stimuli presented in a pre-saccadic epoch; this relative pre-saccadic enhancement in the region of the saccade target may be related to the allocation of visual attention (Deubel & Schneider, 1996).

In a second set of experiments, we measured peri-saccadic chromatic contrast sensitivity for few crucial positions (see insets in Figure 5). In agreement with previous results (Burr et al., 1994; Diamond et al., 2000), we found sensitivity to be enhanced after the completion of the eye movement, with a peak effect of about 0.3 log unit occurring some 100 ms after saccade offset, uniform across the range of tested positions (which spanned some 15 deg of visual angle, at or above gaze level). Extending previous investigations, we observed the same enhancement of chromatic contrast sensitivity following simulated saccades, which suggests that the post-saccadic enhancement may be a by-product of the spurious retinal motion due to the movement of the eyes. Importantly, this finding dissociates the post-saccadic enhancement of chromatic sensitivity from saccadic suppression of luminance sensitivity, since the latter cannot be reproduced with a saccadic-like motion of the visual display (as demonstrated by Diamond et al., 2000).

Visual perception integrates relevant features (e.g., motion or form) across saccadic eye movements in a non-retinotopic coordinate system (Melcher, 2005; Melcher & Morrone, 2003), suggesting the possibility that saccade-related visual phenomena occur in coordinates attached to

the external space. Here, we found that, when considering a screen-centered coordinate system, strongest peri-saccadic suppression of luminance contrast sensitivity occurred for different stimulus regions at different points in time relative to saccade onset. In contrast, the time courses of peri-saccadic suppression were aligned for different stimulus positions defined in retinal coordinates, with the maximum reduction of sensitivity (by a factor of about 0.5 log unit) occurring for stimuli presented immediately after the saccade onset. When plotting sensitivity as a function of the retinal coordinates of the stimuli, we found that peri-saccadic sensitivity was scaled by an approximately constant factor relative to sensitivity during steady fixation. Based on this finding, we conclude that peri-saccadic suppression can be best described as occurring in a retinotopic frame of reference, reducing sensitivity by a divisive factor that is constant across the retinal space.

A spatially homogeneous peri-saccadic reduction of contrast sensitivity specific to luminance signals is consistent with the hypothesis that saccades selectively suppress neural responses in the M pathway, via dynamic gain control mechanisms (Burr et al., 1994; Diamond et al., 2000; Ross, Burr, & Morrone, 1996). The selective impairment of the M pathway can also account for the specificity of saccadic suppression to low-frequency luminance modulations, which constitute the preferential stimulus for this system. A dynamic reduction of gain is consistent with the present and previous (Burr et al., 1999, 1994) results: It is divisive, implying a reduction of neural responses proportional to the response amplitude, hence predicting the observed reduction of sensitivity by a constant scaling (divisive) factor across the retina. The dynamic gain adjustment may be triggered by extraretinal signals: a copy or corollary of the oculomotor command interfering with visual inputs before the detection stage (Diamond et al., 2000). Concurrent evi-

dence in support of a peri-saccadic gain reduction was recently obtained based on an equivalent noise analysis approach (T. L. Watson & Krekelberg, 2011). Physiological evidence supports the existence of a corollary discharge signal, relayed from the superior colliculus to the frontal eye fields through a specialized thalamic nucleus (Wurtz, 2008).

While psychophysical data are consistent with the hypothesis of a differential impact of saccades on the M and the P systems, little work has been dedicated to investigating the effect of saccades on the third geniculocortical pathway, the K system. Although the physiological properties of this system appear to be extremely heterogeneous, K cells are believed to be the primary target of color-opponent S-cone signals (Hendry & Reid, 2000) and S-cone isolating stimuli (i.e., stimuli modulated in chromatic contrast along the blue–yellow axis) have been employed in psychophysical studies to estimate the contribution of the K pathway to visual sensitivity (e.g. Sumner, Adamjee, & Mollon, 2002). Testing peri-saccadic sensitivity to this class of stimuli would provide information on the effect of saccades on activity in the K pathway; to our knowledge, no study has undertaken this investigation. The present experiments cannot address this issue, because the stimuli we employed (luminance modulations or modulation in chromatic contrast along the red–green axis with minimal stimulation of the S cones) were not designed to selectively stimulate the K pathway.

Neurophysiological investigations have revealed clear correlates of peri-saccadic suppression. Both electrophysiological measures in monkeys (Bremmer et al., 2009; Ibbotson et al., 2008) and fMRI experiments in humans (Kleiser, Seitz, & Krekelberg, 2004) revealed peri-saccadic suppression of visual responses in relatively high-level visual areas, notably the motion-sensitive area MT. As for earlier visual structures, fMRI studies in humans indicate suppression of visual re-

sponses in retinotopically defined V1 and in LGN (Sylvester, Haynes, & Rees, 2005; Vallines & Greenlee, 2006) and TMS results suggest a pre-cortical origin of peri-saccadic suppression (Thilo et al., 2004). However, electrophysiological recordings in monkeys indicate that M, P, and K cells in LGN behave similarly during saccades; LGN and V1 responses are not or weakly suppressed peri-saccadically and they are, in fact, enhanced after the saccade (Leopold & Logothetis, 1998; Reppas et al., 2002). In an attempt to reconcile these findings, (Wurtz, 2008) has recently proposed that suppression occurs at subcortical stages other than the LGN. The superior colliculus (SC) is a likely candidate given its involvement in the preparation of saccadic eye movements and the plausible prevalence of inputs from the M pathway to this structure. Recent results (Berman & Wurtz, 2011) support this line of reasoning, showing that saccadic suppression of visual responses in SC is accompanied by similar suppression in neurons of the inferior pulvinar compartment of the thalamus that are connected to the cortical area MT.

It can be concluded from our current data that saccades produce a selective suppression of sensitivity to luminance contrast, constant across a wide range of retinal eccentricities and compatible with an extraretinal origin. Saccades also cause an enhancement of sensitivity to chromatic contrast, but this should be considered separately from peri-saccadic suppression, since it is not specifically linked to the active execution of a saccade and may emerge as a by-product of the rapid whole-field retinal motion resulting from the movement of the eyes.

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Chapter 2: Spatio-temporal topography of saccadic overestimation of time

Abstract

Fast rapid eye movements (saccades) induce visual misperceptions. A number of studies in recent years have investigated the spatio-temporal profiles of effects like saccadic suppression or perisaccadic mislocalization and revealed substantial functional similarities. Saccade induced chronostasis describes the subjective overestimation of stimulus duration when the stimulus onset falls within a saccade. In this study we aimed to functionally characterize saccade induced chronostasis in greater detail. Specifically we tested if chronostasis is influenced by or functionally related to saccadic suppression. In a first set of experiments, we measured the perceived duration of visual stimuli presented at different spatial positions as a function of presentation time relative to the saccade. We further compared perceived duration during saccades for isoluminant and luminant stimuli. Finally, we investigated whether or not saccade induced chronostasis is dependent on the execution of a saccade itself. We show that chronostasis occurs across the visual field with a clear spatio-temporal tuning. Furthermore, we report chronostasis during simulated saccades, indicating that spurious retinal motion induced by saccade is a prime origin of the phenomenon.

Introduction

Saccades are fast ballistic eye movements that bring objects of interest into the fovea. With each saccade the projection of the visual scene sweeps across the retina at high speed. Yet, this drastic change in retinal input remains largely unnoticed and the world around us is perceived as stable. However, when probed with transient visual stimuli, perception is often far from veridical.

As an example, the perceived location of flashed stimuli is shifted in the direction of the saccade (Honda, 1989) or compressed towards the saccade target (Morrone, Ross, & Burr, 1997; Ross, Morrone, & Burr, 1997). Sensitivity to luminance contrasts with low spatial frequencies is actively suppressed (saccadic suppression) while sensitivity to stimuli with high spatial frequencies, as well as to color contrasts remains unchanged (Burr et al., 1994) or is even enhanced shortly after the saccade (Burr & Morrone, 1996; Burr et al., 1994; Knöll et al., 2011). These changes to perception are hardly ever noticed during everyday life.

One saccadic misperception that is easily demonstrated in a modern environment is saccadic chronostasis, also called the *stopped clock illusion* (Yarrow et al., 2001). When making a saccade to a clock, the first second often seems to last longer than the subsequent ones. Yarrow et al. (2001) demonstrated this perceptual effect by asking subjects to saccade to a counter. At a set portion of the saccade, the counter changed from '0' to '1' for a variable duration before it continued to count up to '4' in intervals of one second. Subjects judged whether the duration of the '1' was longer or shorter than the duration of the following one-second intervals. Yarrow and colleagues found that the duration of the '1' had to be shorter than the subsequent intervals in order to be perceived as lasting equally long. That is, the duration of the first interval was overestimated compared to the other intervals. This overestimation exceeded the duration of the saccade by about 50ms and increased by the same amount by which saccade duration increased (as induced by larger saccade amplitudes). Based on two further findings Yarrow et. al. concluded that the onset of a stimulus at the saccade target is antedated to the time at which an efferent signal occurred, e.g., the efference copy of the saccade command) to preserve object-constancy across saccades. (i) When the stimulus was moved at about 200°/s with the eyes stationary, thereby

simulating the visual consequences of a saccade, no chronostasis was observed. (ii) For two probe onset times (one fifth and four fifth of saccade duration), time was overestimated in a way that could be interpreted as antedating the onset of the stimulus to the same point in time prior to the saccade. When considering the physical duration of the stimulus, the overestimation was increased by the same amount the probe onset occurred later in the saccade.

Saccade induced chronostasis is not the only change in temporal perception during saccades. Morrone, Ross, & Burr (2005) reported that the perceived duration of an interval defined by two large horizontal bars, flashed in the retinal periphery around the time of a saccade, was compressed. In a critical time window just before saccade onset, the perceived temporal order of presentation of the two bars was even reversed. The underestimation of duration occurred for a period of about 300ms around saccadic onset. This compression of perceptual time was also present when the interval was defined by vertical bars placed near the saccade target.

In order to disentangle these two seemingly contradictory results of *overestimation* (reported only at the saccade target) and *underestimation* of time around the time of saccades, Georg & Lappe (2007) asked whether chronostasis could also be found at positions other than the saccade target. The authors used the same paradigm as Yarrow et al. (2001), but with the counter placed either at the saccade target or midway of the saccade trajectory. Since Georg and Lappe only found an overestimation of duration at the saccade target, they concluded that chronostasis is, unlike the compression of time, a rather local mechanism.

Our groups recently studied the spatio-temporal profile of saccadic suppression (Knöll et al., 2011). We found that contrast sensitivity was reduced during saccades across the visual field by

a constant factor in an eye-centered frame of reference. However, due to the differences in contrast sensitivity at different retinal eccentricities, the absolute sensitivity during the saccade was strongly dependent on the location of the stimulus in the outside world. Sensitivity was highest near the center of the saccade trajectory and reduced towards both sides (perpendicular to saccade direction) of this location. Perisaccadically, minimum sensitivity occurred later for stimuli near the fixations point as compared to stimuli near the saccade target. This temporal asymmetry occurred, however, only in a head-centered reference frame. The timing was symmetric for different positions in an eye-centered reference frame.

These results might be of critical relevance concerning the above mentioned study of Georg & Lappe (2007). It has been suggested by Yarrow and colleagues that the degraded perception of the stimulus onset, caused by saccadic suppression, might be a necessary condition for chronostasis to occur (Yarrow & Rothwell, 2003; Yarrow, Whiteley, Haggard, & Rothwell, 2006). One could thus expect chronostasis not to occur at the center of the saccade trajectory, where perisaccadic contrast thresholds are lowest. Chronostasis could thus still be a global mechanism, but absent or reduced at some locations. In this case we should be able to find chronostasis at other positions where peri-saccadic contrast sensitivities were similar to that at the saccade target. The time at which chronostasis first occurs could also differ for different locations, given the differences in time course of peri-saccadic contrast sensitivity at different screen positions.

We thus aimed to investigate the influence of saccadic suppression on chronostasis. In the first experiment we tested for chronostasis at the point of initial fixation, where we expected perisaccadic contrast sensitivity to be similar to that of the saccade target. In experiment two we expanded our sampling space by measuring the time course of chronostasis at the same two posi-

tions and additionally midway of the saccade trajectory. Experiment three aimed to investigate the visibility of the used stimuli during the saccade and the dependence of chronostasis on saccadic suppression. Finally, experiment four explored the dependence of chronostasis on an eye-movement related signal by mimicking the visual consequences of a saccade by means of a fast rotating mirror.

General methods

Stimuli and Procedure

All experimental procedures were in line with the declaration of Helsinki. Experiments 1 through 3 were performed at the Philipps-Universität Marburg (Germany). Experiment 4 was carried out at the Neuroscience Institute of the CNR in Pisa (Italy).

Experiments were conducted in a dark room with visual stimuli presented on a CRT screen (Experiments 1-3: Philips Brilliance 202P7; Experiment 4: Barco Calibrator) running at 100Hz with a resolution of 1152x864 pixels and viewed from a distance of 57cm, covering the central 39°x29° of the visual field (Experiment 1: 33cm and 62°x49°, respectively). The stimuli were generated on a PC using the in-House OpenGL/C++ software Neurostim (http://neurostim.sourceforge.net). Eye position data were recorded with an infrared eye tracker running at 500Hz (Experiment 1: SR Research Eyelink II, Experiments 2-4: SR Research Eyelink 1000). They were used for online control of behavior and stored for later offline analysis.

The general paradigm is depicted in Figure 1. In all experiments, subjects were either asked to fixate a given target or to make a visually guided saccade. A small square (constant stimulus, 1.5°x1.5°, Experiment 1: 0.85°x0.85°) was shown for the duration of a trial at one of three possible positions (*constant stimulus*). This square either served as the initial fixation point, as the saccade target or it was placed midway of the saccade trajectory. The square changed its appearance (decrease in luminance, except the second part of Experiment 3: change in color) at a variable time relative to the onset of the saccade. After a variable *probe* duration, it changed back to its original appearance for 1000ms before changing the appearance (identical to that of the probe) for a *test* duration of 500ms (Experiment 3.1: 50ms). It finally returned to its initial appearance until the participant pressed a key indicating whether the probe or the test duration appeared to have lasted longer. In the first part of Experiment 3 participants additionally specified whether or not they had seen both stimuli.

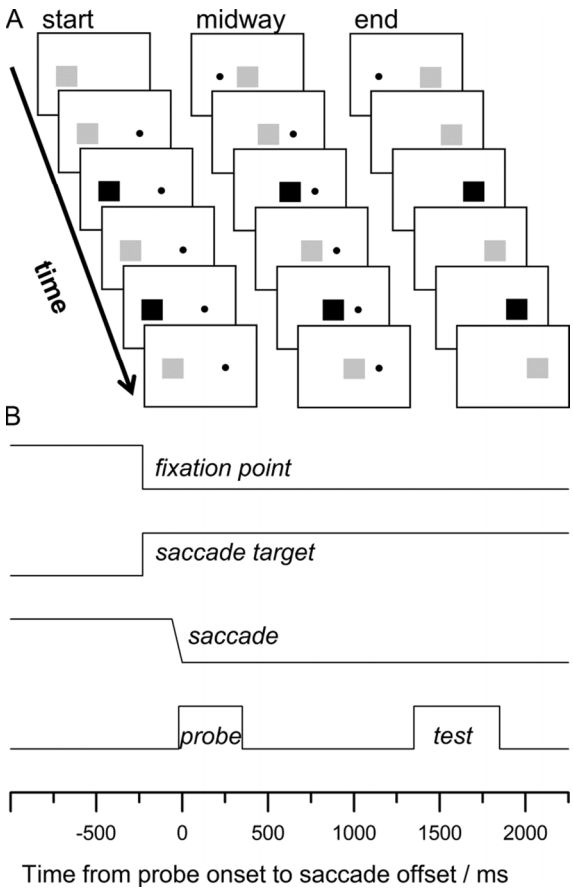

Figure 1: A Schematic representation of the stimuli and procedure used in this study (A). The images are not drawn to scale. Depending on the experiment the probe and test could be located either at the start or the end point of the saccade of midway of the saccade trajectory. B: The Time course of event occurrences. Participants initially fixated at the fixation point, which (if not occluded by the square) turned off while simultaneously the saccade target appeared (unless occluded). Participants saccaded to the saccade target. Around the time the square, constantly visible throughout each trial, changed in appearance for a probe duration. The duration had to be judged to an otherwise identical test duration that started 1000ms after the offset of the probe.

In saccade conditions, depending on the location of the probe, the saccade was cued either by the disappearance of the fixation point (0.5° back disc), by the appearance of a saccade target (0.5° back disc) or both. The square, constantly shown throughout the trial, occluded the fixation point or saccade target when located at the same position, i.e., the cue at the location of the probe was omitted. We had chosen this approach to avoid visual changes at the location of the probe and target unrelated to the task of judging the duration.

