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Dynamic sensorimotor integration: implications for movement cancellation and visual stability

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# Table of Contents

1. General Introduction ................................................. 5  
   1.1. Visual Perception ........................................... 6  
   1.2. Saccadic eye movements ...................................... 10  
   1.3. Visual stability ................................................ 13  
   1.4. Predictive remapping ......................................... 17  
   1.5. Predictive remapping without a saccade? .................... 19  
   1.6. A proxy for cancellation? .................................... 22  
   1.7. What is stabilized? ............................................ 23  
   1.8. Computations for visual stability ............................. 26  
   1.9. Outline of this thesis .......................................... 30  

2. No Peri-saccadic Mislocalization with Abruptly Cancelled Saccades. 33  
   2.1. Introduction ................................................... 34  
   2.2. Materials and Methods ........................................ 35  
   2.3. Results .......................................................... 41  
   2.4. Discussion ........................................................ 47  
   2.5. Acknowledgements .............................................. 51  

3. Neuromuscular markers of movement cancellation in reach control 53  
   3.1. Introduction ................................................... 54  
   3.2. Materials and Methods ........................................ 56  
   3.3. Results .......................................................... 62  
   3.4. Discussion ........................................................ 78  
   3.5. Supplementary Methods ....................................... 84  

4. Causal inference for spatial constancy across saccades ........... 89  
   4.1. Introduction ................................................... 90  
   4.2. Materials and Methods ........................................ 92  
   4.3. Results .......................................................... 102  
   4.4. Discussion ........................................................ 109  

5. Summary & General Discussion .................................... 115  
   5.1. Summary ........................................................ 116  
   5.2. No peri-saccadic mislocalization with abruptly cancelled saccades ........ 116  
   5.3. Neuromuscular markers of movement cancellation in reach control .......... 117  
   5.4. Causal inference for spatial constancy across saccades .................. 118  
   5.5. Point of no return in motor control ........................... 118  
   5.6. Anticipation for something that never comes? .................. 120  
   5.7. Future Research ............................................... 121  
   5.8. The point of no return in transsaccadic integration .................. 122  
   5.9. Alternative (unconscious) decision strategies .................. 123  
   5.11. Conclusion .................................................... 126
1. General Introduction
1.1. Visual Perception

When we open our eyes we instantly see a coherent detailed colorful world with meaningful objects located in 3D space. Somehow our brain is able to rapidly interpret the light that is reflected or emitted from objects in our field of view, a process called visual perception. I emphasize ‘interpret’, because vision is fundamentally a cognitive ability (from Latin cognoscere, meaning to know or learn). We construct what we see, meaning that we may perceive things that are not really there (called illusions) or we may correctly infer things that are not, or just partly, visible.

![Figure 1.1. The eye. A) Horizontal cross section showing basic anatomy. The light sensitive layer (retina) is drawn as thick black line. Note that the image that falls on the retina is inverted due to the lens. B) Photoreceptor density within the retina at various eccentricities expressed in pie charts. Pie surface area represents photoreceptor count. Note that cones dominate at the fovea.](image)

Visual perception starts with our two eyes. The eye ball has a diameter of about 24 mm (in adults), and the retina at the back of the eye, a layer of photoreceptor cells, can be regarded as the sensor of a sophisticated ‘130-megapixel camera’. When light enters the pupil, the lens bends the light to create a sharp image on the retina (see Figure 1.1A). Within the retina, the photoreceptor cells transform the image into electrochemical signals. In humans, the retina contains two kinds of photoreceptors: rods and cones. Cones can discriminate color, but they are not very sensitive and do not respond to low luminance light. Rods are very sensitive to light changes, but cannot discriminate color. Therefore, when our environment is rather dark (e.g. moonlight), only the rods are active, producing an image of only shades of grey. When an object emits or reflects enough light, color discrimination is
possible because each cone is sensitive to either red, green or blue light. The relative activity of these three types together allows us to see all colors of the rainbow. Cones and rods are not spread out homogeneously on the retina (Purves, et al. 2001). The central part of our vision, the fovea, is populated almost exclusively with cones (see Figure 1.1B). The ratio of red/green/blue cones is about 10:5:1, respectively (De Valois & De Valois, 1993). In the periphery, color discrimination is low, but the abundance of rods enables us to have a wide field of view under low luminance conditions.

Vision has evolved over >500 million years to aid in the survival and successful reproduction of organisms. Because different organisms face different challenges, we find a large variety of eyes and how they are positioned across species. For example, prey animals often have lateral placement of the eyes producing a wide field of view, whereas predators have frontal placement of the eyes producing better depth perception. Furthermore, some species cannot discriminate color, while others can discriminate colors beyond our visible spectrum (e.g. insects and birds). There are also large differences in spatial acuity, which is for a large part determined by the number of photoreceptor cells in the retina. For example, the Copilia (a tiny water creature) has only a single photoreceptor per eye (Gregory et al., 1964) while we humans have around 150000 photoreceptors per mm$^2$ in our central vision.

At the fovea, the dense packing of cone photoreceptors creates a ‘hot spot’ in terms of visual acuity. If we want to visually inspect an object, we have to look straight at it to see the detail. Although the fovea is only a few degrees in visual angle, it allows us to see a single hair at several meters distance. Why is the fovea not larger? It is said that if the entire retina were of the quality of the fovea, the optic nerve (2-3mm diameter in humans) would be 200 times as thick (Carpenter, 1977). This would not only be problematically large in structural terms, it would also require much more effort to interpret the incredibly detailed image. Thus, researchers consider the retina with a central fovea a good tradeoff.

The electrochemical signals from the retina are transferred to the brain via the optic nerve. The spot where the retina and optic nerve connect is called the optic disk. Because of the connection the optic disk does not contain any photoreceptors, therefore it is also known as the blind spot (Figure 1.1A). The visual signals travel via the optic nerve to the optic chiasm (see Figure 1.2). At the optic chiasm, the optic nerve fibers on the nasal side of each retina cross
over to the opposite side of the brain. This means that the image of the left visual hemi-field is passed to the right thalamus (see below), and from there to the right primary visual cortex, and vice versa. Although each hemisphere receives information from the opposite visual hemi-field, connections between the hemispheres (e.g. via the corpus callosum) allows to combine information from both hemi-fields into a single coherent representation (e.g. Houzel and Milleret, 1999; Berlucchi and Rizzolatti, 1968). However, for simplicity in the following text only connections within a single hemisphere are considered.

![Figure 1.2. Optic chiasm. The left visual hemi-field is highlighted with orange; the right hemifield is highlighted with green. Within the optic chiasm, visual information on the nasal side of each retina cross over to the opposite side of the brain.](image)

The visual stream reaches the thalamus at the lateral geniculate nucleus (LGN), a central relay station hidden deep in the brain (see Figure 1.3A). From there the information is passed on to the primary visual cortex (V1), located at the backside of the brain. It is believed that in V1, basic visual features are extracted like form, depth, motion, and color (Livingstone and Hubel, 1987, 1988). The primary visual cortex is connected in turn to various other areas throughout the brain.

A very influential hypothesis is that the information leaving V1 travels further via two pathways or streams (Ungerleider and Mishkin, 1982; Mishkin, Ungerleider, and Macko, 1983; Goodale, 1995; Milner & Goodale, 1995): the ventral stream to the temporal lobe, and the dorsal stream to the parietal lobe (Figure 1.3A). These are also called the “what” and the “where” (or “how”) pathway. This is because the ventral stream seems to be involved in object identification (“what”; form, color) and may contain the neural substrate of conscious visual perception, whereas the dorsal stream appears to be more involved in object locations, and the execution of voluntary visually guided
actions (“where/how”; motion, location). Although this is a simplified view, as the dorsal and ventral streams are heavily interconnected and thus not working independently (e.g. Milner and Goodale, 2008; McIntosh and Schenk, 2009, Medendorp et al. 2016, Smeets and Brenner, 2006; De Brouwer et al., 2015), it roughly illustrates how the brain is organized. In this thesis we will be mainly focusing on the “where” pathway.

Figure 1.3. Visual perception and saccade generation. A) Visual information flows via the optic chiasm to the LGN and the superior colliculus. From the LGN, the information goes to V1. Illustrated is the dual pathway hypothesis: dorsal pathway to the parietal lobe (purple) and the ventral pathway to the temporal lobe (red). B) The extraocular muscles are controlled by brainstem circuitry, containing omnipause neurons, that receives the eye movement command from the SC. The SC in turn receives information from various brain areas, including FEF and LIP.

Neurons in visual brain areas respond to only a small part of the image that falls on the retina. In other words, neurons have a receptive field (RF). The concept of a RF is an important one (see Figure 1.4). We generally find that neurons closer to the raw visual input (LGN, visual cortex) have a small RF: when a dot of light is flashed at one specific location in space, stimulating a specific part of the retina, the presence of the stimulus will alter the firing activity of the neuron. In ‘higher’ parts of the cortex we find that RFs increase in size, meaning that such a ‘higher’ neuron receives signals from multiple ‘lower’ neurons. Another property observed in many visual areas is that nearby
regions on the retina project to nearby regions in the cortex. In other words, these areas have a retinotopic organization. This means that if you would record activity from a grid of adjacent neurons from, for example, the primary visual cortex, you would be able to directly infer what is viewed. But it would be a distorted image, because proportionally the information from the fovea receives much greater representation in the cortex than the periphery does (e.g. Cowey and Rolls, 1974), which we call cortical magnification.

**Figure 1.4.** Receptive field (RF). A) While a monkey is presented with visual stimuli, activity of a neuron is recorded using a thin electrode. Line of gaze is shown as a dashed line. When lights are flashed throughout the display, the recorded neuron becomes activated as illustrated by the small horizontal lines, 'spikes,' on the monitor. B) Closer examination reveals that the neuron is activated only when one specific location is stimulated relative to the current line of gaze (dashed line). The RF of this neuron is located up-left relative to gaze.

### 1.2. Saccadic eye movements

Evolution has brought us three pairs of small but strong muscles per eye, the extraocular muscles (see Figure 1.5), which are able to rotate the eyeball very quickly, exceeding 700 degrees per second for large movements (Clark and Stark, 1975; Carpenter, 1977). This not only allows bringing the high resolution fovea quickly directed to different parts of the environment, it also allows fixation of a moving object (by means of smooth pursuit eye movements) or compensation of head movements relative to gaze. In this thesis we will focus on the first function: eye movements that are intended to change the optical content of the retinal image by shifting gaze to another part of the environment. These movements are typically very rapid, hence
their name: saccadic eye movements (from French word ‘saquer’, meaning to pull violently). While you read this text your eyes jump in a very coordinated fashion from one part of the sentence to the next. The duration of each (small to medium-sized) saccade is roughly a function of the amplitude (in visual degrees): duration = 2.2 * amplitude + 21 ms (Carpenter, 1977).

The extraocular muscles are controlled via three cranial nerves (per eye) leaving the brainstem (see Figure 1.3B). Within the brainstem a complex saccade generation circuitry is implemented, which is heavily interconnected with the cerebellum, which is thought to adaptively fine-tune the premotor command (Kheradmand and Zee, 2011). Within the brainstem circuitry, a group of neurons called the omnidirectional pause neurons are found (or omnipause neurons; OPN) that inhibit all saccade-generation regions when the eye is stationary. When the OPN are inhibited, their inhibitory influence on the saccade-generation regions is lifted and the so-called burst neurons instantly start to create the brief bursts of activity needed to innervate the extraocular muscles (Fuchs et al. 1985). The OPN can therefore be regarded as a gate: when the saccade parameters (direction, amplitude) are set, only a single trigger signal is required to initiate the rapid execution of the intended movement.

The brainstem receives the movement command mostly from the superior colliculus (SC; see Figure 1.3B and Figure 1.6). In the SC the desired change in eye position and the trigger signal are found (see Sparks, 2002;
Hanes and Wurtz, 2001). Interestingly, neurons that generate saccades are arranged topographically in the SC: Electrical stimulation of neurons located more to the front (rostral) produce small saccades whereas neurons located more at the back (caudal) produce large saccades (Robinson, 1972; Schiller and Stryker 1972). Furthermore, the direction of the saccade is coded from medial to lateral (i.e. from center to side). The SC is regarded as one of the primary centers of reflexive stimulus-driven saccades (Baluch and Itti 2011; Guillery 1995; Harting et al. 1980; Shipp 2004). It receives direct input from the retina via the thalamus and the visual cortex (Fries, 1984) in order to react quickly to, for example, an unexpected flash of light that draws your attention. To decide where your eyes will go, the SC contains a so-called priority map. This map is retinotopically organized and it represents multiple target locations together with a salience rating (Krauzlis, Liston, and Carello, 2004; Wolf et al., 2015).

To perform voluntarily driven saccades, the SC receives movement commands typically from the frontal eye fields (FEF, see Figure 1.3B). Also neurons in FEF have so-called movement fields that specify where to go (e.g. Hanes & Schall 1996). Likewise, the FEF are thought to be involved in planning eye movements using a priority map. For example, FEF increase activity after a target was flashed (Olivier, Dorris, and Munoz, 1999) thereby holding the location in memory, and only until the neural activity reaches a certain threshold a saccade is executed to that location (Hanes and Schall, 1996). The FEF are also suggested to have a general role in spatial attention (Thompson, Biscoe, and Sato, 2010). For example, electrical stimulation of the FEF (in monkeys) at a low intensity causes attention to shift to corresponding parts of the visual image without evoking an eye movement to them (Moore and

![Figure 1.6. Saccade circuitry. The extraocular muscles are stimulated by the motor neurons, which in turn are stimulated by the burst neurons. Together with the OPN, this forms the (simplified) brainstem saccade circuitry. The saccade parameters are provided by the SC (aim). The OPN normally inhibits the burst neurons. This inhibition is lifted when the OPN themselves are inhibited by the SC (trigger). The burst neurons will then stimulate the motor neurons and keep the OPN inhibited (latch) while the saccade is being executed.](figure16.jpg)
The FEF are complemented with the supplementary eye fields (SEF), a structure that is also involved in visually guided eye movements (e.g. Purcell, Weigand, and Schall, 2012; Stuphorn, 2015), but is hypothesized to be specialized in performing spatial transformations for saccade sequences (Olson, Gettner, 1995; Chen, Wise, 1995) and executive control (Stuphorn and Schall, 2006).

A further key region involved in saccade generation is the lateral intraparietal area (LIP, see Figure 1.3B), located in the dorsal visual stream. Like FEF, area LIP is a visuomotor region containing a priority map to direct both attention and saccades (Foley et al. 2014; Ipata et al., 2009; Bisley and Goldberg, 2003; Gnadt and Andersen, 1988; Andersen and Buneo, 2002), and there is still debate on whether its activity merely reflects visual attention versus motor intention (e.g. Steenrod, Phillips, and Goldberg, 2013). Interestingly, neurons in LIP and FEF appear to have three-dimensional RFs (Ferraina, Paré, and Wurtz, 2000; Fukushima et al. 2002; Genovesio and Ferraina, 2004; Gnadt and Mays, 1995), thus are also sensitive to the depth of targets in the world. The connection between LIP and FEF (see Figure 1.3B) is thought to be mainly for visuomotor processing (Pouget et al., 2009), whereas the connection from LIP to the SC has been involved in fast reflex-like saccades (the so-called express saccades).

1.3. Visual stability

In the example depicted in Figure 1.3, gaze is initially fixated at the girl on a bike. Then a saccade was made to the building. As is illustrated Figure 1.7, during a saccade the image is blurred due to the fast rotation of the eye, and after the saccade all objects have changed their retinal positions (the world has shifted to the left in the example). When we are awake, we make about 2-3 saccades per second (Yarbus, 1967), which produces a highly unstable retinal image over time. In contrast, our subjective experience tells us that normally the visual world appears as very stable. We can easily tell whether movement on the retina is the result from real motion in the world or from our own actions (except when e.g. when we have drunk too much). Thus, somehow the brain is able to keep track of object locations, or create visual stability, by taking our own movements into account.

How does our brain achieve visual stability, or in other words, how can
it tell whether we or the world is moving? This is an ancient riddle (Melcher, 2011) that has still not been resolved today. But, there have been interesting developments in the past few decades. What follows below is a brief overview, ending with the first research question of my thesis.

How does the brain deal with the motion blur during a saccade? It appears that the retinal motion during each saccade is simply suppressed. Indeed, retinal stimulation during a saccade often escapes awareness (e.g. Bremmer, et al., 2009). This can also be demonstrated by simply looking at your own eyes in a mirror: you are not able to perceive your own eyes making saccades, hence during a saccade you are virtually blind. This phenomenon is called saccadic suppression (Volkman, Schick and Riggs, 1978). Note that particularly motion signals seem to be suppressed (e.g. Ross et al., 2001), thus a brief flash of light during a saccade may still be visible.

How does the brain deal with the change in object locations on the retina? One proposal is that the visual system factors out the image shift that a saccade has produced from only image-based (i.e. retinal) information (e.g. Longuet-Higgins and Prazdny, 1980). Because typically parts of the pre-saccadic and post-saccadic image overlap, the images can be ‘glued’ together creating a panoramic image in a similar way as photo editing software is able to create a panoramic photo from several shots. According to Gibson (1966), the world is stable ecologically, and motion of the entire image must be due to a saccade rather than motion in the world. In other words, we may use the structure of the image (everything moves versus a part moves) to infer if the world is stable and we also may have a strong assumption of stability to begin with. Instead of gluing images together, it has also been hypothesized that after each saccade object locations are calculated anew, thereby eliminating the need of some

Figure 1.7. Visual stability. Saccades create a motion blur during the eye movement and change the retinal location of objects, but in our subjective experience the visual world remains perfectly stable. How is this possible?
form of transsaccadic memory for locations (Bridgeman et al., 1994). Also the philosopher Daniel Dennett argues against a sophisticated transsaccadic memory (Dennett, 1991). He argues that the world itself can serve as a visual memory: we may believe that we have a rich and complete image of the world in our head, but this is a mere illusion that follows from the fact that when we want to see detail, we almost instantly see the detail by means of a quick saccade to the object of interest (see Blackmore et al., 1995; O’Regan, 1992).

Another influential account of visual stability is the saccade target theory (Currie et al., 2000) or the reference object theory (Deubel, Schneider, and Bridgeman, 2002) which does expand on a form of transsaccadic memory. It is hypothesized that before a saccade is executed some features of the image, in the saccade target area in particular, are stored in memory. Once the saccade is completed, these features are searched at or near the fovea where the saccade target object should be located if it had remained stationary. If the features are found, visual stability is assumed. If not, then there was movement in the world and visual stability is violated. This transsaccadic memory could be regarded as a part of short-term visual memory (Irwin, 1991).

In contrast to a purely image-based or retinal account of visual stability, there has been a growing interest in extra-retinal accounts. These theories propose that the brain is able to differentiate sensory information arising from its own actions from those that arise from the environment by actively taking the movement command into account. For example, you are unable to tickle yourself because the sensation is predicted together with the tickle movement, effectively cancelling out the ticklish feeling. In the light of visual stability, the term extra-retinal means that also some other signal than the retinal signal is used, namely a copy of the motor (i.e. efference) command, which has been referred to as the efference copy.

Already in 1876, von Helmholtz proposed that the brain may achieve visual stability by using an efference copy or what he called the “Willensanstrengung”, translated “effort of will” (von Helmholtz, 1925). He observed that when you gently pressed against your eyeball, thereby creating eye movements without extraocular muscle activity, you see the world move (see Figure 1.8). He reasoned that with passive movement of the eyeball, no efference copy is made to compensate for the motion on the retina. It should be noted that this experiment does not prove actual compensation per se. It could also be that the “effort of will” only suppresses the retinal motion during
active movement and that visual stability is achieved purely retinally. About 75 years later, von Holst and Mittelstaedt (1950) and Sperry (1950) performed experiments that more directly made the case that the efference copy is used to compensate for retinal motion. Von Holst and Mittelstaedt rotated the head of a fly 180°, so that the head was upside down (see Figure 1.8). This inverted not only up/down, but also left/right visual information for the fly. Thus, when the fly rotates to the left, the retinal image appears to move to the left as well, instead of to the right which would be normally the case. In other words, any expected retinal motion is now in the opposite direction. The researchers found that the fly started to move chaotically in circles.

Sperry (1950) performed a similar experiment with fish (see Figure 1.8). He rotated one eye 180° and blinded the other, so to the fish up/down were reversed as well as front/back. Like the fly, the fish started to move in circles. Clearly, voluntary movement was accompanied with an expectation of how it should affect the visual world. Some researchers took this one step further, as they paralyzed the extraocular muscles of human participants using curare injections (Kornmüller, 1931; Stevens et al., 1976). Indeed, participants reported that when they attempted to make a saccade, the visual world appeared to jump into the direction of the planned eye movement. This supports the idea that the contribution of the saccade itself is subtracted from the retinal flow, thereby creating visual stability.

Figure 1.8. The efference copy. The world seems to move when pushing the eyeball, suggesting that normally the eye movement command is used to create visual stability. The existence of such an efference copy was demonstrated in experiments in which the head of a fly and the eye of a fish was rotated, thereby inducing a mismatch between the expected consequence of movement and the actual retinal input.
1.4. Predictive remapping

In 1988, Gnadt and Andersen reported about an interesting property of some LIP neurons. They recorded from a LIP neuron in a monkey and determined the neuron’s receptive field. Then, the monkey performed a saccade to a remembered target location. Interestingly, although the target never physically appeared in the neuron’s RF, when a saccade brought the remembered location in the RF, the neuron became active as well. Duhamel, Colby and Goldberg (1992) explored this further and their main finding, predictive remapping, will be explained using Figure 1.9. They also recorded from LIP in a monkey and determined the RF of a target neuron. Let’s assume that the RF is positioned as indicated in Figure 1.4. That is, the neuron responds to visual signals in an area located to the north-west of fixation. In the example, when the girl is fixated, the church tower is in the RF (Figure 1.9A). However, the upcoming saccade will bring a different part of the image in the RF. In the example, when the building to the right is fixated, a cloud in the sky will be in the RF. While recording from the same neuron, Duhamel et al. presented a stimulus at one of two locations: the location that is occupied by the RF before the saccade, or the location where the RF will be positioned after the saccade (future RF). They found that normally the neuron responded only to targets within the RF, as expected, but when a saccade was planned, shortly before its execution the neuron also started to respond to targets appearing at the future RF. Then, after the saccade the neuron’s response went back to normal: the RF is now at the new location and the neuron only responds to targets there. This phenomenon, shifting RFs, has been interpreted as showing predictive remapping: representations of object locations are transferred parallel to the upcoming saccade to neurons that will be stimulated by these locations after the saccade, in order to anticipate the upcoming retinal shift.

Also in other brain areas, neurons with shifting RFs have been found, including the FEF (Umeno and Goldberg, 1997), SC (Walker et al., 1995), and the visual cortex V2, V3 (Moore et al., 1998; Nakamura and Colby, 2002). It has also been suggested that the RF shifts are not always parallel to the saccade but can also move towards the saccade target instead (Zirnsak and Moore, 2014; Neupane, Guitton, and Pack, 2016). Also in studies involving human participants comparable observations have been made using brain imaging techniques (e.g. Sereno, Pitzalis, and Martinez, 2001; Medendorp et al., 2003;
Heiser and Colby, 2006; Chang and Ro, 2007; Merriam, Genovese, and Colby, 2007). How these brain areas achieve such spatial remapping is currently unknown.

Some researchers have suggested that the efference copy, needed to guide the shifting RFs, originates from the SC because there the direction and amplitude for both stimulus-driven and voluntary saccades are found that drive the brainstem circuitry. Indeed, a pathway has been identified from the SC via the thalamus to the FEF that contains an efference copy of the saccade that may cause the RFs to shift (see Sommer and Wurtz, 2008; Wurtz, Joiner, and Berman, 2011 for a review). Another pathway may be from the SC via the pulvinar to visual motion area MT (Berman and Wurtz, 2011). However, these observations may only be a part of the story and more pathways will likely be

Figure 1.9. Predictive remapping. A) A planned saccade is about to bring a different part of the world within the neuron’s RF, which is termed the Future RF. B) Top row: the recorded neuron’s RF is similar to Figure 1.4. When a saccade is planned (red arrow), the neuron will still respond to the visual stimulus. However, the execution of the saccade brings the stimulus outside of the RF. Middle row: the neuron will not respond to any other stimulus locations. Bottom row: The future RF is an exception: when a saccade is planned, the neuron starts to respond also to the location where the RF will be positioned after the saccade. This is interpreted as evidencing predictive remapping. After the saccade, the neuron returns to the state where it responds only to its single designated RF.

Some researchers have suggested that the efference copy, needed to guide the shifting RFs, originates from the SC because there the direction and amplitude for both stimulus-driven and voluntary saccades are found that drive the brainstem circuitry. Indeed, a pathway has been identified from the SC via the thalamus to the FEF that contains an efference copy of the saccade that may cause the RFs to shift (see Sommer and Wurtz, 2008; Wurtz, Joiner, and Berman, 2011 for a review). Another pathway may be from the SC via the pulvinar to visual motion area MT (Berman and Wurtz, 2011). However, these observations may only be a part of the story and more pathways will likely be
discovered that deal with implementing visual stability.

When exactly does the spatial update take place? The predictive remapping appears to start already 50-100 ms before saccade onset as indicated by the shifting receptive fields (e.g. Duhamel et al., 1992). It may be possible that during this period you change your mind and decide to look elsewhere, or you cancel the planned saccade completely. What would happen then? To maintain visual stability, the update should be installed only when the execution of a planned saccade cannot be aborted anymore (Sommer and Wurtz, 2008).

1.5. Predictive remapping without a saccade?

Intuitively, when a saccade is considered it would seem logical that a new visual prediction is made only after committing to the execution of the saccade. The trigger signal in the SC to the brain stem is sent out approximately 20 ms before the extraocular muscles start to contract (Horwitz and Newsome, 1999), which would be actually too late for it to be causing the receptive field shifts which start around 50-100ms before saccade onset. Some visual neurons that modulate their activity as a function of gaze direction show anticipatory activation even 150 ms before saccade onset (e.g. Morris et al., 2012), which is as early as ~50 ms after the appearance of the saccade target. Does this mean that the remapping starts already during the saccade planning phase, where cancellation is still possible? To investigate this in human subjects we combined two classic cognitive psychology paradigms in the first study of this thesis: peri-saccadic flash localization and countermanding, which will be briefly described below.

