Exp Brain Res (2004) 155: 231–244
DOI 10.1007/s00221-003-1717-9

RESEARCH ARTICLES

Árni Kristjánsson · Myriam W. G. Vandenbroucke · Jon Driver

When pros become cons for anti- versus prosaccades: factors with opposite or common effects on different saccade types

Received: 12 February 2003 / Accepted: 9 September 2003 / Published online: 6 December 2003

Abstract In five experiments we compared prosaccade and antisaccade performance in normal human observers. This was first examined for visual stimulation in temporal or nasal hemifields, under monocular viewing. Prosaccades were faster following temporal than nasal stimulation, in accordance with previous results. The novel finding was that the *opposite* pattern was observed for antisaccades, consistent with a difficulty in overcoming a stronger prosaccade tendency after temporal-hemifield stimulation. A second experiment showed that these results were not simply due to antisaccades following nasal stimulation benefitting from being made towards a temporal place-holder. Prosaccades and antisaccades were then compared for visual versus somatosensory stimulation. The substantial latency difference between prosaccades and antisaccades for visual stimuli was eliminated for somatosensory stimuli. Antisaccades can thus benefit in relative terms when the competing prosaccadic tendency is weakened; but two further experiments revealed that not all manipulations induce opposing outcomes for the two types of saccade. Although reducing the contrast of visual targets can slow prosaccades and conversely speed antisaccades, this was not the case at the lowest contrast level used, where both types of saccade were slowed, thus indicating some *common* limiting source. Moreover, warning sounds presented shortly before a visual target speeded both prosaccades and antisaccades. These results illustrate that several factors which slow prosaccades can speed antisaccades (consistent with competition between different pathways); but also reveal some notable exceptions, where both types of saccade are slowed or speeded together, suggesting some common pathways that may precede competition over the direction of the saccade.

Keywords Saccades · Antisaccades · Spatial attention · Visuomotor control · Crossmodal integration

Á. Kristjánsson (✉) · M. W. G. Vandenbroucke · J. Driver
Institute of Cognitive Neuroscience, University College London,
17 Queen Square,
London, WC1N 3AR, UK
e-mail: a.kristjansson@ucl.ac.uk
Tel.: +44-20-76791123
Fax: +44-20-79168517

Introduction

A popular paradigm for comparing stimulus-driven spatial behaviour versus more volitional spatial behaviour is to compare prosaccades and antisaccades (e.g. see Everling and Fischer 1998 for a review). A prosaccade requires an eye-movement from the currently fixated locus directly to the location of a peripheral stimulus (typically, a visual event), usually as soon as it appears. By contrast, antisaccade tasks typically require an eye-movement of equivalent amplitude to be executed rapidly, but now in the opposite direction. To achieve this, the natural tendency to move the eyes towards the new stimulus (sometimes called the 'visual-grasp reflex') has to be overcome in order to direct a voluntary saccade in the opposite direction (see Everling et al. 1998; Everling and Fischer 1998; Forbes and Klein 1996; Kristjánsson et al. 2001; Rafal 2002; Rafal et al. 2000; Schlag-Rey et al. 1997). Antisaccades tend to have higher latencies (by at least 10%; Everling and Fischer 1998; Leigh and Zee 1999), may be less accurate (Krappmann et al. 1998), and usually have lower peak velocities than prosaccades (Leigh and Zee 1999).

In the present experiments, we manipulated factors that are known or suspected to influence the latency of prosaccades, and examined whether the manipulation had similar or opposite effects on the latency of antisaccades. In this way we tested whether a particular manipulation that speeds prosaccades will also speed antisaccades; or whether instead antisaccades can be slower when prosaccades are faster. Our initial motivation for doing this stemmed from recent experiments by Kristjánsson et al. (2001), so for brevity we focus on

that study in our Introduction here; but the many other precedents for comparing prosaccade and antisaccade latencies are considered in the course of describing each of the new experiments in this paper, and also in the "General discussion".

Kristjánsson et al. (2001) examined prosaccade and antisaccade latencies for peripheral visual stimulation, while manipulating the extent to which visual attention was engaged by a different visual discrimination task performed at around the same time. Their main finding was that while attentional engagement in another visual task slowed prosaccades, it had the seemingly paradoxical effect of *speeding* antisaccades under certain conditions. These opposite effects may become less paradoxical when one considers that weakening a prosaccadic tendency may naturally facilitate antisaccades, by reducing the time taken to overcome the urge to make a prosaccade, before executing the antisaccade. Suppressing the prosaccade in order to make the antisaccade may take longer when the prosaccadic tendency is stronger. In Kristjánsson et al.'s (2001) study, the speeding of antisaccades was specifically observed when the visual discrimination task preceded the antisaccade stimulus by 200–500 ms, suggesting that attentional diversion from the prosaccade reflex had to come at a specific time to benefit the antisaccade in this particular paradigm. Here we tested more generally the conditions under which speeding or slowing of prosaccades can lead to the opposite effect on antisaccades, across a variety of paradigms.

In a situation where faster prosaccades do indeed reflect a stronger prosaccadic 'reflex', then antisaccades might presumably be delayed due to the difficulty of overcoming the stronger prosaccadic tendency. Note that this should arise whenever the factor that speeds prosaccades has its principal effect within a pathway that is involved in automatic generation of saccades (e.g. including, but not necessarily restricted to, the tectal pathway involving the superior colliculus; see Goldberg 2000; Sparks and Barton 1993; Schall and Thompson 1999). On the other hand it is possible that prosaccades could also be speeded by some factors that influence some stage(s) of processing that are *common* to both prosaccades and antisaccades. If so, antisaccades might then presumably be speeded by the same manipulation that speeds prosaccades. To give an illustrative example, consider a simplistic S-R model with successive stages (cf. Sternberg 1969), in which an initial sensory processing stage must hypothetically first complete registration of the location of a sensory signal, before this can then be transformed into the command for a prosaccade or an antisaccade at a hypothetically later response stage. Any stimulus manipulation that delays prosaccades would be expected also to delay antisaccades by a similar amount on such a model, if the delay arose at the common sensory stage hypothesized to precede either type of motor response in a strictly serial manner.

Note that, in principle, either type of result (or intermediate outcomes) might thus be found (i.e. manipulations that delay prosaccades might either speed or delay antisaccades), but with the type of outcome being specific to the factor manipulated. Manipulations that speed prosaccades but delay antisaccades (i.e. producing opposite effects on the two types of saccade) might then be intepreted as specifically strengthening the tendency for a prosaccadic reflex, by influencing pathways that generate such prosaccadic tendencies; whereas manipulations that have common effects on pro- and antisaccades (i.e. speeding or slowing both together) might be interpreted as influencing stages or pathways that are common to the two types of eye-movement, rather than reflecting competition between the two processes.

The overall aim of the experiments we describe here was thus to examine the generality of faster antisaccades being found in the context of slower prosaccades (cf. Kristjánsson et al. 2001), while also searching for any exceptions to that pattern.

Experiment 1: saccades after stimulation in temporal versus nasal hemifields

Rafal et al. (1991) monocularly tested subjects who made prosaccades to visual targets presented in the temporal or nasal hemifield (while the other eye was covered by an eye patch). They found reliably faster average prosaccade latencies for temporal targets. They suggested that this might be due to stronger projections to the superior colliculus in the midbrain from the nasal hemiretina (which receives stimulation from the temporal hemifield) than from the temporal hemiretina (see also Posner and Cohen 1980; Shulman 1984).

Although there has since been some controversy over whether temporal/nasal asymmetries are a unique hallmark of the collicular pathway (e.g. see Sumner et al. 2002), the fact remains that prosaccades are typically faster to temporal visual targets than to comparable nasal targets. But to our knowledge, no study has ever tested for any temporal/nasal asymmetry in the latency of *anti*saccades.

Contrasting predictions for antisaccade performance in this respect can be derived from alternative accounts. On the Rafal et al. (1991) hypothesis that temporal visual targets drive stronger 'reflex-like' prosaccades (possibly via a tectal pathway), then saccading *away* from a temporal target, in an antisaccade task, should be slower than saccading away from a nasal target, because a stronger prosaccadic tendency has to be overcome before the antisaccade can be executed. Alternatively, it might in principle be that visual targets in the temporal hemifield are simply detected quicker by the visual system than nasal targets. If so, then antisaccades triggered by a temporal stimulus should be quicker than those triggered by a nasal stimulus (just as for prosaccades), leading to the opposite prediction to that derived from the Rafal et al. (1991) account.

Materials and methods

Observers

Six observers (two female, four male), ranging in age from 22 to 37 years (mean age 26.6 years), were recruited from the subject pool of the Institute of Cognitive Neuroscience at University College London. They were paid £7.50 for participation. All were naive regarding the purpose of the experiment. All observers in this experiment and the following ones gave their informed consent to participate in the study. This study and the others presented in this paper had been approved by the local ethics committee, and observers have informed consent, in accordance with the Declaration of Helsinki.