In fixation conditions, visual stimulation was identical to saccade conditions, except that the onset of the probe was determined relative to the mean saccade latency and participants held fixation at the location of the saccade target throughout a trial, unless stated otherwise. The fixation point at the location of the saccade target was visible throughout the trial, unless the probe was also placed at that location. In such case no fixation point was visible at the location of the saccade target, as it was occluded by the square.

Trials were automatically discarded and repeated later, if the participants showed an eye blink in the interval from the beginning of the trial to the end of the test duration, or if they failed to produce the required eye movement.

Data analysis

Eye traces from the left eye were analyzed offline. Saccades were detected by a velocity criterion of 200°/s. The start and end of a saccade were then defined by the last and first sample with a velocity below 20°/s, respectively. In saccade conditions, trials with saccade latencies less than 50ms or more than 300ms were discarded from further analysis. This was also done for trials

where the start and end position of the saccade differed by more than 1° or 2° from the expected start and end point of the saccade, respectively.

In saccade conditions, trials were sorted according to the time of probe onset relative to the off-set of the saccade and split into bins of 20ms. To obtain population results, data were pooled across participants. Datasets were balanced by reducing the number of trials from each participant in a given condition (fixation/saccade and stimulus position) to the minimum number off trials from any participant in that condition, but not for different bins within each condition. Psychometric functions were obtained by fitting cumulative Gaussians to the responses of the participants and the physical duration of the probe with the psignifit toolbox (Wichmann & Hill, 2001a, 2001b), which provided estimates of the 50% threshold (PSE) along with bootstrapped standard errors and 95% confidence intervals based on 1999 simulations. The distributions of the estimated parameters from these simulations were also stored for later statistical comparisons. The PSE of the probe describes the *subjective duration* at which it appeared identical to the test duration. It is important to note that a reduction in *subjective duration* implies an overestimation of the probe duration and vice versa.

Comparisons between two subjective durations were done by first subtracting the two distributions of the simulated estimates from each other. That is, each estimate from one distribution was subtracted by each estimate from the other distribution, resulting in 1999x1999 estimates of the difference. The difference was considered significant if the 5% quantile was larger than zero (one sided test) or if the 2.5% and 97.5% quantiles had identical sign (two sided test). One sided tests were used to determine the point in time for which the subjective duration was for the first

time lower than during fixation (onset on chronostasis) when analyzing the time course of chronostasis. Otherwise two sided tests were used.

Experiment 1

In this experiment we tested the hypothesis that chronostasis does occur at other positions than the saccade target. Specifically, we tested perisaccadic duration perception with probes either at the saccade target or the fixation point and compared it to corresponding conditions during steady fixation.

Methods

Participants

Seven naïve observers participated in Experiment 1 (age range: 22-27, 4 female). All had normal or corrected-to-normal vision.

Stimuli and Procedure

The general paradigm is depicted in Figure 1. At 500 to 1000ms after trial onset, participants were cued to make 25° horizontal rightwards saccades centered along the horizontal meridian. A gray square (0.85° width and height, 70% background luminance) shown throughout the trial on a white background (87cdm^2) located either at the start or end point of the saccade (*constant stimulus*). The detection of a saccade triggered the constant stimulus to change to a black *probe* (<1cdm^2) for a duration of 200—800ms chosen randomly for each trial. The change occurred between 30ms and 10ms before the end of the saccade. Subjects judged the duration of the probe relative to an otherwise identical *test* duration of 500ms that started 1s after the offset of the probe. As a control, duration perception was also measured during fixation for both probe

positions while fixating at the location of the saccade target. A total of 12143 valid trials were recorded with 10269 contributing to the population analysis. The remaining 1874 trials were removed in order consider the same number of trials per subject for each condition.

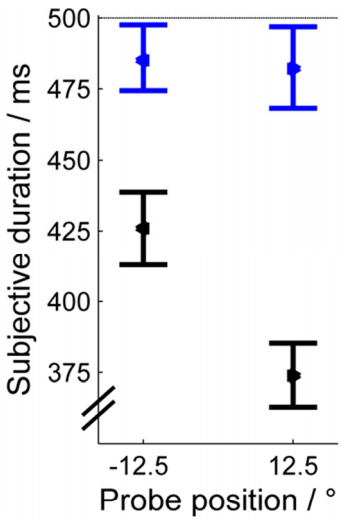

Figure 2: points of subjective duration equivalence at the fixation point (-12.5°) and the saccade target (12.5°) during fixation (blue lines) and perisacdically (black lines) for the population. PSEs are reduced perisacadically both at the saccade target and at the fixation point compared to PSEs during fixation. The dashed line indicates the test duration. Error bars show 95% confidence intervals.

Results

The subjective durations, i.e. the points of subjective equivalence (PSE) of the probe duration, are shown for the population in Figure 2. The perisaccadic PSE for probes located at the saccade end point (at 12.5°) was significantly reduced by about 110ms compared to the PSE during fixation ($p < 0.05$, see section 2.2. for detail). In other words, the perisaccadic probe had to be shorter during the saccade to be perceived lasting as long as a stimulus shown during fixation, demonstrating chronostasis. As expected from our hypothesis, the PSE at the saccade start point (at -12.5°) was also significantly reduced compared to fixation. The extent of overestimation of duration (about 60ms) was however significantly smaller compared to that at the saccade end point. The average saccade duration was 72ms.

Discussion

We demonstrated for the first time that saccadic chronostasis is not limited to the location of the saccade target but occurs also at the location of initial fixation. The amount of temporal overestimation (compared to fixation) at the saccade end point was comparable to previously reported data. Yet at the saccade start point it was only half the value. Participants reported that the probe

at the latter location was sometimes difficult to detect due to the combination of large eccentricity and small stimulus size. However, any related changes in the visibility of the probe onset should have also affected the control condition.

Experiment 2

To further characterize temporal overestimation as a function of spatial position, we next measured the time course of chronostasis at the start and end point of the saccade and at the midway of the saccade trajectory.

Methods

Participants

Five new naïve observers participated in Experiment 2 (age range: 20-31, 4 female). All had normal or corrected-to-normal vision.

Stimuli and Procedure

Stimuli and Procedure were identical to Experiment 1 with the following exceptions. In order to reduce detection problems due to retinal eccentricity, saccade size was reduced to 20°, and the size of the square was increased to 1.5°. The gray square remained at 70% of the background luminance, with the background luminance changed to 60cdm^2. The square could now also be located midway of the saccade trajectory (0°). In saccade conditions, the onset of the probe occurred -150ms to 100ms around the expected offset of the saccade. As a control, duration perception was also measured during fixation for all three probe positions while fixating at the location of the saccade target. A total of 39546 valid trials were recorded with 36225 contributing to the population analysis.

Results

In Figure 3 the subjective duration of the population is plotted for all three positions against the time of probe onset relative to saccade offset. Subjective duration was reduced not only at the saccade target (10°, cyan lines) and the saccade start point (-10°, orange lines), but also midway of the saccade trajectory (0°, magenta line). Well before the saccade (-140ms before saccade offset, mean saccade duration: 65ms), subjective duration was similar to that observed during fixation. Subjective duration started to drop first at the saccade end point (-120ms), followed by the central position (-80ms) and finally at -60ms to saccade offset at the saccade start point. It then progressed to drop at different rates to a peak reduction that occurred at about the time of saccade offset for all positions. Contrary to our expectation, the peak reduction as compared to fixation was stronger at the central position (-164ms) than at both the saccade target (-75ms) and the fixation point (-69ms). About 60ms after the saccade, perceived duration was back to the level during fixation, but continued to an underestimation of duration afterwards. This underestimation was again stronger at the central position (118ms) than at both the saccade target (42ms) and the fixation point (45ms).

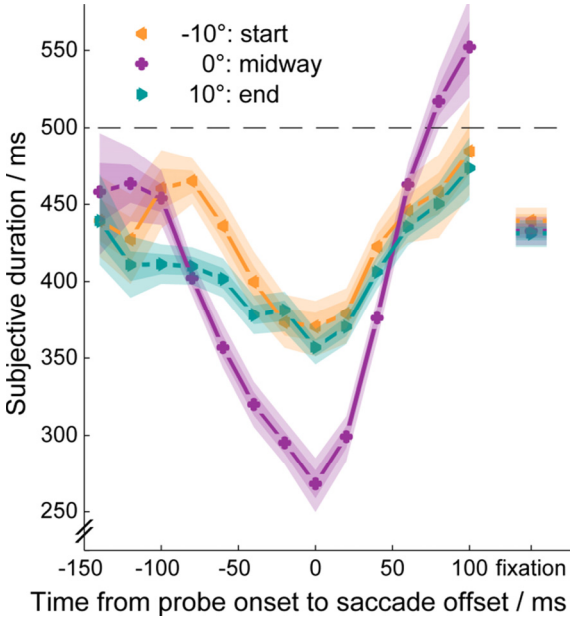

Figure 3: time course of chronostasis. Subjective durations (PSE) at the three measured positions (saccade start: orange, midway of the trajectory: magenta, saccade end point: cyan) as a function of probe onset relative to the offset of the saccade. These colors are also used in subsequent figures for corresponding conditions. PSEs are reduced perisacadically at all three positions compared to PSEs during fixation (rightmost data points). The dashed line indicates the test duration. Dark shaded regions indicate the SEM, light shaded region the 95% confidence intervals.

Discussion

Perceived duration was overestimated at all three positions, peaking at the time of saccade offset. This finding was unexpected, given our hypothesis that chronostasis should be reduced midway of the saccade trajectory due to higher contrast sensitivity at this position compared to the other two. Instead we found the amount of chronostasis to be largest at this position. It has been demonstrated that the fast motion of an image can degrade visual perception independently of saccadic suppression under certain conditions (Campbell & Wurtz, 1978; Diamond et al., 2000). It is possible that the contrast sensitivity to the stimuli used in our experiment was not only affected by saccadic suppression, but also by these presumably passive modulations of perception (a more detailed consideration can be found in the general discussion).

It should be noted that the overestimation observed at the saccade target was reduced compared to the value found in Experiment 1. Perceived durations tested at the saccade target and the fixation point were similar near the end of the saccade. This difference to Experiment 1 might be a result of the slight change of the stimuli in Experiment 1 and Experiment 2 (larger stimulus in Experiment 2).

Experiment 3

In Experiment 2 we found chronostasis midway of the saccade trajectory with a maximum overestimation of duration of 164ms as compared to fixation. We speculated that visibility of our stimulus was strongly reduced even at this point of the saccade trajectory. To test this explicitly, we measured the duration necessary to detect the probe at different times relative to the saccade. The goal of this approach was also to determine what duration of a stimulus would go unnoticed and might thus not be available for later processing.

To test if chronostasis occurs also in the absence of saccadic suppression, we measured the perceived duration for isoluminant stimuli presented at the saccade target in a second part of this experiment. We chose for this approach because saccadic suppression had been shown to not affect the detection sensitivity of such stimuli (Burr et al., 1994).

Methods

Participants

The same participants as in Experiment 2 participated in the first part of this experiment, while only 4 of the 5 participants completed the second part.

Stimuli and Procedure

The following differences to Experiment 2 were applied. Part 1: The probe was always located midway of the saccade trajectory, with a reduced test duration of 50ms and range of probe durations from 0ms (i.e. no probe was shown) to 100ms. Subjects not only reported the perceived duration but also stated whether or not they had seen both the probe and test duration. A total of 12262 valid trials were recorded with 11035 contributing to the population analysis. Part 2: The probe was always shown at the saccade target. In half of the sessions, stimuli and background were identical to those used in Experiment 2. In the other half, the square was initially red, shown on a yellow background. During the probe and test duration the square was green, isoluminant to both the background and the previously red square. Isoluminance was established using the minimum flicker technique (Boynton, 1979). Sessions were recorded in alternating order, with a balanced starting with either type across subjects. Duration perception was

tested during fixation, as well as at three times relative to the saccade offset: 0, 70 and 420ms. A total of 9811 valid trials were recorded with 9548 contributing to the population analysis.

Results

Duration threshold

The duration necessary for a stimulus to be detected in 50% of the trials (duration threshold) is shown in Figure 4A as a function of time relative to saccade offset. Well before and after the saccade as well as during fixation, the duration threshold was below 10ms, i.e. stimuli were typically visible even if they were presented for only one frame. Approximately 20ms before saccade onset, however, duration threshold started to increase, peaking at saccade onset with a value of 45ms (mean saccade duration: 61ms). The duration threshold then declined again and was back to normal approximately 20ms after saccade offset.

Chronostasis for isoluminant stimuli

The comparison of subjective durations (PSE) for isoluminant (green) and luminant (cyan) stimuli is shown in Figure 4B. In both cases, the subjective duration was reduced compared to fixation and to post-saccadic probe onset times. Chronostasis was larger for luminant (112ms) than for isomluminant probes (74ms). PSEs obtained during fixation differed between the isoluminant and the luminant condition (33ms difference).

Discussion

The duration threshold for detecting a stimulus was highly elevated during the saccade midway of the saccade trajectory. Elevation started about 80ms prior to saccade offset, i.e. 20 prior to saccade onset. This implies that the probe onset could not be seen at a location where detection

thresholds are known to be lower than at the saccade target. It supports the idea that saccadic suppression is not the only factor causing an imprecise percept of the probe onset in our experiment. The value of maximum duration threshold (45ms) was less than the overall duration of the saccade. In other words: even if the probe onset coincided with saccade onset, a stimulus shown only for the duration of the saccade did not go completely unnoticed. The value of maximum duration threshold was also significantly smaller than the amount of chronostasis (165ms) found in Experiment 2. Accordingly, chronostasis can cause an overestimation of duration by more than three times the duration that would go unnoticed if a stimulus appeared exclusively during the time of a saccade onset.

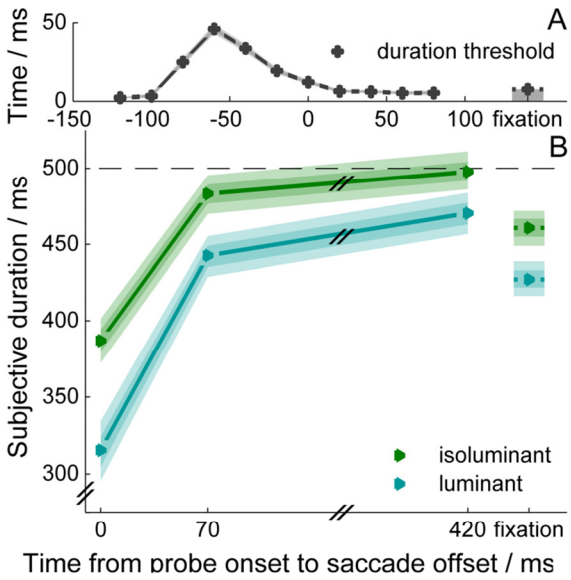

Figure 4. A: The duration threshold, i.e. the duration necessary for the probe to be visible in 50% of the trials, as a function of time to saccade offset. B: Subjective duration for luminant probes (cyan lines) and probes isoluminant (green lines) to the constant stimulus and to the background. Dark shaded regions indicate the SEM, light shaded region the 95% confidence intervals.

Another important finding of this experiment was that chronostasis also occurred for isoluminant stimuli. Such stimuli are known to be unaffected by saccadic suppression (Burr et al., 1994). This result excludes saccadic suppression as a necessary condition for chronostasis to occur. It does, however, not exclude chronostasis to be caused by an imprecise perception of the onset of a stimulus. Given the reduced chronostasis for isoluminant as compared to luminant stimuli, we conclude that the motion of the retinal image probably caused a sufficiently unclear probe onset to allow chronostasis to occur for isoluminant stimuli, and that the additional reduction in visi-

bility due to saccadic suppression for luminant stimuli increased the overestimation of duration observed in that condition.

Experiment 4

It is generally assumed that chronostasis relies on an eye-movement related signal. In previous studies, chronostasis did not occur when a probe was moved on the screen, while the participants kept fixation (Yarrow et al., 2001; Yarrow, Haggard, & Rothwell, 2004). It is possible that the speeds of 200°/s to 240°/s used in these experiments were not sufficient to cause an imprecise percept of stimulus onset. If chronostasis was to occur when the image is moved fast enough to be perceived as blurred, chronostasis would be a purely visual effect, independent of an efferent signal.

In this last experiment, we therefore tested if an eye-movement related signal was necessary for chronostasis to occur by using a mirror that moved the image of the screen at saccadic speeds.

Methods

Participants

Two naïve subjects and one author participated in this experiment. All had normal or corrected to normal vision.

Apparatus

A small galvanometer mounted mirror (M3S, GSI now Cambridge Technology) was placed closely before the participants' right eye, allowing participants to monocularly view a CRT placed at a total distance of 57cm. The mirror was controlled by the stimulus presentation program by sending a sequence of target voltages (corresponding linearly to the angle of the mir-

ror) using a 12-Bit USB-IO-Board (1208-FS, Measurement Computing). The signal was generated at 10kHz in a way that moved the mirror with a Gaussian speed profile by 18° within 46ms (v_{mean}=390°/s, v_{max}=890°/s). An identical second IO-Board was used to record a copy of that signal as well as the output of phototransistors placed on the CRT at 5kHz. This allowed a precise synchronization of the signal to the time of the stimuli presented. The left eye was shielded from visual input and its movements were recorded. In simulated saccade conditions, the recorded signal of the mirror command was analyzed analogous to the eye traces in saccadic conditions.

