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Suppression of displacement in severely slowed saccades

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Abstract

Severely slowed saccades in *Spinocerebellar ataxia* have previously been shown to be at least partially closed-loop in nature, their long duration means that they can be modified in-flight in response to intrasaccadic target movements. In this study, a woman with these pathologically slowed saccades could modify them in-flight in response to target movements, even when saccadic suppression of displacement (SSD) prevented conscious awareness of those movements. Thus, SSD is not complete, in that it provides perceptual information that is sub-threshold to consciousness but which can still be effectively utilised by the oculomotor system. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Saccadic suppression of displacement; Slowed saccades; Visuomotor control; Feedback; *Spinocerebellar ataxia*

1. Introduction

During saccades (fast gaze-shifting eye movements) displacements of objects in the visual world are much less likely to be reported than are equivalent movements occurring during periods of fixation. This degradation of vision has become known as saccadic suppression of displacement (SSD). Outside the laboratory, the effect can be illustrated by our inability to detect the image of our own saccadic eye movements in a mirror, even though we can easily detect other peoples' saccades (Dodge, 1900). In a typical laboratory experiment, a subject is asked to follow a jumping target with their eyes. The target is displaced again slightly during the saccade to the new target position. If the stimulus characteristics are appropriate, then this second displacement is not perceived at a conscious level (Bridgeman, Hendry, & Stark, 1975; McConkie & Currie, 1996; MacAskill, Muir, & Anderson, 1999b).

The motor system, however, is able to make use of intrasaccadic displacements to adjust its performance at an unconscious level. Such a target movement simulates a dysmetria, or inaccuracy, of the saccadic motor control system in that the eyes do not appear to land at the intended target location. If the intrasaccadic displacements are of a consistent size and direction, the system then adapts itself to reduce the apparent error. After a large number of trials, the landing position of the eyes corresponds to the anticipated final location of the target, rather than to where the target was originally seen (McLaughlin, 1967).

Adaptation of saccadic metricity also occurs naturally. Saccades are normally ballistic, or open-loop, movements. They are of such short duration that visual feedback cannot be used to alter their characteristics in-flight, so the size of a saccade must be programmed before its initiation. To maintain the accuracy of the system, the gain of the eye-movement-control signal must be adaptable over time in order to take account of ageing. In this way saccadic eye movements in healthy people remain accurate until at least the age of 70, despite the ageing processes that the saccadic system is subject to at a neural and muscular level (Fuchs, Reiner, & Pong, 1996).

The characterisation of saccades as open-loop fails, however, in those cases where saccadic movements are grossly slowed due to pathological processes. Zee, Opti-

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can, Cooke, Robinson, and Engel (1976) made the first quantitative measurements of such severely slowed saccades. Examining two teenagers with spinocerebellar degeneration, Zee et al. found their voluntary saccadic eye movements to be grossly slowed (by up to 90%), yet still to obey saccadic main sequence type relationships (functions relating the maximum velocity and duration of saccades to their amplitude (Bahill, Clark, & Stark, 1975). Zee et al. presented their subjects with several tasks wherein a target was displaced intrasaccadically. For example, in one condition, the target 'jumped 20° in one direction and after an interval, …, jumped 20° further in the same direction to a final position 40° from the starting position' (p. 243). A normal subject's response in such a situation is to program and execute a saccade to the first (20°) position. As normal saccades are ballistic, a second saccade to the final (40°) position can only be executed after the eyes come to rest at the now erroneous 20° position. However, Zee et al. found that their two subjects could make a single (slow) saccade to the 40° position; i.e. they could alter the characteristics of their saccade by responding to the changing target behaviour in-flight (see Fig. 3 for a graphical example and Fig. 6 for a quantitative portrayal of such performance). Thus, it can be reasoned that normal saccades are only open-loop because they are too brief to encompass the reaction time required to respond to changing target behaviour. For slowed saccades, lasting hundreds rather than tens of milliseconds, there is sufficient time for visual information to be used to guide the eyes accurately toward a target when that target moves during the course of the saccade.

The methodology employed by Zee et al. (1976) (intrasaccadic target displacement) is the same as that used to demonstrate SSD. However, a 20° intrasaccadic displacement is so large as to be certainly perceptible, as SSD can conceal only much smaller displacements (MacAskill et al., 1999b). We, therefore, decided to investigate whether displacements sufficiently small to go undetected by normal subjects could be detected by a subject with slowed saccades. At a motor level, subjects with slowed saccades are able to respond intrasaccadically to target displacements. Does this imply their conscious awareness of those displacements?