It is known that people make systematic localization errors of stimuli presented near the time of a saccade. One of such situations is when you ask someone to indicate the location of a bar of light that was flashed around the time of a saccade (see Figure 1.10A). When the flash occurs just before saccade onset, the flash is systematically mislocalized. This is found in complete darkness or under normal light situations, but with different systematic pattern outcomes (e.g Bischof and Kramer, 1968; Ross et al., 1997). Here, I will only consider the peri-saccadic localization errors found for saccades in a normal illuminated room. As depicted in Figure 1.10, when a flash (orange bar) is presented at saccade onset between the current fixation point,
left circle, and the saccade end point, right circle, it is incorrectly perceived as being very close to the saccade end point (orange dots in Figure 1.10B). Several researchers have linked this phenomenon to shifting receptive fields, or to updating using an eye position signal more generally (Ross et al., 1997; Burr and Morrone, 2010; Hamker et al., 2008; Morris et al, 2012). Such so-called peri-saccadic localization errors (peri- meaning around the time of) can begin ~100 ms before saccade onset, consistent with shifting RF timings, and the error peaks when the flash is presented around saccade onset. Thus, peri-saccadic flash mislocalization might serve as a tool to determine the presence of anticipatory/predictive remapping. Do we find localization errors when a saccade is planned but abruptly canceled just before its execution?

**Figure 1.10.** Peri-saccadic mislocalization. A) A saccade is made from the left circle to the right circle. Around the time of the saccade, a vertical bar was flashed in-between these targets (orange). B) Position (horizontal axis) is plotted against time (top to bottom). Eye position (green line) shows a horizontal saccade. When the bar was flashed around saccade onset, the perceived location of the bar (orange dots) becomes strongly biased towards the saccade end point.

The countermanding paradigm can be used to bring someone in a state where he or she abruptly cancels a planned saccade (Logan and Cowan, 1984). With a countermanding task, a movement to a target is required on the majority of trials. In a subset of trials, however, a stop signal is required on the majority of trials. In a subset of trials, however, a stop signal is presented at some time after target presentation. When a stop signal is presented, no movement is allowed anymore. The time interval between target and stop signal onset, the stop signal delay, has a large influence on the outcome: when the target is
rapidly followed by a stop signal it is easy to inhibit the movement, but when the stop signal delay is long the movement planning has progressed too far and the movement cannot be inhibited anymore. The saccadic countermanding task used in Chapter 2 is illustrated in Figure 1.11. As a stop signal the fixation target is colored red. The outcome is typically modeled as a race between a GO process, initiated by the target, and a STOP process, initiated by the stop signal (Logan and Cowan, 1984; Logan, 1994). In the example of the race in Figure 1.11, the stop signal came on too late to inhibit the saccade. The smaller the stop signal delay, the more likely that the saccade is successfully inhibited/canceled, because the STOP process is initiated relatively earlier thus more likely to win (see Figure 1.11C). We can assume that movement planning is more progressed on trials with a long stop signal delay, hence for the current research question these trials are most interesting.

In our first experiment (Chapter 2) we brought participants in a ‘saccade generation mode’ by presenting a sequence of saccade targets with variable length. Participants effectively followed a jumping dot across the screen. During the presentation of the final saccade target, a vertical bar was flashed (random timing) at one of two locations (see Figure 1.10). Sometimes also a stop signal appeared (after the stop signal delay). After each sequence, the participant indicated the perceived horizontal location of the flash, irrespective of being

Figure 1.11. Countermanding saccades. A) A rightward saccade is planned and ultimately executed, despite the occurrence of a visual stop signal (red) after which movements should be inhibited. B) The GO process that leads to movement execution and the STOP process that leads to movement cancelation can be modeled as a race. The first process that crosses a threshold (dashed line) wins. C) The earlier the stop signal, the easier to inhibit the movement. Vice versa: The later the stop signal, the more likely the GO process wins the race. Countermanding is used in Chapter 2.
successful on the stopping task. Now the question is, do we see localization errors with successfully canceled saccades, particularly when movement planning is progressed almost up to the point of no return? We found that the answer to this question to be no. This suggests that predictive remapping is contingent on the execution of a saccade, i.e. mere preparation of a saccade is not enough.

1.6. A proxy for cancellation?

In the peri-saccadic mislocalization study described above, mislocalization was largest when the flash appeared at the moment of saccade onset. A problem with canceled saccades is that there is no saccade onset (per definition) to which the flash time can be aligned. We resolved this issue (Chapter 2) by inferring when the saccade would have happened on the basis of previous performance. Although this worked out well, the predicted saccade reaction times were not very precise. Would it be possible to get a within-trial measure of when exactly a planned movement is initiated and canceled, without the manifestation of that movement? This is the objective of the study described in Chapter 3.

Saccadic eye movements are very fast and typically once a saccade has started it will proceed until completion. Thus, determining when a canceled saccade would have happened, or when the cancelation did occur exactly, cannot be done without neural recordings. Recent findings on eye-head orienting movements, however, showed that in reaction to a stop signal sometimes the head slightly moved toward the target while the eyes kept fixation (Goonetilleke, Doherty, and Corneil, 2010). Interestingly, the muscles that stopped the neck from moving any further, the antagonist muscles, were recruited at a time consistent with the estimated time to process the stop signal (Goonetilleke, Doherty, and Corneil, 2010; Goonetilleke, Wong, and Corneil, 2012). In other words, it appears that the finish time of the STOP process can be determined by recording the antagonist muscle using electromyography (EMG) following a stop signal. Likewise, the finish time of the GO process can be determined by recording the agonist muscle following target onset. These measures could be obtained because the head is endowed with inertia, meaning that movement generation is slower compared to saccades and stopping the movement requires active braking.
In Chapter 3, we determined if the finish times of the GO and STOP processes could also be determined with arm movements. To this end, we ran a countermanding experiment involving whole-arm reaching movements while recording intramuscular activity from upper-limb muscles (see Figure 1.12). We found that indeed the moment of movement onset and movement inhibition could be determined, even when the arm did not appear to move at all. Having established this, future experiments could be devised that exploit this neuromuscular marker to understand perception-action relationships in health and disease.

Figure 1.12. Countermanding reaching movements. A) Experimental setup. Thin-wire electrodes are placed inside upper-limb muscles contributing to movement generation and braking. Hand position feedback and targets are viewed via an up-faced mirror. B) Recorded hand position and muscle activation during a trial. Although the stop signal appeared relatively late (red dashed line), the movement was stopped mid-flight due to antagonist muscle recruitment (stop reaction). The stop signal and stop reaction are related to the start and finish time of the STOP process, respectively, as studied in Chapter 3 of this thesis.

1.7. What is stabilized?

Now we have looked into the question of when the ‘image stabilization’ occurs, we will now turn to the question of what is stabilized exactly. Figure 1.7 suggests that the whole retinal image from each fixation is mapped and stored into a large photographic memory, but this is, however, very unlikely. As you probably know from experience, looking and seeing are two different things. The first relates to the retinal image, the second to what gets extracted from it. Clearly, not all aspects, or parts, of the image are attended equally. But what is attention, and how does it relate to visual stability?

Because there is typically far more information in the retinal image than can be perceived at once, the visual system selectively samples only the most important information given our current goals, needs, and desires. This
selection process is called visual attention. Unattended objects or aspects are not fully processed and therefore escape awareness (Mack and Rock, 1998; Rock and Mack, 1992; Mack, 2003). Saccades are an important manifestation of this selection process, as they bring relevant parts of the scene on the high-resolution fovea. Therefore, this mechanism has also been referred to as overt attention. However, also when the eyes are stationary we selectively sample information, using covert attention. Covert attention allows you to attended to (or notice) various objects in the visual periphery without making a saccade to them. As noted before, both overt and covert attention seem to share neural maps in SC, FEF, parietal cortex (e.g. Desimore, Wessinger, Thomas, and Schneider, 1990) and are possibly controlled by closely related mechanisms (Rizzolatti, Riggio, Dascola, and Umilta, 1987).

![Figure 1.13. Remapped versus new retinal image. A) In this illustration, gaze is initially directed to the house. B) Then, a saccade is made to the car. C) To determine if the car had moved, the memory of the pre-saccadic image must be consulted. D) This memory must be remapped (shown in grey scale) to align it with the post-saccadic image. Although the position of the car seems to have changed, the car was previously seen from peripheral vision, making its location estimate less reliable. What inference should be made? The solution to this problem is not straightforward, and studied in Chapter 4 of this thesis.](image)

To maintain a stable representation of space, every time a saccade is made retinal object locations must be remapped. This is needed to keep track of objects, to which future actions may be planned. When the eyes lands, object correspondence must be established by somehow comparing the remapped representation with the new retinal image (see Figure 1.13). We have learned
from, for example, multiple object-tracking experiments that a very important factor for object correspondence is spatiotemporal continuity (e.g. Scholl, 2001; Spelke, 1990). Tracking an object is possible only when it moves coherently through space and time, thus without sudden jumps or disappearances (e.g. Scholl and Pylyshyn, 1999; St. Clair, Huff, and Seiffert, 2010). To this end, the attentional system appears to continuously predict future locations of objects in order to maintain object correspondence (e.g. Verghese and McKee, 2002), and the quality of these predictions depends on the amount of attentional resources available (e.g. Atsma, Koning, and van Lier, 2012; Kerzel, 2003a; Iordanescu, Grabowecky, & Suzuki, 2009). Likewise, remapped representations across saccades probably contain only attended objects (e.g. Cavanaugh and Wurtz, 2004; Gottlieb, Kusunoki, and Goldberg, 1998; Wurtz, 2008).

However, attention is not all or none. An unattended object may escape awareness, but that does not mean it is not processed. How else would it be possible to voluntarily shift attention to a previously unattended location? Some crude neural representation must be already present to make a shift in attention possible. Thus, the quality of the remapped representation likely varies heavily between objects. The more attention an object received before the saccade, the higher the quality of its remapped representation. Interestingly, saccades appear to be always preceded by a strong covert shift of attention to the saccade target region (Irwin, 1992; Deubel and Schneider, 1996; Kowler et al., 1995), which suggests that the quality of its remapped location should be very good. However, experiments have shown that when during a saccade the saccade target (or another object) is abruptly displaced by a few degrees, it is often incorrectly perceived as stable (Bridgeman, Hendry, and Stark, 1975). This effect or illusion is called saccadic suppression of displacement (SSD; see Figure 1.14) and at first glance it seems to suggest that the overall quality of

![Figure 1.14. Saccadic suppression of displacement. When during the saccade an object is abruptly displaced, the change often goes unnoticed.](image-url)
remapping is poor.

However, Deubel and colleagues (1994; 1998) have shown that with a simple manipulation SSD largely disappears: when the target is blanked for 50 to 300 ms, so the target is not visible immediately after the saccade, even tiny displacements are readily noticed (see Figure 1.15). It seems that the blank violates spatiotemporal continuity, after which the visual system regards the reappearance as a new object. This new representation then does not interfere with the old (remapped) location estimate. But why would the visual system typically ignore these accurately remapped location estimates when an object is abruptly displaced by a few degrees during a saccade? And are the remapped estimates really ignored? This is addressed in our final study in which we compare behavioral data to a computational model, which will be introduced below.

Figure 1.15. Post-saccadic blanking. Saccadic suppression of displacement is strongly attenuated when the displaced object reappears after a brief delay.

1.8. Computations for visual stability

Vision can be regarded as a kind of computation involving various variables. Here, the variables will be object location estimates. But, as with any biological system, the estimates contain noise, meaning that the visual system represents the locations with some uncertainty. There are quite some sources that add noise to the remapped location estimates. For example, the precision of the retina is limited, neural transmission is variable, memory decays over time, and the efference copy is a noisy signal. We may assume that the visual system has learned how noisy our sensorimotor system is and takes this into account.

A popular framework which provides a unifying way to think about how the brain deals with uncertainty is the Bayesian decision theory. This theory allows to combine sensory input with prior knowledge in a statistically optimal way, which reduces the negative consequences of noise
and variability. Central is the idea that a belief can be represented with a probability, a number between zero and one. A zero means “I don’t believe it at all” and a one means “absolutely certain”. This can be formally written as \( P(\text{belief}) \). For example, when I ask you if it is going to rain and you hold the belief \( P(\text{rain coming}) = .3 \) then you would say “probably not”. However, when I mention that I just saw a very dark cloud approaching, you will integrate this new information \( P(\text{rain coming}|\text{cloud approaching}) = .9 \) and reply “very likely”.

![Figure 1.16](image)

**Figure 1.16.** Bayesian approach. The representation of location estimates can be modeled as probability density distributions. A) The remapped estimate from the pre-saccadic image is shown in grey. The estimate based on the post-saccadic image is shown in red. The peak (or mean) of a distribution represents the most likely true location. B) In general, when two estimates are integrated, the new (orange) estimate will have a peak at the statistically most optimal location by taking into account the uncertainty (width) of the estimates. Integration reduces uncertainty, hence integration is preferred when estimates can be integrated.

A location estimate can be modeled using a Gaussian probability density function (see Figure 1.16A). The wider the curve, the more noise or uncertainty. The mean or top of the curve represents the most likely location when no other source of information is included. Now, imagine that you are in a completely dark room and a light was briefly flashed in your visual periphery. Because the flash was hard to see, you are not very certain about its location. To reduce this uncertainty, you can combine the estimate with prior knowledge that tells you that hard-to-perceive stimuli tend to be located in your central vision. As can be seen in Figure 1.16B, optimal integration results in a more precise (i.e.
less noisy) estimate but unfortunately also induces a foveal bias, which has actually been reported in the literature (e.g. Brenner, Mamassian, and Smeets, 2008). This shows that while integrating (i.e. combining) information may be often advantageous as it reduces uncertainty, it could give rise to incorrect conclusions when the wrong kind of information enters the equation.

Niemeier, Crawford, and Tweed (2003) asked participants to indicate the remembered initial location of an object that was abruptly displaced during the saccade. Interestingly, they found that when the displacement was not very large, localization was strongly contracted to the object’s post-saccadic (incorrect) location. In contrast, with large displacements this effect disappeared and localization became more veridical. The authors formulated these SSD findings by the integration of the remapped location with a prior that reflects the assumption that objects typically remain table during a saccade. In other words, the remapped location information is downgraded because stability can be generally assumed. A problem with this model however, is that even when the displacement would be absurdly large, the model would still predict an influence of the prior. This is probably not realistic, because when a displacement is obvious it would not make sense to combine the

**Figure 1.17.** Bayesian causal inference model. The task is to report the pre-saccadic location of the object. A) To determine whether two estimates can be integrated, the probability that both originate from a common source is calculated, written as P(C|mv). B) Next, two parallel representations are created: one with integration (orange), and one without integration (i.e. segregation; purple). C) The statistically most optimal response (cyan) is calculated by weighing these parallel representations based on P(C|mv).
remapped estimate with the post-saccadic estimate in any way. Instead, it could be expected that in this case both location estimates will be processed independently, like with a post-saccadic blank (Figure 1.15). But, how does the brain know when to integrate signals and when to process them independently in the computations to obtain visual stability?

According to the framework we present in Chapter 4, the brain has to estimate the causal relationship between the pre-saccadic and post-saccadic signals to establish to what degree they can be integrated or when they should be kept apart, which not only depends on the precision of these signals but also on their spatiotemporal difference. Our simplified model is shown in Figure 1.17. In the first step of this model, the probability that the object remained stable during the saccade, \( P(C|mv) \), is computed based on the relative discrepancy between the signals \( m \) and \( v \) (see Figure 1.17). In the second step, two new estimates are computed, one based on the optimal integration of \( m \)
and $v$, and the other solely on $v$, the segregated estimate. In the third and last step, the integrated and segregated estimates are weighted based on $P(C|mv)$. This means that when $P(C|mv)$ equals 1, the system is confident that the object remained stable and the response is based only on integration. When $P(C|mv)$ equals 0, the system is confident that the object was displaced and the response is based only on segregation.

In Chapter 4 we used SSD task in which three object locations had to be remembered across a saccade (see Figure 1.18). During the saccade one object jumped to a new location while the remaining two objects were removed from the display. After the saccade, the initial location of the object had to be reported. We found that our model closely resembled the data, which suggests that the brain indeed considers two possibilities simultaneously: the object has moved or the object has remained stationary. When an object was only slightly displaced during a saccade, stability was judged more probable and localization relied more heavily on the (more precise) integrated estimate. This shows that the remapped location estimates are not discarded at all. Instead, the brain uses all sources of information in a statistically optimal way.

1.9. Outline of this thesis

The goal of this thesis is to obtain a better understanding of the processes involved in visual stability. In Chapter 2 we use peri-saccadic mislocalization, a phenomenon linked to retinal remapping of object locations to investigated whether remapping can be initiated by the mere preparation of a saccade. We show that with canceled saccades, no ‘peri-saccadic’ mislocalization is present, supporting the notion that saccade execution is a prerequisite for remapping.

In Chapter 3 we again study countermanding, but now with arm reaching movements. We hypothesize that in contrast to saccades where the stop signal reaction time can only be estimated indirectly, countermanded arm reaching movements involve antagonist muscle activity that actively brakes the movement, which may reflect the stop signal reaction time directly. We show that indeed the onset of antagonist recruitment following a stop signal can be regarded as a within-trial stop signal reaction time.

In Chapter 4 we investigate the computations involved in visual stability using a saccadic suppression of displacement task. We show that we can model localization behavior based on Bayesian decision theory, meaning that the
brain estimates the causal relationship between pre-saccadic and post-saccadic signals to establish to what degree they can be integrated or when they should be kept apart.

In the final Chapter 5 a summary of the thesis is provided and some implications of this work are discussed including suggestions for future research.
2. No Peri-saccadic Mislocalization with Abruptly Cancelled Saccades

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2.1. Introduction

Saccadic eye movements quickly reposition our line of sight to scan the world around us. During saccades, the image of the world shifts on our retina. Nevertheless, we perceive our visual world as being stable, which suggests that representation of the visual world is integrated over multiple saccades. In 1867, Von Helmholtz proposed that the brain achieves visual stability by using a copy of a movement command - the efference copy - to adjust perception for the corresponding eye movement. In essence, using efference copy, the brain differentiates sensory information arising from its own actions from those that arise from the environment.

Today there is accumulating neurophysiological evidence that the brain incorporates efference copy of a saccade to achieve visual stability (for a review see Wurtz et al., 2011). For example, Morris et al (2012) recently showed that updating of the cortical representation of the eye position starts before saccade initiation. Furthermore, neurons in various cortical and subcortical areas have retinotopic receptive fields that are not fixed to gaze, but shift in the direction of the saccade just before the eyes start to move. Such neurons have been identified in the lateral intraparietal area (e.g. Duhamel et al., 1992), the frontal eye fields (FEF; e.g. Umeno and Goldberg, 1997), the superior colliculus (SC; e.g. Walker et al., 1995), and earlier visual areas like V4 (e.g. Moore et al., 1998), V3, and V2 (Nakamura and Colby, 2002).

A psychophysical phenomenon that has been linked to shifting receptive fields is the systematic mislocalization of brief stimuli presented around the time of a saccade (Ross et al., 1997, Burr and Morrone, 2010; Hamker et al., 2008). Such peri-saccadic localization errors can begin around 100 ms before saccade onset, and peak when flashes are presented around saccade onset. It has been suggested that the mislocalization is driven, at least in part, by presaccadic activity of neurons in the SC or FEF (e.g. Hamker et al., 2008). This implies that the mere preparation of a saccade may contribute to peri-saccadic mislocalization (Hamker et al., 2005; 2008; 2011; Cicchini et al, 2013). If so, can peri-saccadic mislocalization be observed when a saccade is planned, but ultimately aborted just prior to its execution?

To answer this question, we designed a novel paradigm that combines a mislocalization task with a countermanding component that occasionally requires saccade cancellation. The countermanding component has a refined
Materials and Methods (Logan and Cowan, 1984), and an extensive literature describes the neurophysiology that underlies saccadic behavior (for a review, see Schall and Godlove, 2012). This literature demonstrates that oculomotor preparation can be highly advanced on cancelled saccades; many saccade-related neurons in the SC and FEF exhibit increasing levels of activity before being abruptly curtailed (Paré and Hanes, 2003; Brown et al. 2008; Hanes et al., 1998), and oculomotor preparation when the head is unrestrained can even initiate orienting head movements on cancelled trials where the line of sight remains stable (Corneil and Elsley, 2005). Inclusion of the countermanding component therefore provides a test of whether preparation alone can drive mislocalization, independent of other peri- or post-saccadic processes.

2.2. Materials and Methods

2.2.1. Participants

Eight naïve human participants (5 male, 3 female, aged 19-30 years) gave informed consent to take part in the experiment. All subjects had normal or corrected-to-normal visual acuity, and were free of any known sensory, perceptual, or motor disorders. The study was part of a research program approved by the ethics committee of the Social Sciences Faculty of the Radboud University Nijmegen. Each subject participated in 4 experimental sessions of approximately an hour each.

2.2.2. Experimental setup

Subjects sat in a dimly lit room (luminance ~0.06 cd/m^2) with their head supported by a chin rest. They operated a two-button computer mouse. Stimuli were controlled using a custom written program in Delphi (Embarcadero, San Francisco, California) software. Visual stimuli were projected onto a screen, using a projector (Sharp PG-M20X) running at 60 Hz with a resolution of 1024 x 768 pixels. The projection screen was placed approximately 90 cm in front of the subject, creating a display with a visual field of 67° x 52°. The top 10 rows of pixels were projected on a wall behind the projection screen, invisible to the subject, but detected by a photo diode to determine the precise onset of stimuli. Binocular eye position was recorded at 500 Hz using a
head-mounted eye tracker (EyeLink II, SR Research Ltd., Ontario, Canada). Because the paradigm is contingent on saccades, saccade onsets were detected and processed on-line using an eye velocity criterion of 150°/s. A saccade was considered inhibited (i.e. cancelled) when the velocity did not reach 50°/s within 500 ms after target onset. A higher velocity threshold was used for detecting a saccade compared to detecting inhibition because otherwise sporadic small saccades could potentially confound the timing of to-be-localized flash. All stimuli were projected on a black background (0.18 cd/m²).

### 2.2.3. Design issues

We combined a countermanding task with a flash-localization task. The design of the paradigm was based on the following considerations. 1) The mislocalization effect should be substantial and should arise as early as possible relative to saccade onset. 2) Saccade reaction times (SRT) should be highly predictable. 3) Saccade preparation should be encouraged as much as possible. We discuss the rationale behind each of these considerations in turn.

First, substantial mislocalization should start early relative to saccade onset to increase the chance of observing mislocalization even on cancelled saccades. To do this, we opted to use large saccade amplitudes (20°; Richard et al., 2009), a low contrast flash (Michels and Lappe, 2004) that was positioned near the fixation point (Maij et al., 2011a; Richard et al. 2009) and referenced to a continuously-visible static ruler that provided a strong visual reference (Lappe, Awater, and Krekelberg, 2000; Awater and Lappe, 2006). As intended, these factors produced a large mislocalization effect that started up to 100 ms before saccade onset (see Results).

Second, since by definition no saccade occurs on successfully cancelled stop trials, SRTs had to be as predictable possible. Such predictability allows us to express the localization error relative to the SRT that would have been produced if the saccade had not been cancelled. To this end, we used a rhythmic (2Hz) sequence of saccades in the beginning of each trial, which is known to reduce SRT variability (Maij et al., 2011a).

Third, to encourage saccade preparation as much as possible once the 2 Hz rhythm was established, the overall length of the saccade sequence was made unpredictable, so that subjects made in total 4-7 saccades per trial. The last saccade of the sequence was the test trial, in which an imperative stop
signal was either presented (2/3rds of all trials) or not. However, the probability of a stop signal occurring on a given saccade varied across the saccade sequence (1/6th, 2/9th, 1/3rd, 2/3rd for the fourth through seventh saccade, respectively). This feature ensured that saccades were usually required, with stopping required on a minority of most saccades within the sequence (i.e., the probability of stopping exceeded 0.5 only when the sequence was 7 saccades long).

2.2.4. Paradigm

Figure 2.1A depicts the course of a trial. A static white ruler (60° x 4.5°, 165 cd/m²) was continuously present at the bottom of the screen. Subjects made the sequential saccades to targets (gray dot, size 0.8°, luminance 28.3 cd/m²) regularly presented at 500 ms intervals. Saccade targets were presented at 20° eccentricity. With the presentation of a new target, the previous target was rendered dark gray for 500 ms, after which it completely disappeared. The final (probed) saccade in the sequence was always in horizontal direction (leftward or rightward) while the other saccades were directed either horizontally or deviated 45° from horizontal. In two-thirds of the trials, the final saccade was accompanied with an imperative stop signal. The stop signal was given by changing the color of the second last target (the current fixation point) to red. The second last target, or stop signal, and the last saccade target disappeared simultaneously 500 ms after the onset of the last saccade target. Near the time of the onset of the final saccade, either inhibited or not, a dark-green vertical bar (0.4° x 7.6°, 0.32 cd/m²) was flashed for 16.7 ms (one frame). The flash was vertically aligned with the fixation and saccade target and positioned either in-between the two targets (inbound flash; +10°) or 10° from fixation into the opposite direction (outbound flash; -10°). The onset time of the flash was chosen randomly from the range of -150 to +50 ms relative to the average SRT of the previous 10 trials. After disappearance of the last saccade target, the dark-green vertical bar reappeared on the far left of the display. Using a mouse, subjects moved it to a location where they had perceived the flash, which they confirmed by clicking the left mouse button. Subjects had to press the right mouse button when the flash was not perceived. The next trial then started.

We varied the stop signal delay (SSD), i.e. the time between the stop cue and the saccade target, using a 1-up/1-down staircase procedure with a step
size of 33.3 ms (2 frames). As a result, the SSD fluctuated around the interval where subjects cancelled about half of the stop trials. Since two out of three trials contained a stop signal, the number of go trials (those without a stop signal), non-cancelled trials (i.e., where a saccade is made despite the stop signal), and cancelled trials were approximately equal.

