Volition and Automaticity in the Interactions of Optokinetic Nystagmus, Infantile Nystagmus, Saccades and Smooth Pursuit

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"So much has been written about nystagmus that there are only two unresolved questions about nystagmus: 1) the origin of the slow phase, and 2) the origin of the fast phase"

- Unknown Source, as cited in Dell'Osso (1982), Congenital nystagmus: Basic aspects

Summary

Volitional target-selecting eye movements, such as saccades or smooth pursuit, are frequently considered distinct and separate from automatic gaze-stabilising eye movements like optokinetic nystagmus or the vestibulo-ocular reflex. This difference is regularly mapped onto brain anatomy, with distinctions made between subcortical, automatic processes; and cortical, volitional ones. However gaze-stabilising and target-selecting eye movements must work together when a moving observer views natural scenes. Yet such co-ordination would not be possible if automatic and volitional actions are sharply divided. This thesis focuses upon interactions between gaze-stabilising and target-selecting eye movements, and how these interactions can aid our understanding of the relationship between automatic and volitional processes.

For a saccade executed during optokinetic nystagmus to accurately land on target, it must compensate for the ongoing optokinetic movement. It was found that targeting saccades can partially compensate for concomitant optokinetic nystagmus. The degree of compensation during optokinetic nystagmus was indistinguishable from compensation due to voluntary smooth pursuit displacements. A subsequent experiment found that locations are similarly misperceived during optokinetic nystagmus and smooth pursuit. Furthermore, saccade end-points are subject to the same perceptual mislocalisations. The next experiment established that fast-phases of optokinetic nystagmus can act like competitive saccades and cause curvature in targeting saccades. Moreover, optokinetic nystagmus fast-phases are delayed by irrelevant visual distractors in the same way as saccades (the saccadic inhibition effect). Lastly, it was established that the fast-phases of Infantile Nystagmus Syndrome also show the saccadic inhibition effect.

In conclusion, target-selecting and gaze-stabilising eye movements show substantial co-ordination. Furthermore these results demonstrate considerable commonalties between 'automatic' and 'volitional' eye movements. Such commonalities provide further evidence there is no sharp distinction between automatic and volitional processes. Instead it is likely there are substantial interconnections between automatic and volitional mechanisms, and volition has a graded influence upon behaviour.

Acknowledgements

First and foremost all my thanks to my supervisors Tom Freeman and Petroc Sumner. This thesis would have been entirely impossible without your constructive guidance, unfailingly creative and productive ideas, and profound debate. I think as a supervisory pair you are an excellent team, and I sincerely hope you take on further students and continue to work together.

Thanks also to all the people who I've met and worked with over the course of my PhD; you are all, without exception, splendid. I apologise I do not have enough space to name you all, but special thanks to Annie Campbell, Brice Dassy, Stephanie de la Smits, Kate Ellis-Davies, Colin Foad, Bethan Fulford, Kerry Gilroy, Mark Good, Emma Hindley, Richard Inman, Bill Macken, Lee McIlreavy, Laura Middleton, Luke Montuori, Becky Pepper, Georgie Powell, Cass Rogers, Alex St. John, Gary Williams and Ben Windsor-Shellard. Thank you so much for making coming into work fun, and drinks after work compulsory. Extra special thanks to those brave and hardy enough to participate in my seemingly endless experiments. Final thanks to Matt Dunn and Jon Erichsen from the School of Optometry, without whom my experiment on INS would not have been possible.

I would also like to thank my friends outside of academia, who have tirelessly pretended interest and provided support over the last few years. Thank you to Josh Cooke, Lloyd Hambridge, Sophie Hammond, Lucy Morgan, Eddie Pigott, Lucy Quarrier, John-Paul Rantac, Kirsty Spearman, Joe Tegeltija and Natasha Wilson. I would be a far more peculiar man coming out of the PhD process without you all there to ground me in the real world.

Lots of love and thanks to my Mum and my sister for everything you've done for me over the last few years. Your unwavering encouragement, help and love for me means so much, and I couldn't have done it without you. All my love and thanks go to my wonderful girlfriend Zoë Mawdsley, who probably bore the greatest burden of keeping me going over the last three and a half years You've done an amazing job, and you've never failed to ask me how my day's gone; despite the answer inevitably being incomprehensible and downright weird.

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Chapter 1: Introduction

In human vision, optimal perception requires an object of regard to fall upon our relatively small fovea (the area of the retina with the highest visual acuity) (Yuodelis & Hendrickson, 1986). This strategy would be incredibly difficult without those eye movements that relocate the fovea to view new objects, those that relocate the fovea to follow a moving object, or those that stabilize the fovea upon an object when we ourselves are moving (Land, 1999). As a moving observer progressing through a rich visual scene, all three of these requirements must be addressed simultaneously (Moeller, Kayser, Knecht, & Konig, 2004). Therefore it seems prudent to assume there must be some way in which gaze-stabilizing eye movements are co-ordinated with those that shift the fovea in response to a moving, or a new target of interest. However there is a strong tendency in the literature to treat gaze-stabilizing and target-selecting eye movements as separate and independent, each generated by discrete neural structures with little communication between them (Sumner & Husain, 2008). In the following sections the main types of gaze-stabilizing and target-selecting eye movements will be outlined, and the different ways in which the literature views gaze-stabilizing and target-selecting eye movements will be considered. Although some authors consider automatic and voluntary eye-movements as distinct (e.g. Post & Leibowitz, 1985; Whiteside, Graybiel, & Niven, 1965); literature that shows the capacity of gaze-stabilizing and targeting eye movements to be co-ordinated is presented, with particular reference to how this might fit in to a framework that considers targeting and stabilizing eye movements to be separate and independent. Lastly, I will outline the characteristics of a pathological involuntary eye movement, infantile nystagmus syndrome, as investigation of this syndrome will be conducted in the final experiment of this thesis.

1.1 Basic Characteristics of Gaze-Stabilizing Eye Movements

1.1.1 *The Vestibular-Ocular Reflex*

The Vestibular-Ocular Reflex (VOR) has a comparatively simple neural substrate, able to be mediated entirely through subcortical structures (Leigh & Zee, 1999); and indeed can result from a reflex arc consisting of just three neurones (Szentágothai, 1950). Consequently it has a very short latency (Collewijn & Smeets, 2000) and is evolutionary very old (Walls, 1962). The VOR signal stems from perturbations of the fluid in the canals of the inner ear (Hess, 2011; Szentágothai, 1950). These perturbations occur whenever the head undergoes acceleration or deceleration (Hess, 2011). This allows rotation of the eyes in order to negate certain components of movement upon the retina which would otherwise occur during head rotations (Hess, 2011). The VOR can also rotate the eye in order to negate some of the movement upon the retina during head translations, however these compensatory eye-movements are more computationally demanding, and depend upon target distance and eccentricity (Angelaki, 2004). As the fluid in the inner ear is not perturbed during prolonged self-motion, the transient VOR is supplemented by a more continuous gaze-stabilizing eye movement, that of optokinetic nystagmus (Waespe & Henn, 1977).

1.1.2 *Optokinetic nystagmus*

Optokinetic nystagmus (OKN) occurs whenever there is large-scale movement upon the retina; therefore it allows the rotatory component of movement within the retinal image to be stabilized as much as possible during self-motion, or when in a moving environment (Distler & Hoffmann, 2011). It consists of two distinct phases, a slow-phase where rotation of the eye occurs at about the same velocity as the viewed motion (at least for speeds of less than 50°/sec [Garbutt et al., 2003]) and a resetting fast-phase which serves to reposition the eye in its orbit (Curthoys, 2002).

The slow-phase involves two components: a pathway mediated through the flocculus which produces the initial rapid rise in eye velocity (Blanks & Precht, 1983; Schiff, Cohen, Büttner-Ennever, & Matsuo, 1990; Waespe, Rudinger, & Wolfensberger, 1985; Zee, Yamazaki, Butler, & Gucer, 1981) and a velocity storage mechanism situated in the nucleus of the optic tract to maintain eye velocity (Cohen, Reisine, Yokota, & Raphan, 1992; Distler & Hoffmann, 2011; Kato et al., 1986; Schiff, Cohen, & Raphan, 1988; Yakushin et al., 2000). The velocity-storage mechanism is thought to be responsible for the phenomenon of optokinetic after-nystagmus, where transient OKN movements continue when an observer is immediately placed into darkness following OKN stimulation (Büttner & Kremmyda, 2007; Chaudhuri, 1990; Cohen, Matsuo, & Raphan, 1977; Freeman & Sumnall, 2005). The resetting fast-phases of OKN are generated by a different neural substrate to the slow-phases, and are attributed to brainstem burst neurons in the reticular formation (Curthoys, 2002; Curthoys, Markham, & Furuya, 1984; Curthoys, Nakao, & Markham, 1981; Hess, Blanks, Lannou, & Precht, 1989; Kitama, Ohki, Shimazu, Tanaka, & Yoshida, 1995).

OKN is sometimes distinguished into two types: look-OKN and stare-OKN (Baloh, Yee, & Honrubia, 1980; Freeman & Sumnall, 2005, Ter Braak, 1936, as cited in Bender & Shanzer, 1983; Fite, 1968). Stare-OKN occurs when participants passively view moving stimuli without trying to track any particular element in the moving display (Kashou et al., 2010). It is characterised by small amplitude slow-phases, interspersed with fast-phases at a frequency of about 3Hz (Cheng & Outerbridge, 1974). Stare-OKN is thought to be mediated entirely sub-cortically (Baloh et al., 1980; Gulyás, Pálvölgyi, Kamondi, & Szirmai, 2007). Look-OKN is elicited when participants are asked to voluntarily track an element within a moving display (Knapp, Gottlob, McLean, & Proudlock, 2008). Look-OKN is characterised by slow-phases of a longer amplitude, and fast-phases of a much lower frequency (Knapp et al., 2008). Look-OKN (unlike stare-OKN) is usually accompanied by cortical activity in

areas associated with pursuit and saccades (Freeman & Sumnall, 2005; Kashou et al., 2010; Konen, Kleiser, Seitz, & Bremmer, 2005; Schraa-Tam et al., 2009). Indeed some researches assume look-OKN is identical to alternating pursuit and saccades (Heinen & Keller, 2004).

1.2 Basic Characteristics of Target Selecting Eye-Movements

1.2.1 *Smooth pursuit*

Smooth pursuit eye movements allow a moving target to remain fixed upon the retina. Unlike OKN, smooth pursuit is a voluntary response to a small moving object, rather than an automatic response to whole-field motion (Heinen & Keller, 2004). Initial pursuit is internally driven by fast visual feedback (thought to be similar to that which drives the initial rapid component of OKN [Gellman, Carl, & Miles, 1990; Heinen & Keller, 2004; Pola & Wyatt, 1985]) and pursuit latency can be as short as 100ms (Robinson, 1965). However following this, extraretinal mechanisms are deployed within a few hundred milliseconds to maintain pursuit if target velocity is constant or predictable (Barnes, 2011).

Smooth pursuit eye movements are generated though many structures at both the cortical and sub-cortical level (Büttner & Kremmyda, 2007). Two of the most heavily implicated areas are in the caudal portion of the superior temporal sulcus, namely the Middle Temporal (MT) area, and the Medial Superior Temporal (MST) area (Heinen & Keller, 2004). Both of these areas are heavily involved in motion processing; however both appear to be crucial for pursuit (Keller & Heinen, 1991). Area MT has been conceptualised as the area which is crucial for the initiation of pursuit (Heinen & Keller, 2004); for example lesions to area MT impair the initiation of pursuit when target motion is within the receptive field of the lesioned area, whilst saccades are unaffected by these lesions (Dursteler, Wurtz, & Newsome, 1987; Newsome, Wurtz, Dursteler, & Mikami, 1985; Pack, Grossberg, & Mingolla, 2001).

Area MST has been conceptualised as an area associated with the maintenance of

smooth pursuit (Heinen & Keller, 2004). For example stimulation of area MST will not produce pursuit, but it can cause acceleration changes to a pursuit eye movement which is already underway (Keller & Heinen, 1991; Komatsu & Wurtz, 1989). These pursuit velocity changes are negated by corrective saccades, showing that perceived location of the target is not disrupted (Komatsu & Wurtz, 1989). Furthermore lesions to area MST will not abolish pursuit, however they adversely affect pursuit gain, showing an inability to match eye velocity to target velocity (Dursteler & Wurtz, 1988).

Frontal cortical areas also seem to play a role during smooth pursuit. For example pursuit gains are much reduced following lesions to the frontal eye fields, and predictive pursuit is abolished by frontal eye field lesions (Keller & Heinen, 1991). Furthermore recording of frontal eye field neurones show they discharge during smooth tracking eye movements (Keller & Heinen, 1991). The role of the frontal eye fields is further confirmed by microstimulation of this area, which produces a detriment to the gain of smooth pursuit movements (Thier & Ilg, 2005).

Smooth pursuit eye movements are executed predominantly through discharges via the pontine nucleus of the brainstem (Keller & Heinen, 1991). The main projections travel directly from the cortex to the brainstem, however a substantial number also travel through the superior colliculus (Thier & Ilg, 2005). Indeed, some authors have claimed that the rostral pole of the superior colliculus plays a role during pursuit eye movements, as activity in this area has been recorded during pursuit (Krauzlis, 2004; Krauzlis, Basso, & Wurtz, 2000). It has been postulated that activity within the rostral pole of the superior colliculus may code for an error signal between gaze location and target position (Krauzlis, Basso, & Wurtz, 1997).

As well as brainstem areas, smooth pursuit is also heavily reliant upon the cerebellum, for example complete cerebellectomy abolishes pursuit (Keller & Heinen, 1991). The cerebellum may exert its influence upon pursuit via connections through the vestibular nuclei (Keller & Heinen, 1991; Thier & Ilg, 2005); and as such the cerebellum may be crucial in co-ordinating pursuit eye movements which are executed simultaneously with a head movement (Thier & Ilg, 2005, see also Section 1.4.2).

1.2.2 *Saccades*

Saccades are fast, discrete eye movements which reorient the fovea upon new targets of interest. Saccades are some of the most numerous movements we make, it is estimated we make around 3-4 every second (Findlay & Gilchrist, 2003). Moreover, they are the fastest motor actions we execute, large saccades may reach speeds of over 500°/sec (Leigh & Zee, 1999), and a saccade from the extreme left to the extreme right of our orbit can peak at 700°/sec (Blake & Sekuler, 2006). The speed of a saccade depends upon its amplitude in a characteristic and stereotypical way – saccades with a longer amplitude have a higher peak velocity; this relationship is called the main sequence (Bahill, Clark, & Stark, 1975). It is thought that the main sequence is an adaptive strategy which allows for the optimal speed-accuracy trade off during saccadic eye movements (Harris & Wolpert, 2006).

The time taken to initiate a saccade is referred to as the saccade latency period. This value is remarkably variable and depends heavily upon the stimulus characteristics of the saccade target (Findlay & Gilchrist, 2003). Saccades which are made in response to suddenly appearing peripheral targets are much faster than saccades made in response to a symbolic cue (e.g. an arrow presented at fixation) (Walker, Walker, Husain, & Kennard, 2000). This is thought to be because exogenously cued saccades are processed by the oculomotor system more rapidly than endogenously generated saccades (Bompas & Sumner, 2011). However the properties of the saccade target itself also influence saccade latency. For example, saccades

are generally faster to stimuli which have greater luminance (Bell, Meredith, Opstal, & Munoz, 2006; Kalesnykas & Hallett, 1994; Wheeless, Cohen, & Boynton, 1967) or have more low spatial-frequency information (Findlay, Brogan, & Wenban-Smith, 1993). Furthermore location of the target plays a role: beyond the central 2° of visual angle (where latencies are long) there is a linear increase between saccade latency and eccentricity of the saccade target (Bell, Everling, & Munoz, 2000; Kalesnykas & Hallett, 1994). Moreover the time taken to initiate a saccade depends upon the existence and location of other stimuli combined with the saccade target. Irrelevant stimuli presented alongside the saccade target (usually termed distractor stimuli) will speed up saccades if they are placed in close proximity to the saccade target, but will slow saccades if they are placed some distance away (Walker, Deubel, Schneider, & Findlay, 1997); this phenomenon is known as the 'Remote Distractor Effect' (Bompas & Sumner, 2009b; Buonocore & McIntosh, 2008; Findlay & Gilchrist, 2003)

Saccadic latency further depends upon the internal state of the observer (Findlay & Gilchrist, 2003). Saccade latencies are much reduced if a delay is imposed between the extinguishing of the fixation point and the presentation of the saccade target – the so-called 'gap effect' (Kingstone & Klein, 1993). This gap effect is thought to arise from two processes: one process is a general warning signal taken from the disappearance of the fixation point. The use of a tone as a warning signal will also speed up saccade latencies (Forbes & Klein, 1996); however the effect of a warning tone is much less than the gap effect, implying a second process is also involved. It is believed that the disappearance of the fixation point allows fixation-related activity in the oculomotor system to disengage; thereby speeding up the processing of saccade-related activity (Kingstone & Klein, 1993). It has been shown that fixation-related activity in primate superior colliculus decreases during this gap

period, which allows saccade-related superior collicular activity to reach an initiation threshold in a shorter period of time (Munoz, Dorris, Paré, & Everling, 2000).

The attentional state of an observer also has a significant role in the time taken to initiate a saccade; attending covertly to a location in space will decrease latencies for saccades to the attended location. For example a valid cue to a saccade target's location will speed up saccade latencies, and an invalid cue will slow latencies (Walker, Kentridge, & Findlay, 1995). Saccade latencies can also be influenced through priming; in the masked prime paradigm subliminally presented primes can speed or slow saccades if they respectively cue valid or invalid responses (Eimer & Schlaghecken, 2001). This priming effect appears to be a general phenomenon of motor actions (Eimer & Schlaghecken, 2001, 2003) and is believed to reflect automatic, sub-threshold activity changes within the motor system (Boy & Sumner, 2010; Eimer & Schlaghecken, 2003)

Although the above research highlights many of the external and internal contributions to variability in saccade latencies, there still appears to be a large amount of variability which cannot be controlled for or manipulated. Under identical experimental conditions, it would not be unusual for a single observer to show saccade latencies between 100 and 500ms (Sumner, 2011). The variability in saccades furthermore shows a characteristic, positively skewed normal distribution (Gilchrist, 2011). These distributions can be modelled from variability in a rise to threshold of saccade related activity (Carpenter & Williams, 1995). In this way, the saccadic system can be conceptualised as having an in-built decision making mechanism, whereby saccades are only executed through the attainment of a criterion value; attainment which is accomplished more rapidly if there is greater incoming sensory 'evidence' to drive the saccadic response (Carpenter, 1999; Carpenter & Williams, 1995). Intrinsic randomness in the rise-to-threshold rate can allow different behavioural responses to be executed; giving the potential for top-down, goal-

directed behaviour to influence the saccadic system (Carpenter, 1999). Indeed saccades are far more likely to be directed to task-relevant targets; the bottom-up features of the visual scene such as contrast or salience are very poor predictors of saccadic landing points (Land, 2006). This in-built delay to allow top-down, goal directed behaviour could potentially account for why saccade latencies are longer, and more variable than would be expected purely from the physiological constraints of the oculomotor system (Sumner, 2011).

Occasionally a bimodal distribution of saccade latency can be observed. This has been attributed to a distinct population of saccades which seem faster than normal, so-called 'express saccades' (Fischer & Weber, 1993; Fischer et al., 1993). Express saccades may reflect an optomotor reflex for orienting to peripheral stimuli (Fischer & Weber, 1993). Therefore the programming of express saccades might not involve any 'higher-level' processing: they have bypassed the in-built delay which gives rise to long, and variable saccade latencies; as such these express saccades may be using an evolutionarily older pathway to 'normal' targeting saccades (Sumner, 2011). Express saccades do not always occur; they depend upon attentional state, practice of the observer, and stimulus characteristics (Gilchrist, 2011; Knox, Amatya, Jiang, & Gong, 2012).

The programming of saccades is partially ballistic, meaning that the end-point is predetermined before the saccade is initiated (Gilchrist, 2011). The ballistic nature of this process can be revealed by the double-step paradigm, whereby a saccade target is relocated during the saccade latency period (Findlay & Gilchrist, 2003). When target perturbation occurs some time prior to saccade initiation, the change in target location can be accommodated by the saccade (Becker & Jürgens, 1979; Gilchrist, 2011). However if target perturbation is within around 80ms of saccade initiation then the saccade will not be modified, and it will land upon the original target location (Becker & Jürgens, 1979). This shows that the ability to correct saccades on-line is limited.

There is a large and complex literature on the neural pathways generating saccadic eye movements (Leigh & Zee, 1999). Saccades are generated through multiple parallel neural pathways descending to brainstem burst neurones in the reticular formation (Cullen & Van Horn, 2011; Leigh & Zee, 1999). These pathways descend from both frontal and parietal cortical areas. It is well established that stimulation of the frontal eye fields and supplementary eye fields in the frontal cortex will produce saccadic movements (Johnston & Everling, 2011) and ablation of the frontal cortex produces deficits in saccade initiation (Lynch, 1992). However, as well as initiating saccades, the frontal cortex also seems to be crucial for the flexible control of saccades. For example, it has been found that two patients with lesions to medial frontal cortex did not show the usual automatic inhibition of saccade responses elicited through masked priming (Sumner et al., 2007), and lesions to frontal cortex are associated with a range of deficits in the antisaccade task (whereby participants must suppress a saccade to a peripheral target, and instead execute an internally generated saccade in the opposite direction [Hallett, 1978]) (Everling & Fischer, 1998; Munoz & Everling, 2004).

The inhibition of the reflexive saccade in the antisaccade task appears to be reliant upon activity in the frontal eye fields; for example correct performance on the antisaccade task in primates is predicted by lower activity in the frontal eye fields (Munoz & Everling, 2004), and TMS of the frontal eye fields makes it less likely that the reflexive saccade will be successfully inhibited (Olk, Chang, Kingstone, & Ro, 2006). However it is not clear where the signal which inhibits activity in the frontal eye fields originates, potentially it is contained within the frontal eye fields themselves (although lesions to the frontal eye fields do not always impair antisaccade performance [Gaymard, Ploner, Rivaud-Pechoux, & Pierrot-Deseilligny, 1999]), or alternatively it could originate from supplementary eye fields or dorsolateral prefrontal cortex (Munoz & Everling, 2004). Dorsolateral prefrontal cortex does

show significantly greater fMRI activation during antisaccades (Muri et al., 1998) and TMS of the dorsolateral prefrontal cortex 100ms prior to target presentation impairs the correct execution of antisaccades (Nyffeler et al., 2007). Although it is unclear whether the dorsolateral prefrontal cortex plays a role directly in oculomotor control during the antisaccade paradigm, or whether it is required for maintenance of the task-requirements in working memory.

Additionally, the dorsolateral prefrontal cortex seems to be heavily involved in the guidance of saccades to memorised locations (Johnston & Everling, 2011). Dorsolateral prefrontal cortex neurones fire during the delay period in a memory-guided saccade task (Funahashi, Bruce, & Goldman-Rakic, 1989), and lesions to primate dorsolateral prefrontal cortex impair memory-guided saccades, but leave immediate, visually-guided saccades intact (Funahashi, Bruce, & Goldman-Rakic, 1993). Human patients with lesions to the dorsolateral prefrontal cortex likewise show impairments in the memory guided saccade task (Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991; Walker, Husain, Hodgson, Harrison, & Kennard, 1998).

The frontal cortex works alongside saccade-related areas in the parietal cortex. It is not entirely clear what role the parietal cortex plays in saccade generation as ablation of parietal cortex does not prevent saccades from being executed (Lynch & McLaren, 1989). Nevertheless, the lateral intra-parietal area receives connections from numerous visual areas, and sends connections to both the frontal eye-fields and the superior colliculus (Paré & Dorris, 2011) and neurones in the parietal cortex respond strongly to both visual stimulation and during oculomotor tasks (Andersen, Essick, & Siegel, 1987). Lateral intra-parietal area neurones respond strongly in delayed and memory-guided saccade tasks, suggesting they may complement some of the processing which underpins goal-directed saccades in the frontal cortex (Paré & Wurtz, 1997). The parietal cortex furthermore seems to be heavily involved in

the shifting of attention (Lynch & McLaren, 1989) and may enhance the flexible control of saccades (Paré & Dorris, 2011). For example, lateral intra-parietal area activity is modulated to discriminate a saccade target from other distractor stimuli prior to saccade initiation, and correct performance on a distractor task can be predicted with some accuracy from preceding lateral intra-parietal neuronal activity (Thomas & Paré, 2007).

Although both frontal and parietal cortices project directly to the brainstem, these connections are meagre when compared those which travel through the superior colliculus (Leigh & Zee, 1999). The superior colliculus receives information from all cortical areas associated with saccades (Carpenter, 1999) as well as directly from the retina (White & Munoz, 2011). The intermediate layers of the superior colliculus contain an organised motor map (Marino, Rodgers, Levy, & Munoz, 2008; Marino, Trappenberg, Dorris, & Munoz, 2011). Stimulation of this motor map will produce a saccade to its corresponding retinal location (Gandhi & Katnani, 2011). Ablation of the superior colliculus results in a temporary deficit in saccade initiation (Pierrot-Deseilligny, Rosa, Masmoudi, Rivaud, & Gaymard, 1991). The superior colliculus is capable of executing oculomotor responses to the presence of visual stimuli, but is reliant upon higher-level processing from the cortex to direct saccades to a particular saccade goal, when there are a number of alternative potential targets available (Carpenter, 1999). In this way the superior colliculus may be conceptualised as an area which receives many inputs, with many competing potential saccade end-points; but which selects one particular saccadic program to be passed onto execution machinery further down in the brainstem (Carpenter, 1999).

Cortical areas also project to the brainstem via the cerebellum, which itself may play a role in short-term saccadic learning and adaptation (Thier, 2011). Furthermore there are pathways to the brainstem through the basal ganglia, which may be ideally placed to have some form of overall control over the saccadic system (Vokoun, Mahamed, & Basso, 2011).

For example, the activity of neurones in the superior colliculus depends upon the activity within the basal ganglia (Vokoun et al., 2011). Pathways also project from subcortical areas back to the cortex via the thalamus; which is postulated to help monitor saccadic movements to allow spatial updating across saccades (Tanaka & Kunimatsu, 2011), for example patients with thalamic lesions are impaired in directing the second saccade in the double-saccade paradigm, implying oculomotor maps were not updated following displacement of the eye due to the first saccade (Bellebaum, Hoffmann, Koch, Schwarz, & Daum, 2006).

1.3 Conceptualising Eye Movements as either Voluntary or Automatic

Most areas of psychology have been built upon theories which draw fundamental distinctions between processes that are automatic, inflexible and can be handled by relatively unintelligent neural mechanisms, and those that are consciously willed, effortful, adaptable and require highly sophisticated neural processes (Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977, 1984). This dichotomy between automatic and voluntary processes remains embedded in many contemporary articles across a variety of disciplines: for example in spatial attention (Barbot, Landy, & Carrasco, 2012; Chica, Bartolomeo, & Lupiáñez, 2013; Ibos, Duhamel, & Ben Hamed, 2013; Macaluso & Doricchi, 2013; McAuliffe, Johnson, Weaver, Deller-Quinn, & Hansen, 2013; Mysore & Knudsen, 2013; D. T. Smith, Schenk, & Rorden, 2012); temporal attention (Lawrence & Klein, 2013); cognition (Lifshitz, Bonn, Fischer, Kashem, & Raz, 2013); motor cueing (Martín-Arévalo, Kingstone, & Lupiáñez, 2013); reading (Feng, 2012); perception (Pfister, Heinemann, Kiesel, Thomaschke, & Janczyk, 2012; Spence & Deroy, 2013); social cognition/perception (Laidlaw, Risko, & Kingstone, 2012) or emotion regulation (R. Viviani, 2013). Similarly, voluntary and automatic actions are clearly distinguished in clinical literature, for conditions ranging from deafness (Bottari, Valsecchi, & Pavani, 2012), to Parkinson's disease (D'Ostilio, Cremers, Delvaux, Sadzot, & Garraux, 2013; van Stockum, MacAskill, & Anderson, 2012; van

Stockum, MacAskill, Myall, & Anderson, 2013; Vervoort et al., 2013), Huntington's disease (Patel, Jankovic, Hood, Jeter, & Sereno, 2012), autism (Vernazza-Martin, Longuet, Chamot, & Orève, 2013) and mild traumatic brain injury (Zhang, Red, Lin, Patel, & Sereno, 2013).

While the interplay between automaticity and volition has relevance to many areas of psychology, to study it, one must choose an effector system as exemplar. Oculomotor control can usefully encapsulate the debate and serve to test specific hypotheses. Oculomotor decisions are the most frequent volitional acts we make, and have been used as models for decisions in general (Carpenter & Williams, 1995; Cutsuridis, Smyrnis, Evdokmds, & Perantonis, 2007; P. L. Smith & Ratcliff, 2004). The underlying machinery is relatively well understood, partly because oculomotor tasks allow simple, easily controlled and easily implemented paradigms that are also well-suited to primate neurophysiology (Bell et al., 2000; Munoz & Everling, 2004; White, Theeuwes, & Munoz, 2011). In this way eye movements are able to link the fields of psychology and neurophysiology.

Moreover intentional eye movements exist alongside gaze stabilisation mechanisms that are paradigmatic exemplars of ancient reflexive behaviour and whose characteristics and neural underpinning has been extensively researched. Typically, the gaze stabilizing eye movements outlined in Section 1.1 (namely VOR and OKN) are thought to be automatic and inflexible (Findlay & Gilchrist, 2003), whereas targeting eye movements such as smooth pursuit and saccades (Section 1.2) are considered as voluntary and adaptable (Walls, 1962). Frequently this distinction is extended into brain anatomy, with voluntary eye movements requiring cortical control, and reflexive eye movements generated entirely subcortically (for a review, see Sumner & Husain, 2008). However, drawing a clear distinction between automatic and reflexive eye movements is often very difficult. For example saccades can automatically be elicited by stimuli that suddenly appear, which has been referred to as a 'visual grasp reflex' (Theeuwes, Kramer, Hahn, & Irwin, 1998; Theeuwes, Kramer, Hahn,

Irwin, & Zelinsky, 1999). Furthermore in the remote distractor paradigm a small population of saccades will inevitably land upon the distractor stimulus (Godijn & Theeuwes, 2002; Walker et al., 1997); and a far larger effect of erroneous saccades can be seen in those saccades which end up directed toward the anti-target in the antisaccade paradigm (Everling & Fischer, 1998; Hallett, 1978).These saccades appear reflexive, therefore to characterise the entire saccadic system as either volitional or reflexive seems problematical.

Smooth pursuit also appears to be underpinned by reflexive systems. For example smooth pursuit eye movements cannot be generated without a moving stimulus to pursue (Heinen & Keller, 2004). Furthermore retinal motion can induce short-latency ocular following responses without active participation by the observer (Barnes, 2011). However smooth pursuit cannot only be a response to retinal motion per se; for example observers track the perceived motion of objects, rather than the corresponding retinal movement (Krauzlis, 2004; Steinbach, 1976). Furthmore there appears to be a predictive element to smooth pursuit; pursuit of a predictable target is far better than would be expected from retinal feedback alone (Dallos & Jones, 1963) and when predictable target motion is suddenly changed, pursuit will briefly continue to follow the previous, predictable pattern (Barnes & Asselman, 1991). Additionally, smooth pursuit continues when the target is occluded for brief periods, especially if target motion is predictable (Becker & Fuchs, 1985).

The partially reflexive nature of saccades and smooth pursuit might seem to cast doubt upon the assertion that they can be categorically labelled as different from automatic eye movements such as VOR and OKN. Furthermore it seems untenable to state that automatic and voluntary eye movements are entirely independent and distinct when one considers the fundamental requirement to co-ordinate targeting and gaze-stabilizing eye movements when an active observer views natural scenes (Moeller et al., 2004). A moving observer must both stabilize the retinal image to allow the highest acuity possible, and

simultaneously move the eyes in order to foveate targets of interest. The interaction between gaze-stabilizing and targeting eye movements will form a central theme in this thesis; both because would logically appear to be a necessary requirement of the oculomotor system, but also beacuase it can easily distill the debate as to whether there really is a sharp dichotomy between reflexive and volitional motor actions.

1.4 Interactions between Gaze-stabilizing and Target Selecting Eye Movements

Gaze-stabilizing and target selecting eye movements must be co-ordinated to some extent to allow accurate foveation in a moving observer. Yet it is difficult to embed this necessary co-ordination within a framework which draws a sharp distinction between automatic gaze-stabilizing and volitional targeting eye-movements. Some authors have claimed that automatic eye movements such as OKN or VOR are not accompanied by efference copies (Post & Leibowitz, 1985; Walls, 1962; Whiteside et al., 1965), which might imply that volitional oculomotor systems would have limited knowledge of the current activity in gaze-stabilizing networks. However, this does not appear to be borne out by research into the co-ordination between gaze-stabilizing and target selecting eye movements; research outlined in the following sections.

1.4.1 *Interactions between saccades and vestibular-ocular reflex*

The interaction between targeting saccades and the vestibular-ocular reflex (VOR) is essential in order to achieve large shifts of gaze. This is because large gaze shifts are often accomplished with a head and an eye movement (Daye, Blohm, & Lefèvre, 2010; Pelisson & Prablanc, 1986). If one were to imagine a large gaze shift to the right, this would be accomplished with both a saccade and a head movement to the right; however this rightward head movement would elicit leftward VOR. As the head movement is executed during the

saccade, summation of the VOR and saccade plans would slow the eye-in-head velocity during head rotation (Cullen, 2004). However, the velocity of the saccade indicates that there is suppression of the VOR during a saccade, the eye continues at nearly the same eye-in-head velocity during the head movement as it did before (Cullen, Huterer, Braidwood, & Sylvestre, 2004; Jürgens, Becker, & Rieger, 1981). Moreover, saccades made concomitantly with a head movement are remarkably accurate (Jürgens et al., 1981) and are no less accurate than saccades executed without head movements (Cullen et al., 2004).

The suppression of VOR during a saccade is very finely co-ordinated; gaze shifts are usually achieved with the eye moving first, followed by the head (Land, 1993, 2006). At the end of the gaze shift the eye lands on target, but the head continues to move, requiring a VOR compensation (Corneil, 2011). This rapid shift from VOR suppression to VOR activation implies a very close co-ordination between the vestibular and saccadic systems. Additionally, information does not only appear to be sent from the saccadic system to the vestibular system; for example a saccade can be executed to a head-fixed target after the eyes have been displaced through VOR (Hansen & Skavenski, 1977). This would imply that VOR activity updates saccadic motor maps.

1.4.2 *Interactions between smooth pursuit and vestibular-ocular reflex*

The same logic of large gaze shifts requiring a co-ordinated eye-head movement extends to pursuing a target over a wide angle; this too would be achieved through a simultaneous smooth pursuit and head movement (Corneil, 2011). For the same reason that VOR would be counterproductive during eye-head gaze-shifts, an active, or unaccounted for VOR signal would be counterproductive during eye-head pursuit (Corneil, 2011). Suppression of the VOR signal appears to exist for smooth pursuit just as it does for saccades (Cullen & Roy, 2004; Cullen & Van Horn, 2011). This suppression occurs far more quickly than could be achieved through the use of reafferent retinal motion, which implies it is an internally

generated mechanism (Lisberger, 1990). Furthermore smooth pursuit is far more accurate under active, rather than passive head movements, again implying the internal motor commands drive the majority of VOR suppression (Cullen & Roy, 2004).

1.4.3 *Interactions between smooth pursuit and optokinetic nystagmus*

OKN slow-phases consist of two processes (Section 1.1.2), an initial rapid rise in eye velocity, and a velocity storage mechanism to maintain eye velocity (Distler & Hoffmann, 2011). The initial rapid rise in velocity does show some parallels with that seen in pursuit (Gellman et al., 1990; Pola & Wyatt, 1985); however the velocity storage mechanism appears to be a different process. For example cortical areas which respond to single target motion do not appear to be sensitive to global motion (Lisberger, Morris, & Tychsen, 1987). Furthermore the velocity storage mechanism of OKN can result in optokinetic after-nystagmus, whereas no, or very little after-nystagmus occurs following repetitious pursuit (Lisberger et al., 1987). A final point of evidence for divergence in these two systems is that velocity storage can take up to 15 seconds to be fully active during OKN, whereas during pursuit it can build up in several hundred milliseconds (Lisberger et al., 1987; Thier & Ilg, 2005).

If it is true that smooth pursuit and OKN are mediated by different neural structures, there must be a considerable amount of interaction between the two mechanisms. This is because in natural environments most smooth pursuit involves tracking a small object against a structured background; which gives retinal stimulation of global movement against pursuit direction (Lindner, Schwarz, & Ilg, 2001). This retinal signal would be the ideal stimulus to evoke OKN in the direction opposite to pursuit movement, therefore accurate pursuit against a background would require suppression of the optokinetic signal (Lindner et al., 2001). This suppression obviously occurs, as otherwise pursuit over a textured background would be very difficult; however suppression of the optokinetic reflexes does not appear to be complete. For

example pursuit gains are around 10-15% less when pursuing a target over a background than when pursuing a single target in the dark (Collewijn & Tamminga, 1984; Masson, Proteau, & Mestre, 1995). Pursuit gains are reduced further when pursuing over a background which moves in a direction opposite to pursuit; and pursuit gains are improved, even to the point above unity, when pursuing a target against a background moving with a pursuit target (Masson et al., 1995; van den Berg & Collewijn, 1986).

Suppression of OKN may occur because motion perception is selectively inhibited during pursuit eye movements. It has been shown that if the background is set in motion during a pursuit movement, it has very little influence when it moves against the direction of pursuit (Suehiro et al., 1999). This implies that there is a suppression of motion perception for movement against pursuit direction (Lindner et al., 2001; Suehiro et al., 1999), which could inhibit the optokinetic reflex. The motion suppression during smooth pursuit appears to be modulated by an extra-retinal signal for pursuit movement; for example it continues even during brief occlusions of the pursuit target, and is much more pronounced than during fixation with a moving background (Lindner & Ilg, 2006).

This close interaction between voluntary smooth pursuit and the optokinetic reflex furthermore underlies how voluntary and automatic movements cannot exist in complete isolation. This reinforces our point that as an active observer in a natural environment gaze-stabilizing and target selecting eye movements must interact closely

1.4.4 Interactions between saccades and optokinetic nystagmus

To my knowledge there has been very little research to date on the accuracy of a goal-directed, targeting saccade executed during optokinetic nystagmus (OKN). A moving observer viewing a natural scene would be expected to make saccades to targets of interest during ongoing OKN movement. Furthermore an accurate saccade needs to take into account

the displacement of the eye produced by OKN that occurs during the saccade latency period. There is some evidence that this happens: for example observers can accurately point at targets displayed during optokinetic after-nystagmus for both seen and unseen pointing (Bedell, 1990; Bedell, Klopfenstein, & Yuan, 1989). This suggests that the motion of the eye due to activity in the optokinetic system is incorporated into higher-level motor actions.

The incorporation of optokinetic commands into saccadic planning would be essential to allow accurate top-down saccades during OKN; however it is also possible that a closer interaction between targeting saccades and OKN is employed by the oculomotor system. Moeller et al. (2004) reported that the natural viewing of scenes during self-motion results in a unimodal distribution of saccades for all stimulus velocities. Thus it would appear that there is very early interaction between gaze-stabilizing OKN and targeting saccades, such that the fast-phases of OKN themselves show target-selecting properties; the targeting of new objects is achieved through a nystagmus fast-phase (Moeller et al., 2004). This would imply a very close co-ordination between automatic OKN and voluntary targeting saccades, and is evidence that the mechanisms underlying the generation of saccades and OKN may be very similar.

Such similarity between the mechanisms generating targeting saccades and optokinetic fast-phases would not be predicted by those that envisage a sharp distinction between automatic and voluntary actions (Post & Leibowitz, 1985; Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977, 1984; Whiteside et al., 1965), however it may well be predicted by those who have proposed that saccades evolved from the development of purposeful top-down control over the fast-phases of nystagmus (Ron, Robinson, & Skavenski, 1972; Walls, 1962). There are already significant overlaps between the requirements of saccades and OKN fast-phases (Ilg, Bremmer, & Hoffmann, 1993). For example, similarly to a saccade, the fast-phase has to be executed rapidly so as to minimise

the amount of time visual perception is disrupted (Harris & Wolpert, 2006). Early evidence that saccades and fast-phases were generated in a very similar way in the oculomotor system came from the observation that the main sequences of saccades and fast-phases were identical (Guitton & Mandl, 1980; Ron et al., 1972). However, closer examination revealed that the main sequences for fast-phases contained slightly longer durations and lower peak velocities (Garbutt, Harwood, & Harris, 2001; Kaminiarz, Königs, & Bremmer, 2009a). Further evidence that fast-phases and saccades share overlapping circuitry is shown in the latency distributions of these two eye movements. For example, the distribution of fast-phase intervals is similar to that of both visually evoked and spontaneous saccades (Carpenter, 1993; Roos, Calandrini, & Carpenter, 2008) and includes very short latencies possibly analogous to express saccades (Carpenter, 1994; Fischer et al., 1993). Additionally, OKN latency distributions can be modelled by accumulator models originally designed for saccades, such as LATER (Carpenter & Williams, 1995; Roos et al., 2008). The comparable main sequences and latency distributions of fast-phases and saccades imply that there are shared mechanisms in the generation of these two eye movements. Indeed for some authors the terms 'fast-phase' and 'saccade' are often used interchangeably when discussing nystagmus (Baloh et al., 1980; Cheng & Outerbridge, 1974).

However, fast-phases of OKN and targeting saccades do show clear differences in the neural structures which generate them. Whilst the fast-phases of OKN are usually considered to be generated entirely through subcortical brainstem areas, such as the reticular formation (Anastasio, 1997; Curthoys, 2002), saccades are thought to also involve processing in higher-level areas such as the superior colliculus, the frontal eye fields and the supplementary eye fields (Scudder, 1988). Accordingly functional imaging suggests that while saccades involve processing in higher-level cortical areas, the fast-phases of OKN do not (Kashou et al., 2010; Konen et al., 2005). However, whilst brainstem regions seem to be the minimum neural

substrate required to generate fast-phases, it is unclear whether higher level oculomotor areas are also involved in fast-phase generation.

1.5 A Case for Modularity

The previous sections argue that gaze-stabilizing and target-selecting eye movements are heavily integrated, and that one cannot draw a sharp distinction between automatic and volitional processes. Furthermore this thesis claims that a moving observer naturally viewing scenes would be best served by a system that did not separate gaze-stabilizing and target-selecting processes into discrete elements. However, there remain strong arguments for why the opposite might be true, and there are potential benefits for having separate gaze-stabilizing and target-selecting systems.