In some situations the human motor system can indeed appropriately utilise visual information which is below the threshold for conscious detection. Stoffregen (1985, 1986) tentatively demonstrated that subjects could use visual (optical flow) information to control their postural sway even when that flow was at a sub-threshold level. In the phenomenon of blindsight, a person can become blind due to damage to visual cortical areas, yet still be able to discriminate between and localise visual stimuli that they deny seeing (Sanders, Warrington, Marshall, & Weiskrantz, 1974; Marcel, 1998). As described by Zeman (1998), '… the loss of certain kinds of activity in certain crucial areas of the visual cortex impairs or extinguishes visual awareness, without necessarily abolishing visually guided behaviour' (p. 1697). Pöppel, Held, and Frost (1973) measured saccades made to 'unseen' targets presented in the scotomata (circumscribed areas of blindness) of four subjects. They found that the magnitude of saccades was related (albeit very weakly) to the angular eccentricity of the target. Zihl (1980) showed that this poor accuracy could be improved markedly over many sessions of practice.

Additional evidence of the ability of blindsighted individuals to use visual information to guide their motor systems has been provided by studies of shape perception. Perenin and Rossetti (1996) described a patient who was unable to give a verbal report of the orientation of a slot when it was presented in his hemianopic field but who could nonetheless accurately place a card in that same slot. Another patient (reported by Goodale, Milner, Jakobson, & Carey, 1991) had agnosia for form, although her vision was preserved, she could not make even simple judgments about shape. Yet she also could accurately post objects through slots of varying orientation.

Normally, we use verbal reports to communicate our conscious awareness of visual events. In blindsight, the motor system acts to communicate, revealing that visual information has penetrated to the cortical areas responsible for controlling the relevant motor response. Such motor reports can convey richer information, however. Bridgeman (1992) described aphasic individuals who, when set written language puzzles, fixated the correct solutions longer even though they failed to solve the problems. Thus, motor reports can be used to circumvent deficits in either verbal communication or in consciousness itself.

Using the SSD paradigm, Bridgeman, Lewis, Heit, and Nagle (1979) moved a visual target during the saccades of normal subjects. The subjects were able to give an accurate motor report of target location (via manual pointing) even when they were consciously unaware that the target had been moved intrasaccadically, i.e. they demonstrated a dissociation between cognitive and motor-oriented systems of position perception. It could, however, be said that what they had actually demonstrated was a dissociation between perception of position and displacement. It is not likely that a person's failure to detect a change in a target's position during a saccade would influence their ability to correctly determine its current position some time afterwards.

Access to an individual with severely slowed saccades gave us a unique opportunity to directly assess the oculomotor system's ability to respond to displacement information. This allowed us to avoid the confounding use of another motor report (pointing) based on posi-

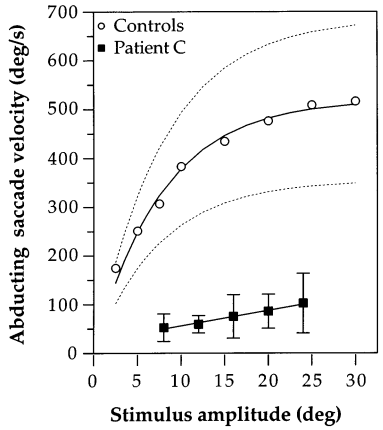

Fig. 1. Main sequence graph showing mean abducting eye peak velocity as a function of saccade size for 11 normal controls (open circles) and for patient C (filled squares). The dotted lines and error bars represent 1.96 S.D. for controls and for patient C, respectively.

tion information. It also provided a true test of the hypothesis that, displacement information is not completely suppressed in saccades but is only selectively available to various levels of the nervous system.

Preliminary results of this experiment have been presented in brief form elsewhere (MacAskill, Anderson, & Jones, 1999a).