Figure 2.1. Experimental setup and basic countermanding findings. (A) Graphical depiction of the three trial types. During the preparation of the test saccade, which occurs after a sequence of 3 to 6 saccades, a stop signal could be given (color change of the fixation point). At various times, a green vertical bar was flashed, which subjects had localize in space after the trial was ended (using mouse control). Go-trials (in green): trials without a stop signal; non-cancelled trials (in blue): trials with a stop signal but a saccade was made; cancelled trial (in red): trials with a stop trial during which the saccade was successfully inhibited. (B) Saccadic reaction time (SRT) of a representative subject subdivided by the saccade-sequence length and trial type. SRT of the non-cancelled trials is significantly lower than that of the go-trials ($p<0.05$). (C) Inhibition functions subdivided by sequence length for the same subject. (D) Stop signal reaction time (SSRT) does not vary systematically with sequence length ($p>0.05$).
Before the actual experiment started, subjects performed approximately 120 trials to familiarize themselves with the paradigm. The eye tracker was calibrated (using a 9 point grid) every time the program failed to detect a fixation, which signaled that the eye tracking error was more than 3° (on average, this happened every 120 trials). Every 50 trials, subjects were allowed to take a small break. Each subject completed 1 session of 300 and 3 sessions of 400 trials on separate days, resulting in 1500 trials per subjects. The total experiment lasted approximately four hours per subject.

2.2.5. Data analysis

We performed offline data analyses in Matlab (The Mathworks, Nattick, MA). A saccade was defined as a period where velocity exceeds 50°/s with the SRT being the time between target and saccade onset. Trials were excluded based on the parameters of the last saccade of a sequence. Note that we conservatively rejected a high number of trials, to reduce the chance that the observed patterns of peri-saccadic mislocalization were confounded by the preparation of incorrect eye movements, a lack of subject vigilance, or blinks. Saccades with a very short (<100ms) SRT were excluded because these were likely generated by anticipation, rather than being directed to the final saccade target (7.6 ± 2.1% and 4.7 ± 1.5%; mean ± SE for the go trials and non-cancelled trials, respectively). We also excluded very late (>400ms) SRTs on the basis of outliers (5.9 ±1.4% and 3.5 ± 0.9%). We also excluded saccades with amplitudes <11°, since these saccades could be directed towards the flash (at 10°) rather than the saccade target (13.6 ± 3.3% and 30.8 ± 3.1%). Furthermore, saccades were discarded when they deviated more than 10° from the correct direction (6.8 ± 1.2% and 11.5 ± 2.2%), when the saccade was preceded by a blink which could interfere with perceiving the stop signal or flash (0.8 ± 0.6% and 0.4 ± 0.2%), and when a saccade was absent (7.9 ± 1.9% of the go trials). In approximately 8.0 ± 1.7% of the go-trials, 8.4 ± 1.9% of the non-cancelled trials, and 0.2 ± 0.1% of the cancelled trials the participant reported not having seen the flash, and these trials were also excluded from analysis. This means that on average, 33.2 ± 2.4% of the go-trials, 46.7 ± 2.7% of the non-cancelled trials and 0.2 ± 0.1% of the cancelled trials, respectively, were discarded. We confirmed that this high percentage of rejected trials did not unduly influence the conclusion presented below, as similar results were obtained if we relaxed
our exclusion criteria so that only ~10% of go-trials and non-cancelled trials were discarded.

Of the remaining trials the localization response was defined in the horizontal direction as the difference between the indicated location and the fixation point. The response was signed positive towards the saccade target and signed negative into the opposite direction. The localization responses were further analyzed as a function of the flash onset time relative to the actual or predicted saccade onset. Mislocalization curves were created based on a running average convolving the errors with a Gaussian of 15 ms window width. The variance of the localization errors was computed using a sliding window of 40 ms.

By definition, successfully cancelled stop trials lack a saccade onset time, relative to which any mislocalization effect can be examined. To resolve this problem, we estimated the onset of the putative saccade in cancelled trials based on a model description of the SRT in the go-trials, as if they were not aborted prior to execution. This linear regression model incorporated the mean SRT of the previous two trials, the SRT of the second-last, third-last and fourth-last saccade within the current sequence, whether the previous trial was a stop trial, whether the previous trial was a cancelled trial, whether the current saccade is left- or rightwards, and the current sequence length. We used the same model to predict the SRTs of both the non-cancelled and successfully cancelled stop trials.

To characterize countermanding behavior, we computed inhibition functions that describe the probability of a non-cancelled (i.e. executed) saccade on a stop trial as a function of SSD. We further computed an estimate of the time needed for saccade cancellation, i.e., the stop signal reaction time (SSRT), using the integration method (Logan, 1994). This method follows from the idea that a saccade escapes inhibition only when the associated SRT is smaller than SSD + SSRT. The probability that a saccade escapes inhibition for a given SSD (from the inhibition function) thus equals the probability that SRT < SSD + SSRT. The go trials can serve as a baseline distribution of SRTs. When, for example, 20% of the saccades are non-cancelled for a given SSD, these saccades can be represented by the 20% fastest saccades of the baseline distribution. The upper bound of these 20% fastest saccades marks the point where SRT ≈ SSD + SSRT. The SSRT can be obtained by simply subtracting the SSD from this SRT. Here, the SSRT was estimated at each SSD. To obtain a
single SSRT value per subject, we averaged the SSRT across SSDs.

2.2.6. Statistical analyses

Both saccadic reaction time (in ms) and saccade amplitude (in deg) were compared between go-trials and non-cancelled trials, using two-tailed paired t-tests. Differences in localization errors (in deg) and their variance (in deg$^2$) were examined using repeated-measures analyses of variance (Anova), with flash location (inbound, outbound), trial type (go, non-cancelled, cancelled), and time (-150 ms, 0 ms re: saccade onset) as independent factors. Differences were considered significant if $p < 0.05$. Post-hoc testing was performed as needed using t-tests. Finally, to address the potential for type II errors ('false-negatives'), we also calculated the 95% confidence intervals of the mislocalization effect in the cancelled trials, as appropriate.

2.3. Results

2.3.1. Saccade behavior resembles previous countermanding studies

Before examining how subjects localized the flash, we first examined whether their overall saccadic behavior varied across saccade sequence, and whether such behavior conformed to the expectations from previous countermanding studies. Saccadic behavior is shown for a representative subject in Figure 2.1B-D. Figure 2.1B shows that compared to the go trials (i.e., without stop signals), non-cancelled stop trials have a shorter saccadic reaction time (SRT). Across subjects, the SRT was significantly shorter on non-cancelled (mean SRT ± SE: 173 ± 8 ms) versus go-trials (185 ± 8 ms; $t(7) = 4.8$, $p < .005$). Saccade amplitude was also slightly smaller for non-cancelled saccades (19.3 ± 0.5°) compared to saccades on go-trials (20.3 ± 0.5°; $t(7) = 4.2$, $p < .005$). Both findings are consistent with previous countermanding results, since preparation on non-cancelled saccades has to proceed on average slightly faster to escape inhibition (Logan, 1994), and because larger non-cancelled saccades can be truncated in mid-flight (Corneil and Elsley 2005). Figure 2.1B also shows that SRT increased for longer sequence lengths, presumably due to the increasing probability of the appearance of a stop signal (see Methods).

Inhibition functions for the representative subject are shown in
Figure 2.1C; as expected, the probability of non-cancelled saccades increased with SSD, because longer SSDs provide less time for saccade cancellation. The inhibition functions in Figure 2.1C, ordered by saccade sequence, shift rightward with longer sequence lengths which mirrors the increasing SRTs (i.e. with a long SRT, inhibiting the saccade after a late stop signal is still possible). The mean SSRT across subjects was 165 ± 37 ms. For the representative subject in Figure 2.1D, the SSRT did not change systematically with sequence length. Also across subjects, a repeated measure ANOVA confirmed that the SSRT is not influenced by sequence length [$F_{3,5} = 2.59, p > .05$]. By contrast, the SSRT showed a significant negative (linear) relationship with SSDs, meaning that they were smaller for longer SSDs [$R^2$ ranged from .966 to .992, $p < .005$ for all subjects]. This observation is consistent with previous countermanding findings: only cancellation processes that proceed quickly can cancel saccades at longer SSDs (Logan and Cowan, 1984). Taken together, the countermanding analyses provide clear evidence that subjects are preparing the saccade, even if that saccade can be suddenly cancelled. The question that we now turn to is: how did subjects localize flashes on go trials without a stop signal, and on either cancelled on non-cancelled trials with a stop signal?

2.3.2. Flash localization when saccades are made

Following previous studies, localization responses were analyzed as a function of the onset time of the flash relative to saccade onset. Figure 2.2A illustrates the localization responses of the representative subject pooling across leftward and rightward saccades of 20° amplitude, as plotted by the black curve. Flashes were presented at either the inbound (+10°) or outbound (-10°) location (see dashed lines). The green and blue curves visualize the average trend of the go and non-cancelled trials, respectively. Consistent with previous reports (e.g. Ross et al., 1997), clear localization errors are observed for targets flashed in the period of about 100 ms before to 50 ms after the onset of the saccade. A repeated measures ANOVA, with flash location (2: inbound, outbound), trial type (2: go, non-cancelled), and time (2: -150 ms, 0 ms re: saccade onset) as independent variables revealed that across subjects localization effects for the go and non-cancelled trials did not differ significantly over time [$F_{1,7} = 0.015, ns$], see Figure 2.3A. This suggests that similar robust mislocalization is observed when a saccade is executed, regardless of the presence of a stop signal.
2.3.3. Flash localization when saccades are cancelled

In successful stop trials, the saccade is initially planned, but aborted prior to its execution. Since these trials lack a saccade onset time relative to which any mislocalization can be examined, we estimated when the saccade would have happened (predicted SRT) using a linear regression model (see Methods). The model revealed a significant correlation ($r = .43 \pm 0.04 \text{ SE}, p < .05$, for all subjects) between the predicted SRTs and the actual SRTs for go trials. Figure 2.2B plots the measured SRT versus the predicted SRT, based on a linear regression model (see methods section), for the go-trials. (C) Localization errors and mean curve aligned to estimated SRT. Cancelled trials in red. (D) Inhibition functions for each session: the probability of erroneously making a saccade as a function of the stop signal delay (SSD). Cancelled trials were subdivided into three approximately equal-sized bins based on the SSD (short SSD, light red; medium SSD, red; long SSD, dark red). (E) Localization errors in the cancelled trials aligned to predicted saccade onset, for the three subdivisions in SSD.

Figure 2.2. Single subject analysis. (A) Localization errors and mean curve (based on a Gaussian moving window of 15 ms) as a function of flash onset relative to saccade onset. Go-trials in green; non-cancelled trials in blue. Black trace: planned saccade. Gray bar: mean saccade duration. Dashed lines: flash locations (-10° and 10°). (B) Measured SRT versus predicted SRT, based on a linear regression model (see methods section), for the go-trials. (C) Localization errors and mean curve aligned to estimated SRT. Cancelled trials in red. (D) Inhibition functions for each session: the probability of erroneously making a saccade as a function of the stop signal delay (SSD). Cancelled trials were subdivided into three approximately equal-sized bins based on the SSD (short SSD, light red; medium SSD, red; long SSD, dark red). (E) Localization errors in the cancelled trials aligned to predicted saccade onset, for the three subdivisions in SSD.
Based on the same model we can also predict the SRT of the non-cancelled stop trials and the successfully cancelled stop trials. This allows a direct comparison of the localization responses for the cancelled trials with the go and non-cancelled trials (Figure 2.2C). For the go and non-cancelled trials (green and blue, respectively) the mislocalization pattern shows a peak at saccade onset, resembling the pattern in Figure 2.2A (the curves are not identical, given the impossibility of perfectly predicting saccade onset). In contrast, the successfully cancelled stop trials (red data points) indicate variable errors but no pattern, regardless of the location of the flash relative to the target. Thus, in contrast to the other trial types, flash localization on cancelled stop trials do not show ‘peri-saccadic’ errors.

Figure 2.3. Mean localization curves across all subjects. (A) Localization curves of go and non-cancelled trials, aligned to saccade onset (B) Localization curves of go, non-cancelled and cancelled trials relative to predicted saccade onset (C) Localization curves of cancelled trials do not differ for short, medium and long SSD ($p>.05$). Shaded areas, standard error of the mean (SEM).
It is important to point out, however, that while the mean curves show no significant modulation, the localization data from cancelled trials may still contain some structure that should also be carefully analyzed to further validate this conclusion. We subdivided the cancelled stop trials into three categories based on the SSD. The rationale for this is that on average preparation will be more advanced for later SSDs before being inhibited. This contention is supported by neurophysiological evidence showing that some saccade-related neurons in FEF and SC ramp up to a higher level of activity prior to cancellation on stop trials with long versus short SSDs (e.g. Hanes, Patterson, and Schall, 1998; Paré and Hanes, 2003; Brown et al., 2008). The subdivision was done session-by-session (see Figure 2.2D). The subdivision groups contained approximately an equal amount of trials. If localization errors arise due to saccade preparation, we would predict larger mislocalization on cancelled trials with long SSDs. However, Figure 2.2E shows overlapping mislocalization patterns for all three SSD categories that did not change over time.

Figure 2.3 summarizes the localization results of all 8 subjects. Across subjects, the go-trials (green) and non-cancelled stop trials (blue) show highly overlapping curves (Figure 2.3A). In Figure 2.3B, we show the same data relative to predicted saccade onset, together with the data of the cancelled trials which resembles the observation of the cancelled trials for the single subject (Figure 2.2C). Indeed, using a repeated measures ANOVA we found that localization errors are significantly influenced by flash location (2: inbound, outbound), trial type (3: go, non-cancelled, cancelled), and time (2: -150 ms, 0 ms re: predicted saccade onset) [$F_{1,7} = 974.4, p < .0001; F_{2,6} = 21.8, p < .005; F_{1,7} = 41.3, p < .0001$, respectively]. Importantly, the interaction between trial type and time was significant [$F_{2,6} = 17.5, p < .005$], which means that the mislocalization effect is not identical for all three trial types. Post-hoc testing revealed that localization changes significantly as a function of time for the go [$t(7) = 6.5, p < .0001$] and non-cancelled trials [$t(7) = 6.2, p < .0001$], but not for the cancelled trials [$t(7) = 1.2, p > .05, 95\% CI: -0.10^\circ to 0.31^\circ$], which is consistent with the notion that the mislocalization effect only occurs when the saccade is executed. Because the confidence interval (CI) is very small, the potential for a type II error (false negative) is low, which means that the present test would not be sensitive enough only if mislocalization is expected to be smaller than $0.31^\circ$. Furthermore, subdividing the cancelled trials in
three SSD categories (Figure 2.3C) does not reveal peri-saccadic localization errors, even for long-SSD trials (95% CI: -0.55° to 0.34°) where preparation is presumably most advanced. A repeated measures ANOVA confirmed that while localization of the inbound vs. outbound flash was not identical \( [F_{1,7} = 1561.1, p < .0001] \), neither the variable time (2: -150 ms, 0 ms) nor the variable SSD (3: short, medium, long) showed a significant effect.

Finally, as Figure 2.2 shows, localization responses also become more variable when the localization target is presented near the time of a saccade. Can saccade preparation alone at least increase the variability of localization of the flash? To test this, we repeated the same analyses, examining the variance of localization relative to saccade onset (variance was calculated using a sliding 40 ms bin, and pooled over flash location). Figure 2.4 shows the variability in localization judgments across subjects. A repeated-measures ANOVA revealed no significant difference between the go and non-cancelled trials \( [F_{1,7} = 0.1, p > 0.05] \). Furthermore, variability changes significantly as a function of time for the go \( [F_{1,7} = 9.1, p < .05] \) and non-cancelled stop trials \( [F_{1,7} = 12.2, p < .05] \), but not for the cancelled trials \( [F_{1,7} < 0.001, ns, 95\% CI: -0.79 to 0.78 deg^2] \). Again, even after subdividing the stop trials (Figure 2.4C), variability does not change over time \( [F_{1,7} = 0.4, ns] \) and no significant difference exist between the three SSD categories \( [F_{2,6} = 1.2, p > .05] \).

![Figure 2.4](image.png)

**Figure 2.4.** Variability of the localization. Variability increases closer to saccade execution (A), but is close to zero in cancelled trials (B), irrespective of SSD (C).

No Peri-saccadic Mislocalization with Abruptly Cancelled Saccades
2.4. Discussion

Briefly flashed objects are mislocalized around saccade onset. It has been suggested that planning an eye movement, not the saccade per se, could at least initiate this visual distortion. To test this hypothesis, we investigated the presence of visuospatial errors using a countermanding paradigm in which a planned saccade is suddenly aborted prior to its execution. Consistent with the literature, we found strong peri-saccadic mislocalization when a saccade was executed, in both go trials as well as non-cancelled stop trials. In contrast, no mislocalization pattern was found when a planned saccade was cancelled close to the point of no return. Similarly, the variability of localization errors was significantly smaller on cancelled compared to non-cancelled saccades, and did not change over time. Our results suggest that the actual execution of the saccade is a prerequisite for mislocalization of briefly flashed objects, rejecting the hypothesis that the preparation of saccade alone evokes such errors.

2.4.1. Did subjects prepare a saccade in the cancelled trials?

One potential criticism is that saccade preparation may not be far enough advanced on cancelled trials to test our hypothesis. However, a number of arguments suggest that the present paradigm pushed the saccadic system close to the point of no return. First, a saccadic sequence enhances saccade planning. Indeed, the reduced reaction time variability on sequences of saccades compared to regular saccades can be explained by a faster and less variable process of saccade preparation (Joiner, Lee, and Shelhamer, 2007). Second, the countermanding behavior we observed conformed to previous studies. Countermanding behavior is typically explained by a race model with stochastically independent accumulating GO and STOP processes, with saccade execution or cancellation being dictated by which process wins. In such a model, the GO process (essentially saccade preparation) may be quite advanced on successfully cancelled trials, depending on the SSD and the progression of the STOP process. While such an independent race model is not entirely consistent with the neurophysiology of the oculomotor system, where gaze-holding and gaze-shifting mechanisms can interact, computational “interactive” race models show that cancelled trials can still feature highly advanced saccade preparation prior to potent cancellation (Boucher et al., 2007). Third, a common response when the head is unrestrained is the “head-
only movement”, where preparation on successfully cancelled gaze shifts is still advanced enough to initiate an orienting head movement to the target (Corneil and Elsley, 2005; Goonetilleke et al., 2010). Head-only movements are most common at the intermediate SSDs that are preferentially sampled by the 1-up, 1-down method of determining SSD used here (Corneil, Cheng, and Goonetilleke, 2013), as this method best balances the GO and STOP processes against each other. Fourth, neurophysiological studies of saccade countermanding show that some saccade-related neurons in the SC and FEF display substantial buildup of activity on cancelled saccades before being abruptly curtailed before saccade execution (Hanes et al., 1998; Paré and Hanes, 2003). In fact, buildup activity in these areas is essentially identical for both cancelled and non-cancelled saccades up until about 40 ms before saccade execution, at which point the activity for cancelled saccades is abruptly curtailed. Thus, even though we cannot know the degree of preparation on a trial-to-trial basis, we can infer that preparation was very advanced, and sometimes just a few tens of milliseconds away from a saccade, especially when the SSD was long.

2.4.2. Why was there no mislocalization effect with cancelled saccades?

The current findings show that even with substantial preparation no trace of peri-saccadic mislocalization arises if the saccade is ultimately cancelled. While the mislocalization effect could easily exceed 5° with non-cancelled saccades, the confidence intervals for cancelled saccades is less than one third of a degree anywhere, indicating that any substantial mislocalization effect in those trials can be ruled out. We consider this a surprising finding given that saccade preparation increases visuospatial sensitivity at the saccade target area (e.g. Zhao et al., 2012). This modulation is presumably caused by the build-up of activity on saccade-related neurons in the SC and FEF, which may project via reciprocal connections to posterior extrastriate areas like LIP, V4, V3 and V2 (Huerta et al., 1987; Stanton et al., 1995; Schall, 1995). Microstimulation in FEF with a current insufficient to evoke saccades nevertheless increases sensitivity in visual cortex at the retinotopic coordinates where the eyes would have been otherwise guided (Moore, Armstrong, and Fallah, 2003). The time course of these pre-saccadic modulations resembles the dynamics of mislocalization
around a saccade (Burr and Morrone, 2010; Hamker et al., 2008). Therefore, it has been argued that although this pre-saccadic sensitivity at the saccade target may be critical for efficient visual processing (and/or visual stability), it distorts the memorized distance between the saccade target and a flashed object, producing peri-saccadic mislocalization (Hamker, 2008; 2011; Lappe, Awater, and Krekelberg, 2000). The distortion of distance arises because the weak position signal of the flash is averaged with the very strong position signal of the saccade target, which subsequently ‘pulls’ the flash towards it. This explanation is also consistent with the idea that receptive fields stretch or shift peri-saccadically which is in turn responsible for peri-saccadic mislocalization (Cicchini et al, 2013; Ross et al., 2001; Burr and Morrone, 2010; Kusunoki and Goldberg, 2003; Tolias, 2001). So, why was there no mislocalization effect with cancelled saccades?

Our results show that mislocalization only arises when the saccade is actually executed. If we assume that peri-saccadic mislocalization reflects the transsaccadic remapping of object information (which might be carried out by shifting receptive fields), the current findings suggest that the remapping starts only when the saccade plan cannot be aborted anymore. This is consistent with the suggestion by Sommer and Wurtz (2008), who argued receptive fields should only shift if the generation of the saccade is inevitable. Viewed from the other end, if remapping is carried out every time we plan a saccade, instability would arise when the plan is aborted prior to execution, which seems like a suboptimal mechanism.

Taking this one step further, it could be suggested from our results that those saccade-related neurons in SC and FEF that do not distinguish between cancelled and non-cancelled trials during their buildup play no major role in the neural mechanisms for visual stability. Interestingly, Ray et al. (2009) showed that visuo-movement neurons in the FEF, which are another functional class of saccade-related neuron, peak in activity only when a saccade is truly inevitable. The authors speculated that this late enhancement begins at a time coinciding with the transition from controlled to ballistic saccade programming, perhaps only then contributing to the update of visual representations associated with the saccade. This functional distinction between saccade-related neurons could explain our results, and an anatomical basis for segregation may exist (Pouget et al., 2009). Linking to the ideas discussed in the previous paragraph, the encoded distance between the saccade target and flashed object may
become distorted only when these visuomovement neurons burst.

2.4.3. Alternative explanations of the present results

While the present findings provide a novel view on the saccadic remapping mechanisms for visual stability, there are other explanations that should be considered. Recently, Maij et al. (2011b; in revision) provided an optimal integration model, explaining peri-saccadic mislocalization as the result of uncertainty in the time of the flash combined with a foveal bias. Their rationale is that peri-saccadic mislocalization occurs because the observer is uncertain about the time of the flash relative to the saccade, and has a prior expectation that any perceived flash must have been close to the fovea. When no saccade is executed there is no ambiguity of where the eyes were at the time of the flash, hence no peri-saccadic mislocalization would occur. This would also be consistent with the idea that pre-saccadic buildup activity in FEF and SC does not play a role in peri-saccadic mislocalization.

There are also suggestions that peri-saccade mislocalization effects are unrelated to making saccades at all. Ostendorf and colleagues (2006) compared flash localization in a condition where a 10° saccade was executed with a condition in which the subject kept fixation but the stimulus display was moved 10° in a fast saccade-like fashion. Mislocalization in the latter condition had a comparable magnitude and time course as the saccade condition. Another recent study, conducted by Zimmermann and colleagues (2013), reported a strong compression of space around a visual anchor. While subjects kept fixation, the anchor was presented, followed by a brief whole-field mask. An object flashed around the time of the mask was mislocalized in the direction of the anchor.

Both these studies suggest that neither the preparation nor the execution of the saccade is a prerequisite for peri-saccadic-like distortions of space. With an actual saccade, a masking effect is provided by the retinal motion-blur, which is not present with saccade planning alone. Based on these results, it can be suggested that when a transient object is presented, the distance from this object towards the currently attended location is distorted in memory. This distorted representation is used only after the occurrence of a visual discontinuity, such as a saccade, mask, or stimulus motion. When no visual discontinuity occurs, localization can be carried out on a purely retinal basis.
without ‘scene reconstruction’, yielding veridical localization. Future research should be conducted to test this proposal.

Finally, the conceptual approach of the present study could be extended to the study of other peri-saccadic phenomena, including saccadic suppression, sluggish internal representations of eye position, or remapping. The abrupt cancellation of saccades just before saccade execution could advance the understanding of whether a given behavioral or neurophysiological phenomenon is driven by saccade preparation or not, independent of peri-saccadic or post-saccadic processes. Our results suggest that peri-saccadic mislocalization is contingent on saccade execution or trans-saccadic memory, and hence helps to constrain the involved neural substrates.

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3. Neuromuscular markers of movement cancellation in reach control

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This chapter is being prepared for submission as:
3.1. Introduction

In a dynamic and uncertain world, sudden events may require the abrupt cancelation of an impending movement. Movement inhibition is an aspect of executive control that can be studied using the countermanding (i.e. stop signal) paradigm (Logan, 1994), which requires subjects to try to cancel an impending movement following presentation of a stop-signal. The countermanding paradigm has a well-grounded theoretical foundation, the race model (Logan and Cowan, 1984), which permits estimation of the average time needed to react to a stop signal (the stop signal reaction time: SSRT). Although the SSRT cannot be empirically measured, its estimation has proven to be useful in neuroscience because it provides a temporal marker to which neural activity can be related, thereby identifying structures plausibly involved in movement cancelation. For example, changes in neural activity related to the cancellation of eye movements are observed in the frontal eye fields and the superior colliculus (Hanes, Patterson, and Schall, 1998; Paré and Hanes, 2003) and for arm movements similar changes have been observed in the dorsal premotor cortex (Mirabella, Pani, and Ferraina, 2011). Furthermore, estimation of the SSRT has clinical relevance for characterizing psychiatric disorders exhibiting poor inhibitory control, for example obsessive compulsive disorder, schizophrenia, or Parkinson’s disease (e.g. Lipszyc and Schachar, 2010; Gauggel, Rieger, and Feghoff, 2004).

In the laboratory, movement cancellation has been typically studied using rapid, effectively ballistic, movements like saccadic eye movements (e.g. Hanes, Patterson, and Schall, 1998; Hanes and Schall, 1996; Schall and Boucher, 2007; Logan and Irwin, 2000), manual button press responses (e.g. Xue, Aron, Poldrack, 2008; Badrya et al., 2009; Kray, Kipp, and Karback, 2009; Logan and Irwin, 2000), or utterances (e.g. Kray, Kipp, and Karback, 2009; Xue, Aron, Poldrack, 2008). Such movements are easily detected, hence performance on trials with a stop signal can be classified into those that were successfully canceled or not. Further, on successfully canceled trials, processes related to movement cancelation must have reached completion prior to the commitment to move.