One such benefit may be efficiency of processing. Gaze-stabilizing and target-selecting are two separate requirements, each with their own purpose; Fodor (1983) argues that for the brain to most efficiently utilize incoming information then that information should only be processed by the necessary brain areas. Therefore, the brain should process information in discrete modules, each specifically tailored to accomplish a particular task (Fodor, 1983, 1985). As processing information is resource-dependent, then natural selection should drive brain organisation to become as efficient as possible, which may mean that a modular organisation is most advantageous (Barrett & Kurzban, 2006).

A further advantage that would come from separating automatic and volitional processes is that different processing strategies could then be employed. Fodor (1983) stated that different information processing strategies could only be employed if modularity existed in the brain. To elaborate: Fodor (1983) drew a distinction between perception and higher cognition; perception requires fast processing of information, at the expense of accuracy of information processing. This allows us to react quickly to incoming sensory evidence, which

may be necessary for our survival, however there is a chance that our interpretation of incoming sensory evidence will be incorrect (thus giving rise to such phenomena as illusions). Higher cognition on the other hand (for example decision making) does not require such rapid responses; furthermore the costs of making an incorrect decision may be higher than the cost of incorrect perceptual interpretation. Therefore nature has it both ways, a trade off is struck such that higher cognition is processed slowly and deliberately, and perception arises from rapid and sometimes inaccurate processing (Fodor, 1983). However, such different processing strategies would not be able to be implemented unless perception and higher cognition were served by separate and discrete modules (Fodor, 1983, 1985).

Whilst I do not wish to debate the cases for and against Fodor's (1983) theories on modularity (however the interested reader may wish to see Barrett & Kurzban, 2006), the above examples highlight that there are indeed potential benefits to modularity. Therefore, although this thesis will argue for integration of volitional and automatic processes, if such a strategy were employed by the brain then it may be that such integration would impinge upon efficient or strategic information processing.

1.6 Infantile Nystagmus Syndrome

The final experiment of this thesis was conducted with participants who show infantile nystagmus syndrome (INS). In the remainder of this chapter, the characteristics of INS will be outlined, with emphasis placed on the visual perception and oculomotor control of those with INS. As INS is a pathological eye movement, it is completely involuntary; therefore represents a very interesting case for comparing automatic and volitional actions.

Infantile Nystagmus Syndrome (INS) describes a syndrome of pathological oscillations of the eyes. It is estimated to affect 10 to 24 in every 10,000 people (Abadi & Bjerre, 2002; Sarvananthan et al., 2009). Oscillations are almost invariably conjugate,

symmetrical and horizontal (Abadi & Dickinson, 1986). Although not usually present at birth, it is commonly established by about three months of age (Ehrt, 2012; Sarvananthan et al., 2009). For this reason the term 'infantile' tends to be used to describe this syndrome, rather than the previous term, 'congenital nystagmus' (Abadi & Bjerre, 2002). While the oscillatory movement of the eyes is continuous (except during sleep [Abadi & Dickinson, 1986]) there is usually a specific eye-in-head position in which intensity of nystagmus is minimal; this is commonly referred to as the null zone (Abadi & Bjerre, 2002).

Whilst twelve types of INS waveform have been identified, waveforms are often grouped into three broad categories: jerk, pendular, or pseudo-pendular (Dell'Osso & Daroff, 1975). Jerk INS is characterised by slow drifts away from fixation with increasing velocity. These are interspersed with resetting fast-phase jumps to bring the fovea back to the desired location. Pendular waveforms consist of slow, smooth eye movements which bring the fovea away and subsequently back to the target. Pseudo-pendular waveforms show the same slow oscillation as the pendular; however there are small fast-phases at either peak of the waveform. These consist of either braking saccades which stop the runaway slow-phase and initiate a slow-phase back to desired gaze location, or foveating saccades which re-establish foveation following the end of a slow phase (Dell'Osso & Daroff, 1976). Usually one can identify points in the waveform where gaze is maintained upon the desired target (these are to be found following the fast-phases of jerk nystagmus and following the foveating fast-phase of pseudo-pendular nystagmus, or at one of the peaks of the pendular waveform). These points are commonly called 'foveation periods' (Abadi & Bjerre, 2002; Dell'Osso, 1986).

Although jerk, pseudo-pendular and pendular waveforms appear very different in form, there are close relationships between all three waveform types. It is has been reported that often jerk or pseudo-pendular waveforms can emerge from pendular nystagmus during infancy (Abadi & Bjerre, 2002; Harris & Berry, 2006a) and adults with jerk nystagmus can

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show pendular oscillations during periods of inattention (Abadi & Dickinson, 1986; Wang & Dell'Osso, 2011). Also it is not uncommon for prolonged recording of nystagmus to reveal expression of more than one waveform type (Abadi & Dickinson, 1986). For this reason all three types of INS are assumed to have a common cause.

Another type of nystagmus which we will consider here is that of latent nystagmus. This type of nystagmus is usually only revealed during the occlusion of one eye and is characterised by slow-phases of *decreasing* velocity (Dell'Osso, 1982). This fundamental difference in the velocity profile of the slow-phase means latent nystagmus is not usually considered to be a sub-type of INS, but is rather a completely different eye-movement (Dell'Osso, 1982). Furthermore it is possible for an individual to show both INS and latent nystagmus (Abadi, 2002).

Figure 1.1 shows example eye-traces we have collected from three participants. Figure 1.1 Panel A displays the jerk waveform (note the increasing velocity of the slow-phase). Panel B shows the pseudo-pendular waveform with braking and foveating saccades at either peak of a slow oscillation. Lastly, Panel C shows the waveform from a latent nystagmus participant (note the decreasing acceleration of the slow-phase).
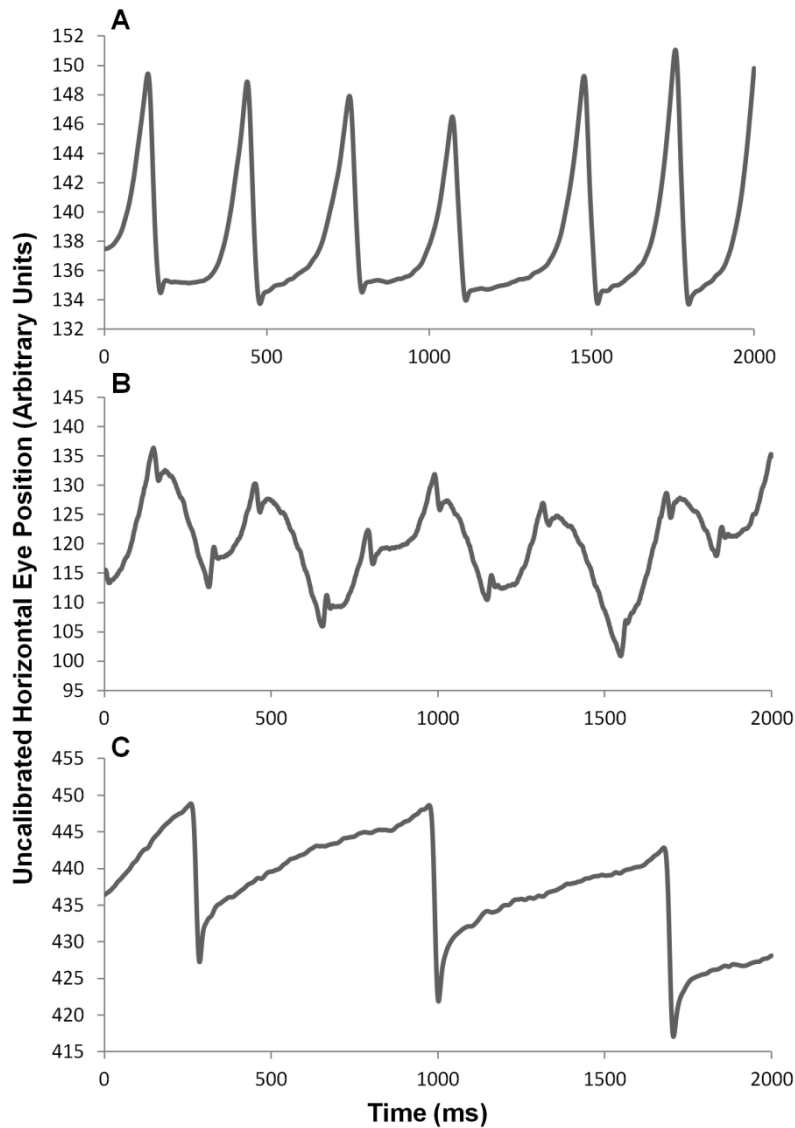
Figure 1.1: Example waveforms from, A: Jerk Nystagmus; B: Pseudo-pendular Nystagmus; C: Latent Nystagmus.

1.7 Aetiology of Infantile Nystagmus Syndrome

The cause of INS is subject to a continuing debate. INS presents alongside a wide range of ocular pathology including (but not limited to) albinism, congenital cataracts, optic nerve hypoplasia, retinal diseases such as achromatopsia, and Down's Syndrome (Averbuch-Heller, Dell'Osso, Jacobs, Jacobs, & Remler, 1999; Ehrt, 2012; Harris, 2011; Sarvananthan et al., 2009). The numerous ocular deficits associated with INS make it difficult to establish a causal relationship, and furthermore a sizable proportion of INS cases do not appear to be

associated with any ocular pathology whatsoever (usually referred to as idiopathic INS) (Harris, 2012; Sarvananthan et al., 2009). Therefore most models seek to explain INS through malfunction of an otherwise intact oculomotor system; rather than through neurological damage or ocular pathology.

1.7.1 *Models based upon gaze-holding malfunction*

An intuitive hypothesis for the occurrence of INS is that it results from a disorder of a gaze-holding network. For example, the intensity of nystagmus is at its lowest during periods of inattention, and increases when fixation attempts are made (Abadi & Dickinson, 1986; Tusa, Zee, Hain, & Simonsz, 1992; Wang & Dell'Osso, 2011). It is proposed that in the normal oculomotor system gaze holding is achieved through feedback from velocity integrators which would cancel out any post-saccadic drifts in fixation (Optican & Zee, 1984). INS is theorized to occur when the sign of this feedback is reversed, making the neural integrator unstable; therefore post-saccadic drifts are amplified resulting in exponentially growing slow-phases (Optican & Zee, 1984).

While this model is intuitive, and can successfully simulate many of the observed INS waveforms (Tusa et al., 1992), it cannot account for pure pendular waveforms (Jacobs & Dell'Osso, 2004). This model also predicts that there should be two null zones, however the existence of two null zones has never been observed empirically (Harris, 1995b). Moreover it cannot account for how some individuals with INS are able to maintain fixation for several hundred milliseconds before the slow-phase is initiated (Jacobs & Dell'Osso, 2004). Furthermore, this model relies on congenital neural misrouting to reverse the sign of the velocity feedback loop; whilst this is possible in achiasmia resulting from albinism, it is difficult to establish how this misrouting would occur in those with idiopathic INS (Abadi, 2002).

1.7.2 *Models based upon smooth-pursuit malfunction*

Some authors believe that the genesis of INS lies in the slow eye movements generated by the smooth pursuit system (Dell'Osso, 1982). This model states that the onset of pursuit is accompanied by a ringing of the pursuit system, which is damped under normal oculomotor functioning (Jacobs & Dell'Osso, 2004). INS results when this ringing is under-damped, which would cause pendular oscillations of the eye. However abnormal feedback loops whose gain is too high cause the characteristic increasing velocity of slow-phases (Dell'Osso, 2006; Harris, 1995; Jacobs & Dell'Osso, 2004). Braking or foveating saccades are then executed to bring the eye back to its desired location (Dell'Osso, 2006; Dell'Osso & Daroff, 1976; Jacobs & Dell'Osso, 2004).

A strength of this model is that it is able to generate normal oculomotor functioning as well as nystagmus (Jacobs & Dell'Osso, 2004) and so mirrors the apparenlty normal saccades and smooth pursuit present in those with INS (Bedell, Abplanalp, & McGuire, 1987; Dell'Osso, 2006; Wang & Dell'Osso, 2007, 2009). Although initially an account only of pendular or pseudo-pendular waveforms, this model has recently been extended to be able to account for jerk nystagmus (Wang & Dell'Osso, 2011). It remains unclear as to why the smooth pursuit system would operate in this way, however it has been theorised that it could be due to early visual deprivation (Harris, 1995b). For example, monkeys reared with visual deprivation during infancy show a deficit in initial smooth pursuit, as well as spontaneous eye movements with the charactersitics of INS (Tusa, Becker, Mustari, & Fuchs, 1994)

1.7.3 *Models based upon saccadic malfunction*

A competing hypothesis attributes abnormality in the saccadic system as the cause of INS. In the normally functioning oculomotor system saccades are initiated by the firing of burst cells, and subsequent fixation is achieved by the steady firing of pause cells (Leigh & Zee, 1999). This model proposes that disorders in these pause cells may give rise to INS by disrupting

saccadic termination (Akman, Broomhead, Abadi, & Clement, 2005; Broomhead et al., 2000). Mathematical modelling of this hypothesis is able to simulate INS waveforms which other models cannot (Akman, Broomhead, Clement, & Abadi, 2006; Broomhead et al., 2000). Recently it has tentatively been proposed that this process occurs from an imbalance in the firing of saccadic burst generators and the fixation-related cells found in the rostral pole of the superior colliculus (Akman, Broomhead, Abadi, & Clement, 2012).

If the saccadic system is abnormal in those with INS then one would expect to see differences in the voluntary saccades of those with INS and normal controls. Indeed early support for this model came from the observation that voluntary saccades made by those with INS had a lower peak velocity, and were more inaccurate than voluntary saccades made by control subjects (Abadi & Worfolk, 1989; Worfolk & Abadi, 1991). However, voluntary saccades made by those with INS appear normal when one takes into account the summation or cancellation effects of a saccade occurring simultaneously with underlying nystagmus movement (Bedell et al., 1987; Jacobs & Dell'Osso, 2004). Furthermore voluntary saccades made by those with INS show the same main sequence as normal individuals, implying the core neural processes are the same in both groups (Dell'Osso, 1973; Dell'Osso, Gauthier, Liberman, & Stark, 1972; Yee, Wong, Baloh, & Honrubia, 1976).

1.7.4 *Models based upon optokinetic reflex malfunction*

A further possibility is that INS arises due to abnormalities in the optokinetic system. This assertion is based largely upon the observation that individuals with INS can show abnormal, reversed OKN (where the slow-phase is against the direction of stimulus motion) (Halmagyi, Gresty, & Leech, 1980; Yee, Baloh, & Honrubia, 1980) or they show no optokinetic response at all (Ehrt, 2012; Leigh, Robinson, & Zee, 1981). Furthermore individuals with INS do not appear to show optokinetic after-nystagmus (transient continuation of OKN observable when participants are immediately placed into darkness following OKN); this could indicate that

those with INS have substantial differences in the neural processes underlying their OKN (Demer & Zee, 1984; Yee et al., 1980). Additionally an animal model of nystagmus has been created using zebrafish with achiasmia; these fish also show reversed OKN (Huang, Rinner, Hedinger, Liu, & Neuhauss, 2006). However whilst achiasmia has some relevance to nystagmus due to albinism, it is hard to see how this can account for idiopathic INS.

The reversed OKN in those with INS is interesting, and an absence of OKN may be used as a method in diagnosing neonates with INS (Ehrt, 2012); however caution must be exercised in reading too much into these results. This is because OKN in those with INS is not a true optokinetic response, rather it is the individual's own INS superimposed upon the OKN waveform (Dell'Osso, Van der Steen, Steinman, & Collewijn, 1992b; Harris, 1995b). Conclusions are further compounded by the possibility that optokinetic stimulation may shift the null zone of an individual with INS, causing unpredictable changes to their nystagmus (Harris, 1995b; Kurzan & Büttner, 1989).

1.7.5 *Models based upon evolutionary developmental biology*

It could be argued that the models mentioned above in sections 1.7.1 through to 1.7.4 emphasise *how* the oculomotor system might generate spontaneous oscillations of the eyes; however they are less clear as to why these oscillations occur in the first place. Contrastingly, one attempt to answer why nystagmus should occur at all comes from the evolutionary-developmental model of Harris (2011). This model notes that there is considerable development of the oculomotor system during infancy (Luna & Velanova, 2011); and proposes that this development seeks toward a state in which oculomotor behaviour maximises visual acuity. This end state is plastic, however in a normally developing system it would settle upon a strategy of affixation of the fovea upon objects of regard, interspersed with rapid reorienting of the fovea toward new objects (i.e. fixation or smooth pursuit interspersed with saccades) (Harris & Berry, 2006a).

Interestingly, during early infancy this 'fixate and saccade' strategy would not actually be the optimal behaviour of the oculomotor system (Harris & Berry, 2006b). This is because vision during early infancy is dominated by low spatial frequencies (Courage & Adams, 1990; García-Quispe, Gordon, & Zemon, 2009) and contrast sensitivity for low spatial frequencies can be improved with the addition of retinal motion (Burr & Ross, 1982). Therefore, paradoxically, the optimal behaviour of the oculomotor system under these conditions is to generate rhythmic eye movements, which closely resemble those waveforms characteristic of INS (Harris & Berry, 2006a, 2006b).

Why then do not all people develop nystagmus? Harris (2011) proposes that under normal conditions oscillatory movements of the eyes are prevented because the smooth pursuit system does not develop until around three months of age (Hofsten & Rosander, 1997). By this age visual acuity is sufficiently developed so that higher spatial frequencies are able to be resolved; thus the optimal strategy for the oculomotor system is no longer to move the eyes continuously, but rather to adopt the 'normal' strategy of fixation and saccades (Harris & Berry, 2006a, 2006b). Therefore, in this model, INS occurs when the smooth pursuit system develops before visual acuity is ready for it. The resolution of higher spatial frequencies does develop in those with INS, often at a slower rate than those without nystagmus (Weiss & Kelly, 2007), but by this time plasticity in the system has ceased, and the nystagmus behaviour is set (Good, Hou, & Carden, 2003; Harris & Berry, 2006b).

The delayed development of visual acuity relative to smooth pursuit might be due to any one of the myriad of ocular deficits associated with nystagmus (Ehrt, 2012). However it is also possible that no ocular deficit exists whatsoever, rather there just so happens to be delayed visual development, or precocious development of the smooth pursuit system (Harris, 2011). The out of order development of these systems may well have a genetic basis, as it has been shown that INS has a mild heritability (Abadi & Bjerre, 2002; Ehrt, 2012).

Hence this model allows for the possibility of a structurally normal oculomotor system, which has settled on a strategy which was optimal and adaptive at a time in which high spatial frequencies were not able to be resolved.

1.8 Visual Perception during Infantile Nystagmus Syndrome

Typically, visual acuity in those with INS is poorer than those with a normally functioning oculomotor system (D. Yang, Hertle, Hill, & Stevens, 2005), although it is possible for certain individuals to have normal visual acuity (Bedell & Loshin, 1991). As INS is associated with a large variety of ocular pathologies, the presence of reduced visual function it is not altogether surprising (Harris, 2011). Nevertheless, even those individuals with idiopathic INS, where no ocular deficit has been found, tend to have poorer visual acuity than control subjects (Abadi & Sandikcioglu, 1975). Logically some of this degradation in visual acuity is attributable to the fact that the eyes are constantly moving, resulting in retinal smearing. For instance acuity is better in those participants who can maintain a longer foveation period, and who can consistently return their fovea to the same location in each waveform (Cesarelli, Bifulco, Loffredo, & Bracale, 2000; Dell'Osso, Van der Steen, Steinman, & Collewijn, 1992a). Additionally, acuity is significantly worse for gratings oriented orthogonally to the slow-phase direction, as these are subject to increased retinal smearing (Abadi & Sandikcioglu, 1975; Dickinson & Abadi, 1992); this grating orientation effect is not seen for normal observers (Meiusi, Lavoie, & Summers, 1992).

However, despite some of the loss in visual acuity attributable to eye motion, the degree of acuity loss is greater than the eye movements themselves would suggest. For example acuity does not seem to be correlated with nystagmus velocity, amplitude or frequency (Bedell & Loshin, 1991; Von Noorden & La Roche, 1983). Moreover acuity is still

superior in a normal observer even if the stimuli they are presented with have the same retinal motion as an individual with nystagmus (Chung & Bedell, 1995).

One of the fundamental characteristics of INS is that despite the constant, involuntary movement of the eyes the perception of oscillopsia (the world moving) is very rare (Bedell, 2000). This is contrary to nystagmus which is acquired later in life through brain injury or disease, where oscillopsia is present (Ehrt, 2012; Sarvananthan et al., 2009). There are multiple theories as to how this perceptual stability is achieved. One possibility is that individuals with INS have a reduced sensitivity to motion. Motion detection thresholds in those with INS have been shown to be significantly higher when motion is in the same direction as the nystagmus waveform (this is true for both horizontal [Dieterich & Brandt, 1987; Shallo-Hoffmann, Bronstein, Acheson, Morland, & Gresty, 1998] and vertical nystagmus [Dieterich, Grünbauer, & Brandt, 1998]).

However, other authors have argued that oscillopsia is prevented through extra-retinal signals accompanying nystagmus movement (Bedell, 2000). The existence of extra-retinal signals is strongly suggested by the observation that if a participant with INS is presented with a stabilized retinal image, then they will report oscillopsia (Leigh, Dell'Osso, Yaniglos, & Thurston, 1988). However spatial stability is still maintained during partial retinal stabilization (Abadi, Whittle, & Worfolk, 1999) and the extra-retinal signal accompanying INS is reported to underestimate eye movement (Bedell & Currie, 1993); therefore a combination of strategies seems to be the likely method by which oscillopsia is prevented.

Extra-retinal signals may also aid veridical perception during nystagmus by attenuating motion smear; for example perceived motion smear is reduced when targets move against the slow-phase direction (Bedell & Tong, 2009). This attenuation of motion smear is significantly greater than normal controls report when they are presented with matched retinal

motion (Bedell & Tong, 2009). The reduction in motion smear is intriguing, as it mirrors the reduction in motion smear seen in normal observers during smooth pursuit (Bedell & Lott, 1996; Tong, Aydin, & Bedell, 2007), smooth vergence eye movements (Bedell, Chung, & Patel, 2004) and the vestibular-ocular reflex (Bedell & Patel, 2005). This might imply that infantile nystagmus and slow eye-movements are generated by the same system in both individuals with INS and normal subjects, lending support to models which state INS arises from activity in the smooth pursuit system (see section 1.7.2).

1.9 Oculomotor Control during Infantile Nystagmus Syndrome

Despite the constant movement of the eyes during INS, it is clear that there is an attempt to maintain gaze upon a particular location (Dell'Osso et al., 1992a; Tusa et al., 1992). Therefore gaze direction may need to be maintained using the same gaze-stabilizing eye-movements as exist in normal observers (Dell'Osso et al., 1992a). Additionally, when an individual with INS wishes to redirect their gaze to another location in space, this must be achieved using the same mechanisms as in the normal oculomotor system; namely smooth pursuit and saccades (Dell'Osso, 2006; Jacobs & Dell'Osso, 2004).

1.9.1 *Gaze stabilizing eye movements during infantile nystagmus*

In the normal oculomotor system, gaze can be stabilized during self motion using optokinetic and vestibular-ocular reflexes (Leigh & Zee, 1999). It has already been mentioned above (section 1.7.4) that the optokinetic reflex can appear absent or inverted in those with INS (Demer & Zee, 1984; Halmagyi et al., 1980; Yee et al., 1980). However, one must be very cautious in assuming this reflects an impaired optokinetic system, as it may be that normal optokinetic nystagmus is hidden by ongoing infantile nystagmus (Harris, 1995b; Kurzan & Büttner, 1989).

Typically the vestibulo-ocular reflex (VOR) is reported to be normal in INS (Dell'Osso, Van der Steen, Steinman, & Collewijn, 1992c). The gaze location of foveation periods remains constant during head movements, with a gain comparable to that of a normal observer (Demer & Zee, 1984; Kurzan & Büttner, 1989). Some individuals with INS show a characteristic head shaking behaviour which could theoretically help to cancel out nystagmus movement if VOR were absent or incomplete (Carl, Optican, Chu, & Zee, 1985). However upon investigation, whilst one subject was able to employ this strategy, it was not seen in any other observers, all of whom had normal VOR function (Carl et al., 1985).

1.9.2 *Smooth pursuit during infantile nystagmus*

None of the models presented above (sections 1.7.1 to 1.7.5) would predict anything other than a normal smooth pursuit system in those with INS. In the model of Jacobs and Dell'Osso (2004) which states that INS is generated by the smooth pursuit system, it is explicitly stated that the pursuit system is functionally intact (see section 1.7.2).

Early investigations into smooth pursuit during nystagmus reported that smooth pursuit was either not present, very inaccurate, or even reversed (Dell'Osso, 1986; Leigh et al., 1981). However these findings failed to take into account the superimposition of the nystagmus waveform upon the smooth pursuit movement (Dell'Osso, 1986; Dell'Osso et al., 1992b). Furthermore smooth pursuit can shift the null zone of nystagmus, causing changes to the waveform which may be interpreted as abnormal pursuit movements (Kurzan & Büttner, 1989). If one assumes that the foveation period represents desired gaze location, then interpolation of gaze location during each foveation period reveals that the target is tracked normally (Dell'Osso, 1986; Dell'Osso et al., 1972; Dell'Osso et al., 1992b). Consequently there is no indication that the smooth pursuit system functions abnormally during INS (Dell'Osso, 2006; Kurzan & Büttner, 1989).

1.9.3 *Saccades during infantile nystagmus*

Voluntary saccades made by those with INS show an identical main sequence to voluntary saccades made by normal observers (Dell'Osso, 1973; Dell'Osso et al., 1972; Yee et al., 1976). This would imply that the saccadic system functions normally in those with INS. However, there are some circumstances in which those with INS appear to exhibit unusual saccadic behaviour; for example rapid target displacements in the direction opposite to the fast-phase are frequently acquired through a slow-phase of nystagmus, rather than a saccade (Bedell et al., 1987; Yee et al., 1976). Whilst this might imply a failure of the saccadic system to acquire the target, it has been postulated that this is actually perfectly normal oculomotor behaviour when one considers the retinal stimulation of a target step during nystagmus (Bedell et al., 1987). As such this behaviour is analogous to some step-ramp oculomotor tasks employed in normal observers (Bedell et al., 1987).

Further experiments revealed that voluntary saccades made by those with INS do appear to be more inaccurate than in normal observers (Worfolk & Abadi, 1991); however this inaccuracy may be due to interactions between the saccades and the fast-phases of nystagmus. For example, when target displacements are made in the same direction as fast-phases then the resulting saccade usually overshoots the target; similarly, when the target displacement is in the opposite direction to the fast-phases then the resulting saccade will undershoot the target (Worfolk & Abadi, 1991). This could be because the desired end-point of the fast-phase and the desired end-point of the voluntary saccade interact in a way analogous to the global effect; therefore the ensuing saccade will be directed to a point between the two loci of activity (Worfolk & Abadi, 1991).

The time taken to plan and execute voluntary saccades during INS also seems to be related to the fast-phase. For example saccade latency seems to be slightly longer in those with INS, and especially long if the target jump is at around the time of the fast-phase (Wang

& Dell'Osso, 2007). This might imply that the processes underlying a fast-phase interfere with the generation of voluntary saccades. Although it is not clear how the perception of targets might be altered around the time of the fast phase (as it is well known that there are perceptual biases around the time of saccades [Ross, Morrone, Goldberg, & Burr, 2001] or OKN fast-phases [Kaminiarz, Krekelberg, & Bremmer, 2007]).

For activity related to the fast-phase to interact with that producing voluntary saccades might imply that they are generated by the same neural networks, which poses the question of whether fast-phases in INS are identical to voluntary saccadic movements? INS is frequently stated to be a nystagmus of the pursuit system (Dell'Osso, 1982; Jacobs & Dell'Osso, 2004) and therefore the fast-phases are nothing more than corrective saccades designed to bring the eye back to an appropriate position (Yee et al., 1976). The fast-phases of INS have reportedly the same main sequence as voluntary saccades made without a visual target (Abadi & Worfolk, 1989). Also the peak latency between fast-phases seems to be the same as the peak intersaccadic latency in normal observers, suggesting these eye-movements are generated by the same system (Bosone, Recci, Roberti, & Russo, 1990). A final point of evidence that saccades and fast-phases are generated by the same mechanisms comes from dynamic overshoots. These are small corrective eye movements executed immediately after a saccade, and in a direction opposite to the saccade. These are present in the fast-phases of INS (indeed they are clearly visible in Figure 1.1), and have the same main sequence as the dynamic overshoots of voluntary saccades (Abadi, Scallan, & Clement, 2000).

Therefore voluntary saccades made by those with INS appear to show the same behaviour as voluntary saccades made by those with a normally functioning oculomotor system. The exception to this is where voluntary saccades interact with the fast-phases of infantile nystagmus; which may be evidence that fast-phases are processed in a very similar way to voluntary saccades.

1.10 Thesis Overview

In the preceding sections the basic gaze-stabilizing and target selecting eye movements have been outlined (Sections 1.1 and 1.2). Furthermore it has been summarised how gaze-stabilizing and target selecting eye movements are frequently considered as separate and distinct – the former being automatic, reflexive and stimulus driven, the latter volitional, top-down and goal-directed (Section 1.3). However, to achieve natural viewing behaviour in an active observer, target selecting and gaze-stabilizing eye movements cannot exist in complete isolation. Evidence of close co-ordination between target selecting and gaze-stabilizing eye movements is reported (Section 1.4). Such co-ordinated behaviour may indicate that automatic and volitional eye movements are not processed entirely separately in the oculomotor system, and therefore it may be the case there is no great distinction between volitional and automatic processes in the oculomotor system. Lastly, the characteristics of INS were outlined. INS represents an invaluable case for testing whether the co-ordination which was found between automatic and volitional eye movements using OKN can extend to a pathological eye movement. This can help inform our views of what automatic and volitional processes entail, and may help elucidate some of the oculomotor functioning of those with INS.

In Chapter 2 to Chapter 6, experimental work will be described which investigates the interface of targeting eye movements and gaze-stabilizing OKN. Chapter 2 will discuss the first experiment: the accuracy of goal-directed saccades executed simultaneously with reflexive optokinetic nystagmus. This experiment was conducted to answer the question of whether reflexive eye movements can spatially update volitional eye movements. If spatial updating between reflexive and volitional eye movements is possible, it would be evidence against a sharp separation between reflexive and volitional actions; and furthermore would

give a potential mechanism for the co-ordination of targeting and gaze-stabilizing eye movements elicited when a moving observer naturally views scenes.

Proceeding directly from this work, Chapter 3 examines whether saccadic behaviour during optokinetic nystagmus is related to the misperception of location which occurs during OKN (Kaminiarz et al., 2007; Tozzi, Morrone, & Burr, 2007), and compares this behaviour to misperception during voluntary smooth pursuit. Chapter 4 begins to look at the influence ongoing OKN can have on saccades by examining saccade curvature during OKN; and Chapter 5 will build upon these results by using the saccadic inhibition paradigm (Reingold & Stampe, 1999, 2000, 2002) to ask whether the fast-phases of OKN share some of the same 'higher-level' characteristics as saccades. Finally, Chapter 6 will move away from optokinetic nystagmus in order to look at infantile nystagmus syndrome. Chapter 6 examines how the saccadic inhibition effect used in Chapter 5 applies to the fast-phases of infantile nystagmus; and what this can tell us about the relationship between targeting saccades, OKN fast-phases and the fast-phases of infantile nystagmus. The conclusions of this work, along with general implications and findings are discussed in Chapter 7.

Chapter 2: The Accuracy of Saccades Executed During Concomitant Optokinetic Nystagmus

2.1 Introduction

As an active observer moving through a scene with numerous sites of attention, eye movements intended to foveate targets of interest must co-occur with eye-movements required to stabilize the retinal image. Intentional foveation and fixation of a specific point is achieved through saccades and smooth-pursuit eye-movements; whereas more automatic gaze stabilization is achieved through multiple processes, notable of which are the vestibular-ocular reflex (VOR) and optokinetic nystagmus (OKN) which rotate the eye in order to negate the rotatory component of retinal motion which would otherwise occur during self-movement (Findlay & Gilchrist, 2003; Heinen & Keller, 2004; Leigh & Zee, 1999). Saccades, smooth pursuit, VOR and OKN have an intimate relationship: all of these eye-movements are elicited during the viewing of natural scenes in a moving observer (Daye et al., 2010; Moeller et al., 2004; Pelisson & Prablanc, 1986) and it has even been proposed that saccades and smooth pursuit arose through the evolution of purposeful control over phylogenetically older reflexive VOR and OKN (Post & Leibowitz, 1985; Ron et al., 1972; Walls, 1962). In spite of this, target selection and gaze stabilizing mechanisms are frequently regarded as independent and discrete processes: the former being top-down, volitional and goal-directed, and the latter bottom-up, reflexive and stimulus-driven (Post & Leibowitz, 1985). There has been very little work to date on saccades made during concomitant gaze-stabilizing processes generally, and especially saccades made during OKN.

An accurate saccade during ongoing OKN requires the saccadic system to integrate the displacement of the eye that occurs during the planning and initiation of the saccade. However some authors claim that automatic and volitional motor actions are separate and

independent, residing in different neural structures (Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977; Theeuwes et al., 1998; Theeuwes et al., 1999). Therefore it is unclear as to whether the necessary interconnections would exist to allow reflexive movements of the eyes to spatially update the motor maps which volitional actions rely upon.

However there is evidence that involuntary eye-movements can update the spatial maps of higher-level motor actions. For example it has been shown that perception of a target's location during rebound nystagmus (an involuntary eye-movement which occurs following prolonged gaze holding at large eccentricities [Leigh & Zee, 1999]) is essentially veridical for short-duration targets (Bedell & Currie, 1992; Currie & Bedell, 1991; Lott & Bedell, 1995). A similar result has also been reported for Optokinetic After-Nystagmus for both seen and unseen pointing (Bedell, 1990; Bedell et al., 1989). Moreover it has been shown that gaze can be returned to an extinguished head-fixed target following displacement due to VOR (Hansen & Skavenski, 1977). This might imply that even reflexive eye movements are able to spatially update motor maps (Bedell et al., 1989). However the ability of top-down targeting saccades to compensate for displacements due to OKN has never been tested before.

A further issue that is not investigated here, but that is of theoretical interest is whether the saccadic system has to 'know' the saccade latency in order to correct appropriately. As saccades have a variable latency, unless the saccadic system can predict the latency of the upcoming saccade it will not know where gaze will be during the OKN movement. This could enable an accurate saccade if the saccadic system had access to the velocity of the optokinetic movement. Alternatively, a moving hill of activity which was updated by eye displacement could allow accurate targeting saccades during OKN, however this strategy would not be as accurate as fore-knowledge of the latency period unless the moving hill of activity had zero lag.

This experiment aimed to test the accuracy of vertical saccades to flashed targets made during concomitant horizontal OKN. Figure 2.1 outlines two different predictions for how saccades might behave under these circumstances. If reflexive OKN is generated through neural mechanisms completely independent and separate from those which generate volitional saccades, then saccades should be insensitive to displacements of the eye during OKN. Therefore saccades would be executed to the retinal location of a briefly flashed target (solid line in Figure 2.1). However, if there is no sharp dichotomy between reflexive and volitional movements then we expect saccades made concomitantly with OKN to be accurate, and arrive at the target's spatial location (dashed line in Figure 2.1).
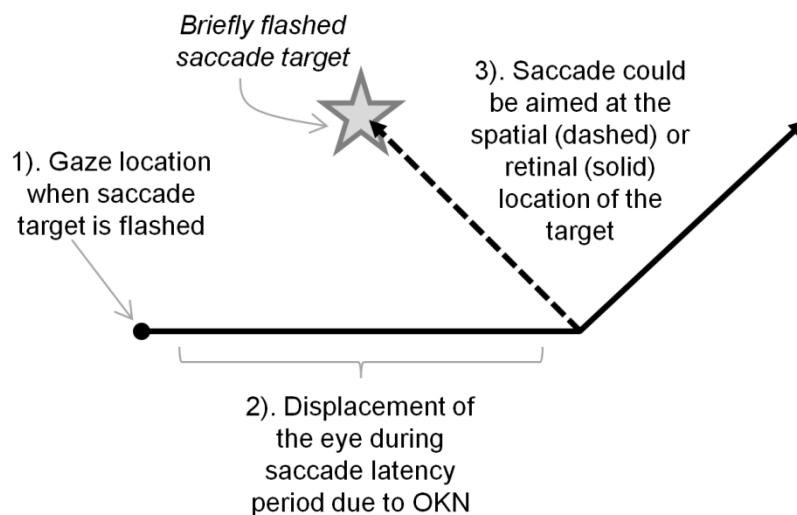
Figure 2.1: A saccade executed simultaneously with OKN will be displaced during the saccade latency period. If the saccadic system is sensitive to OKN activity, then the saccade may compensate for the displacement and land on the spatial location of the target (dashed line). However if these two eye movements are programmed in isolation then the saccade may be insensitive to any displacement and instead be directed to the retinal location of the target (solid line).

Therefore, this method was used to investigate whether saccades are able to compensate for gaze-stabilizing OKN. This further allowed exploration of whether there are interconnections between these reflexive and volitional eye movements.

2.2 Experiment A1 – Saccadic Compensation for OKN Displacements

The aim of the first experiment was simply to investigate whether saccades could compensate for OKN displacements and land upon a briefly presented target's spatial location (see Figure 2.1). The task required observers to view a band of randomly moving dots in order to elicit a strong horizontal optokinetic reflex, and then make a targeting saccade to targets flashed briefly above or below the band of OKN dots.
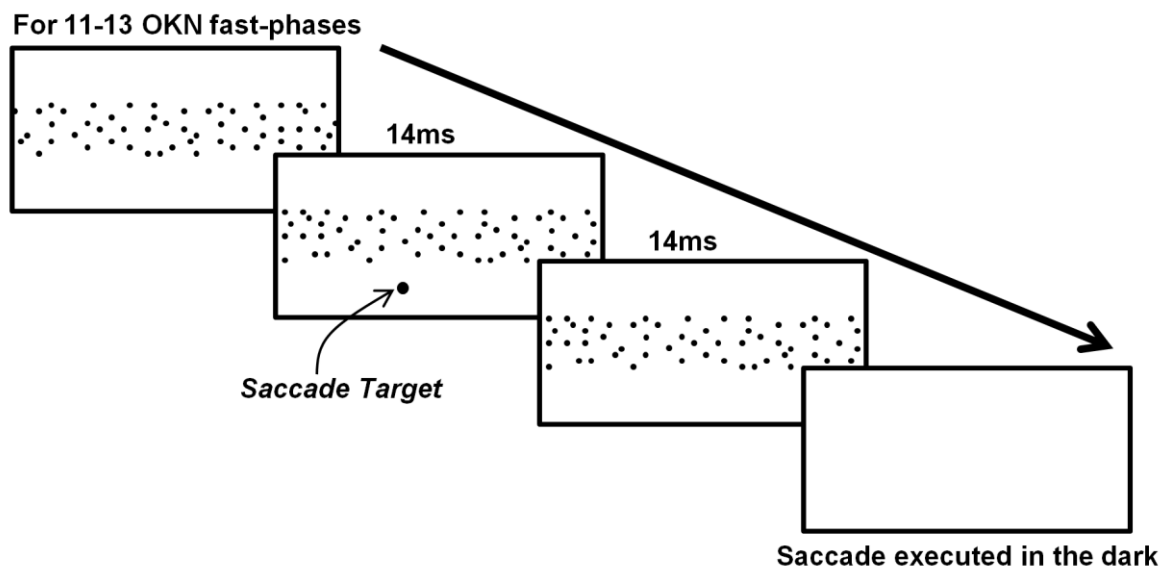
Figure 2.2: Stimuli used in this experiment. OKN is elicited using a horizontally moving band of random dots. After 11-13 waveforms a saccade target is presented for 14ms. Observers have to execute a top-down saccade to the location of the flashed target.

2.2.1 *Participants*

Four observers (three female) ranging from 22 to 24 years of age participated in this experiment in exchange for payment. This sample size was chosen as it is consistent with previous experiments that examined similar oculomotor tasks (e.g. 3-5 participants used to examine the behaviour of saccades during concomitant smooth pursuit: Hansen, 1979; Ohtsuka, 1994; Schlag, Schlag-Rey, & Dassonville, 1990; Van Beers, Wolpert, & Haggard, 2001). Sample size was fixed before testing commenced. All participants self-reported

normal vision. Two participants had previous experience with eye-tracking and two did not. All experimental procedures were approved by the ethics committee of the School of Psychology, Cardiff University.

2.2.2 *Materials*

Unless otherwise stated, the materials in this section were common to all experiments.

In all experiments stimuli were rendered using OpenGL software running on a Radeon 9800 Pro graphics card. Stimuli were displayed through rear projection using a Sony Multiscan projector (VPH 1272QM) onto a large screen (2.08×1.56 meter, 1024×768 pixels) at a refresh rate of 72Hz. The screen had an embedded Fresnel lens, which collimated light evenly throughout the display. Gamma correction was achieved using standard techniques. Only the central 'green' cathode ray tube of the projector was used, and 0.9 neutral density filter was placed over the projector. Other than the presented stimuli the lab was completely dark.

Participants were seated 140cm from the screen and viewed the stimuli binocularly. Their head position was maintained through the use of a chin and forehead rest. Eye movements were recorded using an SR Eyelink 2000 eye-tracker mounted on the chin and forehead rest. The eye-tracker recorded eye-movements at a rate of 1000Hz using standard video based technology. All experiments used the same calibration procedure. Participants were required to fixate nine points arranged in a 3×3 square grid. Each calibration point was separated by 10°. Calibration points were accepted manually. Calibration accuracy was checked by the experimenter prior to commencing recording, and calibration was repeated if necessary.

2.2.3 *Stimuli*

OKN was elicited by presenting observers with a band (16° high, 73° wide) of coherently moving random dots (radius = 0.3°, brightness = $0.1cd/m^2$, density of 0.5 $dots/deg^2$, speed 32°/sec, randomly either to the left or right). The 4° at extreme left and extreme right of the display were faded so that the horizontal edges were indiscernible; this was to limit the use of the horizontal screen edge as a fixed external reference point. Between each trial a blank screen of brightness $0.38cd/m^2$ was displayed for five seconds to stop participants from dark-adapting during the experiment, which might have allowed them to perceive the external stationary features of the room; which can disrupt OKN. To allow other stimuli to be presented at specific points in the OKN waveform, on-line detection of fast-phases was achieved using a velocity criterion of 92°/sec. On 25% of trials the band of dots remained stationary to measure saccades without concomitant OKN, and on the other 75% of trials the band of dots moved at 32°/sec, randomly either to the left or right. This stimulus was used to elicit OKN in all further experiments.