Stimuli and Procedure

Two conditions were recorded in this experiment. Saccade conditions were identical to the saccade condition in Experiment 2, with three exceptions. The probe was always located midway of the saccade trajectory. Probe onset was timed to occur at 0, 70 or 420 ms after the offset of the saccades and participants made 18° leftward saccades (9° to -9°) instead of 20° rightward ones. In simulated saccade conditions participants kept fixation, while a mirror was moved to mimic an 18° leftward saccade. Possible probe onset times where 200, 100, 50, and 25ms before and after offset of the simulated eye movement or directly at its offset. In order to suppress initiation of saccades upon appearance of the saccade target, the initial fixation point was not turned off in this condition. A total of 6717 valid trials were recorded with 6678 contributing to the population analysis.

Results

Subjective stimulus durations during simulated and real saccades are shown in Figure 5. The magenta line depicts the subjective duration when participants viewed the screen monocularly through a mirror and performed 18° leftward saccades. The onset of the probes could occur at

three possible offsets to the end of the saccade. As expected from the previous experiments, the subjective duration was reduced at the offset of the saccade, quickly recovering thereafter. Subjective duration was also reduced when subjects kept fixation while the mirror was moved in a Gaussian speed profile at saccadic speeds (blue line). Like with saccades, the reduction was strongest at the offset of the mirror movement, as compared to both before and after movement. The time course of this modulation was comparable to that observed during real saccades (see Figure 5).Yet, the PSE did not recover as quickly, remaining strongly reduced until 50ms after the movement. About 100ms after the movement it was back to a level comparable to that before the movement.

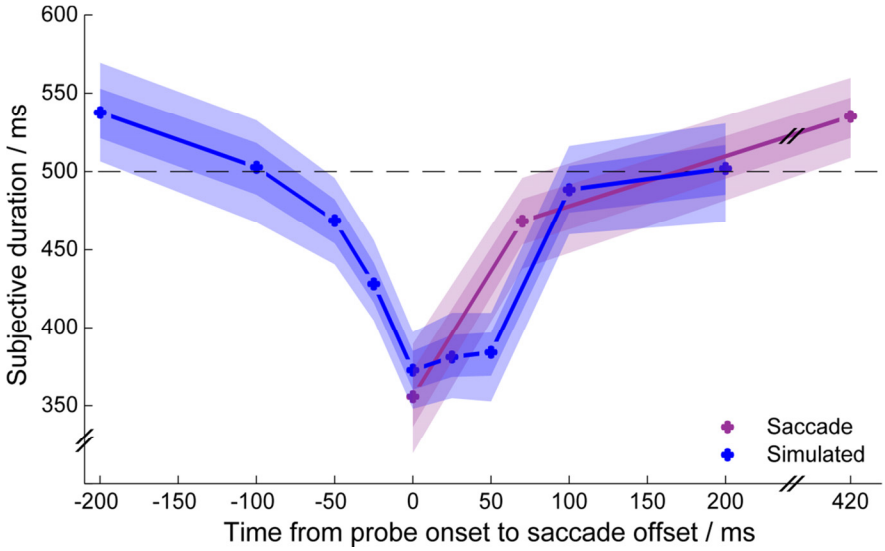

Figure 5. Subjective duration during real (magenta lines) and simulated (blue lines) saccades. In both cases, strongest reduction of PSEs occurred at the end of the movement. Dark shaded regions indicate the SEM, light shaded region the 95% confidence intervals.

Discussion

The results of this experiment clearly demonstrate that chronostasis can occur even in the absence of a saccade and thus in the absence of an efferent signal. The time course of chronostasis during simulated saccades, however, was prolonged, lasting up to 50ms after the end of the mirror movement compared to real saccades (Experiment 2) where subjective duration started to return to normal immediately after saccade offset. This result is consistent with the small overes-

timation observed by Georg & Lappe (2007) in the condition of simulated saccades. The overestimation in this case was smaller than the one we observed. However, in Georg & Lappe (2007) the retinal motion between the simulated and real saccade was not exactly equated. Given the pronounced temporal profile for simulated saccades, we can also exclude that the overestimation reflects an attentional or temporal order effect as observed during fixation for stationary stimuli (Rose & Summers, 1995).

In similar experiments where the visual motion of a saccade was also simulated with a mirror, neither saccadic suppression (Diamond et al., 2000) nor the peri-saccadic compression of space (Morrone et al., 1997) was found to occur in the absence of eye movements. In another experiment the pattern of spatial mislocalization appeared to be compression-like for saccades but shift-like for simulated saccades (Honda, 1995). The dependence of temporal perception for stimuli similar to those used in experiments of compression of time and perceived inversion of temporal order (Morrone et al., 2005), has also been investigated with simulated saccades (Binda, Cicchini, Burr, & Morrone, 2009). In an audio-visual temporal order judgment task, two subjects had to specify whether a transient isoluminant stimulus was presented earlier or later than a short sound. The visual stimulus was presented within 25ms before a saccade. To be perceived as occurring synchronous, the stimulus had to be presented 100ms prior to the sound. For simulated saccades, no such postdating was found. The independence of chronostasis on an eye-movement thus clearly dissociates it from many other saccadic misperceptions.

General Discussion

In a set of experiments we demonstrated that chronostasis is neither limited to the location of the saccade target (Experiment 2) nor to the execution of saccades (Experiment 4). Also, the peri-

saccadic overestimation of durations does not just compensate for the duration a stimulus that is perceptually 'invisible' due to saccadic suppression (Experiment 3), and differs for different positions (Experiment 2).

Visibility of probe onset without saccadic suppression

The amount, by which the subjective durations were reduced, differed between single experiments of our study and also between different stimulus positions. Yarrow et al. (2004) also reported different effect sizes in different experimental setups. They attributed this to subject specific differences, given that distinct groups of subjects participated in the different experiments. While this could also explain the differences found between Experiment 1 and Experiment 2 for stimuli at the saccade target, it cannot explain the differences we found for the different positions within Experiment 2 and between luminant and isoluminant stimuli in the second part of Experiment 3, as these were carried out by the same group of participants. We rather consider chronostasis to be highly dependent on the visibility of the stimulus onset. The differences would then result from differences in visibility, which can be affected by a variety of factors such as eccentricity of the stimuli in the visual field. If an imprecise probe onset is modulating chronostasis, saccadic suppression alone thus cannot have been the source of the imprecise probe onset, as chronostasis also occurred during simulated saccades and for isoluminant stimuli.

It has been shown that a broad frequency stimulus (the image of a room), shown for the duration of a saccade, can be perceived as smeared due to the blurring of the retinal image (Campbell & Wurtz, 1978). While the image was not perceived as blurred when the presentation exceeded the duration of the saccade by about 20ms, the peri-saccadic part of the stimulus was, most like-

ly not clearly perceived. The image was also perceived as undistorted when shown for only up to 5ms during the saccade. This effect is thus likely to be independent of saccadic suppression, which also occurs for short stimulus presentations and stimuli optimized to reduce motion blur (Burr et al., 1994). In our experiment, a stimulus was constantly shown throughout a trial at the location of the probe and could thus contribute to motion blur even if the onset of the probe itself occurred at the very end of the saccade.

Another, possibly related, degradation of visual perception due to fast image motion has been reported by Diamond, Ross, and Morrone (2000) for stimuli presented on a patterned background. The background consisted of small patches, each with a luminance randomly chosen to be the minimum or maximum screen luminance. Contrast sensitivity was strongly reduced during a saccade but importantly also when the image was moved by a mirror. The authors of this study demonstrated that the reduced visibility can be explained by the image motion of the patterned background. When the stimuli were shown on a homogenous background, decreased contrast sensitivity was only found during saccades. This clearly separates saccadic suppression from reductions in stimulus visibility due to image motion. As with chronostasis in our present study, the recovery of contrast sensitivity in their study was prolonged when the image was moved as compared to the saccadic condition. The background in our experiment was not patterned, yet the constant stimulus (the square) could have been sufficient to cause a (local) reduction in stimulus visibility.

It is thus likely that the onset of the probe was not optimally perceived even in the absence of saccadic suppression. While the conditions necessary for chronostasis to occur may typically on-

ly exist during saccades (imprecise percept of probe onset due to saccadic suppression and/or the high speed of the retinal image), it is definitely not dependent on the saccade itself.

Taking this consideration a step further, one could argue that it might be the background motion induced reduction in contrast sensitivity described by Diamond, Ross, and Morrone (2000) that mediates a large part of chronostasis. If a constant stimulus would cause a local reduction in contrast sensitivity, it could explain why chronostasis could not be found when the counter was displaced during the saccade (Yarrow et al., 2001). If this was the case, the constant stimulus would not be required for object constancy as previously assumed, but only to cause an imprecise onset of the probe. Other stimuli, where saccadic suppression alone causes such a sufficiently imprecise perception of its onset, might be able to cause chronostasis without a constant stimulus at the position of the probe.

It is important to note that the overestimation of duration was not necessarily completely unrelated to saccadic suppression. The overestimation was larger for luminant than for isoluminant stimuli. Also, the time at which chronostasis first occurred differed for the different stimulus positions with the same pattern observed for saccadic suppression (Knöll et al., 2011).

Chronostasis midway of the saccadic trajectory

At first glance our demonstration of chronostasis midway of the saccade trajectory appears to be contradicting the results of Georg & Lappe (2007), who did not find chronostasis at this location. In fact, our hypothesis based on the spatio-temporal profile of peri-saccadic contrast sensitivity (Knöll et al., 2011) was, that stimuli shown during saccades midway of the saccade trajectory should have a higher visibility than at the saccade target. The stimuli used in our experiments

however could not be detected at this location for a large proportion of the saccade (first part of Experiment 3), allowing for chronostasis to occur. We conclude that the stimuli used by Georg & Lappe (2007) might have not been affected by motion blur as much as our stimuli and might thus have been peri-saccadically visible midway of the saccade trajectory, but not at the saccade target. The comparably high spatial frequencies of digits (the stimuli) might have allowed to adequately perceive the stimulus when it was foveated during the saccade. Given that the onset of the probe occurred after the eye passed the screen center (two thirds of saccade duration), it is possible that the visibility of the stimulus, during the saccade and before probe onset, might have limited the occurrence of chronostasis. Thus visibility of the stimulus (a counter) might be the reason chronostasis at the midway of the saccade trajectory could not be found in their study.

No common target of antedating

Yarrow and colleagues proposed that the onset of the perisaccadic probe is antedated to a fixed point in time, relative to the onset of a saccade; namely to the occurrence of an efferent trigger, e.g. an efference copy of the saccadic motor command (Yarrow, Johnson, et al., 2004). This view was further supported by an experiment in which they asked subjects to judge the onset of the probe relative to an auditory time marker (Yarrow et al., 2006). The authors confirmed that the overestimation of the duration is in fact caused by antedating the onset of the probe. While the fact that we found chronostasis for simulated saccades excludes an efferent signal as a 'target' for this antedating, the onset could still be antedated to the start of the image motion or the start of visual degradation of the stimulus onset.

The first possibility would predict the onset to be antedated to a fixed point in time. Our results show strong variations of the amount by which a stimulus is overestimated with stimulus positions, thereby excluding this option. This finding also supports the second possibility, as the time course of perisaccadic contrast sensitivity also varies with the location of a stimulus on the screen. Thus the onset of chronostasis varies in a similar pattern as visual degradation due to saccadic suppression. However the visual degradation (the duration necessary for a stimulus to be detected) started to increase only at about 20ms before saccade onset (Experiment 3), while the duration was overestimated by 100ms in addition to the saccade duration. It thus seems unlikely that the onsets of the stimuli are antedated to a particularly marked event.

A possible mechanism of chronostasis

It has been demonstrated that the perceived temporal order of two stimuli, shown in rapid succession at the same location, can be reversed when the second stimulus had a lower contrast than the first (Bachmann, Põder, & Luiga, 2004). If the reason for this reversal is that the onset is perceived to occur earlier for a stimulus with low than with high contrast, this explanation might also be able to explain chronostasis. The contrast sensitivity is reduced during the saccade and recovers afterwards. The onset of a probe that is switched on during the saccade might thus only be detected as soon as the contrast detection threshold returns to a value below the probe's contrast. It might thus be encoded like an onset of (subjectively) very low contrast. This could cause the onset to be perceived as earlier compared to the onset of the test stimulus and would result in chronostasis. It is, however, unclear if the changes in temporal perception described by Bachmann et. al can be as large as the overestimations in duration of up to 164ms observed in this study.

Another probable possibility is that the speed of the constant stimulus (and of the probe) directly influenced the perceived duration. Motion is known to influence temporal perception. A stimulus with high speed is perceived to last longer than slow stimuli (e.g. Brown, 1931; Kaneko and Murakami, 2009; Tomassini et al., 2011). Watson & Krekelberg (2009) have demonstrated that a peri-saccadic stimulus that is not consciously perceived can nevertheless induce a shape contrast illusion. Even though the peri-saccadic part of the stimulus and its motion were not available to conscious perception, it might have still contributed to the perception of the probe's duration.

Other forms of chronostasis

Overestimation of durations have also been reported in the tactile (Park, Schlag-Rey, & Schlag, 2003; Yarrow & Rothwell, 2003) and auditory (Hodinott-Hill, Thilo, Cowey, & Walsh, 2002) domain. While Park et al. (2003) and Yarrow & Rothwell (2003) assumed that chronostasis may be a mechanism related to actions in general (as opposed to being specific for saccades), Hodinott-Hill et al. (2002) argued that the overestimation of durations is due to arousal. As evidence, that action is not a necessary condition for chronostasis to occur, they related the results of Yarrow et al. (2001) to those of Rose & Summers (1995) who had reported an increased perceived duration for the first and partly also the last of a sequence of visual stimuli. While this phenomenon could explain an overestimation of duration of a sequence, it should have also affected the control condition, in which the same sequence was shown while participants kept fixation. In fact, in all our control conditions, the first stimulus had to be slightly shorter to be perceived lasting as long as the second. But this overestimation was much smaller than the chronostasis found in the main conditions. A follow up study, however, demonstrated duration overestimations unrelated to

the execution of actions in the auditory domain (Alexander, Thilo, Cowey, & Walsh, 2005). The authors argued that the saccade acted as a cue as to when the duration judgment has to be done, causing an increase in arousal, which causes an increased rate of a hypothetical internal clock and thus the duration to be overestimated. Some discrepancies however remained, making it unclear how these results translate to the visual system. First, arousal should also be increased when the counter is unexpectedly displaced during the saccade. Chronostasis, however, was not found under such circumstances. Secondly, arousal should be independent of the location of the counter in general. Yet, we and others found strong modifications of chronostasis with probe position. It is also unclear why the overestimation would scale with saccade duration in this framework, as small and large saccades should be similarly effective cues as to when the duration judgment is to be made. The demonstration that the duration overestimation is caused by an antedating of the stimulus onset (Yarrow et al., 2006) is another strong hint, that arousal cannot easily explain the results of saccade induced chronostasis.

Conclusions

Chronostasis is not a saccade-specific mechanism, limited to occur at the location of the saccade target. It rather reflects a global mechanism for duration estimation of visual stimuli. The overestimation of durations could be caused by an active compensation in conditions where the exact time of a stimulus onset is not clearly perceived, but it appears possible that it is a passive result of how the time of a stimulus onset is predicted by the visual system in general.

Acknowledgments

The research leading to these results has received funding from the European Union (project MEMORY, FP6-NEST; FP7- STANIB -IDEA grant) and from the Deutsche Forschungsgemeinschaft (GRK-885-NeuroAct and FOR 560). We thank Judith Vornwald for help with data acquisition.

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Chapter 3: Responses in macaque area MT to single and double pulse stimulation during fixation and saccades

Abstract

Visual perception is altered in many ways during saccades. One example is a peri-saccadic loss of visual sensitivity to luminance contrasts (saccadic suppression). The neuronal substrates for these perceptual changes are largely unknown and there is an ongoing debate about the stage of visual processing at which saccadic suppression occurs. To investigate a potential role of the middle temporal area (MT) in peri-saccadic perception, single cell responses to single and pairs of stimuli were recorded in the macaque area MT during fixation and around the time of saccades. During fixation, responses to the second of a pair of stimuli were strongly reduced even with the largest tested stimulus onset asynchrony (SOA) of 107ms. For SOAs of less than 50ms, there was no detectable response to the second stimulus. The reduction of responses was accompanied by an increase in response latency to the second stimulus. Responses to stimuli shown during the second half of a saccade were also reduced, but the latencies remained unchanged. The reduced response was comparable to that of a stimulus shown during fixation with approximately one seventh of the contrast. When the second of two stimuli was shown during a saccade, no additional reduction to that observed during fixation occurred and the increase in latency was partially counteracted. These results are well in line with a possible contribution of areas of the dorsal stream in saccadic suppression.