Apparatus and stimuli

The saccade targets were provided by two red LEDs, each 8 degrees to the right or left of fixation. Another red LED was positioned centrally to provide an initial fixation point. The fixation point and the two peripheral targets were at the same height in the visual field. Two further LEDs (one red and one green) were placed side by side 1.5 degrees directly below the central fixation point. These were used on each trial to indicate (see below) whether a prosaccade or an antisaccade was required for the subsequent peripheral trigger-stimulus. The luminance of the red LEDs was 55 cd/m^2, while that of the green LED was 30.8 cd/m^2 (this means that the near-centre red and green LEDs differed in luminance as well as colour, but since their role was only to indicate *symbolically* the type of saccade required, as explained below, then there was no requirement for isoluminance).

Stimulus presentation was controlled through the parallel port of a Pentium PC computer. The computer sent pulses through this port to switch on or off any of the five LEDs. The pulse sequence was controlled with custom software (XGEN, www.mricro.com). The LEDs were mounted on a black panel and connected via a relay box to the parallel port. Observers wore a black felt eye-patch over one eye. Eye tracking for the unpatched eye was performed with a SKALAR IRIS (Model 3500) infrared eye tracker, which measured eye position at a rate of 500 Hz. To linearize horizontal eye position with respect to the measurements of this system, eye-position signals were calibrated at the start of each block of trials, by obtaining the output from the eye tracker at the locations of each of the two peripheral targets as well as at the central fixation point. Eye traces were saved to disk during the experiment and analyzed off-line to obtain the latency, amplitude and peak velocity of each saccadic eye-movement. The amplitude of the observed prosaccades and the antisaccades was relatively uniform, probably due to the fact that a visible landing point was available in both cases (see below). Head position was stabilized with a chin rest.

Procedure

A trial started when the red central fixation light was turned on. Subsequently (700–1,700 ms later, as determined randomly in 1-ms steps), the central red or green LED that was 1.5 degrees immediately below central fixation was switched on. The central red LED indicated symbolically that a prosaccade would be required on that trial, while the central green LED indicated an antisaccade trial. The trial type (pro or anti) was unpredictable from one trial to the next. The subsequent peripheral saccade trigger-signal was provided by illuminating either one of the two red LEDs at 8 degrees to the right or left of the central fixation LED (either side was equally likely on each trial). This peripheral trigger LED was switched on 1,200–1,800 ms (determined randomly for each trial in 1-ms steps) after the onset of the central LED that had indicated symbolically whether a pro- or anti-saccade was to be made on that trial. At exactly the same time the central fixation LED was switched off. The peripheral LED, serving as the saccade trigger-signal, was visible for 1,500 ms, after which a new trial started.

The experiment was conducted in a dimly lit sound-proof booth. The positions of all LEDs were still visible, however, even when they were not illuminated, so that a visible landing point was available even for the antisaccades. Note that when a landing-point marker is visible in this way rather than being absent, the *latency* of antisaccades typically seems relatively unaffected compared to when no such marker is visible, while the peak velocity and landing point accuracy of the antisaccades can increase (e.g. Edelman et al. 2001).

Observers were told to execute the saccades as quickly as they could, but were also told to try to avoid making many errors (such as an erroneous prosaccade when an antisaccade was required). The experimenter monitored each eye-trace on a monitor during tests, and told the observer if he/she was making too many errors. After some practice observers were able to keep their errors to an acceptable level, typically 10% or less, which is a representative finding for normal observers making antisaccades (e.g. Hallet 1978; Hallett and Adams 1980; Everling and Fischer 1998).

On different blocks of trials, either the left eye or the right eye was patched, thus reversing which visual hemifield (left or right) served as temporal versus nasal. In total, each observer participated in 4 blocks of 60 trials (two blocks for each eye patch), with left and right saccades being intermingled in a random sequence within each block, as was the instruction to make pro- or antisaccades. Whether or not antisaccade and prosaccade trials are intermingled within a block has been shown not to be critical for their overall characteristics (e.g. Hallett and Adams 1980; Cherkasova et al. 2002), although there can be some task-switching effect (like, for example, overall slowing of saccade latencies) when there is only a very short time interval between the signal indicating which type of saccade is to be made, and the saccade stimulus itself (e.g. 200 ms as in Weber 1995; see also Cornelissen et al. 2002). Moreover, prosaccades may be somewhat slower when intermingled unpredictably with antisaccades, possibly due to error-avoidance strategies.

Results and discussion

The mean saccadic reaction times (RTs) across the six observers are presented in Fig. 1, separately for pro- versus antisaccades, and for saccades triggered by illumination of a peripheral LED in the temporal visual hemifield versus the nasal hemifield. Prosaccades were faster when made to an LED in the temporal hemifield (mean=253.5 ms) than for a target in the nasal hemifield (mean=274.1 ms), echoing the findings of Rafal et al. (1991). The critical new result is that the *opposite* pattern was observed for antisaccades, with faster saccade latencies when the antisaccade was triggered by a peripheral LED in the *nasal* hemifield (mean=311.2 ms) than for a trigger in the temporal hemifield (mean=328.6 ms). A 2×2 ANOVA [with the factors of saccade-type (pro versus anti) and trigger hemifield (temporal versus nasal)] revealed a significant main effect of saccade type (faster pro- than antisaccades, $F_{(1,5)}$=105.3, p<.001) but no significant overall effect of hemifield ($F_{(1,5)}$=0.004, p=n.s.). Critically, a significant crossover interaction was observed between these two factors ($F_{(1,5)}$=18.3, p<.01) showing a differential effect of trigger-stimulus hemifield (nasal/temporal) on the latencies of the two types of saccades. Pairwise post hoc comparisons confirmed that while prosaccades were significantly faster following temporal than nasal triggers (paired t_5=3.4, p<0.01), the reverse applied significantly

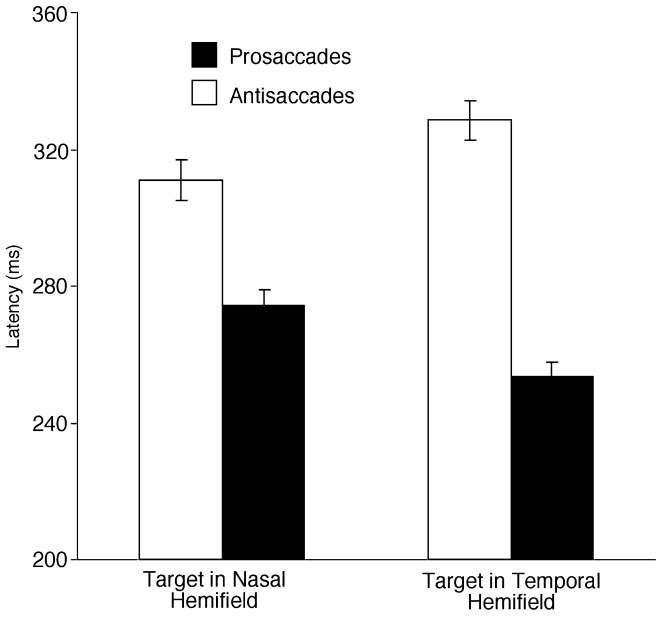

Fig. 1 Mean latencies of pro- and antisaccades as a function of the hemifield (nasal or temporal) in which the peripheral visual trigger stimulus for the saccade appeared, during monocular viewing, for Experiment 1. The *error bars* denote the standard errors of the mean

for antisaccades (paired t_5=2.9, p<0.02), thus producing the crossover interaction in the 2 by 2 ANOVA.

These results accord well with the predictions derived from Rafal and colleagues' account (e.g. Rafal et al. 1991, 2000). According to Rafal et al. (1991), targets in the temporal hemifield drive a stronger prosaccadic tendency than targets in the nasal hemifield (which they suggested might be due to corresponding asymmetries in the neural pathway from the retina to the superior colliculus). For the first time, we also tested here for any temporal/nasal asymmetries with *anti*saccades, and found the opposite pattern to that observed for prosaccades. Saccade latencies were slower when observers saccaded away from a signal in the temporal hemifield than when saccading away from a nasal signal. This argues against the idea that temporal signals are simply detected quicker overall by the visual system, suggesting instead that they really do trigger a stronger tendency for prosaccades (as proposed by Rafal et al. 1991), which then takes more time to overcome when an antisaccade must be made in the direction opposite to the saccade signal (see, e.g. Kristjánsson et al. 2001).