The saccade target consisted of a dot with a radius of 0.6 degrees ($1.06cd/m^2$). This was presented for 13.8ms (one frame at a refresh rate of 72Hz). The target was positioned either 10° above or below the vertical centre of the screen (therefore 2° above or below the band of OKN-dots) and was randomly presented within 4° to either side of the horizontal centre of the screen (in subsequent experiments it was noted that presenting the target within 4° of the centre of the screen might create a bias whereby participants would saccade towards the centre of the screen, therefore in all experiments subsequent to this the horizontal location of the target was presented at ±4° of current gaze location). Presentation of the saccade target was yoked to the participants' eye movement to allow greater control of when the target was presented with respect to the nystagmus waveform. The target was presented following 11, 12 or 13 nystagmus fast-phases (order randomised) and was presented 110, 160 or 260ms

following a fast phase (detected on-line using a velocity criterion of 92°/sec). The target onset times were chosen to allow the fast-phase to be completed (from pilot data this was estimated at taking 60ms) plus a variable time of 50, 100 or 200ms (randomly selected) to allow saccades to be elicited early, in the middle, or near the end of the slow-phase of the nystagmus. If a baseline trial was conducted (using a stationary display of dots) then the target was triggered based upon the time it would take to reach the desired number of fast-phases were the nystagmus operating at a frequency of 3Hz (an approximation of fast-phase frequency [Cheng & Outerbridge, 1974]). The order of stimuli are illustrated in Figure 2.2.

2.2.4 Procedure

Participants were sat at the eye-tracker in an otherwise dark room and told that they should passively view the band of moving dots, and upon appearance of the target, execute a saccade to the target (or the target's location if it had extinguished) as quickly and as accurately as possible. Recording was split into 10 blocks, each composed of 40 trials. Each block began with a calibration. Each trial began with a drift correct, which the participant initiated with a mouse-click. A dot 0.6° in radius was presented in the centre of the screen. Participants were required to fixate this dot for 300ms while gaze location was recorded. Any discrepancy between the recorded and actual location of the eye was then corrected for off-line on a trial-by-trial basis. The experimental trail began immediately following the drift correct. The band of random dots was viewed until the target was presented, at which point the band was extinguished. This means that any eye-movements which occurred during the saccade latency period were conducted in the dark. It has been shown that the eye will continue to move for around a second following cessation of OKN or smooth pursuit stimuli (Gellman & Fletcher, 1992; Leigh & Zee, 1999). Once the target had been presented, there was a delay of 1000ms in which the saccade could be made, followed by an inter-trial interval of 300ms before the

initiation of the next drift correct. Between each block of 40 trials the lights were turned on and participants were given the opportunity to take a short break.

2.2.5 *Data Analysis*

All eye-movement recordings were analysed off-line line using Matlab software (version 2010a, Mathworks Inc.). Analyses were all performed using custom-written Matlab code. Before any data analysis, eye traces were smoothed using a Gaussian filter (SD = 16Hz). Saccades were detected using a velocity criterion of 100°/sec, with the start of the saccade taken to be the time at which the velocity first rose above 20°/sec. Fixation was detected when the eye did not deviate by more than 0.3° over a 100ms period.

Fast-phases of the OKN waveform were identified using a combination of acceleration (location of zero-crossing), eye-velocity (average velocity across the fast-phase of at least 60°/sec), local minima and maxima of position, and direction of motion (fast-phases nearly always travel against stimulus motion). Detected fast-phases had to be more than 40ms apart to be accepted by the analysis program. To determine the velocity of slow-phases, an average velocity was calculated disregarding the 50ms immediately after and immediately prior to a fast-phase. If slow-phase velocity was over 1.5 × stimulus velocity then the detected slow-phase was flagged as an error. All trials were visually checked by the experimenter before being included in the final analysis.

Many experiments conducted in this thesis are within subjects designs. To graphically represent the data, the procedure for showing error bars outlined by Cousineau (2005) was adopted. This procedure subtracts each data point from that participant's overall mean value, and this value is then added to the grand mean. This creates a dataset where the individual differences have been removed, and the standard deviation of this new data set is used to create the error bars. This method is useful because small differences in conditions, when

present for the majority of subjects, can be significant when subjects are substantially different from one another. Partialling out the between-subject variability allows the differences between conditions to be more clearly visible. This method is not the only possible way to show error bars which are not subject to between-subject variability, the most notable alternative being that of Loftus and Masson (1994). However, the method of Cousineau (2005) was adopted because it has certain advantages over the method of Loftus and Masson (1994). For example Loftus and Masson's (1994) method utilizes the results of inferential statistics to construct error bars, which can be paradoxical since often graphical representation is required to anticipate the results of analyses. Moreover Loftus and Masson's (1994) method provides a single error bar size which may mask information about the differences in variances across conditions, and requires assumptions about which error term to use to construct the error bars if there are multiple factors present. Also Loftus and Masson's (1994) method requires adherence to the assumption of sphericity (Baguley, 2012).

However there are limitations to the use of Cousineau's (2005) method; for example normalisation to a single score will cause all scores to become correlated, which will bias variance to be lower than expected, especially for data with a large number of levels (Morey, 2008). Moreover these intervals are designed to graphically show a pattern of a set of means for informal analysis; they are not intended to mimic hypothesis tests or to serve as a 'visual statistic' (Baguley, 2012). This cannot be a criticism of the test as the method was never designed to be a 'visual statistic', nevertheless confusion will arise if the error bars are interpreted as a visual representation of a statistical test (Baguley, 2012).

These data analysis methods were used in every experiment in this thesis, unless otherwise stated.

2.2.6 Results

A typical eye trace is shown in Figure 2.3 with the uncompensated (red) and compensated (blue) vectors marked on. The saccade taken in Figure 2.3 was typical in this experiment as it takes an angle roughly half-way between the compensated and uncompensated angles.
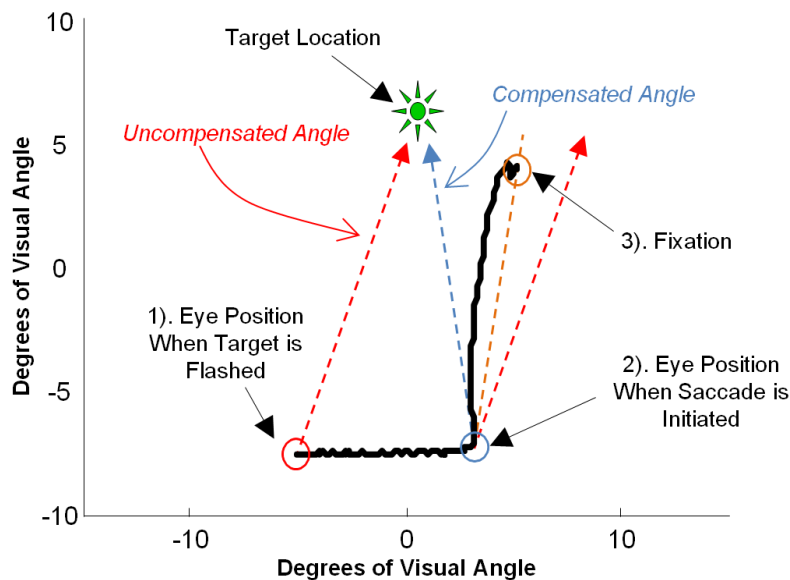
Figure 2.3: Typical eye trace from this experiment (black line). 'Uncompensated' (red) and 'compensated' (blue) angles are overlaid to show the two predictions of this experiment.

In order to determine the degree of angular compensation an index was computed to compare the saccade angle taken relative to that which would have taken the saccade to the target's spatial (compensated) or retinal (uncompensated) location. Here, 'compensation' refers to the ability of the saccadic system to adapt for displacements on the eye due to OKN; thus the 'compensated angle' would direct the saccade to the target's spatial location (denoted by the blue line in Figure 2.3), and an 'uncompensated angle' would direct the saccade at the target's retinal location (red line, Figure 2.3). Therefore a percentage 'compensation index' was calculated using the following equation:

$$Compensation\ Index\ = 100\ \times \frac{Saccade\ Angle\ Taken\ - Uncompensated\ Angle}{Compensated\ Angle\ - Uncompensated\ Angle}$$

It follows that a compensation index of 0% describes a saccade which is completely uncompensated for the intervening eye movement, and a compensation index of 100% would indicate that the saccade angle had completely compensated for the intervening eye-movement.

The mean compensation index for the four participants was 48.1% (SD = 1.9%), indicating that approximately half of the displacement due to OKN was compensated for by the targeting saccade. The distribution of compensation indices for data pooled across all four participants is shown in Figure 2.4.
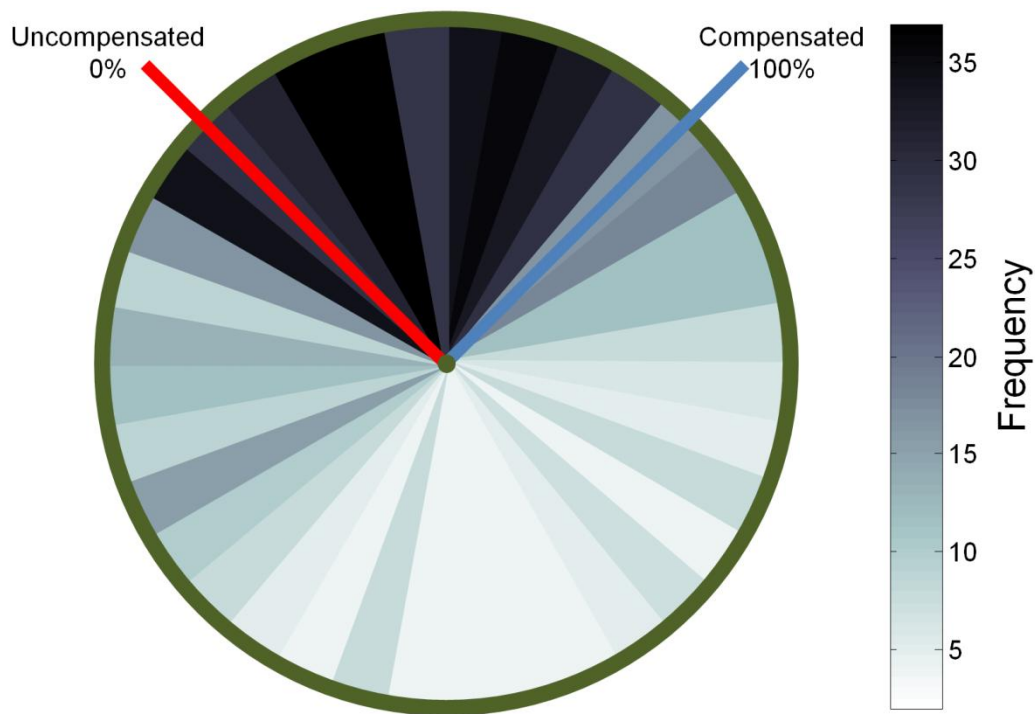
Figure 2.4: Saccades are represented based on their relative difference between the uncompensated and compensated vectors (the compensation index). For example, a saccade which compensated for exactly half of the displacement (a compensation index of 50%) would fall at the 12 o'clock position in this diagram. The number of saccades which fall at certain values of the compensation index is represented by the change in colour. Data has been pooled from all participants.

The distribution shown in Figure 2.4 reveals that the average compensation index of 48.1% does not stem from the targeting saccades being either compensated or not, otherwise Figure 2.4 would show a bimodal distribution.

As the compensated and the uncompensated vector become closer, the compensation index tends to infinity. In practice, this will tend to amplify the noise in the data, potentially making interpreting the index difficult. As an alternative, the distance travelled by the eye during the latency period was correlated with the horizontal component of error at fixation. This analysis is useful because the distance the eye travels in the latency period is the difference between the spatial and retinal locations of the target. Therefore if compensation were complete, we would expect no systematic relationship between the distance travelled during the latency period and the horizontal error. However a complete lack of compensation will result in a strong correlation between these two measures, with a slope of one. Figure 2.5 shows an example of such a correlation from one observer.
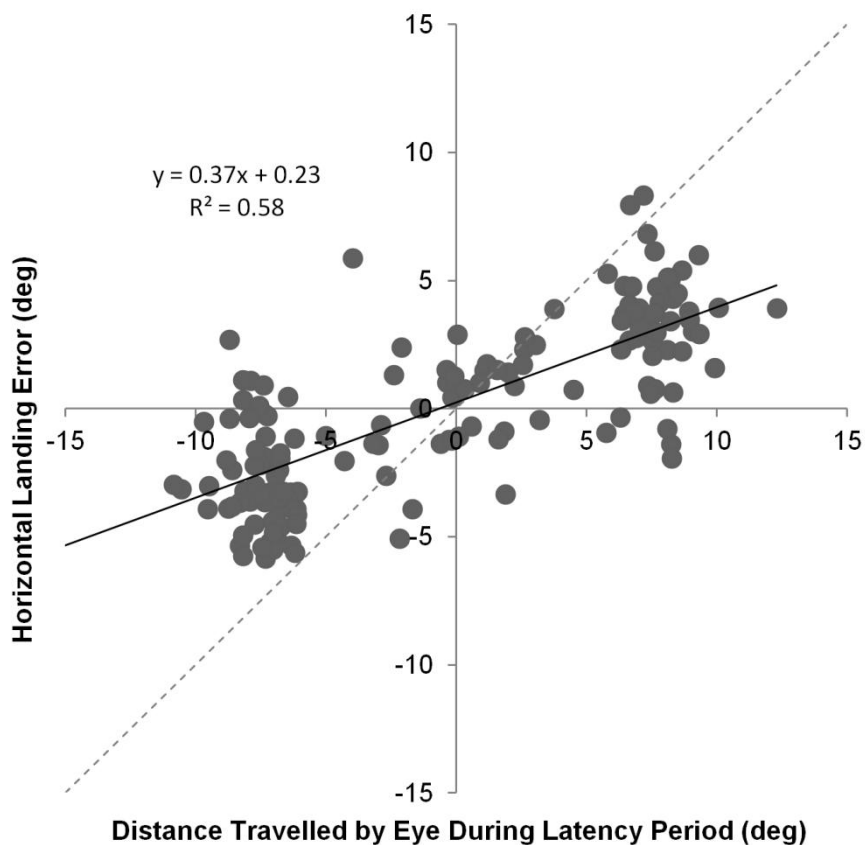
Figure 2.5: Correlation between the distance the eye travels during the saccade latency period and the horizontal error at fixation. Solid line shows the line of best fit, dashed line shows a slope of 1. Data taken from participant two.

As Figure 2.5 shows, there is a correlation between the distance the eye travels during the saccade latency period and the horizontal landing error; however the slope of the regression line is less than one (a slope of one is illustrated by the dashed line in Figure 2.5). The slight clustering evident in Figure 2.5 is due to a divergence between trials where there was a fast-phase during the saccade latency period (thus taking the distance travelled during the latency back towards zero) and trials where no such fast-phase occurred. A strong correlation with a slope of less than 1 was found in all participants; and individual slopes are shown in Table 2.1 along with the *r*-value, and the significance level of the correlation.

| Participant	r - values	Value of Slope	p - value
1	0.69	0.42	< 0.001
2	0.76	0.37	< 0.001
3	0.76	0.38	< 0.001
4	0.61	0.31	< 0.001

Table 2.1: Slope values, *r*-values and significance level for each participant for the correlation between distance travelled by the eye during the saccade latency period, and the horizontal landing error.

As clearly shown in Table 2.1 each participant shows a strong correlation between the distance travelled during the saccade latency period, and the horizontal error at fixation. This means that the error at fixation is systematically related to the amount of displacement due to OKN, however the magnitude of the error is less than we would expect given the size of the displacement. Thus targeting saccades executed during OKN appear to be partially sensitive to the ongoing eye-movement.

Many of the saccades that were recorded in this experiment show a tendency to undershoot the target, which has been extensively reported as normal saccadic behaviour (Becker & Fuchs, 1969; Harris, 1995a; Henson, 1978; Weber & Daroff, 1971), and indeed is visible in the typical eye trace shown in Figure 2.3. One potential concern is whether the partial compensation observed in this experiment is due to such undershoots – if angular compensation for the optokinetic displacement is correct, however the saccade does not reach the target position due to a natural undershoot, then this may give rise to a pattern of behaviour consistent with partial compensation. It is also possible that saccadic undershoots are due to uncertainty in target location, and therefore saccades that undershoot will show less accurate angular compensation. In order to address this possibility, saccadic amplitudes were extrapolated in order to measure the error that would have occurred had the saccade reached the target's vertical location. A pattern of partial compensation was observed when

using these extrapolated eye positions, indicating that the eye was not merely stopping short while heading in the right direction. As a further analysis, saccade amplitude gain was correlated with the angular compensation index, these plots are visible in Figure 2.6.
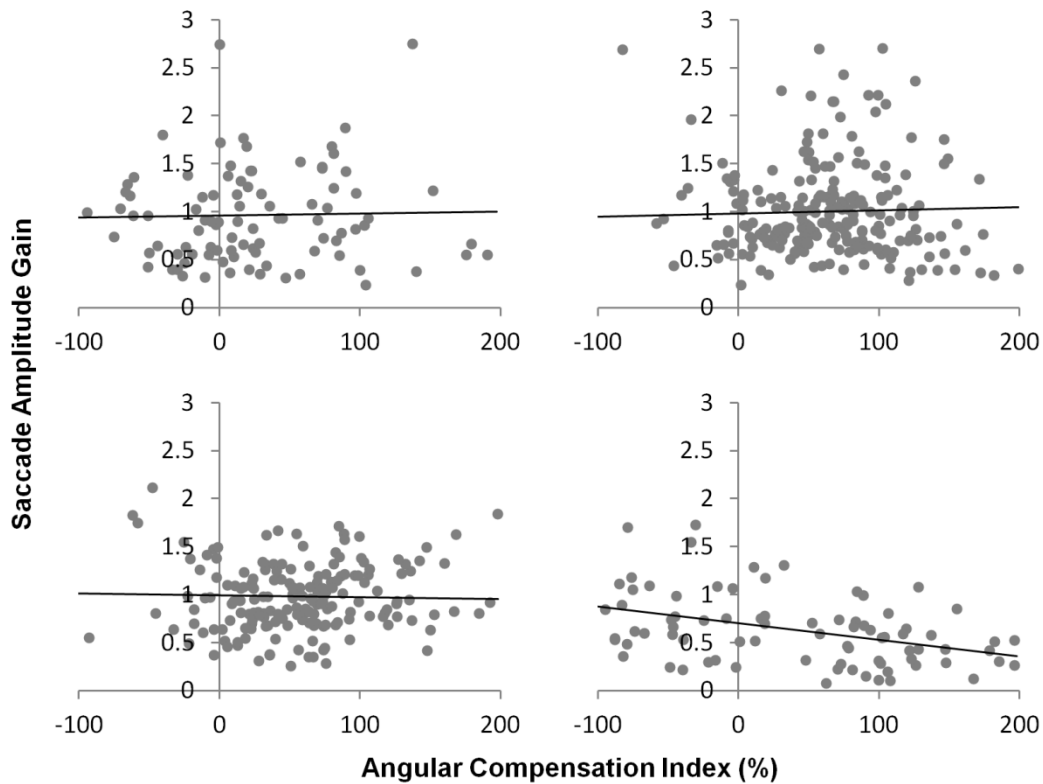
Figure 2.6: Correlations between the Compensation Index and saccadic amplitude for each of the four observers.

It was found that only one participant showed a significant correlation between the compensation index and saccadic amplitude (the bottom right plot in Figure 2.6) ($r = -0.35$, $p < 0.001$). It is not clear why this participant showed a correlation when the other three did not, however the correlation was negative, implying that a lower amplitude gain was associated with more accurate angular compensation. This is the opposite to what would be expected if saccadic undershoots indicated a greater uncertainty about target position; therefore there does not appear to be any evidence that saccadic compensation would be more complete if saccades did not undershoot the target.

2.2.7 *Discussion*

It is clear from Figure 2.4 and Figure 2.5 that saccades made to targets during OKN deviate in a systematic way. The results suggest that some compensation occurs during nystagmus but is incomplete. Under the experimental conditions investigated, half the angle required for the eye to land correctly on the target was compensated for on average. However these averages do not seem to stem from a combination of saccades being either compensated or not, otherwise Figure 2.4 would show a bimodal distribution.

These data agree with studies showing partial compensation for perceived location during involuntary eye-movements (e.g. Bedell & Currie, 1991; Bedell & Currie, 1993). It is less clear how these results sit alongside reports which show that pointing to short-duration targets during rebound nystagmus or optokinetic after-nystagmus is essentially veridical (Bedell, 1990; Bedell et al., 1989). However, it is unclear how different reference frames such as eye-movements and pointing responses might operate. For example, pointing responses do not have an equivalent dissociation between retinal and spatial co-ordinates; they can only ever be to the perceived egocentric direction of an object. For example, as time elapses between stimulus and response, eye position becomes increasingly dislocated, whereas a pointing response would remain constant.

The results of Experiment A1 suggest that the saccadic system cannot exist in complete isolation from the optokinetic system – if this were the case then no compensation for OKN displacements would be evident. The sharp dichotomy between reflexive and volitional eye movements often alluded to in the literature is therefore questionable. The results also show however that the compensation is not complete – displacements due to OKN introduced systematic errors in saccadic accuracy. This could mean that reflexive OKN has a limited ability to update the spatial maps of the saccadic system. However, to draw this conclusion it would need to be demonstrated that saccadic compensation is improved if the

displacement is due to a voluntary eye movement. This formed the basis for the next experiment: comparing the accuracy of saccades executed during concomitant stare-OKN, look-OKN and smooth pursuit.

2.3 Experiment A2 – Comparison of the Accuracy of Saccades Executed during Stare-OKN, Look-OKN and Smooth Pursuit

In the previous experiment optokinetic nystagmus was used as an example of a reflexive gaze-stabilizing eye movement. However, there are two different types of OKN, a volitional type and a reflexive type. These two OKN types are usually referred to as 'stare-OKN' and 'look-OKN'. Stare-OKN is commonly assumed to be a reflexive eye movement, one which is elicited when participants are required to passively view a moving screen; whilst look-OKN requires the observer to track a specific point of their choosing in the display, and is assumed to be more akin to deliberate pursuit eye-movements (Knapp et al., 2008). In Experiment A1 it was assumed (although not specifically manipulated) that participants were conducting reflexive stare-OKN. Whether look- or stare-OKN is being elicited should be taken into account as there are some fundamental differences between look- and stare-OKN (Kaminiarz, Königs, & Bremmer, 2009b; Kashou et al., 2010; Magnusson, Pyykkö, & Jäntti, 1985). Stare-OKN and look-OKN have different patterns of neuronal activity, with look-OKN (unlike stare-OKN) activating cortical areas associated with volitional pursuit and saccades (Kashou et al., 2010; Konen et al., 2005; Schraa-Tam et al., 2009; however see also Dieterich et al., 2009; Gulyás et al., 2007). This suggests that look-OKN is more akin to a volitional eye-movement, and some authors consider look-OKN to be nothing more than alternating saccades and smooth pursuit (Heinen & Keller, 2004).

The comparison between look-OKN and stare-OKN is useful because it is a potential paradigm to directly compare a volitional and a reflexive eye movement using the same

stimulus; only the instructions to the participant are changed between conditions. It was also decided to introduce a condition of saccades made during concomitant smooth pursuit. Although some authors consider the slow-phase of look-OKN to be the same as a smooth pursuit eye movement (Heinen & Keller, 2004) there are some important differences between these two eye movements which may mean the results from one do not necessarily apply to the other. For example, during look-OKN there is a large full-field display, whether classic pursuit experiments use a single target. It has been shown that if the target moves along with a textured background, pursuit accuracy is improved (Heinen & Watamaniuk, 1998; van den Berg & Collewijn, 1986); possibly because the peripheral stimulation due to the background gives a larger amount of information to motion processing areas (Heinen & Keller, 2004; van den Berg & Collewijn, 1986). Furthermore large-scale motion upon the retina can activate very short-latency reflexive ocular-following movements which may change the dynamics of the tracking eye movements during look-OKN, these ocular following responses are not found with single pursuit targets of less than 20° in size (Gellman et al., 1990). Moreover lesions to the parietal lobes can impair foveal pursuit, but leave full-field pursuit intact, suggesting different neural pathways exist for single-target and large display pursuit (Baloh et al., 1980).

Another basic difference between look-OKN and pursuit is that during look-OKN the participant does not have to actively seek out a specific pursuit target, this allows the participant to determine their own pursuit amplitude and duration, and they can make a returning saccade at a point at which they choose. Smooth pursuit tasks, on the other hand, demand far more rigid eye trajectories which are determined by the stimuli utilized. It has been found that if participants are presented with textured backgrounds which they are able to pursue at leisure, pursuit gains are better than if participants are required to pursue a single target stimulus, even if it too is given the same textured background (Niemann & Hoffmann,

1997). Therefore it seems that pursuit velocities are most accurate when the participant has the opportunity to determine their own pursuit trajectory (Niemann & Hoffmann, 1997). Although it is unclear whether differences in pursuit accuracy would affect the accuracy of simultaneously executed saccades.

This experiment aimed to investigate whether saccades are any more accurate when they are executed during a concomitant volitional eye movement instead of a reflexive eye movement. Therefore the accuracy of saccades during reflexive stare-OKN was compared to the accuracy of saccades executed during volitional look-OKN or smooth pursuit. Saccadic accuracy during look-OKN does not appear to have been tested previously; however there is some literature on the accuracy of saccades executed to targets during smooth pursuit. The results of experiments investigating saccadic accuracy during pursuit have not been entirely consistent, with some authors concluding that the saccade can compensate for a displacement due to pursuit (Hansen, 1979; Ohtsuka, 1994; Schlag et al., 1990; Van Beers et al., 2001), others concluding that saccades cannot compensate for smooth pursuit displacements (McKenzie & Lisberger, 1986), and others reporting that the compensation is only partial, ranging from an average of 27% compensation (Gellman & Fletcher, 1992) to 62% (Daye et al., 2010). These differences may be due to experimental methods, for example Schlag et al. (1990) reported that longer target durations allowed for greater compensation. The divergence in results may also be related to task requirements, experiments where saccades are initiated as quickly as possible tend to show low compensation, whereas those in which the saccade is not made quickly tend to find compensation is possible, despite the fact that pursuit continues during the delay between target presentation and saccade execution (Blohm, Missal, & Lefèvre, 2005; Blohm, Optican, & Lefèvre, 2006). Therefore there are contradicting results in the literature on executing a saccade during pursuit, and the true

behaviour of the oculomotor system may well depend on taking into consideration the precise metrics of its operation (e.g. the saccadic latency period).

Therefore the aim of this experiment was to investigate whether the accuracy of saccades would be any different if the displacement was due to a volitional eye movement: namely either look-OKN or pursuit. There were three different conditions of smooth pursuit. The purpose of this was to try to isolate some of the similarities and differences between pursuit and look-OKN. Therefore the pursuit target either appeared on its own (single-target pursuit), or superimposed upon the moving display of OKN dots (full-field pursuit), or it was superimposed upon a static display of OKN dots (static-background pursuit). This allows a basic measure of saccadic accuracy during pursuit (single-target pursuit); a measure where there is equivalent peripheral stimulation to look-OKN (full-field pursuit); and a measure where there is there are the equivalent background contours to look-OKN, but without the motion stimulation (static-background pursuit). The static background condition also allows the investigation of interactions between the smooth pursuit and the optokinetic systems; because pursuit over a background will result in retinal motion which should drive OKN (see Section 1.4.3). The full-field pursuit and static-background pursuit conditions meant that the top and bottom of the band of dots was maintained in both pursuit and look-OKN/stare-OKN conditions, as these give a strong vertical contour.

2.3.1 *Participants*

This experiment was conducted on five participants, three of whom were female. This sample size is consistent with previous literature (see Section 2.2.1) and was fixed prior to the experiment commencing. Participants ranged in age from 22 to 25 years. Two of the participants had participated in Experiment A1, and three were naïve to this paradigm. All participants self-reported normal vision.

2.3.2 *Stimuli*

The stimuli used to elicit stare-OKN were identical to those outlined in Experiment A1. For look-OKN the stimuli displayed were exactly the same as during stare-OKN, however some changes had to be made to the timing of the targeting saccade target. The longer durations of look-OKN slow phases mean that the target delays used in Experiment A1, and the stare OKN condition (50, 100 and 200ms into the slow-phase) are no longer appropriate, as these will all elicit saccades during the first half of the slow-phase. From pilot data, it was found that the mean duration of the slow-phase under these experimental conditions was around 650ms. Therefore the idea of an early, middle and late saccade was kept, however the timings were made more continuous so that the target presentation would be kept unpredictable. Thus targets could be triggered early (50-200ms), in the middle (201-350ms) or at the end of the nystagmus waveform (351-500ms). The precise delay was randomly determined. Furthermore, during Experiment A1 and the Stare-OKN condition, saccades were triggered after 11, 12 or 13 fast-phases. There were two reasons to change this for the look-OKN condition: firstly because look-OKN is under voluntary control, this would make it more predictable as to when the target would appear, and secondly, as the frequency of look-OKN is much lower than stare-OKN, trials would be much longer. Therefore the triggering criterion was changed, such that saccades could be triggered from anywhere between 5-13 fast-phases, this kept the target presentation unpredictable, and gave a greater spread of trial durations.

The larger amplitudes of look-OKN mean that the distance to the saccade target is highly variable, saccades made at the beginning or end of the slow-phase have to travel much further than they would during stare-OKN. To account for this, the target's location was altered with respect to gaze location, such that the target was presented within four degrees either side of the horizontal location of gaze.

The pursuit target consisted of a single dot 0.3° in radius (the same size as the dots used to elicit OKN). During full-field pursuit and static-background pursuit, this target was distinguishable from the OKN-dots by its brightness of 1.06cd/m^2, which was clearly brighter than the OKN dots which were 0.1cd/m^2. During full-field pursuit the pursuit target moved at the same speed (32°/sec) and in the same direction as the OKN-dot display, therefore all movement in the display was coherent. During static-background pursuit the band of OKN dots did not move. In all pursuit conditions the pursuit target moved at a constant speed of 32°/sec for 30°, and then stepped back 30°. The pursuit target's horizontal location at the start of the trial was up to three degrees either side of the centre of the screen (randomly determined) and the 30° amplitude of the pursuit target's motion was centred on the screen, with a random shift by up to 6°. The pursuit target's vertical location was always in the middle of the screen. The saccade target's location and onset during the pursuit condition was determined in exactly the same way as described in the look-OKN condition.

2.3.3 Procedure

Participants were initially given instructions as to the task requirements (stare-OKN, look-OKN or pursuit). For stare-OKN participants were asked not to track any particular dot in the display, but not to allow the band of dots to become blurred. In look-OKN conditions participants were asked to pick any particular dot, and follow it across the screen for as long as they liked, and then return their gaze to the other side of the screen to track another dot. For pursuit, participants were asked to follow the course of the single bright dot only, regardless of whether it appeared superimposed upon a moving or static background. In all conditions participants were asked to saccade to the vertically-presented saccade target as quickly as they could. Standardised written instructions were given to ensure that every participant performed each condition in a similar way. Each condition was divided up into five blocks; each block consisted of 40 trials. The order of conditions (stare-OKN, look-

OKN, single-target pursuit, full-field pursuit or static-background pursuit) was randomised for each participant, and each condition was completed as a single block of trials.

2.3.4 Results

In order to check that the manipulation to elicit either stare- or look-OKN was successful, the amplitudes and frequencies of nystagmus in these two conditions were compared. Figure 2.7 shows example eye traces from one participant in both the stare-OKN (A) and look-OKN (B) conditions.
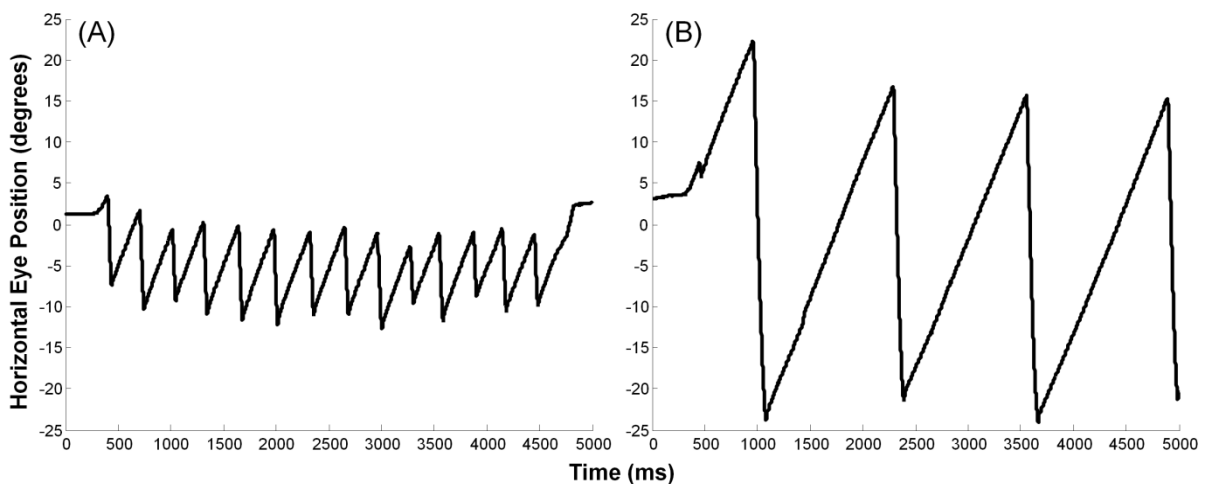
Figure 2.7: Example eye traces from one participant. *A*, shows a stare-OKN trial, whilst *B* shows a look-OKN trial. Note the characteristic small amplitude, but high frequency waveform of stare-OKN.

There are clear differences between the two nystagmus waveforms in Figure 2.7; and the stare-OKN waveform shows the small amplitude, high frequency nystagmus characteristic of this type of eye movement (Freeman & Sumnall, 2005). The mean amplitude for stare-OKN was 11.29° (SD = 7.30°), whereas the mean amplitude for the look-OKN condition was 29.41° (SD = 5.64°). These differences were significant ($t(4)$ = -4.97, p = 0.008; effect size[1]:

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[1] The correlation coefficient *r* is employed as the effect size for *t*-tests. This measure of effect size is beneficial as it is constrained to lie between 0 and 1, where 0 would indicate the manipulation has no effect upon outcome,

$r = 0.93$). The expected pattern in nystagmus frequency was also found, with stare-OKN showing a higher frequency (Mean = 2.59HZ, SD = 0.52Hz) than look-OKN (Mean = 1.22Hz, SD = 0.48Hz). This difference in frequency was also significant ($t(4) = 4.26$, $p = 0.013$, $r = 0.91$). The higher frequencies and smaller amplitudes of stare-OKN suggest that the standardised instructions given to participants were successful in eliciting either the reflexive or the volitional types of OKN (Cheng & Outerbridge, 1974).

Before progressing onto the main effects, I checked whether the main finding of Experiment A1 was replicated (however as two participants contributed data to both Experiment A1 and A2 this was not a true replication in the statistical sense). As shown in Section 2.2.6, correlations were analysed between the distance the eye is displaced during the saccade latency period, and the horizontal component of error at fixation. The pattern of results shown in Experiment A1 was once more found in the stare-OKN condition of this experiment: there were significant correlations for each participant, however the slope of the line of best fit is less than one, these data are shown in Table 2.2. This again suggests that partial compensation for the OKN displacement is possible.

Participant	r - values	Value of Slope	p - value
1	0.84	0.37	< 0.001
2	0.85	0.44	< 0.001
3	0.73	0.44	< 0.001
4	0.54	0.21	< 0.001
5	0.37	0.24	< 0.001

Table 2.2: *r*-values, slopes and *p*-values for the correlation between distance travelled during the saccade latency period and horizontal error in the stare-OKN condition.

and 1 indicates the manipulation has a perfect effect upon the outcome (i.e. manipulation explains 100% of the variance). An r of 0.10 is considered to be a small effect, an r of 0.30 a medium effect, and an r of 0.50 is a large effect (Field, 2005)

The main aim of this experiment was to investigate whether saccadic accuracy would be any different if saccades were executed during a reflexive or a volitional eye movement. Therefore horizontal error at fixation was correlated with the distance travelled by the eye during the saccade latency period for each condition. Recall from Experiment A1 that in the correlation between displacement distance and horizontal error, greater compensation would be associated with lower slope gradients, however, in order to aid interpretation of the data a compensation measure of '1 – slope value' was used to indicate compensation, thus it follows that higher compensation measures indicate greater compensation.
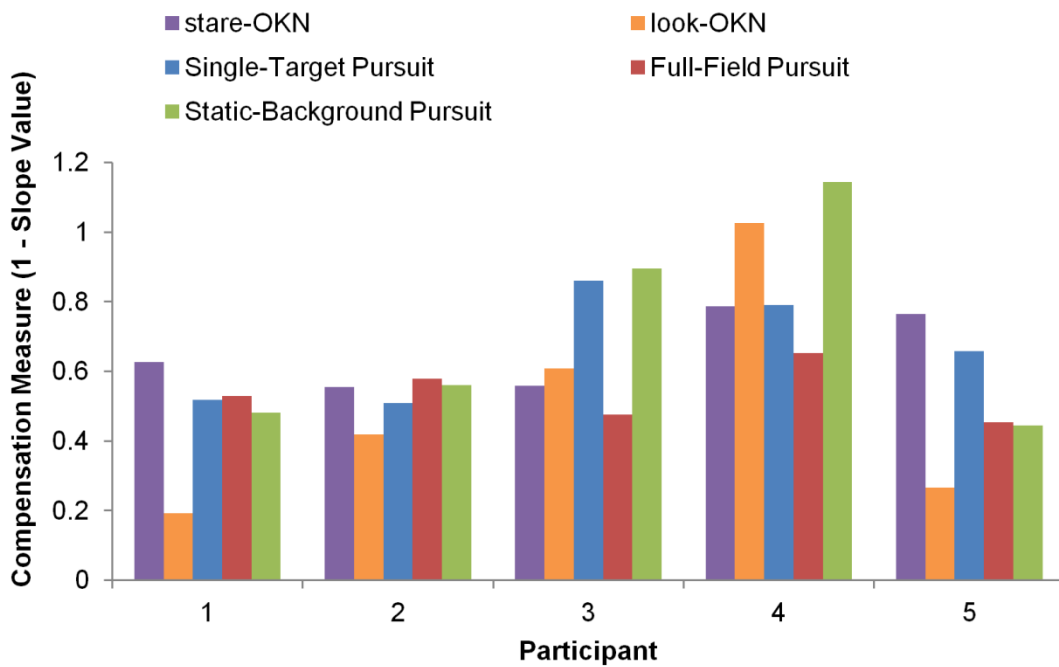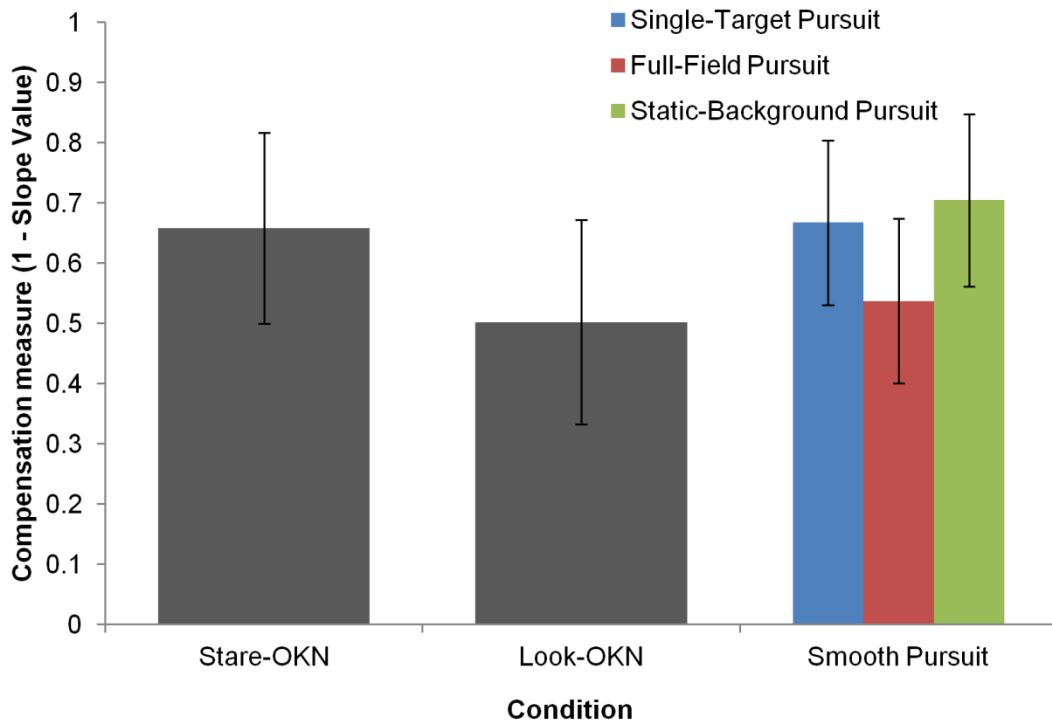
**Figure 2.8:** *A).* **Mean compensation measure (1 - slope values) across all condition types. A higher compensation measure denotes better compensation for the displacement during the saccade latency period. Error bars show ±1 × Standard Deviation with variance attributable to individual differences partialled out in line with Cousineau's (2005) method.** *B).* **Individual data across all conditions.**

As shown in Figure 2.8, there is little indication that the different conditions caused any change in the compensation measure ($F_{(4,16)}$ = 1.49, p = 0.252, effect size[2]: η^2 = 0.27). This suggests that there is no change in saccadic accuracy when considered relative to displacement distance. Furthermore there is no reduction in absolute error for volitional eye-movement displacements (shown in Figure 2.9); indeed all eye-movement conditions show larger absolute errors at fixation than those seen in stare-OKN. The effect of eye-movement condition on horizontal error was found to be significant ($F_{(4,16)}$ = 6.31, p = 0.003, η^2 = 0.61); this effect appears to be driven largely by the consistently high errors observed in the look-OKN condition as in all participants errors were largest during look-OKN. It should be noted that mean error is a crude measure in this respect, it is clear from Figure 2.8 and Figure 2.5 that error depends upon the distance the eye moves during the saccade latency period; therefore this has to be accounted for when considering the meaning of mean error (i.e. a low mean error might mean better saccadic compensation, or it may simply mean that there was less displacement during the saccade latency period). Therefore the compensation measure that utilizes the slope when displacement is correlated with error is a more meaningful dependent variable (i.e. Figure 2.8). Mean error is shown here because it has direct relevance to the perceptual biases reported in Chapter 3; and therefore it allows the interested reader to compare results obtained here with those reported in Chapter 3.