However, many of our movements in daily life are not ballistic. Reaching, for example, involves moving a multi-segmental body part that is endowed with considerable inertia. Studies in double-step or related tasks show that
reaching movements are under control throughout their entire trajectory, meaning that a new movement can supersede a movement already in progress (for review, see Battaglia-Mayera et al., 2014; Gaveau et al., 2014). The definition of success on stop trials with such movements is arbitrary and accordingly subjective (de Jong et al., 1990; McGarry and Franks, 2003): is a successfully canceled movement one that is stopped just prior to reaching the movement goal, or one that features absolutely no sign of an overt movement? Adding to this question, a movement that appears to be successfully canceled may still exhibit initial patterns of muscle recruitment too weak to overcome the system’s inertia. One could argue that even in such cases “central” cancellation has failed (e.g. Burle et al, 2002).

The arbitrariness of a definition for cancellation aside, there are potential benefits in studying the cancellation of reaching movements. Chief amongst these is that stopping an inertial object in mid-flight requires active recruitment of antagonist muscles. It has been hypothesized that such muscle recruitment is a direct manifestation of a stopping process initiated by the stop signal (Goonetilleke, Doherty, and Corneil, 2010). This hypothesis predicts that the interval between stop signal and antagonist muscle onset, the antagonist latency, provides a direct measure of the SSRT that is available on a single trial basis. Studies on canceling orienting head movements made during gaze shifts support this hypothesis, showing that the antagonist latency for neck muscle recruitment aligned best to stop signal onset and co-varied with conventional estimates of SSRT both within and across subjects (Goonetilleke, Doherty, and Corneil, 2010; Goonetilleke, Wong, and Corneil, 2012). More recent work has revealed trial-by-trial modification of antagonist latencies based on immediate trial history (Corneil, Cheng, and Goonetilleke, 2013), showing proactive adjustment of movement cancellation processes beyond what could be gained via SSRT estimates.

The goal of the current study is to compare the timing of changes in muscle recruitment to conventional estimates of movement cancellation, using whole-arm reaching movements. Reaching movements offer an alternative platform in which to test the hypothesis of how antagonist muscle recruitment indexes movement cancellation. Participants generated and occasionally attempted to cancel reaching movements executed in the horizontal plane with the right arm (see Figure 3.1A for the setup). Simultaneously, we recorded intramuscular activity from upper-limb muscles contributing to movement
generation and braking. We present a remarkably rich dataset showing a clear response in the motor periphery to a stop signal even with movements that have barely begun, showing that the antagonist latency can be used as a within-trial measure of the stop signal reaction time.

3.2. Materials and Methods

3.2.1. Participants

Nine people (2 female, mean = 30.2 ± 8.7 years old) participated with informed consent and received payment. None reported any neurological deficits and all had normal or corrected-to-normal vision. All procedures were approved by the University Research Ethics Board for Health Science Research at the University of Western Ontario and were in accordance with the Declaration of Helsinki. Participants were aware that they could terminate testing at any time. Two of the authors participated in this study (pp3 and pp7) and were known with the specific goals of the experiment. Their results did not differ from the remaining participants who were naïve to the experimental goals.

3.2.2. Apparatus

Parts of the apparatus and electromyographic (EMG) recording setup have been described previously (Wood et al., 2015). Briefly, participants performed leftward or rightward reaching movements in the horizontal plane while holding the handle of a robotic manipulandum (InMotion Technologies) with the right arm (Figure 3.1A). X and y positions of the manipulandum were recorded at 600Hz. A custom build air sled was positioned under the right elbow to remove friction. Real-time feedback of hand position was displayed as a red dot (0.08 m diameter) against a white background on a down facing LCD (Sony Bravia KDL-46V3000, output 150Hz, input 60Hz), viewed via an up facing mirror. A photo diode, placed at target height at the right edge of the display, recorded target onset times. Unlike previous studies with this apparatus (Wood et al., 2015; Gu et al., 2016), we did not apply any background loading force to the arm.
3.2.3. Electromyography (EMG)

EMG activity from the clavicular head of the right pectoralis major (PEC) and posterior deltoid (DELT) were recorded using two pairs of intramuscular fine-wire electrodes and one surface electrode per muscle. Both muscles are proximal muscles that span the shoulder joint and are recruited very early during whole-arm reaching movements (Karst and Hasan, 1991); accordingly they are good candidates for expressing activity related to active braking of a reaching movement. In our setup, PEC and DELT are active prior to leftward or rightward movements of the right arm, respectively (Wood et al., 2015). While surface recordings lead to equivalent conclusions about the control of whole arm reaching, we will focus on the intramuscular recordings here because of its high signal-to-noise ratio which allows detection of transient muscle recruitment. With two participants (pp1 and pp4), however, surface recordings of DELT were used because intramuscular recordings were lost. For each intramuscular recording, we inserted two monopolar electrodes (A-M Systems) staggered by ~1 cm to enable recording of multiple motor units. For PEC, insertions were aimed ~1 cm inferior to the inflection point of the clavicle. For DELT, insertions were aimed at the middle belly of the posterior deltoid. Intramuscular EMG activity was recorded with a Myopac Junior system (Run Technologies, low-pass filter modified to 2 kHz). Surface EMG was recorded with doubled-differential electrodes (DE-2.1, Delsys Inc., Natick, MA, USA), placed on the same muscle fiber belly, but displaced from the intramuscular electrodes. Both the surface and intramuscular EMG signals were digitized at 4kHz.

3.2.4. Behavioral Task

Participants performed a center-out reaching task with a countermanding component, which required them to move to a peripheral target on most trials (termed no-stop trials; 70% of all trials), but cancel this movement when a stop signal appeared (termed stop trials; 30% of all trials). On no-stop trials, participants were instructed to move with their hand, as soon as possible, to a black target dot appearing randomly 0.2 m left or right from the central starting position, at a visual angle of roughly 20° relative to the central fixation point. After hand position intersected the peripheral target, the target was jumped back to the starting position and was colored yellow, changing back
to black only once the hand position returned within 0.01 m radius of the starting location. The next target appeared after a 1-2 second fixation interval. On stop trials, the black target dot jumped back to the central position after a predetermined delay (stop signal delay; SSD). The participant was instructed to try to keep the hand at the central position on stop trials.

A stop trial was considered successfully canceled when the hand remained within the 0.01 m radius from the starting position, and noncanceled when the hand crossed the 0.01 m radius (Figure 3.1B). No feedback was given regarding the outcome of a stop trial. The SSD was varied adaptively via a 1-up/1-down staircase with a step size of ~28 ms, so that participants were

Figure 3.1. Experimental setup countermanding task, and analysis. (A) Participants hold a robotic manipulandum with the right hand and associated muscles were recorded using both surface and intramuscular EMG. Stimuli and virtual hand position were viewed via a mirror. (B) Each trial started by fixating a central starting position. After 1-2 seconds a target appeared either to the left or right, which needed to be intersected as fast as possible. In 30% of the trials, the target jumped back to the starting position after a delay (SSD), instructing participant to withhold planned the movement. If the hand remained within 0.01 m from the start location, the stop trial was classified as successfully canceled; otherwise noncanceled. (C) Integration method. At each SSD, the SSRT is determined by subtracting the SSD from the point that subdivides the RT distribution into the proportion of noncanceled trials from the inhibition function. This point differentiates those trials that would have escaped inhibition on stop trials (which lie below the point of subdivision) from those that would have been canceled, had a stop-signal been provided (above the point of subdivision). (D) Overview of the measures from kinematics and EMG. Movement RT is defined as the point when the hand crosses the 0.01 m radius around the starting location.

A stop trial was considered successfully canceled when the hand remained within the 0.01 m radius of the starting position, and noncanceled when the hand crossed the 0.01 m radius (Figure 3.1B). No feedback was given regarding the outcome of a stop trial. The SSD was varied adaptively via a 1-up/1-down staircase with a step size of ~28 ms, so that participants were
able to cancel movements within these constraints on approximately half of all stop trials. Because the input and output refresh rate of the display did not match, there was some scatter in actual SSD timings (standard deviation ~15 ms). Therefore, SSD was binned (16.67 ms width) for the estimation of the stop signal reaction time (see below).

Each participant completed one session with a total of 1600 trials, preceded by at least 100 practice trials. After each block of 200 trials, the experiment was paused for at least two minutes. The experiment, including electrode placement, took about two hours.

3.2.5. Kinematic analysis

Data analyses were performed offline. Hand position recordings were analyzed in the left-right dimension only. Movement reaction time (RT) was defined as the interval between target onset and the moment the hand crossed the 0.01 m radius of the starting position. Note that for both the RT measure and the classification of stop trials the same, albeit arbitrary, criterion was used. To quantify movement amplitude for all trials (including canceled stop trials) we computed the maximum deviation of hand position, relative to the position at target onset, in the direction of the target within one second after target onset.

3.2.6. Race model

Performance in the countermanding paradigm can be analyzed within the framework of a race model (Logan and Cowan, 1984). In this model, the outcome of a stop trial depends on which process finishes first: the ‘go process’ or the ‘stop process’. The go process is initiated by target appearance and results in a movement towards the target upon completion. The stop process is initiated by the stop signal, the reappearance of the central fixation target, and results in movement inhibition upon completion. The two processes are assumed to proceed independently (Logan & Cowan, 1984).

One way the SSRT can be inferred is via the “integration method” (Hanes and Schall, 1995; Logan, 1994), which requires the RT distribution from no-stop trials, and the inhibition function that plots the proportion of noncanceled trials as a function of SSD (Figure 3.1C). At each SSD, the SSRT is determined by subtracting the SSD from the point that subdivides the RT distribution into the proportion of noncanceled trials from the inhibition function. The
rationale here, given the assumed independence of the stop and go processes, is that this point differentiates those trials that would have escaped inhibition on stop trials (which lie below the point of subdivision) from those that would have been canceled (above the point of subdivision) had a stop-signal been provided. As suggested elsewhere (Logan, 1994), we avoided SSDs where the probability of movement fell below 0.1 or exceeded 0.9. The SSRT estimates at qualifying SSDs are then averaged to derive a single SSRT estimate.

Another way of estimating the SSRT is via the “mean method”. In this method, a Weibull function is fit to the inhibition function, and the SSD at which P(move) = 0.5 is extracted. This SSD is then subtracted from the mean of the RT distribution to extract the SSRT.

In theory, SSRTs estimated via either method are equivalent. With the exception of some analyses which test assumptions of the race model that require SSRT estimates at each SSD, SSRTs were estimated by averaging the results of the mean and integration methods.

3.2.7. Determining the onset and offset of muscle recruitment

Central to our experimental aim is the timing of changes in muscle recruitment. Defining both the onset and offset of bursts of EMG activity within a single trial at a high temporal resolution is not straightforward, given the variability of background EMG activity before target onset, and the fact that recruitment on canceled stop trials was often very brief and small. Hence, rather than adopting a simple algorithm based on an amplitude threshold (e.g., 2-3 standard deviations above mean baseline activity), we developed an algorithm for detecting recruitment timings based on the work by Liu et al. (2015). This work exploits the fact that the distribution of the logarithmic power of the EMG signal can be characterized by a mixture of Gaussian normal distributions, including a low-power baseline distribution and a high-power ‘burst’ distribution (see Figure 3.2). Using this Gaussian mixture model (GMM), at each time point (0.25 ms steps) a burst presence probability was estimated for several frequency bands in parallel, and together with a clustering algorithm the burst of interest was extracted (see supplementary methods). The algorithm was run from 110 ms after target onset to avoid the stimulus-locked response (SLR), which is a burst of EMG recruitment time-locked to peripheral stimulus onset (Wood et al., 2015; Pruszynski et al., 2010;
Gu et al., 2016). All detected onset and offset times were visually inspected by a trained observer, using a custom written Matlab program, and corrected if needed (6.7% of instances).

**Figure 3.1D** provides an overview of the within-trial measures that are used throughout the paper. The agonist latency is defined as the interval between target onset and the start of the detected agonist burst. The agonist offset latency is defined as the interval between stop signal onset and the end of the agonist burst. The agonist offset latency was included as an alternative measure to the antagonist latency. It should be noted, however, that the agonist offset is less pronounced in the EMG signal and therefore less reliable than the antagonist onset. The antagonist latency is defined as the interval between stop

**Figure 3.2.** Burst detection algorithm. A Discrete Fourier Transform was applied to separate the EMG power into several frequency bands (one band shown). The power distribution was parameterized using three normal distributions: two baseline distributions (blue) which are fitted on a the baseline period (500 ms before target onset), and one burst distribution with higher power (red) which complements the baseline fit to describe the trial period (2000ms after target onset). At each trial, these distributions were fitted anew using the previous three no-stop trials (one trial shown). From this fit only the burst distribution was used, together with the baseline fit of the current trial, to compute the burst probability over time. The burst probability vectors of the three frequency bands that dissociated best between burst and baseline were averaged to get to a single p(burst) vector. Finally, using a low pass filter the burst of interest was extracted.
signal onset and the start of the detected antagonist burst.

### 3.2.8. Data exclusion

A trial was excluded from the analyses when the antagonist latency was higher than 350ms (1.0% of all stop trials) or when the agonist offset latency was negative (i.e. agonist withdrawal occurred before stop signal onset) (1.0% of all stop trials), because these occurrences are not linked to prompt processing of the visual stop signal. Furthermore, when on visual inspection the burst onset appeared ambiguous because of a gradual increase in muscle recruitment, or when an initial small movement was followed by the actual movement a few hundred milliseconds later, the trial was excluded (3.8%). For one participant (pp8) agonist onsets could not be determined reliably because of tonic co-contraction before movement onset. Therefore, agonist onset markers of this participant were discarded.

### 3.2.9. Model simulations

According to the independence assumption within the race model, manifestations of the go or stop processes are independently sampled from their underlying distributions. To test this assumption against our data, bootstrapping simulations were conducted by taking random samples from the observed agonist latency, antagonist latency, and also the SSD distributions. Each such sample produced an interval between agonist and antagonist burst onset, which we then used to predict the movement amplitude that would be associated with such an interval (using a fitted cumulative Gaussian). If the distribution of simulated movement amplitudes did not resemble the data at, for example, each SSD, the assumption of independent recruitment of the agonist and antagonist would be violated.

### 3.3. Results

We studied whole-limb reaching movements in a countermanding task. We hypothesized that the timing of antagonist muscle recruitment arises from the completion of the stop process, and hence should relate to both stop signal onset and the estimated SSRT. First we describe behavior on the basis of hand position recordings in the conventional manner used in countermanding
3.3.1. Continuous control of movement

Figure 3.3 shows the hand movement traces of a typical participant, which is representative of our sample. In this plot, each trace represents a single trial, aligned to the onset of either the right or left target. In the no-stop trials (Figure 3.3A), without the stop signal, the movement amplitudes scatter closely around the target (rotated histograms to the right of the movement traces), meaning that this participant performed as requested. As expected, there is substantial variance in movement RT, as summarized by the respective RT distributions above or below the movement traces.

In stop trials, the target suddenly jumps back to the central position after a variable stop signal delay (SSD), instructing the participant to withhold the movement. In Figure 3.3, we have segregated data from stop trials depending on whether the participant generated a movement beyond the 0.01 m criterion (Figure 3.3B, noncanceled trials) or not (Figure 3.3C, canceled trials). In Figure 3.3B and C, the SSD ranged between 125 and 425 ms (color-coded SSD histograms just above position traces; note how intermediate SSDs were sampled most often), with the colored RT distributions and the hand position traces corresponding to these trials. Interestingly, a substantial proportion of noncanceled movements failed to attain the target, but were arrested in midflight (e.g., compare movement amplitude histograms on the sides of Figure 3.3B to those on Figure 3.3A). Furthermore, compared to the no-stop trials, the noncanceled trials lack the late RTs (Figure 3.3B) suggesting that even the latest stop signals can lead to the cancellation of (presumably) long RT movements. The color-coding of the movement RT distributions support this further: for short SSDs (red/orange), only movements with short RTs escape inhibition. In contrast to the movement traces on noncanceled trials, the movement traces on canceled trials (Figure 3.3C) barely deviate at all. Note as well that there are more canceled trials with early SSDs compared to
noncanceled trials, and more noncanceled trials with late SSDs than canceled trials. These observations are consistent with the notion that the probability of successfully canceling a movement increases the earlier the stop signal is presented.

Figure 3.3. Movement traces with absence and presence of a stop signal. Traces are drawn until the point of maximum deviation towards the target. Stop trials (panels B, C) are color coded by SSD. SSD histograms indicate the proportion of times a given SSD was sampled. (A) In the absence of a stop signal all movements are ~0.2 m as indicated by the tilted histograms at the right. (B) When a movement was made despite a stop signal, movement amplitudes varied considerably. Note how earlier stop signals are accompanied with earlier but smaller movements, as indicated by the histograms. (C) When the movement was successfully canceled, amplitudes are typically less than 0.01 m.

Figure 3.4. Histogram of movement amplitudes with all participants stacked for no-stop trials (A) and noncanceled trials (B). Vertical dashed line indicates target position.
As mentioned, the reach behavior of this participant was representative of our sample. In Figure 3.4, we represent movement amplitudes of all participants on no-stop trials (Figure 3.4A) versus noncanceled stop trials (Figure 3.4B). Note how movement amplitude for noncanceled trials ranged from the target location down to the 0.01 m boundary between canceled and noncanceled movements. This observation reinforces the non-ballistic nature of these whole-arm reaching movements, meaning that the stop signal continues to be processed even after movement onset so that commenced movements can still be canceled before reaching the target.

In Figure 3.5, we quantify a number of other observations both for the representative participant (top row), and across our sample (bottom row). The inhibition function (Figure 3.5A,B) shows the proportion of noncanceled stop trials as a function of SSD, and as expected, the proportion of noncanceled trials increases the later the stop signal is presented (Lappin and Eriksen, 1966). All participants exhibited this pattern, although there was substantial inter-subject variability in where this function was centered along the x-axis, which generally reflects differences in RTs between participants. As described in the
Methods, the inhibition function can be used along with the RT distributions to estimate the SSRT, i.e. the amount of time needed to react to the stop signal. Across our sample, SSRTs derived from the integration method and mean method were very similar ($R^2 = .96$) justifying our averaging of both SSRT estimates. SSRTs averaged $244 \pm 28$ ms (mean ± standard deviation), ranging from 215 to 295 ms, which conforms well with previously reported SSRTs for a variety of manual responses (e.g. Boucher et al., 2007; Mirabella et al., 2006; Brunamonti, Ferraina, and Paré, 2012).

3.3.2. Movements conform to the race model

The race model makes a number of predictions about reaching behavior that we can test against our observations. For example, the race model predicts that SSRTs should decrease for longer SSDs, since only those stop processes that proceed faster can produce movement inhibition in such cases (Logan and Cowan, 1984). To test this, we used the integration method to estimate the SSRT at each SSD, and plotted this as a function of SSD. As shown for our representative participant (Figure 3.5C) and across our sample (Figure 3.5D), SSRTs did decrease as a function of SSD (negative correlation $p < .05$; except pp3 and pp8). Thus, as predicted by the race model, SSRTs generally did decrease for longer SSDs.

Another way of testing the race model is to see how well the RT on noncanceled trials across SSDs could be predicted by subdividing the RT distribution on no-stop trials into the proportion of trials that would or would not have escaped inhibition, had a stop signal been provided at that particular SSD (see Figure 3.1C; Logan and Cowan, 1984). This test also predicts that RTs for noncanceled trials should increase for progressively longer SSDs, since longer SSDs permit more slowly proceeding go processes to still escape inhibition. Indeed, across our sample, the observed RTs of noncanceled trials did increase with SSD (Figure 3.5E,F; positive correlations, all $p < .005$), although we found that the trend in observed RTs was generally steeper than the predicted RTs, with RTs at higher SSDs being particularly underestimated (e.g., as shown in Figure 3.5E).

Finally, our observation of highly variable amplitudes for noncanceled movements is consistent with both the non-ballistic nature to these movements, and the continued processing of the stop signal after the movement is
launched. As mentioned above, we found a straightforward relationship between SSD and movement amplitude: movement amplitudes tended to be greater for longer SSDs, both in our representative participant (Figure 3.5G) and across our sample (dots in Figure 3.5H; all positive correlations, \( p < .05 \)). The finding that movements progress further for more delayed stop signals was accurately captured with race model simulations (see Methods; lines in Figure 3.5H; \( R^2 = .86 \pm .11 \), all \( p < .005 \)), which supports the notion of independent recruitment of agonist and antagonist muscles for going and stopping, respectively.

### 3.3.3. Muscle recruitment on no-stop trials

Having established that participants performed in a manner consistent with a race model, we now turn to the profile of muscle recruitment accompanying this task. In particular, we address the question of whether changes in muscle recruitment can provide a proxy of movement cancelation.

To address this question, we measured surface and intramuscular EMG activity of the right PEC and right posterior deltoid muscles (Figure 3.1A,D). In our setup, PEC and DELT contribute to either leftward or rightward planar movements of the right upper limb as an agonist muscle, respectively, and would be expected to contribute to active braking of movements proceeding in the opposite direction as an antagonist muscle. Figures 3.6 and 3.7, respectively, show intramuscular PEC and surface DELT activity during the classified trials types (no-stop, noncanceled and canceled), vertically stacked by either movement RT (A-D) or movement amplitude (E, F). Surface EMG recordings are shown for DELT in this example because intramuscular recordings were lost with this participant. Further, we wish to show that similar observations can be made using either intramuscular or surface recordings. The trial-by-trial timing of various events are marked by colored dots, including burst onset and offset times for agonist and antagonist muscles, time of target and stop signal onset, and movement RT.

We focus first on the recruitment of these muscles during no-stop trials (Figure 3.6A,B for PEC; Figure 3.7A,B for DELT), aligned to target onset and vertically sorted by movement RT. Note that we have organized these figures based on the agonist or antagonist action of the muscle in question, hence leftward (for PEC) and rightward (for DELT) movements are shown
Figure 3.6. Rectified and smoothened intramuscular EMG data recorded from PEC of the representative participant. EMG voltage above baseline is proportional to the darkness of the grey shade. Colored dots show the timing of variety of events (see legend). Trials are vertically stacked based on movement RT (A-D) or movement amplitude (E,F).
Figure 3.7. Surface activity recorded from the DELT muscle of the representative subject. Same format as Figure 3.6. Note that left column represents rightward movements, for which DELT acts as an agonist.
Neuromuscular markers of movement cancellation in reach control

in Figure 3.6A and 3.7A respectively. As would be expected of muscles that serve to move the arm, large bursts of activity preceded the movement RT (i.e. point where position exceeds 0.01 m) in both PEC (Figure 3.6A, by 123 ms) and DELT (Figure 3.7A, by 112 ms). Across our sample, the interval between agonist onset and RT tended to be greater in PEC for leftward movements than in DELT for rightward movements (154 ± 32 ms vs. 135 ± 27 ms, respectively).

On a trial-by-trial basis, burst onset was highly correlated with RT in both muscles ($R^2 = .94 \pm .02$ (PEC) and $R^2 = .94 \pm .04$ (DELT)). Similarly, when these muscles served as antagonists on no-stop trials, they both exhibited a prominent burst of activity that led movement offset across our sample by 197 ± 19 ms (PEC) or 220 ± 24 ms (DELT). These profiles of recruitment are entirely consistent with the first two phases of the triphasic profile of activation that accompany rapid movements of an inertial object toward a goal (Hallett, Shahani, and Young, 1975), with the third phase (agonist muscle recruitment) being visible for the shortest latency movements in Figure 3.6A and 3.7A. One point that should be stressed, since a different interval was observed on many noncanceled stop trials (below), is that the interval between agonist burst onset and antagonist burst onset was quite substantial (e.g., 226 ± 57 ms for PEC activation following rightward movement onset).

One other aspect of muscle recruitment that is apparent on no-stop trials is the banding of PEC recruitment that begins ~100 ms after target presentation, with muscle activity increasing after leftward target presentation (Figure 3.6A) and decreasing after rightward target presentation (Figure 3.6B), before progressing through a series of ~15 Hz oscillations in advance of the main burst of muscle recruitment. This feature is what we and others have termed the stimulus-locked response (SLR) (Pruszynski et al., 2010; Wood et al., 2015; Gu et al., 2016). Across our sample, the SLR was detectable on no-stop trials in 8 of 9 participants on PEC and to a lesser degree in 2 of 9 participants on DELT.

3.3.4. Muscle recruitment on stop trials

We now turn to the profiles of muscle recruitment observed on stop trials (Figure 3.6C-F, and 3.7C-F), subdividing such data based on target direction and the classification of performance into noncanceled or canceled stop trials (above and below the dashed line, respectively). Stop trial data in Figures 3.6
and 3.7 are aligned either to target onset (C,D; cyan lines or dots) or stop signal onset (E,F; red lines or dots). On noncanceled trials, there is a relationship between stop signal onset and the latency of the agonist burst relative to target onset (Figure 3.6C, 3.7C): late noncanceled movements escaped inhibition only when the stop signal was late. Agonist muscle activity commenced as expected, but compared to no-stop trials, the duration of the agonist burst was shorter (123 ± 30 ms compared to 207 ± 84 ms, mean and std across participants), and a burst of antagonist muscle activity started earlier relative to target onset on stop versus no-stop trials (241 ± 49 ms compared to 284 ± 57 ms). These features (foreshortening of the agonist burst, and earlier antagonist recruitment) are consistent with the large variability in the amplitude of noncanceled trials, due to the arm movements being arrested in mid-flight.

Two further observations of muscle recruitment on stop trials are important to note. First, there were a number of instances of detectable muscle recruitment even on canceled trials, in which the hand did not leave the starting position. More specifically, we found agonist activation on 38 ± 15% (PEC) and 31 ± 15% (DELT) of those trials (mean ± standard deviation across participants). Antagonist activation on PEC and DELT occurred at an even higher rate, appearing on 67 ± 10% and 40 ± 23% of canceled trials, respectively. Second, as is particularly apparent in Figure 3.6C and 3.6D, the SLR persisted on stop trials, regardless of whether the movement was canceled or not. Clearly, despite the absence of overt movement of the limb, both PEC and DELT are being recruited on many canceled stop trials.