On some trials observers made saccades in the wrong direction. For the prosaccades, these errors occurred on 1.9% of the trials for targets in the temporal hemifield, but on 3.1% of the trials for nasal targets. For the antisaccades these error rates were 9.3% and 8.7% for trigger stimuli in the temporal and nasal hemifields, respectively. Although this pattern of error rates is in overall accord with the latency results, the differences between nasal and temporal targets were not significant for either the prosaccades ($F_{(1,5)}$=1.56, n.s.) or the antisaccades ($F_{(1,5)}$=1.27, n.s). The peak velocities of the saccades were 385°/s and 367°s for nasal and temporal prosaccades, respectively; and 312°/s and 299°/s for the nasal and temporal antisaccades respectively. This difference in peak velocities was significant for the prosaccades ($F_{(1,5)}$=4.32, p<.05), but not for the antisaccades ($F_{(1,5)}$=1.91, n.s). The interaction between the two terms was not significant ($F_{(1,5)}$=1.89, n. s.) The standard deviations of landing points of the saccades were 0.61° for the temporal prosaccades and 0.60° for the nasal prosaccades. Standard deviations for the temporal antisaccades were 0.67°, and 0.70° for the nasal antisaccades, but there was no main effect of hemifield ($F_{(1,5)}$=0.45, n.s.). The effect of saccade type on deviations of landing point was significant ($F_{(1,5)}$=3.6; p<.05), but the interaction between those two main effects was not ($F_{(1,5)}$=0.89). The fact that the difference between landing point accuracy for prosaccades and antisaccades was only small is probably due to the desired landing points being visibly marked for both types of saccade in the present study. It should also be mentioned that the prosaccade latencies in the experiment were relatively high (253 ms into the temporal hemifield and 274 ms into the nasal hemifield), compared with the latencies often found for stimulus driven prosaccades (e.g. ~200 ms; Leigh and Zee 1999).[1] The precise reasons for this are unknown, but it should be kept in mind that our subjects were inexperienced with oculomotor tasks and were wearing an eye patch over one eye. It is also possible that the intermingling of pro- and antisaccades may have led to some strategy for minimising direction errors. But while such factors might have contributed to the overall latency for prosaccades (see also Experiment 3 below), the important point of the present study was not overall latency per se, but rather the differential effect of temporal versus nasal trigger signals, which had opposite effects on pro- versus antisaccades.

The next experiment was a control study designed to address one potential difficulty in interpreting the findings of Experiment 1.

Experiment 2: saccades into temporal or nasal hemifields triggered by central cues

The results of Experiment 1 showed an opposite latency pattern for the effects of temporal or nasal stimulation on pro- versus antisaccades, when scored in terms of whether the peripheral *trigger-signal* for the saccade arose from within the temporal or nasal hemifield. However, if one considers instead the hemifield *to which the eye got directed*, one could argue that the pattern is in fact the same for the two types of saccades; that is, faster performance when the eye must be directed towards the temporal hemifield to produce a correct response. In the case of prosaccades, the visual signal triggering the faster response arose within the temporal hemifield; in the case of antisaccades, the signal triggering a faster antisaccade arose in the opposite (nasal) hemifield instead, but one might argue that following this signal the anti-saccade was then directed to a visible landing point (the unilluminated

[1]We are grateful to an anonymous referee for raising this issue.

LED on the other side) in the *temporal* field. As we noted previously, the possible landing points were indeed tonically visible throughout Experiment 1, despite not being phasically illuminated in the temporal hemifield on trials requiring an anti-saccade away from a nasal target.

Moreover, an extreme sceptic might even suggest that the asymmetries in the observed latencies for Experiment 1 could in principle somehow reflect some artefactual property of how the limbus eye-tracker used here picks up its signal via infrared reflection from the iris and the sclera. Faster saccades were in effect always observed for leftward eye-movements from the left eye (as for a pro-saccade to a temporal target for this eye, or an anti-saccade with that eye in response to a nasal signal), and for rightward eye-movements from the right eye. Although any measurement artefacts seem unlikely to have produced the substantial latency effects we observed in Experiment 1, it is a logical possibility that must be addressed, given that the extent of visible sclera will differ for the nasal versus temporal side of each eye, and the tracker relies on reflectance from the sclera and the iris.

To address any such potential criticisms of Experiment 1, the next study was a control experiment, in which saccade latencies were again monocularly recorded for leftward versus rightward movements with either eye. These saccades were now made to peripheral LEDs which were always tonically visible (as in Experiment 1) but were now *never* phasically illuminated (unlike Experiment 1). The direction for the required saccade was now indicated by a central symbolic cue on each trial. If the previous results were due to the temporal versus nasal location of a visible landing point (albeit an *unilluminated* one, in the case of the previous antisaccades), or due to some measurement artefact when measuring scleral reflectance for nasal versus temporal movements with each eye, then the latency advantage when saccading towards a temporal landing-point should be found once again. However, if the results of Experiment 1 were indeed due to the hemifield in which a *peripheral* saccade trigger-stimulus was *phasically* illuminated, as we have hitherto assumed, then we should now observe no nasal/temporal differences, because no peripheral illumination now took place.

Materials and methods

Six new observers (four female and two male, aged 18–26 years; mean age 22.1 years) were recruited from the same subject pool as before. They were again paid £7.50 for participation, and were naive regarding the purpose of the experiment.

The methodology here was as for Experiment 1 except as noted below. The required direction of the saccade (i.e. left or right) was now signalled centrally and symbolically, by illuminating one or other of the two central LEDs directly below the initial fixation LED. For half of the subjects the green central LED signalled that a rightwards saccade of 8 degrees to the rightward landing point was required; while the red one signalled that a leftwards saccade was required. This was reversed for the other half of the subjects. Peripheral location markers for the saccade landing points were tonically visible throughout (as in Experiment 1), but were now *never* phasically illuminated.

Again each observer wore an eye patch over one eye; on 2 blocks of 80 trials the patch was over the right eye, and on 2 blocks of 80 trials the patched eye was the left eye. The order in which each eye was patched was counterbalanced over the six observers.

Results and discussion

The latencies for saccades into the temporal or nasal hemifield no longer differed. Saccades into the nasal hemifield had a mean latency of 253.6 ms, while saccades into the temporal hemifield had a mean latency of 256.5 ms, with no significant difference (t_5=0.876, n.s) between the two. The temporal/nasal effects previously shown in Experiment 1 were thus now eliminated. This shows that the critical factor behind the results of Experiment 1 could not have been whether the *landing point* for the saccade was temporal or nasal (nor any artefact when measuring temporal versus nasal saccades with a limbus tracker, which was implausible in any case). The critical factor in Experiment 1 must instead have been whether the *phasic peripheral stimulation* to signal the saccade arose in the temporal or nasal hemifield. No phasic peripheral stimulation ever occurred in Experiment 2, since we now used central cues to indicate the required saccade direction instead; and no effect was now observed. This supports the interpretation of Experiment 1 in terms of Rafal et al.'s (1991, 2000) account. Peripheral visual stimulation in the temporal hemifield evidently triggers a stronger prosaccade tendency than nasal stimulation, leading to faster prosaccades towards it than for nasal stimulation, but to slower antisaccades away from it than for nasal stimulation. This is consistent with the general notion of competing pathways for prosaccades versus antisaccades (e.g. see Forbes and Klein 1996; Kristjánsson et al. 2001; Schlag-Rey et al. 1997; Everling et al. 1998; Everling and Fischer 1998; Rafal et al. 2000).

Directional error rates in Experiment 2 were 4.3% for the centrally cued saccades that should have been made into the temporal hemifield, and 3.6% for the centrally cued saccades that should have been made into the nasal hemifield. This difference was not significant ($F_{(1,5)}$=0.98, n.s.) as expected given the arbitrary and counterbalanced nature of the central cues indicating the required directions. The peak velocities of saccades were 298°/s and 312°/s for saccades into nasal and temporal hemifields, respectively, with no significant difference between the two (t_5=1.2, n.s). The standard deviations of landing points of the saccades were 0.75° for saccades into the nasal hemifield, and 0.83° for temporal saccades, again with no significant difference between the two (t_5 = 0.86, n.s).

Experiment 3: saccades triggered by tactile versus visual stimuli

Most eye-movement research has focused on saccades directed by visual stimulation (see, e.g. Leigh and Zee

1999) with some studies of saccades triggered by auditory stimuli (e.g. Zambarbieri et al. 1995). There have been substantially fewer studies of saccades triggered by somatosensory stimuli (though see Amlot et al. 2003; Groh and Sparks 1996a, 1996b, 1996c; Neggers and Bekkering 1999; Blanke and Grüsser 2001). Groh and Sparks (1996a, 1996b, 1996c) published an initial series of experiments on this, investigating the behavioural characteristics of haptically cued saccades in both humans and monkeys, as well as gathering single-cell neurophysiological data from collicular neurons in the monkeys. One basic finding was that prosaccadic latencies for tactile stimuli were typically slower than for visual stimuli at comparable eccentricities (by about 50 ms for humans; see also Neggers and Bekkering 1999). Somatosensory saccades also tended to be less accurate than visually triggered saccades (Groh and Sparks 1996a; see also Amlot et al. 2003).

To our knowledge, there has not yet been any systematic research on antisaccades following tactile trigger stimuli, nor any formal comparison of tactile pro- versus antisaccades against the visually triggered equivalents. If tactile stimuli tend to elicit a weaker prosaccadic tendency than visual stimuli, then one might expect less of a latency cost when comparing antisaccades to prosaccades for tactile stimuli than for visual stimuli. By contrast, if the tendency for slower prosaccades to tactile stimuli than visual stimuli reflects, say, a delay in initial detection of the trigger stimulus, then antisaccades triggered with tactile stimuli might be slower than antisaccades triggered with visual stimuli by a similar amount to the tactile delay found for prosaccades. To investigate such possibilities, we tested pro- and antisaccade performance in response to visual or somatosensory targets in an intermingled experimental design.