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[2] The eta-squared effect size measure is common when using an ANOVA analysis. It is equivalent to an R^2 value (therefore η^2 of 0.01 is a small effect, η^2 of 0.09 is medium, and η^2 of 0.25 is a large effect).
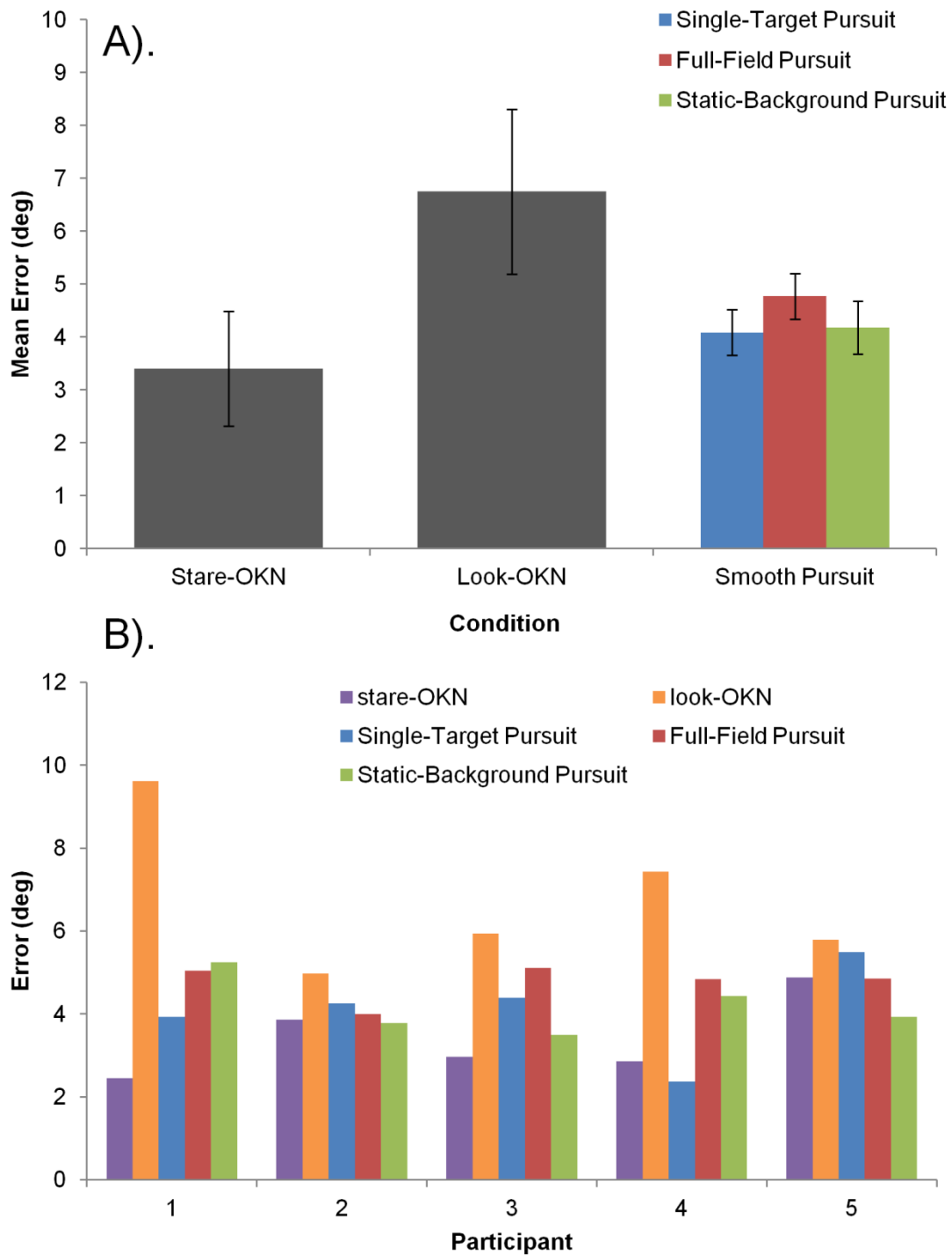
Figure 2.9: *A).* **Mean horizontal error at fixation across all condition types. Lower errors would imply better compensation. Error bars show ±1 × Standard Deviation with variance attributable to individual differences partialled out in line with Cousineau's (2005) method.** *B).* **Individual errors are plotted for all conditions.**

Therefore there is no indication that saccades were more accurate when the displacement during the saccade latency period is due to a volitional eye movement. No difference in saccade latency was found between the conditions ($F_{(4,16)} = 1.30$, $p = 0.314$, $\eta^2 = 0.24$); individual and mean latencies are shown in Table 2.3.

Participant	OKN		Pursuit		
	Stare	Look	Single-Target	Full-Field	Static-Background
1	466.1	487.7	436.4	426.0	540.6
2	296.8	268.0	265.1	263.6	285.9
3	292.7	292.0	299.5	272.0	284.5
4	433.8	410.2	450.7	390.6	383.6
5	339.6	416.9	341.2	333.7	352.0
Mean	**365.8**	**375.0**	**358.6**	**337.2**	**369.3**

Table 2.3: Saccade latencies (in ms) for each observer and each condition.

These data also allow further investigation of whether look-OKN is equivalent to smooth pursuit. The data replicated the previous findings of Heinen and Watamaniuk (1998) and van den Berg and Collewijn (1986) that pursuit gains are higher when pursuit is accompanied with a moving background (Mean = 0.89, SD = 0.05) than with a static background (Mean = 0.70, SD = 0.10) or no background (Mean = 0.69, SD = 0.07). This increase in gain with a moving background was significant ($F_{(2,8)} = 51.05$, $p < 0.001$, $\eta^2 = 0.93$). There was no difference in gain between look-OKN and full-field pursuit ($t(4) = -0.62$, $p = 0.568$, $r = 0.30$), therefore the data did not replicate the finding of Niemann and Hoffmann (1997) that pursuit gains are improved if the participant is able to determine their own pursuit trajectory. Previous researchers found that saccadic accuracy following pursuit displacements were improved at longer saccade latencies (Schlag et al., 1990). There was no evidence of this being the case in our data in any of our conditions. Table 2.4 displays the correlation coefficients when saccade latency was correlated with error.

Participant	OKN		Pursuit		
	Stare	**Look**	**Single-Target**	**Full-Field**	**Static-Background**
1	-0.06	0.43	0.51*	0.26	0.43
2	-0.03	0.12*	0.12	0.20*	-0.05
3	0.07	0.26*	0.17	0.20*	-0.01
4	-0.14	0.08	0.04	-0.08	-0.01
5	0.31*	-0.33	0.03	-0.16	-0.09

* $p < 0.01$

Table 2.4: Correlation coefficients between saccade latency and horizontal error.

2.3.5 *Discussion*

Experiment A2 repeated the results found in Experiment A1 - the saccadic system appears to be able to partially compensate for displacements of the eye due to reflexive optokinetic nystagmus. Whilst this would imply that volitional and automatic eye movements cannot be processed in complete isolation, it was unclear whether saccades would be better able to compensate for displacements due to another volitional eye movement. The results of this experiment did not find any evidence that saccades are any more accurate if the displacement of the eye was due to volitional look-OKN or smooth pursuit. If saccades are no more accurate following a displacement of the eye due to a volitional eye movement then this implies that the volitional saccadic system has interconnections to the reflexive OKN system which are no different than the interconnections to other volitional eye movement systems. Therefore, the oculomotor system does not appear to distinguish between volitional and automatic eye movements in this case, which is further evidence there is no great difference between volitional and automatic actions.

Differences between look-OKN and Pursuit

The gain of the slow-phase of look-OKN was no different than the pursuit condition which was also given a moving background (full-field pursuit), which is consistent with authors who claim that the slow-phase of look-OKN is analogous to a smooth pursuit eye-movement

(Heinen & Keller, 2004). However, saccades executed during look-OKN showed the largest amount of error for every one of the five participants, which was not the case for the full-field pursuit condition. It is not clear why errors for look-OKN are largest, when errors for smooth-pursuit and stare-OKN did not differ. One possibility is that the task-requirements in look-OKN demand more attention; for example the participants have to determine their own eye-movement trajectory, and decide when to make a returning saccade. By its very definition, look-OKN demands attention – if participants were to passively view the stimuli then stare-OKN would be elicited. Indeed, some claim that look-OKN contains within it bursts of stare-OKN which occur during brief periods of inattentiveness (Cheng & Outerbridge, 1974). Dual-task paradigms have shown that executing an attentionally demanding task whilst making saccades adversely affects saccade accuracy (Castet, Jeanjean, Montagnini, Laugier, & Masson, 2006; Kowler, Anderson, Dosher, & Blaser, 1995; Stuyven, Van der Goten, Vandierendonck, Claeys, & Crevits, 2000). Perhaps it is possible that participants found look-OKN to be more attentionally demanding than full-field pursuit, and this limited the ability of saccades to compensate for displacements which occur during the saccade latency period? Nevertheless, the accuracy of saccades made during stare-OKN did not appear to be any different than during smooth pursuit, which implies the volition of the eye movement was not a factor in the poorer accuracy of the targeting saccades.

Differences between the Pursuit Conditions

This experiment did replicate earlier work showing that pursuit gain was more accurate with a moving background (Heinen & Watamaniuk, 1998; van den Berg & Collewijn, 1986). However, the ability of saccades to correct for pursuit displacements did not differ between the three conditions. In this experiment, on average participants were able to compensate for 41.11% (SD = 14.69%) of the displacement due to smooth pursuit. It is hard to say how this figure compares to previous literature because the amount of compensation depends upon

precise factors such as the target duration, and the latency of the saccade (Blohm et al., 2005; Blohm et al., 2006). However compensation for smooth pursuit has been reported as both lower (Gellman & Fletcher, 1992; McKenzie & Lisberger, 1986) and higher (Daye et al., 2010; Schlag et al., 1990) than the figure found in this experiment – this implies there is nothing exceptional about the data collected here.

The Role of Saccade Latency

Previous studies have reported that top-down, targeting saccades are able to compensate for displacements of the eye due to smooth pursuit if saccade latencies are longer (Blohm et al., 2005; Blohm et al., 2006). This effect was not found in this experiment in any of the five conditions. Although some individual correlations between error and latency did show significant positive correlations, these were not consistent across conditions or observers. Furthermore there were no significant negative correlations between error and latency, as would be expected if longer latencies were associated with reduced errors. However, this may simply be due to a lack of variance in the saccadic latencies found here. For example, Schlag et al. (1990) reported that saccadic compensation for smooth pursuit displacements did occur; however in their task saccade execution was delayed until the smooth pursuit target extinguished (130-300ms after the saccade target was displayed, the smooth pursuit target was moving throughout). Thus the longer latencies which allowed greater compensation were externally imposed upon the saccadic system; whereas longer latencies in this experiment were the product of intrinsic noise in the saccade latency distribution. Therefore the results of this experiment do not necessarily contradict those who find that compensation is possible at longer latencies. It is not altogether clear whether longer saccade latencies due to an externally imposed delay are even analogous to longer latencies due to internal noise of the saccadic system; they presumably result from entirely different mechanisms (Sumner, 2011) and may affect the oculomotor system in very different ways.

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Fast-Phase Frequency: A Confound between Conditions

Although this experiment did not find any evidence that saccades were better able to compensate for displacements due to volitional look-OKN or pursuit there are some issues which need to be addressed before this conclusion can be made confidently. For example, there are differences between the eye-movement conditions beyond their volitional nature. An example of this difference is clearly visible in Figure 2.7; stare-OKN has a much higher frequency of resetting fast-phases than look-OKN or pursuit. It might be possible that the higher frequency of resetting fast-phases has an effect on the ability of the saccadic system to compensate for the displacements of the eye. Some authors believe that fast-phases and saccades are manifestations of the same eye movements (Guitton & Mandl, 1980; Ron et al., 1972); if this were true then it would be highly likely that activity in the fast-phase system could affect the generation of the top-down targeting saccade. However, it is unclear what role, if any, fast-phase frequency played in this experiment because it was not specifically manipulated; therefore Experiment A3 aimed to study this issue.

2.4 Experiment A3 – Investigating the Role of Fast-Phase Frequency in Saccadic Compensation

This experiment aimed to investigate what effect the frequency of resetting fast-phases or saccades might have on saccadic accuracy. Unfortunately, manipulating the frequency of the resetting fast-phase is not altogether easy for either stare-OKN or look-OKN. In stare-OKN fast-phases tend to occur at a rate of about three times a second, unless stimulus speeds are very slow (Cheng & Outerbridge, 1974; Freeman & Sumnall, 2005). At very slow stimulus speeds it is likely that the low-frequency of resetting fast-phases is due to participants implicitly switching to look-OKN from stare-OKN (Cheng & Outerbridge, 1974). Therefore

the frequency of fast-phases in stare-OKN cannot really be manipulated, which means that the effect of frequency can only be examined using look-OKN or smooth pursuit.

There is no way to precisely set a frequency of look-OKN, this is because the participant is free to determine their own eye trajectory, and make a returning fast-phase whenever they choose. However, the relative frequency can be indirectly manipulated by changing the stimulus speed: participants tend to make the returning fast-phase at a particular gaze eccentricity, therefore faster stimulus speeds will result in a higher frequency of resetting fast-phases in look-OKN. This means that frequency and slow-phase speed become confounded during look-OKN; fortunately, they are separable in stare-OKN and pursuit conditions, so these conditions can act to control for the effects of stimulus speed. Note that smooth pursuit is the only condition where both frequency and speed can be directly manipulated. For stare-OKN, the resetting frequency is fixed.

It had been previously found in Experiment A2 that there was no difference in saccade accuracy following displacements due to single-target pursuit, full-field pursuit or static-background pursuit. Therefore only one type of pursuit was included in this experiment, which was the full-field pursuit condition; as this condition was most similar to look-OKN, and the only one with a comparable slow-component gain to look-OKN.

2.4.1 *Participants*

This experiment was conducted on five participants, three of whom were male. Ages ranged from 23 to 25 years. Two of the participants had previously participated in Experiment A2 and Experiment A1; three of the participants were naïve to this paradigm. All participants self-reported normal vision. This sample size is consistent with previous literature (see Section 2.2.1) and was fixed prior to the experiment commencing.

2.4.2 *Stimuli*

The stimuli displayed for stare-OKN and look-OKN were exactly as described in Experiment A2. The stimuli displayed for the pursuit condition were exactly as described in Experiment A2 for full-field pursuit.

For each eye movement type, two different stimulus speeds were presented. In one condition stimuli moved at 32°/sec (the same as in Experiment A1 and A2), and in a second condition, stimuli moved at 40°/sec. In the smooth pursuit conditions, four different pursuit amplitudes (summarised in Table 2.5) allowed the manipulation of frequency independently of the two stimulus speeds.

Condition	Velocity (°/sec)	Amplitude (°)	Frequency (Hz)
1	32	30	1.067
2	32	20	1.6
3	40	37.5	1.067
4	40	25	1.6

Table 2.5: Stimulus properties of the four pursuit conditions. Two different stimulus velocities are tested, each at a particular amplitude, resulting in two different saccade frequencies. This allows the investigation of the effects of frequency independently from stimulus velocity.

The four pursuit conditions were designed to produce just two different frequencies of resetting saccades. The purpose of this was to isolate any effects due to frequency, independent of stimulus velocity or amplitude. If frequency does affect saccadic accuracy, then it would be expected that saccadic accuracy in condition 1 would equal that in condition 3; and saccadic accuracy in condition 2 would equal that in condition 4. Furthermore, accuracy in conditions 1 and 3 would be different to those in conditions 2 and 4.

Because of the two different stimulus velocities, the onset of the targeting saccade target had to be adjusted depending on the velocity condition. For stare-OKN, stimulus

velocity should not alter slow-phase duration; therefore the onset times for both conditions were identical to those in Experiment A2. For look-OKN with stimulus speeds of 40°/sec, the saccade target was triggered randomly from 110-560ms following fast-phase detection; this shorter time accounts for the shorter duration of slow-phases which occurs at faster stimulus speeds. For the 32°/sec look-OKN condition timings were kept exactly as described in Experiment A2. For the pursuit condition, two different timings were used based upon whether the pursuit has a high or low frequency of resetting saccades (see Table 2.5). At a high frequency of resetting saccades, the target was triggered 110-460ms following resetting saccade detection, and with a low frequency the saccade target was triggered between 110-760ms following resetting saccade detection.

Therefore, to summarise, this experiment had three eye movement conditions, stare-OKN, look-OKN and smooth pursuit. Within each of these eye-movement conditions there were two stimulus speeds (32°/sec or 40°/sec) and within the pursuit conditions there were two levels of pursuit frequency. This was implemented to allow a spread of resetting saccade/fast-phase frequencies, to see if there would be any effect of frequency upon compensation.

2.4.3 Procedure

There were eight experimental conditions: the four pursuit conditions (Table 2.5), and stare-OKN and look-OKN conducted at either 32°/sec or 40°/sec stimulus velocities. The order in which each participant completed the eight conditions was randomised. Each condition was split into five recording blocks, each of which consisted of 40 trials. Before each condition the participants were given standardised written instructions as was done in Experiment A2. The characteristics of the trials were exactly the same as described in Experiment A2.

2.4.4 Data Analysis

Data were analysed as described in Experiment A1 (Section 2.2.5), however additional multiple regression analyses were run in order to try and investigate the independent contributions of eye-movement type (stare-OKN, look-OKN or pursuit) and resetting saccade/fast-phase frequency. The regression model also contained a predictor of saccade latency, because in previous experiments latency influences the accuracy of saccades executed during smooth pursuit (Blohm et al., 2005); therefore this was a variable of interest to this experiment. Details of the regression model are found in the results section (Section 2.4.5).

2.4.5 Results

By pooling data across stimulus speeds and frequencies and looking only at the effect of eye-movement, this experiment replicated the results of Experiment A2 (although two participants were common in both this Experiment and Experiment A2, meaning this was not a true replication). There was no significant difference in the compensation measure ($F(2,8) = 0.64$, $p = 0.554$, $\eta^2 = 0.14$). These data are shown in Figure 2.10.
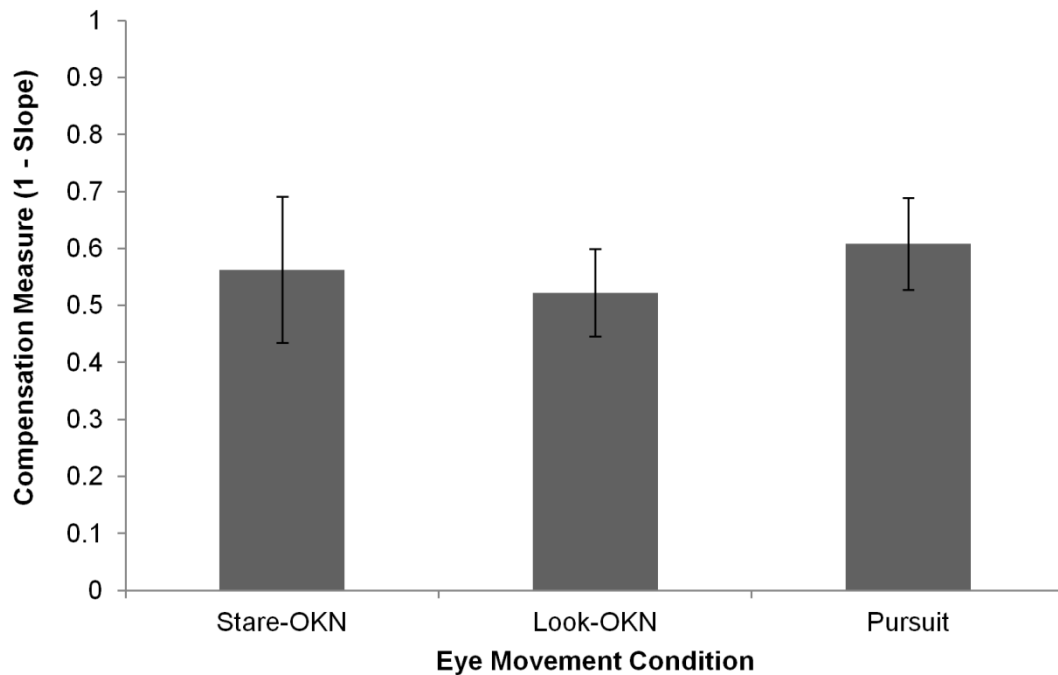
Figure 2.10: Mean compensation measure (1 - slope values) across each eye movement condition. Data has been pooled across different conditions of stimulus speed, and in the case of pursuit, different pursuit amplitudes. Error bars show ±1 × Standard Deviation with variance attributed to between subjects error removed (Cousineau, 2005).

Horizontal error did depend upon the type of eye movement which occurred during the saccade latency period ($F_{(2,8)}$ = 12.95, p = 0.003, η^2 = 0.76), however this effect was again driven by large errors in the look-OKN condition, these data are shown in Figure 2.11.
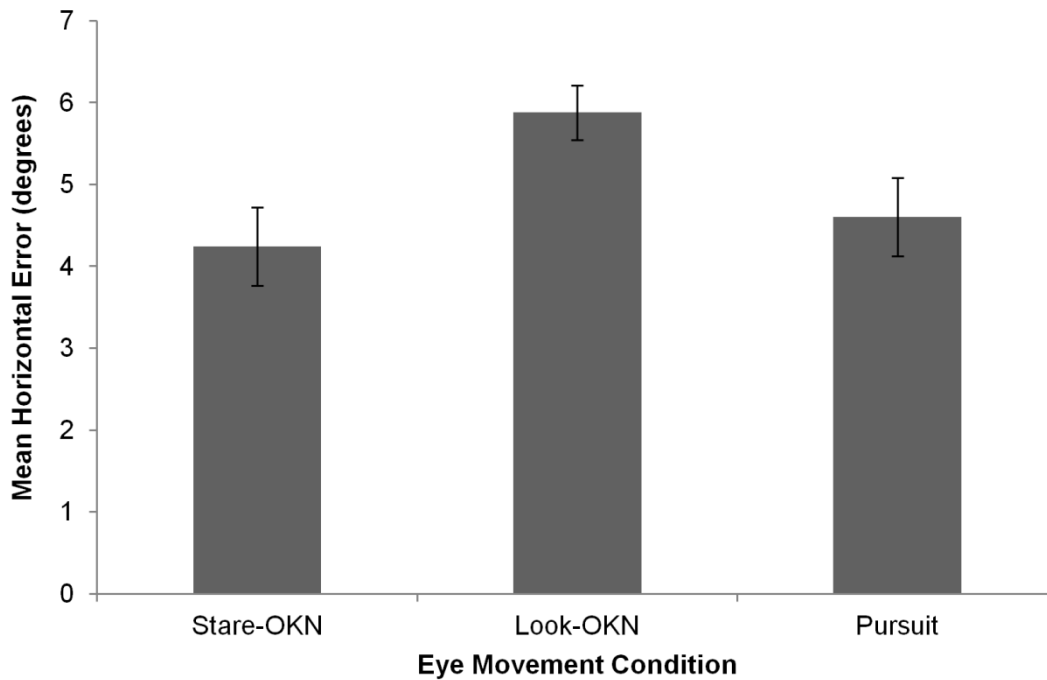
Figure 2.11: Mean errors across each eye movement condition. Data has been pooled across different conditions of stimulus speed, and in the case of pursuit, different pursuit amplitudes. Error bars show ±1 × Standard Deviation with variance attributed to between subjects error removed (Cousineau, 2005).

The amount of horizontal error from displacements due to look-OKN was larger than those for stare-OKN; this increase was present in all five observers ($t(4) = -5.45$, $p = 0.005$, $r = 0.94$). Moreover, all observers showed larger errors for look-OKN than pursuit ($t(4) = 4.27$, $p = 0.013$, $r = 0.91$). Errors did not appear to be any different between stare-OKN and pursuit, only two observers showed a decrease in error between stare-OKN and pursuit ($t(4) = -0.88$, $p = 0.427$, $r = 0.40$).

This experiment aimed to investigate effect of fast-phase or resetting saccade frequency upon targeting saccade accuracy. The mean frequencies are shown in Figure 2.12; where conditions have been divided based on whether they stimuli were presented at 32°/sec or 40°/sec, and, for the pursuit condition, whether the pursuit ramp had a short or long amplitude.

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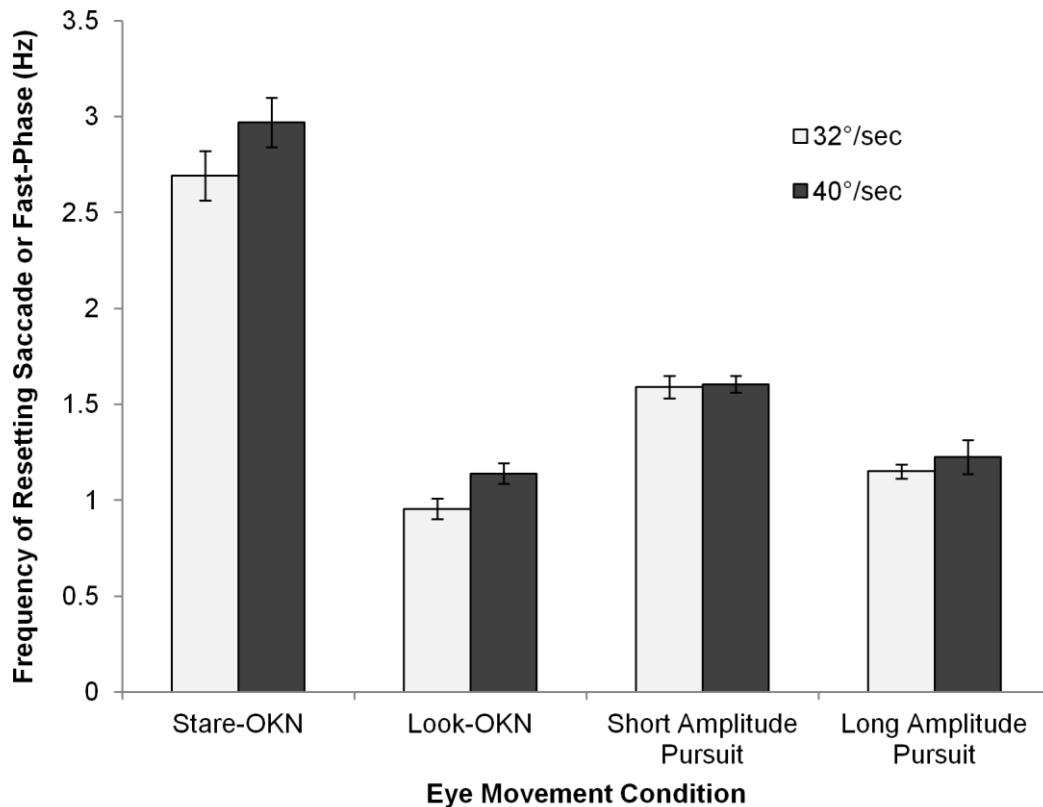
Figure 2.12: The frequency of resetting saccades or fast-phases in each condition. Light bars denote trials with a velocity of 32°/sec, dark bars denote a velocity of 40°/sec. Different pursuit amplitudes were employed to create two high frequency and two low frequency conditions, independent of stimulus velocity. Error bars show ±1 × Standard Deviation with variance attributed to between subjects error removed (Cousineau, 2005).

As noted previously (Freeman & Sumnall, 2005), there was a far higher fast-phase frequency for stare-OKN compared to look-OKN; indicating stimulus instructions were successful in eliciting either reflexive stare- or volitional look-OKN. This experiment concerns the relative change in frequency in both stare-OKN and look-OKN due to an increase in stimulus velocity. A factorial ANOVA conducted only on stare-OKN and look-OKN conditions revealed that although frequencies were significantly higher in stare-OKN ($F(1,4) = 43.72$, $p = 0.003$, $\eta_p^2 = 0.92$) and were significantly higher at faster stimulus speeds ($F(1,4) = 8.20$, $p = 0.046$, $\eta_p^2 = 0.67$), there was no significant interaction between eye movement type and

stimulus speed ($F(1,4) = 1.86$, $p = 0.244$, $\eta_p^2 = 0.32$). This would suggest that stare-OKN frequency was manipulated by stimulus speed, which was not predicted based upon previous literature (Cheng & Outerbridge, 1974). Therefore it was decided not to collapse data across stimulus speeds in the stare-OKN condition as was initially intended, but to instead create high and low frequency stare-OKN conditions based upon stimulus speed. Although this may not be the ideal analysis, frequency will be accounted for directly in a subsequent regression analysis (see below). Also note that analyses were performed collapsing data across stare-OKN conditions as was originally intended and the same results were obtained.

Unsurprisingly, changes in frequency were the clearest for the pursuit condition, where the experimenter has the ability to directly manipulate frequency. Looking only at the four pursuit conditions, stimulus velocity did not have a significant effect on frequency ($F(1,4) = 3.14$, $p = 0.151$, $\eta_p^2 = 0.44$), whereas the set amplitude did ($F(1,4) = 84.92$, $p = 0.001$, $\eta_p^2 = 0.96$). There was no significant interaction between amplitude and speed ($F(1,4) = 3.06$, $p = 0.155$, $\eta_p^2 = 0.43$). Thus it was concluded that pursuit can be divided up based on set frequency to create low and high frequency conditions independent of stimulus speed. The effect of frequency on the compensation measure is shown in Figure 2.13.
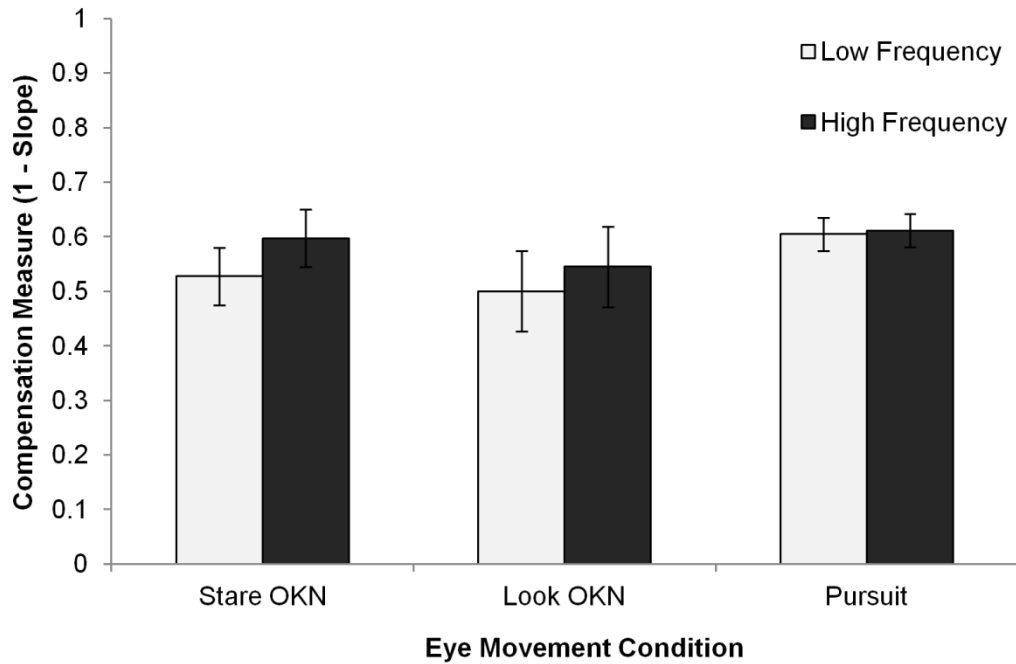
Figure 2.13: Compensation measure (1 – slope) for the correlation between displacement and error in each condition. Errors are shown for conditions of high or low resetting saccade/fast-phase frequency. Error bars show ±1 × Standard Deviation with variance attributed to between subjects error removed (Cousineau, 2005).

There was no indication that frequency had any influence on the slope compensation measure ($F(1,4) = 3.50$, $p = 0.135$, $\eta_p^2 = 0.47$). The impact of frequency on horizontal error is shown in Figure 2.14. Although there was a slight reduction in error for higher frequencies in both look-OKN and pursuit, these reductions were not significant ($F(1,4) = 0.66$, $p = 0.811$, $\eta_p^2 = 0.16$).
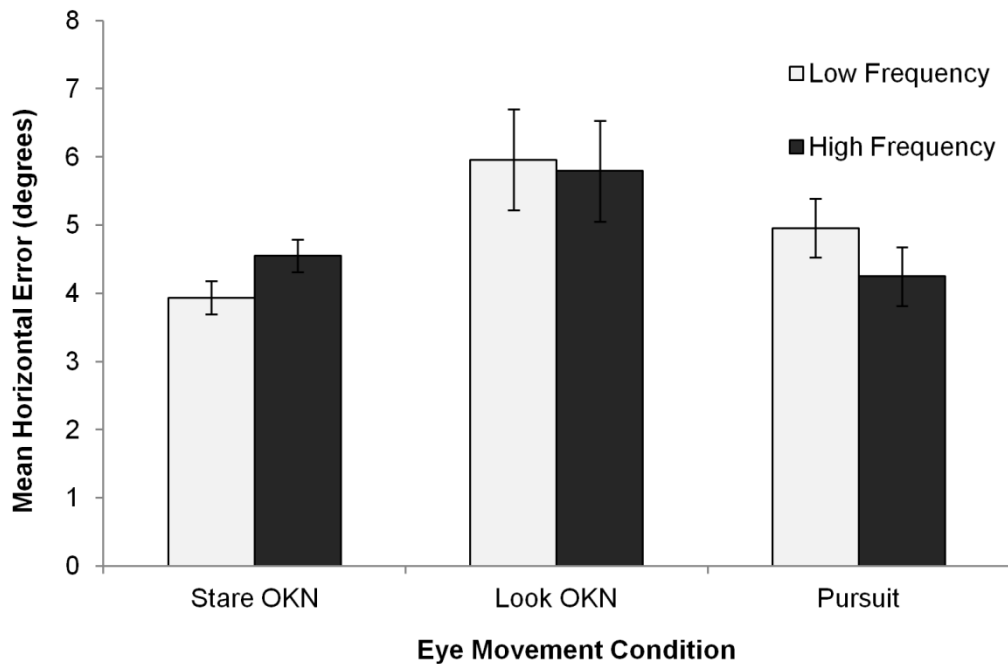
Figure 2.14: Horizontal error for conditions of high or low resetting saccade/fast-phase frequency. Error bars show ±1 × Standard Deviation with variance attributed to between subjects error removed (Cousineau, 2005).

It is possible that the lower errors in the higher frequencies could be attributable to the shorter amplitudes of the eye movement, the shorter amplitudes would be taken into account with the compensation measure (Figure 2.13). However similar results were found using either compensation measure or absolute error as the dependent variable.

Regression Analyses

The regression analyses aimed to investigate the influence of eye-movement condition (stare-OKN, look-OKN or pursuit) while accounting for any differences which could be attributed to frequency. The regression model allows frequency to be inputted as a continuous variable. This is useful because every saccade in this experiment will have a measurable error at fixation, but also it has a type of displacement (stare-OKN, look-OKN or pursuit), and a frequency of resetting fast-phases associated with it. Therefore one way these data can be

analysed is to use multiple regression methods. In a paper by Lorch and Myers (1990) it is stated that:

> "An alternative method of analyzing repeated measures data is to conduct a
> single regression analysis on the entire data set . . . each individual observation
> constitutes a separate "case" in the analysis and must be coded with respect to
> all independent variables". (Lorch & Myers, 1990, p. 154).

This means that the regression analysis is conducted upon the entire dataset, across all conditions and observers. Each "case" of the regression analysis is a particular saccade. Each saccade must then be tagged by which participant it comes from, and which in eye movement condition it occurred. Lorch and Myers (1990) describe how this can be achieved:

> "The independent variables include not only the predictors of interest but also
> subjects … Because subjects is a nominal variable, it must be coded as a vector
> of N - 1 dummy variables. . . After the coding is complete, the variables must
> be entered into the regression equation in several steps. The initial step is to
> partition the variability into between-subjects and within-subjects components.
> This is accomplished by entering the vector of subject variables as a single
> block to account for the between-subjects variability". (Lorch & Myers, 1990,
> p. 154).

This means that every data point can be assigned to a particular participant. By including this information in the regression model, variance in horizontal error can be attributed to whichever participant executed the saccade. Therefore 'participant' becomes an independent variable. By entering 'participant' into the regression model as an initial step, any between-subjects variance can be accounted for prior to investigating the influence of the other independent variables (e.g. eye movement condition, or frequency).

'Participant' was coded for with four standard dummy variables (see Field, 2005). Eye-movement condition was coded for with dummy variables such that stare-OKN formed the baseline category (Davis, 2010; Howell, 2007). This allowed the two dummy variables to represent the change in error between stare-OKN and look-OKN, and the change in error between stare-OKN and pursuit.

The model was run hierarchically in two stages. The first was to enter in the participant variables as a predictor to attribute as much variance in error as possible to between subject effects (following Lorch & Myers, 1990). Subsequent to this 'frequency', 'latency' and 'eye-movement condition' were entered into the model. The predictor variable of saccade latency was included because previous work has shown that the ability of saccades to compensate for smooth pursuit displacements depends upon the saccade latency (Blohm et al., 2005; Blohm et al., 2006).

The initial step of the regression analysis was to enter a categorical variable of participant in order to account for the between-subjects variance. This resulted in a significant initial step of the regression (R^2 = .04, $F(4,3078) = 10.67$, $p < 0.001$), showing a significant amount of the variance in error at fixation was attributed to between-subjects variability. The results of the second step of the regression analysis are shown in Table 2.6.

	B	SE B	β
Saccade Latency	0.02	0.01	0.06*
Frequency	-0.20	0.10	-0.01
Stare-OKN vs. Look-OKN	1.68	0.24	0.24**
Stare-OKN vs. Pursuit	0.56	0.19	0.09*

Note: R^2 for Step 1(Participant as predictor) = .014; ΔR^2 = .048 for Step 2 ($p < 0.001$). *$p < .01$; **$p < .001$

Variances accounted for by 'Participant' as a predictor have been left out for clarity

Table 2.6: Results from second step of multiple regression model. '*B*' = Unstandardized Beta Coefficient; '*SE B*' = Standard Error of Coefficient; '*β*' = Standardized Beta. The variance accounted for using Participant as a predictor has been left out of the table for clarity.

There are a number of notable points shown in Table 2.6, firstly frequency had no effect on the magnitude of error at fixation; this implies that it does not have an effect on the ability of saccades to compensate for displacements during the saccade latency period. There was a significant effect of latency ($t = 3.20$, $p = 0.001$), however this effect was very slight (beta = 0.02) and was in the opposite direction to what was predicted (i.e. longer latencies were associated with an increase in error). The difference in error between stare-OKN and look-OKN was significant ($t = 7.05$, $p < 0.001$), showing that whilst controlling for any effects of frequency and latency, errors were still significantly larger during look-OKN. The regression analysis also found that errors were significantly larger for pursuit than during stare-OKN ($t = 2.98$, $p = 0.003$).

2.4.6 *Discussion*

This experiment did not find any evidence that the accuracy of targeting saccades was influenced by resetting saccade or fast-phase frequency. Splitting the data up by resetting saccade/fast-phase frequency did not affect the magnitude of errors at fixation, nor the values of slopes when displacement is correlated with error. Furthermore, controlling for frequency

in a regression model did not change the effect of eye-movement condition on saccade accuracy. This means that there are two main conclusions which can be drawn from Experiment A3. The first is that no improvement in saccade accuracy was observed when the displacement was due to a volitional eye movement; this is exactly what was found in Experiment A2, and is further evidence that the saccadic system is as integrated with reflexive eye movements as it is with volitional eye movements. The second conclusion is that the results of Experiment A2 do not appear to have been unduly influenced by nystagmus frequency, which could have been a confounding variable between stare-OKN and the other eye movement conditions.

The regression model revealed a slight, but significant effect of saccade latency on fixation error. However, this effect is opposite to that which has been reported previously (Blohm et al., 2005; Blohm et al., 2006), longer saccade latencies were associated with larger errors at fixation. Although this experiment, like Experiment A2, did not utilize the same experimental manipulation as previous literature (e.g. Schlag et al., 1990). Experiments showing that longer latencies allow saccades to compensate for smooth pursuit displacements explicitly delayed the execution of the targeting saccade through the use of a cue (Schlag et al., 1990); whereas in the experiments reported here longer saccade latencies were the result of naturally occurring variance within the oculomotor system. It is not possible to equate internal, intrinsic motor variability with externally imposed delays; therefore one cannot say that the results of these experiments contradict those who report that delayed saccades are able to better compensate for smooth pursuit displacements.

Nevertheless, it is interesting that the regression model found longer saccade latencies to be associated with larger errors at fixation. Perhaps this could be due to the fact that longer saccade latencies mean that the eye travels further during the saccade latency period, making an accurate saccade more difficult to program. However, this relationship is far from clear

cut, a resetting saccade or fast-phase can occur during the targeting saccade latency; and a longer saccade latency increases the time during which this can happen. Therefore, paradoxically, a longer saccade latency may result in a smaller displacement distance if a resetting saccade or fast-phase is executed. An important point to note is that the regression model shows that the eye movement condition still has a significant effect upon saccade accuracy (with the smallest errors for stare-OKN) when the effect of saccade latency is held constant.

2.5 General Discussion of Chapter 2

This chapter aimed to address two research questions. The first is whether top-down targeting saccades are able to compensate for displacements due to reflexive optokinetic nystagmus. This relatively specific research question was motivated by a desire to understand how gaze co-ordination might be achieved when a moving observer views natural scenes. Additionally, this research hoped to address a second, more general question of whether there are interactions between reflexive and volitional eye movement systems.

The results presented here suggest that targeting saccades are partially sensitive to displacements of the eye from reflexive OKN. This contradicts those who claim that involuntary eye movements are not accompanied by extra-retinal signals (Post & Leibowitz, 1985; Wertheim, 1994; Whiteside et al., 1965), however this is very consistent with the results showing perceived location is partially sensitive to involuntary eye movements (Bedell & Currie, 1993; Currie & Bedell, 1991). The ability of saccades to compensate partially for optokinetic eye movements gives a potential mechanism which could help to co-ordinate targeting saccades made during self-motion. The results obtained here would suggest that this mechanism is not the only method by which targeting saccades can correct for involuntary gaze-stabilizing movements; because partial compensation would never allow a

completely accurate targeting saccade. A clear difference between the experimental paradigm used here and saccades under natural viewing conditions is that these experiments used open-loop saccades – there was no retinal information provided to help the saccadic system correct for any displacements. It is possible that retinal information could supplement the interaction between OKN and targeting saccades. For example, the double step paradigm has shown that retinal information can modify a saccade landing point up until about 80ms before the saccade is executed (Becker & Jürgens, 1979). A co-ordination of extra-retinal optokinetic information and retinal feedback might allow saccadic accuracy during gaze-stabilizing eye movements.