Next, we asked whether the timing of muscle events, and in particular the timing of agonist offset and antagonist onset, relate to the timing of the stop signal. To examine this, we realigned stop-trial data on stop signal onset (panels E and F of Figures 3.6 and 3.7). This greatly tightened the trial-by-trial variability in the timing of agonist muscle offset (e.g., orange histograms in panels E) and antagonist muscle onset (e.g., magenta histograms in panel F), compared to the variability observed when this data is aligned to target onset (panels C and D). Across our sample, realigning data to stop signal onset reduced the standard deviations of the antagonist onset distributions by a factor of 2.0 ± 0.9 for PEC (p < .005), and 1.8 ± 1.0 for DELT (p < .005). The agonist offset distributions were reduced in a similar way (2.0 ± 1.5 (p < .005) for PEC and 1.6 ± 0.6 (p < .01) for DELT, respectively). Across participants, average antagonist latencies for PEC and DELT were 182 ± 16 ms and 195 ± 18 ms,
respectively. Average agonist offset latencies were 165 ± 22 ms (PEC) and 164 ± 18 ms (DELT).

Another feature of panels E and F in Figures 3.6 and 3.7 is that trials have been stacked in order of increasing movement amplitude (this is possible even for canceled trials, since such trials often included minute movements that did not exceed the position threshold). Ordering trials this way re-emphasizes how larger movements are associated with earlier agonist onsets (blue dots) relative to the stop signal (i.e., note how agonist muscle bursts start earlier for larger non-canceled trials). In contrast, both agonist offsets and antagonist onsets appear to relate with movement amplitude in a reciprocal way: the earlier the antagonist onset, the less motion occurred (Figure 3.6F and 3.7F).

![Figure 3.8](image)

**Figure 3.8.** Averaged EMG activity aligned to burst onset for no-stop (black traces) and stop trials with large (red), intermediate (green), and small (blue) movement amplitudes. Traces are averaged across all trials and participants. The left panel (A) shows the burst profile of the agonist muscle (irrespective of movement direction). The right panel (B) shows the burst profile of the antagonist muscle.

Although not visible in Figure 3.6 and 3.7, because of the chosen grayscale, we also observed that antagonist muscle recruitment on stop trials was typically more brisk than on no-stop trials. This is illustrated in Figure 3.8, which shows the average burst profile of the agonist and antagonist muscles, pooled across participants, for either no-stop trials (black traces) or stop trials associated with a variety of different movement amplitudes (colored traces). The initial ~100 ms of agonist muscle recruitment (Figure 3.8A) was largely similar on no-stop trials and larger non-cancelled movements (red and green traces), but muted for smaller non-cancelled movements (blue traces).
In contrast, the initial ~100 ms of antagonist muscle recruitment was more gradual on no-stop trials than on larger non-cancelled movements, and the recruitment on even the smallest non-cancelled peaked within the first 50 ms. The different profiles of antagonist muscle recruitment on no-stop versus stop trials support our contention that such recruitment relates to a triphasic pattern of activation linked to target onset on no-stop trials, but active braking linked to stop signal onset on stop trials.

Taken together, these finding support our hypothesis that characteristics of muscle recruitment, such as the antagonist onset, provide a proxy measurement of the stop signal reaction time.

### 3.3.5. Antagonist latencies relate to SSRTs

Another way of testing our hypothesis is to examine relationships between the antagonist latency and the SSRT. For example, fluctuations of SSRT across and within subjects should be reflected by comparable fluctuations in the antagonist latency.

Indeed, across participants, we observed that the individual SSRT estimates correlated with the mean antagonist latencies (Figure 3.9A; $r = .86$, $p < .01$). This result agrees with previous work on neck muscle recruitment during orienting head movements (Goonetilleke et al. 2010, Goonetilleke et al. 2012): subjects with longer SSRTs tended to have longer antagonist latencies. On average, antagonist latency preceded the estimated SSRT by $57 \pm 5$ ms (mean $\pm$ SEM).

To further investigate the relationship between SSRT and antagonist latency, we examined how the antagonist latency changed with SSD. Recall that SSRT decreases for longer SSDs (Figure 3.5D), since a late stop signal allows less time for stopping. According to our hypothesis, antagonist latency should also decrease with SSD. However, to examine this relationship, it is necessary to take movement amplitude into account, since longer SSDs are associated with larger movements and longer antagonist latencies. Thus, we examined the relationship between antagonist latency and SSD as a function of binned movement amplitude (0.025 m bins), averaged across all participants. As shown in Figure 3.9B (with SSDs grouped into quartiles for convenience), antagonist latency was negatively correlated with SSD over most movement amplitudes. Thus, with the movement amplitude taken into account, antagonist latency
decreased for later SSDs, as predicted by the race model. Another way of investigating the relationship between antagonist latency and SSD that permits the collapsing across movement amplitude is to subtract the average antagonist latency per movement amplitude. Doing so produces a single antagonist-latency-SSD curve for each subject, and as predicted by our hypothesis, this again revealed a negative relationship between antagonist latency and SSD for most subjects, as was observed for SSRTs (compare Figure 3.5D and 3.9C).

Figure 3.9. Relationships between SSRT and antagonist latency. Color codes as in Figure 3.4. (A) Mean antagonist latency correlates with SSRT across subjects. (B) Across subjects antagonist latency is negatively correlated with SSD over the whole range of movement amplitudes. (C) After subdividing data from stop trials into quartiles based on movement amplitude, antagonist latency is negatively correlated with SSD. (D) Positive relationship between SSRT and antagonist latency within and across subjects (stop trial data divided into quartiles based on SSD). (E) Between-subject correlation between SSRT variance and antagonist latency variance. (F) Same as C, but now for each participant the mean observation from each block of 200 trials is depicted (dots). Lines show linear fits, all having positive slopes.

To directly compare antagonist latency and SSRT, we split the SSDs into quartiles, and then plotted the observed antagonist latency against the estimated SSRT at each quartile. As shown in Figure 3.9D, doing so revealed a positive relationship between antagonist latency and SSRT in 7 of 9 participants.

We also extracted the variance of the antagonist latency, and found that this measure correlated positively with the SSRT variance estimated through the integration method (Figure 3.9E; $r = .82$, $p < .01$). Thus, as predicted by our
hypothesis, participants with more variable SSRTs also tended to have more variable antagonist latencies.

Finally, we investigated if the antagonist latency and SSRT co-fluctuate over time. Previous research indicates that participants may shift priority between going and stopping during the course of the experiment (e.g. Bissett and Logan, 2011; Corneil, Cheng, and Goonetilleke, 2013). Our hypothesis predicts that slowing or speeding of SSRT over time should also be reflected in the antagonist latency. To analyze this aspect of our data, we compared the estimated SSRT and the mean antagonist latency in each of the eight blocks of 200 trials (Figure 3.9F). In all participants, we found a positive relationship between antagonist latency and SSRT, although linear fits to this relationship did not reach significance in most participants due to scatter ($p > .05$). This shows that even within participants, the SSRT appears to be related to the antagonist latency.

Taking together, the antagonist latency and SSRT appear to co-vary both between and within participants, in support of the hypothesis that the antagonist latency provides a proxy for the SSRT.

3.3.6. Opposing and scaled post-error adjustments

We now turn to analyses that would not be possible using conventional estimates of SSRT, given that many trials are needed for such estimates. A well-documented effect in countermanding tasks is post-error slowing of the RT on no-stop trials (Emeric et al., 2007; Boehler et al., 2011; Bissett et al., 2012; Enticott et al., 2009). Previous work on countermanding eye-head gaze shifts has shown that the antagonist latency on neck muscles is also adjusted based on recent trial history (e.g. Corneil, Cheng, and Goonetilleke, 2013), but that the direction of such an adjustment is the opposite of that observed for RTs. Such results were attributed to strategic shifts in the balance between movement generation and inhibition. Here we looked for a similar trial history effect on both movement generation and inhibition using a ‘triplet analysis’ (Nelson et al., 2010) that allows assessment of the change in agonist latency relative to target onset, or antagonist latency relative to stop signal onset, across different trial sequences (see Figure 3.10A). Such a triplet analysis accounts for any long-term fluctuations in these measures across different blocks of trials, and permits pooling across participants if one references all observations to
the n-1 trial.

The effect of trial history on movement generation was assessed via the change in the agonist latency (blue bars in Figure 3.10B). In agreement with previous studies (Nelson et al., 2010; Corneil et al., 2013), agonist latency decreases across a nostop-nostop-nostop sequence \((t(7) = -4.6, p < .005)\), reflecting a hastening in movement generation across no-stop trials with an intervening no-stop trial. In contrast, if the intervening trial was a stop trial, agonist latency increased \((t(7) = 2.38, p < .05)\). Moreover, this post-error slowing across such sequences scaled with movement amplitude (right side of Figure 3.10B), being moderate for small movements (which would include canceled and small amplitude noncanceled trials) and progressively larger the larger the movement on the intervening stop trial (significant linear correlation in 5 out of 8 participants, mean \(r = .24, p < .05\)). Thus, not only was a movement generation process delayed after an intervening stop trial, but the magnitude of the delay scaled with the magnitude of any error that the subject made on the intervening trial.

**Figure 3.10.** Triplet analysis. (A) Graphical depiction of the logic of the analysis, wherein the change in a metric across the n-1 and n+1 trial is assessed as a function of the intervening trial. For movement generation, the change in agonist latency on no-stop trials is assessed across intervening stop or no-stop trials. For movement inhibition, the change in antagonist latency on stop trials is assessed across intervening stop or no-stop trials. (B) When the intervening trial was a no-stop trial (left of dashed line), both agonist latency (blue) and antagonist latency (magenta) decreased. When the intervening trial was a stop trial (right of dashed line), agonist latency increased but antagonist latency decreased as a function of movement amplitude on the intervening trial.
To examine the effect of trial history on movement inhibition, we conducted a similar analysis on antagonist latency (magenta in Figure 3.10B). First, in contrast to previous findings on the effect of trial history on the antagonist latency (Corneil et al., 2013), antagonist latency extracted from stop trials decreased if an intervening trial was a no-stop trial ($t(8) = -2.37, p < .05$). Our failure to replicate previous findings may relate to the effect of movement amplitude on the n-1 and n+1 stop trials on the antagonist latency, which could obscure any effects on trial history. In contrast, this analysis revealed a pattern of decreasing antagonist latency across an intervening stop trial, with this decrease also scaling with the size of the error made ($r = -.44, p < .05$). Thus, in contrast to the slowing effect of an intervening stop trial on movement generation, assayed through the agonist latency, larger errors tended to hasten movement inhibition, assayed through the antagonist latency.

In sum, we found proactive trial-to-trial adjustments in both the agonist and antagonist latency across trial sequences with an intervening stop trial, in line with opposing reprioritization of movement generation or inhibition with immediate trial history.

### 3.3.7. Fewer trials required for accurate measures of antagonist latency than SSRT

In the final analysis, we examine how many trials are required to obtain reasonably accurate measures of either the antagonist latency or SSRT. Requiring fewer trials would be beneficial for studies on children or patients, or for paradigms featuring multiple experimental conditions. To answer this question, we performed post-hoc simulations in which an “x” number of trials was randomly taken from the dataset. Under the assumption that after 1600 trials (with 30% stop trials) we obtained the true value of the SSRT or antagonist latency, we asked how many trials would be required to obtain measures than lay within 10 ms of this true value (this 10 ms criterion is arbitrary, but seems reasonable given that SSRTs double in children with ADHD (Alderson et al., 2008)). Across our sample, about 440 ± 98 trials (mean ± SEM) were required to derive SSRTs that lay within the 10 ms range. In contrast, only 64 ± 14 trials (only 30% of which are stop trials) were required to obtain average antagonist latencies that lay within the 10 ms range. This analysis illustrates that the antagonist latency could be used to provide a more rapid
3.4. Discussion

We studied cancelation of whole-arm reaching movements in humans and recorded EMG at the upper limb muscles involved in the initiation and stopping of the movement. We build on the notion that the arm is under control throughout the trajectory, in contrast to fast brief movements like small saccadic eye movements. We found evidence supporting our hypothesis that the antagonist muscle recruitment following a stop signal, which effectively arrests motion in mid-flight, offers a trial-by-trial proxy for the stop signal reaction time, thereby providing a further perspectives on the timing and nature of movement cancellation beyond what could be gained via SSRT estimates. Measurements of the timing of antagonist muscle recruitment relative to a stop signal, which can also be obtained via surface recording techniques, converge on stable values within less than 100 trials, offering a potentially far more rapid means to assess inhibitory control in young or patient populations compared to standard estimates of the SSRT.

3.4.1. Mid-flight cancellation

We found that processing of the stop signal continued after movement onset for each participant. For the representative participant in Figure 3.6 this can be directly observed near the top of panel E where several trials show agonist recruitment before the stop signal was even presented, yet these movements were still braked in midflight. The notion of control here likely relates to the overall movement duration, since the ~300 ms needed to complete the full 20 cm reaching movement offers ample time to prematurely stop the arm. Such mid-flight control may not be available for briefer duration saccadic eye movements (e.g. <50ms up to ~10° movement), although larger eye-head gaze shifts can certainly be arrested or adjusted in mid-flight (Corneil et al., 1999; Corneil and Elsley, 2005).

Associated with inhibitory control, we observed clear signs of active braking; as expected, stopping an inertial object like the arm requires not only withdrawal of agonist muscle activity, but also antagonist muscle recruitment. For example, in our EMG recordings we observed a tight relationship between
antagonist muscle onset and the size of the produced movement, with a brisker recruitment profile following a stop signal than on uninhibited movements. Furthermore, movement amplitudes conformed with race model predictions, scaling with SSD in a manner that was consistent with independent recruitment of the agonist muscles (in reaction to target onset) and antagonist muscles (in reaction to the stop signal).

The wide range of amplitudes on stop trials emphasizes the arbitrariness of the stopping criterion that divides stop trials into canceled or noncanceled trials. Our stopping criterion was effectively 5% of the movement amplitude on no-stop trials, which resulted in SSRT estimates that compared well to those already in the literature (Boucher et al., 2007; Mirabella et al., 2006; Brunamonti, Ferraina, and Paré, 2012). In a post-hoc analysis, we simulated countermanding performance using different stopping criteria. When the stopping criterion was set more leniently (i.e., a larger window defining successful stopping), the estimated SSRT increased for each participant. For example, when a criterion of 0.1 m was used (i.e., 50% of movement amplitude), the SSRT was inflated by 42 ± 17 ms (mean ± standard deviation). When reaching the target was the criterion instead (i.e. 95% of movement amplitude), SSRT estimates were 72 ± 34 ms higher. Thus, for whole-arm reaching movements, the choice of stopping criterion impacts estimates of the SSRT. This issue, and the arbitrariness of what definition constitutes success, is likely inherent to the study of control of any inertial, non-ballistic movement. In this respect, assessment of antagonist latency relative to stop signal offer the additional advantage of offering less variable, and more empirical, measures of stop signal processing.

Why were our SSRT estimates about 60 ms higher than the average antagonist latencies? Previous work on antagonist latencies in neck muscles have attributed a difference between SSRT and antagonist latency to the efferent lag of the system (Goonetilleke et al. 2010). However, instead of being 60 ms higher, they found that the SSRT estimates were ~20 ms lower than the antagonist latency. Because here the SSRT did not precede, but followed the antagonist latency, the discrepancy cannot be explained by the efferent lag. What else could explain our 60 ms difference? Possibly, the choice of stopping criterion. Indeed, our post-hoc simulations showed that SSRT estimates matched antagonist latencies if the stopping criterion was set to a more restrictive ~0.002 m. This again demonstrates that the antagonist latency may
offer a more robust measure of stop signal processing than the SSRT.

3.4.2. The stimulus-locked response escapes inhibition

In our EMG recordings we observed a brief stimulus-locked response around 100 ms after target onset which is thought to reflect a visual grasp reflex (e.g. Corneil and Munoz, 2014). Studies have shown that the SLR is related to movement generation, as it manifests on agonist and antagonist muscles in a manner consistent with a brief movement towards the stimulus, and scales in magnitude with movement RT (Pruszynski et al., 2010; Wood et al., 2015; Gu et al., 2016). In our data the SLR was present even on successfully canceled movements, suggesting the SLR is not subjected to the control exerted on the primary processes that move the arm toward the target. A future study will examine the timing and magnitude of the SLR on stop trials more closely.

3.4.3. Trial-by-trial proxy for cancellation

In line with previous reports (Goonetilleke et al., 2010; 2012) the antagonist latency and the SSRT co-varied both between and within subjects, demonstrating their relatedness. Both the SSRT and the antagonist latency conformed with predictions of the race model. For example, shorter SSDs required faster stop processes to reach a comparable performance, which was observed on both the antagonist latency and SSRT.

Although the race model assumes a stochastic independence of the go and stop processes, for which we indeed found evidence, we also found functional dependence of the go and stop processes based on trial history. More specifically, after a stop trial the antagonist latency was expedited while the agonist latency was delayed, reflecting an increase in priority for stopping at the expense of movement generation. Such opposing effects have been reported previously (Corneil et al. 2013). Further, when the fixation target was removed 200 ms before target onset, this ‘gap’ reduces reach RTs but simultaneously increased SSRTs (Mirabella, Pani, and Ferraina, 2009; but see Stevenson, Elsley, and Corneil, 2009 for different findings in the oculomotor domain).

A novel observation in our dataset is how the magnitude of adjustment on either agonist or antagonist latency depended on the error amplitude on the intervening stop trial. Thus, not only is the dynamic balance of stopping versus
going affected by the presence of a stop trial, but adjustments depend on the performance on that stop trial in a continuous as opposed to binary fashion. This aspect of the data shows a sensitivity to performance that was not observed in the oculomotor domain (Corneil et al., 2013). Such dynamic balancing in motor generation and inhibition may be linked to selective attention (Mirabella, Pani, and Ferraina, 2009; Salinas and Stanford, 2013). Before a motor command is executed, the appropriate response must be selected and planned, and before that, the visual target needs to be identified and processed. In daily life, the latter will probably affect timings of movement generation, and cancellation, the most: the abundance of visual information in the world challenges the selection of the most relevant stimulus and hence delays the appropriate response. Although the visual information in the current task can hardly be regarded as abundant, selective attention could play a role in that a stronger attentional focus on the periphery (i.e. where targets appear) speeds up the go process at the expense of a weaker attention on the central stop signal, slowing down the stop process (or vice versa when stopping is prioritized). Alternatively, the priority for going versus stopping may be implemented not by spatial attention, but by the speed of response preparation. Future research could test this hypothesis, for example by combining a visual discrimination task that probes spatial attention, with a countermanding task similar to the current study. If it is attention based, visual discrimination should mimic the agonist/antagonist latency balance.

However, in contrast to the effect of a stop trial, a no-stop trial did not affect the agonist and antagonist latency in an opposite way like in Corneil et al. (2013). Both agonist and antagonist latencies decreased across sequences with intervening no-stop trials, although the adjustment to antagonist latency was smaller. Our failure to replicate previous findings may relate to the effect of movement amplitude on the n-1 and n+1 stop trials on the antagonist latency, which could obscure any effects on trial history. The lack of replication may also relate to pooling antagonist latencies across PEC and DELT, given that the within-subject antagonist latencies for PEC and DELT were not identical. A proper analysis would require having sufficient stop-nostop-stop sequences with similar movement directions and error amplitudes on the n-1 and n+1 trials, but such sequences were very rare even though subjects completed 1600 trials.

Taken together, given the similarities with previous reports involving head
movements (Goonetilleke et al., 2010; 2012; Corneil, Cheng, and Goonetilleke, 2013), the findings strongly support the hypothesis that the antagonist latency can be regarded as within-trial proxy for the SSRT for non-ballistic movements in general. Note as well that the conclusions reached from the head movement literature were derived from a relatively small subset of “head-only” errors on stop trials where the head moved even though gaze remained stable on the fixation point. The antagonist latency results reported here are based on a much larger number of trials.

3.4.4. Implications

The antagonist latency has considerable advantages over the traditional SSRT. First of all, it objectively assesses the time elapsed after stop signal onset and, unlike the SSRT, is unaltered by changes in arbitrary stopping criterion. Second, it provides a within-trial measure of stop signal reaction that can be exploited, as was shown here, to provide evidence for trial history effects on movement cancellation. This cannot be done in the same manner as with SSRT estimates, although a variety of task designs have shown proactive adjustments in SSRTs (e.g. Bissett and Logan 2012; Verbruggen and Logan 2009). Third, we found a high occurrence of antagonist recruitment on stop trials (~75%), even on successfully cancelled trials with movement amplitudes at a sub-millimeter scale. Thus, even in the absence of a noticeable movement, a reaction to the stop signal could be picked up with EMG. Fourth and last, in case an individual's SSRT needs to be assessed, the antagonist latency provides much quicker convergence to a stable value compared to the SSRT, requiring less than 100 trials when compared to the hundreds of trials required to estimate SSRTs. A more rapid conference to a stable value for antagonist latencies compared to the SSRT could be prove beneficial for assessments of inhibition in clinical or pediatric populations that may not be amenable to experiments requiring a large number of trials. Finally, given that such populations may not wish to undergo insertion of invasive fine-wire EMG electrodes, it is encouraging that the abrupt changes in antagonist muscle recruitment was easily detectable with surface recordings.

However, there were also some disadvantages with using the antagonist latency in manner described in the current study. First, EMG recordings during a single trial are quite noisy, requiring non-trivial burst detection
algorithms. Second, the limb is a complex and asymmetric motor plant. In our case, PEC and DELT were not recruited symmetrically as agonist-antagonist pairs, as was the case of bilaterally recorded neck muscles (Goonetilleke, Doherty, and Corneil, 2010). In our configuration, PEC likely acted as more of a prime mover than DELT, being recruited sooner relative to target onset when it acted as an agonist, and sooner relative to stop signal onset when it acted as an antagonist. Given this asymmetry, the level and timing of exerted control many not be identical for PEC and DELT. As mentioned previously, such asymmetry is especially problematic in the triplet analysis where certain trial sequences are considered (e.g. stop-nostop-stop sequence with all left targets is very rare). Also, in our design we used a single staircase for stepping the SSD up and down, regardless of movement direction, whereas it may have been better if the SSDs were adjusted for each direction independently to keep performance in each direction at 50%. To avoid these issues, future studies on reaching movements may wish to examine control of movements only in only a single direction (e.g. future studies may wish to examine the contribution of PEC during the attempted cancellations of leftward movements of the right arm).

These complexities aside, measurements of antagonist latency as a proxy for movement cancelation may prove beneficial for other directions of research. The fact that the finish time of the stop process can be estimated on a single trial basis creates the opportunity to identify brain structures involved in movement control without relying on an average time required for cancelation. Related to this, Burle et al. (2016) found a within-trial transient increase in the strength of response inhibition using frontal EEG recordings. In this study, a colored target indicated whether a left or right hand button press was required. While only the color was relevant, the location of the target created an incongruency (Simon effect). When aligning EEG to EMG onset that preceded the button press, they observed response inhibition on incongruent trials, but primarily when the occurrence of incongruent trials was low. In other words, they observed an active inhibitory neural mechanism that is sensitive to context (Burle et al, 2016). Hypothetically, the strength of response inhibition relates to the antagonist latency. The stronger the response inhibition, the faster the antagonist latency. Future research could address this question. Clearly, with non-ballistic movements, EMG activation starts before the decision is made to proceed with the movement (see also Servant et al., 2015; Burle et al., 2002).
This allows one to align neurophysiological signals to the EMG onset times to infer where, when and how the correct response is chosen.

In conclusion, we found strong evidence that the antagonist latency can provide a within-trial proxy for the SSRT. Furthermore, fluctuations of the antagonist latency with recent trial history proceed largely in the opposite direction of movement RT. This within-trial measure of response control may prove beneficial in research on movement control in both healthy and clinical populations.

### 3.5. Supplementary Methods

#### 3.5.1. Burst presence probability

The signal from the differential EMG electrodes can be modeled by Gaussian normal distributions when the log power is taken (Liu et al. 2015). We estimated EMG power by applying a Discrete Fourier transform (4ms, 16 samples Hamming window, with 0.25ms overlap) resulting in 9 distinct frequency bands (0, 250...2000Hz). Power values were expressed in decibels referenced to the band’s mean power: $x = 10\log_{10} \frac{P}{P_0}$. We look into the frequency domain, because muscle activity generates EMG oscillations in a broad range of frequencies, of which some contain more information about the presence of a burst than others. Initially each band was analyzed independently. In a later step, only the frequency bands that dissociate well between burst presence/absence were included in the decision (see section burst extraction).

Unlike Liu et al. (2015) we will define not two, but three states of the muscle: rest ($s = 1$, low power), baseline activity ($s = 2$, intermediate power), and burst ($s = 3$, high power). Thus, our GMM consists of three components:

$$p(x) = \sum_{k=1}^{3} p(s = k) p(x|s = k)$$  \hspace{1cm} (1)

where $p(s = k)$ is the a priori probability of state $k$, which is modeled by parameter $\Phi_k$ (with $\sum_{k=1}^{3} \Phi_k = 1$), and $p(x|s = k)$ being the conditional probability distribution of observing log power value $x$ given state $k$, modeled by a normal probability density function:

$$p(x|s = k) = N(x|\mu_k, \sigma_k^2) = \frac{1}{\sigma_k \sqrt{2\pi}} e^{-\frac{(x-\mu_k)^2}{2\sigma_k^2}}$$  \hspace{1cm} (2)
where $\mu_k$ and $\sigma_k^2$ are the mean and variance of the Gaussian distribution for the given hypothesis $s = k$. Using Bayes rule, we can infer the probability of state $k$ given $x$ as follows:

$$p(s = k|x) = \frac{p(s = k)p(x|s = k)}{p(x)} = \frac{\Phi_k N(x|\mu_k, \sigma_k^2)}{\sum_{m=1}^{3} \Phi_m N(x|\mu_m, \sigma_m^2)} \quad (1)$$

This forms the basis for estimating the burst probability $p(s = 3|x)$. The parameter set $\Phi \triangleq \{\Phi_1, \Phi_2, \Phi_3\}$, $\mu \triangleq \{\mu_1, \mu_2, \mu_3\}$, and $\sigma \triangleq \{\sigma_1, \sigma_2, \sigma_3\}$ are estimated for every trial in a series of steps. These steps follow from the following assumptions:

1. In the 500ms period preceding target onset, which we will refer to as the baseline period, the state of the muscle is either rest ($s = 1$) or baseline ($s = 2$), but never burst ($s \neq 3$). Thus, the probability density of $x$ in the baseline period can be estimated with a two component GMM.