Within each block of trials the observers could now expect to perform any of the following four tasks: a prosaccade in response to a somatosensory target, or to a visual target; or an antisaccade in response to a somatosensory target or a visual target. Visual and tactile stimuli were presented at effectively the same external location (see Fig. 2a). The observers placed the thumb of each hand on vibrators that were located at the same location as the LEDs that marked the intended landing points (at 8 degrees on the left or right) for all of the four saccade types tested.

Materials and methods

Observers

Six new observers (five female and one male, aged 18–23 years, mean age 20 years) were recruited from the same subject pool as before. They were again paid £7.50 for their participation, and were naive about the purpose of the experiment.

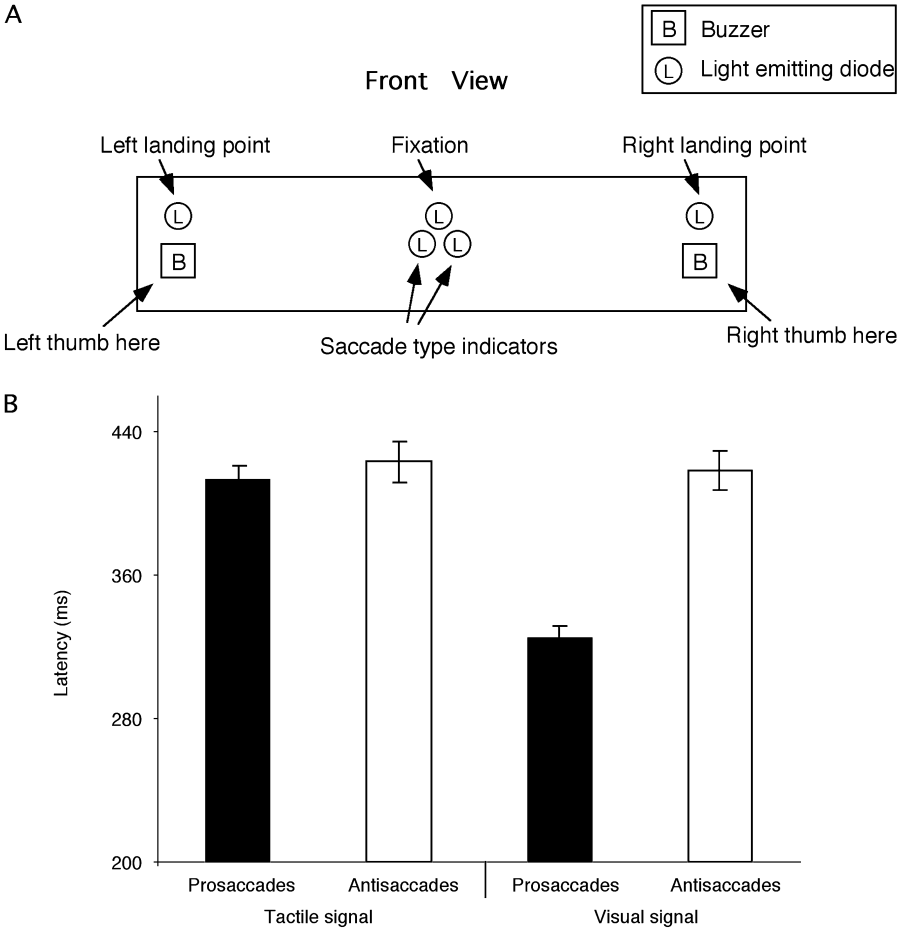

Fig. 2 **A** Schematic of the apparatus used in Experiment 3, with visual or tactile stimulation at equivalent external locations on the left and right. **B** Mean latencies of pro- and antisaccades as a function of whether the peripheral trigger signal for the saccade was visual or tactile, in Experiment 3. The *error bars* denote the standard errors of the mean

Apparatus and procedure

Subjects now binocularly viewed a black panel on which five LEDs, and two Oticon-A bone-conduction vibrators were mounted (see Fig. 2a). The tips of the thumbs of each hand were placed on the vibrators. The basic methodology was similar to Experiment 1, except that on half of the trials within each block the trigger stimulus for the saccade was now an unseen vibration to one or other thumb, rather than the illumination of a peripheral LED. When vibrator stimulation was switched on via the parallel port of the PC, a signal generator induced a 200-Hz sine wave signal (duration 500 ms). This signal was amplified and transmitted to the two bone conductors serving as vibrators.

As in Experiment 1, two LEDs placed 1.5 degrees immediately below the fixation point indicated symbolically whether a prosaccade or an antisaccade was to be made. They were switched on 1,200–1,700 ms before the saccade target was presented. If the red central LED was switched on the subjects were required to make a prosaccade towards the next peripheral stimulus (be this tactile or visual); while if the central green LED was switched on they had to make an antisaccade in the direction opposite to the peripheral stimulus. To mask any sounds from the vibrators (in order to prevent auditory signalling of the timing of the vibration), subjects wore headphones throughout each block that emitted continuous white noise supplied by a white noise generator, effectively masking any sound made by the vibrators. Visual or tactile trigger stimuli were intermingled in a random sequence, as was the requirement to make a pro- or antisaccade. Each subject participated in 4 blocks of 80 trials each.

Results and discussion

Figure 2b plots the mean latencies across subjects. The most striking result from this experiment is that there is a much smaller (now non-significant) latency difference between tactually cued pro- and antisaccades, than for the visually cued pro- and antisaccades, which showed the usual standard substantial difference. An initial repeated measures ANOVA analysis revealed significant main effects on latencies of saccade type (pro- or anti-; $F_{(1,5)}$=4.37, p<.01) and of cue type (visual or tactile; $F_{(1,5)}$=7.28, p<.01) and also a significant interaction between the two ($F_{(1,5)}$=2.77, p<.01). The ANOVA analyses were followed up with post hoc tests (see below).

For saccades after a visual trigger, the results replicate many previous observations. Antisaccade latency was substantially slower than the latency of visual prosaccades (means of 418.5 ms and 324.7 ms respectively, t_5=6.97, p<0.005). The error rates (saccades initially made in the wrong direction) were marginally higher for the anti-saccades than the prosaccades (11.7% and 10.9% respectively) but this small difference was not statistically significant (t_5=0.69, n.s.).

The pattern was qualitatively different for saccades following a somatosensory trigger stimulus. There was only a small numerical difference between the latencies of the haptically cued prosaccades (mean of 416.2 ms) and antisaccades (mean of 423.6 ms), which did not reach conventional significance levels (t_5=1.24, p>.4). The error rates were also quite similar for the two types of saccades when cued by the somatosensory stimulus (14.6% and 15.6% for pro- and antisaccades respectively (t_5 = 0.56, n.s.).

The peak velocities for the visual prosaccades were 391°/s while for the visually cued antisaccades they were 284°/s. For the somatosensory saccades the peak velocities were 312°/s and 275°/s for the pro- and antisaccades respectively. The differences in peak velocities were statistically significant within the visually cued saccades ($F_{(1,5)}$=2.18, p<.01), but not for the tactually cued ones ($F_{(1,5)}$=1.38, p>.1). Finally, the standard deviation of landing points was lowest for the visual prosaccades (standard deviation 0.29°), for the visual antisaccades it was 0.68°; while for the tactile saccades the landing point standard deviations were 0.73° and 0.91° for the pro- and antisaccades respectively. For these landing point deviations the main effect of pro- versus antisaccades was significant ($F_{(1,5)}$=5.64; p<.01) as was the main effect of visual versus tactile saccades ($F_{(1,5)}$=4.12, p<.05), but the interaction was not significant ($F_{(1,5)}$=1.56, n.s.).

The exact latency for somatosensory saccades will presumably differ somewhat for different tactile stimuli (e.g. tactile prosaccades might have been somewhat faster and/or less variable in landing point if a more salient tactile stimulus was employed). But the present results nevertheless provide an illustrative case where slowing of prosaccades (here by using tactile rather than visual trigger stimuli) can lead to less of a latency cost for antisaccades in comparison with prosaccades. This general outcome, whereby delaying prosaccades either helps antisaccades (as in Experiment 1) or leads to less of a cost for making an antisaccade (as in Experiment 3) is of course consistent with the notion of competing pathways for more 'reflexive' prosaccades (as for visual trigger stimuli) versus more 'controlled' antisaccades (e.g. Forbes and Klein 1996; Kristjánsson et al. 2001; Schlag-Rey et al. 1997; Everling et al. 1998; Everling and Fischer 1998; Rafal et al. 2000), such that antisaccades are delayed (in comparison with prosaccade latencies to the same trigger stimulus) whenever a strong prosaccadic tendency must be overcome (as for visual trigger stimuli).

In the final two experiments here, we sought any exceptions to this, which might arise in cases where prosaccades are speeded or delayed by factors influencing a pathway that is *common* to both pro- and antisaccades, rather than by influencing pathways specific to prosaccades that then compete with antisaccade generation.