2. Method

2.1. Subjects

2.1.1. Patient C

The subject was a female aged 36–39 over the course of the experiments reported here, with a 20 year history of autosomal dominant spinocerebellar ataxia (SCA Type 2, see Buttner et al. (1998) for a description of the oculomotor findings in the various spinocerebellar ataxias). In addition to cerebellar ataxia, incoordination and dysarthria, there was cranio-cervical dystonia, areflexia and extensor plantar responses. Nerve conduction studies confirmed a mild axonal sensory neuropathy. There was mild conjugate limitation of upgaze and markedly slowed saccades in all directions. Smooth pursuit and the vestibulo-ocular reflex were normal. Uncorrected visual acuity was $6/9-1$ in the left eye and $6/12-2$ in the right. Colour vision was preserved and there was no optic atrophy or retinal pigmentary change. Blepharospasm (involuntary blinking) was frequent and controlled periodically with botulinum toxin injections.

Main sequences (relating the velocity and duration of voluntary saccades to their amplitude) are shown in Figs. 1 and 2. Patient C's velocities and durations are

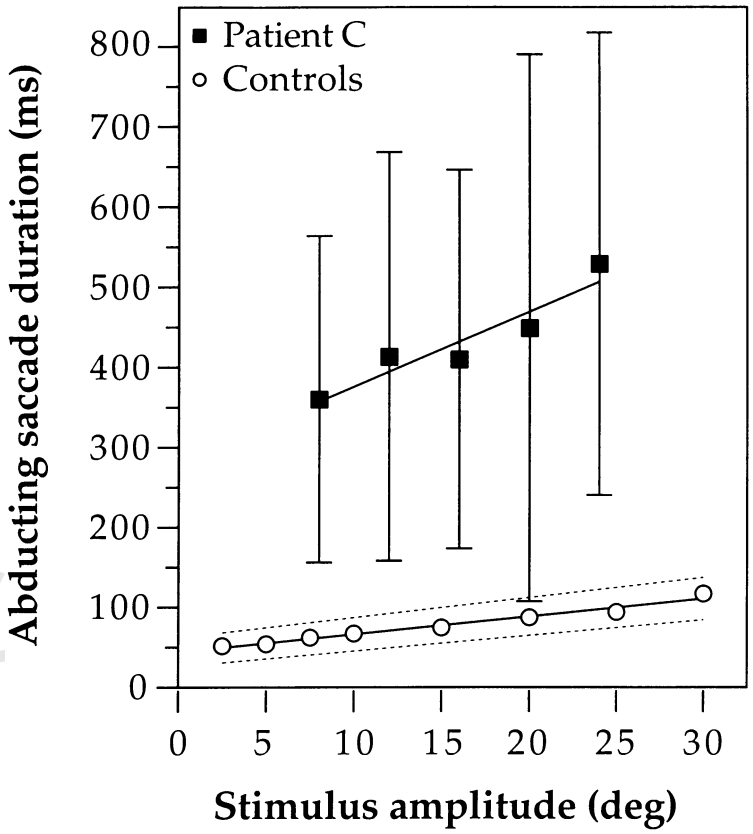

Fig. 2. Main sequence graph showing mean duration of abducting saccades as a function of saccade size for 11 normal controls (open circles) and for patient C (filled squares). The dotted lines and error bars represent 1.96 S.D. for controls and for patient C, respectively.

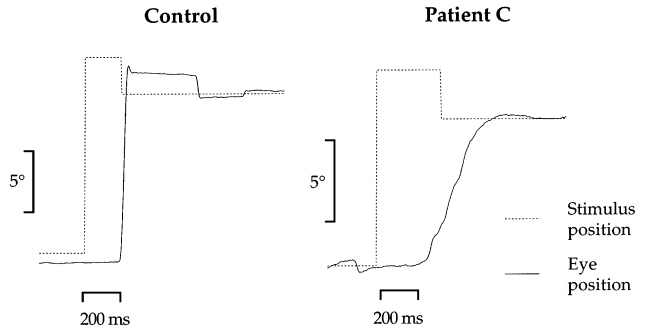

Fig. 3. Representative recordings of target position (dotted lines) and corresponding eye position (solid lines) in the intrasaccadic displacement paradigm (upward deflection of the trace indicates a movement to the right, downward to the left). The control subject (left-hand graph) made a primary saccade to follow the initial target movement. Only after an interval were corrective saccades made to bring the eyes to the final target location. However, patient C (right-hand graph) was able to modify her saccade in-flight, such that her eyes moved directly to the final target location.

clearly abnormal and are similar to the lesser affected of the two cases reported by Zee et al. (1976). The fast phases of optokinetic nystagmus were as slowed as her voluntary saccades, falling on the same main sequence. Compensatory eye movements in response to rapid impulsive head turning were of normal velocity. Fast phases of vestibular nystagmus were not measured.