Introduction

Even though we move our eyes several times a second to fixate new objects of interest, we do not take notion of the periods in between, where the image projected on the retina moves at very high speeds (saccades). This is easily demonstrated by watching one self's eyes through a mirror while moving the gaze from one eye to the other: The eyes appear as stationary - the motion of the eyes is not perceived. This is, at least in part, caused by a mechanism called saccadic suppression, which causes the detectability of certain stimuli to be reduced during saccades (Burr et al., 1994; Diamond et al., 2000; Knöll et al., 2011). Because saccadic suppression does not occur when the visual input caused by the saccade is simulated with a rotating mirror (Diamond et al., 2000), it is considered to be an active neural mechanism.

Not all stimuli, but only those possessing specific attributes, are peri-saccadically suppressed. Saccadic suppression occurs for stimuli modulated in luminance, but not in color and only for stimuli with low spatial frequencies (Burr et al., 1994). These are stimulus properties typically being processed by the magnocellular but not parvocellular visual pathway. These findings led to the assumption that saccadic suppression occurs at the latest stage of visual processing where the magno- and parvocellular path are still strictly segregated, namely at the lateral geniculate nucleus (LGN). The view of a pre-cortical source of saccadic suppression was fostered by results from transcranial magnetic stimulation (Thilo et al., 2004) which did not show a peri-saccadic reduction in detectability of phosphenes generated in the primary visual cortex (V1). That is, signals generated in V1 did not seem to be suppressed during saccades.

Results from electrophysiological experiments, however, are more diverse. While responses in the LGN have indeed been shown to be affected by eye movements (Fischer et al., 1996; Reppas

et al., 2002), these changes were not limited to the mangocellular layers of the LGN and manifested mainly in an enhancement of activity. Reported decreases in LGN activity around the time of saccades also occurred when the visual motion of the saccade was simulated by a rotating mirror (Noda, 1975). A peri-saccadic reduction of neural activity in the middle temporal (MT) and medial superior temporal (MST) areas has been found in a number of studies (Bremmer et al., 2009; Ibbotson et al., 2008; Thiele et al., 2002). Bremmer et al. (2009) expanded these results by reporting a reduction of signals during saccades in areas MT, MST and the ventral and lateral intraparietal areas (VIP and LIP, respectively). The time-courses of peri-saccadic excitability in MT, MST and VIP were qualitatively similar to behavioral data reported in psychophysical experiments (Diamond et al., 2000). All these areas are part of the dorsal stream and receive strong input via the magnocellular path. Yet, the response changes during saccades differed greatly between these areas. From these results, Bremmer et al. suggested that different, demand specific, mechanisms are at play in different visual areas during saccades and that visual input is suppressed at a later visual stage than the LGN. Further evidence for a cortical contribution in saccadic suppression comes from a behavioral study by Chahine and Krekelberg (2009) in which saccadic suppression was effected by changes of the visual input in the hemifield opposite to the stimulus. Because the LGN only encodes for one hemifield, this change in saccadic suppression cannot arise in the LGN alone.

If saccadic suppression results from neuronal changes at a stage other than the LGN, the question arises whether the highly differentiated stimulus characteristics of saccadic suppression could also result from neuronal changes in the higher visual areas of the dorsal stream. As a first step in answering this question, we focused on neural responses in area MT. We characterized

the responses of MT neurons to single static stimuli of different contrast and duration during fixation and saccades.

We aimed to test whether the reductions of peri-saccadic responses in MT are compatible with a causal relationship of MT in saccadic suppression. In such case, one could expect neural responses to high contrast stimuli, presented during saccades, to be comparable to responses to low contrast stimuli during fixation. We thus measured the peri-saccadic response to high contrast stimuli and compared it to the responses to stimuli of different luminance levels presented during fixation. This allowed to test whether the peri-saccadic response reductions in MT are quantitatively compatible with the hypothesis that saccadic-suppression results from changes in neural processing in this area.

Perception of time can also be perturbed during saccades and even during fixation. In a short interval around the time of saccade onset, the second of a sequence of two transient stimuli can be perceived as being the first (Morrone et al., 2005). An inversion of perceived temporal order was also reported during fixation when two stimuli were shown at the same location in rapid succession with the second stimulus having a lower contrast than the first (Bachmann et al., 2004). In order to characterize the dynamics of responses to sequences of short, stationary stimuli in MT, we also measured responses to pairs of stimuli, separated in time by different intervals both during fixation and saccades.

Methods

Experimental setup

At the beginning of each recording session the dura mater was penetrated with a guide tube. Parylene-C-coated tungsten electrodes (0.7–3 MΩ at 1 kHz; FHC; Bowdoin, ME, USA) were lowered using a microelectrode drive (NAN Instruments; Nazareth, Israel). Neural activity was recorded at 25 kHz with Alpha Lab (Alpha Omega Engineering; Nazareth, Israel). Action potentials were detected using a threshold criterion and sorted using the KlustaKwik algorithm (http://klustakwik.sourceforge.net) to obtain single unit activity. An Eyelink 1000 eye tracker (SR-Research; Ontario, Canada) was used to record eye movements of the left eye. Stimuli were generated using the in-House C++/OpenGL based software Neurostim (http://neurostim.sourceforge.net) and displayed on a 20'' CRT monitor (Sony GDM-520) running at 150 Hz and placed 57 cm in front of the eyes, covering the central 40° x 30° of the visual field.

We functionally identified MT cell activity with a circular motion stimulus (Bremmer, Ilg, et al., 1997; Krekelberg & Albright, 2005; Schoppmann & Hoffmann, 1976). Once a cell was isolated and its preferred motion direction identified, we determined the position of the cells' receptive field (RF). This was done by briefly presenting patches (5° x 5°) of coherently moving dots in a random sequence at 36 possible positions within a 6 x 6 grid (Krekelberg & Albright, 2005).

Visual Stimuli

A schematic diagram of the visual stimulation and the paradigm used is illustrated in Figure 1. In all trials, the monkey had to keep fixation or make a cued saccade. In each trial of the main

experiment, one or two white large horizontal bars (40° x 3°; equal energy white: x=0.33, y=0.33; CIE color space) were presented on a black background (monitor black: 0.2 $cd \cdot m^{-2}$) in a dimly lit room (Figure 1A). In fixation trials, each presented bar could be centered on either of two positions: At the vertical center of the previously mapped receptive field (RF) or 15° above or below the RF, depending on whether the RF was located below or above the screen center, respectively. In trials where only one bar was shown, it could be presented at one of five luminance levels (1, 3, 9, 27 or 64 $cd \cdot m^{-2}$). The range of possible durations of the bar depended on the position. When presented at the location of the RF, the duration could vary in four steps from 6.7ms to 113.3ms (1, 3, 5 or 17 frames), while it was always shown for one frame when located outside the receptive field. In trials with two bars, these were shown with a stimulus onset asynchrony (SOA) of 2, 4, 8 or 16 frames and were visible for one frame each (Figure 1B). The first bar always had a fixed luminance of 64 $cd \cdot m^{-2}$. Luminance of the second bar could have one of the values used in single bar trials. Responses to trials without visual stimulation, both during saccades and fixation, were also recorded for a subset of cells.

Fixation and saccade trials were presented in separate blocks of 6 and 4 trials, respectively (Figure 1C). At the beginning of a fixation block, a red fixation point was shown on the center of the screen. About 300ms after the subjects' gaze was within 3° of the fixation points' location, the first stimulus was presented. While the monkey kept fixation, the other five trials were presented with at least 300ms between the last stimulus of a trial and the first stimulus of the next trial, resulting in a block duration of about 3.5 seconds. Upon successful completion of a block, the animal was rewarded with a drop of juice. Otherwise, i.e. if the monkey made an eye blink or

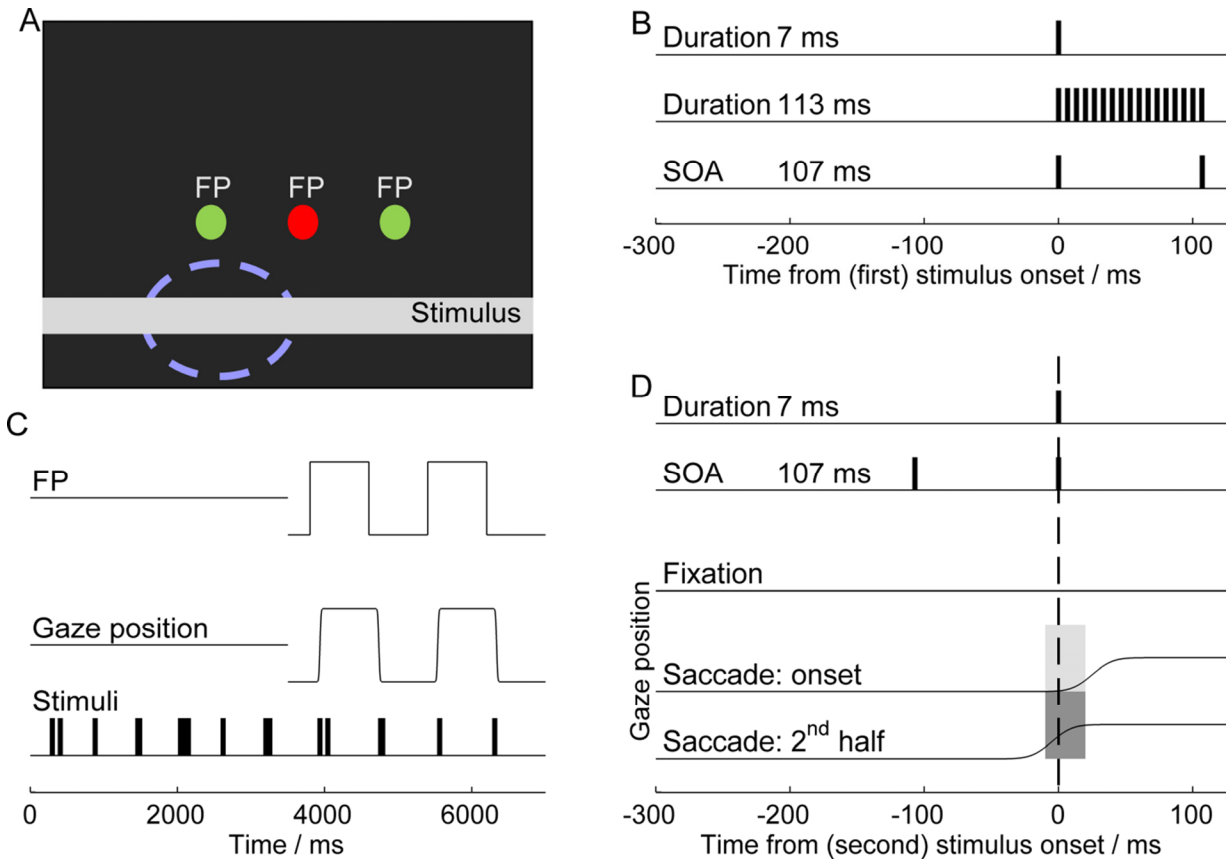

Figure 1. Paradigm. (A) Schematic image of the presented stimuli. The Stimulus was presented at the vertical center of the RF (indicated by the dashed blue ellipse), while the monkey either fixated centrally (red dot) or made saccades between the two positions indicated by the green dot. Note that the different color is chosen for illustration purposes and was always red in the experiment. The second possible stimulus location (15° above the first) is not shown, as this data was not analyzed here. (B) Time course for three conditions, a one frame stimulus (top), a 17 frame stimulus (middle) and a pair of one frame stimuli with a stimulus onset asynchrony of 16 frames. The time of the second stimulus onset is identical to the last frame of the 17 frame stimulus. (C) Event time course of a fixation block (6 trials) followed by a saccade block (4 trials). Top line indicates the position of the Fixation point. The middle line indicates the schematic gaze position, closely following the fixation point. In each trial one or two stimuli with different duration/stimulus onset asynchrony and luminance were presented (bottom line). (D) For one frame stimuli, data was binned depending on the occurrence of the stimulus onset relative to saccade onset. For pairs of stimuli, it was the distance of the saccade to the onset of the second stimulus that determined the bin. The first bin included onsets of 10ms prior to saccade onset to 20ms after saccade onset (saccade onset). The second bin was from 20ms to 50ms after the onset of the saccade (second half of the saccade).

looked away from the fixation point, the block was automatically aborted and repeated. Each fixation condition was repeated on average 8 times.

Saccade trials were identical to fixation trials, except that the monkey performed 15° saccades and that only conditions with maximum luminance of the bars were used. When only one bar

was presented, the stimulus was timed for the stimulus offset to occur at about the time of the onset of a cued saccade. In trials with two bars, it was the onset of the second bar that was timed to occur at about the time of a saccade (Figure 1D). At the beginning of a block of 4 trials, a fixation point was shown 7.5° to the left of the screen center. About 300ms after the subject first fixated the fixation point, it disappeared and a new fixation point appeared 7.5° to the right of the screen center, to which the monkey had to saccade as fast as possible. Saccades were performed in alternating directions for the remaining 3 trials within each block, with at least 300ms between the last presentation of a stimulus in one trial and the disappearance of the fixation point in the next trial. The horizontal extent of the bars ensured that their horizontal ends lay outside the cells' RF. The visual input within a cell's RF thus did not change due to the saccades. The subject was again rewarded upon successful completion of a block of trials. In addition to the criteria defined above, blocks were also aborted if the animal did not make a correct saccade within 50ms and 300ms after a change of fixation point. Each saccade condition was repeated 12 times on average.

Data analysis

Only responses to stimuli presented at the location of the RF were analyzed here. A total of 68 cells were recorded. For 15 of these cells, the signal could be isolated for more than 50 percent of the planned duration of the experiment. For the first 6 of these 15 cells, the offset of the stimulus was sometimes incorrect by one frame. Also, the maximum SOA and duration was set to 100ms instead of 106.7ms. Alignment of data to the actual onset of the stimuli was however always accurate. Removing these cells from analysis did not qualitatively change the results. For the remaining 9 cells, timing was completely accurate.

Single cell and population signals

For each cell, trials were sorted by presentation condition. In saccade trials, the onset of the saccade was extracted from automatic saccade detection events generated by the eye tracker. In these trials, conditions were further split up by the stimulus presentation time relative to the saccade onset (see Figure 1D), to include either trials with the stimulus presentation around the time of saccade onset (-10ms to 20ms relative to the onset of the saccade) or during the second half of the saccade (20ms to 50ms; average saccade duration 44ms). For each trial, the time of occurrence of action potentials (spikes) was determined relative to the stimulus onset of either the first or the second stimulus. Spike density functions (Richmond, Optican, Podell, & Spitzer, 1987) were computed by temporally filtering the occurrence of spikes (with 1ms precision) with a 20ms triangular filter separately for each condition.

The mean response of the population of all recorded cells was calculated by first normalizing spike densities to the maximum activity during the presentation of a single one frame stimulus during fixation at maximum luminance and averaging these normalized responses afterwards.

Difference to linear summation

In order to analyze if the response to two stimuli might result from a linear summation of the responses to each stimulus, an estimated linear response was calculated. This was done by adding, for each trial with two stimuli, the spike densities of each single stimulus response, with one of the responses shifted in time by the stimulus onset asynchrony of that trial. The linear responses were then averaged across trials of each condition. Finally, the difference between the measured and the linear response was computed to obtain a time resolved representation of nonlinear components of the response.