The observation that top-down saccades are sensitive to optokinetic movements suggests that volitional and automatic oculomotor systems cannot exist in complete isolation. This is evidence against those that claim automatic and voluntary motor actions are processed in separate and independent neural structures (Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977, 1984), and compliments research which suggests that automatic and volitional actions exist on the same continuum and use shared neural networks (McBride, Boy, Husain, & Sumner, 2012). Moreover, these experiments have also shown that the sensitivity of the saccadic system does not depend upon whether the displacement is due to a reflexive or a volitional eye movement. If it does not matter to the saccadic system whether the displacement is involuntary or not, then this suggests that automatic and volitional actions are processed in the same way by the oculomotor system and there is no appreciable difference between them. If this is true then is implies that the way in which automatic and volitional eye movements are processed is very similar indeed; and they should not be considered as separate and distinct.

In summary, these experiments have demonstrated that there do appear to be mechanisms by which top-down targeting saccades can compensate for reflexive OKN.

These compensatory mechanisms are no weaker than those which exist for volitional eye-movement displacements. These two conclusions are strong evidence that there is no separation of automatic and volitional eye movement generation in the oculomotor system, and show that mechanisms do exist to allow targeting eye movement to be co-ordinated with gaze-stabilizing eye movements. However there remain further questions about the behaviour of targeting saccades during optokinetic eye movements. One such question concerns the perceptual experiences which accompany the oculomotor behaviour. As mentioned above, the data collected here are consistent with research showing there is partial compensation for misperceptions of location during reflexive eye movements (Bedell, 1990; Bedell & Currie, 1992; Bedell et al., 1989). However, it is not at all clear as to whether motor control and perceptual experience are governed by the same information (Aitsebaomo & Bedell, 1992; Hansen & Skavenski, 1977; Lott & Bedell, 1995). Furthermore, if one proposes that the generation of reflexive and volitional eye movements utilizes a common neural mechanism, then one would also assume that misperceptions of location would be the same for both reflexive OKN and volitional pursuit. The examination of these questions formed the basis of the experiments in the next chapter.

Chapter 3: Oculomotor Control and Perceived Location during Optokinetic Nystagmus

3.1 Introduction

This thesis has so far established that a saccade executed during optokinetic nystagmus or pursuit can only partially compensate for the displacement of the eye which occurs during the saccade latency period. However, it is unclear whether the targeting saccade is directed toward the perceived location of the saccade target, or whether there is a dissociation between the oculomotor behaviour of the saccadic system and the perceptual judgement. This research question is relevant for two reasons: the first is that there is clear evidence that perceived locations are not veridical during optokinetic nystagmus or pursuit; and the second is that there are already reported dissociations between oculomotor action and perception. The evidence supporting these two claims will be discussed below. This chapter aims to compare the perceptual mislocalisations during OKN and smooth pursuit, and to investigate whether the behaviour of targeting saccades executed during OKN and smooth pursuit is dissociable from the perceptual judgement.

Perceptual Mislocalisations during Optokinetic Nystagmus and Smooth Pursuit

During smooth pursuit eye movements the locations of flashed stimuli are misperceived in the direction of the eye movement (Brenner & Cornelissen, 2000). The error is proportional to the velocity of the eye, and it is estimated that the error is about the distance which the eye will travel in 100ms (Brenner, Smeets, & van den Berg, 2001). Part of this error is accounted for by the time it takes for retinal signals to reach the brain, however neuronal delay times can only account for about 50ms of the 100ms discrepancy (Brenner et al., 2001). However, it is hypothesised that the retinal signal is combined with the efference copy of the eye-command; which necessarily specifies a point ahead of instantaneous eye position because it

codes the desired future location of gaze (Brenner et al., 2001; Klier & Angelaki, 2008). Thus at the time at which retinal and extra-retinal signals are combined, the retinal signal is generated from a point behind instantaneous eye position, and the extra-retinal signal specifies a point ahead of instantaneous eye position; combination of these two errors can account for the observed 100ms delay (Brenner et al., 2001).

Further evidence that the mislocalisation is due to a mismatch between extra-retinal and retinal signals is that mislocalisations appear to depend upon the movement of the eyes, not the movement of the target. In an experiment whereby the pursuit target could change direction unexpectedly, mislocalisation was related to the change in gaze after the target flash, not the change in target direction (Rotman, Brenner, & Smeets, 2004a). Furthermore, mislocalisation occurs during the pursuit latency period, before the eye is in motion but when the efference copy would be specifying the future position of the eye (Schütz, Braun, & Gegenfurtner, 2011). However, the mismatch between retinal and eye position signals cannot fully explain all of the characteristics of mislocalisation during pursuit. For example mislocalisations are considerably reduced if the pursuit is over a structured background (Brenner et al., 2001; Schütz et al., 2011), and fixed external reference points aid localisation greatly even if they are brief flashes which occur after the target (Noguchi, Shimojo, Kakigi, & Hoshiyama, 2007).

One also might wish to consider what the relationship is between misperceptions during smooth pursuit and the flash-lag effect. The flash-lag effect was first tested with moving and stationary stimuli while the observer maintained fixation; it was found that constant, moving stimuli were perceived as being ahead of brief flashes (Nijhawan, 1994). It was believed that this effect was due to a mechanism that was designed to compensate for neural transmission delays in the visual system; this could be achieved if the position of a moving stimulus is extrapolated by the visual system and its perceived location is therefore

ahead of its actual spatial position (Nijhawan, 1994). Brief flashes, on the other hand, are not subject to such extrapolation of position, and, *as long as fixation is maintained*, they are perceived veridically (Nijhawan, 1994, 2001). Of course we are interested in the situation whereby the moving stimulus is pursued, and eye movements make the situation more complex. Nijhawan (2001) investigated the flash-lag effect during pursuit eye movements and discovered that flashes are misperceived in the direction of pursuit (therefore the same finding as Brenner et al., 2001), which Nijhawan (2001) interpreted as a form of flash-lag effect. Nijhawan (2001) hypothesised that the flash-lag effect is due to the visual system not compensating for brief flashes (they are perceived as being at their retinal locations) therefore by the time retinal stimulation reaches 'higher' perceptual areas the flash will be perceived as being shifted in the pursuit direction due to the movement of the eye. However, Brenner et al. (2001) state that the degree of mislocalisation is too great to be due to retinal transmission delays alone, and postulate that the effect is also due to a predictive extra-retinal signal (as described fully above).

Nijhawan (2001) also consider an alternative option, that a stationary stimulus shown during smooth pursuit will have retinal motion in the direction opposite to pursuit. This might cause a flash-lag effect such that a stationary stimulus shown during pursuit is misperceived in the opposite direction to the eye movement; it is unlikely that this would occur in this experiment as brief flashes (such as are employed here) create a negligible amount of retinal motion (Nijhawan, 2001); however if it were discovered that our stimulus is misperceived in the opposite direction to pursuit then the a flash-lag effect due to retinal motion of a stationary stimulus could be a potential explanation.

During reflexive OKN there are misperceptions of location that appear very similar to those observed during pursuit; targets are mislocalised in the direction of the slow-phase (Kaminiarz et al., 2007; Tozzi et al., 2007). The misperception temporarily decreases shortly

before a fast-phase, and temporarily increases after the fast-phase has been competed (Kaminiarz et al., 2007; Tozzi et al., 2007). Interestingly, the decrease and subsequent increase of mislocalisation around the fast-phase is consistent with mislocalisations which occur during saccades (Kaminiarz et al., 2007). Around 100ms before a saccade is executed, perceived locations shift in the direction of the saccade; and shortly after the saccade is completed, positions are misperceived against the direction of the saccade (Honda, 1991). This pattern is thought to occur because in anticipation of the saccade there is a remapping of receptive fields, such that neurones will respond to stimuli which will be within their classical receptive fields upon completion of the saccade (Klier & Angelaki, 2008; Ross et al., 2001). For example, neurones in the lateral intraparietal area respond up to 80ms before a saccade to stimuli which will fall within their classical receptive field after the saccade is completed (Duhamel, Colby, & Goldberg, 1992). The rebound in mislocalisations after the saccade might be due to the resetting of receptive fields back to their classical organisation being slower than the saccade itself (Ross et al., 2001).

This pattern of perisaccadic mislocalisation can account for the temporary changes in perceived location seen during OKN fast-phases. This mechanism is briefly outlined in Figure 3.1.
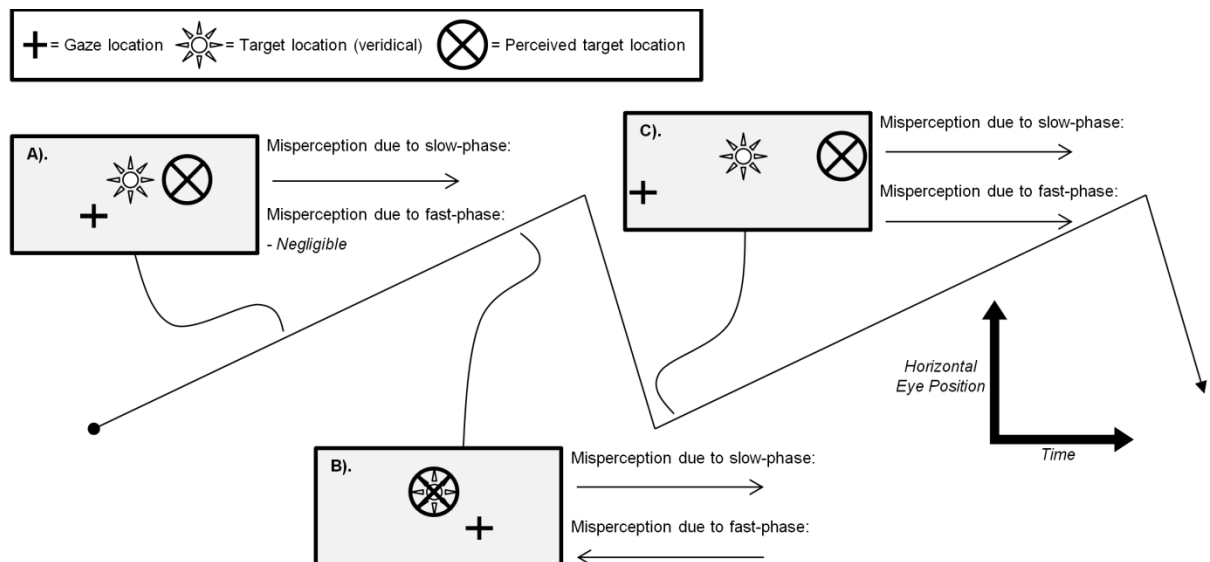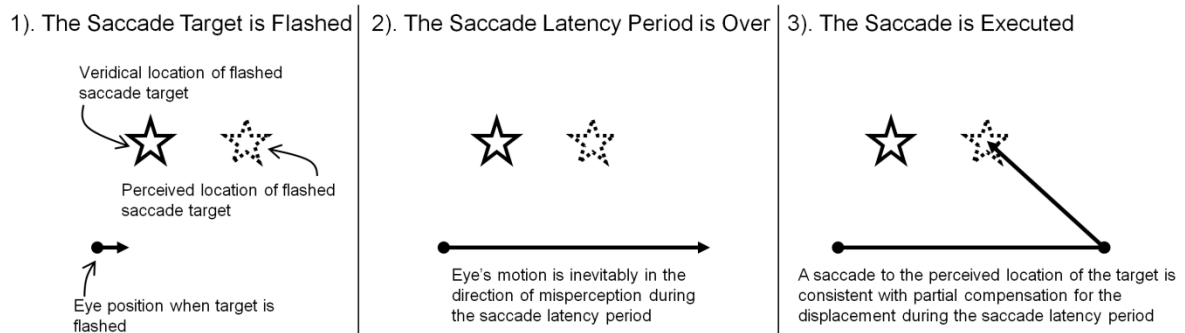
Figure 3.1: The time course of mislocalisations during OKN can be explained through interactions between misperceptions arising from the slow-phase and the fast-phase. Centre line illustrates horizontal gaze-location during OKN. *A).* **In the middle of the slow phase, targets are misperceived in the direction of the eye movement.** *B).* **Shortly before a fast-phase, misperceptions due to slow- and fast-phases are in opposition, thus perceptual mislocalisations are temporarily reduced.** *C).* **Shortly after a fast-phase misperceptions are in the same direction, therefore perceptual mislocalisations are temporarily increased.**

By combining the pattern of mislocalisation during pursuit and during saccades in the way outlined in Figure 3.1, one can very neatly explain the pattern of perceptual mislocalisation seen during OKN (Kaminiarz et al., 2007; Tozzi et al., 2007). This would suggest that perception during the slow-phase is analogous to perception during smooth pursuit, and perception during the fast-phase is analogous to perception during saccades. There is some support for the notion that perception during pursuit is analogous to that during OKN slow-phases. For example it has been found that sensitivity to chromatic and high-spatial frequency stimuli is enhanced during smooth pursuit (Schütz, Braun, Kerzel, & Gegenfurtner, 2008), and a similar enhancement is observable during OKN slow-phases (Schütz, Braun, & Gegenfurtner, 2009). However differences in perception during pursuit and OKN have been found. For example, mislocalisations during pursuit appear to be greatest

for stimuli presented in the hemifield which the eyes are travelling towards (Königs & Bremmer, 2010); this does not appear to be the case during OKN slow-phases (Kaminiarz et al., 2007). Furthermore, the perception of auditory targets appears to follow the pattern of visual mislocalisations during OKN (Königs, Knöll, & Bremmer, 2007); however this does not appear to be the case during smooth pursuit (Königs & Bremmer, 2010). During smooth pursuit auditory targets have been found to be mislocalised in the direction of the smooth pursuit eye movement, as are visual stimuli, however it was found that auditory targets are subject to an expansion of space; whereas visual localisation was subject to a compression of space (Königs & Bremmer, 2010).

There are furthermore differences between the patterns of mislocalisation seen during fast-phases and during saccades. For example, prior to a saccade there is a compression of visual space towards the saccade end-point (Ross et al., 2001). This compression of has not been found during the fast-phases of OKN (Tozzi et al., 2007). It is possible that there is no compression of space prior to OKN fast-phases because they do not have an explicit visual goal (Tozzi et al., 2007).

Thus there are well-documented mislocalisations which occur during smooth pursuit and OKN; and there are similarities in the pattern of misperceptions between smooth pursuit and the OKN slow-phase. Because locations are shifted in the direction of eye-motion it is possible that results of Chapter 2 show that the saccadic system is similarly affected by the misperceptions. For example, if misperceptions are is the direction of the eye movement, then when the targeting saccade is executed during OKN it will have moved in the direction of the eye movement. A saccade to the target's perceived location will result in a pattern of saccadic behaviour consistent with partial compensation for the displacement of the eye. This process is outlined in Figure 3.2.

1). The Saccade Target is Flashed | 2). The Saccade Latency Period is Over | 3). The Saccade is Executed

Veridical location of flashed saccade target

Perceived location of flashed saccade target

Eye position when target is flashed

Eye's motion is inevitably in the direction of misperception during the saccade latency period

A saccade to the perceived location of the target is consistent with partial compensation for the displacement during the saccade latency period

Figure 3.2: If a saccade target is presented during OKN or smooth pursuit, then it will be misperceived in the direction of the eye movement. Thus, a resulting saccade to the perceived location of the target will give a pattern of saccadic accuracy consistent with partial compensation for the eye's displacement

However, although the results of Chapter 2 are consistent with the literature of misperceptions of location during smooth pursuit and OKN, it is unclear whether the behaviour of the oculomotor system actually does follow the perceptual experience. Some authors claim that perceptual judgements and oculomotor behaviour use different sources of information; this research is discussed in the following section.

Dissociations between oculomotor actions and perception

There are several examples in the literature of apparent dissociation between the information available to eye movements and to perception. Here I will outline two prominent examples. It is well-established that stimuli which appear very shortly before a saccade is executed are misperceived in the direction of the saccade (Honda, 1991; Ross et al., 2001). However, the double step paradigm has revealed that if a target appears shortly before the execution of a saccade (i.e. during the time in which locations are misperceived), then a second saccade can be accurately executed to the location of this target (Hallett & Lightstone, 1976). This would suggest that there is a dissociation between the information used to form a perceptual judgement, and that which guides the saccadic eye movement. Other motor actions have also

been reported as being unaffected by perceptual mislocalisations; for example participants can accurately strike hammer blows toward stimuli which are presented near the time of a saccadic eye movement (Hansen & Skavenski, 1985).

However, other authors have found that saccades are in fact aimed at the perceptual locations of targets flashed near to saccadic eye movements (Dassonville, Schlag, & Schlag-Rey, 1992; Honda, 1989); thus the discrepancy between the perceptual judgement and the oculomotor behaviour found by Hallett and Lightstone (1976) may be methodological. It is possible that the results of Hallett and Lightstone (1976) were due to a relatively long target duration which may have given enough retinal feedback to allow accurate saccades (Dassonville et al., 1992). Moreover the delay between the first and second saccade targets was constant, this may have allowed saccadic adaptation to occur which biased the saccade end point (Dassonville et al., 1992). Another potential methodological factor is that the saccade target was always in the same place, and revealed to the observer at the end of each trial (Honda, 1989). Varying time between targets and using shorter-duration flashes reveals that saccades are affected by mislocalisations in the same way as perceptual judgements (Dassonville et al., 1992; Schlag & Schlag-Rey, 2002).

Dissociations between action and perception during smooth pursuit eye movements have also been reported. Stimuli flashed during a smooth pursuit eye movement are misperceived in the direction of pursuit (Brenner et al., 2001), however Hansen (1979) reported that participants could accurately strike the location of a target flashed during pursuit with a hammer blow. Accurate motor actions during pursuit would suggest that the motor system has access to up-to-date information about eye position, whereas the perceptual system does not. However, this result has not always been found, other authors have reported that motor actions during smooth pursuit are biased in the same way as perceptual judgements (Kerzel, Aivar, Ziegler, & Brenner, 2006; Rotman, Brenner, & Smeets, 2004b). It

is unclear why Hansen (1979) found accurate motor responses during pursuit whereas other researchers did not. One possibility is that Hansen (1979) used very high luminance flashes; it has been found that localisation is better for a bright flash in the dark (Kerzel et al., 2006). However the luminance of the target was not reported by Hansen (1979) so this remains only a possible reason for the divergence in the literature.

Therefore there is some evidence that there are dissociations between perceptual experience and motor actions during eye movements; however the evidence is not entirely consistent. This chapter aimed to investigate whether the behaviour of targeting saccades described in Chapter 2 followed the perceptual experience of where the saccade targets were located. However, prior to this experiment a short control study was carried out to investigate the influence of stimulus motion on perceived locations of objects. This was done in order to be more confident that any effects observed during smooth pursuit or OKN were due to the eye movements themselves, not stimulus motion.

3.2 Experiment B1 - Influence of Retinal Motion on Perceived Location

During smooth pursuit the velocity of the eye will lag behind stimulus velocity to a certain degree (Collewijn & Tamminga, 1984). The same is true for the slow-phases of OKN, eye velocity is slower than the stimulus velocity (Garbutt et al., 2003). This means that despite the tracking movements of the eyes during pursuit and OKN, there will still be movement upon the retina.

It has been shown that when fixation is maintained, a moving stimulus can cause the perceived location of briefly presented stimuli to be shifted in the direction of the motion (Whitney & Cavanagh, 2000). This shift in perceived location occurs even if the test stimuli and the moving stimuli are separated by a considerable distance (Whitney, 2002) or if the test stimuli are also in motion (Whitney & Cavanagh, 2002). The misperception from moving

stimuli appears to be a very low-level effect, even modulating the retinotopic mapping of area V1 (Whitney, Goltz, et al., 2003). Furthermore a moving stimulus affects manual reaching responses as well as perceptual judgements, suggesting it has a common effect on both perceptual and motor pathways (Whitney, Westwood, & Goodale, 2003).

Because eye velocity will never exactly match stimulus velocity, there will inevitably be some degree of retinal motion during OKN or smooth pursuit. Therefore this experiment aimed to investigate what effect this retinal motion might have on perceived locations.

3.2.1 *Participants*

This experiment was conducted on five participants, all of whom had participated in previous eye-tracking experiments. Ages ranged from 23 to 25 years and three participants were male. This number of participants was fixed prior to commencing experimentation, and is consistent with previous studies, all of which employed 3 or 4 participants in order to detect psychophysical effects (Whitney & Cavanagh, 2000, 2002; Whitney, Goltz, et al., 2003; Whitney, Westwood, et al., 2003).

3.2.2 *Stimuli*

This experiment used the same band of random dots used to elicit OKN in Chapter 2. This band of dots is described in full in Section 2.2.3. From Experiment A2 (Section 2.3) it was found that the mean eye velocity during slow-phases was 26.3°/sec; therefore the band of dots moved at 5.7°/sec (estimated to be the speed at which stimuli moved upon the retina during OKN: stimulus speed = 32°/sec, eye velocity = 26.3°/sec, therefore retinal slip velocity = 5.7°/sec). Fixation was maintained through the use of a single dot of radius 0.3°. This dot was distinguishable from the band of moving background dots by its brightness of 1.06cd/m^2, whereas the background was only 0.1cd/m^2. However, as the fixation point was still hard to distinguish from the background, a ring was placed around it, which had a radius

of 1°, and a thickness of 0.1°. During OKN stimulation participants will tend to unconsciously maintain their average gaze location in a direction opposite to stimulus motion, this is known as contraversion (Garbutt, Harwood, & Harris, 2002). From previous experiments it was found that the mean degree of contraversion was 4.9°, with a standard deviation of 6.2°. Therefore the fixation location was determined randomly from a Gaussian distribution with a mean of 4.9 and a standard deviation of 6.2; the mean value was positive for leftward stimuli, and negative for rightward stimuli. The band of moving dots was displayed for 3700 to 4500ms, whereupon the target was presented. This consisted of a single dot, 0.6° in radius, presented for 14ms. It was positioned horizontally within 4° of gaze location and at ±10° vertically. Therefore the target was presented in the same way that the saccade target was presented in Experiment A2. The band of moving dots remained on the screen for 14ms following the extinguishing of the target stimulus, and the screen was blank until the test stimulus was presented 200ms later. Perceived location of the target was ascertained using judgements of whether the test was to the left or the right of the target using a staircase procedure.

3.2.3 *Staircase Design*

Participants indicated the perceived location of the target by reporting whether a presented test stimulus was to the left or the right of the target stimulus. This test stimulus was a single dot (radius = 0.6°, brightness $1.06cd/m^2$) presented for 100ms, and was always presented at the same vertical location as the target. The test was presented 200ms after the target stimulus, during which the screen was dark. Two-hundred milliseconds appears to be a long enough time for the influence of motion to dissipate; for example 200ms following motion reversal the perceived location of stationary objects also reverses (Whitney & Cavanagh, 2000). The test stimulus was initially presented 3° horizontally from the target, and the initial

step size was 1°. Following each reversal the step size decreased by half, and the staircase ended after six reversals.

Eight staircases were run in parallel: Four each for either leftward or rightward stimulus motion, within which two began with the test presented 3° to the left of the target, and two began with the target 3° to the right of the target (one staircase for targets appearing above the band of dots, one staircase for targets appearing below the band). The degree of perceptual mislocalisation was indexed by taking the mean position of each reversal.

3.2.4 Procedure

Participants were sat at the eye-tracker and a calibration performed. Before each trial began a bright screen of 0.38cd/m^2 was displayed for 2000ms in order to keep participants from dark-adapting during the experiment and being able to perceive the furniture of the lab (as this might allow perceived locations to be based upon fixed external reference points). Participants were instructed to maintain fixation upon the bright dot, ignoring the moving band of dots, and that a target would flash above or below the band of dots. Participants were asked not to saccade to the target flash, but to keep their eyes on the fixation point throughout the trial. 200ms following the target flash the test stimulus was presented for 100ms and participants indicated whether it was to the right or the left of the target with a left or right mouse click. Participants were allowed 1200ms in which to make this judgement, if a response was not made in this time the trial was discounted. Following the response there was an intertrial interval of 1000ms before the next trial began. Participants were given the opportunity to take a break every 25 trials.

3.2.5 Results

Perceptual mislocalisations were calculated from the mean locations of staircase reversals. These data are shown in Figure 3.3.
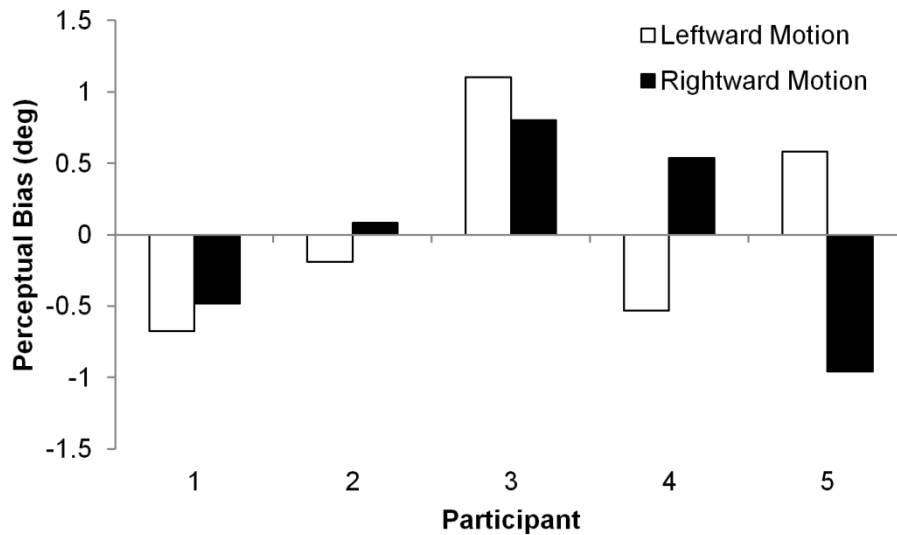
Figure 3.3: Individual perceptual mislocalisations of a target flashed while viewing leftward (white) or rightward (black) motion. Positive values indicate a rightward bias, whereas negative values indicate a leftward bias.

There did not appear to be a consistent effect of stimulus motion on perceived locations; there were substantial individual differences in the pattern of results found. Although participants 2 and 4 showed biases in the direction of motion, participant 5 showed a bias against the direction of motion. Furthermore participant 1 showed a general leftward bias, whilst participant 3 showed a general rightwards bias.

3.2.6 *Discussion*

From the results of Whitney and Cavanagh (2000) one would expect perceived location to be shifted in the direction of the presented motion. Although this pattern was found in participants 2 and 4, it was not seen in the other three participants. It is not entirely clear why this experiment did not replicate the findings of Whitney and Cavanagh (2000); however one possibility is that the stimulus motion in this experiment was too slow, the moving stimulus of Whitney and Cavanagh (2000) had a velocity of 13.4°/sec, however in this experiment stimulus velocity was 5.7°/sec.

Although this experiment aimed to investigate the effect of misperception of location that would occur under conditions of retinal motion it is unclear how the Whitney effect would interact with eye movements themselves, and this is listed as an unresolved question in Whitney (2002). To my knowledge, the Whitney effect has only ever been tested while the eyes are stationary. However, stimulus motion may is still cause perceptual effects even if it is not accompanied by retinal motion, for example the flash lag effect is found even when they eyes track the moving stimulus and retinal motion is zero (Schlag, Cai, Dorfman, Mohempour, & Schlag-Rey, 2000), as such one could say that the flash-lag effect occurs as a general effect of stimulus motion, not retinal motion per se. Therefore although we can state that retinal slip velocities expected during OKN are not sufficient to produce a Whitney effect, it is unclear whether one would occur due to the stimulus motion itself (i.e. if stimulus velocity was 32°/sec). Nevertheless, the results of this experiment mean we can conclude that any consistent mislocalisations that are observed during OKN or pursuit can be better attributed to the eye movements themselves, rather than any motion on the retina (as has been employed thus far in testing the Whitney effect). However, any general effect of veridical stimulus motion (i.e. not only retinal motion) upon the perceptual effects observed during OKN or pursuit remains an open question.

3.3 Experiment B2 – Perceived location during OKN and Pursuit

This experiment investigated the extent to which locations are misperceived during OKN or pursuit eye movements. Previous research has shown that locations are misperceived in the direction of the eye movement during the slow-phases of OKN, however this misperception decreases before a fast-phase, and temporarily increases after a fast-phase (Kaminiarz et al., 2007; Tozzi et al., 2007). This experiment investigated these effects using a different measure of perceived location. The experiments of Kaminiarz et al. (2007) and Tozzi et al. (2007) used an on-screen ruler for the participant to judge the location of flashed targets. In the case

of Kaminiarz et al. (2007) this ruler was presented at the end of the trial, and in Tozzi et al. (2007) it was continuously present, however was in the periphery of the visual field throughout. It is possible that the marks on the ruler are also mislocalised during OKN, therefore it may not be the best measure in order to establish the perceived location of target flashes. It was hoped that the use of left-right judgements on test stimuli in a staircase (as was performed in Experiment B1) would help to confirm the results of Kaminiarz et al. (2007) and Tozzi et al. (2007).

The main aim of this experiment was to directly compare the mislocalisations during OKN and those during alternating smooth pursuit with resetting saccades. This would allow investigation of whether the degree of mislocalisation during OKN slow-phases is similar to that seen during smooth pursuit. Furthermore, I wished to observe whether errors decrease and subsequently increase around the resetting saccade movement, as they do for fast-phases. If this is found to be the case, then it would be good evidence that the pattern of mislocalisations during fast-phases is the same as during saccades. Moreover, a common pattern of perceptual mislocalisations during OKN and smooth pursuit with resetting saccades would further indicate that reflexive and voluntary eye movements are generated in very similar ways, and there are commonalities in the way they are programmed by the visuomotor systems.

3.3.1 *Participants*

Five participants were used in this experiment. All of whom had participated in Experiment B1. This number of participants was fixed before the experiment started, and is consistent with previous literature on the perception of location during eye movements (between 3 and 9 participants: Brenner et al., 2001; Kaminiarz et al., 2007; Königs & Bremmer, 2010; Tozzi et al., 2007).

3.3.2 *Stimuli*

For the OKN condition stimuli were exactly as described in the OKN condition of Experiment A2; however the saccade target in Experiment A2 was now used as a perceptual target, the subjective location of which was determined using a staircase. The same was true for the smooth pursuit condition, which was exactly the same as the 'Full-Field Pursuit' condition described in Experiment A2 (thus the band of moving OKN dots was maintained alongside the pursuit target).

3.3.3 *Staircase Design*

The start position of the test stimulus was always ±6° from the veridical location of the target. The initial step size was 2°, and this halved following each reversal. The staircase ended after nine reversals had occurred. Six staircases were conducted in parallel, in three of which the difference between the target and the initial test position was in the same direction as stimulus motion, and in the other three, the difference between target and test was in the opposite direction as target motion. Three staircases were used in each condition so that the target could be presented early, in the middle, or late into the slow-phase. For the OKN condition this meant presenting the target 60ms, 110ms or 160ms following the detected fast-phase; for the smooth pursuit condition the target was triggered 140ms, 290ms or 590ms from the detected resetting saccade. Other features of the staircase were exactly as described in Experiment B1.

3.3.4 *Procedure*

All participants initially conducted the OKN condition, where they were given the same viewing instructions as in Experiment A2 in order to elicit reflexive stare-OKN. Participants viewed a bright screen ($0.38cd/m^2$) for 2000ms before each trial to prevent dark adaption. Subsequently they viewed the band of moving dots until the target appeared; participants were asked not to make an eye movement to the target. 200ms later the test stimulus was

presented, and participants were given 1200ms to make a response. The procedure was exactly the same for the smooth pursuit condition, however participants were asked to follow the single bright dot only (as in the full-field pursuit condition of Experiment A2).

3.3.5 *Results*

The perceptual mislocalisations were represented relative to stimulus motion. Therefore positive mislocalisations indicate a misperception in the direction of the slow-phase; this is what would be predicted based on previous literature (Kaminiarz et al., 2007; Tozzi et al., 2007). Figure 3.4 shows the degree to which participants mislocalised the test stimulus in both OKN and pursuit.
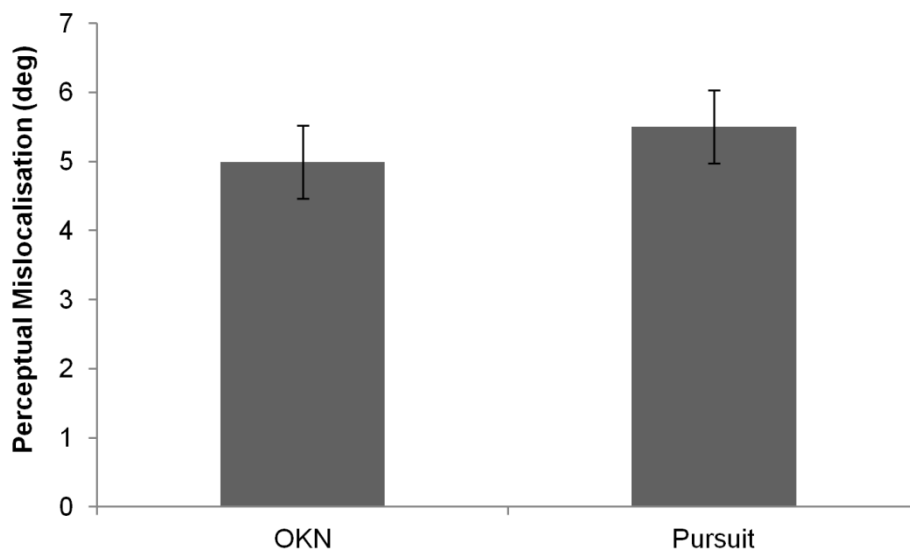
Figure 3.4: Perceptual mislocalisation during reflexive OKN or volitional pursuit. Mislocalisations are reported relative to stimulus direction, thus a positive mislocalisation indicates that the target was misperceived in the direction of the slow-phase eye movement. Error bars show ±1 × Standard Deviation with individual differences removed (Cousineau, 2005).

Figure 3.4 shows that participants clearly mislocalised the target in the direction of the slow-phase of the eye movement. One-sample t-tests comparing the mislocalisations to zero show that this was significant for both OKN ($t(4) = 15.31$, $p < 0.001$, $r = 0.99$) and pursuit ($t(4) =$

14.77, $p < 0.001$, $r = 0.99$). There was no evidence of a difference between mislocalisations for OKN and pursuit ($t(4) = -1.07$, $p = 0.343$, $r = 0.47$).

Staircases were separated based upon whether the target was presented early, in the middle, or late into the slow-phase of the eye movement. Previous research has found a temporary reduction in error before the fast-phase, and a temporary increase after the fast-phase (Kaminiarz et al., 2007; Tozzi et al., 2007). Therefore it follows that errors should be largest shortly into the slow-phase (as a fast-phase has just been completed) and smallest late into the slow-phase (as the system is preparing to make another fast-phase). Perceptual mislocalisations split by staircase timing are shown in Figure 3.5.
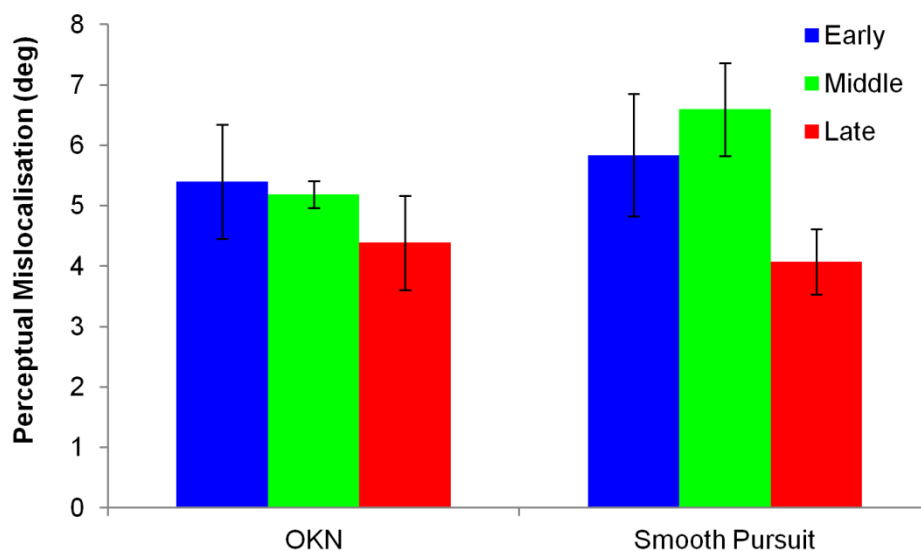
Figure 3.5: Mislocalisations relative to stimulus motion based upon when during the slow-phase the target was presented. The target could be presented early in the slow-phase (i.e. shortly after a fast-phase), in the middle, or late into the slow-phase (i.e. presumably shortly before a fast-phase). Error bars show ±1 × standard deviation with individual differences removed (Cousineau, 2005).

The degree to which target locations were misperceived did not depend upon whether the participant was executing OKN or pursuit ($F(1,4) = 1.16$, $p = 0.343$, $\eta_p^2 = 0.22$). However, as shown in Figure 3.5 there was a reduction in error for those targets presented late on in the

slow-phase. The effect of when during the slow-phase the target was presented was found to be significant ($F_{(2,8)}$ = 5.84, p = 0.027, η_p^2 = 0.59). There does not appear to be a consistently high error for those targets presented early on in the slow phase. However, the on-line detection of fast-phases does not necessarily guarantee that the target will be presented at the desired point in the slow-phase. For example, during the delay between the detected fast-phase and target presentation another fast-phase may occur (especially if the delay is long as in the 'Late' conditions). This would result in the time between the last fast-phase and target presentation being shorter than desired. Furthermore the presentation might erroneously detect a fast-phase when none actually occurs; this could be caused by blinks or catch-up saccades. This means that the time between the last fast-phase and target presentation is longer than desired. One way to overcome these limitations is to confirm the time between fast-phases and target presentations using off-line analysis and group targets based on when they actually occurred relative to the last fast-phase. These data can then be used to construct psychometric functions, which are shown in Figure 3.6.
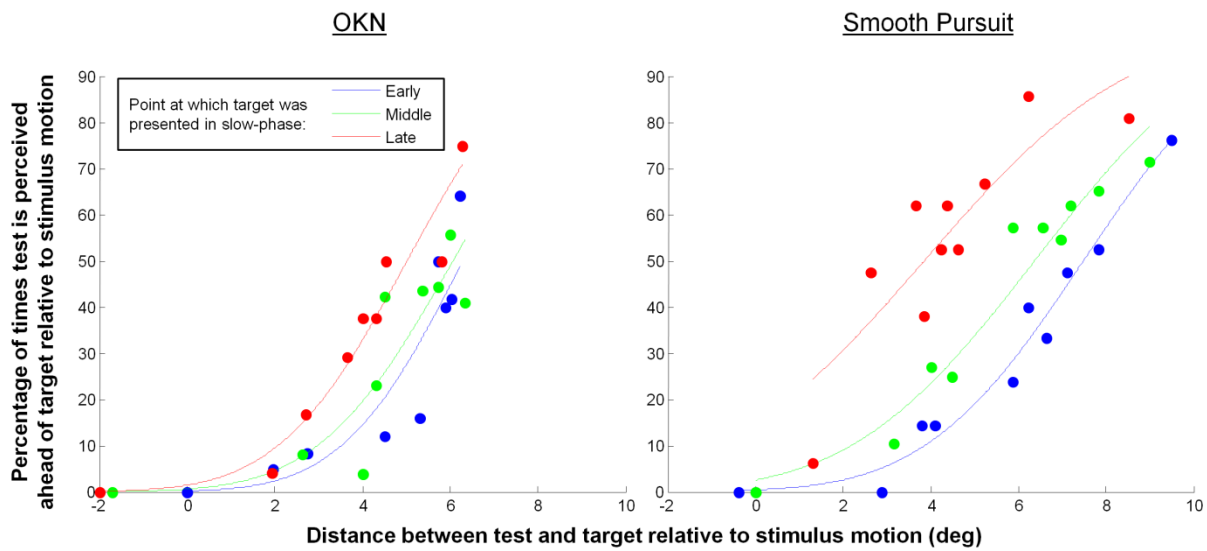
Figure 3.6: Psychometric functions showing the perceptual mislocalisations apparent when the target is presented early in the slow-phase, in the middle, or at the end of the slow-phase. Data has been pooled from all participants. Fits are from a Maximum Likelihood Model (Klein, 2001).

Figure 3.6 shows that error is greatest when the target is presented early in the slow-phase (i.e. shortly after a fast-phase) and is reduced when the target is presented near the end of the slow-phase. By taking the point of subjecting equality it was found that the effect of whether the target was presented early, in the middle or late into the slow-phase was found to be significant ($F_{(2,8)} = 15.08$, $p = 0.001$, $\eta_p^2 = 0.84$). Perceptual mislocalisations did not differ between OKN or pursuit ($F_{(1,4)} = 0.12$, $p = 0.748$, $\eta_p^2 = 0.03$). There was a significant interaction between the effect of slow-phase timing and whether the condition was OKN or pursuit ($F_{(2,8)} = 5.15$, $p = 0.037$, $\eta_p^2 = 0.56$), indicating that the extent to which fast-phase or saccade timing effected mislocalisations depended upon whether the participant was executing OKN or pursuit.

Previous literature found that during pursuit errors were largest when the target was presented in the retinal hemifield which the eyes were moving towards (Königs & Bremmer, 2010); however this effect was not previously found during the slow-phases of OKN (Kaminiarz et al., 2007). The data here did not find that localisation errors were any different depending upon the hemifield in which the target was presented ($F_{(1,4)} = 0.58$, $p = 0.491$, $\eta_p^2 = 0.13$), and there was no interaction between hemifield and whether the eye movement was pursuit or OKN ($F_{(1,4)} = 1.91$, $p = 0.239$, $\eta_p^2 = 0.32$).

3.3.6 *Discussion*

This experiment found that targets are indeed mislocalised in the direction of the slow-phase during OKN, just as they are mislocalised in the direction of the eye movement during pursuit. This is consistent with previous literature showing mislocalisations during OKN or pursuit (Brenner et al., 2001; Kaminiarz et al., 2007; Tozzi et al., 2007). Therefore measurements from a staircase procedure give the same conclusions as the use of an on-

screen ruler employed by Kaminiarz et al. (2007) and Tozzi et al. (2007). The data collected here suggest that the degree of localisation error does not depend upon whether the observer is executing reflexive OKN or smooth pursuit; this implies that there are commonalities in the way the perceptual system processes reflexive or volitional eye movements.