Zee et al. (1976) conclusively demonstrated that the slow refixational movements of their subjects were in fact saccades and not some substituted slow eye movement, such as 'voluntary smooth pursuit'. The similarity of patient C's pathology and her main sequence data for both voluntary saccades and fast phases of optokinetic nystagmus also point to the saccadic nature of her refixations.

2.1.2. Controls

Three groups of control subjects were utilised. Normal main sequence data were provided by 11 neurologically normal controls (mean age 42 years, range 27–62) to provide a comparison with the patient's saccadic velocities and durations.

The control group for Section 2.3.1 comprised eight experimentally naïve, neurologically normal subjects (mean age 24 years, range 17–43, six female) recruited to compare displacement detection ability.

The control group for Section 2.3.2 comprised two females (age 26 and 44) and one male (age 54), all neurologically normal. One subject was experienced in eye movement recordings, but all were naïve to the purposes of this experiment. The subjects were recruited to provide a control comparison to the patient's ability to modify saccades in-flight.

2.2. Apparatus

Eye movements were recorded using an IRIS (Skalar Medical, The Netherlands) infrared limbus tracker. A computer-generated stimulus (a red square target subtending 0.75°) was video front-projected on to a large screen. The subjects sat on a chair 1.72 m from the screen, with a bite bar providing head stabilisation. A 486 PC controlled the screen display, and its keyboard was used by subjects to give keypress responses. A second 486 PC stored the eye movement data, detected saccades in real time, and transferred this information to the other PC for control of eye movement contingent display changes. At the end of each test, the keypress information was automatically incorporated into the eye movement data file for offline analysis.

Calibration was performed prior to each trial block, with the subject alternately fixating three point targets at 15° left, centre, and 15° right. Signal gain and offset and sensor position were adjusted in an iterative process until the eye position signal corresponded to the three target values (the IRIS is linear within this range).

2.3. Procedure

2.3.1. Experiment 1: quantifying extent of SSD

Subjects were instructed to follow the target as it jumped horizontally by 8, 12, 16, 20 or 24° (see Fig. 3). After a saccade toward the new target position was initiated, the target was displaced centripetally by 1, 2, 3, or 4° on two-thirds of the trials (i.e. displacement ratios of 0.04–0.50). On the remaining one-third of trials, the target was not displaced at all ('catch trials', included to allow assessment of subjects' false alarm rate). Subjects were instructed to report awareness of an intrasaccadic target displacement after each trial by pressing a key. The final position of the target at the end of the trial then served as the initial target position for the following trial. Prior to data collection commencing, subjects performed a standard reflexive saccadic test, followed by sufficient practice on the detection task to ensure that the task requirements were understood.

For control subjects, a mean 27 ms (range 0–39 ms) elapsed from saccade initiation until the target could be displaced. This comprised a mean 15 ms to reach and detect the 30 deg s^{-1} velocity threshold indicating saccade initiation, a further 5 ms for the computer to move the target, and a mean delay of 7 ms due to the screen refresh rate. As the mean control saccade duration was 62 ms (range 40–110 ms), the system was always capable of producing target displacements within the duration of control subjects' saccades and also within the much longer saccadic duration of patient C. For controls, this minimum delay was incremented by either 0, 10, 20, 35, 50, or 65 ms on each

trial to assess differences in detection between targets displaced intrasaccadically and those displaced after the eyes had come to rest. For patient C, the variable delays were necessarily larger, and are described in Section 3.

For controls, fully crossing the saccade sizes, displacement sizes (including zero displacement), and delays yielded 180 trials. These were split into four tests of 45 trials, each lasting about 100 s, with a short rest break and instrument recalibration between.

All subjects performed these trials in both light and dark conditions. Control subjects performed both conditions in the same session, in two blocks balanced for order across subjects. Patient C performed the two conditions on several separate sessions. In the dark condition, the room was completely dark, with the red stimulus being the only visible feature. Room lights were turned on in the rest periods between tests to minimise dark adaptation. In the light condition, both the target and a light background (a homogeneous light blue/grey marble texture) were projected on the screen, leading to ambient lighting of the entire room.