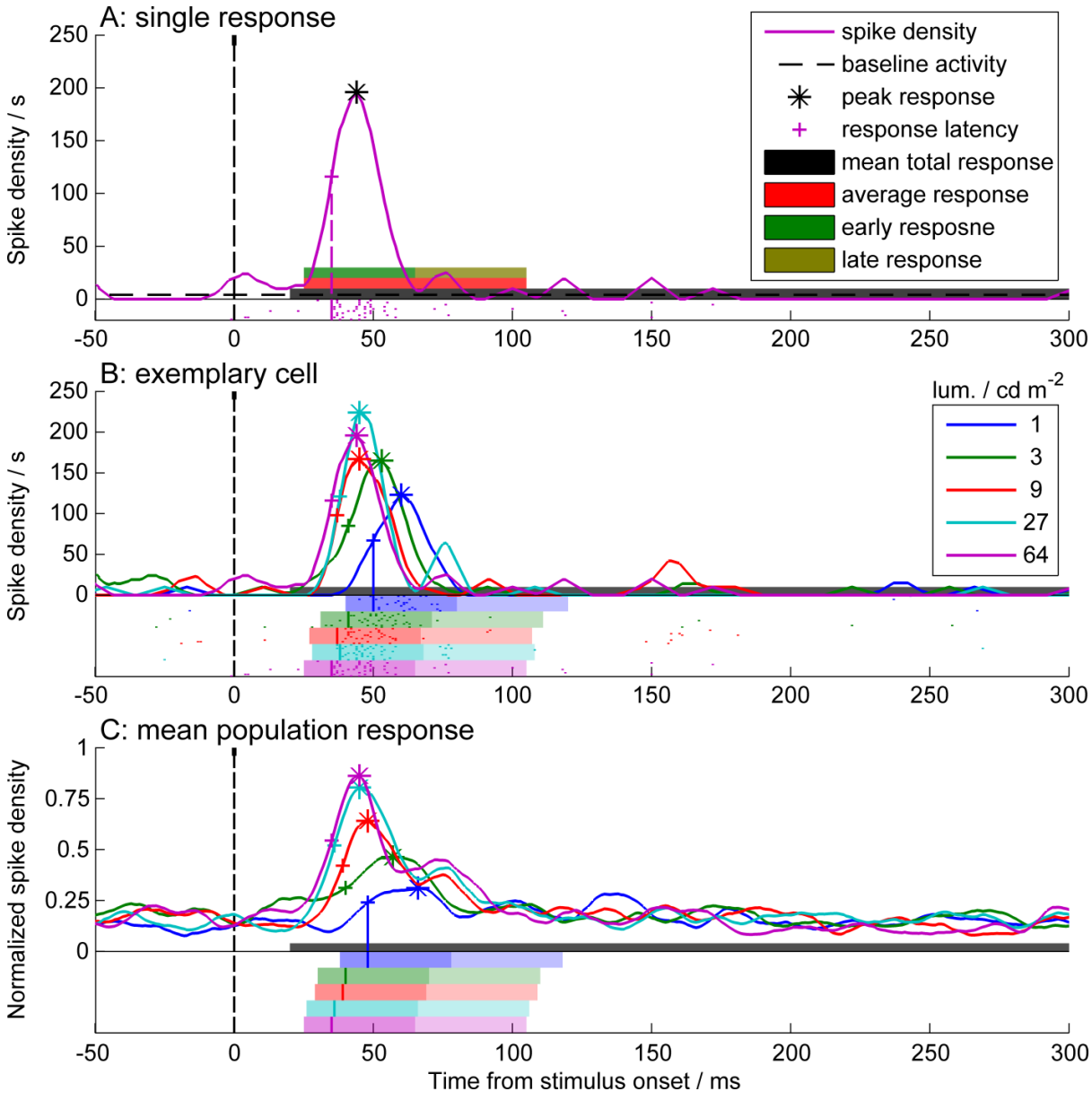

Figure 2. Luminance dependence of responses to one frame stimuli during fixation. (A) The parameters extracted from responses, shown for the response to a 64 cd·m^{-2} stimulus from an exemplary cell. Rasters below the curve indicate individual spikes occurring at different times, sorted by different trials (y axis). The resultant computed spike density is indicated by the magenta curve. The peak response and response latency (half peak time) are shown as an asterisk and a plus, respectively. Filled areas indicate the different averaging windows for the spike density to obtain the mean total response (black), average (red), early (green) and late (yellow) response. (B) Responses from the exemplary cell to the five different stimulus luminance levels. Display is analogous to A, except that the early and late responses are shown below the curves along with the spike raster diagram in different shades of the color encoding a given stimulus luminance. Note that the averaging was computed on the spike density however. (C) Mean population response shown as the data in panel B. Since the mean population response is normalized, no spike raster diagrams are shown.

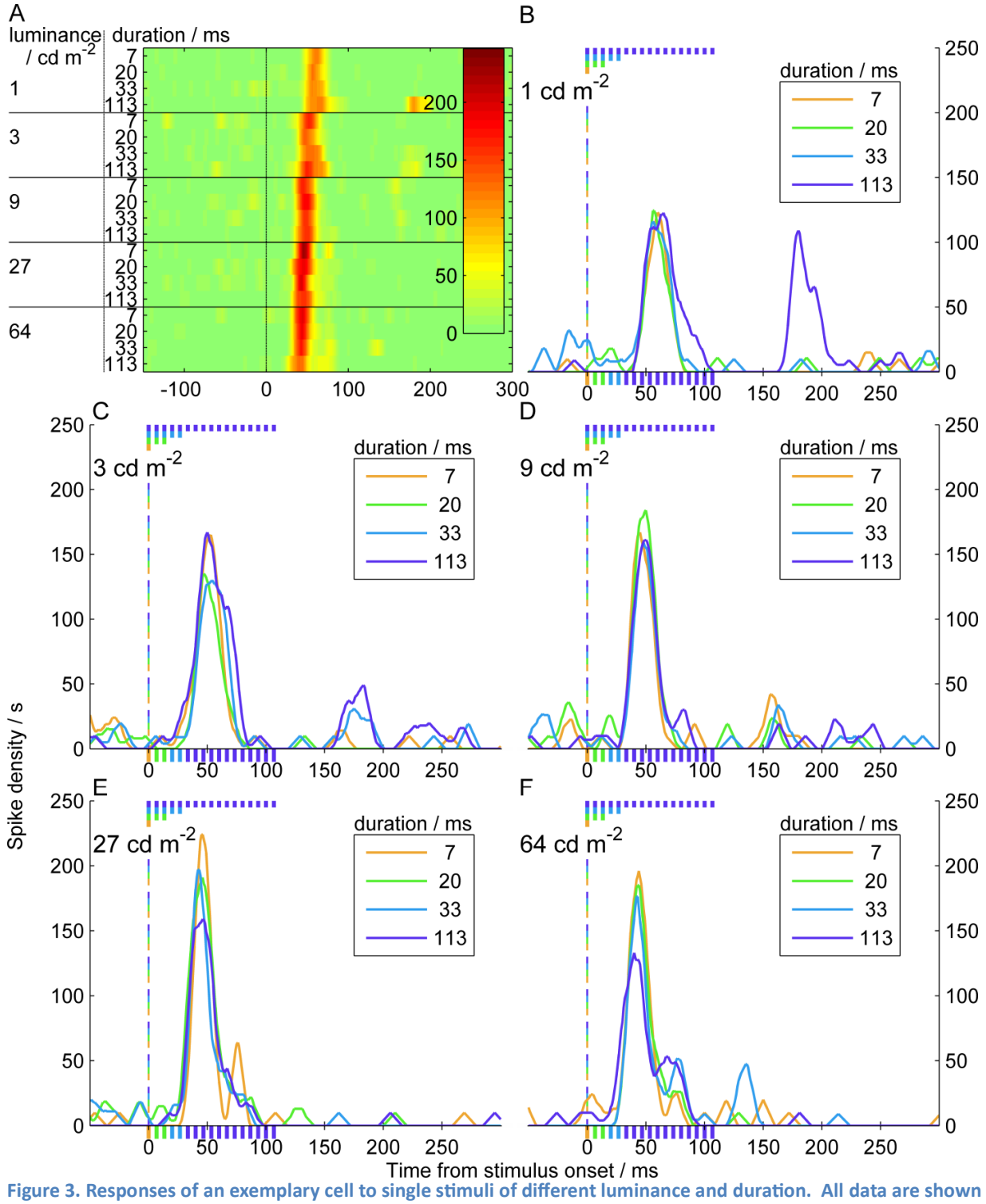

Figure 3. Responses of an exemplary cell to single stimuli of different luminance and duration. All data are shown as a function of time to stimulus onset. (A) A colormap of the responses sorted by luminance and duration. Green indicates no response, red indicates maximum response. (B-F) Each panel includes responses for different stimulus durations at a given luminance (as indicated in the top left corner of each panel). Colored horizontal lines indicate the time of each presented stimulus frame.

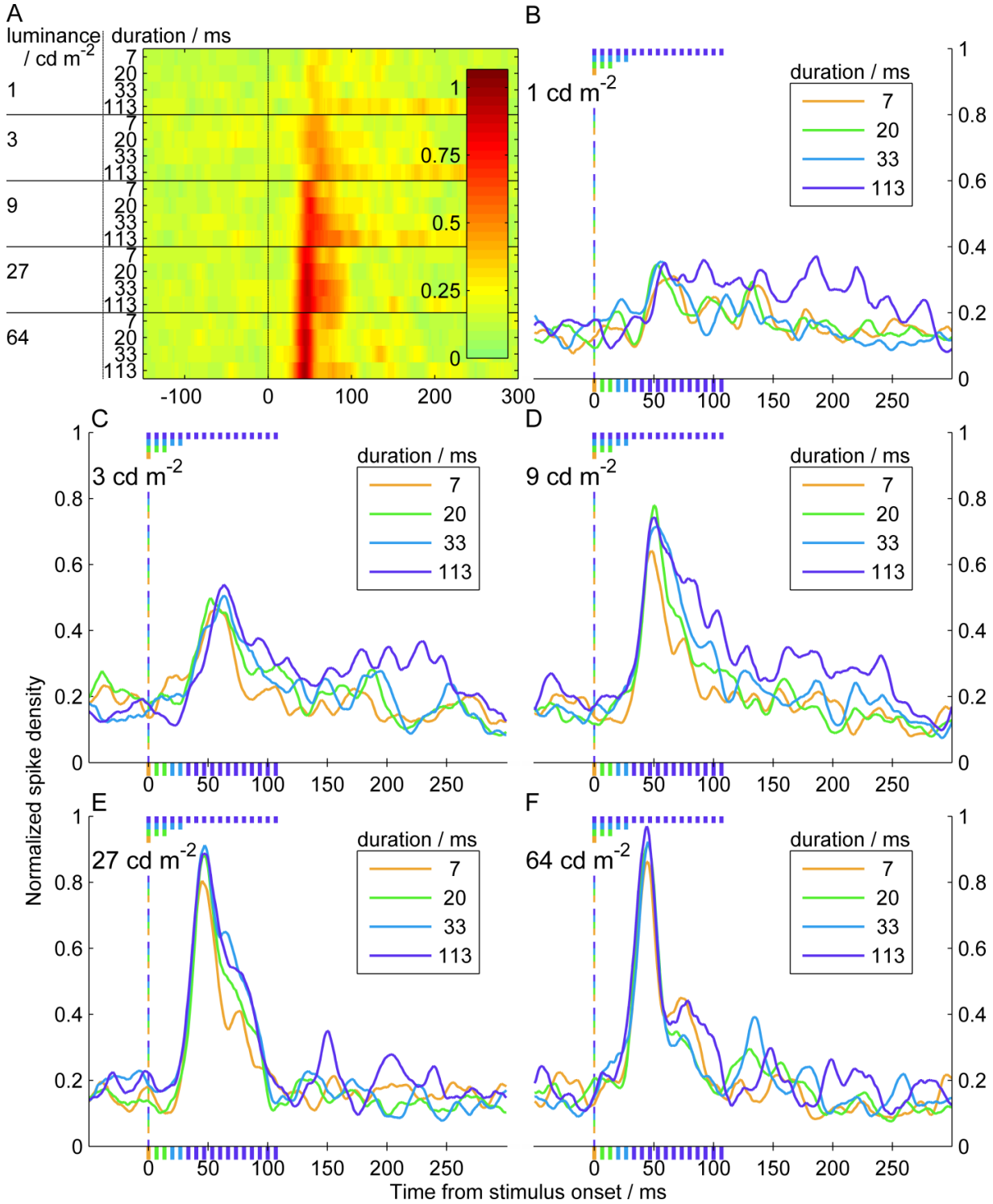

Figure 4. The mean population response to single stimuli of different luminance and duration. Data is shown analogous to Figure 3.

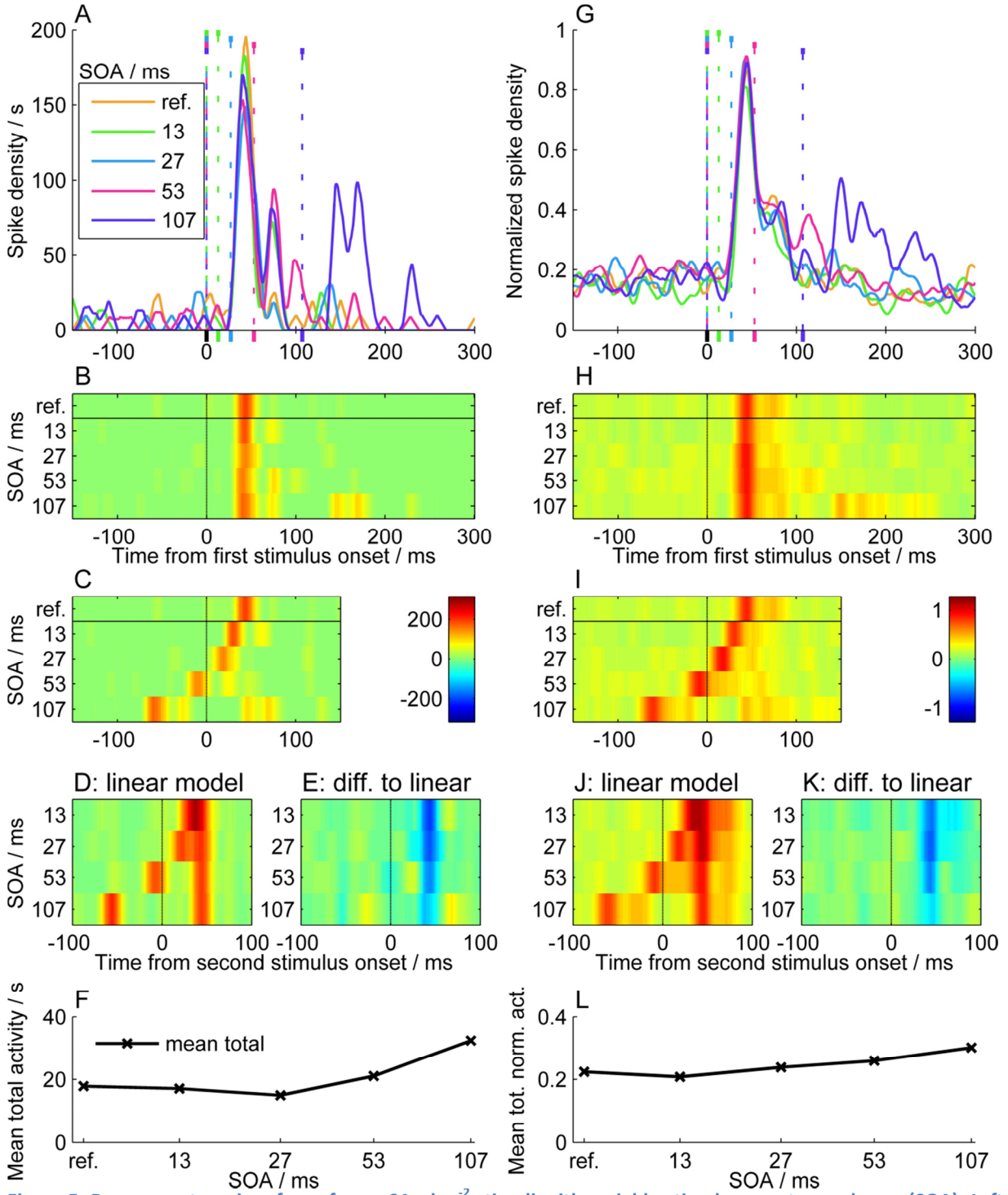

Figure 5. Responses to pairs of one frame 64 cd·m^{-2} stimuli with variable stimulus onset asynchrony (SOA). Left (A-F): Data from the exemplary cell. Right (G-L): The mean population response. In panels A and B the responses are shown relative to the onset of the first stimulus. Panel C displays the same data aligned to the onset of the second stimulus. Panels D and E show the response expected from linear addition and the difference from measured data, respectively. (F) Mean total response as a function of the SOA. Response of a single one frame stimulus with identical luminance is displayed as a reference (ref.). (G-L) analogous to Panel A through F, respectively.