In this experiment the pattern of mislocalisations observed during OKN is consistent with the results of Kaminiarz et al. (2007) and Tozzi et al. (2007). Errors were lowest when the target was presented late into the slow-phase (therefore presumably presented shortly before a fast-phase) and errors were highest when the target was presented early on in the slow-phase (therefore shortly after a fast-phase). The same pattern of mislocalisation was observed during alternating smooth pursuit and resetting saccades; which suggests that the changes in mislocalisation around the OKN fast-phase are very similar to the changes in mislocalisation which occur around voluntary saccades (Honda, 1989; Ross et al., 2001). This further reinforces the point that voluntary and automatic eye movements appear to have common consequences for the perceptual system. There was a significant interaction between the eye movement type (OKN or pursuit) and the time at which the target was presented during the slow-phase. This is observable in the way in which the psychometric functions have different distances between each other depending on whether OKN or pursuit is executed (see Figure 3.6). This might imply that there are differences in the way fast-phases and slow-phases interact compared to smooth pursuit and saccades. However it is hard to directly make this comparison because voluntary resetting saccades cannot occur at the same high frequency as OKN fast-phases. This means that the distribution of times between the start of the slow-phase and target presentation is much tighter during OKN than during smooth pursuit. Therefore although one can say that the relative pattern of mislocalisation is the same in both OKN and pursuit, comparing the absolute values obtained may not be possible. One way to overcome this problem would be to take specific target presentation

times relative to a fast-phase or resetting saccade, and compare these across OKN and pursuit. Unfortunately there was not enough data collected in this experiment to make this analysis tenable.

Previous authors have stated that the perceptual error during smooth pursuit is about the distance which the eye will travel in 100ms (Brenner et al., 2001); this would equate to an error of around 3.2° for this experiment, however errors were actually much higher than this, with a mean error of 5.25°. It is not altogether clear why errors were larger than one would predict for this experiment, however one possibility is that it is due to the targets used. A component of the 100ms delay Brenner et al. (2001) found was due to delays in the transfer of retinal signals to visual cortex. The targets used by Brenner et al. (2001) were presented much more centrally (1.24° compared to 10°) and were considerably brighter (5cd/m^2 compared to 0.38cd/m^2) than those used in this experiment. Saccadic reaction times are faster for higher luminance stimuli (Doma & Hallett, 1988; Wheeless et al., 1967); which implies that higher luminance signals are transferred and processed more rapidly. Therefore the larger errors seen in this experiment may be a result of increased neural delays due to a lower luminance target.

This experiment did not find that mislocalisations were any greater for targets presented in the hemifield which the eye is travelling towards. An effect of hemifield has been reported for smooth pursuit (Brenner et al., 2001; Königs & Bremmer, 2010), although it has not been previously found during OKN (Kaminiarz et al., 2007). It is not clear why this experiment has not found an effect of hemifield for the smooth pursuit condition, when other experiments have (Brenner et al., 2001). One possibility is that the hemifield effect only occurs when pursuit is not interspersed with resetting saccades. It is already clear from the data collected here that the resetting saccades influence the mislocalisations during smooth

pursuit. Therefore it is possible that the hemifield effect only emerges from simple, uninterrupted pursuit, which is what previous studies showing the hemifield effect employed.

Although this experiment ascertained that there appears to be a common perceptual mislocalisation during OKN and pursuit, it is still unknown as to whether targeting saccades executed during OKN or pursuit follow the same pattern of mislocalisation. This research question formed the basis for the subsequent experiment.

3.4 Experiment B3 – Comparison of Perceived Location and Saccadic Accuracy during Optokinetic Nystagmus and Smooth Pursuit

Some authors have claimed that motor actions are not affected by the perceptual mislocalisations (Goodale & Westwood, 2004; Haffenden, Schiff, & Goodale, 2001; Hallett & Lightstone, 1976; Hansen, 1979; Hansen & Skavenski, 1985; Króliczak, Heard, Goodale, & Gregory, 2006; Schwartz, Moran, & Reina, 2004). This would suggest that motor actions are controlled by different sources of information to perceptual judgements. However, other researchers have found the opposite to be true, motor actions and perceptual judgements are similarly affected by concomitant eye movements (Dassonville et al., 1992; Honda, 1989; Rotman et al., 2004b; Schlag & Schlag-Rey, 2002). This experiment investigated whether the behaviour of saccades executed during concomitant OKN or smooth pursuit was dissociable from the perceptual mislocalisations found in Experiment B2. To achieve this, the paradigm used in Experiment A2 (Section 2.3) was run in parallel with the paradigm in Experiment B2. This allowed direct comparison of saccadic accuracy and perceptual judgements within participants.

3.4.1 *Participants*

This experiment was conducted using seven participants. Four of the participants had previously participated in Experiment B2, and three participants had not participated in any

previous experiments. Ages ranged from 23 to 27 years of age, and three participants were female. The number of participants was fixed before the study commenced, and is consistent with previous literature (see Section 3.3.1) and previous experiments, whilst allowing for additional naïve participants in order to ensure no practice effects were observable.

3.4.2 *Stimuli*

The saccadic accuracy conditions were exactly the same as the stare-OKN and full-field pursuit conditions described in Experiment A2. Perceptual judgement conditions were as described in Experiment B2; however some changes were made to the staircase design, these changes are outlined below.

3.4.3 *Staircase Design*

This experiment used eight staircases. Four staircases which began with the test stimulus presented in the direction of stimulus motion, and four where the test began in the opposite direction to stimulus motion. In each of the four staircases the target was triggered at a different time following on-line fast-phase detection. For the OKN condition the times were 110, 160, 210 or 260ms following fast-phase detection; for pursuit the times were 140, 260, 380 or 500ms. If the test was presented ahead of the target (relative to stimulus motion), then it began from 10° away from the target; if the test was presented behind the target (relative to stimulus motion) then it began at 1° from the target. The initial step size for the staircase was 4°, which was halved after each reversal. All other characteristics of the staircase were the same as described in Experiment B2 (Section 3.3.3).

3.4.4 *Procedure*

There were four conditions in this experiment, saccadic accuracy in either OKN or smooth pursuit, and perceived locations in either OKN or smooth pursuit. The order in which participants completed the conditions was randomised. The procedure for saccadic accuracy

during OKN and pursuit was exactly the same as that described for the OKN and full-field pursuit conditions of Experiment A2 (Section 2.3). For the perceived location conditions the procedure was as outlined in Experiment B2 (Section 3.3.4).

3.4.5 *Results*

During the saccadic accuracy conditions, error was taken to be the mean horizontal distance between fixation and the saccade target, relative to stimulus direction (i.e. errors which are in the same direction as stimulus direction are positive). This allows direct comparison with the magnitude of perceptual mislocalisation; which is also expressed relative to stimulus motion. These data are shown in Figure 3.7.
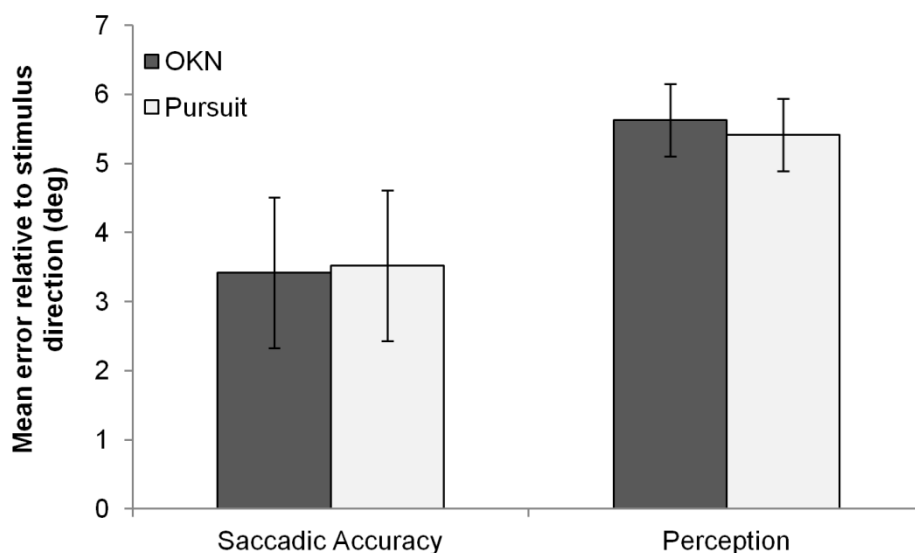
Figure 3.7: Mean saccade errors and perceptual mislocalisations during OKN and smooth pursuit. Errors are expressed relative to the direction of stimulus motion (thus positive values indicate errors in the direction of the stimulus motion). Error bars show ±1 × standard deviation with individual differences partialled out (Cousineau, 2005).

Saccadic accuracy was very similar in both OKN and smooth pursuit, this is clearly visible in Figure 3.7 and is consistent with the results of Experiments A2 and A3 (Sections 2.3.4 and 2.4.5). Furthermore the degree of perceptual mislocalisation was very similar in both OKN

and pursuit, which is in line with the findings of Experiment B2. Whether the participant was conducting OKN or pursuit did not have a significant effect upon errors ($F(1,6) = 0.01$, $p = 0.925$, $\eta_p^2 < 0.01$). Saccades appear to be more accurate than perceived locations in both OKN and pursuit; accordingly there was a significant influence of whether the response was oculomotor or perceptual ($F(1,6) = 15.97$, $p = 0.007$, $\eta_p^2 = 0.73$); there was no interaction between response type and eye movement type ($F(1,6) = 0.36$, $p = 0.568$, $\eta_p^2 = 0.06$).

The time at which the target was presented during the slow-phase influenced perceptual mislocalisations in the same way as was described in Experiment B2. Psychometric functions of pooled data showing this effect are shown in Figure 3.8.
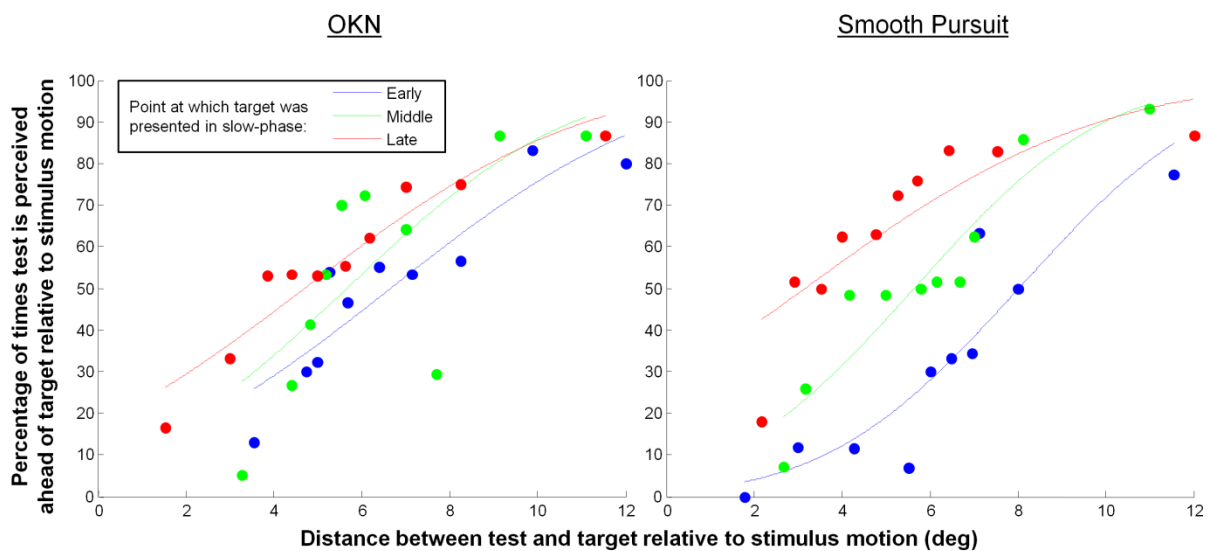
Figure 3.8: Psychometric functions showing the perceptual mislocalisations apparent when the target is presented early in the slow-phase, in the middle, or at the end of the slow-phase. Data has been pooled from all participants. Fits are from a Maximum Likelihood Model (Klein, 2001).

The same pattern of results was observed in Figure 3.8 as was observed in Experiment B2. Targets are mislocalised to the greatest extent when they appear shortly after the beginning of the slow-phase (therefore, just after a fast-phase). Mislocalisation is reduced when the target appears late in the slow-phase (thus presumably shortly before the onset of a fast-phase). The

effect of slow-phase timing was found to be significant ($F(2,12) = 45.18$, $p < 0.001$, $\eta_p^2 = 0.88$). There was no effect of whether the participant conducted OKN or smooth pursuit ($F(1,6) = 0.50$, $p = 0.507$, $\eta_p^2 = 0.08$), and there was no significant interaction ($F(2,12) = 2.81$, $p = 0.089$, $\eta_p^2 = 0.33$).

In the 'saccadic accuracy' conditions, horizontal error at fixation was plotted as a function of time during the slow-phase that the saccade target was presented. These data have been pooled across participants and are shown in Figure 3.9.
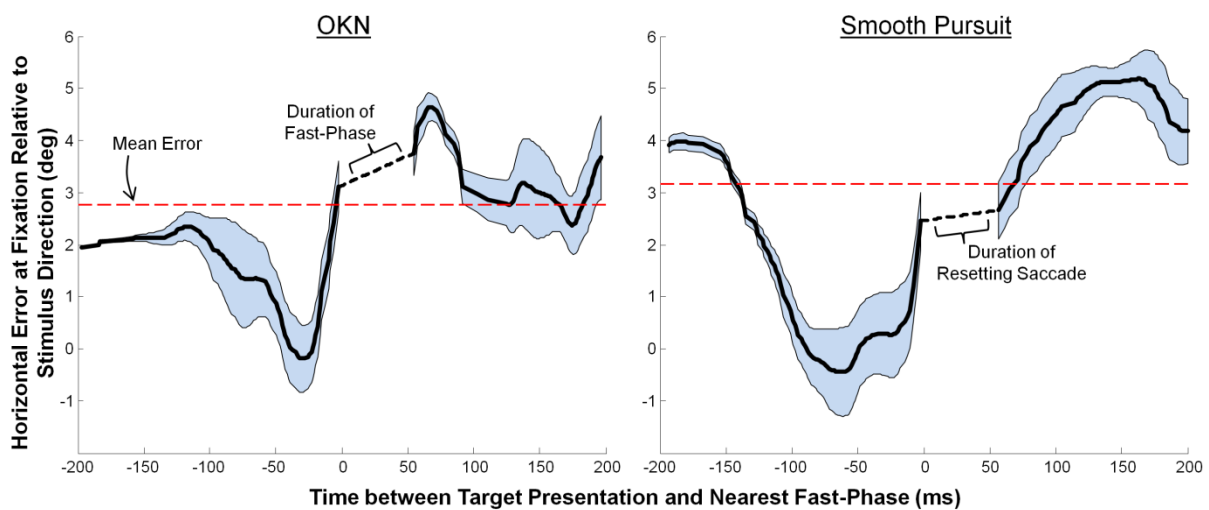
Figure 3.9: The magnitude of horizontal error at fixation is shown as a function of the time between target presentation and the closest fast-phase or resetting saccade (thus fast-phase or resetting saccade is executed at 0ms). Data has been pooled from all participants. The sleeve represents ±1 × standard error. Data taken with a bin size of 1ms and smoothed with a Gaussian filter (SD = 12Hz). The mean error is shown by dashed red line. Data was not kept if the target was presented during the fast-phase or resetting saccade, the gap in the distribution has been interpolated with a dashed line.

The pattern of errors in Figure 3.9 shows that targeting saccades become more accurate if the saccade target is presented shortly before a fast-phase or resetting saccade. There also seems to be an increase in error for targets presented shortly after the fast-phase or resetting saccade has completed.

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The effect of hemifield was also analysed in this experiment. Unlike Experiment B2, there was a significant effect of hemifield, with larger errors when the perceptual or saccade target was presented in the hemifield which the eyes were travelling towards (F(1,5) = 13.96, p = 0.013, η_p^2 = 0.74). The effect of hemifield is shown in Figure 3.10.
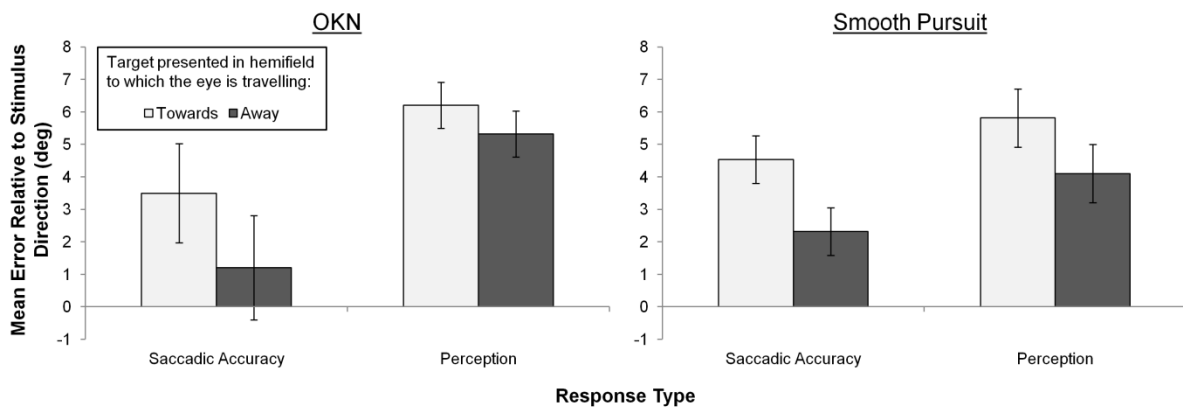
Figure 3.10: Error as a function of whether the target was presented in the retinal hemifield which the eye was travelling towards (light bars) or away from (dark bars), for both saccadic and perceptual tasks in OKN and pursuit. Error bars show ±1 × Standard Deviation with individual differences removed (Cousineau, 2005).

There was no significant interaction between the hemifield and whether the response was a saccade or perceptual judgement (F(1,5) = 5.30, p = 0.070, η_p^2 = 0.515) or between the hemifield and whether the concomitant eye movement was OKN or pursuit (F(1,5) = 0.083, p = 0.785, η_p^2 = 0.02). There was no significant three-way interaction between hemifield, response type, and eye movement type (F(1,5) = 0.66, p = 0.453, η_p^2 = 0.12). Post-hoc paired t-tests revealed that errors were significantly larger during pursuit for both perceptual judgements ($t(6)$ = 2.52, p = 0.045, r = 0.51) and saccades ($t(6)$ = 4.01, p = 0.007, r = 0.73); however there was no significant effect of hemifield during OKN for either perceptual judgements ($t(6)$ = 1.65, p = 0.150, r = 0.31) or saccades ($t(5)$ = 1.77, p = 0.137, r = 0.34). However only the effect of hemifield on saccadic accuracy remains significant when taking a Bonferroni-corrected alpha value of 0.0125.

3.4.6 *Discussion*

This experiment revealed that saccades executed during OKN or smooth pursuit show errors which are in the direction of the eye movement. This direction of error is the same as perceptual mislocalisations which occur during OKN (Kaminiarz et al., 2007; Tozzi et al., 2007) or smooth pursuit (Brenner et al., 2001; Kerzel et al., 2006). However, saccades are more accurate than perceptual judgements; fixation locations were closer to the target than the subjective experience of target location. This may tie in with those authors who claim that perceptual judgements and actions are generated using different streams of information (Hansen, 1979; Hansen & Skavenski, 1977). However, there are two reasons to question the assertion the saccades executed in this experiment were directed using information which was different from that which formed the perceptual judgement. The first reason is that saccade end-points were still biased in the direction in which the eyes were moving; thus perception and oculomotor control share the same bias. Secondly, saccadic accuracy followed the same pattern of mislocalisations seen during OKN or smooth pursuit. Targeting saccades were most accurate when the saccade target was presented shortly before fast-phase or resetting saccade, and were least accurate when the target was presented shortly after the resetting saccade or fast-phase. This pattern of saccadic accuracy mirrors that of the subjective experience of locations during OKN fast-phases (Tozzi et al., 2007) or saccades (Ross et al., 2001); and is evidence that the targeting saccade is programmed using the same information as the perceptual judgement. This pattern of saccadic accuracy also is in line with authors reporting that saccades are affected by perisaccadic mislocalisations (Dassonville et al., 1992; Honda, 1989).

Thus a common pattern of errors suggests targeting saccades are programmed using the same information as that which perceptual judgements are based upon; however saccades are more accurate than perceptual judgements would suggest. How can these two findings be

resolved? One possibility is that saccadic generation and perception are based upon the same information, but the oculomotor system has access to this information before the perceptual system does. It is assumed that the mislocalisations which are evident during pursuit arise from a delayed retinal signal being combined with an extra-retinal eye position signal specifying the future position of the eye (Brenner et al., 2001; Rotman et al., 2004a). If the retinal signal reaches oculomotor areas before it reaches perceptual areas then the mismatch between retinal signals and extra-retinal eye position signals will be reduced for oculomotor actions relative to perceptual experience. This hypothesis would be consistent with what is known about the physiology of the visuo-motor system. For example retinal information can reach the superior colliculus directly via the retinotectal pathway, which means visual information can be processed by the superior colliculus before that information reaches visual cortex via the retino-geniculo-cortical pathway (Munoz & Everling, 2004). It has been previously shown that short-wave cone distractors take longer to affect saccade initiation than do classic luminance distractors (Bompas & Sumner, 2009a). This is hypothesised to be because short-wave cone distractors are invisible to the retinotectal pathway, and therefore must exert their influence through the longer retino-geniculo-cortical pathway (Bompas & Sumner, 2009a). This study highlights the fact that the time taken for retinal stimulation to reach brain areas has a demonstrable effect upon behaviour. It is possible that saccades were more accurate in this experiment because the retinal information reached oculomotor areas such as the superior colliculus before it reached visual cortex; this shorter delay would reduce the mismatch between retinal and extra-retinal signals. This could potentially account for why targeting saccades and perception follow the same pattern of errors; but errors overall are reduced for oculomotor behaviour.

This experiment did find the effect of hemifield which has previously been reported, during pursuit errors are greatest for targets presented in the hemifield which the eyes are

travelling towards (Königs & Bremmer, 2010), but this effect does not occur for OKN (Kaminiarz et al., 2007). This result was not found in Experiment B2. It is unclear why the effect was found in this experiment, but not in the previous one. It could be simply an issue of statistical power, this experiment had seven observers, whereas only five participated in Experiment B2. However there were also some minor changes to the staircase procedure between this experiment and Experiment B2: the starting point of the test was offset in the direction of the mislocation, meaning more of the distribution where the participant perceived the test as behind the target relative to stimulus motion was able to be sampled, and eight staircases were run in parallel instead of the six which were used in Experiment B2, meaning more data was collected within each participant. Interestingly, the effect of hemifield was similar for both perception and saccadic accuracy: saccades were more inaccurate to targets presented in the hemifield the eye was travelling towards in the pursuit condition only – this is the same pattern of results as for perceptual judgements. This could be taken as further evidence that oculomotor and perceptual systems are using the same sources of information.

3.5 General Discussion of Chapter 3

The experiments presented in this Chapter aimed to address two research questions: whether the pattern of targeting saccade accuracy shown in Chapter 2 followed the same pattern as perceptual mislocalisations, and to investigate any potential differences between misperceptions during OKN and pursuit. The results of Experiment B3 suggest that saccades are processed using the same source of information as that which generates the subjective experience of where the saccade target is located. However, saccades are more accurate than perceptual judgements. This reduction in error could be due to faster transfer of retinal information to oculomotor areas, resulting in a smaller mismatch between retinal and extra-retinal signals.

How do the findings of this chapter influence the way in which the results of Chapter 2 are interpreted? Before this question can be answered one important difference between the motivations of this chapter and Chapter 2 must be addressed: the experiments of Chapter 2 were concerned with how the displacement of the eye during the saccade latency period was represented by the saccadic system; it was shown that the saccadic system is able to partially accommodate for the movement of the eye during the saccade latency period. However, the perceptual experiments outlined in this chapter do not have this same notion of displacement after target presentation; instead they are more concerned with the subjective location of the target, the concept of saccade latency is meaningless in the context of the perceptual experiments. This raises an interesting possibility, perhaps the results of Chapter 2 were not due to incomplete compensation for the saccade latency displacement, but rather the error stems from the starting point of the oculomotor system being incorrect. To elaborate, in Chapter 2 it was presumed that the eye would land upon the target's veridical location if compensation were complete, however if the retinal signal for the target was combined with a mismatched extra-retinal eye-position signal (as is assumed to occur for perceptual judgements [Rotman et al., 2004a]) then the oculomotor system would not have access to the saccade target's veridical location in the first place. If this were the case then complete compensation for the saccade latency period would appear as though it were only partial.

This possibility, although interesting, does not actually change the overall conclusions of Chapter 2. For example it was concluded that here was a mechanism which might allow saccades to be updated by OKN slow-phases or pursuit; this mechanism must still exist, indeed it may actually be far more accurate than the results of Chapter 2 suggest. If such a mechanism did not exist, and the oculomotor system did not have access to the target's veridical location (as is postulated above) then we would expect saccade landing points to show a negative compensation, and for saccades to show an error even greater than the

distance travelled during the saccade latency period. This was not found, showing that such a compensatory mechanism must exist in some form.

Chapter 2 also concluded that targeting saccades were as integrated with OKN as they were with smooth pursuit. The experiments in this Chapter found a very noticeable similarity in the results obtained during OKN or smooth pursuit, further reinforcing the view that there is considerable commonality between the neural processing underlying reflexive OKN and volitional smooth pursuit. One notable difference between smooth pursuit and OKN was that Experiment B3 replicated the effect of hemifield for smooth pursuit (Königs & Bremmer, 2010), but failed to find a significant effect of hemifield for OKN; which is consistent with the results of Kaminiarz et al. (2007). This effect of hemifield was not found for either OKN or smooth pursuit in Experiment B2; however this may have been due to slight methodological differences between experiments, or due to a lack of power in Experiment B2.

It is difficult to establish how important the hemifield effect is, because researchers are currently unsure of why it occurs in the first place. Van Beers, Wolpert and Haggard (2001) speculated that there is not one gaze signal which is used to compensate for eye-movements, but a set of gaze-signals, each compensating error in a different region of visual space. Therefore, the hemifield which the eye is travelling towards is integrated with different gaze signals to those used in the hemifield which the eye is travelling away from (Van Beers et al., 2001). Alternatively, Königs and Bremmer (2010) postulate that the tendency to overestimate eccentricities during pursuit (Kerzel et al., 2006; Rotman et al., 2004b) coupled with a shift in pursuit direction produces the observed differences between hemifields. However, the neural basis underlying the hemifield effect remains unclear (Königs & Bremmer, 2010). Nevertheless if there are differences between localization during OKN or smooth pursuit, these are subtle when compared to the overarching pattern of results; for

example the magnitude of mislocalisation during OKN and pursuit is very similar, and the pattern of errors around a resetting fast-phase is very similar to the pattern of errors around a resetting saccade. Differences between OKN and pursuit are likely to exist; they are two different eye movements which serve two different purposes. However the extent of similarities which have been found seem to imply that there is a considerable amount or shared processing in voluntary and automatic eye movements.

In summary, although there may be differences in perceived locations during OKN and smooth pursuit, on the whole they are subject to the same degree of perceptual bias. This perceptual bias appears to be related to the movement of the eyes, and not the act of viewing a moving stimulus. Saccades executed during concomitant OKN or smooth pursuit show similar patterns of errors as those seen during perceptual tasks, implying the same source of information is used to guide oculomotor control as well as perception.

The experiments outlined in this chapter, and in Chapter 2 have mainly used the comparison between OKN slow-phases and smooth pursuit as examples of reflexive and volitional eye movements. However, there is another comparison which can be made, the comparison between OKN fast-phases and saccades. Although some of the issues surrounding the relationship between OKN fast-phases and saccades have been touched upon (e.g. a common pattern of mislocalisation during fast-phases and resetting saccades) the links between these two eye movements have not been examined in much detail. The relationship between fast-phases and saccades will be further examined in the next two chapters, starting with Chapter 4, which sought to establish whether OKN fast-phases can cause curvature in top-down targeting saccades.

Chapter 4: Saccade Curvature due to the Activity of OKN Fast-Phases

4.1 Introduction

The research presented so far has mainly focussed upon the slow-phase of OKN, by investigating the ability of top-down saccades to compensate for slow-phase displacements, and examining the perceptual effects which occur during OKN slow-phases. Furthermore, there have been explicit comparisons with voluntary smooth pursuit eye movements throughout. In this chapter the focus will shift to the fast-phase of OKN; the rapid jump which repositions the eye in between tracking slow-phases. Instead of a comparison between slow-phases and smooth pursuit, this experiment compared fast-phases and saccades. However the aim remains to investigate the interactions between voluntary eye movements and automatic ones.

Research outlining the relationship between fast-phases and saccades has been discussed in detail previously (Section 1.4.4). However, to summarise, saccades and fast-phases are thought to share the same brainstem execution machinery (Bense et al., 2006; Curthoys, 2002; Lueck & Kennard, 1990). Evidence for this comes from the observation that saccades and fast-phases have very similar main sequences (Garbutt et al., 2001; Guitton & Mandl, 1980; Kaminiarz et al., 2009a; Ron et al., 1972) and very similar latency distributions (Carpenter, 1993, 1994; Roos et al., 2008). However, automatic stare-OKN fast-phase generation is not thought to involve processing in brain areas higher than the brainstem, such as the superior colliculus, or saccade-related cortical regions (Collewijn, 1975; Kashou et al., 2010; Konen et al., 2005; Schraa-Tam et al., 2009). Indeed some models of the fast-phase system (Anastasio, 1997) are explicitly models of the saccadic system (e.g. Scudder, 1988) with top-down input via the superior colliculus removed. However, while the end characteristics of saccades and fast-phases (i.e. the main sequence and latency distributions)

appear very similar, whether generation of fast-phases is subject to the same pre-processing as saccades has, as far as I am aware, never been investigated.

In order to investigate the possible interaction between fast-phases and saccades at a 'higher' level than the brainstem execution machinery, an experiment using saccade curvature was conducted. Saccade curvature is a readily observed, simple behavioural phenomenon. Although saccades naturally curve by a certain amount, which is usually attributed to imperfect co-ordination between pairs of ocular muscles (Smit & Gisbergen, 1990; P. Viviani, Berthoz, & Tracey, 1977), saccade curvature has also formed a strong basis from which to investigate a diverse range of behaviours. For example saccade curvature is thought to index inhibitory and excitatory processes in saccade target selection (Hermens, Sumner, & Walker, 2010; Walker, McSorley, & Haggard, 2006), is thought to reflect the online updating of saccades to changing targets (Findlay & Gilchrist, 2003; Findlay & Harris, 1984) and is even thought to measure attributes such as attention and visuo-spatial working memory (Theeuwes, Olivers, & Chizk, 2005; Van der Stigchel, 2010; Van der Stigchel, Meeter, & Theeuwes, 2006).

Saccade curvature can also result from competition from, and inhibition of, oculomotor plans. It has been demonstrated that the appearance of a distractor stimulus will cause saccade trajectories to curve away from the distractor location (McSorley, Haggard, & Walker, 2004, 2005). This is thought to occur because distractor-induced activation on an oculomotor map is inhibited below baseline activity, which biases saccade direction away from the distractor's location. Under certain specific conditions it has also been found that saccades can curve towards distractor locations; this tends to occur if the saccade is made very rapidly (a latency of around 200ms) and if the saccade target's location is unpredictable (McSorley, Haggard, & Walker, 2006, 2009; Walker et al., 2006). Curvature towards a distractor has been hypothesised to arise because inhibition of distractor-induced activity

takes some time, therefore short-latency saccades are executed at a time when distractor-induced activity is above baseline, causing a trajectory bias toward the distractor location (Van der Stigchel, 2010; Walker & McSorley, 2008). Thus curvature toward a distractor's location only occurs when competition on an oculomotor map has been unresolved.

It is possible that similar unresolved competition between oculomotor plans is the cause of saccade curvature due to the parallel planning of two different saccade trajectories (McPeek & Keller, 2000, 2001, 2002). The parallel planning of two saccades would give rise to two competing sources of activity on an oculomotor map that would be in close temporal proximity, and therefore may be subject to unresolved inhibition (Walker & McSorley, 2006). This could result in competition between the two saccade end-points and produce a saccade that is curved, such that the trajectory of a saccade executed due to one locus of activity deviates towards the other site of activity (McPeek, Han, & Keller, 2003; McPeek & Keller, 2000). These competing sites of activity have been hypothesised to reside in the intermediate layers of the superior colliculus (McSorley et al., 2004; Walker & McSorley, 2008; Walker et al., 2006), as shown in Figure 4.1.
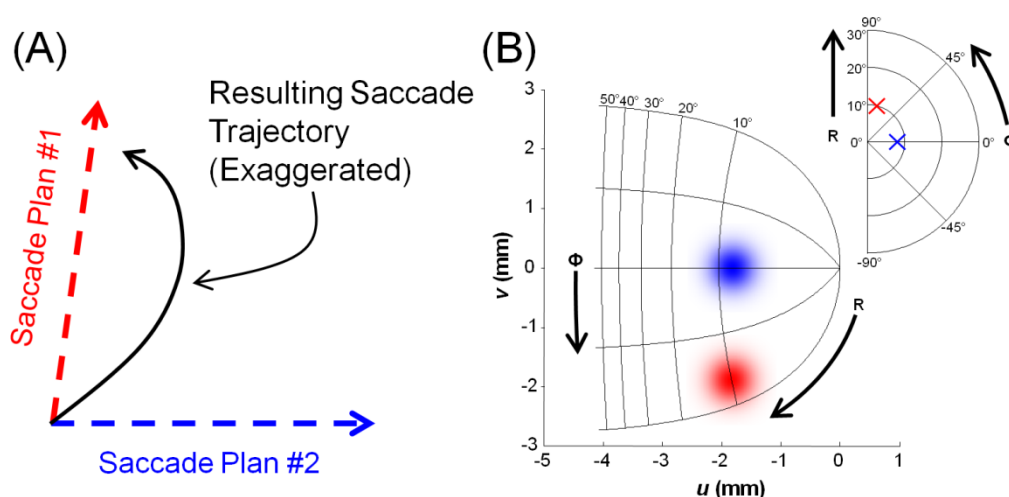
Figure 4.1: *A*, **The parallel planning of an oblique and a horizontal saccade can result in the execution of a single, curved saccade; which deviates toward the competing end**

point if completion between end-points is unresolved. This is thought to occur due to competing sites of activity in the superior colliculus, which biases the initial saccade direction (shown in panel *B*). Sites of activity in the superior colliculus are illustrated for the horizontal saccade of 8°, and the oblique saccade of 10° (insert shows the oblique saccade [red] and horizontal saccade [blue] endpoints in retinal co-ordinates; formulas to convert retinal co-ordinates to SC locations taken from Marino et al., 2008).

Activity in two different locations on the superior colliculus (assuming competition between these two locations is unresolved) would cause the initial saccade direction to be aimed at an intermediate point between the two locations, in a way analogous to the global effect (McSorley et al., 2004; Walton, Sparks, & Gandhi, 2005). However, the superior colliculus only codes for the end-points of saccades, it cannot alter the saccade trajectory to produce a curved saccade. The curvature itself has been attributed to corrective feedback from structures downstream of the superior colliculus such as the cerebellum (McSorley et al., 2004; Quaia, Lefèvre, & Optican, 1999) and the brainstem (Walton et al., 2005), which cause the saccade to curve back towards the desired end-point.

If fast-phase activity is dealt with in the oculomotor system in the same way as saccades, then the parallel planning of a fast-phase and a saccade might cause saccade curvature in the fast-phase direction. Curvature towards the fast-phase direction was predicted because the parallel planning of two saccades tends to cause curvature towards the second site of activity, rather than away from it (McPeek & Keller, 2001) and our saccade target would appear at an unpredictable location, which increases the chance of unresolved competition (Walker et al., 2006). Therefore it was hypothesised that goal-directed vertical saccades executed during horizontal OKN should be curved in the direction of the fast-phase end-point, especially when the saccade is initiated around the time of fast-phase generation (see Figure 4.2B). To test this hypothesis, observers were required to make vertical saccades during horizontal OKN.

4.2 Methods

4.2.1 *Participants*

Eight observers participated this experiment, five of whom were female and age range was 22-28 years. All reported normal vision. Six observers had previously participated in eye tracking experiments. All participants gave informed consent, and all procedures were vetted by the Ethics Committee for the School of Psychology, Cardiff University. The low-level effect of saccade curvature should not differ between subjects. Indeed, previous studies which have used oculomotor competition to elicit curvature show large effect sizes (N = 53 reported effects, mean r = 0.80, SD = 0.15) (Doyle & Walker, 2002; Hermens et al., 2010; McSorley et al., 2004, 2009; Nummenmaa & Hietanen, 2006; Theeuwes et al., 2005; Van der Stigchel, Meeter, & Theeuwes, 2007; Van der Stigchel & Theeuwes, 2005, 2006; van Zoest, Van der Stigchel, & Barton, 2008; Walker et al., 2006; White et al., 2011). Sample size calculations (Soper, 2014) show that eight participants will give a power value of 80% with an effect size of 0.80 (alpha = 0.05), therefore eight participants were tested, and this value was fixed before the experiment began.

4.2.2 *Stimuli and Procedure*

OKN was elicited using the same band of dots as described in Section 2.2.3. Participants viewed the band of OKN-eliciting dots until a bar appeared above or below the dots, to which the participants were instructed to make a targeting-saccade (see Figure 4.2A). The targeting-saccade stimulus was triggered after 11, 12 or 13 detected fast-phases, plus a variable delay of 110-300ms. On 25% of trials (randomly determined) the band of dots did not move to allow baseline measures of targeting saccades without concomitant OKN. Target onset in baseline trials could not be yoked to an OKN waveform, therefore onset time was calculated

as if fast-phases had occurred three times a second (Cheng & Outerbridge, 1974; Freeman & Sumnall, 2005; Kolarik, Margrain, & Freeman, 2010).

The target stimulus was a horizontal line stretching across the width of the screen and positioned $\pm 10°$ from centre of the screen, line height = $0.3°$, brightness = $1.24cd/m^2$. It was displayed for 50ms. The band of OKN-eliciting dots remained on the screen for 14ms following the target stimulus, meaning the targeting saccade was conducted in the dark. It has been shown that OKN will continue for around a second following extinguishing of all stimuli (Gellman & Fletcher, 1992; Leigh & Zee, 1999). A period of 1000ms was therefore allowed for the targeting saccade, followed by an inter-trial interval of 300ms. The experiment was split into 10 blocks of 40 trials each, and no more than five blocks were completed in a single day.
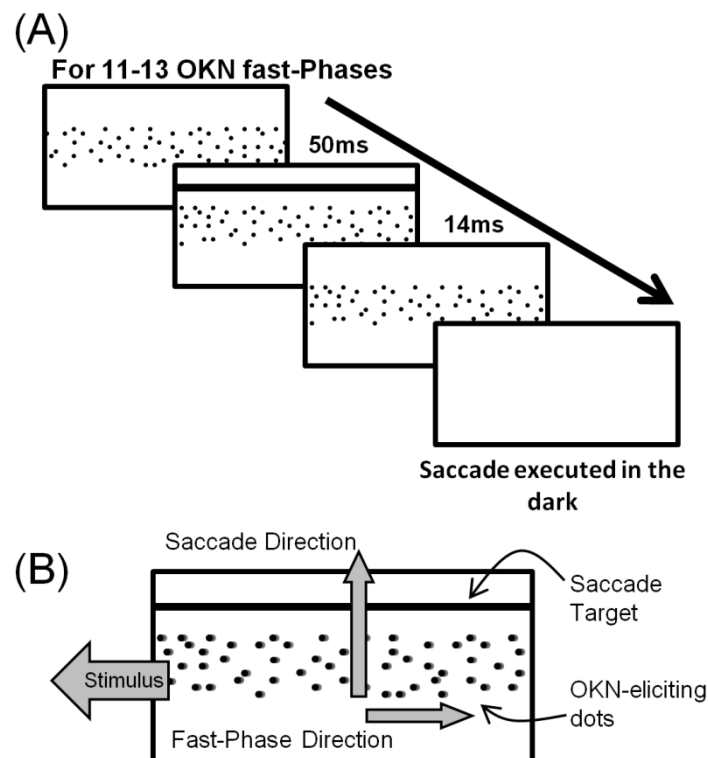
Figure 4.2: Illustration of method for eliciting curvature from an OKN fast-phase. *A*, **OKN was induced by a horizontally moving band of random dots. A saccade target line was presented above or below these dots following 11-13 fast phases. The participant was simply required to lift or lower their gaze to the vertical location of the line.** *B*, **At**

the time of the saccade it is hypothesised there could be two motor commands programmed in parallel, the targeting saccade (vertical component) and an OKN fast-phase (horizontal component).

4.2.3 *Data Analysis*

All eye traces were analysed using the procedures outline in Section 2.2.5. To express the magnitude of saccade curvature, the amplitude and direction of all saccade trajectories were first normalised. A second-order polynomial was then fitted to each saccade trajectory, and the coefficient of the quadratic term was taken to directly represent the magnitude of curvature (following Ludwig & Gilchrist, 2002).

4.3 Results

During OKN, it was found that 54.6% of the targeting saccades had curvature in the competition-predicted direction (i.e. saccades that curved in the direction of the fast-phase). However, given that saccades are rarely exactly straight (P. Viviani et al., 1977), Figure 4.3 plots the amplitude of curvature in OKN compared to the baseline condition for both group and individual data. All participants showed larger curvature when deviations were in the competition-predicted direction, compared to baseline or deviations that were not in the competition-predicted direction (see Figure 4.3A). Seven of the eight participants also showed an increase in the magnitude of curvature from baseline for those deviations that were not competition-predicted (Figure 4.3B).