2.3.2. Experiment 2: quantifying ability to modify saccades in-flight

The range of displacement sizes and intrasaccadic timings of Section 2.3.1 were not well suited to quantitatively demonstrating the ability of patient C to modify her saccades in-flight. To do this, the basic methodology was unchanged from Section 2.3.1, but the target parameters were altered. Initial target steps were of 8, 12, or 16°. Intrasaccadic displacements could be centripetal or centrifugal and were 0, 12.5, 25, 50, or 75% of the initial step. The delay between saccade detection and intrasaccadic target displacement was set at 0 ms for each trial so as to maximise the amount of time available to respond to the displacement during a saccade. The intertrial interval ranged from 2500 to 3500 ms. One hundred sixty-two trials were split into six tests of 27 trials each, lasting approximately 85 s with a short rest break and instrument recalibration before each one. The fixed intrasaccadic displacement timing allowed both patient C and the controls to perform exactly the same tests.

3. Results

3.1. Experiment 1

A preliminary exploratory session with patient C revealed that the timings used for control subjects made it impossible for her to perform the task. Consequently, the minimum intertrial interval was increased from 2000 to 3000 ms for her so that she could keep up with the trials. The fixed delay increments of 0–65 ms between saccade detection and target displacement produced a range of intra- and post-saccadic displacement times for control subjects. However, with the greatly extended duration of patient C's saccades (Fig. 2), such delays all resulted in intrasaccadic displacements, allowing no comparison with her ability to detect displacements occurring postsaccadically. Subsequently, a more sophisticated timing regimen was devised for patient C, based on a main sequence from her preliminary recording. This allowed us to predict her mean saccadic duration for a saccade of a given size. The target displacement was then timed to occur at a given offset (a range of constants between -300 and $+200$ ms) of this predicted duration. In effect, this led to the same situation as for controls that displacements occurred at a range of times, either intrasaccadically or postsaccadically. However, for patient C, the delay between saccade initiation and subsequent target displacement was usually much larger in absolute terms due to her much longer saccade durations (Fig. 3).

Subsequently, patient C performed six sessions over a 6-month period in the light background condition. Each session comprised five tests of 30 trials each. The large number of sessions reflected the severity of the patient's blepharospasm that the majority of trials had to be discounted from analysis due to contamination by blinks, requiring additional sessions to build up a useful number of observations. Of 900 trials, 265 were able to be analysed. Of these, 108 contained intrasaccadic target displacements and 64 contained postsaccadic ones. The remainder were catch trials where no target displacement occurred. It should be noted that the catch trials included no false alarms; i.e. patient C never indicated that a displacement had occurred when, in fact, it had not. Six of the control subjects also had a zero false alarm rate with the remaining two exhibiting 2 and 20% rates. No consistent adaptation of saccadic amplitude was observed in control subjects' data due to the variable intrasaccadic displacement size and frequent catch trials with no displacement.

Fig. 4 illustrates that a person with severely slowed saccades can still exhibit saccadic suppression of displacement. Patient C's hit rate (the proportion of displacements correctly reported) is near zero when displacements occur in the early portion of a saccade, and approaches unity when they occur postsaccadically. There is a sharp transition between the two states, occurring during the latter half of the saccade and the early post-saccadic period. Even though this subject's saccades are of long duration and low velocity, saccadic suppression of displacement clearly remains a strong effect.

Saccadic suppression in this subject clearly adheres to the time course for healthy subjects first quantified by Bridgeman et al. (1975). Fig. 5 illustrates another characteristic of SSD, the increase in hit rate with increasing

displacement ratio (the ratio of the size of the intrasaccadic displacement to the size of the saccade), i.e. a 1° displacement during a 24° saccade is much less detectable than the same sized displacement during an 8° saccade.

Having demonstrated that, in light conditions patient C exhibited SSD which was similar in form to that of normals, we then tested her in dark conditions. Testing was conducted in two sessions on consecutive days, 3 months following the last light session. Only two sessions were required as the subject had 3 days earlier received a botulinum toxin injection that had largely abolished her blepharospasm, whilst not interfering with ocular motility. Of 300 trials, 131 were able to be analysed. Of these, 70 contained intrasaccadic target displacements, 19 contained postsaccadic ones, and the remainder were catch trials. The false alarm rate was again zero. The functions described in Figs. 4 and 5 were very similar to the results obtained in this condition. The detectability of displacements did not, therefore, alter between light and dark conditions for patient C.