2. If an arm movement is made towards the target, then the probability density of $x$ in the 2000 ms period after target onset resembles the GMM of the baseline period, but with a third component added ($s = 3$) with $\mu_3 > \mu_2 > \mu_1$.

3. We expect the burst state parameters $\Phi_3$, $\mu_3$, and $\sigma_3$ to change slowly over time (e.g. fatigue), while the baseline parameters are expected to change more rapidly from trial to trial (e.g. because of posture changes).

For each trial, a three component GMM was defined. To accomplish this, first a two-component GMM was fitted to the signal in the baseline period. Fits were performed using Matlab's fminsearch function by maximizing the log likelihood of the signal under the model. The baseline fit provided the trial's $\mu_1, \mu_2$, $\sigma_1, \sigma_2$, and preliminary weights $\Phi_2$ and $\Phi_1$ with $\Phi_2 = 1 - \Phi_1$. Preliminary, because they will be lowered once $\Phi_3$ is known. Note that not all trials contain movements: a muscle may never reach state 3 given a stop trial. Thus, to estimate the third component ($\Phi_3$, $\mu_3$, and $\sigma_3$) we used the previous three no-stop trials where we know the muscle has reached the burst state at some point. Estimating the burst parameters this way also avoids "double dipping" which may skew onset timings for the stop trials where burst presence is low. We took the previous three no-stop trials instead of a single
no-stop trial as are reference, because this resulted in more robust fits. For these three reference trials, the baseline periods were taken together and fitted using a two-component GMM. Then, for these trials the 2000 ms periods after target onset were taken together and fitted with the three-component GMM in which the first two components were adopted from the two-component fit with their weights ($\Phi$) fixed proportionally to each other. Now the full GMM for the reference trials is known, only $\Phi_3$, $\mu_3$, and $\sigma_3$ are transferred to the current trial’s GMM fit with the first two weights now being: $\Phi_1 = \tilde{\Phi}_1 (1 - \Phi_3)$ and $\Phi_2 = \tilde{\Phi}_2 (1 - \Phi_3)$.

Once the three-component GMM for a given trial is established, the probability of the muscle being in burst state at the $i$’th time point $p(s_i = 3|x_i)$ could be computed using equation 3. However, doing it this way ignores the variability between samples nearby in time. We know that the muscle transitions in and out of a burst state only now and then, thus including not only $x_i$ also a few nearby samples makes the state estimate more reliable. However, we should be aware not to include samples that are to close in time because they are likely highly correlated due to our 16-sample Fourier window. We found that including $x_{i-8}$, $x_i$, and $x_{i+8}$ into the computations at time point $i$ provided reliable results. The burst probability at $x_i$ is now given by:

$$p(s_i = 3|x) \approx \frac{p(s_i = 3) \prod_{u=-1}^{3} p(x_{i+8u}|s_i = 3)}{\sum_{m=1}^{3} p(s_i = m) \prod_{u=-1}^{1} p(x_{i+8u}|s_i = m)} = \frac{\Phi_3 \prod_{u=-1}^{3} N(x_{i+8u}|\mu_3, \sigma_3^2)}{\sum_{m=1}^{3} \Phi_m \prod_{u=-1}^{1} N(x_{i+8u}|\mu_m, \sigma_m^2)}$$  (4)

### 3.5.2. Burst extraction

The parameter and state estimations were carried out for all 9 frequency bands independently. The inferred weighting parameter $\Phi_3$ provided a clue to which extent a frequency band could dissociate between burst presence and absence. When $\Phi_3 < .005$ or $\Phi_3 > .3$ we excluded the band for that trial, because it is unlikely that the muscle was in a burst state less than 0.5% or more than 30% of the time in the previous go trials.

Next, the overlap of the distribution $p(s = 3|x)$ with the combined distribution $p(s = 1|x) + p(s = 2|x)$ was estimated for each frequency band. The frequency bands showing the least overlap were included for the actual burst detection, as these could dissociate best between the burst and no-burst states (up to three bands were included). The burst probability vectors of
the included frequency bands were averaged, and transformed into a binary vector: \[ p(s = 3|x) > p(s = 1|x) + p(s = 2|x). \]

Because hand kinematics were recorded, we used temporal markers where hand velocity into the target direction crossed 0.01 m/s to specify intervals in which bursts are expected. When hand velocity did not cross 0.01 m/s within a trial, bursts that occurred up to 1 second after target onset were considered. Bursts occurring before 110 ms after target onset were discarded, to avoid the SLR (Pruszynski et al., 2010; Wood et al., 2015; Gu et al., 2016).

The agonist onset was expected to occur not earlier than 150 ms before velocity crossed 0.01 m/s. The antagonist onset was expected to occur not earlier than 80 ms before velocity crossed 0.01 m/s. In addition, the antagonist onset should be later than the agonist onset. To remove noise from the binary vector, bursts (a cluster of adjacent ones) that had a duration of less than 4 ms were removed. Then, bursts were concatenated when they were less than 50 ms apart. The first detected burst was considered the burst of interest, taking into account the intervals in which bursts were expected.
4. Causal inference for spatial constancy across saccades

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4.1. Introduction

During saccadic eye movements, the image of the world shifts across our retina. Despite these shifts, we perceive targets as having world-stable positions, and have no problem to act upon them whenever necessary. It has been suggested that a combination of predictive and feedback mechanisms subserve this faculty, referred to as spatial constancy (Medendorp, 2011).

In the literature, spatial constancy has been studied by using motor and perceptual tasks. Using motor tasks, it has been shown that we can look or reach accurately to the remembered position of a target after an intervening saccade (see Medendorp, 2011 for review). Using arm movements, Vaziri et al. (2006) recently tested the hypothesis that the brain computes the position of a reach target after a saccade based on the optimal integration of predicted and actual sensory feedback. In their paradigm, participants first made a saccade after they briefly foveated a visual target in complete darkness. The brain is known to predict the new retinal position of this target after the saccade by internally remapping its representation relative to gaze (Medendorp, 2011; Duhamel, Colby, and Goldberg, 1992; Wurtz, Joiner, and Berman, 2011). Next, the target was post-saccadically viewed for a variable duration, slightly displaced relative to its initial position, before the participant reached at it. Results show that reach endpoints had smaller variance than was possible based on the predicted (i.e. remapped) estimate or the actual post-saccadic estimate alone, consistent with integration. The authors further demonstrated that the uncertainty of the post-saccadic target position, which was modulated by varying its viewing time, affected its weight in the integration process.

From a perceptual perspective, it has been shown that the sensitivity to perceive the displacement of a visual target severely drops during a saccade. In fact, target displacements up to one third of the saccade amplitude typically go unnoticed, which is known under the term saccadic suppression of displacement (SSD; e.g. Bridgeman, Hendry, and Stark, 1975). Remarkably, blanking the target briefly after the saccade, before it reappears at a displaced position, significantly improves the sensitivity to the displacement (Deubel, Schneider, and Bridgeman, 1996), as does merely changing some characteristic of the saccade target, such as its form or polarity (Demeyer et al., 2010; Tas, Moore, and Hollingworth, 2012). This has led to the notion that the visual system a priori assumes that a target will not move or change during the
saccade. If this assumption is broken, as with the blank, form change, or with large displacements, it causally regards the post-saccadic target as a new object, and computes the old position using retinal and extraretinal signals.

Niemeier et al. (2003) formulated the SSD findings from an optimal integration perspective by combining visuomotor signals with a prior that reflects the assumption that targets are not displaced during the saccade. As predicted by their model, behavioral reports show that SSD has a nonlinear relationship with the size of the target displacement. While with small displacements the localization of the initial pre-saccadic target was strongly contracted to the post-saccadic target, this integration effect was reduced with larger displacements, making localization more veridical.

But how does the brain know when to integrate signals and when to process them independently in the computations to obtain spatial constancy? From a perceptual viewpoint, Vaziri et al. (2006) essentially used a blanking paradigm, thereby ignoring the possible assumption that visual targets typically do not move during saccades. Despite the blank, which is assumed to indicate that sources are unrelated, their results show optimal integration of the pre-saccadic target information and actual post-saccadic target position. Also in the model of Niemeier et al. (2003), the spatial constancy computations are unconditioned to causality: integration always occurs even with large target displacements.

In the present study, we test the role of causal inference in the computations to obtain spatial constancy. According to this framework, the brain has to estimate the causal relationship between the pre-saccadic and post-saccadic signals to establish to what degree they can be integrated or when they should be kept apart, which not only depends on the precision of these signals but also on their spatiotemporal difference (Wozny, Beierholm, and Shams, 2010; Kording et al., 2007). Based on the pre-saccadic input, it could be hypothesized that initially foveated representations are less susceptible to SSD than non-foveal representations because their remapped representations are more precise, triggering a segregation strategy. Based on the post-saccadic input, it could be proposed that if the post-saccadic target is presented only briefly, its representation is too weak to infer a target displacement, making the brain rely most heavily on an integration strategy in the later localization of the target. But if the post-saccadic target is viewed longer, displacements may become better detectable, triggering a segregation strategy, especially with
large displacements.

Here, we test these hypotheses by varying the duration of the post-saccadic display in an SSD task for displacements of the initial fixation target, the saccade target and a non-foveated peripheral target. Because previous studies reported direction-specific SSD, (e.g. Whipple and Wallach, 1978; Mack, 1970) we test for both parallel and orthogonal displacements relative to the direction of the saccade.

We show that spatial constancy is not based on the exclusive integration of pre-saccadic target information and actual post-saccadic sensory feedback nor does it follow from an a-priori assumption that targets do not move during saccades. Our results suggest that spatial constancy naturally follows from the principles of causal inference involving two possible causal structures: one where the pre- and post-saccadic percepts represent the same stable object (i.e. have a common cause), and one where two distinct objects are perceived (i.e. no common cause).

4.2. Materials and Methods

4.2.1. Participants

Twelve naïve participants (eight females, average age 25.7 ± 0.6 years, mean ± SEM) participated in the experiment, all with normal or corrected-to-normal vision. The study was part of a research program approved by the ethics committee of the Social Sciences Faculty of Radboud University. Each participant participated in four experimental sessions of approximately 1 h each and informed consent was given beforehand. One participant did not complete all sessions because the eye-tracker helmet felt uncomfortable. We discarded her data.

4.2.2. Experimental Setup

Participants sat in a dimly lit room with their head supported by a chin rest. They operated a two-button computer mouse. Stimuli were controlled using a custom-written program in Delphi (Embarcadero) software. Visual stimuli were displayed on a 19-inch CRT monitor (Philips 109B) using a vertical refresh rate of 100 Hz and a resolution of 1024 x 768 pixels. The monitor
was positioned about 30 cm in front of the participant’s eyes, encompassing 61° x 46° (HxV) of the visual field. A photodiode was placed over the bottom-left corner to determine the precise onset and displacement of the visual stimuli with respect to eye movements. Binocular eye position was recorded at 500 Hz using a head-mounted eye tracker (EyeLink II; SR Research). The eye tracker was calibrated using a 9-point grid. A saccade was detected online using a position threshold of 1.5°. Participants were allowed to take breaks every 400 trials. After each break the eye tracker was recalibrated and as needed during testing, for example when the program failed to detect a fixation at the start of a trial.

4.2.3. Experimental protocol

We tested participants in an SSD task with three target positions, each of which contained a gray shape (circle, square, or triangle, all 1° size). Figure 4.1 presents a graphical depiction of a trial. At the start of the trial, the three target shapes appeared 15° apart at equilateral triangular positions against a light-grey background. The shapes designated the fixation target (FT), the saccade target (ST), and a peripheral non-target (NT). The specific shape of each target was held constant for each participant (e.g. the triangle was

![Figure 4.1](image)

**Figure 4.1.** Graphic representation of a trial. Each trial started with the presentation of three objects, of which the FT (here: triangle) was foveated. After an auditory go cue, a horizontal saccade was initiated to the ST (here: circle). Upon detection of the saccade, two objects were removed from the screen while the other was displaced (here: orthogonal). The displaced object remained visible for 50 ms, 300 ms or until the response was given (~1000 ms). The remembered pre-saccadic location of this object was indicated using a computer mouse.
always the FT), but counterbalanced across participants. The participant was instructed to first foveate the FT, i.e. the triangular target in Figure 4.1. After the participant had kept fixation for a random duration of 200-500 ms (discouraging anticipatory saccades), an auditory signal (1kHz sine-wave beep, 60 ms) instructed the participant to saccade to the ST. The saccade was always in horizontal direction, either leftward or rightward in randomized order. The NT appeared midway between the ST and FT, above or below (randomized). The exact position of these targets relative to the screen’s center was varied (over a range of 27° horizontally and 20.6° vertically, flat distributions) in order to deter learning the exact location of the targets on the monitor. During the saccade, at on average 36 ± 8.3 ms (mean ± std) after saccade onset, one of the three targets was displaced, while the other two were removed from the display. The target displacement (−5, −3, −2, −1, −½, 0, ½, 1, 2, 3, or 5 degrees) was parallel or orthogonal to the saccade. The displaced target remained visible for 50 ms, 300 ms, or for about 1000 ms until a response was given, the ‘1000 ms’ condition. The time between saccade offset and the response was kept constant such that memory decay of the pre-saccadic scene was similar for the three viewing conditions. Together, this defined 792 trial types (i.e. 2 saccade directions, 3 targets, 2 NT locations, 11 displacement sizes, 2 displacement directions (parallel vs. orthogonal), and 3 viewing durations). For our first six participants, the 50 ms and 300 ms condition were randomly presented in the first three experimental sessions; the 1000 ms condition was tested in a separate session. For the other group of participants, the three viewing time conditions were fully mixed in all four sessions. No significant differences between both groups were found.

Participants gave their response using a mouse cursor (small crosshair) indicating the pre-saccadic position of the displaced target, which they confirmed by clicking the left mouse button. The cursor appeared always 300 ms after the displacement occurred. Participants performed each trial type 4 or 5 times. In case the saccade endpoint deviated more than 5° from the ST location, a red screen was shown for 1000 ms after a response was given. Eye blinks that triggered the target to jump were also followed with a red screen. If the participant did not know about which of the three targets to report, he or she had to shift the cursor to the left border of the display, before clicking the mouse button. Before the actual experiment started the participant completed a series of practice trials until s/he felt comfortable with the task.
4.2.4. Data analysis

We performed offline data analyses in Matlab (The Mathworks, Natick, MA). Trials in which the target displacement did not occur during the saccade (eye velocity < 50°/s for offline analysis) were discarded (14.6 ± 2.0%; mean ± SEM). Trials in which the post-saccadic target was not perceived (2.7 ± 0.7%) and trials with localization responses that were closest to a target other than the original position of the post-saccadic target were also discarded (3.7 ± 1.4%). We also discarded trials with a red screen (2.7± 0.6%). As a result, each participant completed on average 2427 ± 111 correct trials. Across participants, saccade duration was 50.7 ± 1.1 ms and saccade amplitude 14.0 ± 0.2°. There was no instruction on saccade reaction time. Average saccade latency, 273.7 ± 45.4 ms (mean ± SEM), was higher than usual, probably because of the memorization of the pre-saccadic positions (cf. Zimmermann, Morrone, and Burr, 2013). The total duration that the targets were displayed before the saccade was on average 1200 ± 60 ms.

Data of four experimental configurations, that is a left/rightward saccade and NT above/below, were pooled by transforming them toward the single configuration shown Figure 4.1A, reducing the number of unique trial types to 198. Localization error was defined relative to the pre-saccadic target location, and was signed positive into the horizontal saccade direction and vertically upwards (see Figure 4.1A).

4.2.5. Mixture model

We modeled the role of causal inference in the computations to obtain spatial constancy. The model has to explain the observed responses of each participant. Our principal model involves a statistically optimal mixture at the trial level of two possible causal structures on the signals available. This 2D model is developed here, formulated along the lines proposed in Körding et al. (2007), to which we will frequently refer for further information. In the subsection ‘Alternative Models’ below we will introduce two variants of this model, also considered by Wozny et al. (2010), involving at the trial level not a mixture of, but a choice between the two possible causal structures.

By estimating the causal relations between the various sources of information the brain attempts to determine whether two percepts belong together or need to be processed independently. More specifically, on each
trial the task of the system is to estimate the pre-saccadic target position on the screen, denoted \( s \), based on two sources of information, the memory-based remapped pre-saccadic position percept, denoted \( m \), and the position percept of the post-saccadic visual stimulus, denoted \( v \). Both entities are available with finite precision only (having some amount of noise) and are represented by probability distributions, which constitute the input to the causal inference model expounded below. First, we briefly describe how we modeled these probability distributions of the single source percepts \( m \) and \( v \).

The distributions of both \( m \) and \( v \) are assumed to be independent 2D Gaussians. It can be expected that the variance of \( m \) has several sources, such as retinal noise during target encoding, remapping noise related to target updating, and noise due to memory decay. Some of the noise sources may be anisotropic (e.g. Niemeier, Crawford, and Tweed, 2003). For simplicity, we do not model these sources but use a combined estimate \( \sigma_m^2 \) for each target position and allow anisotropy. Thus, \( \sigma_m^2 \) is estimated per target position, both for the parallel and orthogonal direction, resulting in 3x2 free parameters for \( m \). For \( v \) we assume its variance to be isotropic, primarily determined by encoding noise. Intuitively, the shorter an object is viewed, the more noisy the position percept. Thus, \( \sigma_v^2 \) is estimated per viewing time condition (irrespective of target), resulting in 3 free parameters for \( v \).

It has further been suggested that participants localize visual targets towards the fovea (e.g. Kerzel, 2002; Brenner, Mamassian, and Smeets, 2008; Maij, Brenner, and Smeets, 2011). We modeled this foveal bias by including a prior, specified as an independent isotropic 2D Gaussian with variance \( \sigma_f^2 \) centered at FT for \( m \) and at the saccadic landing point for \( v \) (see Figure 4.2A), and by interpreting the percepts \( m \) and \( v \) as the results of an optimal Bayesian integration process of accurate sensory signals \( \tilde{m} \) and \( \tilde{v} \), respectively, with this prior. As a consequence, the center of \( m \) is not at the true target position, but shifted in the direction of FT by the fraction \( \frac{\sigma_m^2}{\sigma_f^2} \) of the distance between these points (see Figure 4.2B). Similarly, the center of \( v \) shifts from the true target position in the direction of the saccade landing point by the fraction \( \frac{\sigma_v^2}{\sigma_f^2} \) of the distance between these two points.

These single source distributions play an essential role in the mixture model, in which the evidence for target position \( s \) given memory information \( m \) and visual information \( v \) takes the form of a probability density function \( p(s|mv) \). Thus, \( p(s|mv) \) is the localization response, given estimates
\( m \) and \( v \). In order to determine this \( p(s|m,v) \) in an optimal way, the system has to process correctly the probabilistic information available in \( m \) and \( v \). That is, the system has to acknowledge that, while there is a direct relationship between \( m \) and \( s \) on each trial, this is not the case for \( v \) and \( s \). Depending on the discrepancy between the two sources of information the system may either see no evidence for a displacement and consider the information \( v \) as relevant for the pre-saccadic position \( s \) to be reported (Figure 4.2C; integration), or it may take \( v \) to refer to a new visual object without a clear relationship with \( s \) (Figure 4.2C; segregation). In short, the system may distinguish two kinds of trials, requiring different forms of \( p(s|m,v) \). In this probabilistic setting the optimal procedure for the system is not to choose per trial one of these forms, but to apply on any trial a mix of both, with the weight for each form equal to the estimated probability of it being the correct one given sources of information \( m \) and \( v \) (Figure 4.2D). Denoting the situation of a trial where both \( m \) and \( v \) derive directly from the pre-saccadic position \( s \) by \( C \) (common cause for \( m \) and \( v \)) and one where \( v \) derives from a different object (the displacement) by \( \bar{C} \) (no common cause for \( m \) and \( v \)), this leads to a mixture model of the representation of \( p(s|m,v) \) (Kording et al., 2007):

\[
p(s|m,v) = p(s|m,v,C) \cdot p(C|m,v) + p(s|m,v,\bar{C}) \cdot p(\bar{C}|m,v)
\]  

(1)

This model consists of three components: (i) \( p(s|m,v,C) \), the distribution of \( s \) given \( m \) and \( v \) when \( v \) is the sensory representation of the true position; (ii) \( p(s|m,v,\bar{C}) \), the distribution of \( s \) given \( m \) and \( v \) when \( v \) does not represent the true position, but a displaced version of it; and (iii) \( p(C|m,v) \), the probability that the current \( m \) and \( v \) are from a trial with common source, with \( p(\bar{C}|m,v) = 1 - p(C|m,v) \) the complementary probability of a trial with \( m \) and \( v \) referring to different positions. We will now discuss the specification of these three components in turn.

(i) **The distribution of \( s \) under the assumption of no displacement.** In this situation both \( m \) and \( v \) are directly informative about the true position \( s \) and this is a case for the standard optimal integration model. By the laws of probability ("Bayes rule") and assuming that \( m \) and \( v \) constitute two independent sources of information of a specific position \( s \) we obtain:
Here, the denominator is a normalizing constant, independent of $s$, while the three factors of the numerator represent, respectively, the likelihoods of remapped position $m$ and visual sensory information $v$ given $s$, and the prior probability of $s$, the probability of $s$ being at a certain spot of the screen independent of any sensory trial information, all of this in trials without a displacement. The first two are the independent 2D Gaussian $m$ and $v$ distributions described above, and the prior for $s$ is taken to be of the same kind, centered at some point $\pi$ of the screen and having anisotropic variance ($\sigma^2_{\pi x}$ and $\sigma^2_{\pi y}$).

(ii) The distribution of $s$ under the assumption of a displacement. In this case, $m$ still derives directly from the true position $s$, but $v$ refers to a different position. Without any systematic relationship between this new position and $s$ it is unclear how $v$ can contribute to the estimation of $s$. The optimal procedure is then to not integrate and disregard $v$. In terms of probability distributions:

$$p(s|m v C) = p(s|m \bar{C}) = \frac{p(m | s C) \cdot p(s | C)}{p(m | \bar{C})}$$

Actually, the distinction between $C$ and $\bar{C}$ trials has only to do with the role of the information $v$ and there is no reason why the likelihood of $m$ or the prior for $s$ would be different for the two kinds of trials. That is, these distributions can be taken identical to their counterparts in the $C$ trials described in (i) above and the consequence is that the specification of $p(s | m v \bar{C})$ coincides with that of $p(s | m v C)$ apart from deleting the contribution made by $v$.

(iii) The probability of the trial having vs. not having a displacement. The data $m$ and $v$ on a specific trial are also informative for assigning optimal relative weights to the estimate for $s$ obtained under the assumption of no displacement (case (i) above) and the estimate under the assumption of a displacement (case (ii) above). Intuitively, the larger the discrepancy between $m$ and $v$ of a given trial, the more evidence that they are not emanating from the same source, i.e., the more evidence for a displacement trial. This can again be made precise by the laws of probability, including Bayes rule:
The latter equation expresses how the probability of the trial outcomes $m$ and $v$ having a common source (no displacement) depends on a prior probability, independent of trial information, for common source trials, $p(C)$, and on the likelihoods of the obtained $m$-$v$ combination for no-displacement (common source) and displacement trials, $p(mv|C)$ and $p(mv|\bar{C})$, respectively. The first of these, the prior common source probability $p(C)$ is simply taken as a free parameter $p_c$ in the model, with $p(\bar{C}) = 1 - p_c$.

As for the $m$-$v$ likelihood in no-displacement trials, this can be mathematically obtained as the weighted average across all possible $s$ positions (Kording et al., 2007). Assuming independence, this can be done for two orthogonal directions separately. For the parallel (i.e. horizontal) direction, indexed by $x$, it follows:

$$p(m_x v_x | C) = \int p(m_x v_x | s_x) p(s_x) ds_x = \int p(m_x | s_x) p(v_x | s_x) p(s_x) ds_x$$

(5)

Given the Gaussian assumptions for $p(m_x | s_x)$, $p(v_x | s_x)$, and $p(s_x)$, this integral has an analytic solution (see Kording et al., 2007):

$$p(m_x v_x | C) = \frac{1}{2\pi \sqrt{\sigma_{m_x}^2 \sigma_v^2 + \sigma_{m_x}^2 \sigma_{\pi_x}^2 + \sigma_v^2 \sigma_{\pi_x}^2}} \times \exp \left[ -\frac{1}{2} \frac{(m_x - v_x)^2 \sigma_{\pi_x}^2 + (m_x - \pi_x)^2 \sigma_v^2 + (v_x - \pi_x)^2 \sigma_{m_x}^2}{\sigma_{m_x}^2 \sigma_v^2 + \sigma_{m_x}^2 \sigma_{\pi_x}^2 + \sigma_v^2 \sigma_{\pi_x}^2} \right]$$

(6)

An analogous equation can be derived for the $m$-$v$ likelihood in the orthogonal (i.e. vertical) direction ($y$), yielding $p(m_y v_y | C)$. The 2-D likelihood $p(mv|C)$ is then obtained as the product of these horizontal and vertical likelihoods.