Experiment 4: saccades triggered by visual stimuli with different contrast levels

Doma and Hallett (1988a, 1988b; see also Reuter-Lorenz et al. 1991; Groh and Sparks 1996a, 1996b, 1996c) found that prosaccades tend to be faster for brighter visual targets (i.e. with higher contrast against the dark surrounding background they used). Doma and Hallett (1988a) further showed that while the typical finding of slower and less accurate antisaccades than prosaccades is found under photopic luminance levels, at scotopic luminance levels this difference can disappear (which was mainly due to the fact that prosaccades now became slower and less accurate

in their study, similar to the tactile versus visual prosaccades in Experiment 3 here).

In our next experiment we examined the latencies of pro- and antisaccades across a fairly wide range of different contrast values for visual trigger stimuli. We reasoned that changes in stimulus contrast which alter the salience of a visual target for pathways involved specifically in prosaccadic tendencies might have opposite effects on prosaccade versus antisaccade latencies (i.e. leading to *faster* antisaccades when prosaccades are delayed by reducing the contrast value of the trigger stimulus). This could happen across a certain contrast range where stimulus salience for prosaccades is affected, rather than stimulus detectability per se, reflecting competition between separate pathways for pro- and antisaccades as previously discussed. However, once contrast is reduced to the level where the visual trigger stimulus now becomes fairly hard to detect, then both pro- and antisaccades might start to become slower together, due to a common pathway (e.g. initial sensory registration of the visual trigger stimulus), then becoming the rate-limiting factor.

If so, then as stimulus contrast is varied over intermediate levels, then antisaccades might become faster whenever prosaccades become slower; whereas at very low levels of contrast, both types of saccade should become slower (as in Doma and Hallett 1988).

Materials and methods

Observers

The six new naive observers (three female and three male, aged 19–30 years, mean age 24.1 years) were drawn from the same pool as before, and were again paid £7.50 for participation.

Stimuli and procedure

Visual stimuli were now presented on a cathode ray tube screen, rather than with LEDs, so that contrast levels could be controlled more precisely. Subjects made prosaccades towards, or antisaccades away from, a displaced fixation cross appearing on a 75-Hz CRT screen with 8-bit color resolution (as in Kristjánsson et al. 2001). Stimulus presentation was controlled by an Apple Macintosh G3 computer using the Vision Shell programming package (see http://www.visionshell.com). The saccade target appearance was triggered by a pulse from the parallel port of the Pentium PC that recorded the eye movements, to the Apple G3 computer through a voltage changing electrical circuit. This was done to synchronize the initiation of eye movement recording on one computer with the presentation of stimuli by the other. Eye movement recording was otherwise similar to that described for the previous experiments.

The saccade target was now presented by abruptly shifting the central fixation cross (size: 0.4 degrees) to a peripheral location (8 degrees at the viewing distance of 30 cm), to the right or left of the initial location. Four different contrast levels were chosen for the peripheral presentations of the cross, following some pilot work on its detectability at different levels (at the lowest level used in the experiment, the peripheral cross could still be reliably detected, but typically only just). The luminance of the screen background was 3.34 cd/m^2, and the four contrast levels between the achromatic peripheral target cross and background were: .133, .324, .475 and .642. The fixation cross that was presented at the centre of the screen at the start of each trial was either red (13.8 cd/m^2) or green (12.4 cd/m^2), determined randomly from one trial to the next. The color of the fixation cross denoted (in a manner counterbalanced across subjects) whether a pro- or antisaccade was to be made when the central fixation cross disappeared and the peripheral saccade target appeared (i.e. when the cross abruptly changed position). Twelve hundred to 1,800 ms after the initial fixation cross appeared centrally, the peripheral saccade target was presented, while the central fixation cross was switched off at the same time. Each observer took part in 4 blocks of 80 trials. The contrast level for the peripheral cross on each trial was determined randomly from one trial to the next.

Results and discussion

The mean saccadic latency results from this fourth experiment are shown in Fig. 3. These results show that, as the contrast of the peripheral trigger stimulus decreased, prosaccades became progressively slower, and were markedly slow at the lowest contrast level, for which the peripheral target cross appeared very dim against the dark background. This is consistent with previous research (e.g. Doma and Hallett 1988a, 1988b). The more novel observation is that at the three higher contrast levels used here, antisaccades, unlike prosaccades, tended to become progressively *faster* as the contrast of the peripheral trigger stimulus was lowered (and as prosaccade latencies accordingly became slower). The pattern at these three higher contrast levels is thus consistent with the general rule of antisaccades becoming faster when prosaccades become slower (see also Experiment 1). However, at the lowest contrast level used here, we found an exception to this general pattern, with *both* pro- and antisaccades now being slowed substantially together, presumably because a common pathway (most likely initial registration of the trigger stimulus) now becomes rate-limiting.

This pattern was confirmed by statistical analysis. A 2×4 within-subjects ANOVA [with factors of saccade type (pro or anti) and contrast (4 levels)] unsurprisingly revealed main effects of saccade-type ($F_{(1,5)}$=132.8, p<.001) and of contrast level ($F_{(3,15)}$=32.3, p<0.001) on the latencies. More importantly, the interaction between saccade type and contrast was also significant ($F_{(3,15)}$=17.37, p<.001). Further post hoc regression analyses were conducted to investigate the source of this interaction. These revealed a significant linear trend for the *pro*saccade latencies as a function of contrast ($F_{(1,22)}$=55,39, p<.0001), with faster prosaccades as contrast level was increased. For *anti*saccade latencies, there was, on the other hand, a significant *quadratic* trend for latencies as a function of contrast ($F_{(2,21)}$=14.37, p<.0001), due to the substantial slowing down at the very lowest contrast level, but with antisaccade latencies otherwise tending to increase as contrast was increased (see antisaccade results for the three higher contrast levels in Fig. 3), i.e. at .324, .475 and .642. Indeed, when only the three higher contrast levels were considered (i.e. now excluding the exceptional result for the lowest contrast

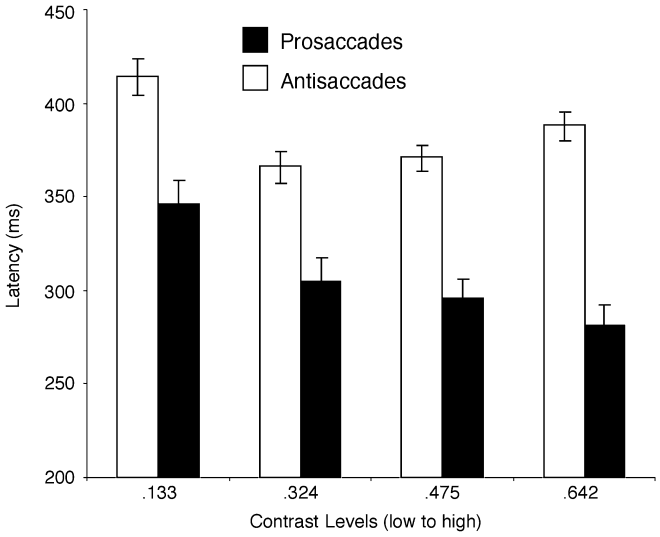

Fig. 3 Mean latencies of pro- and antisaccades as a function of the four different contrast levels for the peripheral saccade targets used in Experiment 4. Note that while both types of saccades were slowest at the lowest contrast level used (*leftmost pair of bars*), for the three other contrast levels prosaccades tend to become faster as stimulus contrast rises, while the opposite pattern applies for antisaccades. The *error bars* denote the standard errors of the mean

used), there was a significant *linear* trend for the antisaccade data against contrast ($F_{(1,16)}$=7.58, p<0.02), with latencies *increasing* reliably with increasing contrast, the opposite result to that found for prosaccades.

The results for the three higher contrast levels thus confirm the inherently competitive relationship between pro- and antisaccades, with the latter tending to become faster as the former become slower. But the present results also identify one exception; at a sufficiently low contrast level, both types of saccade now become slowed together, most likely as a result of the initial registration of the trigger stimulus becoming slower and now becoming a common rate-limiting factor.

Table 1 summarizes the error rates, peak velocities and landing point error for the four contrast levels as a function of saccade type. The directional error rates were significantly higher for the antisaccades versus the prosaccades, while the opposite was the case for the peak velocities (Fs=3.0 or higher, p<.05). None of the effects of contrast on error rates, peak velocities and landing error was significant, nor the interactions between contrast and saccade type (ps>.05).

Experiment 5: alerting effect of warning sound on visual prosaccades and antisaccades

Our final experiment in this paper sought to determine whether another factor might have *common* effects on both pro- and antisaccades (as for the lowest contrast value, but not the three higher contrast values, in the preceding experiment), namely, the presence of a warning sound to produce nonspatial alerting.

A central warning sound is capable of speeding reaction times in many different tasks, and is thought to have this general effect primarily by means of nonspatial alerting or arousal (e.g. see Posner 1978; Posner and Petersen 1990). Thus if prosaccades to a peripheral visual stimulus following a central warning sound are faster than those on unwarned trials, one likely explanation for this effect would be in terms of *nonspatial* alerting (rather than specifically in terms of any *spatial* influence that might enhance the prosaccadic tendency, as might apply instead if a sound appears at the same peripheral location as a concurrent visual target, e.g. see Harrington and Peck 1998; Ross and Ross 1981; Stein and Meredith 1993; Tassinari and Campara 1996). If a central warning sound can speed prosaccades in a manner that does not reflect a specifically spatial influence on prosaccade pathways, then it might speed antisaccades in a similar manner, rather than producing the opposite pattern for antisaccades as happens when competing pathways are influenced rather than a common pathway.