A non-parametric measure of sensitivity is the 'area under the curve' (AUC) of the receiver operating characteristic (ROC, a plot of the hit rate as a function of false alarm rate for an individual). Taking the inflationary effect of the false alarm rate on the hit rate into account, this statistic produces a 'pure' measure of sensitivity. An AUC of 1.0 corresponds to perfect sensitivity, whereas an AUC of 0.5 corresponds to chance performance. AUC can be estimated using a single hit rate/false alarm rate pair (McNicol, 1972) using a simple formula (Boice & Gardner, 1988). For patient C, AUC = 0.80 in both light and dark conditions. For controls, it was slightly decreased in the dark compared to the light condition (AUC = 0.73 and 0.80, respectively; $P < 0.05$, Wilcoxon matched pairs test).

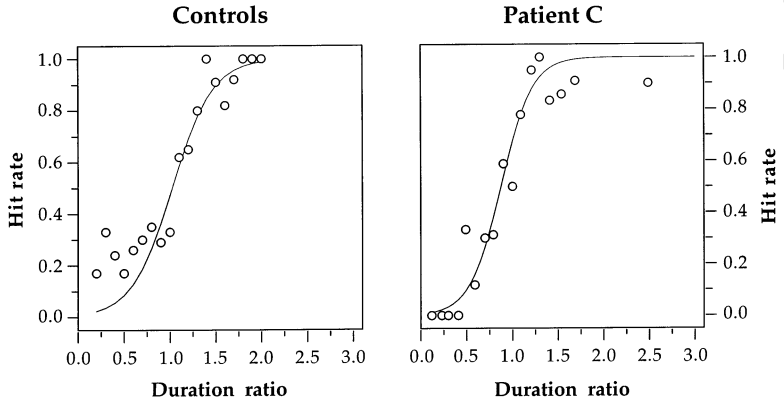

Fig. 4. The probability of detecting target displacements as a function of whether the displacement occurred during the saccade (shaded region) or after (the duration ratio is the delay between saccade initiation and the subsequent target displacement, divided by the duration of the saccade. A ratio of ≤ 1 indicates intrasaccadic displacement; > 1 indicates postsaccadic displacement). Both patient C and the controls exhibit the typical pattern of SSD, with minimal displacement detection during the early part of the saccade followed by a steep increase in detection continuing into the post-saccadic period. The curves of best fit in Figs. 4 and 5 are derived from logistic regression.

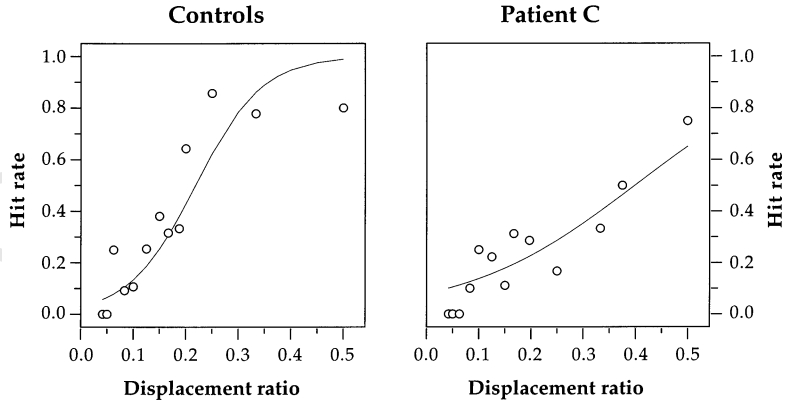

Fig. 5. Probability of detecting intrasaccadic displacements as a function of the ratio of the size of that displacement to the size of the saccade to the target (the 'displacement ratio'). None of the postsaccadic displacement trials shown in Fig. 4 are included. Both patient C and the control subjects exhibit the increase in detection with increasing displacement ratio noted by Bridgeman et al. (1975).