As to the $m$-$v$ likelihood in displacement trials, we note that $m$ and $v$ are regarded independent, not connected by a common $s$, and thus their weighted averages across $s$ positions have to be computed independently (Kording et al., 2007). This amounts to:

$$p(m_x v_x | \bar{C}) = p(m_x | \bar{C}) \cdot p(v_x | \bar{C}) = \int p(m_x | s_x) p(s_x) ds_x \cdot \int p(v_x | s_x) p(s_x) ds_x$$

(7)

which given our Gaussian assumptions has again an analytical solution, now as
a product of two Gaussians (Kording et al., 2007):

$$p(m_x v_x | \bar{c}) = \frac{1}{2\pi\sqrt{(\sigma_{m_x}^2 + \sigma_x^2)(\sigma_v^2 + \sigma_{\bar{c}}^2)}} \exp \left[ -\frac{1}{2} \left( \frac{(m_x - \pi_x)^2}{\sigma_{m_x}^2 + \sigma_x^2} + \frac{(v_x - \pi_x)^2}{\sigma_v^2 + \sigma_{\bar{c}}^2} \right) \right]$$

In combination with the analogous expression for the vertical direction, we achieve

$$p(mv | \bar{c}) = p(m_x v_x | \bar{c}) \cdot p(m_y v_y | \bar{c}).$$

Figure 4.2. Mixture model. The pre-saccadic location of the NT (square) is reported after a transsaccadic displacement of 10° to the right. Objects in red represent visible targets; the white objects depict the veridical target locations. Representations of location estimates, modeled as 2D Gaussians, are shown as dark ellipses. (A) Before the saccade, all three objects are encoded with the foveal prior \(f\) (light grey blob) being centered at the triangle, the FT. After the saccade, the displaced target’s position and identity (NT here) are encoded with \(f\) now being centered at the saccade landing position. (B) Based on the NT’s pre-saccadic \(m\) and post-saccadic \(v\) representations, both biased by \(f\), the probability of a single stable object, \(p(C | mv)\), is computed. In case \(m\) and \(v\) are unrelated the best solution is to segregate and ignore \(v\). If \(m\) and \(v\) derive from the same object, the best solution is to integration all signals. (D) The two solutions in (C) are weighted according to the probability that \(m\) and \(v\) are related. The localization response follows from

$$p(s | mv) = p(s | mvC) \cdot p(C | mv) + p(s | mv\bar{C}) \cdot p(\bar{C} | mv).$$

4.2.6. Model fitting and evaluation

The model contains 15 free parameters to fit 2D localization data from 198 different conditions: 3 target positions (FT, ST, NT) x 11 displacement sizes (-5° to 5°) x 2 displacement directions (parallel, orthogonal) x 3 viewing times (50, 300, 1000 ms). Six parameters are used to estimate \(m\); three
parameters are used for $v$ (see Mixture Model). The remaining six parameters describe the priors: one for the foveal bias ($\sigma_f^2$), four for the $x,y$ position (allocentric) and anisotropic variance of $\pi$, and finally one for the general expectation of perceiving a common source ($p_C$). These parameters were fit to all localization responses simultaneously for each participant (mean: 2589 data points) using Matlab’s `fminsearch` with 1000 searches (random initial parameter values) per participant. In every iteration of the search process, each condition was simulated 10000 times. These distributions were then compared (using $0.1^\circ$ bins) to the actual localization data in order to estimate the likelihood of the data given the model. Across iterations, the parameters were adjusted until an optimal fit was reached, i.e., the loglikelihood was maximized.

4.2.7. Alternative models

The above mixture model assumes a causal inference process that is fully statistically optimal. Of course, it is questionable whether the brain can attain such absolute optimality. To test for this, we additionally fitted two variants of the mixture model, suboptimal in the statistical sense, following proposals by Wozny et al. (2010). These two alternative models use the same ingredients as the mixture model, but differ by the response rule applied. On each trial, given an estimate of $p(C|mv)$, the common-cause probability of the trial, this probability is not used for weighting the common-cause, $p(s|mvC)$, and no-common-cause, $p(s|mv\bar{C})$, distributions of the target as in Eq. (1), but for choosing one of these. While making such a forced choice is not optimal, the choice itself can be made in an optimal way and this constitutes the first alternative model (referred to as model selection): per trial just choose the more likely causal structure, i.e., if $p(C|mv) > 0.5$, choose $p(s|mvC)$ otherwise choose $p(s|mv\bar{C})$. The second alternative model (referred to as probability matching) amounts to one more step away from optimality: here the choice between the two causal structures is again guided by the common-cause probability of the trial, but now according to the principle of probability matching: with probability equal to $p(C|mv)$ choose $p(s|mvC)$ and with complementary probability $p(C|mv)$ choose $p(s|mv\bar{C})$.

The model fitting procedure for the two alternative models is identical to the one for the mixture model described above (e.g. same number of free
parameters) and log-likelihoods are compared to determine which model describes the data best for each individual participant.

### 4.3. Results

Participants were tested in a saccadic suppression of displacement task in which they had to indicate the pre-saccadic position of either the fixation target, the saccade target or a peripheral non-foveated target that was displaced parallel or orthogonal during a horizontal saccade (Figure 4.1). The displaced target was subsequently viewed for three different durations (50, 300 or ~1000 ms).

Figure 4.3 shows the performance of a typical participant, plotting the localization errors (red dots) of the three target positions (rows: FT, ST, NT) as a function of parallel and orthogonal target displacement, respectively, separately for the three post-saccadic viewing times. Blue shaded areas represent best-fit model predictions, and will be discussed below. Data points should fall along the horizontal dashed line if the participant correctly remembered the pre-saccadic target location and ignored the target displacement after the saccade. In contrast, if the position of the post-saccadic target (dashed diagonal line) interacts with memory for the pre-saccadic position of the target, the data should diverge from the horizontal line and linearly relate to the size of the target displacement.

The localization responses of this participant indicate a mixture of these two patterns. While localization errors become larger with increasing target displacements, beyond a certain target displacement they transition back to smaller errors. Thus, with increasing target displacement, there appears to be a shift in the proportion of responses that are contracted to the post-saccadic target vs. the ones that are unaffected by it. This pattern can be seen in all panels.

Figure 4.4 depicts the localization errors, averaged across participants. The pattern of localization errors is similar to the results of the single participant shown in Figure 4.3, particularly the bias toward the post-saccadic target for small displacements and the loss of this contraction for large displacements. Below, this will be interpreted as the outcome of a mixture model balancing integration and segregation processes, but this qualitative structure can already be confirmed by standard statistical analysis. The distinction between small and large displacements is not a sharp one, of course, and could, in a functional
sense, depend on target position, viewing time and direction of displacement. Therefore, we took for the following analyses the displacements with absolute value strictly smaller than 2° (0, ±0.5, ±1°) as “small” and the displacements with absolute values strictly greater than 2° (±3, ±5°) as “large”. (Replicating the analyses with the ±2° displacements added to either the “small” or “large” group turned out to yield very similar results.)

An analysis including the three targets (FT, ST, NT), the three viewing times (50, 300, ~1000 ms), the two directions (parallel, orthogonal) and the “small” displacements (0, ±0.5, ±1°), showed a significant positive linear effect of displacement on localization error ($F_{1,10} = 28.7, p < .001$). This effect was

![Figure 4.3. Performance of a single participant. Red dots represent localization responses and blue shaded areas represent the response probabilities, $p(s|mv)$, according to the best-fit model predictions. Left three columns, localization errors for parallel target displacements; right three columns, errors for orthogonal displacements. Horizontal dashed line represents veridical localization, i.e. the segregation strategy. Dashed diagonal line represents the displacement of the post-saccadic target. With small displacements, errors deviate toward the diagonal line for the three targets; this pulling effect appears stronger with longer viewing durations.](image-url)
present across targets, viewing times and directions, but it was moderated by these factors. For instance, post hoc comparisons revealed that the regression slope of localization error on displacement was less steep for FT trials than for ST and NT trials, the latter two not differing significantly. This is in line with the notion that because FT is initially foveated, it is represented more precisely than ST and NT, and therefore less influenced by its post-saccadic location. As for viewing time, the slope was generally less steep for 50 than for 300 ms, with no significant difference between 300 and ~1000 ms. Overall, parallel displacements produced a steeper slope than orthogonal ones. The moderating effects of viewing time and direction, however, were not present for all targets (a 2nd order interaction). For the FT, slopes were not significantly different across viewing times and directions, although they tended to be steeper for parallel than orthogonal displacements ($p = 0.06$). For ST trials, there was no moderating effect of time, but a very clear effect of direction ($p < .001$), with a steeper slope for parallel displacements. In contrast, the NT trials showed no moderation of the slope by direction, but they did show a very clear effect of time: here the slope was significantly steeper for ~1000 ms than for 300 ms ($p = .018$), as well as for 300 ms compared to 50 ms ($p = .025$). All in all, this makes for a complicated collection of results, which have in common across all conditions, however, a positive linear effect of small displacements on localization error.

This linear relationship between displacement and localization error does not extend to the large displacements. Choosing either the positive large displacements $3^\circ$ and $5^\circ$, or the negative displacements $-3^\circ$ and $-5^\circ$ revealed no effect of displacement on localization error ($p = .87$ and $p = .28$, respectively) in an analysis including the target, viewing time, and direction factors.

To explain these effects, we modeled the role of causal inference in the computations to obtain spatial constancy. Our principal model involves a statistically optimal mixture at the trial level of two possible causal structures on the signals available (see Methods and Figure 4.2). For each participant, the model was fit to all localization errors simultaneously. For the participant in Figure 4.3, the best-fit model is shown by the blue shaded curves. The shade intensity represents the model’s likelihood of localization errors ($p(s|mv)$). The model adequately predicts the positive slope in the errors as observed with small but increasing target displacements. This positive slope reflects the model’s weight on the assumption that the pre- and post-saccadic percepts

Causal inference for spatial constancy across saccades
originate from the same stable target (i.e. have a common cause), so they can be integrated to estimate a more precise but biased response (in the direction of the post-saccadic target). Along the same lines, the model also accounts for the effects of post-saccadic viewing time, the increase of which causes a more precise post-saccadic representation resulting here in a steeper slope in the localization error (i.e. a stronger contraction or pull to the post-saccadic target). Finally, the model infers that for large target displacements, the pre- and post-saccadic percepts likely stem from different causes, for which it is optimal to not integrate but rather disregard the post-saccadic percept. As a result, the probability of a localization response toward the displaced target decreases, which matches with the transition to smaller errors as observed in the data.

Figure 4.4. Mean localization errors across participants. Mean responses are shown as dots (error bars, SEM) and mean model fits as continuous lines (shaded areas, SEM). Format as in Figure 4.3.
The continuous lines in Figure 4.4 depict the best-fit predictions from the model, averaged across participants. As shown, these curves display a good correlation with the localization errors ($R^2 = .65 \pm .06$ and $R^2 = .85 \pm .03$ for the parallel and orthogonal direction, respectively, across participants; see the section Mixture Model for details about the fitting procedure).

The best-fit parameter values (see Table 4.1) give insight in the precision with which the target positions are recovered from memory when computing the localization responses ($\sigma_m$; see Figure 4.5A). A two-way analysis on the $\sigma_m$ values revealed significant effects of both target ($F_{2,9} = 31.9$, $p < .001$) and displacement direction ($F_{1,10} = 5.2$, $p = .045$), as well as a significant interaction effect ($F_{2,9} = 25.9$, $p < .001$). The interaction is expressed by the finding that this effect is mostly driven by the orthogonal displacements (see Figure 4.5A). Post hoc comparisons revealed that NT is memorized with a lower precision than FT and ST. Thus, while both ST and NT are viewed in the periphery before the saccade, ST is memorized with higher precision than NT. No significant difference was found between the estimated parameters for FT and ST.

Figure 4.5B depicts the model’s prediction of the precision of the post-saccadic target ($\sigma_v$) for the three viewing times. Here the effect of viewing time is significant ($F_{2,9} = 7.5$, $p = .012$) and, as expected, post hoc comparisons reveal precision to improve (lower sigma values) both from 50 to 300 ms viewing ($p = .004$) and from 300 to ~1000 ms viewing ($p = .008$).

Table 4.1
Best-fit parameter values for all eleven participants. All values are in degrees except probability $p_C$. Position of $\pi$ is expressed relative to FT.

<table>
<thead>
<tr>
<th>$\sigma_{mx}$</th>
<th>$\sigma_{my}$</th>
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Causal inference for spatial constancy across saccades
As the mean data show, there are also errors in the absence of any target displacement. The model explains this by the combined effect of the foveal prior ($\sigma_f = 4.6^\circ \pm 0.27^\circ$, mean $\pm$ SEM) and the allocentric prior $\pi$. The location and precision of the allocentric prior are plotted in Figure 4.5C, showing that it is centered in between the three target locations, and has a substantial width ($\sim12^\circ$) compared to the inferred precision values of both the remapped, pre-saccadic target representations (Figure 4.5A) and post-saccadic information (Figure 4.5B).

![Figure 4.5](image)

**Figure 4.5.** Mean parameters of best-fits. (A) Average $\sigma_m$ across participants (error bars, SEM). The orthogonal component of the memorized positions appears to be more precise than the parallel component for FT and ST, but not for NT. (B) Average $\sigma_v$ across participants. Variability of the post-saccadic-target representation decreases as a function of viewing duration. (C) Prior $\pi$, positioned relative to FT, representing where objects are generally expected to appear. All values are in degrees.

Finally, in the model, the general degree by which participants’ localization responses were influenced by the displaced target is captured by parameter $\mathbf{p}_C$, which represents the prior probability that the target remains stable. Its value was on average 0.45 ± 0.1 (mean $\pm$ SEM), but Table 4.1 shows that this parameter varied substantially among the 11 participants. This prior in combination with the information of $m$ and $v$, results in a posterior probability that the target has not moved, $p(C|m,v)$, as a function of target displacement.

Figure 4.6 shows that the average $p(C|m,v)$ is close to one for small displacements, suggesting integration of pre- and post-saccadic targets. For
larger target displacements, the curves fall off, suggesting more evidence that pre- and post-saccadic representations stem from different sources. The curves also illustrate the effect of viewing time: when the post-saccadic target is viewed only briefly, inferring causality becomes more difficult, resulting in a more gentle decline of $p(C|mv)$ with increasing displacements.

The above results follow from fits of a mixture model that assumes a causal inference process that is fully statistically optimal. For comparison, we also fitted two variants of this model, model selection and probability matching (see Methods). The models differ by the response rule applied (see Methods). Across our participants, on average the log-likelihood differences of these models with the mixture model were $344 \pm 124$ and $125 \pm 49$, respectively, indicating that the mixture model (average log-likelihood -17262) outperforms

![Figure 4.6](image-url)

**Figure 4.6.** Inferred probability of a common cause $p(C|mv)$ as a function of target displacement. Probabilities are based on the best-fit parameters, separated by target location (rows: FT, ST, NT), displacement directions (columns: parallel/orthogonal) and post-saccadic viewing duration (in color). Shown are the mean values across participants and standard error (shaded areas). This probability, which can be interpreted as the complementary probability of perceiving the displacement, optimally weights the integration and segregation strategy.
its variants. Since the three models share the same parameters, using an AIC or BIC instead of the log-likelihood criterion in the model comparison would not change this conclusion. For one participant (number 6) no clear difference between the mixture model and model selection was found (log-likelihood difference < 3); for two other participants (number 9 and 11), a probability matching strategy was ranked before the mixture model.

4.4. Discussion

In the current study we modeled and tested the role of causal inference in the computations for spatial constancy across saccades. According to our model, the brain has to estimate whether pre-saccadic and post-saccadic signals reflect a stable or an unstable visual world, which depends on the spatiotemporal difference between these signals and on their precision. We operationalized the problem experimentally by using the saccadic suppression of displacement paradigm. Participants viewed three targets, with one of them the fixation point, the other the saccade target and the third a peripheral target. After the saccade, one of these three remained for different viewing durations, but often at a slightly displaced position, and participants had to indicate which location it had prior to the saccade. Our results show that: 1. the integration of the pre- and post-saccadic target positions declines as a function of their spatial separation, 2. different targets show different strengths of SSD, and 3. viewing time of the post-saccadic target changes the strength of SSD. Our model could account for all these findings, which will now be discussed in more detail.

We replicated the non-linear localization response pattern previously reported by Niemeier and colleagues (2003), but modeled it in a different way. Sensory signals are inherently noisy. This means that even in the case of a completely stable world the pre- and post-saccadic percepts may show some false discrepancy which should be ignored by the brain. In the model of Niemeier and colleagues a spatial window of stability is created by integrating a displacement vector (i.e. the visual discrepancy) with a prior centered at zero displacement. This predicts that localization is pulled to the post-saccadic target, irrespective of the size of the displacement. The present model goes a step further, and considers this pulling effect from a causal inference perspective, stating that pre-saccadic and post-saccadic percepts should be integrated when their discrepancy is relatively small but should be segregated
when the displacement increases. More specifically, it infers the probability
that a common cause underlies the pre- and post-saccadic percepts. The
model dealt with these considerations in an optimal manner, i.e. on any
trial it applied a mix of both integration and segregation, each weighted by
its respective probability as based on the precision of both percepts, thereby
minimizing quadratic error in the long run. Of course, there are alternative
forms by which the brain could process the inference about the common cause
(see Wozny, Beierholm, and Shams, 2010). For example, the brain could also
select per trial which causal structure is most likely, and accordingly process
the trial in a binary fashion either by integration or by segregation. In most
participants, we found that our weighted averaging model better described the
data than a model involving binary selection or a model based on the principle
of probability matching.

In the comparison of the fits of the three models described, Wozny et al.
(2010) found the last and least optimal variant, probability matching, the clear
winner in a multisensory perception experiment. It must be noted, however,
that our experimental setting differs principally from that of Wozny et al. and
of other applications of the mixture model known to us (Kording et al., 2007;
Rohe and Noppeney, 2015). They deal with multisensory perception, where
bimodal cues (typically auditory and visual) are available to be combined if
there is evidence they belong to the same object, even though each unimodal
cue is in itself sufficient to solve the task (e.g., localize an object). Data for
either unimodal condition (just the auditory cue or just the visual cue) can be
obtained without changing the task. In our case, there are two complementary
representations in one modality (vision) and a division in an experiment with
“just the pre-saccadic remapped memory information” and one with “just the
post-saccadic visual information” is not sensible. Consequently, the outcome
of the model comparison might well be different for our case.

As predicted by our model, we found strong integration when the target
displacements were small, characterized by low response variability but large
biases toward the post-saccadic target. Increasing the size of the displacement
lowers the probability of a common cause (Figure 4.6) which results in smaller
localization errors (Figure 4.4). The inferred probability of a common cause
can directly be interpreted as the strength of SSD. As shown previously (e.g.
McConkie and Currie, 1996), displacements up to one third of the saccade
amplitude typically show strong SSD. However, we have found differences in
the strength of SSD between targets and displacement directions.

We showed that the differences in strength of SSD between targets reflect differences in the precision of the pre-saccadic target representations upon recall. The regression analysis suggests that FT is represented more precisely than ST and NT, while the model fits showed that both FT and ST were represented more precisely than NT. We lack a clear explanation for this difference, but as shown in Figure 4.4, the model generally underestimates the pulling effect of ST and overestimates this for FT. For both FT and ST, localization is better with orthogonal than parallel target displacements, which can be explained by the anisotropy in the precision of their memories. This anisotropy may result from the noisy eye position signals that are used to remap the target representation across saccades (Niemeier, Crawford, and Tweed, 2003). Indeed, our participants showed about twice as much scatter in the saccade end points in the direction of the saccade than orthogonal to it (1.27 ± 0.05° and 0.73 ± 0.03°, respectively, mean standard deviation ± SEM). The estimated parameters of the mixture model indicate that memory precision of FT and ST is also about two times worse parallel than orthogonal (see Table 4.1), which suggests that noise sources related to eye position sense play a role in the coding of these representations (Niemeier, Crawford, and Tweed, 2003). The memory of NT, which we found to be less precise than ST and FT, appears to be more variable in the orthogonal than along the saccade direction. Although we cannot explain all the differences in the strength of SSD among the three targets, an important factor may relate to how the brain has coded the visual scene in memory, which we will discuss next.

It has been suggested that across saccades the brain stores a structural description of the target display in memory (e.g. Carlsonadvansky and Irwin, 1995). For example, in a task where participants have to remember a pattern of dots, it was shown that the relative positions of the dots could be recalled independent of absolute spatial information (Irwin, 1991). After a saccade, the saccade target could serve as an anchor to which the structural description is related (McConkie and Currie, 1996; Irwin and Robinson, 2014; Currie et al., 2000). Connecting this finding to the present experiment suggests that participants encoded the equilateral triangle constituted by the three targets. In our experiment, however, the majority of trials had no ST present after the saccade. If the structural description of the target display would then be anchored to the eyes’ landing position instead, it would predict a positive...
relationship between the saccade landing error and localization error. Indeed, we found a small but significant correlation for ST in almost all participants (mean $r = .18$). In the same vein, this notion could also explain why the ST was recovered with higher precision from memory than NT although both were pre-saccadically presented at equal eccentricities. If participants indeed stored a structural description as an equilateral triangle, there may be some variability in the size of the triangle from trial to trial. This variability would bear out in more response variability in the orthogonal direction of NT, as we have found. Furthermore, previous work has shown that a group of random static dots are typically remembered closer to each other than they actually were (Sheth and Shimojo, 2001; Dent and Smyth, 2006), like our participants did. Our model explains this observation using an allocentric prior, positioned at about the center of the target display, albeit with some variability among participants. This is consistent with current models of efficient coding in visuospatial memory, which propose that people code a display in terms of summary characteristics, such as its center of mass (e.g. Alvarez and Oliva, 2008; Ariely, 2001).

Despite relative coding accounts, as described above, there is also ample evidence that the brain keeps target representations in a dynamic register (for a review see Wurtz, Joiner, and Berman, 2011). These representations, coded in eye-centered coordinates, must be updated when the eyes move. In support, several brain regions have been identified that contain neurons with visual receptive fields (RFs) that are normally fixed to one position of the retina but briefly shift in anticipation of a planned saccade to the position the RF will occupy after the saccade (e.g. Duhamel, Colby, and Goldberg, 1992; Walker, Fitzgibbon, and Goldberg, 1995; Nakamura and Colby, 2002). Although it is currently unknown how the brain transfers object information across shifts of the RFs, it could be an important mechanism in order to achieve space constancy (e.g. Cavanagh et al., 2010). In our experiment, the three target representations would be shifted in the opposite direction of the upcoming saccade. After the saccade a one-to-one comparison can be made between the post-saccadic retinal input and the predicted input to assess visual stability. It could be hypothesized that in anticipation of a saccade a given receptive field shifts in the accurate direction but with a less accurate amplitude. This seems plausible given that saccades to a target typically show more variability in amplitude than direction. While this would be consistent with the observed SSD differences for FT and ST, this is not the case for the NT. VanRullen
(2004) has argued that while the visual world translates homogeneously during a saccade, its cortical representation does not because the amount of cortex dedicated to a certain sized patch of the retina varies, especially as a function of retinal eccentricity. One possibility is that these non-homogenous shift of RFs introduces noise orthogonal to the saccade in the periphery, which may explain our results for the NT. A precise mapping of shifting RFs would be needed to test this hypothesis.

Alternatively, one could speculate that the observed differences between target locations reflect distortions due to RFs that shift not in parallel but towards the ST in anticipation of a saccade (Zirnsak et al., 2014; Tolias et al., 2001). Although it has been suggested that this anticipatory transient increase in density of receptive fields around the saccade target underlies the boost in attention around the ST area, and thus is beneficial for space constancy for that target, it may be that the encoding of peripheral targets becomes distorted because of these RF shifts. The representation of a target like NT may become stretched or displaced towards the ST, resulting in a compressed memory. Future research should investigate whether these RFs do indeed distort perception.

In our experiment, we not only displaced the target but also manipulated the post-saccadic viewing time. In general, longer viewing increases its pulling effect on the localization response. Recently, Zimmerman et al. (2013) performed a SSD task in which the viewing time of the pre-saccadic target was varied. They showed that when the pre-saccadic target is briefly viewed, i.e. < 0.5 s, displacement detection performance is low. Here, we modeled viewing time as a factor that changes the precision of the target representation. Indeed, the longer the target was visible, the higher its precision. In terms of our model, the viewing time manipulation by Zimmerman and colleagues would affect $\sigma_m$ which in turn affects the probability of perceiving a common cause $p(C|mv)$. In other words, the system is generally more likely to integrate when the representation of the pre-saccadic target is noisy, hence displacement detection performance is low. In our experiment, decreasing the viewing time of the post-saccadic target did generally lower the detection performance as well (i.e., increase $p(C|mv)$). The latter may not be directly obvious from the localization responses which show the strongest pulling effect with the longest viewing duration. The explanation is as follows. Although the integration strategy receives less weight with long viewing, the post-saccadic target
representation is more precise, which has an opposite effect and ultimately pulls localization towards it.

A final point of discussion relates to model parameter $p(C)$, which represents the a priori probability that the world remains stable. We found a considerable variability among participants for this parameter. In most participants, the $p(C)$ estimates can be regarded low, given that in daily life objects rarely jump while we scan the world. We consider it plausible that the experimental context and task instruction, which explicitly mentions the possibility of displacements, alters $p(C)$. For example, if you know beforehand that a certain scene will contain a lot of instability, it seems logical to lower $p(C)$ and thus become more skeptical regarding the feasibility to integrate percepts.

Taken together, we showed that integration of the pre- and post-saccadic target representations can be modeled using principles of causal inference. When representations follow from spatially close target locations, integration is strong. In contrast, when targets are further apart, integration weakens, depending on precision of involved representations.
5. Summary & General Discussion
5.1. Summary

The goal of this thesis was to obtain a better understanding of the processes involved in visual stability and movement cancelation. In Chapter 2 we studied peri-saccadic mislocalization, a phenomenon linked to retinal remapping of object locations. We investigated whether this remapping can be initiated by the mere preparation of a saccade. We found no mislocalization with canceled saccades, supporting the notion that saccade execution is a prerequisite for remapping. A problem with cancelled saccades is that the moment of cancellation cannot be determined directly. Therefore, in Chapter 3 we again studied movement cancelation, but now regarding arm reaching movements. We hypothesized that in contrast to saccades, where the stop signal reaction time can only be roughly estimated, countermanded arm reaching movements involve antagonist muscle activity that actively brakes the movement, which may directly reflect the stop signal reaction time. Our data support this notion: the onset of antagonist recruitment following a stop signal can be regarded as a within-trial stop signal reaction time. In Chapter 4 we investigated the computations involved in visual stability using a saccadic suppression of displacement task. We found that we could model the behavior based on Bayesian decision theory, meaning that the brain estimates the causal relationship between pre-saccadic and post-saccadic signals to establish to what degree they can be integrated or when they should be kept apart. Below a more detailed summary of each study is given, followed by some concluding remarks.