Much research has been conducted on the so-called "gap-effect", whereby prosaccade latencies are faster if a central visual fixation stimulus is extinguished shortly before the peripheral trigger stimulus appears (e.g. see Fischer and Boch 1983; Fischer and Weber 1993; Ross and Ross 1980, 1981; Saslow 1967). Although this gap-effect may sometimes involve a nonspatial warning effect as one contributory component (see Taylor et al. 1999), the usual gap effect is also known to involve other influences, such as a tendency to keep the eye on the currently fixated stimulus when present, which competes with the tendency for prosaccades to any peripheral event (Edelman and Keller 1996, 1998; Fischer and Boch 1983; Fischer and

Table 1 Direction errors, peak velocities and landing point error from Experiment 4 as a function of saccade type and contrast level

Saccade type	Contrast levels	Direction errors (% of trials)	Peak velocities (°/s)	Landing point error (degrees; SD)
Prosaccades	.133	4.9	344	.78
	.324	2.5	382	.65
	.475	3.7	376	.61
	.642	2.4	374	.59
Antisaccades	.133	8.7	286	1.24
	.324	9.5	301	1.01
	.475	10.7	294	.89
	.642	8.9	315	.91

Weber 1993; Munoz and Wurtz 1995a, 1995b; Sparks et al. 2000); plus a possible role for attention to the fixated stimulus (Fischer and Breitmeyer 1987). Thus, while gap-effects can involve temporal warning influences, in their prototypical form they may not provide a pure measure of this temporal effect alone. So, although it is now well known that antisaccades as well as prosaccades can be speeded by the offset of a currently fixated stimulus in the gap paradigm (e.g. Reingold and Stampe 2002; Fischer and Weber 1992; Craig et al. 1999), albeit sometimes less strongly than for prosaccades (Reuter-Lorenz et al. 1995), the role of *temporal warning* alone in such results remains uncertain. Accordingly, in our final experiment we focused on a relatively pure manipulation of just temporal warning, without manipulating the presence or absence of visual stimulation at fixation.

Subjects performed pro- or antisaccades triggered by a peripheral visual stimulus, while unpredictably either a brief central sound preceded the visual trigger stimulus (to produce nonspatial alerting), or *no sound* was presented during the trial. We expected that a preceding auditory warning sound should speed prosaccades, via the well-known nonspatial alerting effect (Posner 1978). The new question was whether antisaccades would likewise be speeded by the warning sound (as expected if this affects an alerting pathway common to both types of saccade), or would instead have the opposite effect on antisaccades (as expected if the warning sound were to specifically facilitate a prosaccadic pathway that competes with antisaccades).

Materials and methods

Observers

Six new naive observers (four female and two male, aged 19–27 years, mean age 23.9 years) were selected from the same subject pool as before, and received £7.50 for their participation.

Stimuli and procedure

The observers were again required to make either prosaccades towards a peripheral visual target, or antisaccades of the same amplitude in the direction opposite to the peripheral target. Unpredictably, a central auditory stimulus either preceded the onset of the peripheral visual target by 200 ms, or no sound was presented during the trial. Whether a central auditory stimulus preceded the saccade target or not was determined randomly from one trial to the next. The observers were instructed to completely ignore the auditory stimulus, as their only task was to perform visually triggered saccades. As in Experiment 4 the colour of the central fixation point indicated whether a pro- or antisaccade was to be made on a trial-by-trial basis (see "Materials and methods" for Experiment 4 for further details). The type of saccade required was thus intermingled within blocks. The mapping of colour type to saccade type was counterbalanced across the different observers. The required saccade type (pro- or anti-) was randomly determined for each trial.

Each trial started with a red or green fixation cross (the colour signalling the required saccade type) at the centre of a CRT display screen (controlled by an Apple Macintosh G3 computer) on a dark grey background (3.34 cd/m^2). Twelve hundred to 1,800 ms after the onset of the fixation cross it disappeared, followed immediately by the peripheral saccade target (a white cross) presented 8 degrees to the right or left of fixation (at a viewing distance of 50 cm). If an auditory warning sound preceded the target, this was presented using two external speakers conjointly (situated to the immediate right and left of the display screen) so that the apparent spatial sound-source was central. The warning sound was a 100-ms approximately sinusoidal middle-C, generated by the Apple G3 PC. This sound was clearly audible and alerting without being uncomfortable [77 dB(A)]. Each observer participated in 4 blocks of 100 trials. Eye movement recording was implemented as before. In all other respects methods were similar to the previous experiments.

Results and discussion

Figure 4 plots the mean saccade latencies for the six observers, with standard errors shown. The clear finding is that when a central alerting sound preceded the peripheral visual trigger stimulus for the saccade, mean saccadic latencies were speeded up. This was the case for both pro- and antisaccades, as compared with those trials without advance auditory warning. Mean prosaccade latencies were 191.96 ms with warning sound, versus 213.5 ms without the warning sound. A similar result was found for antisaccades, again with faster latencies on auditorily warned versus unwarned trials (226.6 ms with warning sound versus 251.3 without). Indeed, the warning effect and the effects of pro- versus antisaccades were found to be additive rather than interactive, as revealed with a repeated measures ANOVA. The main effect of saccade type on latency was reliable ($F_{(1,5)}$=65.17, p<.001) as was the main effect of auditory warning ($F_{(1,5)}$=35.88, p=.002), while the interaction between these two factors was not close to being significant ($F_{(1,5)}$=.139, p=.72), showing

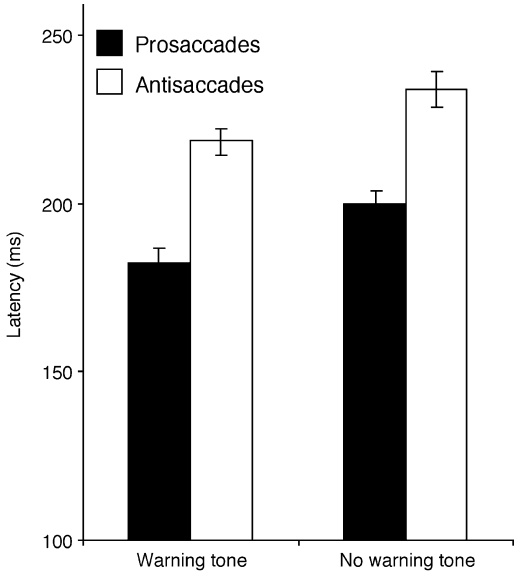

Fig. 4 Mean latencies of pro- and antisaccades across the six observers in Experiment 5, as a function of whether an alerting sound preceded (by 200 ms) the visual signal that triggered the saccade; unwarned trials had no such sound at any point. The *error bars* denote the standard errors of the mean

that the benefit of auditory warning was constant across pro- and antisaccades.

For prosaccades the directional error-rates were 4.2% when no sound was presented, and 4.3% with auditory warning. For the antisaccades these directional error rates were 7.6% and 8.4% respectively. The main effect of saccade type on these error rates was significant ($F_{(1,5)}$=55,5, p<.001), but there was no effect of auditory warning, and no interaction (both p values >.169). The mean peak velocities for prosaccades were 308.81°/s with auditory warning and 314.82°/s without. For antisaccades these peak velocities averaged 271.05°/s and 269.35°/s respectively. Peak velocities were thus significantly higher for pro- than antisaccades ($F_{(1,5)}$=20.05, p=.007), but auditory warning produced no main effect on peak velocities, or interaction with the effect of saccade type (both ps>.4). The standard deviations of the landing points were also unaffected by auditory warning, but were higher for antisaccades (mean 1.12°) than for prosaccades (mean .68°), which is common when no landing point is visible for antisaccades as in this particular experiment (see also Kristjánsson et al. 2001)

The critical new result of this experiment is that auditory warning signals can speed up both pro- and antisaccades in a similar manner, suggesting that the auditory warning influences a common pathway for the two types of saccade, rather than producing a specific benefit for prosaccades that then competes with antisaccade generation.

General discussion

In the experiments presented here we compared pro- and anti-saccades under a variety of different conditions, to examine the generality of previous findings that antisaccades may be speeded by factors that delay prosaccades and vice versa. Such an outcome, when observed, suggests competing pathways for pro- versus anti-saccades, so that antisaccades are slowed whenever a stronger prosaccadic tendency must first be overcome.