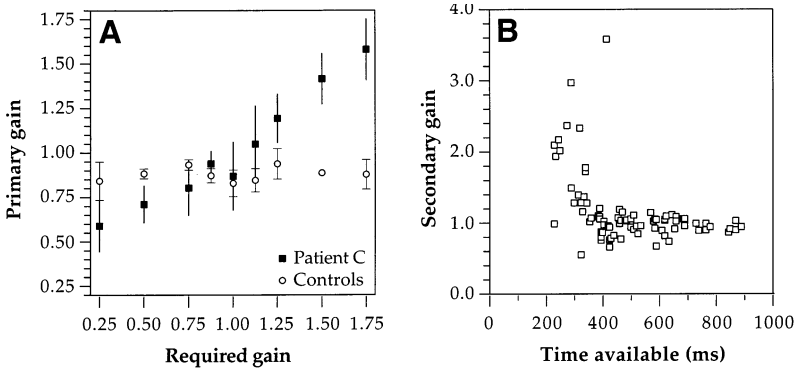

Fig. 6. (A) On the y-axis is the size of the primary saccade relative to the size of the initial target step (primary saccade gain). On the x-axis is the 'required gain', which is the gain required of the saccade to the initial position if it were to reach the final target position in one step (for an initial step of 8° to the right and then an intrasaccadic displacement of 2° to the left, the required gain would be $6/8° = 0.75$). For patient C (filled squares), able to modify saccades in-flight, the primary gain should equal the required gain (i.e. data points should fall on the line $y = x$). For control subjects' ballistic saccades (circles), the primary saccade should always be to the initial location and, thus, the primary gain should be constant at slightly less than 1.0 and, thus, their data points should fall on a horizontal line. These predicted patterns are evident in this figure (error bars = 1 S.D.); (B) data from patient C only. On the y-axis is 'secondary gain', the ratio of the size of the primary saccade to the saccade size needed to fixate the second target position. This measures the inaccuracy of the primary saccade, relative to the final target position. The independent variable on the x-axis is the duration of the saccade minus the time from the initiation of the saccade until the target was displaced, i.e. the time available during the saccade to respond to the intrasaccadic displacement. Data points are calculated for each trial on which an intrasaccadic displacement occurred. Open-loop type saccades (those with a secondary gain markedly different from 1.0) occur mostly when the patient had less than 400 ms during the saccade to respond to the displacement.

3.2. Experiment 2

Patient C completed one session in light background conditions and another in the dark. Data from these two sessions did not differ in terms of saccade trajectory control and, thus, were collapsed to form a single pool of 95 valid trials from a possible 324 (again many trials were excluded due to contamination by blinks). Consequently, control subjects performed the task in one session in the light background only.

The results of Section 3.2 are reported in Fig. 6. On the y-axis of Fig. 6A is the size of the primary saccade divided by the size of the initial target step (the standard 'primary saccade gain'). On the x-axis is the 'required gain', which is the gain required of the saccade to the initial position if it were to reach the final target position in one step. For example, if the target made an initial step of 8° to the right and then an intrasaccadic displacement of 2° to the left, the required gain would be $6/8° = 0.75$. For a control subject making ballistic saccades, the primary saccade should always be to the initial location and, thus, the primary gain should be reasonably constant at approximately 1.0, regardless of the required gain, and, thus, data points should fall on a horizontal line (note that, gain adaptation effects are not seen due to the variability of the intrasaccadic step in both size and direction). For a subject able to modify saccades in-flight, the primary gain should equal the required gain (i.e. data points should fall on the line $y = x$), as the eyes are able to move directly toward the final location rather than be committed to a single saccade to the first location.

The data confirm these predictions. The control subjects' data follow a horizontal line (linear least squares fit, $y = 0.01x + 0.87$) consistent with the typical saccadic undershoot in response to suddenly appearing targets (Becker & Fuchs, 1969; Lemij & Collewijn, 1989). Patient C's responses are clearly closer to the line, $y = x$ (linear least squares fit, $y = 0.68x + 0.34$), indicating an ability to substantially modify her saccades in response to intrasaccadic movements. Her performance in the light ($y = 0.72x + 0.25$) was very similar to that in darkness ($y = 0.65x + 0.39$), and, thus, the data were collapsed to form a single set.