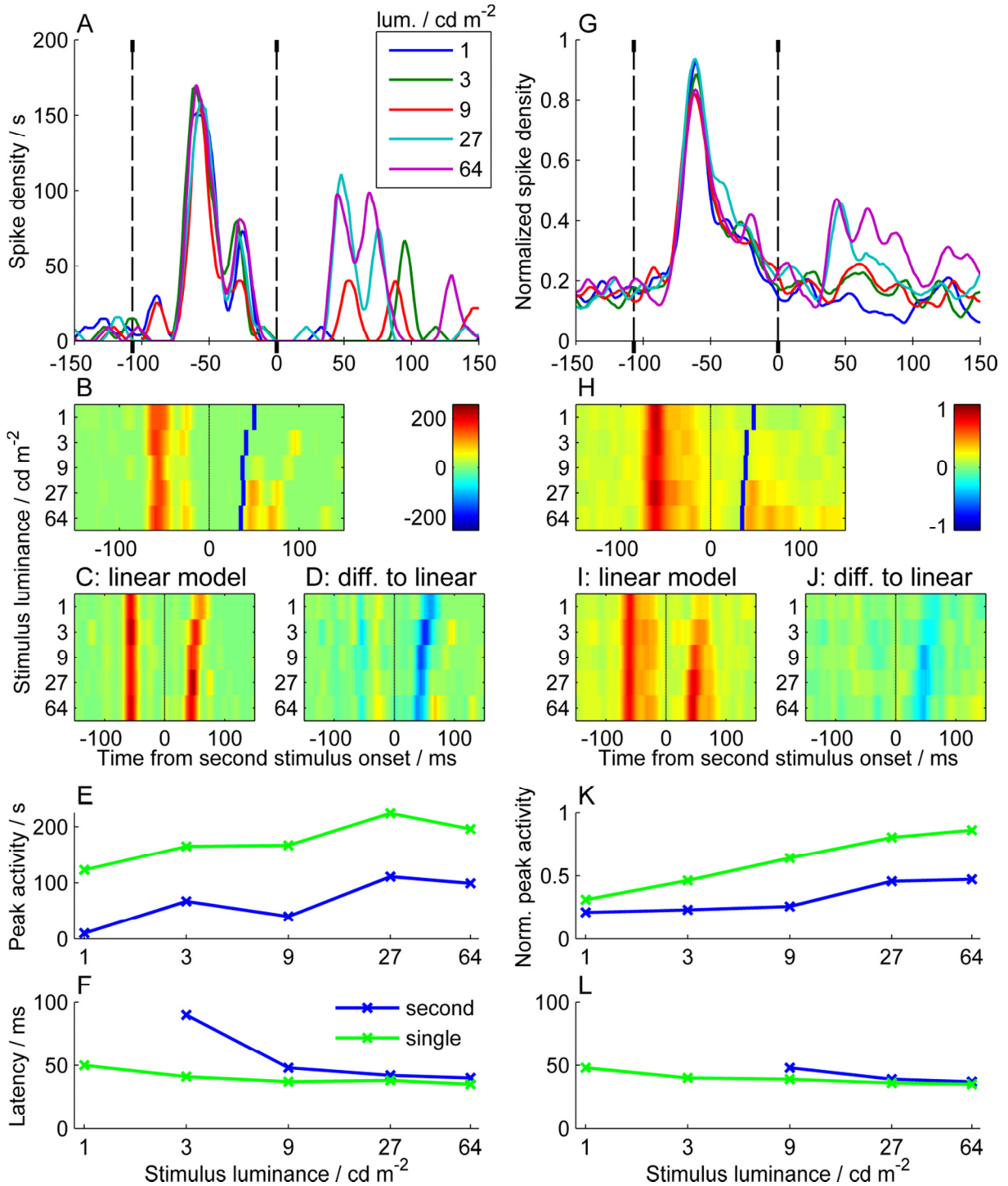

Figure 6. Responses to pairs of stimuli with different luminance of the second stimulus (maximum SOA). Left (A-F): Data from the exemplary cell. Right (G-L): The mean population response. Panels A through D show data relative to the onset of the second stimulus. Panel A and B show the measured response, while panels C and D show the response expected from linear summation and the difference from measured data, respectively (see methods). The peak activity (E) and latency (F) is shown as a function of stimulus luminance for the second stimulus (blue) and single one frame stimulus as a reference (green). (G-L) analogous to Panel A through F, respectively.

Extracted parameters

A number of response parameters were extracted per condition for each cell for later analysis (see Figure 2A for a visualization of the parameters). The *peak response* was defined as the maximum activity that occurred either after the first/single stimulus or after the second stimulus was presented. The *response latency* was defined as the first time the response reached half the value between the peak response and baseline activity. As the response latency is defined by the time to half peak, the activity will have typically started to increase earlier. The *average response* was computed as the mean response between 10ms before to 70ms after the response latency. The average response was sometimes further split up into an *early* and *late response period*, each covering half the interval used for the average response. The *mean total response* defines the mean response from 20ms up to 250ms after the occurrence of the first stimulus. If the peak response did not exceed the baseline activity by three standard deviations of the baseline activity, the latency was discarded from further analysis.

Statistical evaluation

In order to test whether these parameters (e.g. the response latency) varied for the population of cells other than by random processes, additional statistical tests were performed. For the 15 cells that could be recorded, a normal distribution of parameters cannot be assumed. Thus, non-parametric tests were used instead. To test if a parameter was influenced by another independent variable (e.g. whether latencies decreased with increasing stimulus luminance), regressions were first fit for each cell individually (one value for each parameter and condition). The slopes of the fits, most importantly the signs, were used in the next step. If the population of cells was affected by the independent variable, the signs of the slopes should result from a binomial dis-

tribution that is different from chance level (0.5). This was next tested using a *sign test*. A Wilcoxon *signed-rank test* was used to test if two parameters differed between two given conditions. A test was considered significant if the probability that the test criterion is met was greater than 95 percent (p<0.05). A trend was defined as a probability of at least 90 percent (p<0.1). A *relative change* in a parameters' value was calculated as the difference divided by the sum of the two values. As it would have not been possible to dissociate a response peak to the second stimulus from a late peak to the first, only cells where the latest response peak to a single flash at maximum luminance occurred no later than 120ms were included in statistical analyses of latencies to a second stimulus.

Results

Data will be presented for an overview for one exemplary neuron with a high signal to noise ratio and for the mean of all measured neurons' normalized responses (mean population response). Statistical analyses were performed by taking each cell's response into account.

Responses to one frame stimuli

We measured the responses of MT neurons to horizontal bars presented at different luminance levels and for different durations during fixation. This was done to characterize responses to stationary stimuli like the ones used to test saccadic suppression (Bremmer et al., 2009) and saccadic perceived inversion of temporal order (Morrone et al., 2005). We first considered responses to stimuli that were displayed for one frame at different luminance levels (Figure 2). Responses to these stimuli are of particular relevance, as they will serve as a comparison to responses to pairs of stimuli and to peri-saccadic stimuli. Both for a single exemplary cell (Figure 2B) and the mean population response (Figure 2C), the peak of activity occurred earlier with increasing lu-

minance. At the same time the peak activity increased. To test if the correlations of latency and peak response with stimulus luminance were not caused by random variations in the response pattern, sign tests were computed using each cells regression slope of the latency and peak versus stimulus contrast as input. These tests confirmed a significant increase in peak response (p<0.01, n=15) and decrease in latency (p<0.01, n=15) with increasing stimulus luminance.

Responses to stimuli of different durations

For the exemplary cell two findings are visible when considering all single stimuli shown during fixation (Figure 3A). First, response latencies were reduced and spike density was increased for higher luminance stimuli. Second, the responses were largely independent of stimulus duration. The responses to different durations were clearly phasic and nearly identical with two exceptions: at the lowest two luminance levels (1 and 3 cd·m^{-2}; Figure 3B and C, respectively), a response to the offset of the stimulus (*offset response*) was present at the longest duration (113 ms), but not for the others. Stimuli shown at the other luminance levels did not evoke an offset response. For the two highest luminance levels (27 and 64 cd·m^{-2}; Figure 3E and F, respectively) the onset response differed for the longest duration: It was reduced compared to stimuli of shorter duration.

The changes in latency and peak response with increasing luminance as well as the phasic nature of the response were also visible in the mean population response (Figure 4). At a luminance of 27 cd·m^{-2} (Figure 4E), the onset response was more sustained, but activity still rapidly returned to a baseline level before the offset of the stimulus with the largest duration occurred. At the three lower luminance levels (Figure 4, panels B-D) responses were also sustained for the longest stimulus duration, with a residual level of activity that remained elevated until at least

100ms after the offset of the stimulus. To further investigate the phasic nature of the response, the early and late responses were analyzed. For both intervals, the relative difference in activity between the shortest and longest stimulus duration was calculated for each stimulus luminance. The relative differences were next averaged across luminance levels to yield one value per cell. Sign-rank tests were then performed on this data, separately for each response period. For both response periods, activity was significantly increased for the longer stimulus duration compared to the shortest (p<0.005 each, n=15), with a relative increase of about 10 and 19 percent for the early and late interval, respectively. Offset responses were not visible at the population level.

Responses to sequences of two stimuli with variable SOA

To understand the responses to two stimuli shown in rapid succession, it is helpful to first consider those cases where both stimuli had identical luminance with variable stimulus onset asynchrony (Figure 5, left: exemplary cell; right: mean population response). When spike densities are aligned to the onset of the first stimulus (Figure 5, panels A, B, G and H) the initial responses to the first stimulus were similar to the response to a single stimulus (reference) independent of the stimulus onset asynchrony (SOA). For the exemplary cell a secondary response to the first stimulus was present for some SOAs at about 75ms. This response cannot be caused by the second stimulus, as it also occurred for the 107ms SOA. In that condition the second stimulus, presented 107ms after the first, cannot have influenced these early responses. For SOAs of 53ms and 107ms responses to the second stimulus were recognizable but reduced compared to single stimulus presentation. For the two shortest SOAs (13ms and 27ms) responses to the second stimulus were missing. When data were aligned to the onset of the second stimulus (Figure 5, panels C and I), the response to the second stimulus was delayed compared to the single stimu-

lus response (for the two larger SOAs). The reduced response to the second stimulus was particularly obvious when the measured response was subtracted from that to be expected from linear summation of the two single stimulus responses (Figure 5, panels D and J). In this view (Figure 5, panels E and K) it can be seen that responses to the second stimulus were absent when the time between the stimuli was small (dark blue at the location of the expected second peak) and that the response was slowly recovering for longer stimulus separations. The mean total response (average spike density from 20ms to 250ms after the first/single stimulus onset) for pairs of stimuli was initially close to that of single stimulus presentations, but started to increase for the two largest SOAs (Figure 5, panels F and L). Fitted regressions of each cells' mean total activity and the SOA with a subsequent sign test confirmed that this increase was significant ($p<0.001$, $n=15$). As a follow up, signed rank tests on the difference between the mean activity for a given SOA to that for the single stimulus presentation were performed for each of the four SOAs. A significant difference to a single stimulus was only found for the highest SOA ($p<0.0002$, $n=15$).

Responses to sequences of two stimuli with variable luminance

As a significant response to the second stimulus was only evident for the largest stimulus asynchrony, responses to different luminance levels of the second stimulus were next analyzed for this very SOA (Figure 6). Responses to the second stimulus can be seen for each but the lowest luminance (Figure 6, panels A, B, G and H). Since the activity to single stimuli was dependent on their luminance, these parameters have to be taken into account when analyzing the difference from the linear summation estimate to the measured response luminance (Figure 6, panels D and J). It is clear that the response to the second stimulus were reduced for all stimulus luminance levels. The peak responses to the second stimulus were decreased by a roughly constant

value when compared to the peak activity of single stimulus presentations with corresponding luminance (Figure 6, panels E and K). For the exemplary cell (Figure 6E), the general shape of the response pattern, with a maximum at 27 cd·m^{-2} and a dip at 9 cd·m^{-2} was similar both for the single and second stimulus presentation. For the mean population response (Figure 6K), peak activity was reduced to the level of baseline activity for the three lowest luminance levels. As a result, the decrease of the peak response compared to the single stimulus presentation was smaller for the stimulus at the lowest luminance. When detectable (see methods), response latencies to the second stimulus were consistently larger than to corresponding single stimulus presentations (Figure 6, panels F and L). The relationship of peak activity and latency might help to estimate whether the response to the second stimulus can be interpreted as a response to a

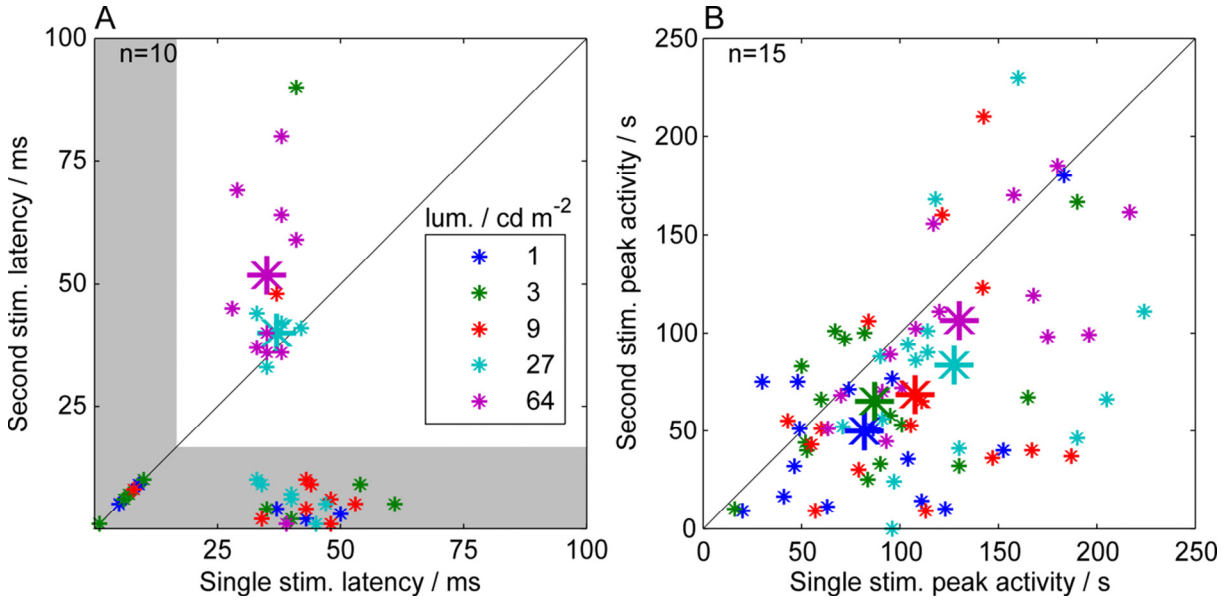

Figure 7. Single cell parameters for the second stimulus (107ms SOA) against those during single stimulus presentation separately for each tested stimulus luminance. (A) The latency for the second (y-axis) against single (x-axis) presentation for each cell. Data points plotted in the gray area along each axis indicate that a latency could not be determined for the other axis. For example for the cluster of latencies shown on the x-axis between 25ms and 60ms, a latency could not be determined to the second of a pair of stimuli. The value on the y-axis is scattered for better visibility of single data points. For data points on the bisecting line in this area, no latency could be determined in either condition. Large symbols indicate the mean of all cells. Data from three cells with very large detected latencies are not shown. The coordinates of these are: (85,122) ms and (132,/) ms at 1 cd·m^{-2} and (/,124) ms at 9 cd·m^{-2}; '/' indicating no detected latency. (B) Peak activity for each cell and stimulus luminance to a single stimulus versus that to the second of a pair of stimuli. Large symbols indicate the mean of all cells.

stimulus of reduced contrast. For the exemplary cell, the peak response to the second stimulus at maximum luminance was even lower than to the single stimulus with lowest luminance. Even though the latency was increased from 35ms to 40ms, it was still lower than the 50ms observed for the single stimulus with lowest luminance. Thus, for the exemplary cell, the measured profile of the response to the second stimulus was different from that to single stimuli of lower luminance. For the mean population response, the peak activity to the second peak was comparable to that of a single stimulus with 3 $cd \cdot m^{-2}$ luminance. In this case the response latency of 37ms was also increased from 34ms and matched that observed for the 3 $cd \cdot m^{-2}$ stimulus. In the single cell comparison of the parameters from the second versus the single stimulus presentation the increase in latency (Figure 7A) and decrease in peak response (Figure 7B) for the second stimulus are also visible. A signed rank test comparing the response latency to the second stimulus at maximum luminance to that during single stimulus presentation at identical luminance confirmed an increase of the latency for the population ($p<0.02$, $n=10$). Significance was not tested for other luminance levels, as latencies to the second stimulus could not be consistently detected for many cells with increasingly lower luminance. A signed rank test on the relative change in peak response to the second stimulus (again at the maximum luminance), compared to the peak response to single stimulus presentations revealed a significant reduction ($p<0.01$, $n=15$). Thus, the response to a second of a pair of stimuli was reduced and delayed.