Saccades that curved in the direction predicted by competition from OKN fast-phases showed significantly greater deviation than those that did not curve in the competition-predicted direction ($t(7) = -4.28$, $p = 0.004$, $r = 0.85$); they were also significantly more curved than the mean unsigned curvature found in the baseline (no OKN) condition ($t(7) = -6.73$, $p < 0.001$, $r = 0.93$). Hence, the greatest amount of curvature found was in the direction

predicted by an interaction between the fast-phase and saccade planning. The increase from baseline for those saccades that curved against the competition-predicted direction (i.e. those predicted by online correction) was also significant ($t(7) = -3.03$, $p = 0.019$, $r = 0.75$); which may reflect some degree of on-line correction of the saccade towards a point on the target line.
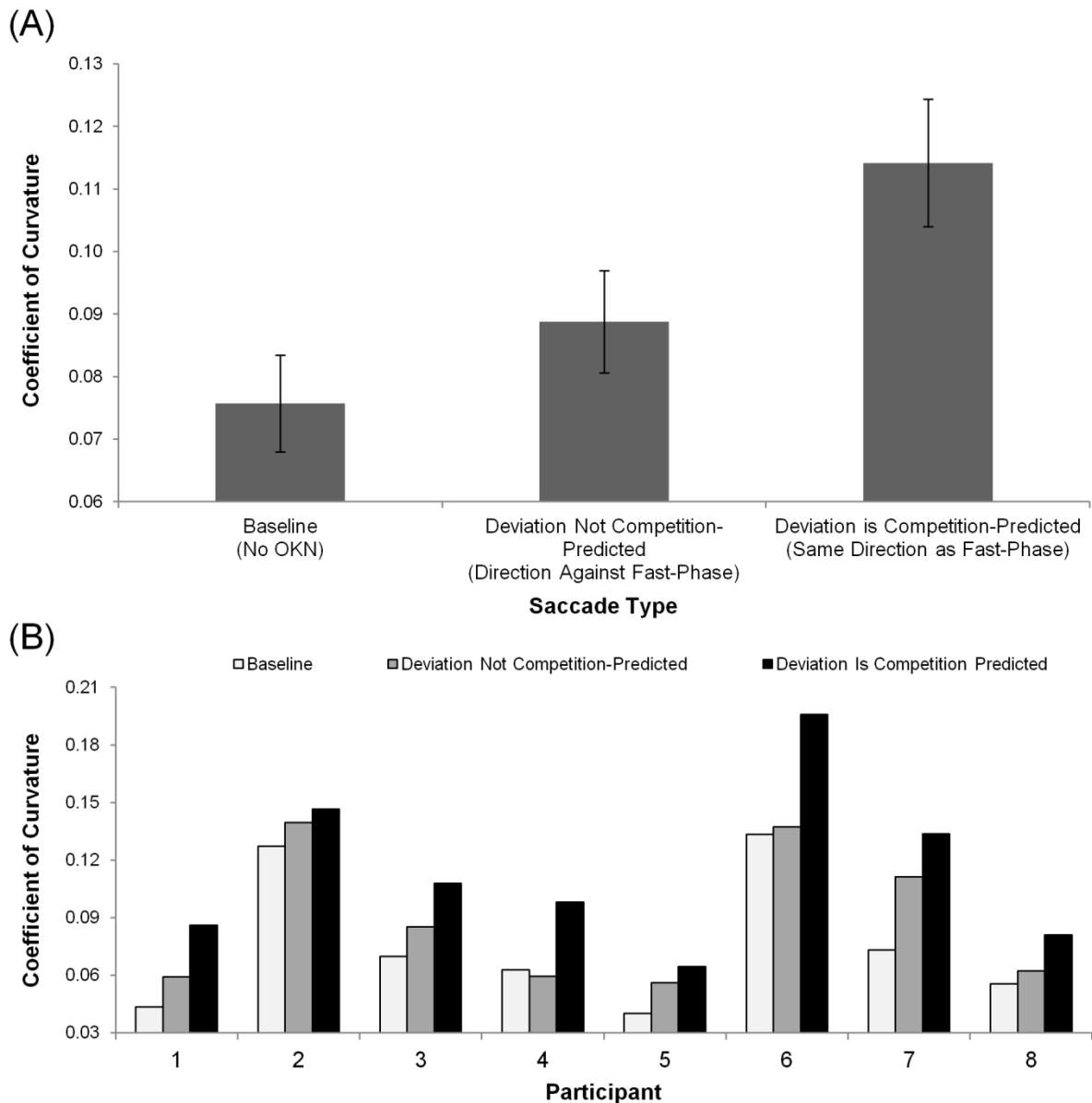
Figure 4.3: *A,* **Competition-predicted saccades that deviate in the direction of the OKN fast-phase (rightmost bar) had significantly larger trajectory deviations than those deviating against the fast-phase, which were not competition-predicted (middle bar); or baseline saccades that are initiated from fixation (leftmost bar). Non-competition-predicted saccades that do not curve in the direction of the fast-phase (middle) show a**

smaller, yet significant increase in trajectory deviations than baseline. Left and rightward curvature is combined for each condition. Error bars show ± 1 x standard deviation with variance attributed to individual differences partialled out in line with Cousineau's (2005) method. ***B*****, Individual data reveals that all participants show the largest deviations for those saccades that curve in the competition-predicted direction (black bars).**

Beyond the simple comparison of curvature in the competition-predicted direction and against it, it was predicted that there would be an association of curvature with timing. Figure 4.4A illustrates the activity in build-up neurons associated with saccades (red) and hypothesised activity for fast phases (blue) during four cycles of OKN. The profiles are based on actual cell recordings from Munoz and Wurtz (1995), their Figure 2, with no adjustment to their temporal dynamics except to remove the initial visual burst seen for target-evoked saccades (the sharp rise in the red profiles) from the putative fast-phase activity; mutual inhibition has not been modelled. If fast phases are programmed like saccades, activity for them would rise to threshold in the saccade network repeatedly at a rate of about 3Hz. Meanwhile, activity for the vertical saccade would rise in response to the onset of each target stimulus (marked by the grey vertical line in Figure 4.4A).

The illustrated activity associated with the first targeting saccade in Figure 4.4A comes about half way between two fast phases and so is least likely to be affected by them. The second saccade comes just after a fast phase; thus its planning overlaps considerably with that of a fast phase, and would incur greater interaction with the fast phase activity. One would also expect a saccade that came just before a fast-phase to be influenced by it, but since the fast phase then does not occur due to cessation of optokinetic stimulation, and OKN is not regular enough to predict exactly when it would have occurred, we have to rely on saccades that follow fast phases to test the hypothesis that saccades initiated near the time of

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fast phases are most likely to interact with fast-phase planning and thus to curve in the 'competition-predicted' direction.
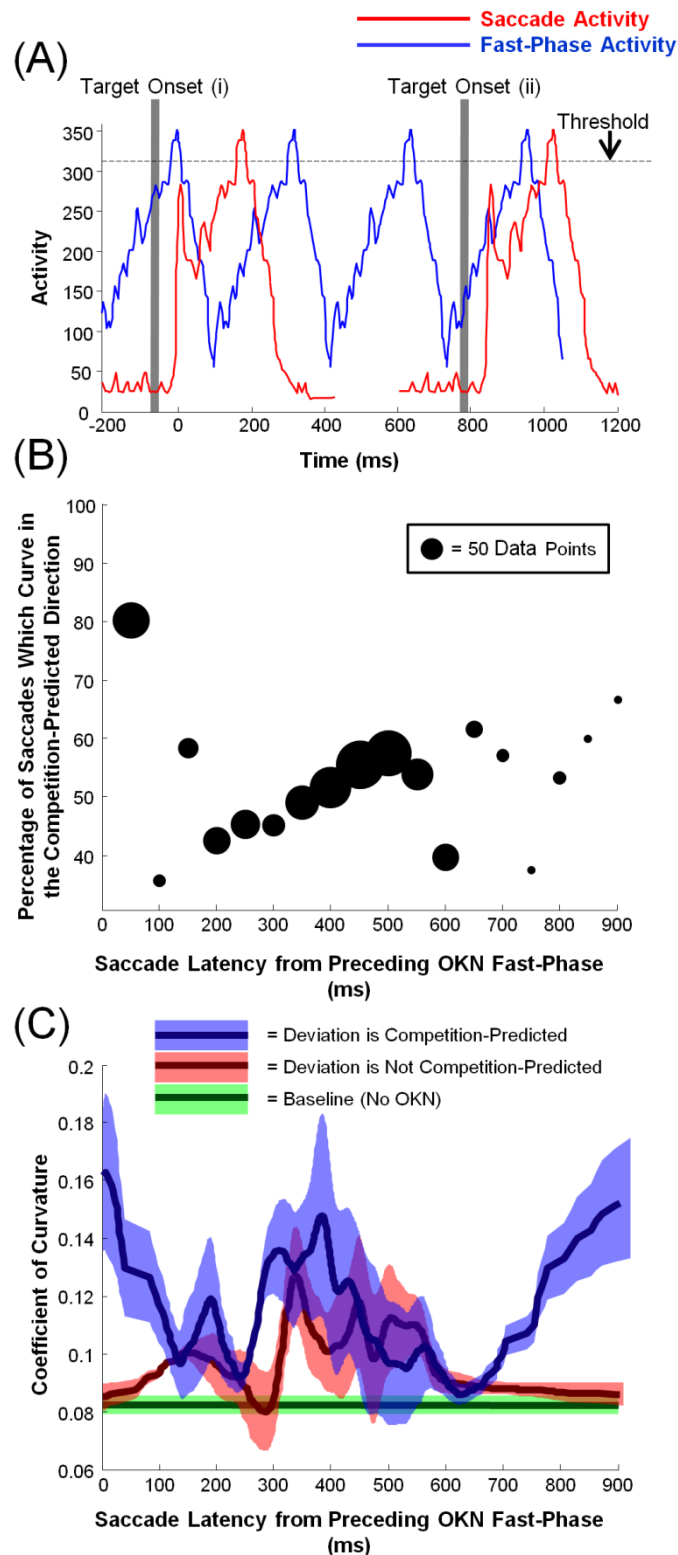
Figure 4.4: *A*, Hypothetical rise to threshold for build-up neurones in SC for both OKN fast-phases (blue line) and targeting saccades (red line) (build-up activity constructed from actual neurophysiological SC recordings by Munoz & Wurtz, 1995). Target onset (i) results in a targeting saccade activity that rises when there is relatively little fast-phase-related activity; however target onset (ii) results in targeting saccade activity that rises when there is concurrent fast-phase activity. *B*, shows that for targeting saccades

made in temporal proximity to a fast-phase, the majority deviate in the direction of the fast-phase. There is a possibility that the proportion of saccades deviating in the direction of fast-phases rises once again for longer delays where an imminent fast phase is likely. There is also a possibility that there is a temporary reversal, where most saccades curve in the direction opposite to fast-phases at around 200-300ms. *C*, shows the magnitude of saccade curvature as a function of time since the last fast-phase for deviation in the competition predicted direction (in fast-phase direction - blue), in the non-competition predicted direction (red), and baseline (no OKN - green). Data has been pooled from all participants. Competition-predicted saccades are largest when executed shortly after a fast-phase, and they are also largest a long time after a fast-phase. Note the baseline value is slightly different to that shown in Figure 4.3; this is due to the mean being taken from pooled data, rather than a mean of individual scores. The sleeve denotes ±1 × Standard Error.

Consistent with the prediction, Figure 4.4B shows that the majority of targeting saccades made shortly after an OKN fast-phase (analogous to target-onset (ii) in Figure 4.4A) deviated in the direction of the fast-phase (competition-predicted direction). This effect then decayed away to a point where targeting saccades were equally likely to deviate in the competition-predicted direction or not. This would be analogous to a saccade to target onset (i) in Figure 4.4A. This accounts for the fact that overall only a small majority (54.6%) of targeting saccades deviated in the competition-predicted direction. There is a trend showing that the proportion curving in the competition-predicted direction rises again for longer latencies – i.e. when one might expect that the next fast phase is imminent. This is also consistent with the predicted saccade-fast phase interactions, but since the fast-phase does not actually occur it is harder to be as sure that this rise is due to fast-phase activity. Furthermore the amount of data for the longest delays is inevitably small (represented by the area of the circles in Figure 4.4B) which makes testing the prediction that curvature is associated with an imminent fast-phase difficult.

Figure 4.4C suggests that saccades that curve in the direction of the fast-phase are largest when they are executed shortly after a fast-phase. In a similar pattern to Figure 4.4B, it appears as though saccades made a long time after a fast-phase are also large, however as

mentioned previously small quantities of data at these long delays make drawing conclusions difficult. For saccades that deviate in the direction non-predicted by competition (i.e. against fast-phase direction), they do not become obviously larger than baseline until a few hundred milliseconds after the fast-phases have been completed.

4.4 Discussion

This experiment found that saccades made during OKN showed significantly greater curvature than that which was shown when saccades were made without concomitant OKN. The effect also showed a systematic relationship with the OKN waveform, with those saccades that deviated in a direction predicted by competition between fast-phases and saccades showing a significantly larger amount of curvature. This implies that the curvature observed in this experiment is due to the summation of two eye-movement plans: a vertical component to bring the eye to the target's location, plus a horizontal component elicited by activity in the optokinetic system to make a fast-phase against the stimulus motion. It is expected that this effect is similar, if not identical to curvature observed during the parallel processing of two saccades to different locations (e.g. McPeek & Keller, 2000, 2001, 2002). Saccade curvature due to the fast-phases of OKN provides evidence that OKN fast-phases can indeed act like competitive saccades. Thus the generation of saccades cannot be independent from the activity of fast-phases, for fast-phases to have a demonstrable effect on the behaviour of saccades they must share common neural networks.

Furthermore, this implies that the common neural network serving saccades and OKN fast-phases is not restricted to brainstem execution machinery, saccade curvature is thought to occur from interactions higher up in the saccadic system. Although some researchers have found evidence that saccade curvature can arise from interactions in cortical areas (White et al., 2011), there is a large amount of evidence suggesting that the initial deviation found in

curved saccades arises from activity within the SC, and it seems most parsimonious to assume the effects noted here are mediated by the superior colliculus as it is anatomically closest to areas currently known to be involved in fast-phase generation. During curved saccades, two sites of activity have been observed in the SC (McPeek & Keller, 2002; Port & Wurtz, 2003) and stimulation of the SC elicits saccade curvature towards the stimulated site (McPeek et al., 2003; McPeek & Keller, 2000). Conversely, inactivation of areas of SC using muscimol causes deviations away from the inactivated area (Aizawa & Wurtz, 1998). It is important to note that competing SC activity is thought to bias the initial direction of the saccade, but it cannot account for the saccade curving back towards the target location; this may arise from corrective mechanisms in the brainstem or cerebellum (McSorley et al., 2004; Quaia et al., 1999; Walton et al., 2005).

It was observed that the majority of targeting saccades executed shortly after an OKN fast-phase were curved in the direction of that fast-phase, and that after 100ms this effect decayed away to the point at which saccades curved roughly equally in and against the direction of OKN fast-phases. It was also found that saccades that deviated in the competition-predicted direction were largest when they occurred immediately after a fast-phase (and perhaps for those saccades that are executed a long time after a fast-phase, i.e. when the next fast-phase is imminent). This is in line with what is known about the time course of SC activity and curvature: for two sites of activity in the SC to elicit saccade curvature towards the competing site of activity they must occur in close temporal proximity (Noto & Gnadt, 2009). This is particularly true for curvature that is in the direction of a competing saccade end-point because the saccade must be executed before inhibitory mechanisms suppress the competing site of activity (McSorley et al., 2005).

Furthermore it is possible that a competing source of activity can be inhibited below baseline, and cause curvature away from that location (McSorley et al., 2004), which here

would induce curvature in the non-competition predicted direction. There is a hint that the majority of saccades do curve in the non-competition predicted direction a few hundred milliseconds after fast-phases have been completed, and deviations in the non-competition predicted direction appear to become larger than baseline only after some time has elapsed following fast-phase completion (see Figure 4.4). However, the time at which deviations in the non-competition predicted direction are above baseline is also accompanied by deviations in the competition-predicted direction being above baseline. This would not be expected if inhibition of the fast-phase was underway, however interpretation of these time-courses is very difficult due to the stochastic nature of OKN – we do not know when a future fast-phase is being planned and therefore inhibited. Moreover, it is possible that deviations in the non-competition predicted direction could be due to on-line corrections rather than inhibition. Nevertheless, if these effects are due to inhibition of the fast-phase, then this would also be good evidence that activity due to OKN fast-phases is able to interfere with saccade execution.

This experiment shows that fast-phase generation can influence the activity in higher-level saccadic areas, which may include the SC. However, if the saccadic system is as integrated with the fast-phase system as this experiment suggests, then the connections should work both ways. Thus it should be possible for the activity in higher-level areas to influence the fast-phases themselves. If a bidirectional, functional connection can be established, then this would lend stronger support to the notion that saccades and OKN fast-phases share more than just low-level brainstem execution machinery. In order to investigate whether activity in areas traditionally considered 'saccadic' (especially the SC) could influence OKN fast-phases, an experiment was conducted which attempted to find the saccadic inhibition effect in the fast-phases of OKN. This experiment formed the basis for the next chapter.

Chapter 5: The Saccadic Inhibition Effect in OKN Fast-Phases

5.1 Introduction

In the previous chapter, evidence was presented that suggested the fast-phases of OKN can act like competitive saccades. It was postulated that activity from OKN fast-phases is represented in higher level saccadic areas, and the superior colliculus was suggested as a possible location for competitive interaction between fast-phases and saccades that caused initial deviations in saccade trajectories. If OKN fast-phases are functionally connected to higher level areas previously only associated with saccade planning, then this would be at odds with currently established ideas about the neural basis of OKN fast-phases (Anastasio, 1997).

The superior colliculus (SC) is not usually included when considering the neural substrate of OKN fast-phases (Anastasio, 1997; Chun & Robinson, 1978; Curthoys, 2002; Curthoys et al., 1984; Curthoys et al., 1981; Hess et al., 1989; Kitama et al., 1995; Precht & Strata, 1980). There is good reason to discount the SC from the optokinetic pathway, for example lesions to the SC have been reported not to affect OKN (Albano & Wurtz, 1982; Collewijn, 1975; Pierrot-Deseilligny, Rosa, et al., 1991; Precht & Strata, 1980; Schiller, True, & Conway, 1980) and maturation of the SC during early development do not seem to be accompanied by any changes to optokinetic responses (Distler & Hoffmann, 1992). However, just because the SC is not necessary for fast-phase generation, this does not mean it is not functionally involved during OKN fast-phases. For example, whist the frontal eye fields and posterior parietal cortex are strongly linked to higher-level saccadic processing, they are not necessary for saccades themselves to be executed (Lynch, 1992; Lynch & McLaren, 1989).

There is evidence which suggests the SC may be functionally involved during the fast-phases of OKN. For example activity has been recorded in the SC during OKN fast-

phases (Schiller & Stryker, 1972) and stimulation of the SC can induce nystagmus-like movements (Bergmann, Costin, Gutman, & Chaimovitz, 1964; Straschill & Rieger, 1973). Furthermore, there are substantial connections between the SC and areas in the reticular formation known to be crucial for the generation of fast-phases (Cohen, Matsuo, Fradin, & Raphan, 1985; Grantyn & Grantyn, 1976; Hikosaka & Kawakami, 1977; Kitama et al., 1995). Interestingly there are also substantial connections between the SC and the nucleus of the optic tract, an area strongly implicated in the generation of the slow-phases of OKN (Büttner-Ennever, Cohen, Horn, & Reisine, 1996; Cardozo, Mize, & van der Want, 1994; Holstege & Collewijn, 1982).

If the SC has a functional connection with the generation of fast-phases, then activity within the SC should be able to modulate fast-phase behaviour. This experiment used the saccadic inhibition paradigm in order to investigate whether SC activity can influence the fast-phases of OKN. Saccadic inhibition was first noted in the patterns of fixations during reading (Blanchard, McConkie, Zola, & Wolverton, 1984; McConkie, Underwood, Zola, & Wolverton, 1985); it was found that around 100ms following a display change there were virtually no saccades made, creating a dip in the saccadic latency distribution when time-locked to the onset of the visual transient. Although originally considered to be due to disruption of reading processes, it has since been shown that the dip in the latency distribution is a lower-level oculomotor effect present in a wide variety of saccadic tasks (Buonocore & McIntosh, 2008; Edelman & Xu, 2009; Reingold & Stampe, 1999, 2000, 2002, 2003). Irrelevant distractor stimuli that appear during the saccade planning period cause a precisely time-locked population of saccades to be inhibited, this leaves a characteristic dip in latency distribution when time-locked to distractor onset, as shown in Figure 5.1 (Buonocore & McIntosh, 2008, 2012, 2013; Edelman & Xu, 2009; Reingold &

Stampe, 1999, 2002, 2004). Typically, the dip onset is approximately 70ms, reaching a maximum at approximately 100ms (Bompas & Sumner, 2011; Reingold & Stampe, 2002).
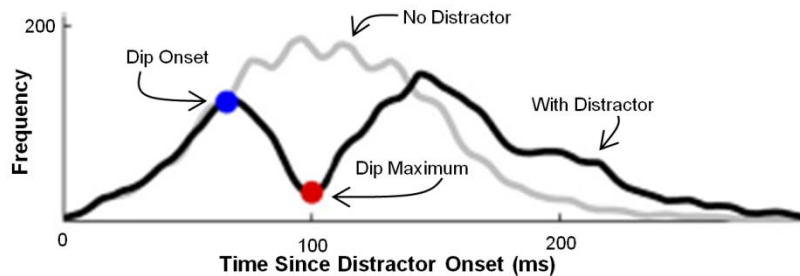
Figure 5.1: The saccadic inhibition effect in a single observer reproduced from Bompas and Sumner (2011). A distractor stimulus is briefly flashed before a saccade is executed to a suddenly appearing target. Plotting the time between distractor flash and saccade initiation reveals a characteristic dip in the distribution (black line). This dip reveals that distractors delay saccades that would otherwise have occurred around 70-150ms later. Distributions were taken with a bin size of 4ms, and smoothed using a Gaussian kernel with a 5ms window and 1ms SD.

Saccadic inhibition is thought to arise because irrelevant distractor stimuli automatically drive activity in saccade-processing areas such as the superior colliculus, which then delays the rise to threshold of saccade-related build-up activity through mutual inhibition (Reingold & Stampe, 2002). This has the effect of reducing the number of saccades occurring shortly after the distractor, causing the dip in the distribution of saccade latencies. Note that the dip begins for saccades that would have occurred 70ms after the distractor; saccades with shorter latencies escape the distractor's influence.

The site of mutual inhibition which gives rise to saccadic inhibition is thought to be the SC (Edelman & Xu, 2009; Reingold & Stampe, 2000, 2002; S.-N. Yang, 2009). Much of the evidence for the effect having a collicular locus come from the observation that the onset of the dip in latency distributions is highly consistent with the sum of conduction and response times to the SC (around 60-90ms, Reingold & Stampe, 2000, 2002). Certainly dip

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onsets are far too rapid to be mediated via a visual cortex pathway (Reingold & Stampe, 2000, 2002) and EEG has shown that cortical changes following distractor onsets occur after the saccadic inhibition dips have passed (Graupner, Pannasch, & Velichkovsky, 2011). Moreover, it has been shown that distractor stimuli can elicit activity in the SC, and sub-threshold micro-stimulation of the SC affects saccades in the same way that distractor stimuli do (Dorris, Olivier, & Munoz, 2007). Furthermore saccadic inhibition is an emergent property of neutrally-inspired SC models (Bompas & Sumner, 2011; Engbert, 2012).

Further evidence for a role of the SC in mediating the saccadic inhibition effect comes from investigations of express saccades, which are saccades which exhibit very short latencies (around 100ms) and are thought to be an optomotor reflex for orienting to peripheral targets (Fischer & Weber, 1993; Fischer et al., 1993). The mechanism thought to underlie the saccadic inhibition effect is believed to be one and the same as that which causes express saccades, i.e. the automatic activation of oculomotor areas by visual stimuli. The onset of saccadic inhibition is very similar to express saccade latencies (Bompas & Sumner, 2011; Reingold & Stampe, 2000, 2002). There is strong evidence that express saccades are mediated by the SC (see Fischer & Weber, 1993), for example lesions to the SC abolish all express saccades (Schiller, Sandell, & Maunsell, 1987). Therefore if express saccades and saccadic inhibition are two sides of the same process, this is good evidence that the site of activity underlying saccadic inhibition is indeed the SC.

A final point of evidence for saccadic inhibition being mediated by the SC is that the onset of distractor dips is lawfully modulated by contrast and chromaticity of distractors in a way which is highly consistent with known changes in conduction times of signals to the SC (Bompas & Sumner, 2011; Buonocore & McIntosh, 2012). A more dramatic change to the distractor stimulus is to render it non-visual and use an auditory distractor. An effect, albeit a smaller one, has been observed for auditory distractors (Pannasch, Dornhoefer, Unema, &

Velichkovsky, 2001; although this was not found by Reingold & Stampe, 2004); it is known that a number of cells in the superior colliculus respond to auditory stimuli (Jay & Sparks, 1984; Pannasch et al., 2001).

Therefore, this experiment sought to investigate whether distractor induced activity can influence the behaviour of fast-phases of OKN. If this is found to be the case, then it would be greater support for an integrated saccade-fast-phase network, with a likely site of interaction being the superior colliculus.

5.2 Method

5.2.1 *Participants*

Eight observers participated this experiment, all reporting normal vision. Four were female and age range was 23-28 years. Six participants had previous experience with eyetracking experiments. All participants gave informed consent and were fully aware of the purpose of the experiment. All procedures had been approved by the School of Psychology, Cardiff University Ethics Committee.

Reingold and Stampe (2004) have previously noted that between participant-variability in saccadic inhibition is very low; indeed they showed that it did not matter whether one used individual or pooled data analysing the dips in the latency distribution. Convergently, the effect sizes in previous studies using saccadic inhibition have been very high (N = 64 reported effects, mean r = 0.82, SD = 0.11) (Buonocore & McIntosh, 2012, 2013; Reingold & Stampe, 2000, 2002, 2003, 2004; Stampe & Reingold, 2002). Similarly to the last chapter, sample size calculations (Soper, 2014) show that eight participants will give a power value of 80% with an effect size of 0.82 (alpha = 0.05), therefore eight participants were tested, and this value was fixed before the experiment began.

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5.2.2 *Stimuli and procedure*

The effect of distractor stimuli was investigated for two eye-movement conditions: standard targeting saccades (Figure 5.2A) or OKN fast-phases (Figure 5.2B). Each condition was conducted on a separate day, with four participants conducting the standard targeting saccade condition first.
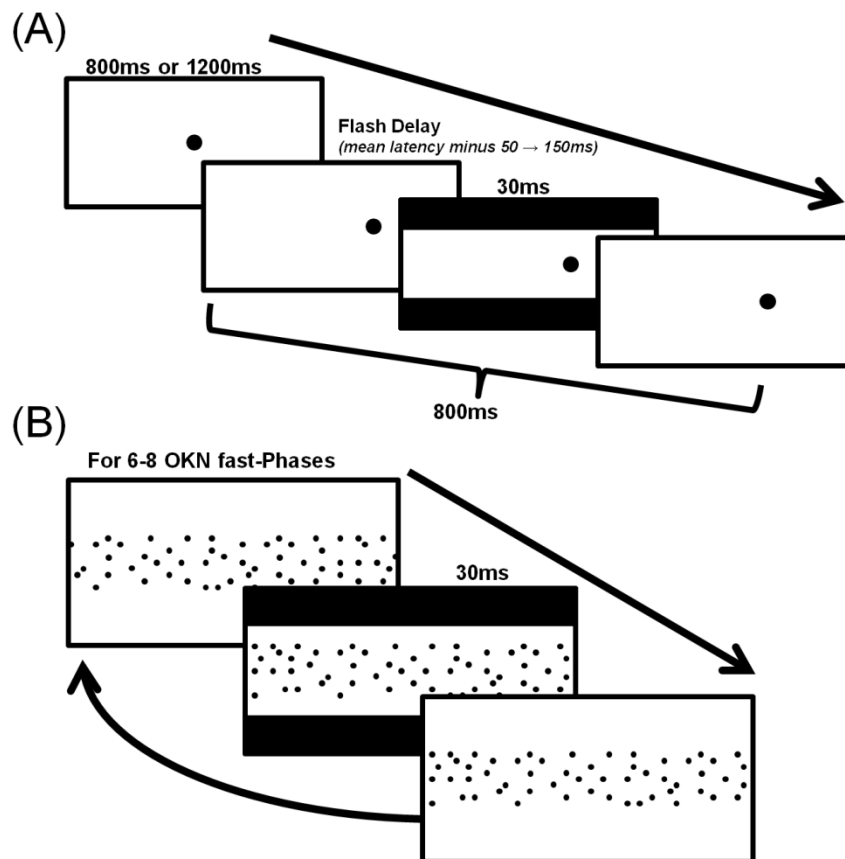
Figure 5.2: Illustrated procedure for testing 'saccadic inhibition' for standard targeting saccades (A) and OKN fast-phases (B). *A*, Participants made a saccade when the dot stepped left or right, while on 50% of trials irrelevant bars (black in illustration) flashed before the saccade was made (Reingold & Stampe, 2002). *B*, OKN was induced by passively viewed random dots moving left or right while irrelevant bars flashed intermittently in order to assess their effect on OKN fast-phases.

The methods used for the standard targeting saccade condition closely resembled those of Reingold and Stampe (2002). A central fixation point (radius = 0.3°, brightness = 1.24cd/m^2) was displayed in the centre of the screen for 800 or 1200ms, whereupon it stepped 8° either

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to the left or the right (see Figure 5.2A). On 50% of trials a distractor stimulus was presented for 30ms, consisting of two bars ($1.24cd/m^2$) that filled the screen from $\pm18°$ vertically outwards. The bars were more peripheral than those used by Reingold and Stampe (2002) in order to accommodate the requirements of the OKN stimuli described below (in a separate experiment the influence of eccentricity was investigated by repeating the saccade condition with bars placed at $\pm7°$). At the start of each block, 50 baseline trials were run without distractors so that the mean saccadic latency of each observer could be measured. Distractors in subsequent trials were then triggered 50-150ms prior to this value (which was updated throughout the experiment using the 50 preceding no-distractor trials), thus ensuring that the expected dip would fall within the distribution of saccades. Observers completed two blocks, each consisting of 50 baseline trials followed by 400 trials, of which half contained a distractor stimulus. Between each block the lights were turned on and participants given the opportunity to rest.

For the OKN condition, nystagmus was elicited by presenting observers with a band (16° high, 73° wide) of coherently moving random dots (radius = 0.3°, brightness = $0.1cd/m^2$, density of 0.5 dots/deg^2, speed 32°/sec, either to the left or right). Between each trial a blank screen of brightness $0.38cd/m^2$ was displayed for five seconds to stop participants from dark-adapting during the experiment, which might have allowed them to perceive the external stationary features of the room, as this can disrupt OKN. Participants were instructed not to track any particular dot in the display, but at the same time to not let the band of dots become blurred. To allow other stimuli to be presented at specific points in the OKN waveform, on-line detection of fast-phases was achieved using a velocity criterion of 92°/sec.

Participants viewed the band of OKN-eliciting dots until 15 fast-phases had been detected. Distractor stimuli were then flashed for 30ms every 6-8 detected fast-phases, at a random time between 85 and 235ms following fast-phase detection (see Figure 5.2B). This

procedure continued for 30 seconds, and constituted one trial. Forty-five of these trials were conducted per block, and there were five blocks in the experiment. The OKN condition therefore produced approximately 2000 distractor stimulus onsets per participant.

5.2.3 *Data Analysis*

All eye traces were analysed according to the procedures outlined in Section 2.2.5. In order to plot and calculate the metrics of saccadic inhibition, latency distributions need to be plotted with respect to distractor onset, not target onset (Stampe & Reingold, 2002). This in turn requires a method to create comparison distributions for the no-distractor trials that have no distractor upon which to time-lock. For the saccade condition a 'phantom' distractor was placed in each no-distractor trial. The phantom distractor onset had the same timing as the previous distractor trial. In the OKN condition a similar procedure was used: phantom distractors were placed in OKN slow phases that did not have actual distractors, based on the timing of the most recent actual distractor trial relative to its preceding fast-phase. Distributions of saccade onsets and fast phase onsets were taken with a bin-size of 1 ms (this being the temporal resolution of the eye-tracker), and were smoothed using a Gaussian filter with a standard deviation of 20ms (from the analysis described in Bompas & Sumner, 2011). The distraction ratio (*(baseline –distractor distribution)/ baseline*) was calculated (as in Bompas & Sumner, 2011; Reingold & Stampe, 2004). The onset of 'saccadic inhibition' was taken when the distraction ratio first rose above 2% (following Bompas & Sumner, 2011).

5.3 Results

The results in Figure 5.3 show that irrelevant distractor stimuli cause a dip in the latency distributions of OKN fast-phases for each participant (left-hand panels). OKN fast-phases therefore display the 'saccadic inhibition' effect. Pooled data distributions (right-hand panels

of Figure 5.3) reveal that this experiment found the standard dip in saccadic conditions (top and bottom), and also that a dip was present in the OKN fast-phase condition (middle).
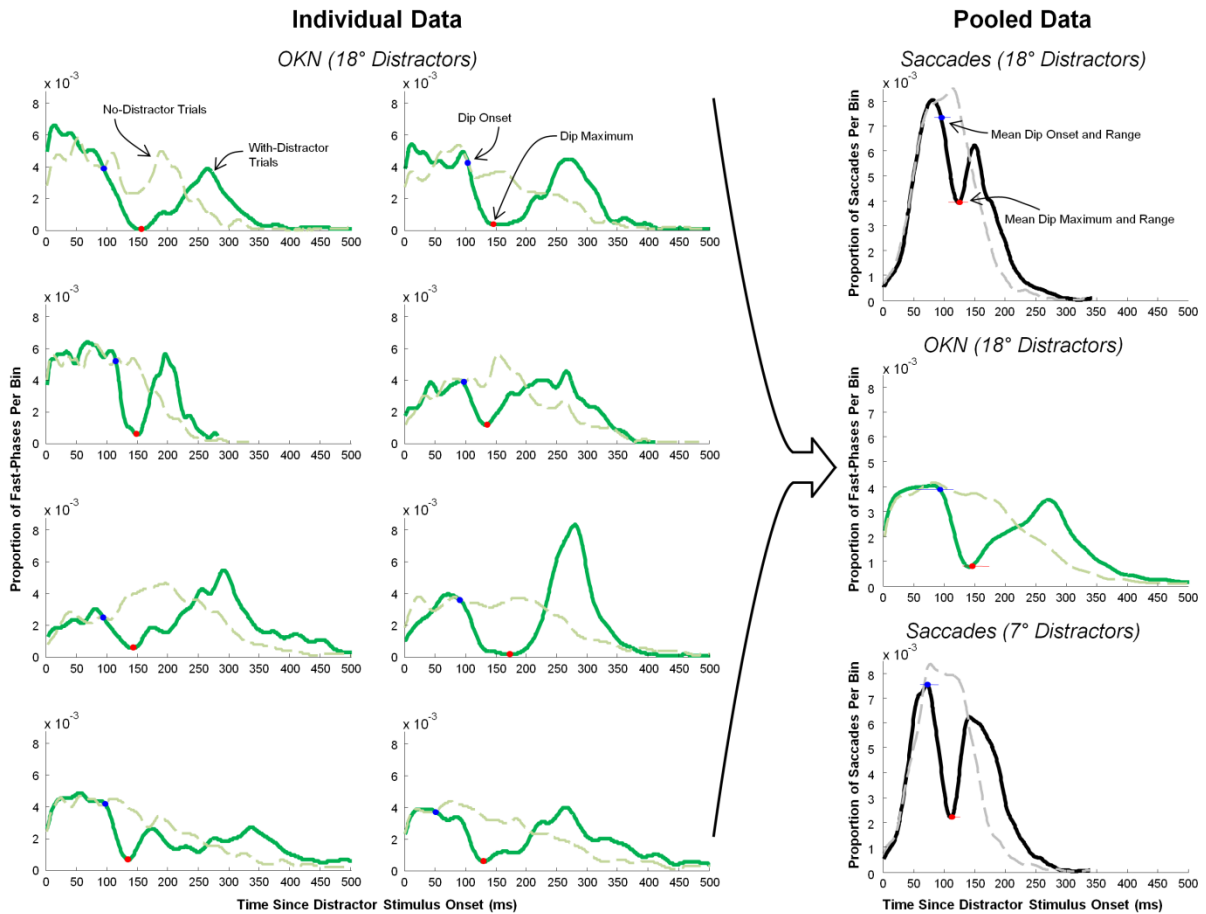
Figure 5.3: Individual data (left-hand panels) of OKN fast-phase latencies with respect to distractor onset for trials with (solid) and without (dashed) flashed distractor stimuli (see analysis section of Methods for how no-distractor distributions are created). The saccadic inhibition effect occurs for OKN fast-phases in all participants. Pooled data distributions (right-hand panels) reveal that a dip in the latency distribution occurs for OKN fast-phases (green) just as it does for targeting saccades. Blue circles represent the mean dip onset, and the horizontal blue error-bars represent the numerical range of dip onsets across participants. Red circles and error-bars represent the mean dip maximum, and the range of dip maxima respectively.

The mean onset time of the dip for the saccadic condition (18° distractors) was 95.75ms (SD = 8.89ms; see blue symbols in Figure 5.3), which is comparable to the mean onset time for the OKN condition (mean = 93.63ms, SD = 18.02ms, $t(7) = -0.28$, $p = 0.79$, $r = 0.11$). For

both OKN and saccades with 18° distractors, mean dip onsets were later than those for saccades with 7° distractors ($t(7) = 4.08$, $p = 0.005$, $r = 0.84$; $t(7) = 4.78$, $p = 0.002$, $r = 0.87$), presumably due to the more peripheral distractors (which have never been tested in previous literature). For saccades with 7° distractors the mean dip onset was 72ms (SD = 10.53ms), in line with previous literature (Bompas & Sumner, 2011; Edelman & Xu, 2009; Reingold & Stampe, 2002).

The amplitude of the dip is expressed as the percentage of saccades or fast-phases inhibited at the dip maximum point. Due to variability in the dip maximum point across participants (represented by horizontal red error bars in the pooled data distributions of Figure 5.3) individual dip amplitudes are larger than the pooled data distribution shown in Figure 5.3 would suggest. There is no indication that the inhibition effect is smaller in amplitude for OKN than for saccades with the same distractors; in fact it is larger, with the distractor inhibiting an average 87.76% (SD = 9.07%) of fast-phases at its peak, whereas only 64.93% (SD = 14.81%) of saccades were inhibited at its peak ($t(8) = 3.39$, $p = 0.012$, $r = 0.79$). Being larger, the dip peak is also later, since the peak depends on the amplitude, given that the onset time is fixed by sensory and motor delays in and out of the oculomotor system (see Bompas & Sumner, 2011).

5.4 Discussion

The presence of an irrelevant distractor causes a dip in the latencies of OKN fast-phases just as it does for saccades. The onset of this dip for both OKN and saccades is later than has been previously reported when the distractor stimuli were presented at a greater eccentricity than in prior studies. Presenting the distractor stimuli more centrally (as is done in established experiments) results in earlier dip onsets that are comparable to previously published results (Bompas & Sumner, 2011; Edelman & Xu, 2009; Reingold & Stampe, 2002). The common

effect that a distractor has on both fast-phases and saccades suggests that they are generated with shared mechanisms. This compliments the results of Chapter 4 in which it was observed that OKN fast-phases can cause curvature in top-down saccades. This saccade-like behaviour in the fast-phases of OKN implies a functional overlap in the programming of fast-phases and saccades in the cortico-collicular network where saccadic inhibition is thought to originate - not just in the brainstem execution circuitry. This would contradict the traditional idea that automatic and volitional actions are distinct and separate from each other, but support the idea that there are close interactions between volitional and automatic processes.

The behavioural effects observed in this experiment may help elucidate some of the neural pathways responsible for the fast-phases of OKN, which currently are far less well-known than those pathways that generate the slow-phase (Waddington & Harris, 2012). One possibility is that the saccade-like effects observed with fast-phases may be attributable to the superior colliculus (SC). This is because the saccadic inhibition effect has strong links to processing in the SC. The onset of the saccadic inhibition effect is highly consistent with the sum of the conduction times from stimulus onset to the SC, and from SC activity to executed saccade (around 60-90ms, Reingold & Stampe, 2000, 2002). Furthermore, sub-threshold stimulation of the SC affects saccades in the same way as distractor stimuli do (Dorris et al., 2007). Additionally, saccadic inhibition is an emergent property of SC models (Bompas & Sumner, 2011; Engbert, 2012). Furthermore, in the previous chapter it was discussed how the initial deviations found in saccade curvature are strongly associated with competing activity in the SC, thus processing in the SC during OKN fast-phases is suggested by two separate paradigms.

The SC is ideally situated to engage in fast-phase related processing, as it has substantial connections between reticular formation areas known to be involved in the generation of the fast-phase of nystagmus (Cohen et al., 1985; Grantyn & Grantyn, 1976;

Hikosaka & Kawakami, 1977; Kitama et al., 1995). I would not claim that the SC is the main site in which fast-phases are generated, as ablation of the SC has little influence on basic fast-phases elicited in standard nystagmus paradigms (Albano & Wurtz, 1982; Pierrot-Deseilligny, Rosa, et al., 1991; Schiller et al., 1980). However this does not preclude the SC from having a strong functional involvement. While brainstem burst and pause neurones are the *minimum* neural substrate required for fast-phase generation, connections to higher-level areas such as the SC may modulate fast-phases (Curthoys, 2002).

Dip onsets were no different for saccades and fast-phases with 18° distractors, implying a common mechanism underlying the effect. However dip onsets for both occurred later than has been previously reported (Bompas & Sumner, 2011; Reingold & Stampe, 2002), which may be due to the eccentricity of the distractors used in this experiment. It is already well-known that the dip onset depends upon the characteristics of the distractor stimulus; this is assumed to reflect changes in the temporal dynamics of SC processing (Buonocore & McIntosh, 2012; Pannasch et al., 2001; Reingold & Stampe, 2004; Stampe & Reingold, 2002). Accordingly, dip onsets elicited by different distractors are predicted to reflect the systematic differences in saccadic latency if those distractor stimuli are used as targets (Bompas & Sumner, 2011). Since there is a gradual increase in saccade latency as target eccentricity increases beyond 2° (Bell et al., 2000; Kalesnykas & Hallett, 1994), one would expect dip latency to increase with the eccentricity of distractors. This is what was found.

It is possible that the saccadic inhibition effect stems from more than one locus. For example sudden visual transients have been shown to affect activity in omnipause neurones as well as the SC (Boehnke & Munoz, 2008; Everling, Paré, Dorris, & Munoz, 1998; Munoz et al., 2000); as such the crucial interactions may be between SC, omnipause neurones or other brainstem circuitry. Similarly, a component of saccadic inhibition may reflect inhibitory

influences from FEF and basal ganglia that impinge upon the SC. However, the data from this experiment indicate that all loci involved in saccadic inhibition are also involved in OKN fast phases - if only a subset were relevant for OKN, one would expect the inhibition effect to be smaller. In fact it was larger than for saccades.

Nevertheless, the conclusion that the saccade network also contributes to OKN does not rely on the observed interactions stemming from the SC, it is merely postulated that the SC seems a likely locus of this interaction. Whilst it is most parsimonious to assume an SC locus, as this is closest to the structures already associated with OKN, there is no reason to rule out contribution from the rest of the network including frontal and parietal cortex. Key features of the 'SC' models accounting for saccadic inhibition also mirror properties of the FEF (Bompas & Sumner, 2011).