Such competing pathways have been previously been proposed on the basis of neuropsychological and neurophysiological findings (e.g. Cornelissen et al. 2002; Everling et al. 1998, 1999; Everling and Munoz 2000; Funahashi et al. 1993; Grosbras et al. 2001; Gottlieb and Goldberg 1999; Guitton et al. 1985; Schlag-Rey et al. 1997; Zhang and Barash 2000), invoking suggestions that 'reflexive' prosaccades may be triggered primarily by tectal pathways involving the superior colliculus (Dorris et al. 1997; Schall and Thompson 1999; Sparks and Barton 1993), whereas voluntary antisaccades may involve cortical modulation from structures such as the frontal eye fields (Chen and Wise 1995; Bruce and Goldberg 1985; Everling et al. 1997, 1999; Everling and Munoz 2000; Funahashi et al. 1993; Schall 1991; Schlag-Rey et al. 1997). Here we found several circumstances where manipulations that delayed prosaccades did speed up antisaccades, but we also found several exceptions to this.

Experiment 1 examined saccades triggered by peripheral stimulation in the nasal or temporal hemifield, under monocular viewing. Replicating previous studies of prosaccades (e.g. Rafal et al. 1991, 2000), we found that prosaccadic latencies were faster for stimulation in the temporal hemifield. As previously suggested (see, e.g. Rafal et al. 1991) this might in principle relate to asymmetries in pathways leading to the colliculus (although see also Sumner et al. 2002). The critical new result here was that the opposite pattern was found for antisaccades, which were faster following nasal than temporal stimulation. This supports the notion of competing pathways, whereby antisaccades are slower when the competing prosaccadic tendency is stronger, as following temporal stimulation. It also shows that stimulation in the temporal hemifield does not speed all types of oculomotor responses (as might have been the case if, say, stimulation in the temporal hemifield was simply detected quicker than in the nasal hemifield). Furthermore, a control study (Experiment 2) confirmed that these results are specific to *peripheral* trigger stimulation in the temporal versus nasal hemifield; no temporal/nasal difference is found when saccading towards a tonically present placemarker in either hemifield, following a central symbolic cue regarding the required saccade direction.

In Experiment 3, we compared pro- and antisaccade performance for visual versus tactile trigger stimuli. Tactile triggers led to a smaller difference between anti- versus prosaccades than did visual triggers, primarily due to slower prosaccades for tactile targets. This provides a further example that slowing prosaccades can lead to reduced costs for antisaccades. Although tactile pro- and antisaccades did not themselves differ much in latency, which might have led to suggestions that both were in fact equally 'endogenous', the landing-point deviations were in fact smaller for tactile pro- than antisaccades, suggesting that the tactile prosaccades may have had some 'exogenous' component.

Experiment 4 returned to considering just visually triggered saccades, now comparing pro- and antisaccades as a function of the contrast level of the peripheral visual stimulus. Across the three higher contrast levels used, prosaccades got increasingly slower as contrast was reduced, whereas antisaccades showed the opposite effect of decreasing in latency as contrast was reduced over these three levels. The opposing outcome for pro- and antisaccades across this contrast range is again consistent with the notion of competing pathways, and with the prosaccadic tendency weakening as the salience of the peripheral stimulus for prosaccadic pathways is reduced, so that less time is then required to overcome the prosaccadic urge when an antisaccade is required instead. However, at the very lowest contrast level used, both types of saccades were slowed considerably together, demonstrating that a common effect can arise when (presumably) a common pathway is affected. Here it seems likely that stimulus detection became a rate-limiting step at the lowest contrast, thus delaying both types of saccade.

Finally, Experiment 5 demonstrated another case in which pro- and anti-saccades can be slowed or speeded together. A warning tone that preceded the saccade trigger stimulus speeded up both types of saccade, suggesting an alerting effect on a pathway that is common to both pro- and anti-saccades. This joint facilitation of latencies for both saccade types is reminiscent of the similar effects previously seen in 'gap' paradigms involving offset of the central fixation stimulus (Craig et al. 1999; Edelman and Keller 1996; Fischer and Boch 1983; Fischer and Breitmeyer 1987; Fischer and Weber 1992, 1993; Munoz and Wurtz 1995; Reingold and Stampe 2002; Sparks et al. 2000), although unlike the present warning sound effects, the gap paradigm is thought to involve not just alerting effects, but also changes in the tendency to maintain central fixation.

When taken together, the present experiments provide additional new evidence for proposals that pro- and antisaccades involve competing pathways, while also illustrating that they may nevertheless share some common pathways prior to the stage(s) at which competition over the saccade direction arises. Experiments 1 and 2 confirm that prosaccades but not antisaccades are enhanced for temporal hemifield stimulation versus nasal stimulation (and, indeed, they demonstrate for the first time that this effect is indeed specific to prosaccades). Experiment 3 shows that the difference between pro- and anti-saccades can vary with the modality of stimulation; Experiment 4 shows that for the visual modality, the contrast level of the stimulation can have differential effects on the pro- versus antisaccade relationship over different ranges of contrast levels; while Experiment 5 shows that both types of saccade can benefit from an auditory temporal warning.

While these results establish the general principle of competing versus common pathways for the two types of saccade that can be studied behaviourally, and identify some of the boundary conditions on whether opposite or common outcomes are observed for pro- and anti-saccades, further work is necessary to identify the neural basis of the various effects. As already mentioned, the temporal/nasal asymmetries observed in Experiment 1 may relate to asymmetries in the tectal pathway to the colliculus. However, since this has been questioned for other temporal/nasal asymmetries (Sumner et al. 2002), it would be useful to address the anatomical issue directly in further work, for example by examining antisaccade performance for the two hemifields in patients or in animals with unilateral collicular lesions (e.g. see Sapir et al. 1999, 2002). Turning to consider the neural basis of the pattern in Experiment 3, there has been little work to date on the neural basis of tactually triggered saccades (though see Groh and Sparks 1996a, 1996b, 1996c), but these could in principle be studied with functional imaging as readily as for the visually triggered case (e.g. see Macaluso and Driver 2003, for some initial fMRI results on commonalities and differences between saccadic activations following visual or tactile stimuli). For the effects of contrast in Experiment 4, one possible hypothesis is that prosaccades may show a contrast-response profile similar to that for magnocellular visual channels, given that visual pathways to the colliculus are primarily of the magnocellular type (e.g. Livingstone and Hubel 1988; Marrocco and Li 1977). By contrast, antisaccade performance as a function of contrast might relate to the combination of magno- and parvocellular inputs to cortical structures. Finally, turning to the effects of auditory warning in Experiment 5, there have been some initial imaging studies of such alerting effects, albeit only in non-saccadic tasks to date (Fan et al. 2002), and a role for ascending noradrenergic arousal pathways has been suggested (e.g. Posner and Petersen 1990). Further studies of the effects we have reported here, using a combination of neuroimaging and/or patient studies, should shed light on the neural basis of the patterns we have identified. For now, the present purely behavioural results demonstrate several situations in which antisaccades can be speeded when prosaccades are slowed, but they also demonstrate some important exceptions to this.

Acknowledgements A.K. was supported by a Long Term Fellowship from the Human Frontiers Science Program (Number LT00126/2002-C/2). J.D. was supported by programme grants from the Medical Research Council (UK) and the Wellcome Trust. Thanks are due to Francesco Pavani, Angelo Maravita, Chris Rorden and Steffan Kennett for technical help.

References

Amlot R, Walker R, Driver J, Spence C (2003) Multimodal visual-somatosensory integration in saccade generation. Neuropsychologia 41:1–15

Blanke O, Grüsser O (2001) Saccades guided by somatosensory stimuli. Vision Res 41:2407–2412

Bruce CJ, Goldberg ME (1985) Primate frontal eye fields. I. Single neurons discharging before saccades. J Neurophysiol 53:603–635

Chen LL, Wise SP (1995) Neuronal activity in the supplementary eye field during acquisition of conditional oculomotor associations. J Physiol 73:1101–1121

Cherkasova MV, Manoach DS, Intriligator JM, Barton JJ (2002) Antisaccades and task-switching: interactions in controlled processing. Exp Brain Res 144:528–537

Cornelissen FW, Kimmig H, Schira M, Rutschmann RM, Maguire RP, Broerse A, Den Boer JA, Greenlee MW (2002) Event-related fMRI responses in the human frontal eye fields in a randomized pro- and antisaccade task. Exp Brain Res 145:270–274

Craig GL, Stelmach LB, Tam WJ (1999) Control of reflexive and voluntary saccades in the gap effect. Percept Psychophys 61:935–942

Doma H, Hallett PE (1988a) Rod-cone dependence of saccadic eye-movement latency in a foveating task. Vision Res 28:899–913

Doma H, Hallett PE (1988b) Dependence of saccadic eye-movements on stimulus luminance, and an effect of task. Vision Res 28:915–924

Dorris MC, Pare M, Munoz DP (1997) Neuronal activity in monkey superior colliculus related to the initiation of saccadic eye movements. J Neurosci 17:8566–8579

Edelman JA, Keller EL (1996) Activity of visuomotor burst neurons in the superior colliculus accompanying express saccades. J Neurophysiol 76:908–926