Peri-saccadic responses to one frame stimuli

The peri-saccadic peak response to stimuli was also reduced compared to fixation for the exemplary cell (Figure 8A), both for stimuli shown around the onset of the saccade (10ms before until 20ms after the onset) and during the second half of the saccade (20ms until 50ms after saccade

onset). There is one important difference in the mean population response (Figure 8B) to the exemplary cell. While the peak response to stimuli shown in the second half of the saccade was also reduced compared to fixation, the response to stimuli around saccade onset appeared to be increased. However, single cell comparison of the peri-saccadic responses in the early, late and

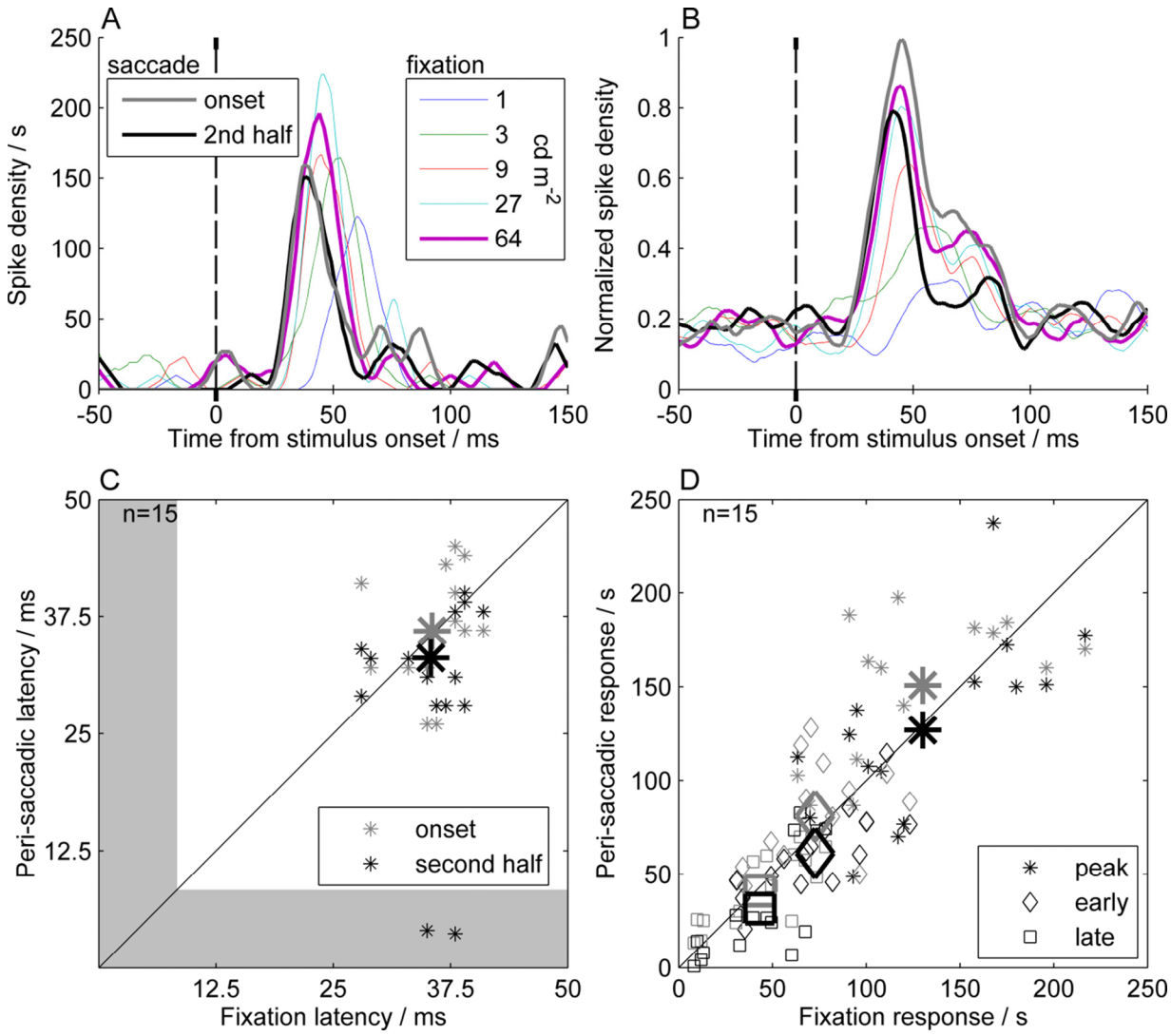

Figure 8. Peri-saccadic responses to single one frame stimulus (64 cd·m^{-2}) presentations. (A+B) The (normalized) spike density to stimuli shown around the onset of a saccade (gray) or during the second half of the saccade (black) as a function of time for the exemplary cell (A) and the mean population response (B). Responses to single stimuli presented during fixation with different luminance levels (same as in Figure 2) are shown as a comparison. (C) Single cell response latencies to stimuli shown during fixation against those during peri-saccadic stimulation. As in Figure 7A, the data plotted within the gray area indicate that no latencies could be determined in the peri-saccadic condition for those cells. (D) Peak (⁎), early (◇) and late (□) response period of single cells during the two peri-saccadic time intervals plotted against those observed during fixation. Large symbols indicate the means.

peak response period to those during fixation showed changes mainly during the second half of the saccade (Figure 8D). The increased response around the time of the saccade is only reflected as a trend in a signed rank test of the relative changes in peak response (p<0.08, n=15) but not significant in the early and late response periods. For stimuli shown during the second half of the saccade, the peak was also not significantly different during fixation. However, both the early and late responses were significantly reduced (p<0.05 and p<0.01, respectively; n=15). On average responses were reduced by about 8 and 25 percent for the early and late period, respectively. The overall reduction of the average response was roughly comparable to the responses to the 9 cd·m^{-2} stimulus for the population mean response and to the response to the 3 cd·m^{-2} stimulus for the exemplary cell. Interestingly, the reductions in response amplitude between the two saccadic intervals compared to the response during fixation were not accompanied by changes in latency. There were no significant changes when comparing each cells' latency in the two saccadic intervals to that during fixation with signed rank tests (Figure 8C; p>0.8 and p>0.16, respectively; n=15).

Peri-saccadic responses to a second stimulus

A possible interaction of mechanisms causing the reduced responses during saccades and to the second stimulus in a sequence of two stimuli was investigated by presenting the second stimulus around the time of a saccade (Figure 9). Only responses for the largest stimulus asynchrony (107ms) are analyzed here. Both for the exemplary cell (Figure 9A) and the mean population (Figure 9B), responses to the second stimulus were reduced compared to a single stimulus during fixation. The relative difference was significant in signed rank tests for the average response in both saccadic intervals (p<0.005 and p<0.02, respectively; n=15), but not for the peak response

(p>0.1 and p>0.3, respectively; n=15). In case of the exemplary cell, the reduction differed for the two saccade time intervals. This difference was however not as pronounced in the mean population response and signed rank tests of the relative difference of the average and the peak re-

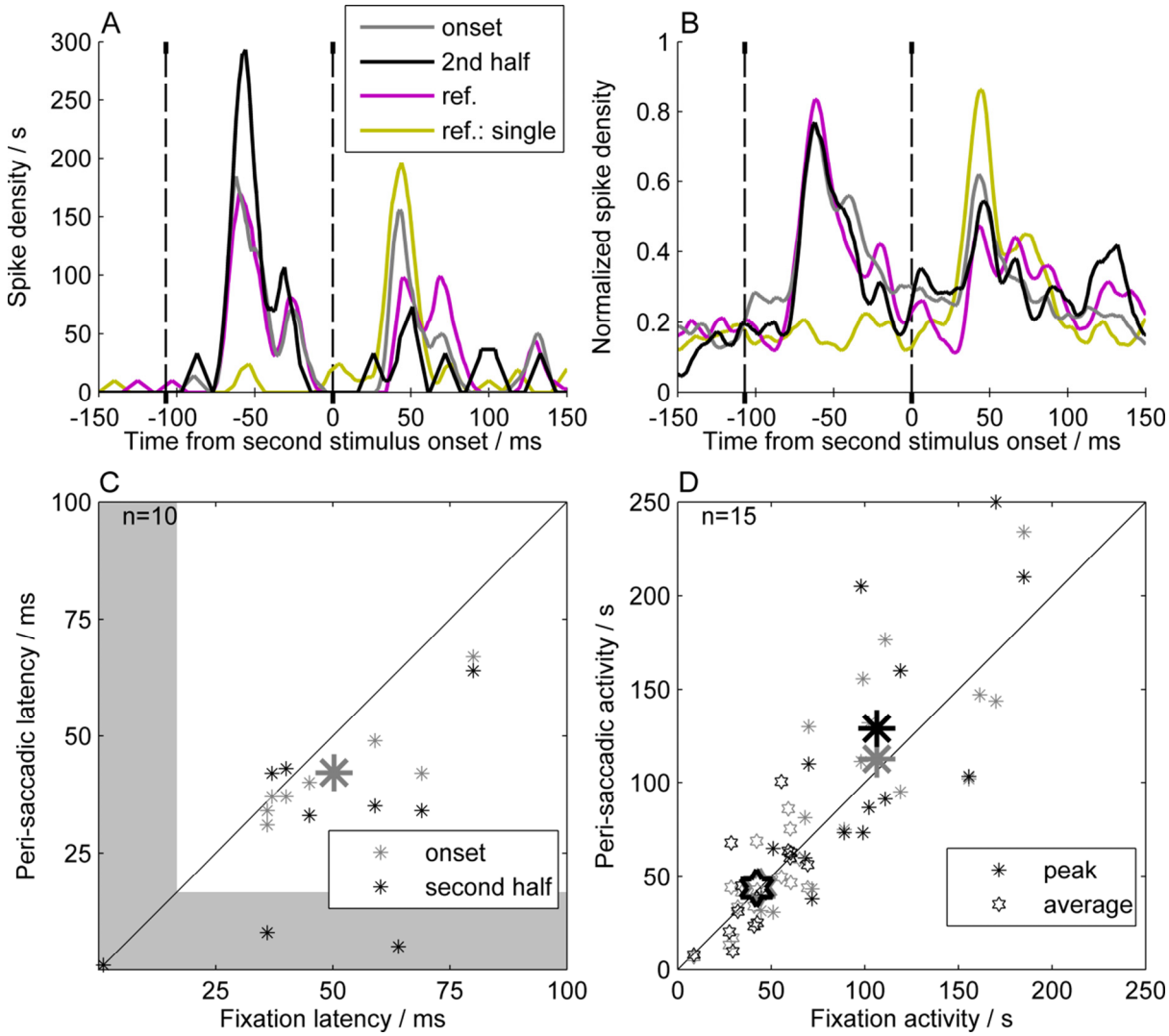

Figure 9. Peri-saccadic responses to double stimulus presentations (SOA 107ms, 64 cd·m^{-2} each). (A+B) The (normalized) spike density to pairs of stimuli shown around the onset of a saccade (gray) or during the second half of the saccade (black) as a function of time relative to the onset of the second stimulus. Data is shown for the exemplary cell (A) and the mean population response (B). The responses to the corresponding single (yellow) and double (magenta) stimulus presentation during fixation are shown as a comparison. (C) Single cell response latencies during to the second of a pair of stimuli shown during fixation against those during an otherwise identical peri-saccadic stimulation. As in Figure 7A, the data plotted within the gray area indicate that no latencies could be determined in the peri-saccadic condition for those cells. Data from one cell with a very large detected latency at coordinates (36, 123) during the second half is not shown. (D) Peak (⋆), average (✱) response period of single cells to the second of a pair of stimuli presented during the two peri-saccadic time intervals plotted against those observed during fixation. Large symbols indicate the means.

sponse between the two saccadic intervals revealed no significant differences between each other. There was a trend towards an increased latency compared to a single stimulus for both intervals (p<0.08 each; n=10). Compared to the latency to a second stimulus during fixation, the per-saccadic latencies were reduced (Figure 9C) and this reduction was significant for second stimuli shown around the onset of a saccade, but not during the second half (p<0.02 and p>0.50, respectively; n=10). For both saccadic intervals, the relative changes of the average and peak responses (Figure 9D) were not significant.

Discussion

Strong suppression of the response to the second stimulus

Single cell responses to a second stimulus, presented shortly after a first, were strongly reduced. Recovery of the signal was first observed for a stimulus onset asynchrony of 53ms but was still significantly reduced for a SOA of 107ms. The reduction of the peak response was accompanied by an increase in response latency for the second stimulus. For the exemplary cell, the peak response to the second stimulus with highest luminance was less than that to the single stimulus of lowest luminance. The latency however did not increase as much as found for that luminance. Also, the variation of the peak response to the second stimulus, compared to the first, preserved the luminance tuning, with the maximum response occurring for stimuli with a luminance of 27 $cd \cdot m^{-2}$. These results indicate that response changes to the second stimulus might be different to that of an otherwise identical stimulus but at a lower luminance. The mean population response to the second stimulus was however comparable to that of a stimulus with a luminance of 3 $cd \cdot m^{-2}$. Data from more cells and a wider range of stimulus luminance levels would be needed to determine whether the relation between response magnitude and latency is preserved for the

second of a pair of stimuli. A rapid suppression of a response to a second stimulus has also been reported in the frontal eye field and the superior colliculus (Mayo & Sommer, 2008). A change in latency however was not found. The increase in latency for the second stimulus is an important difference between the results in this study and those reported for these two brain areas.

For motion stimuli, an inhibition of responses to a second stimulus has been previously reported for area MT (Priebe, Churchland, & Lisberger, 2002) but not for transitions between optical flow stimuli in MST (Paolini, Distler, Bremmer, Lappe, & Hoffmann, 2000). In the experiment by Priebe et al. (2002), two brief motion stimuli (64ms duration) were shown with a variable interval in between. They found an exponential recovery of the response to the second motion with an average recovery time constant of 100ms after the offset of the motion. When the two motion phases followed another without a gap, an increase in response latency was reported if the first direction of motion was different to that of the second motion (Priebe & Lisberger, 2002). When both motion periods were in a similar direction, no changes in latency were found. Our results extend these findings by showing for the first time that a transient response to a non-moving short stimulus presentation is sufficient to cause these changes in response amplitude and latency in area MT.

These results might relate to an inhibition of transparent motion signals in MT that has been first demonstrated by Snowden, Treue, Erickson, and Andersen (1991). When two random dot patterns are simultaneously moving in the same depth plane but in opposite directions, the response in MT (but not in V1) to the motion in the preferred direction is reduced. This inhibition of one motion stimulus by another was best described by a gain control mechanism. A flashed stimulus has motion energy in all directions and this has previously been proposed to play a

role in the reduction of responses to flicker (Born & Bradley, 2005; Qian & Andersen, 1994). Assuming that this gain control mechanism is not instantaneous (as in Qian, Andersen, & Adelson, 1994) but slightly delayed (or with larger time constants than the excitatory input), the part of the motion energy in the cells' preferred motion direction might initially drive the cell at an unchanged gain. The components in other directions could cause a delayed reduction of the gain and thus inhibit the response to the second stimulus. Similarly, the increased latency found by Priebe & Lisberger (2002), when a first motion was directed opposite to a second, might reflect the temporal dynamics of the recovery from this gain mechanism.

Response to peri-saccadic stimulus not identical to low luminance stimulus

When stimuli were presented during a saccade, the exemplary cell showed a decreased response. Such a peri-saccadic reduction has previously been found by Bremmer et al. (2009). However for the mean population response shown in this study (Figure 8B), the response was only reduced when the stimulus was shown in the second half on the saccade. Although the decrease of the peak response was not consistent across the population of recorded cells, the average responses during the early and late response periods were significantly reduced, with the larger relative change occurring in the late response period. The lack of a significant response reduction for stimuli presented around the onset of a saccade also appears to be contradicting the known time course of saccadic suppression measured in psychophysical experiments. Data from more cells will be required to increase statistical power but also nonvisual saccade related neuronal responses need to be excluded as a potential source of an increased measured response. For stimuli shown during the second half of the saccade, the peri-saccadic decrease in response had a similar pattern to that reported by Bremmer et al. (2009). Even though the peak

response was reduced in the population, the average response around the time of the peak was also not significantly different from that during fixation. Only slightly after the peak (≈80ms after stimulus onset) did a larger reduction of activity occur. The results reported here for the second half of the saccade are thus in line with those of Bremmer and colleagues.

Latencies were not significantly altered during saccades compared to fixation. For investigations of peri-saccadic responses to motion stimuli in MT, reduced response latencies have even been reported during saccades (Price, Ibbotson, Ono, & Mustari, 2005; Thiele et al., 2002). In both studies the saccade induced retinal image motion of a random patterned stimulus was compared to a comparable motion of the stimulus during fixation. The difference to these results might lie in the differences of the stimuli. The motion stimuli had a speed profile and thus probably did not elicit the maximum response at the beginning of the eye-movement. In our case bright stimuli were presented, resulting in short but very strong responses. It might thus be that the latency to these stimuli was already close to the physiological limit, whereas the stimuli used by Price et al. and Thiele et al. apparently allowed for further reductions in latency along the way of the visual processing. The image that emerges from the results of this study is of a not or only slightly reduced and undelayed initial response, with an increased reduction in the later portion of the early and late response periods. Thus the responses to a peri-saccadic stimulus appear to be fundamentally different to a stimulus at low luminance presented during fixation.

Average response to single peri-saccadic stimuli compatible with psychophysical results

It is likely that a number of areas of the brain are involved in the detection of luminance thresholds and the number of areas contributing to the changes in perceptual sensitivity during saccades may be limited to a subset of these. An area that strongly contributes to the decrease of

perceptual contrast sensitivity during saccades should thus have modulations in the neuronal response compatible with a reduction of at least the sensitivity observed in comparable behavioral studies. Perceptual contrast sensitivity has been shown to decrease by a factor of up to 10 during saccades (Burr et al., 1994; Diamond et al., 2000). From this one may expect the neuronal response to drop down to a level in the approximate range of a 6 $cd \cdot m^{-2}$ stimulus. The peri-saccadic average response during the second half of the saccade was roughly reduced to a level of a 9 $cd \cdot m^{-2}$ stimulus. Given that the amount of saccadic suppression decreases for stimuli shown on a dark background (Burr et al., 1982; Chahine & Krekelberg, 2009) the reduction of the response to that of a stimulus with approximately one seventh of the luminance is well within the expected range. Our results for stimuli presented during the second half of the saccades are thus compatible with the idea, that the peri-saccadic reduction of MT activity reflects the reduction of perceptual contrast sensitivity caused by saccadic suppression. They show that even though response changes may be small, the impact on perception might be large. It has to be noted, that these results cannot exclude the possibility of saccadic suppression occurring at a previous stage of visual processing.