5.2. No peri-saccadic mislocalization with abruptly cancelled saccades

Every saccadic eye movement that we make changes the image of the world on our retina. Yet, despite these retinal shifts, we still perceive our visual world to be stable. Efference copy from the oculomotor system to the visual system has been suggested to contribute to this stable percept, enabling the brain to anticipate the retinal image shifts by remapping the neural image. A psychophysical phenomenon that has been linked to this predictive remapping is the mislocalization of a stimulus flashed around the time of a saccade. If this mislocalization is initiated by saccade preparation, one should also observe
localization errors when a saccade is planned, but abruptly aborted just prior to its execution. We tested this hypothesis with human subjects using a novel paradigm that combines a flash localization task with a countermanding component that occasionally requires saccade cancellation. Surprisingly, we found no trace of mislocalization, even for saccades cancelled close to the point of no return. This strongly suggests that the actual execution of the saccade is a prerequisite for the typical localization errors, which rejects various models and constrains neural substrates. We conclude that peri-saccadic mislocalization is not a direct consequence of saccade preparation but arises after saccade execution when the flash location is constructed from memory.

5.3. Neuromuscular markers of movement cancellation in reach control

Movement inhibition is an aspect of executive control that can be studied using the countermanding paradigm, which requires subjects to try to cancel an impending movement following presentation of a stop-signal. Success can be modeled as a race between a GO process, initiated by the target, and a STOP process, initiated by the stop signal. Most studies in the literature have examined fast or ballistic movements even though many movements in daily life are non-ballistic, and under control throughout their entire trajectory. A potential benefit in studying the control of non-ballistic movements is that antagonist muscle recruitment used to stop the movement mid-flight may explicitly mark the finish time of the STOP process. Studies on canceling orienting head movements support this hypothesis, showing that antagonist neck muscle recruitment aligned best to stop signal onset and co-varied with conventional estimates of stop signal reaction time (SSRT) both within and across subjects (Goonetilleke, Doherty, and Corneil, 2010; Goonetilleke, Wong, and Corneil, 2012). Here, human participants performed a center-out reaching task with a countermanding component. We used intramuscular electromyography to record from upper-limb muscles contributing to movement generation and braking. The data show a clear response in the motor periphery to a stop signal even with movements that have barely begun. Congruent to the head movement work, we find that antagonist recruitment timings co-varied with SSRT estimates both within and across subjects. Furthermore, we observed that antagonist recruitment was influenced by immediate trial history,
something that conventional SSRT analyses cannot show. These data strongly support the notion that the onset of antagonist recruitment following a stop signal, the antagonist latency, can be used as a within-trial measure of the stop signal reaction time.

5.4. Causal inference for spatial constancy across saccades

Our ability to interact with the environment hinges on creating a stable visual world despite the continuous changes in retinal input. To achieve visual stability, the brain must distinguish the retinal image shifts caused by eye movements and shifts due to movements of the visual scene. This process appears not to be flawless: during saccades, we often fail to detect whether visual objects remain stable or move, which is called saccadic suppression of displacement (SSD). How does the brain evaluate the memorized information of the pre-saccadic scene and the actual visual feedback of the post-saccadic visual scene in the computations for visual stability? Using a SSD task, we test how participants localize the pre-saccadic position of the fixation target, the saccade target or a peripheral non-foveated target that was displaced parallel or orthogonal during a horizontal saccade, and subsequently viewed for three different durations. Results showed different localization errors of the three targets, depending on the viewing time of the post-saccadic stimulus and its spatial separation from the pre-saccadic location. We modeled the data through a Bayesian causal inference mechanism, in which at the trial level an optimal mixing of two possible strategies, integration vs. separation of the pre-saccadic memory and the post-saccadic sensory signals, is applied. Fits of this model generally outperformed other plausible decision strategies for producing SSD. Our findings suggest that humans exploit a Bayesian inference process with two causal structures to mediate visual stability.

5.5. Point of no return in motor control

With reflexive movements, the time between stimulus and response – the reaction time (RT) – is relatively small and fairly constant. With voluntary movements, however, RTs are typically longer and more variable because more deliberations are involved. Given the stimulus, the brain takes time to
decide how and when a goal is achieved best given the current circumstances. This requires that the stimulus is not perceived as a distinct entity, but is incorporated in its context, and is evaluated in relation to our current state of body and mind. Many factors affect RT. When in a state of arousal, for example in a life-threatening situation, decisions regarding movement execution may be made more rapidly than in a state of relaxation.

Motor control is also thought to be hierarchical, meaning that the abstract movement goal ("I want to go there") is transformed into concrete and specific muscle commands ("muscle X will be activated at time T...") following some hypothetical stages. Somewhere along this hierarchy, the movement plan is progressed so far that it passes a point of no return: muscles will be recruited no matter what. Indeed, the point of no return appears to be located after perception, at the level of response selection (see Logan, 2015 for a review).

Our findings in Chapter 2 suggest that the execution of a saccade and the remapping of the retinal image to correct for the visual perturbations induced by the saccade have a common underlying point of no return. We found peri-saccadic mislocalization effects, a behavioral marker of a neural remapping process, only to occur when one is committed to the execution of the saccade. Would this suggest that there is a point of no return for saccadic eye movements?

Interestingly, in Chapter 3, we found little evidence for a point of no return in arm reaching movements. We observed that reaching movements could be stopped mid-flight. To my surprise, even on a substantial amount of successfully canceled trials, agonist and antagonist muscles were subsequently activated. This suggests that on all stages of the motor control hierarchy, including the very last stage during which the muscles are activated, the movement can still be aborted. This is in line with earlier studies that tried to determine the point of no return with hand movements (De Jong et al., 1990; Ko, Alsford, and Miller, 2012). These studies showed that the point of no return for squeezing movements and key presses is also very late, even after movement initiation.

With this in mind, is there a true point of no return for saccades? Typically, the point of no return for saccades is estimated by determining the time at which the saccade plan cannot be altered anymore by some stimulus prior to saccade execution. It has been found that when the saccade target object is displaced up to ~60 ms before saccade onset, the saccade could be
influenced by this new target position (Findlay and Harris, 1984; Ludwig et al., 2007; Walshe and Nuthmann, 2015). Furthermore, when a large distracting flash is presented during saccade preparation, the flash appears to inhibit the execution of saccades 60-70 ms after flash onset (e.g. Reingold and Stampe, 2002). This would lead to the conclusion that for saccades, the point of no return is around 60–70 ms before onset. However, a recent study showed that a pre-saccadic flash is able to decrease the amplitude of a saccade (by up to 15%) even when the saccade was launched only 20 ms after the flash (Buonocore, McIntosh, and Melcher, 2016). Given that visual information can reach the SC within 35–47 ms (Rizzolatti et al. 1980), the authors suggest that the omnipause neurons may have been stimulated by the flash, effectively stopping the saccade mid-flight. While the saccade may not be fully canceled, this finding by Buonocore et al. suggests that some form of control is still possible after saccade. Thus, saccades are not ballistic (see also Corneil et al., 1999). Returning to the question whether a point of no return exists for saccades, the answer would be probably yes. But, like with arm movements, a point of no return (if any) is at a very late stage.

5.6. Anticipation for something that never comes?

If the point of no return is very late with saccades, till 20 ms before a saccade would actually take place, why would receptive fields start to shift already 100 ms before saccade onset? I would like to speculate on this paradox a little here. Sommer and Wurtz (2008) argued that receptive fields should shift only if the generation of the saccade is inevitable. Their argument follows from the intuition that when a saccade is canceled prior to execution, remapping would be counterproductive as it would create visual instability. An alternative notion that I would like to put forward is to regard the shifting receptive fields as a form of mental imagery. That is, what would I see if I moved my eyes there. It is known that visual imagery recruits visual areas (e.g. Ganis, Thompson, and Kosslyn, 2004; Slotnick, Thompson, and Kosslyn, 2005), and when one imagines making a movement, the predicted consequences of the imagined movement can be readily dissociated from the actual current state of the body. I would therefore hypothesize that RFs shift to create a hypothetical future state without yet committing to the underlying movement plan. The function of the shifting RFs may therefore be twofold: first, to create a mental image
that can be exploited to plan subsequent (eye) movements if the saccade is committed, and second, to compare remapped object locations after the saccade to detect transsaccadic changes. In this light, it would make sense that the RFs shift relatively early in the planning phase, because this leaves enough time to reevaluate the movement plan.

This begs the question for why we did not find peri-saccadic mislocalization without saccades. Continuing my speculation following this "two-fold" hypothesis, it is possible that peri-saccadic mislocalization is a product of only the second function of shifting RFs: to compare the predicted retinal image with the actual post-saccadic retinal input after the saccade was committed. Without a saccade, the RFs may have shifted briefly during consideration of the saccade, but this shift was not yet used to create visual stability. In the Discussion of Chapter 2, we discussed the visuomovement neurons in FEF as observed by Ray et al. (2009). These cells showed diverging activity around 40 ms before saccade onset, and only when a saccade was carried out. Possibly this signal then causes the transition from the hypothetical reality (function one) to the expected reality (function two). I think it would be interesting to test this hypothesis, for example by combining neurophysiological recordings of shifting RFs with a countermanding paradigm. In the following paragraph I will suggest a purely behavioral experiment.

5.7. Future Research

I speculated that the initial role for shifting receptive fields is not for visual stability per se, but to create a mental image that can be exploited to plan subsequent (eye) movements if the saccade would be committed. Interestingly, the findings of a behavioral study by Rolfs et al. (2011) could be interpreted this way, and adding a countermanding-like component would provide a test of this hypothesis.

Recall that briefly before a saccade starts, attentional performance near the saccade target location is typically enhanced (see General Introduction). Rolfs et al. found that this pre-saccadic attentional enhancement also shifts to those retinal locations that targets would cover after the saccade. For a single saccade, this means that the attention boost at the saccade target shifts to the fovea, because the fovea would be covered by the saccade target after the saccade. More interesting, however, is the case of a double-step saccade: if the
saccade sequence would be first rightward - and then upward, then the findings by Rolfs et al. predict that before the first saccade would begin, attentional enhancement will be found at the location straight above the fovea: the retinal location where the second saccade target will be located after completing the first saccade. They argue that because of the updating process that predicts the retinal consequence of the first saccade, the remapping of the second saccade target gives rise to the attentional enhancement at this otherwise irrelevant spot in space.

The authors note that one function of the pre-saccadic remapping is likely to plan a subsequent saccade. It would be interesting to test what would happen in case two movement plans compete. Possibly, two "hypothetical realities" are considered simultaneously which would give rise to presacacdic attentional enhancement at two otherwise irrelevant spots in space. If this is true, and attentional remapping is found relating to the discarded movement plan, this may suggest that remapping is not solely for post-saccadic visual stability.

5.8. The point of no return in transsaccadic integration

In Chapter 2 we found no peri-saccadic mislocalization with prepared but abruptly canceled saccades. We interpreted this finding as showing that transsaccadic updating of location information commences only after the point of no return. In Chapter 3 we observed that the point of no return for reaching movements is very late, after the response began, and that antagonist muscle recruitment signifies the "point of return" as it appears to be a proxy of the stop signal reaction time. In Chapter 4, one could argue that we also studied a point of no return, but not for action but for perception. When an object was quickly displaced during a saccade, the brain decides whether the mismatch between prediction and retinal input is due to internal or external noise. Once this (unconscious) decision is made, there is no going back anymore. A striking example related to our study is the McGurk illusion regarding audiovisual speech recognition (you can find several examples on Youtube). In this illusion, sound is altered by visual information (McGurk and MacDonald, 1976). Once the brain decides that vision and sound originate from the same source, they are fused or integrated into one percept, and you cannot separate them anymore. Likewise, we showed that the memory of the pre-saccadic location an object is altered by its post-saccadic location.
This effect was successfully modeled using Bayesian causal inference. In this model, whether a pre-saccadic and post-saccadic position signal reflect a stable or an unstable object was estimated based on the spatiotemporal difference between these signals and on their precision. This stability estimate, expressed as $p(C|mv)$, is a probability: for example, given the remembered pre-saccadic location and the perceived post-saccadic location of an object $p(C|mv)$ may equal .3 which means the probability of an object displacement is judged to be 70%. We showed that when participants were asked to indicate the remembered location, they did not discard the post-saccadic location completely but instead based their response for 30% (following the example) on the integrated (pre- and post fused) estimate and 70% on the segregated estimate. Although it may intuitively seem better to strictly choose either segregation or integration per trial, participants seem to have applied a weighted average of both segregation and integration, which is actually statistically most optimal. It is statistically most optimal, because it minimizes the mean squared error of the spatial estimates in the long run. Assuming that our findings are sound, I think it is fascinating that evolution has built a brain that deals with uncertainty in such an elegant manner.

5.9. Alternative (unconscious) decision strategies

In Chapter 4, we found that the majority of participants used a weighted average of integration and segregation. Interestingly, there are also reports in the literature that show that people sometimes rely on other types of decision strategies. For example, in a study by Wozny, Beierhold and Shams (2010), participants had to indicating the perceived location of an auditory and visual stimulus that were presented together in time, but at different locations in space. They found that most participants did not adopt a weighing strategy as we found in our study, but instead relied on a decision strategy called probability matching. In this alternative strategy, on each trial either complete segregation or integration is chosen, but this choice is not based on the most likely causal structure (common cause = integration versus separate causes = segregation). Instead, the choice is based on the probability of the causal structure, meaning that in case $p(C|mv)$ was 30%, integration is chosen 30% of the time and segregation is chosen 70% of the time. This seems very irrational and suboptimal.
Why is probability matching suboptimal? Imagine that you participate in a game with multiple rounds in which you have to guess each time which one of two face-down playing cards has a higher value. Imagine further that each time the two cards are drawn from two separate piles, pile A and pile B, and that cards from pile B win 70% of the time, which you will learn after playing a few rounds. Given that the cards are drawn from each pile at random, the best strategy would be always to pick the card drawn from pile B, because then you would win 70% of the time. However, it appears that people often do not do this, but instead apply probability matching, meaning that they will pick the card from pile B in 7 out of 10 times (with a success rate of \(0.7 \times 0.7 + 0.3 \times 0.3 = 58\%\)). Why would that be? Gaissmaier and Schooler (2008) argued that people are generally poor in judging whether a sequence of events is completely random. Instead, people often try to find non-random patterns in order to be more successful at the task at hand, which is directly causing the probability matching because that helps in finding the pattern.

In the abovementioned audiovisual localization task, the authors argued that exploration and pattern finding was likely the reason why their participants adopted the probability matching strategy. Why did our participants preferred the averaging strategy over probability matching? Possibly our participants were more inclined to believe that they were facing real randomness in the stimulus conditions. One reason why this could be the case is that we included a very large number of conditions (e.g. three targets with each four displacement directions), which discourages pattern-finding. Thus, in designing future experiments it may be very important to consider the possibility that participants will try to find regularities in the stimuli which may give rise to unexpected behavior. That is, the behavior may be interpreted as suboptimal by the experimenter, while in fact it may be optimal when one considers a pattern-finding strategy.

5.10. Bayesian causal inference: a brain’s theory of everything?

Given that we found optimal integration of location information across saccades, are other object attributes such as color, orientation, or form also transaccadically integrated? It has been shown that the perceived color of a post-saccadic stimulus was influenced by the to-be-ignored color it had
before the saccade (Wittenberg, Bremmer, and Wachtler, 2008). Another study changed the reliability of the pre- or post-saccadic color, by overlaying a variable amount of colored grain (Oostwoud-Wijdenes, Marshall, and Bays, 2015). Indeed, the authors found that color information was integrated in an optimal way. However, in this study, the researchers only slightly changed the color across a saccade, which will evoke integration instead of segregation as predicted by Bayesian causal inference. Probably, when the discrepancy in colors is rather large, there will be less or maybe no integration at all. Perhaps, people use a weighting strategy that incorporates both integration and segregation per judgment, or probability matching. Another alternative is that people may choose integration or segregation based on which causal structure is more likely. This would be an interesting question for future research.

Regarding the orientation of an object, there have also been reports that orientation information is remapped (e.g. Melcher, 2007), and integrated across saccades (Granmor, Landy, and Simoncelli, 2015). In the study by Granmor et al. (2015) it was found that although the pre- and post-saccadic stimulus orientations were combined by linear integration, people generally displayed a systematic overweighting of the fovea. Again, in this study, principles of causal inference were not taken into account, and possibly the high resolution of the fovea triggered segregation instead of integration.

The integration of form across saccade has also been studied. In the study by Demeyer et al. (2010), an ellipsoid saccade target was elongated or shortened during a saccade. The authors reported that people combined the pre- and post-saccadic forms, but this did not reduce variance as you would expect from optimal integration. One problem is that shapes do not tend to be very constant on the retina when the observer or the object moves. Thus, for object tracking the shape of the object may play only a minor role. Possibly, when instead an action needs to be performed with the target object (e.g. grasping it), there may be value in integrating its shape across saccades to form a more precise motor plan. This hypothesis could be tested in for example a virtual reality environment in which a saccade triggers a shape-shift of a to-be-grasped object. Note that in all these future studies, causal inference should be taken into account because fusion of two percepts may depend on their discrepancy.
5.11. Conclusion

The highly dynamic world we live in challenges our perceptions and actions. Relevant stimuli may appear, disappear, or move, which may render certain actions to be (in)appropriate through time. Our actions themselves also add to this complexity as they change the (apparent) state of the world, and perturb inputs to our perception systems. In this thesis we explored how the human brain deals with this uncertainty, regarding arm movements and in particular regarding saccadic eye movements. The image shift that a saccade induces is anticipated by making a prediction of the post-saccadic image through space and time. The process of fusing this prediction with the actual post-saccadic visual input appears to follow rules that can be regarded as statistically optimal. Furthermore, to cope with a rapidly changing environment, the brain is able to withhold a movement until the very last moment, which creates a large flexibility. When a saccade was withheld just before its execution, perception was not affected by any prediction of the post-saccadic image, suggesting that this flexibility in motor control does not compromise visual stability. This shows that millions of years of evolution has created a brain that is both very flexible and accurate in perceiving and acting in a highly dynamic world.
6. Appendix
6.1. References


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6.2. Nederlandse Samenvatting

Dynamische sensomotorische integratie: implicaties voor het annuleren van beweging en visuele stabiliteit

Een mens maakt op een dag tienduizenden oogbewegingen om in onze rijke visuele omgeving relevante details goed te kunnen zien. Deze typische snelle oogbewegingen worden saccades genoemd. Tijdens iedere saccade schiet het beeld van de wereld over je netvlies. Ondanks zo'n plotselinge verandering blijf je de wereld als stabil waarnemen. Dit in tegenstelling tot wanneer je met je vinger voorzichtig je oogbol laat bewegen. Hoe kan dit?

De vraag 'hoe wordt visuele stabiliteit gecreëerd' is al eeuwenoud. Recente neurowetenschappelijke bevindingen suggereren dat ons brein dit doet door op de visuele consequenties van saccades te anticiperen: voordat een saccade begint wordt voorspelt hoe deze de waarneming zal gaan veranderen. Na de saccade wordt vervolgens aan de hand van deze voorspelling bepaald of de objecten nog op dezelfde plek staan, of zijn verplaatst (zie hoofdstuk 1 van dit proefschrift).

Een doel van dit proefschrift is om de processen rondom visuele stabiliteit beter te begrijpen. Daarnaast wordt het annuleren van een geplande (oog)beweging onderzocht. Immers, als het brein anticipiert op een geplande beweging, dan heeft het abrupt annuleren van die beweging consequenties: voorspellingen moeten wellicht op het laatste moment worden herzien.

In hoofdstuk 2 van dit proefschrift hebben we peri-saccadische mislokalisatie bestudeerd, een fenomeen dat in verband wordt gebracht met de interne herberekening van objectlocaties, anticiperend op een saccade. We hebben onderzocht of deze anticipatie al plaatsvindt in een stadium waarin het annuleren van de saccade nog mogelijk is. Dit is gedaan door proefpersonen een reeks saccades te laten maken door een verspringende stip te laten volgen op een scherm. Vervolgens werd rond het tijdstip van de laatste saccade van een reeks (i.e. peri-saccadisch) een 'flits', een vertikaal staafje, kort getoond waarvan de positie onthouden en (met een muis) gerapporteerd moest worden. Mensen maakten systematische lokalisatiefouten als de flits vlak voor, tijdens of na de saccade verscheen. Echter, soms werd aan het eind van een
reeks een stop signaal gegeven waarna de proefpersoon de laatste saccade niet mocht uitvoeren. Hoe later dit stopsignaal gepresenteerd werd ten opzichte van het saccade-doel, hoe verder gevorderd de planning, hoe moeilijker het was om de saccade te onderdrukken. Een dergelijke stoptaak wordt ook wel countermanding genoemd. We vonden vreemd genoeg geen tekenen van mislokalisatie wanneer een geplande saccade succesvol was onderdrukt. Dit suggereert dat objectlocaties intern pas herberekend worden als de uitvoering van een geplande saccade onvermijdelijk is. Ook alternatieve interpretaties worden besproken.

Hoofdstuk 3 van dit proefschrift richt zich op het annuleren van een geplande beweging. Voor de analyses met betrekking tot succesvol onderdrukte oogbewegingenis in hoofdstuk 2 gebruikgemaakt van een ruwe schatting om te bepalen wanneer de oogbeweging zou zijn uitgevoerd wanneer deze niet onderdrukt zou zijn geweest. Hoewel deze schatting afdoende bleek wordt in hoofdstuk 3 dit tijdstip exact bepaald, met behulp van (intramusculaire) elektromyografie (EMG). Hiervoor hebben we gebruik gemaakt van armbewegingen, en niet van oogbewegingen. De reden is dat saccades van zeer korte duur zijn en oogspieractiviteit lastig te meten is. Beter geschikt zijn bewegingen die onderhevig zijn aan massatraagheid, zoals de arm. In het countermanding experiment beschreven in hoofdstuk 3 hebben we ons gericht op reikbewegingen van de rechterarm. We laten zien dat bij het merendeel van de succesvol onderdrukte bewegingen minieme spieractiviteit kan worden opgepikt welke gerelateerd is aan het starten en onderdrukken van de armbeweging. We vinden tevens dat fluctuaties van deze voorheen onzichtbare reactietijden deels kunnen worden verklaard aan de hand van countermanding succes in voorgaande trials. Toekomstige experimenten zouden deze bevinden kunnen gebruiken om de perceptie-actie koppeling beter te begrijpen in het gezonde en pathologische brein.

Terugkomend op de vraag hoe het brein visuele stabiliteit creëert wordt in hoofdstuk 4 van dit proefschrift onderzocht hoe interne representaties van objectlocaties van voor en na de saccade worden gecombineerd. Zintuigen geven ruizige signalen waardoor het voorspelde en daadwerkelijke beeld na een saccade nooit gelijk zullen zijn. Hoe bepaalt het brein dan of een object is verschoven tijdens de saccade? Als een object verplaatst wordt tijdens een saccade vinden mensen het doorgaans lastig om dit te detecteren. In ons onderzoek hebben we hier gebruik van gemaakt. Aan proefpersonen werden
drie objecten getoond op een scherm waarvan de locaties onthouden moesten worden: een fixatiedoel, een saccadedoel en een perifeer doel. Tijdens de saccade van het fixatiedoel naar het saccadedoel werd abrupt een van de objecten verschoven, de andere twee verdwenen van het scherm. Van het object dat zichtbaar was na de saccade, het post-saccadisch object, moest de proefpersoon de herinnerde pre-saccadische locatie aangeven. Zoals verwacht op basis van de literatuur werd deze herinnering beïnvloed door de locatie van het post-saccadisch object. Deze beïnvloeding was een niet-lineaire functie van de tijd dat het post-saccadisch object zichtbaar was, de grootte van de verplaatsing en de richting van de verplaatsing. Alle resultaten konden succesvol worden gemodelleerd met Bayesiaanse causale inferentie: Na iedere saccade wordt eerst de waarschijnlijkheid bepaald dat de pre- en post-saccadische representaties veroorzaakt zijn door een stabiel object. Ruigheid van de representaties speelt hierbij een belangrijke rol: als de representaties heel precies zijn (weinig ruis) zal een kleine verplaatsing gemakkelijk worden opgemerkt. Vervolgens wordt de waarschijnlijkheid van een stabiel object gebruikt om integratie (i.e. combineren van pre en post) versus segregatie te wegen, om uiteindelijk tot de meest optimale schatting te komen. Dit suggereert dat visuele stabiliteit niet alles-of-niets is: het alternatieve scenario (het object bewoog of het object stond stil) wordt continu meegewogen.

Afsluitend wordt in hoofdstuk 5 van dit proefschrift gereflecteerd op bovenstaande onderzoeken. Zo wordt er stilgestaan bij de vraag of er een ‘point of no return’ bestaat bij het plannen van bewegingen (waarschijnlijk wel, vlak voor de beweging begint). Daarnaast wordt er gespeculeerd dat de interne herberekening van objectlocaties, anticiperend op een saccade, misschien in eerste instantie plaatsvindt om alvast vervolgbewegingen te plannen en pas in tweede instantie voor het creëren van visuele stabiliteit. Tot slot wordt de generaliseerbaarheid van het model uit hoofdstuk 4 besproken en worden er suggesties gedaan voor vervolgonderzoek.
6.3. Donders Graduate School for Cognitive Neuroscience

For a successful research Institute, it is vital to train the next generation of young scientists. To achieve this goal, the Donders Institute for Brain, Cognition and Behaviour established the Donders Graduate School for Cognitive Neuroscience (DGCN), which was officially recognized as a national graduate school in 2009. The Graduate School covers training at both Master's and PhD level and provides an excellent educational context fully aligned with the research programme of the Donders Institute.

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For more information on the DGCN as well as past and upcoming defenses please visit:
http://www.ru.nl/donders/graduate-school/donders-graduate/
6.4. Dankwoord

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6.5. About the author

Jeroen Atsma was born the 9th of June 1987 in Oss, the Netherlands. He finished his secondary education at the Philips van Horne SG in Weert in 2011, after which he started the BSc Computer Science program at the Radboud University Nijmegen. However, he soon got interested in a more mysterious 'computer', namely the human brain. In 2009 he obtained his BSc degree in Cognitive Psychology and in 2011 his MSc (research) degree in Cognitive Neuroscience at the Radboud University with a thesis on visual attention and multiple object tracking, under the supervision of Rob van Lier and Arno Koning. In 2011 Jeroen started as a PhD student in the Sensorimotorlab group at the Donders Institute for Brain, Cognition and Behavior in Nijmegen, under the supervision of Pieter Medendorp and Femke Maij. His research focused on sensorimotor integration; in particular how the brain creates a coherent and stable world when making eye movements. In addition, he studied mechanisms in inhibitory control and movement cancelation, under the supervision of Brian Corneil. The current doctoral thesis is the result of this work.