Patient C's performance was still some way from a perfect $y = x$ performance level. On a number of trials, she did make distinct double saccades towards the final target position rather than a single smooth movement. The reason for this apparently open-loop behaviour is shown in Fig. 6B. Here, the dependent variable is 'secondary gain' that, the ratio of the actual size of the primary saccade to the size needed to fixate the second target position in one step, calculated for each trial on which an intrasaccadic displacement occurred. This gives a measure of the inaccuracy of the primary saccade, relative to the final target position. The independent variable on the x-axis is the duration of the saccade (mean 542 ms, range 265–970 ms) minus the time from the initiation of the saccade until the target was displaced (mean 48 ms, range 22–152 ms). Thus, this was the time available during the saccade to respond to the intrasaccadic displacement. Note that there are no data points where $x < 200$, indicating that, on every trial, the subject had at least 200 ms to

respond. Open-loop-type saccades are those with a secondary gain not approximately equal to 1.0, and it can be seen that these occur mostly when the patient had less than 400 ms to respond to the displacement. In this situation, the saccade was committed to finishing at the original location before a corrective saccade could be programmed and executed. Note that patient C's primary saccade latency in this experiment was a mean 231 ms (range 130–330 ms), and that the latency of her corrective saccades (measured from those trials with secondary gain >1.5) was a mean 163 ms (range 60–290 ms), i.e. when discrete corrective saccades existed, they exhibited the same 70 ms decrease relative to primary saccades, as seen in controls (Leigh & Zee, 1999). Also, of interest is that most open-loop corrections occurred in response to overshooting target movements (note the large number of secondary gain values $\gg 1$, but relatively few $\ll 1$).

4. Discussion

Several findings emerge from this study. Firstly, we have demonstrated that, extremely slowed saccades can be modified in-flight in response to intrasaccadic displacements. Although this was earlier reported by Zee et al. (1976), we have extended these authors' observations with more quantitative evidences (Fig. 6), and show the circumstances in which this closed-loop control is not exhibited. Our subject with slow saccades reverted to closed-loop corrective saccades when there was not sufficient time during the primary saccade to change the saccade trajectory. In this situation, it was primarily overshooting target movements for which the subject was unable to compensate in-flight. Particularly, when they occur late in a saccade, overshoots can require the eyes to reverse direction and, thus, corrective movements may be harder to execute than adjusting the eye movement trajectory further in the same direction. The oculomotor system has a strong predilection to make corrective saccades in the same direction as the primary saccade (Henson, 1978). However, in this experiment, the eyeball dynamics on each trial were likely to be the dominant factors in making the execution of oppositely-directed corrections difficult.

Secondly, SSD can still occur in a person with extremely slow saccades and the characteristics of that suppression are similar to those of controls with normal velocity saccades. For example, the time course of suppression closely followed the duration of the saccade (Fig. 4), even though patient C's saccades were longer than control saccades by an order of magnitude. Patient C's sensitivity to intrasaccadic displacements did not vary between light and dark conditions, although the control group did show a small but statistically significant decrease in sensitivity in the dark. The only similar published comparison we are aware of was by Deubel, Schneider, and Bridgeman (1996) who measured the ability to detect discrete displacements of a small red laser spot in complete darkness. They found performance to be essentially identical to that in an illuminated environment, but examined only two subjects. Our results are consistent with those of Lappe, Awater, and Krekelberg (2000) who investigated a related phenomenon. They found that the mislocalisation of perisaccadic flashed targets was greater in the dark than in conditions where visual contextual information was available. They reasoned that, in the dark, the only target position information available is derived from an eye position signal. Richer and more accurate information is available from vision, and this may also explain why our control subjects were better at detecting changed target positions in the light.

The third and most important finding of this study is that in-flight saccadic modification can occur even when SSD prevents conscious awareness of intrasaccadic displacements. This adds to earlier studies (Pöppel et al., 1973Zihl, 1980) showing that the oculomotor system can utilise information that is not available to conscious awareness. We have also confirmed the finding of a dissociation between cognitive reports (e.g. verbalisation, key pressing) and motor reports (e.g. pointing) of displacement detection (Bridgeman et al., 1979). By using eye position itself as the dependent variable, however, we avoided confounding position and displacement perception. The dissociation between conscious and motor reports shows that SSD does not necessarily produce a 'suppression' of displacement information but rather allows different levels of the CNS selective access to that information. We concur with Blackmore, Brelstaff, Nelson, and Troscianko (1995) who claimed that displacement information is not so much suppressed during saccades as simply neglected, at least at a cognitive level. This demonstration of a dissociation between the motor and cognitive systems accords with the contention of Hallett and Lightstone (1976) that, 'there are almost certainly differences between the sensory processes leading to perception and those leading to saccadic eye movements' (p. 99).

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