Peri-saccadic response change different for second and for single stimulus

When the second of two stimuli was shown during the saccade, peak responses were comparable and not statistically different to the response of a second stimulus shown during fixation. The mechanisms of reduction as a consequence of (a) a preceding stimulus and (b) a saccade thus appear to interact in a way that an already reduced response due to a preceding stimulus is not subject to further reductions due to a saccade. Furthermore, the peri-saccadic response latency to the second stimulus at the onset of the saccade did not increase as much as when pre-

sented during fixation. Both these changes are an interesting difference to the presentation of a peri-saccadic single stimulus where the opposite was found (response, but not latency, decreased significantly). Responses to peri-saccadic stimuli of different luminance should be obtained in further experiments. This would allow determining whether these discrepancies are caused due to the lower response amplitude alone, or by the mechanisms involved in the reduction of the response to a second stimulus.

The results shown in this experiment have an impact on the interpretation of results from previous experiments. The mapping of receptive fields using reverse correlation is a powerful method. Different stimuli are shown in random sequences and, under the assumption of linearity, the typical stimuli that cause a neuron to respond can be calculated (e.g. De Boer & Kuyper, 1968; Eckhorn, Krause, & Nelson, 1993; Hartmann et al., 2011). At least in the case where the changes in contrast are high and the stimuli are shown in rapid succession, the response of a stimulus is unlikely to be independent of a prior stimulus. This could potentially hinder the analysis of the full dynamics of the receptive fields of the measured cells. The mentioned experiment on peri-saccadic activity in the LGN (Reppas et al., 2002) was also carried out using rapidly changing stimuli and this might have influenced the changes of neuronal activity observed. We did not find additional reductions in responses due to a saccade when responses were already reduced. This opens the possibility that the method used in the prior experiments prevented saccade related reductions in activity to be detected.

Encoding of temporal information

For stimuli presented during a saccade, the response to latency relationship changed compared to fixation; with latencies being shorter than to be expected from the response magnitude. For

single stimulus presentations during the second half of the saccade, the latency remained unchanged, but the average response was reduced. For the second of a pair of stimuli shown at saccade onset, the average response did not decrease further and the latency was reduced compared to a second stimulus shown during fixation. If the mechanism responsible for the perception of the timing of stimuli had access to the dynamics of the response-latency relationship during fixation, it could correct for different latencies (due to different stimulus luminance levels and preceding stimuli). If the correction would not take the peri-saccadic changes in this relationship into account, it would then result in an overcompensation; as latencies are not increased as much as the average response would indicate. The occurrence of a peri-saccadic stimulus would thus be antedated. This could relate to psychophysical reports of the inversion of perceived temporal order and reduction of perceived interval duration (Morrone et al., 2005).

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General discussion and outlook

In this thesis I psychophysically investigated the spatio-temporal profile of saccadic suppression and saccade induced chronostasis in humans. In a neurophysiological approach, I measured responses in area MT of a macaque monkey to evaluate a possible involvement of MT in the emergence of these perceptual modulations during saccades.

Response suppression of a second stationary stimulus in area MT

As a part of the experiments reported in chapter 3, that aimed to determine the temporal dynamics of responses to sequences of stationary stimuli in MT, I was able to show for the first time, that responses of neurons in macaque area MT to a second of a pair of stationary stimuli were strongly suppressed. There was no significant response for the second stimulus, when the interval between the two stimuli was short, i.e. in the order of 50ms or less. But even at an onset asynchrony of 100ms the response was not fully recovered to the level found for a single stimulus. The time scale of more than 100ms for the neuronal response to return to its original state might relate to the integration of visual information over time for motion encoding. Two studies, one psychophysical (Burr, Ross, & Morrone, 1986) and one physiological (Mikami, Newsome, & Wurtz, 1986), found that visual motion information can be integrated over a time of about 100ms. When two stimuli are presented at the same location and their information is integrated, the response of a motion selective cell should be reduced, as the speed is zero. With the inhibition of other stimuli at the same position as a first, one could thus argue that motion sensitivity can arise if responses from locations at the same position and in the direction opposite to the cells preferred motion direction are inhibited. As such, the initial onset response and the follow-

ing inhibition might reflect fundamental components of MTs direction selectivity for visual motion. It has to be noted, however, that cells of FEF and the SC have similar characteristics with respect to the inhibition of a second stimulus (Mayo & Sommer, 2008) and it is unclear why FEF and SC would be subject to similar dynamics in the framework just described.

Saccadic suppression

In the first chapter of this thesis I was able to demonstrate that peri-saccadic contrast thresholds are reduced by a constant temporally aligned factor across the visual field when analyzed in a retinal frame of reference. When analyzed in screen coordinates, this was not the case and the time course of contrast sensitivity varied with stimulus position. It is thus likely, that the neuronal changes that contribute to saccadic suppression also occur in neurons encoding stimuli mainly in an eye-centered frame of reference. This would put limits to possible acting locations of saccadic suppression. It has recently been suggested that saccadic suppression may not result from response changes at an early stage of visual processing, but rather in higher visual areas (Bremmer et al., 2009). The authors found that the excitability of neurons in monkey areas VIP, MST and MT match the temporal profile of saccadic suppression. However a population of neurons in area VIP of the macaque brain have been shown to encode visual stimuli in a non-retinal frame of reference (Duhamel et al., 1997). Taken together, area VIP can thus be regarded an unlikely source of the reduced peri-saccadic reduction of contrast sensitivity. While there is currently a dispute about a possible spatiocentric encoding in the human area MT+ (Crespi et al., 2011; Gardner et al., 2008; d' Avossa et al., 2007), neurophysiological recordings in the macaque have not found evidence for this in the monkey brain (Hartmann et al., 2011; Ong & Bisley, 2011).

While Bremmer et al. (2009) showed a qualitative agreement of neuronal responses and psychophysical results, it remained unanswered whether the reduction of neural excitability to about 80 percent is sufficient for the reduction in perceptual contrast sensitivity to about 10 percent to that during fixation. To test this, I measured the responses of single cells in the macaque area MT to stimuli of different luminance during fixation and to the stimulus of maximum luminance during saccades (chapter three). At least for stimuli presented during the second half of the saccade, responses were reduced compared to fixation. The reduced response was comparable to a stimulus during fixation with one seventh of the luminance. This is slightly less than the factor of ten reported in psychophysical experiments. However, saccadic suppression is known to be inversely correlated with background luminance (Burr et al., 1982; Chahine & Krekelberg, 2009). In this experiment, a low background luminance was used and thus a smaller reduction of the perceived contrast sensitivity is expected. Additionally, other areas involved in the perception of luminance stimuli might be subject to even stronger perisaccadic response changes. As such my data are well in line with the view of a contribution of MT in the detection of luminance contrasts and the variation in detectability around the time of saccades. However the consequences of these results for the understanding of saccadic suppression face two limitations. First, even if MT responses are peri-saccadically reduced, it is unknown whether MT is actually involved in luminance detection tasks at all. To test this directly it might be worth to measure the contrast sensitivity while manipulating the activity in this area or other areas of interest. One method to do this would be to temporally deactivate the area by physically cooling it or by local injection of a GABA agonist (e.g. muscimol) and measure the monkeys contrast sensitivity in a psychophysical task using low spatial frequency luminance contrast stimuli which are known to be sup-

pressed during saccades (Burr et al., 1994). Second, assuming that MT is necessary for contrast detection tasks, it still cannot yet be said whether the changes arise in this area or are passed on from other (lower) visual areas. Studies using electrophysiological recordings in the LGN have found that peri-saccadic changes of activity were typically not limited to the magnocellular path and also occurred when the visual input of a saccade was simulated. Thus, at least at first sight they failed to show a pattern of saccadic suppression consistent with psychophysical results (Fischer et al., 1996; Noda, 1975; Reppas et al., 2002). However the stimuli used in the experiments of Fischer et al. (1996) were not optimized to selectively target saccadic suppression. In their experiment a patterned background was used. With this kind of background, contrast sensitivity can be reduced, even for simulated saccades (Diamond et al., 2000). In the experiment by Reppas et al. (2002) a full field stimulus was used that randomly changed between two extreme luminance levels in rapid succession. As shown in chapter three, for area MT (but also for the CS and FEF (Mayo & Sommer, 2008)) the responses to a stimulus can be strongly suppressed when another stimulus was presented shortly before. When the second stimulus was shown during a saccade, I found no further reduction of the response to occur and latencies to increase less than during fixation. Hence, if the responses to sequential stimuli are similar in the LGN to those in MT, the rapid stimulation used by Reppas and colleagues may have hindered a more pronounced reduction in neuronal activity. But even with this limitation, the reduction found just after the onset of a saccade was close to a value of about 80 percent compared to well before a saccade (Figure 4C of that article), for magno- and parvocellular neurons combined. The response of parvocellular neurons alone to modulations with cone isolating stimuli was only reduced to about 95%, again when considering only the early postsaccadic component. Even

though a postsaccadic enhancement of activity was found, these results might still be compatible with a large proportion of saccadic suppression being mediated at the level of the LGN. In this framework, the differences in saccadic suppression found by Bremmer et al. (2009) between different higher visual areas of the dorsal stream might result from input to these areas from other non-cortical routes (most notably from the SC via the pulvinar, as described by Berman & Wurtz, 2011) and from different receptive field properties of the neurons in the different areas as well as input from higher visual areas. Input from the CS could include both visual input as well as non-visual eye-movement related signals.

New insights about saccade induced chronostasis

The results of the experiment described in the first chapter of my dissertation also inspired a reexamination of properties of the peri-saccadic overestimation of time: chronostasis. I found that saccadic suppression occurs in an eye-centered frame of reference. As a consequence, it varies considerably between different stimulus locations in a head-centered frame of reference (chapter 1). My hypothesis that chronostasis might be linked to saccadic suppression and that, as a result, chronostasis could differ for different stimulus locations on the screen, was confirmed by the results described in chapter two. I found that, unlike previously assumed, chronostasis is not limited to the location of the saccade target. Instead it appears to occur throughout the visual field. The spatio-temporal profile found for chronostasis was however different than initially anticipated. The hypothesis (based on previous results by Georg & Lappe, 2007) was that chronostasis would be lowest midway of the saccade trajectory. Yet the opposite was found. This discrepancy could be explained when taking two other sources of degradation of visual perception into account. First, when stimuli are shown throughout a saccade they are

perceived as blurred (Campbell & Wurtz, 1978). Second, stimuli shown on a structured background can cause a reduction of contrast sensitivity even in the absence of saccades (and thus absence of saccadic suppression) when the image motion is fast (Diamond et al., 2000). A stimulus was always present at the location of the probe whose duration was to be judged. It is thus likely that the perception of the probe's onset was not only influenced by saccadic suppression, but also by these image motion induced effects. And as a result duration was even strongly overestimated at the location where saccadic suppression was expected to be lowest.

Chronostasis was previously believed to be triggered by an efferent signal, i.e. the efference copy of the saccadic motor command (Yarrow et al., 2001). In chapter 2 I also showed that chronostasis occurred even in the absence of saccades, when the image was moved by a mirror at saccadic speeds. With this result, it can be concluded that an eye-movement related signal is not necessary for chronostasis to occur. Instead chronostasis is induced by the retinal motion and appears to depend on the visual degradation of the perceived probe onset.

However, there is at least one remaining aspect, which requires further investigation. In the original report of chronostasis, the authors proposed that chronostasis occurs to preserve object constancy and that, as a result, it would only occur if a stimulus was present before the saccade at the location of the probe. Without this pre-saccadic stimulus there would be no object constancy to preserve and thus no chronostasis. They provided evidence for this idea, by showing that no chronostasis occurred when the probe was notably shifted during the saccade, so that it was at a different location than the perisaccadic stimulus. In chapter two I discussed that, much like the structured background used in the experiment of Diamond et al. (2000), the pre-saccadic stimulus might merely serve as a source for a reduced contrast sensitivity. This could be tested

by measuring perceived duration with or without a pre-saccadic stimulus as well as with or without a structured background. If chronostasis was to occur in the absence of a pre-saccadic stimulus when a structured background was used, it would demonstrate the role I proposed for this component in chronostasis.

Decoding of temporal information

It has been shown that chronostasis is caused by a predating of the probe's onset (Yarrow et al., 2006). As discussed in chapter two, this predating might reflect a more general mechanism of temporal perception. The response to the peri-saccadic probe onset is likely to be reduced to that during fixation. An assumption that is backed up by the peri-saccadic responses to transient stimuli measured in area MT in chapter three and by previous investigations (Bremmer et al., 2009; Ibbotson et al., 2008). The time that passes since the onset of the probe could be estimated by the size of the residual neuronal response at any given time after the onset. A very recent stimulus will have a larger residual response, than one that occurred earlier. A reduced response amplitude could thus be (mis-)interpreted as the residual response of a stimulus that occurred earlier; resulting in predating.

This concept of temporal perception could also explain why the temporal order can be systematically perceived as inverted when the second stimulus is either shown during a saccade (Morrone et al., 2005) or at a lower contrast during fixation (Bachmann et al., 2004). The responses in area MT to peri-saccadic stimuli and to the second of a pair of stimuli reported in chapter three were strongly reduced in both cases and as a result could also be predated. The time constants of the signal delay found in area MT are, however, too small to allow decoding of temporal information when the stimuli are separated by more than 100ms, as the activity is already

back to baseline at that time. The responses from MT might provide input to other areas with longer decay constants. As information has to be preserved for longer durations for tasks involving the memorization of objects, areas involved in short term memory also appear relevant in this context. The monkey area 7a is part of the dorsal stream and receives input from area MST, which receives input from area MT. This area had been proposed to play a role in spatial working memory (Constantinidis & Steinmetz, 1996). It might thus be worth to investigate the functional role of area 7a in this context.

An alternative explanation could be that the system is compensating for neuronal latencies when judging time. Since responses of MT neurons reported in chapter 3 to stimuli with low luminance were accompanied by larger latencies, the compensation could be dependent on the response size, with larger compensation for lower response sizes. Despite the reduced response, latencies did not increase in the peri-saccadic conditions. This could result in an overcompensation of latencies at a later stage of visual processing that is based on MT responses. If this hypothetical mechanism was to also cause chronostasis, it would require overcompensation by more than 140ms. Thus at least for chronostasis, overcompensation alone appears to be an unlikely source. Another mechanism was proposed to account for the perceived inversion of temporal order during saccades (Binda, Cicchini, Burr, & Morrone, 2009; but see also Diamond, 2002). The authors explain changes in temporal perception in a model as the consequence of the remapping of visual information found to occur in areas LIP (Duhamel et al., 1992) and other visual areas (e.g. Hall & Colby, 2011). They assume the visual response to linearly shift to the neurons that would encode for them after the saccade within a duration identical to the duration of the saccade. The signal causing this change, however, is assumed to start slightly before the onset of the

saccade. If a stimulus is shown shortly before the onset of a saccade, the initial part of the signal might still be encoded by the neuron that would encode for it during fixation. The shifting of information would then cause the signal to be cut off for that neuron and a part of the neuronal response to be distributed across other cells encoding for locations along the trajectory of the planned saccade. Therefore, the peak of the neuron with the strongest response would be shifted towards an earlier time. For stimuli presented after the shifting signal started, the effect would be opposite: the peak would be shifted towards a later point in time. While this model performed reasonably well for the situation examined, there are other situations where it might fail. The model critically depends on a change of the visual response as the information is shifted across the neuronal map. If however a large stimulus was to be used that is oriented along the direction of the saccade, the neuronal input could stay unchanged during the remapping phase (except for the edges of the stimulus). As a result the changes in temporal perception should not (or only in a much reduced form) occur. But this is exactly the kind of stimulus used by Morrone et al. (2005) to first demonstrate the saccadic perceived inversion of temporal order.

Saccadic suppression occurs in an eye-centered frame of reference with reductions in contrast sensitivity compatible to the reductions in single cell activity I measured in the macaque MT. While chronostasis is influenced by saccadic suppression and the visual changes induced by a saccade, it is not in itself dependent on eye-movement related signals.

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Erklärung

ich versichere gemäß §10(c) der Promotionsordnung, dass ich meine Dissertation

Spatio-temporal representations during eye movements and their neuronal correlates

selbstständig, ohne unerlaubte Hilfe angefertigt und mich dabei keiner anderen als der von mir ausdrücklich bezeichneten Quellen und Hilfen bedient habe. Ich habe alle vollständig oder sinngemäß übernommenen Zitate als solche gekennzeichnet sowie die Dissertation in der vorliegenden oder einer ähnlichen Form noch bei keiner anderen in- oder ausländischen Hochschule anlässlich eines Promotionsgesuchs oder zu anderen Prüfungszwecken eingereicht.

Marburg, 16.08.2012 _____

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