Edelman JA, Keller EL (1998) Dependence on target configuration of express saccade-related activity in the primate superior colliculus. J Neurophysiol 80:1407–1426
Edelman JA, Intriligator J, Barton JJ (2000) Is poor antisaccade performance in human due to the absence of a visual target, or to reflex suppression? Soc Neurosci Abstr 25:362.3
Everling S, Fischer B (1998) The antisaccade: a review of basic research and clinical studies. Neuropsychologia 36:885–899
Everling S, Munoz DP (2000) Neuronal correlates for preparatory set associated with pro-saccades and anti-saccades in the primate frontal eye field. J Neurosci 20:387–400
Everling S, Krappmann P, Flohr H (1997) Cortical potentials preceding pro- and antisaccades in man. Electroencephalogr Clin Neurophysiol 102:356–362
Everling S, Dorris MC, Munoz DP (1998) Reflex suppression in the antisaccade task is dependent on prestimulus neural processes. J Neurophysiol 80:1584–1589
Everling S, Dorris MC, Klein RM, Munoz DP (1999) Role of primate superior colliculus in preparation and execution of anti-saccades and pro-saccades. J Neurosci 19:2740–2754
Fan J, McCandliss BD, Sommer T, Raz A, Posner MI (2002) Testing the efficiency and independence of attentional networks. J Cogn Neurosci 14:340–347
Fischer B, Boch R (1983) Saccadic eye movements after extremely short reaction times in the monkey. Brain Res 754:285–297
Fischer B, Breitmeyer B (1987) Mechanisms of visual attention revealed by saccadic eye movements. Neuropsychologia 25:73–83
Fischer B, Weber H (1992) Characteristics of "anti" saccades in man. Exp Brain Res 89:415–424
Fischer B, Weber H (1993) Express saccades and visual attention. Behav Brain Sci 16:553–567
Forbes K, Klein RM (1996) The magnitude of the fixation offset effect with endogenously and exogenously controlled saccades. J Cogn Neurosci 8:344–352
Funahashi S, Chafee MV, Goldman-Rakic PS (1993) Prefrontal neuronal activity in rhesus monkeys performing a delayed antisaccade task. Nature 365:753–756
Goldberg ME (2000) The control of gaze. In: Kandel E, Schwartz JH, Jessel TM (eds) Principles of neural science, 4th edn. McGraw-Hill, New York, pp 782–800
Gottlieb J, Goldberg ME (1999) Activity of neurons in the lateral intraparietal area of the monkey during an antisaccade task. Nat Neurosci 2:906–912
Groh JM, Sparks DL (1996a) Saccades to somatosensory targets. I. Behavioral characteristics. J Neurophysiol 75:412–427
Groh JM, Sparks DL (1996b) Saccades to somatosensory targets. II. Motor convergence in primate superior colliculus. J Neurophysiol 75:428–438
Groh JM, Sparks DL (1996c) Saccades to somatosensory targets. III. Eye-position-dependent somatosensory activity in primate superior colliculus. J Neurophysiol 75:439–453
Grosbras MH, Leonards U, Lobel E, Poline JB, LeBihan D, Berthoz A (2001) Human cortical networks for new and familiar sequences of saccades. Cereb Cortex 11:936–945
Guitton D, Buchtel HA, Douglas RM (1985) Frontal lobe lesions in man cause difficulties in suppressing reflexive glances and in generating goal directed saccades. Exp Brain Res 58:455–472
Hallett PE (1978) Primary and secondary saccades to goals defined by instructions. Vision Res 18:1279–1296
Hallett PE, Adams BD (1980) The predictability of saccadic latency in a novel voluntary oculomotor task. Vision Res 20:329–339
Harrington LK, Peck CK (1998) Spatial disparity affects visual-auditory interactions in human sensorimotor processing. Exp Brain Res 122:247–252
Krappmann P, Everling S, Flohr H (1998) Accuracy of visually and memory-guided antisaccades in man. Vision Res 38:2979–2985
Kristjánsson Á, Chen Y, Nakayama K (2001) Less attention is more in the preparation of antisaccades, but not prosaccades. Nat Neurosci 4:1037–1042
Leigh RJ, Zee DS (1999) The neurology of eye movements, 3rd edn. Oxford University Press, Oxford
Livingstone M, Hubel D (1988) Segregation of form, color, movement, and depth: anatomy, physiology, and perception. Science 240:740–749
Macaluso E, Driver J (2003) Multimodal spatial representations in human parietal cortex: evidence from functional imaging. In: Siegel AM, Andersen RA, Freund H, Spencer DD (eds) Advances in neurology: the parietal lobe. LWW, Philadelphia
Marrocco RT, Li RH (1977) Monkey superior colliculus: properties of single cells and their afferent inputs. J Neurophysiol 40:844–860
Munoz DP, Wurtz RH (1995a) Saccade-related activity in monkey superior colliculus. I. Characteristics of burst and build-up cells. J Neurophysiol 73:2313–2333
Munoz DP, Wurtz RH (1995b) Saccade-related activity in monkey superior colliculus. II. Spread of activity during saccades. J Neurophysiol 73:2334–2348
Neggers SWF, Bekkering H (1999) Integration of visual and somatosensory target information in goal-directed eye and arm movements. Exp Brain Res 125:97–107
Posner MI (1978) Chronometric explorations of mind. Erlbaum, Hillsdale
Posner MI, Cohen Y (1984) Attention and the control of movements. In: Stelmach GE, Requin J (eds) Tutorials in motor behaviour. North Holland, Amsterdam, pp 243–258
Posner MI, Petersen SE (1990) The attention system of the human brain. Annu Rev Neurosci 13:25–42
Rafal R (2002) Cortical control of visuomotor reflexes. In: Stuss DT, Knight RT (eds) Principles of frontal lobe function. Oxford University Press, Oxford, pp 149–158
Rafal R, Henik A, Smith J (1991) Extrageniculate contributions to reflex visual orienting in normal humans: a temporal hemifield advantage. J Cogn Neurosci 3:322–328
Rafal R, Machado L, Ro T, Ingle H (2000) Looking forward to looking: saccade preparation and control of the visual grasp reflex. In: Monsell S, Driver J (eds) Control of cognitive processes: attention and performance XX. MIT Press, Cambridge, USA, pp 155–174
Reingold EM, Stampe DM (2002) Saccadic inhibition in voluntary and reflexive saccades. J Cogn Neurosci 14:371–388
Reuter-Lorenz PA, Hughes HC, Fendrich R (1991) The reduction of saccadic latency by prior offset of the fixation point: an analysis of the gap effect. Percept Psychophys 49:167–175
Ross LE, Ross SM (1980) Saccade latency and warning signals: stimulus onset, offset, and change as warning events. Percept Psychophys 27:251–257
Ross SM, Ross LE (1981) Saccade latency and warning signals: effects of auditory and visual stimulus onset and offset. Percept Psychophys 29:429–437
Sapir A, Soroker N, Berger A, Henik A (1999) Inhibition of return in spatial attention: direct evidence for collicular generation. Nat Neurosci 2:1053–1054
Sapir A, Rafal R, Henik A (2002) Attending to the thalamus: inhibition of return and nasal-temporal asymmetry in the pulvinar. Neuroreport 13:693–697
Saslow MG (1967) Effects of components of displacement-step upon latency for saccadic eye movements. J Opt Soc Am A Opt Image Sci Vis 57:1024–1029
Schall JD (1991) Neuronal activity related to visually guided saccadic eye movements in the supplementary motor area of rhesus monkeys. J Neurophysiol 66:530–558
Schall JD, Thompson KG (1999) Neural selection and control of visually guided eye movements. Annu Rev Neurosci 22:241–259
Schlag-Rey M, Amador N, Sanchez H, Schlag J (1997) Antisaccade performance predicted by neuronal activity in the supplementary eye field. Nature 390:398–401
Shulman GL (1984) An asymmetry in the control of eye movements and shifts of attention. Acta Psychol (Amst) 55:53–69
Sparks DL, Barton EJ (1993) Neural control of saccadic eye movements. Curr Opin Neurobiol 3:966–972

Sparks DL, Rohrer WH, Zhang Y (2000) The role of the superior colliculus in saccade initiation: a study of express saccades and the gap effect. Vision Res 40:2763–2777

Stein BE, Meredith MA (1993) The merging of the senses. MIT Press, Cambridge, USA

Sternberg S (1969) The discovery of processing stages: extensions of Donders' method. In: Koster WG (ed) Attention and performance II. North Holland, Amsterdam, pp 276–315

Sumner P, Adamjee T, Mollon JD (2002) Signals invisible to the collicular and magnocellular pathways can capture visual attention. Curr Biol 12:1312–1316

Tassinari G, Campara D (1996) Consequences of covert orienting to non-informative stimuli of different modalities: a unitary mechanism? Neuropsychologia 34:235–245

Taylor TL, Klein RM, Munoz DP (1999) Saccadic performance as a function of the presence and disappearance of auditory and visual fixation stimuli. J Cogn Neurosci 11:206–213

Weber H (1995) Presaccadic processes in the generation of pro and antisaccades in human subjects—a reaction time study. Perception 24:1265–1280

Zambarbieri D, Beltrami G, Versino M (1995) Saccade latency toward auditory targets depends on the relative position of the sound source with respect to the eyes. Vision Res 35:3305–3312

Zhang M, Barash S (2000) Neuronal switching of sensorimotor transformations for antisaccades. Nature 408:971–975