In summary, the results of this experiment show that activity in the saccadic network can affect the behaviour of fast-phases, and the previous chapter examining saccade curvature demonstrated that the activity in the fast-phase system can affect the behaviour of saccades. This suggests that fast-phases and saccades have more than just a superficial similarity stemming from shared peripheral motor circuitry; they are also subject to some of the same pre-processing. On the basis of these data, it appears that cortico-collicular saccade network is functionally involved in the modulation of OKN fast-phases. These findings therefore provide further evidence that automatic and volitional actions are more strongly integrated than is often thought, and builds upon work that suggests there is no sharp dichotomy between automatic, inflexible movements and voluntary, adaptive movements (McBride et al., 2012). In the next chapter the relationship between voluntary and involuntary eye movements was further investigated by examining whether the saccadic inhibition effect also extends to the fast-phases of Infantile Nystagmus Syndrome.

Chapter 6: Saccadic Inhibition in the Fast-Phases of Infantile Nystagmus Syndrome

6.1 Introduction

Previously it was demonstrated that the saccadic inhibition effect also occurs for the fast-phases of optokinetic nystagmus; implying that OKN fast-phases have a strong functional similarity to voluntary saccades and may be processed by higher level 'saccadic' areas. Subsequently it was investigated whether the same effect would also occur for the fast-phases of those with Infantile Nystagmus Syndrome (INS). The purpose of this experiment was two-fold: (1) to ascertain more certainly whether the fast-phases of INS are saccadic; and (2) to gather evidence of whether the saccadic system in those with INS functions in the same way as it does in normal observers. There is currently strong evidence that the fast-phases of INS are basically the same movements as saccades. For example both fast-phases and saccades have the same main sequence (Abadi & Worfolk, 1989), the same peak intersaccadic interval (Bosone et al., 1990), and the same characteristics of dynamic overshoots (Abadi et al., 2000). Furthermore voluntary saccade latency (Wang & Dell'Osso, 2007) and accuracy (Worfolk & Abadi, 1991) seem to be related to the concurrent activity of fast-phases.

However, one clear difference between voluntary saccades and the fast-phases of nystagmus is in the consciously willed nature of these two movements. INS is characterised as an involuntary movement of the eye, and individuals with INS are not aware of the fast-phases which they make (Harris & Berry, 2006a). Therefore, the low-level automatic nature of fast-phases means that they might not be modified by external stimuli in the same way as voluntary saccades.

If it is established that fast-phases of infantile nystagmus do show the saccadic inhibition effect, then not only will this provide evidence that fast-phases are saccadic in

nature, but will also imply that the saccadic system in those with INS is subject to the same processing as it is in normal observers. This would be further evidence that the saccadic system in those with INS is normal, which would be evidence against the theories presented in Section 1.7.3 that INS is caused by deficits in the saccadic system (Akman et al., 2005; Akman et al., 2006; Broomhead et al., 2000). Furthermore it would compliment theories which state that those with INS have a basically normal oculomotor system (Harris, 2011).

6.2 Methods

6.2.1 *Participants*

This experiment used twelve observers, five of whom were female. Ages ranged from 19 to 83 years, with an average age of 47.5 years. Eleven participants were diagnosed with INS, and one showed manifest latent nystagmus. Of the eleven participants with INS, five had a pseudo-pendular type, and the remainder showed jerk nystagmus. No participants presented with pure pendular nystagmus. Table 6.1 summarises the participant information.

Participant	Gender	Age	Waveform	Pathology
DB	M	53	Jerk	Idiopathic
GS	M	28	Jerk	Idiopathic
GT	M	59	PP	Idiopathic
JC	M	69	Jerk	Idiopathic
JC2	F	54	Jerk	Idiopathic
JS	M	55	Jerk	Idiopathic
JT	M	24	PP	Idiopathic
KL	F	60	LN	Idiopathic
LF	F	19	Jerk	Idiopathic
NB	M	44	PP	Idiopathic
RC	F	22	PP	Possible Albinism
RW	F	83	PP	Possible Albinism

Table 6.1: Participant details for INS distractor experiment. PP = Pseudo-pendular, LN = Latent Nystagmus.

The number of participants was not fixed before the experiment began, rather we tested as many participants as we were able to recruit before a certain date, therefore cessation of testing was not due to any facet of the data collected.

6.2.2 *Stimuli and Procedure*

Unlike previous experiments, participants were not calibrated. This was because knowledge of absolute eye position is not necessary, the onset of the fast-phase can be determined using the relative change in eye-position. Although it is possible to calibrate an eye which is constantly moving, this requires specialist, custom-made calibration algorithms. Designing an experiment which requires no calibration means this paradigm can easily be adopted by others should they attempt modification or replication of this work.

During this experiment participants were asked to maintain fixation upon a single target. This consisted of a green dot (radius $0.5°$, brightness $1.24cd/m^2$). Some individuals with INS do not find it comfortable to maintain gaze straight ahead (Abadi & Dickinson, 1986), therefore before the experiment began target location was adjusted so that the participant could comfortably maintain gaze upon the target with their head in the correct position in the eyetracker.

The experiment consisted of forty trials, each of which lasted for thirty seconds. During this time the participant maintained gaze upon the target while the same distractor bars used in Chapter 5 (presented from $\pm10°$, brightness $1.24cd/m^2$) were flashed intermittently. This means that the distractor bars were presented more centrally than the OKN condition in Chapter 5; therefore, based upon the results of Chapter 5, one would expect dip onset to be earlier for INS fast-phases then for OKN fast-phases. In Chapter 5 the distractor stimuli were presented based upon the timing of the fast-phase. This was not possible in this experiment as on-line detection of fast-phases using a velocity criterion would

require prior calibration. Therefore, a simpler method was adopted whereby the distractor stimuli were flashed at intervals of 750 to 1250ms. Figure 6.1 outlines the stimuli used in this experiment.
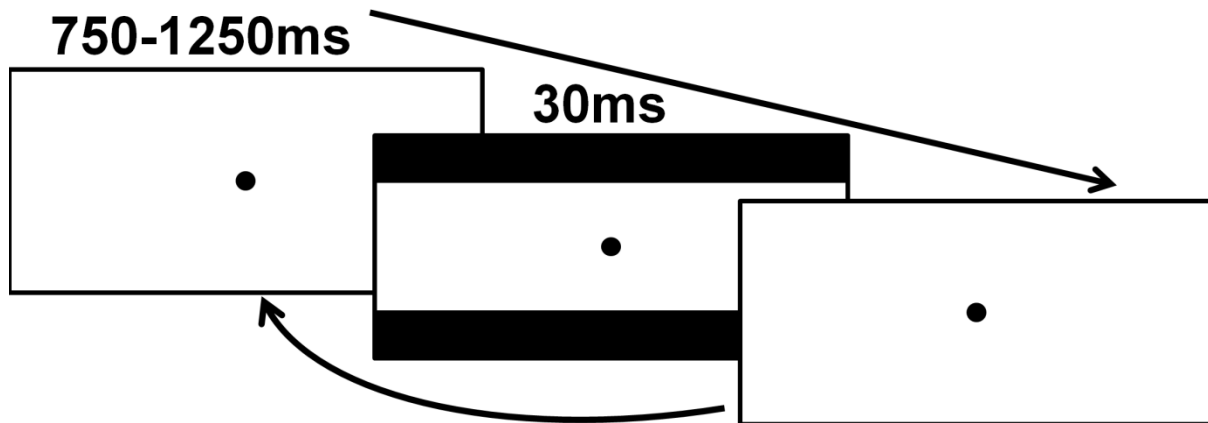
Figure 6.1: Stimulus outline for INS distractor experiment. Gaze is maintained upon a single fixation target while distractor stimuli flash every 750-1250ms. Distractor bars are presented at ±10°, which is more central than used during the OKN experiment.

At the end of each trial a blank screen was presented and the participant given the opportunity to rest. The participant initiated the next trial with a mouse click.

6.2.3 *Data analysis*

As mentioned above (section 6.2.2) no calibration was performed for this experiment. Therefore eye position is expressed by arbitrary units rather than degrees of visual angle. Fast-phases were detected using a velocity criterion which was manually adjusted until automatic fast-phase detection corresponded to those detected by visual inspection of the waveform. The onset of the fast-phase was determined when velocity fist rose above a particular value, also set by correspondence to the visual inspection of waveforms. The accuracy of this fast-phase detection was visually inspected for every distractor stimulus onset. This then allowed measurement of the latency between each distractor flash and the subsequent fast-phase.

In order to compare the distribution of fast-phase latencies with respect to a distractor, it is necessary to create a distribution of fast-phase latencies in a 'no-distractor' condition, just as in Chapter 5. The method adopted in this experiment was to create an array of random time points throughout the dataset upon which to measure fast-phase latency. Random time points were chosen every 750 to 1250ms (the same timing as the distractors) and the next fast-phase following each random time point was used in the distribution for the 'no-distractor' condition. This procedure was then repeated 100 times (with different random time points each time) to create a very large dataset for use as a no-distractor condition. Fast-phases which did actually follow a distractor flash were then removed from this dataset, which left distributions of between 68,000 and 107,000 data points per participant (depending upon fast-phase frequency).

This method is different from the OKN fast-phase distractor experiment, and was chosen for two reasons. The first was that the high frequency of resetting fast-phases in some of those with INS (it is not uncommon for fast-phase frequency to be as high as 8Hz [Abadi & Bjerre, 2002]) meant that is was possible the distractor flash would have carry-over effects into the subsequent waveforms. Therefore it is not advisable to use the subsequent waveforms to create the no-distractor distribution as was done in the OKN experiment. The second reason was that this method allowed for large datasets to be created, which was beneficial as I did not get the chance to collect as much data on the INS participants as was possible with the normal observers in the OKN experiment.

Just as in the OKN distractor experiment distributions of latencies in both the distractor and no-distractor conditions were taken with a bin-size of 1ms (the temporal resolution of the eyetracker), and smoothed using a Gaussian filter of 20ms (following Bompas & Sumner, 2011). The distractor ratio (*(baseline – distractor distribution)/ baseline*) was calculated (as in Bompas & Sumner, 2011; Reingold & Stampe, 2004). The onset of the

effect was taken when the distractor ratio first rose above 2% (following Bompas & Sumner, 2011).

6.3 Results

The data shown in Figure 6.2 clearly demonstrate that all participants show evidence of a dip in their distribution of fast-phase latencies when time-locked to distractor onset. Therefore the saccadic inhibition effect extends to the fast-phases of INS as well as the fast-phases of OKN.
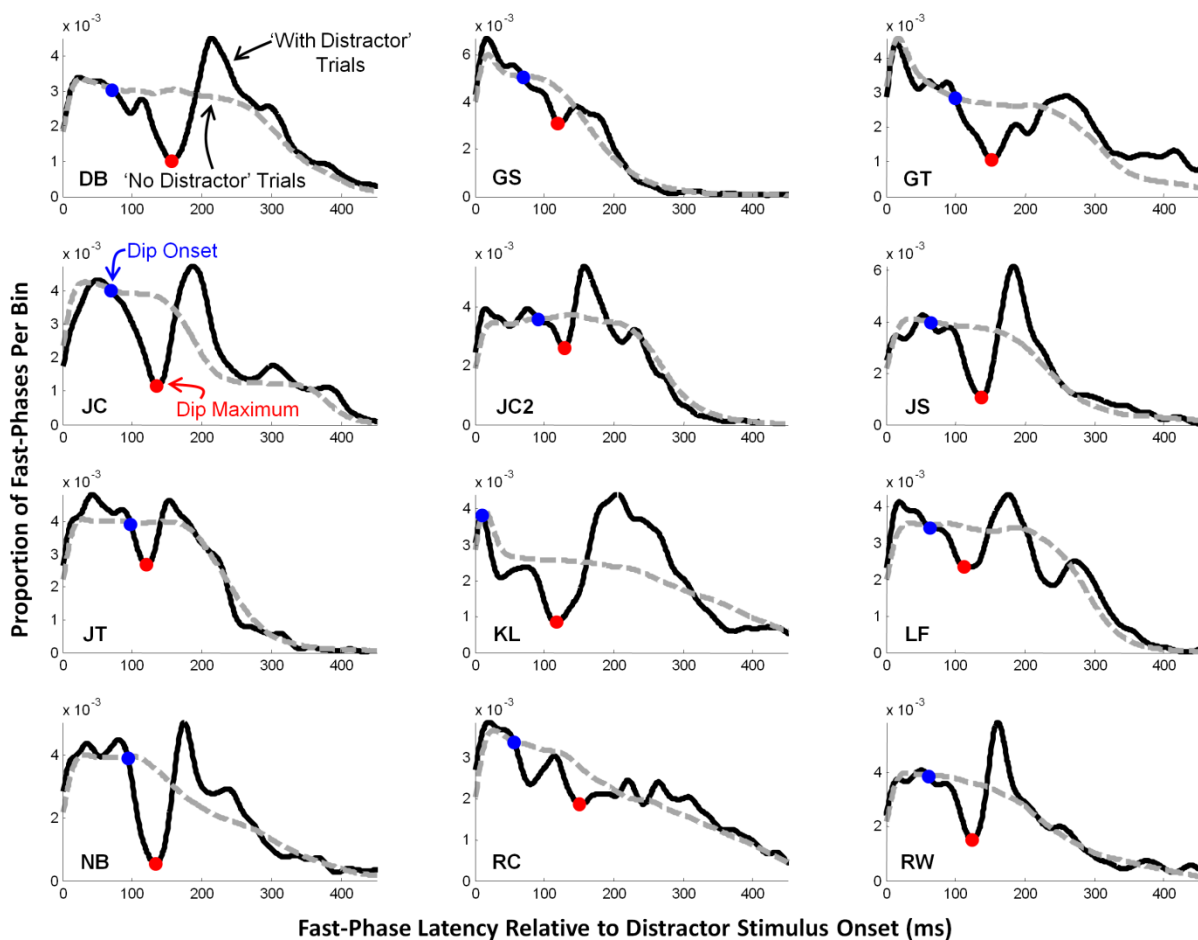
Figure 6.2: Individual data showing distributions of fast-phase latencies relative to distractor stimulus onset (solid line). Also shown are distributions relative to random time points, which form the 'no-distractor' condition (dashed line). Blue circles denote detected dip onsets; red circles denote detected dip maxima. Plots are annotated with participant label.

Although there is some individual variation between dip onset and amplitude, this does not appear to be consistent across waveform type. Mean dip onset for jerk nystagmus was 72ms (SD = 10ms), whereas it was 82.7ms (SD = 20.9ms) for pseudo-pendular nystagmus. At the dip maximum point, on average 50.4% (SD = 21.2%) of jerk fast-phases were inhibited, and 53% (SD = 21.8%) of pseudo-pendular fast-phases were inhibited. There were no significant differences in either dip onset ($t(9)$ = 1.1, p = 0.3) or dip amplitude ($t(9)$ = 0.27, p = 0.8). Participants with suspected albinism (RC and RW) and the participant with latent nystagmus (KL) also appear to have inhibition effects which appear typical to the other participants. Therefore, data was pooled across all participants for comparison with previous experiments, the pooled data distributions are shown in Figure 6.3.
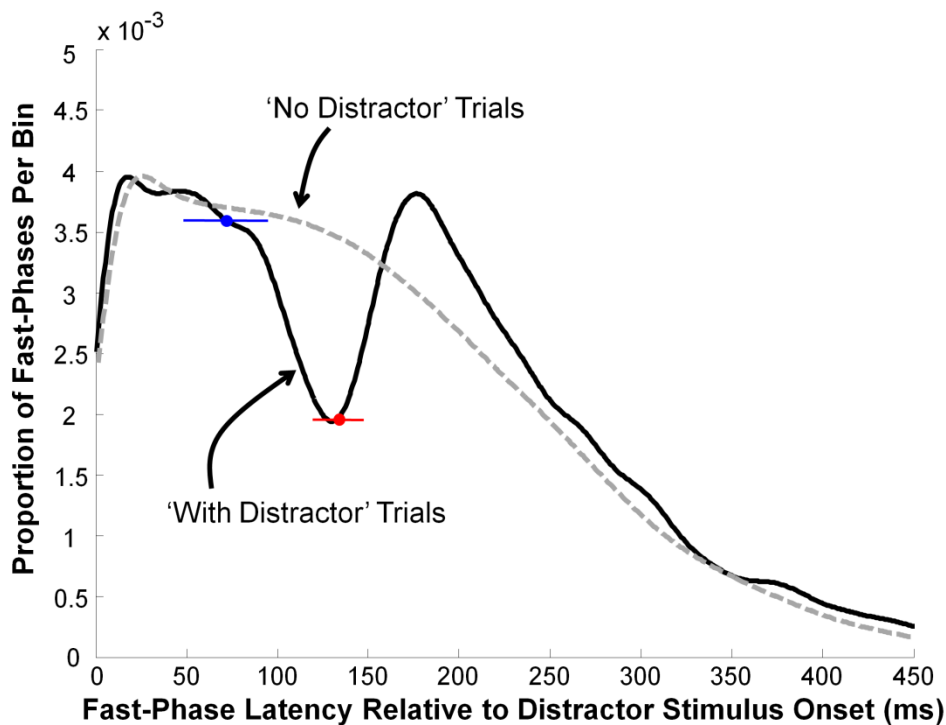
Figure 6.3: Pooled data distributions of fast-phase latency with respect to distractor onset. Blue circle shows the mean dip onset time, and the red circle shows mean dip maximum time. Blue horizontal and red horizontal lines show ± 1 × standard deviation of dip onsets and dip maxima respectively.

The mean dip onset time of 71.4ms (SD = 23.3ms) is very comparable with that which was observed in the saccade condition of Chapter 5 (mean = 73.7ms, SD = 12.48ms), and the published results of previous literature (Bompas & Sumner, 2011; Edelman & Xu, 2009; Reingold & Stampe, 2002). As expected, INS dip onset was earlier than reported for the OKN condition, which used more peripheral distractors (±18° for OKN, ±10° for INS, see Section 5.4 for discussion of the effect of eccentricity on dip onset). Dip amplitude of INS fast-phases (mean = 52%, SD = 20.4%) was lower than had found in the saccades of normal observers in Chapter 5 (mean = 71.6%, SD = 3.8%); this difference was marginally significant ($t(14) = 2.1$, $p = 0.055$).

6.4 Discussion

This experiment demonstrates that the saccadic inhibition effect occurs for the fast-phases of INS just as it occurs for the fast-phases of OKN and for voluntary saccades. This implies that the fast-phases of INS and OKN are generated by neural mechanisms which have a considerable overlap with those which generate saccades. It also implies that the interaction between sensory and motor activity in the saccadic system of those with INS is basically normal, as the onset of the saccadic inhibition effect is highly consistent with that of normal observers.

6.4.1 *Relationship between the fast-phases of INS, of OKN, and saccades*

These results compliment theories that the fast-phases of INS are fundamentally saccadic eye movements (Abadi et al., 2000; Abadi & Worfolk, 1989; Bosone et al., 1990). These results also suggest that the fast-phases of INS and the fast-phases of OKN are generated by similar mechanisms; something which has previously been postulated (Harris & Berry, 2006a), but which, to my knowledge, has not been backed up with any experimental evidence before.

These results suggest a shared mechanism between the generation of saccades and fast-phases, which supports the hypothesis that voluntary saccade end-points can be biased toward the end-point of INS fast-phases (Bedell et al., 1987; Worfolk & Abadi, 1991). This also lends support to the supposition that the reason voluntary saccade latencies are longer when target steps are around the same time as fast-phases, is because fast-phase processing disrupts voluntary saccade generation (Wang & Dell'Osso, 2007).

No obvious differences between the inhibition effect in pseudo-pendular nystagmus and jerk nystagmus were observed. Whilst it cannot be ruled out that a difference might be observed were data collected on more individuals, there is no reason to hypothesise that such a difference would occur. This is because both jerk and pseudo-pendular waveforms are considered to be manifestations of the same nystagmus phenotype (Abadi & Bjerre, 2002; Abadi & Dickinson, 1986; Harris & Berry, 2006a; Wang & Dell'Osso, 2011). However, within the pseudo-pendular waveform there is a distinction drawn between the fast-phase which occurs at the peak farthest from desired gaze location (dubbed a 'braking fast-phase', this serves to halt the runaway slow-phase an initiate a slow-phase back toward target location), and the fast-phase which occurs at target location (dubbed the 'foveating fast-phase', the alignes the fovea with desired gaze locaton) (Dell'Osso & Daroff, 1976). Unfortunately, without calibration one cannot be sure which one of the fast-phases is braking, and which is foveating; however no distinction was made during the analysis, rather the next fast-phase was taken whichever it may be. As there is no discernable difference between the effect in those with jerk nystagmus and pseudo-pendular nystagmus (see Figure 6.2) it seems that one can assume that the braking and the foveating fast-phase are both similarly affected by the distractor stimulus. This would indeed tie in with the finding that voluntary saccade latency is prolonged equally by target steps around the time of a foveating or a braking fast-phase (Wang & Dell'Osso, 2007). This would suggest that despite the different requirements

of these two fast eye-movements (Dell'Osso & Daroff, 1976) they are generated by the same neural mechanisms.

One individual with latent nystagmus was tested in this experiment, which is thought to be a different eye-movement from INS (Dell'Osso, 1982). With one observer it would not be judicious to comment on differences or similarities between INS and latent nystagmus; however it is possible to say that a clear saccadic inhibition effect was observed (Figure 6.2, participant KL). This at least allows the conclusion that the saccadic inhibition effect appears to be a ubiquitous pheonomenon in all the fast-phases of nystagmus which have been tested here.

6.4.2 *The significance for aetiological models of INS*

Whilst I did not investigate voluntary saccades per se, the fact that there was a saccadic inhibition effect in those with INS implies that sensory and motor activity interact in the saccadic system in the same way as occurs in normal observers. This would be consistent with previous claims that the saccadic system is basically normal in those with INS (Abadi et al., 2000; Bedell et al., 1987; Dell'Osso, 1973; Dell'Osso et al., 1972; Yee et al., 1976). Intact saccadic functioning would be contrary to those models which claim INS results from saccadic abnormality (Akman et al., 2005; Akman et al., 2006; Broomhead et al., 2000). Moreover, proponents of these models have recently suggested that INS may be due to an imbalance in the firing of fixation-related cells in the rostral pole of the superior colliculus (Akman et al., 2012). As the saccadic inhibition effect is strongly liked to activity interactions in the superior colliculus (Bompas & Sumner, 2011; Reingold & Stampe, 2002) then the presence of saccadic inhibition in INS fast-phases implies that the superior colliculus is functionally intact. Thus one would not expect for INS to be a result of superior colliculus malfunction.

Furthermore, these results support models which assume the oculomotor system is basically normal in those with INS (Harris, 2011; Jacobs & Dell'Osso, 2004). These findings are certainly consistent with the idea that INS is the result of an intact oculomotor system which has settled on a pattern of behaviour which was adaptive during infancy, but which is now inappropriate (Harris, 2011; Harris & Berry, 2006a). If this was the case, one would expect to find the same oculomotor effects in both those with INS and normal observers; this is indeed what was found.

6.4.3 Could there be a top-down influence over infantile nystagmus fast-phases?

I believe these results are a clear indication that the fast-phases of INS can take on externally modified behaviour in a saccade-like fashion. Therefore, despite the involuntary nature of INS fast-phases I do not envisage a fundamental distinction between saccades and fast-phases. This means that one would expect to see other saccade-like behaviours in the fast-phases of INS. Indeed, it has been reported that when visual target displacements are small, observers with INS are likely to acquire them with an ordinary fast-phase, rather than making a distinct saccade (Worfolk & Abadi, 1991; Yee et al., 1976).This implies that the fast-phases of INS can take on targeting properties, which would require some form of top-down influence to modify the end-point of the fast-phase.

A top-down effect on fast-phases is also very consistent with the observation that fast-phase frequency depends upon a conscious attempt to maintain fixation. For example, a conscious effort to fixate seems to be related to more frequent fast-phases, and periods of inattention can induce slow pendular oscillations (Abadi & Dickinson, 1986; Wang & Dell'Osso, 2011). It is not simply visual stimulation which gives rise to this effect, rather it seems related to levels of arousal or mental effort, for example fast-phase intensity (frequency $\times$ amplitude) increases when the participant performs mental arithmetic with their eyes closed (Abadi & Dickinson, 1986). Furthermore the nystagmus waveform appears to be

modulated to aid visual functioning when viewing stimuli in a low-stress situation (Wiggins, Woodhouse, Margrain, Harris, & Erichsen, 2007). This would suggest that the INS waveform is in some sense adaptive to current visual demand.

Whilst these modifications of INS appear to be related to higher cognitive functions, it is not likely that fast-phases can be consciously modified to the same extent as voluntary saccades. However, rather than assuming a sharp dichotomy between voluntary and reflexive eye-movements I instead propose that there is a graded influence of top-down goal-directed behaviour on more reflexive movements such as fast-phases of INS. Thus it might be assumed that INS fast-phases are not completely automatic and inflexible, but are able to be voluntarily influenced to a certain degree. However the degree to which INS could be subject to complete conscious control is not clear, for example it has been found that certain individuals with INS have the ability to wilfully turn off their nystagmus completely (Tusa et al., 1992).

This experiment revealed that the execution of INS fast-phases is inhibited by a distractor stimulus in the exactly the same way as voluntary saccades and the fast-phases of OKN. This suggests that there is considerable communality between these three fast eye movements, and that the fast-phases of INS are basically saccadic in nature. These results are further evidence that aetiological models of INS which emphasise a functionally intact oculomotor system (Harris, 2011; Jacobs & Dell'Osso, 2004) are more plausible that those with predict oculomotor abnormalities (Broomhead et al., 2000; Optican & Zee, 1984). Furthermore, these results tie into previous findings that there is some degree of conscious influence over the behaviour of INS fast-phases (Abadi & Dickinson, 1986; Bedell et al., 1987; Tusa et al., 1992; Wiggins et al., 2007; Worfolk & Abadi, 1991) and suggest that there is no sharp dichotomy drawn between voluntary saccades and automatic INS fast-phases.

Rather they are manifestations of the same eye-movement with a graded influence of top-down volition.

Chapter 7: General Discussion

7.1 Summary of Findings

The overarching aim of this thesis was to investigate the interactions between voluntary and automatic eye movements. In Chapter 2 it was shown that voluntary, top-down, targeting saccades were able to partially compensate for a displacement of the eye which was due to automatic stare optokinetic nystagmus. Furthermore, the targeting saccade was as accurate during stare-OKN as it was during look-OKN or smooth pursuit. Subsequently, Chapter 3 found that locations were similarly misperceived during both stare-OKN and smooth pursuit. Targeting saccades executed during OKN or pursuit also appeared to be similarly directed to the perceived location of the targets, although fixations were more accurate than perceptual judgements. Chapter 4 moved on to examining interactions between saccades and the fast-phases of stare-OKN. It was established that fast-phases can act like competitive saccades and cause curvature in top-down targeting saccades. This suggested that fast-phases are processed in areas of the oculomotor system which are usually only associated with saccade generation, and the superior colliculus was suggested as a potential example of such an area. The saccadic inhibition experiment presented in Chapter 5 gave greater support to the idea that 'higher' saccadic areas (such as the superior colliculus) are functionally involved in the processing of OKN fast-phases. Lastly, Chapter 6 revealed that the saccadic inhibition effect also occurs for the fast-phases of infantile nystagmus syndrome, potentially indicating that saccades, OKN fast-phases and INS fast-phases all share common mechanisms of generation.

7.2 Conceptualising Eye Movements as either Voluntary or Automatic (Reprise)

A common finding over the course of this thesis is that eye movements considered automatic and volitional show very similar behaviour. The slow-phases of OKN and smooth pursuit

both update saccadic motor maps to the same extent. Perceptual experience is similarly affected by OKN slow-phases and smooth pursuit. OKN fast-phases can cause saccade curvature, and are also inhibited by distractor stimuli in the same way as saccades. Furthermore the fast-phases of INS show the saccadic inhibition effect. Wherever similarity has been sought between automatic and volitional eye movements, it has been found.

Differences between automatic and volitional actions, if they have been found, have been remarkably subtle. For example, localization during pursuit depended upon whether the stimulus appeared in the hemifield which the eyes had been travelling towards, but this was not detected during OKN; as has been previously reported (Kaminiarz et al., 2007). However, such small differences do not appear to justify those authors which state that automatic and volitional processes are completely independent and served by separate neural structures (Post & Leibowitz, 1985; Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977, 1984; Whiteside et al., 1965). Therefore, the results of this thesis would compliment theories which do not draw a sharp distinction between automatic and voluntary processes, and instead view automaticity as a graded phenomenon, with gradual levels of top-down influence (McBride et al., 2012; Sumner & Husain, 2008).

Whilst the experiments presented in this thesis are not the first to suggest that there is integration between automatic and volitional actions, these experiments do make some notable advancements. For example, most studies which have examined the interface between reflexive and volitional actions have made comparisons using top-down and reflexive saccades (Godijn & Theeuwes, 2002); however drawing an unambiguous distinction between voluntary and reflexive saccades can be difficult. OKN is much more clearly considered a low-level, reflexive eye-movement, thus one can conclude more unequivocally that there is no distinct segregation between reflexive and volitional systems.

Furthermore, many previous studies have concluded that there is integration between automatic and volitional processes by demonstrating that there is a degree of automaticity in processes which are usually considered as voluntary and flexible. For example, the automatic facilitation and inhibition of actions in the masked prime paradigm (Boy & Sumner, 2010; Eimer & Schlaghecken, 2001, 2003) or the automatic capture of saccades (Theeuwes et al., 1998). However, this thesis demonstrates that the relationship also runs in the other direction; it has been shown that actions which were previously considered as automatic and inflexible can show behaviour consistent with volitional movements. For example saccade curvature and the saccadic inhibition effect revealed saccade-like behaviour in the fast-phases of OKN and INS.

Flexible modulation of automatic effects has been previously reported, for example exogenous attention to a location can enhance the priming strength of a subliminally presented prime (Marzouki, Grainger, & Theeuwes, 2007; Sumner, Tsai, Yu, & Nachev, 2006). Moreover subliminal priming can only occur if there is shared meaning between cues and targets, it has been found that arrows will not automatically prime responses to letter targets (Eimer & Schlaghecken, 1998). Furthermore the same subliminal prime can activate or inhibit actions based on its context. Wokke, van Gall, Scholte, Ridderinkhof and Lamme (2011) associated a stimulus with either a 'Go' or 'No-Go' response, and the association switched on a trial-by-trial basis. The trial-by-trial context of the stimulus also changed the way in which subliminal primes were processed, revealing that there is flexible, goal-directed control over automatic responses (Wokke et al., 2011). The results of this thesis reveal that such flexible modulation can also occur for motor actions which some consider to be entirely automatically and sub-cortically generated.

The experiments presented here can also help to distinguish between differing models of how stimulus-driven and consciously-willed eye movements interact. Some studies have

suggested that reflexive and volitional saccades are programmed by distinct populations and engage in a first-past-the-post winner-takes-all race, without directly interacting (Theeuwes et al., 1998; Theeuwes et al., 1999; Walker & McSorley, 2006). Conversely others have argued for a competitive integration model where reflexive and volitional eye-movements exist on a common motor map and inevitably influence one another (Godijn & Theeuwes, 2002). We have shown that OKN demonstrably interacts with targeting saccades. This implies reflexive and volitional eye-movements do indeed exist on a common motor map and lends support to competitive integration models.

7.3 Putative Neural Connections between Automatic and Volitional Eye Movements

If one is to claim that automatic and volitional eye movements are served by a single, integrated system, then an obvious question to ask is how this system would be implemented in the brain. Areas traditionally considered to be 'saccadic' are frontal and parietal cortices (Andersen et al., 1987; Johnston & Everling, 2011; Lynch, 1992; Paré & Dorris, 2011; Paré & Wurtz, 1997) and the superior colliculus (Munoz & Wurtz, 1995; Pierrot-Deseilligny, Rosa, et al., 1991; Schiller & Stryker, 1972). These areas are not usually considered to play a functional role during OKN.

The slow-phase and fast-phases of OKN are mediated by separate structures. The flocculus and the nucleus of the optic tract are responsible for slow-phase generation (Blanks & Precht, 1983; Kato et al., 1986; Schiff et al., 1990; Zee et al., 1981) whereas the fast-phase is generated through the reticular formation (Curthoys, 2002; Curthoys et al., 1984; Curthoys et al., 1981; Hess et al., 1989). There are notable connections between the superior colliculus (SC) and the nucleus of the optic tract (NOT) as well as between the SC and the reticular formation (Büttner-Ennever et al., 1996; Cardozo et al., 1994; Hikosaka & Kawakami, 1977;

Holstege & Collewijn, 1982; Kitama et al., 1995). These significant connections between the SC and areas associated with OKN provide a potential way in which higher level saccadic areas might integrate with lower-level oculomotor processes.

The connections between the SC and the fast-phase generating reticular formation are not altogether surprising; it has been claimed for many years that saccades and OKN fast-phases share the same brainstem execution machinery (Ron et al., 1972). However, it is less clear why there should be such substantial connectivity between the SC and the NOT; connections which are just as substantial as those which exist between the SC and the reticular formation (Büttner-Ennever et al., 1996).

Although some authors have dismissed the connections between the SC and the NOT as unimportant for the generation of OKN (Holstege & Collewijn, 1982), others have postulated that they may be used to suppress voluntary fixation and saccades during OKN (Büttner-Ennever et al., 1996). There does appear to be functional connectivity between these two areas, for example some neurones in the NOT change their firing rate in response to any saccades (Mustari & Fuchs, 1990). Perhaps this connection could be the mechanism which allows the updating of saccadic motor maps by OKN slow-phases, as observed in Chapter 2. The NOT is thought to be responsible for velocity storage during OKN (Kato et al., 1986; Mustari & Perachio, 1994), therefore it is not inconceivable that a connection between NOT and SC could allow the updating of saccadic motor maps during OKN. As the experiments presented in Chapter 2 found no difference between the accuracy of saccades executed during either OKN or smooth pursuit, it is likely that the same neural mechanism is responsible in both cases. Accordingly, the NOT is also involved in the maintenance of smooth pursuit; lesions to the NOT impair both OKN slow-phases and pursuit (Mustari & Perachio, 1994; Yakushin et al., 2000). Therefore it is possible that the connections between the SC and the NOT could be responsible for updating saccades during both OKN and smooth pursuit.

Connections between saccadic areas and those responsible for the generation of fast phases have been discussed in detail in Chapter 5, however, to summarise, it seems likely that activity in the SC can influence, and can be influenced by fast-phases. This could be served by the known connections which exist between the SC and the reticular formation (Cohen et al., 1985; Grantyn & Grantyn, 1976). However, the observation that the SC is involved in the processing of fast-phases would open up the possibility that other 'higher' level areas (such as frontal and parietal cortices) could feed into fast-phase generation (and vice versa). Thus it is possible to envisage a single, integrated and unified oculomotor system; where stimulus-driven and internally-generated eye movements are programmed using overlapping neural pathways.

7.4 Potential Benefits of Integrating Automatic and Volitional Eye Movements

Thus far the argument has been put forward for an integrated oculomotor system – one where automatic and volitional eye movements are processed using an overlapping and interconnected mechanism. However, what benefits would such a system bring? What behaviours would an integrated system allow that would not be possible using separate automatic and volitional modules? One possibility that has already been touched upon is that it would allow the co-ordination between target selecting and gaze-stabilizing eye movements. Such co-ordination must occur when a moving observer naturally views scenes, and the results of Chapter 2 confirmed that such co-ordination does take place. An integrated system with clear connections between the automatic gaze-stabilizing and voluntary target-selecting eye movements could achieve such co-ordination, and would allow a moving observer to most efficiently act in a rich visual scene.

Another possibility is that top-down influences could modify the behaviour of 'automatic' eye movements in a task-relevant way. In Chapter 4 to Chapter 6 evidence was presented which showed that processing in areas traditionally considered as saccadic was affected by, and could affect the generation of involuntary nystagmus fast-phases. So far the putative influence of cortical areas upon OKN has been limited to the delivery of visual information to, and between subcortical areas. For example, binocular connections mediated via the cortex are assumed to be the mechanism that allows monocular OKN symmetry to develop (Distler & Hoffmann, 1992; Lewis, Maurer, Chung, Holmes-Shannon, & Van Schaik, 2000; Schor, Narayan, & Westall, 1983). Furthermore OKN slow-phase gain and symmetry can be affected by ablation of the SC (Flandrin & Jeannerod, 1981) or the visual cortex (Montarolo, Precht, & Strata, 1981). However, it is possible that cortical areas could influence OKN in a task-relevant manner, and could be just as relevant to the control of OKN fast-phases as they are for saccades. Under natural viewing conditions moving observers do not appear to make fast-phases and saccades separately; rather the fast-phases of OKN have target-selecting properties (Moeller et al., 2004). Some models of fast-phase generation (Anastasio, 1997; Curthoys, 2002) are explicitly models of saccade generation (e.g. Scudder, 1988) with top-down input from the SC removed. Adding the cortico-collicular network back into the model would account for how fast phases can also target specific stimuli. It may be that the cortico-collicular network is silent during OKN only in experimental lab conditions where there are no interesting objects in the visual scene for targeting (Kashou et al., 2010; Konen et al., 2005). The ability of fast-phases to take on goal-directed, targeting behaviour would show a very high degree of co-ordination between volitional and automatic processes.

The same process may also occur for the slow-phases of OKN. Although this thesis did not try and modify the slow-phases of OKN in a task relevant manner, there is evidence that slow-phases of the vestibulo-ocular reflex (VOR) can take on goal-directed behaviour.

For example, the gain of VOR slow-phases is significantly improved if participants, without any visual stimulation, simply imagine looking at an earth-fixed target (Barr, Schultheis, & Robinson, 1976). The gain can be significantly reduced if participants imagine fixating a target which moves with their head movements (Barr et al., 1976). Moreover, the gain of VOR can be shifted up or down if participants imagine tracking a target which is moving slightly faster or slower than their head movement (Melvill-Jones, 1994). Additionally, VOR gain can shift following adaptation to an imagined moving target (Melvill-Jones, Berthoz, & Segal, 1984). Therefore, reflexive slow-phases can be adjusted according to current behavioural needs. Connectivity with higher level, goal-directed areas would be necessary to achieve such task-relevant adjustment.

7.5 The Evolution of Volitional Actions from Automatic Reflexes

Previous authors have postulated that purposeful saccades evolved through cortical areas developing the ability to commandeer the older, sub-cortical fast-phase circuitry (Ron et al., 1972; Walls, 1962). It it also possible that smooth pursuit evolved in a very similar fashion, through the purposeful control of slow-phase generating systems (Gellman et al., 1990; Walls, 1962). However, the results of this thesis imply a much closer co-ordination between saccadic areas and OKN areas. For saccades to spatially update themselves due to OKN displacements then there must be a delivery of information from the OKN machinery to saccadic areas. Similarly, the same could be said if OKN fast-phases can cause saccade curvature. Saccadic inhibition suggests that OKN fast-phases interact with saccadic areas before they are initiated. Such close ties between saccadic and optokinetic regions implies that there is more integration than would be expected if separate saccadic modules are simply commandeering the OKN machinery. Rather, it implies there are no separate saccadic and OKN modules, but a single, integrated system, with free-flowing information from upper to lower levels and vice versa.

This view may be able to help eludicate the way in which volitinal saccades evolved in the first place. Instead of the sudden emergence of specialised saccadic areas to take over the ancient optokinetic systems, it would be more parsimonious to imagine that the progressive and linear development of the optokinetic system itself eventually allowed saccadic behaviour as we know it today. If the optokinetic system were made gradually more sensitve and complex; for example targeting fast-phases to specific points, or tailoring slow-phases to track a particular object of interest; then eventually the automatic, stimulus-driven nature would give way to flexibe, adaptive, controlled behaviour. Accordingly, I would argue for a shift in perspective: it is not that OKN sometimes takes advantage of the cortico-collicular 'saccade' network to achieve slightly more clever, flexible behaviour (e.g. targeting fast-phases, or tracking slow-phases). Rather, the cortico-collicular 'saccade' network is not originally a 'saccade' network at all (as viewed for laboratory saccades made by a stationary observer); rather it developed as a network for guiding OKN fast phases to specific objects of interest for active moving animals. Similarly, the smooth pursuit system may have originally developed from increasing flexibility in how the optokinetic system tracks retinal movement. In this view, we have a potential model for how an automatic system becomes a voluntary one.

This model does not have to be restricted to eye movements, indeed it could be extended to shape our thinking of how any volitional action first evolved. For example, consider the last time you walked down a street. The action of walking is stereotyped, repetitive, highly automated and was achieved by very ancient ancestors. Yet when you walk down a street puddles are missed, adjustments are made to accommodate raised kerbs or slippery patches, and your feet generally avoid stepping on unsavoury items. Thus, like so many human behaviours, there is inherent flexibility and selection (or 'volition') alongside

automaticity, and it is the interplay between these characteristics that lies at the heart of what it is for humans to make actions.

One analogy is that of an automatic pilot or cruise control system and a human pilot or driver. The cruise control system is sufficient as long as the road and traffic are predictable, but if anything tricky or unusual is perceived, the driver takes over command. In the view proposed here, the 'driver' is not a distinct mechanism from the 'cruise control' that operates in parallel and occasionally takes command; rather the cruise control system incrementally becomes more sophisticated and able to flexibly handle all the tricky situations. Avoiding puddles is just as much part of 'walking' as is putting one foot in front of the other; the more sophisticated job is not run by a distinct system.

Therefore the linear and progressive development of early, stimulus-driven, automatic actions might eventually allow flexible, voluntary behaviours. The co-ordination between automatic, gaze-stabilizing and volitional, targeting eye movements serves as a very useful illustration of how this process might operate. This thesis advocates the view of an integrated and unified oculomotor system, which is exactly what would be predicted by the evolutionary argument proposed here.

7.6 Concluding Remarks

This thesis serves as another example of how simple oculomotor behaviours serve as useful effector systems to enable research into much broader cognitive processes. Although a number of different tasks and paradigms have been adopted in this thesis, the finding which links every experiment is that automatic and volitional processes share considerable similarity. These close interactions between automatic and volitional eye movements would never be predicted by those who envisage reflexive and volitional processes as distinct and separate. However, the existence of a close interaction between automatic and volitional

processes makes a lot of sense. For it allows the co-ordination between gaze-stabilizing and target selecting eye movements; it might enable gaze-stabilizing movements to take on flexible, goal-relevant behaviour; and it may even serve as an illustration of how volitional behaviour could evolve from automaticity in the first place. I believe that achieving understanding of how basic movements and actions work, and crucially, how they work alongside one-another, can inform our thinking on some of the fundamental mysteries of human psychology.

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