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RESEARCH ARTICLE

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Stimulation in the rostral pole of monkey superior colliculus: effects on vergence eye movements

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Abstract Recent studies have indicated that the superior colliculus (SC), traditionally considered to be saccade-related, may play a role in the coding of eye movements in both direction and depth. Similarly, it has been suggested that omnidirectional pause neurons are not only involved in the initiation of saccades, but can also modulate vergence eye movements. These new developments provide a challenge for current oculomotor models that attempt to describe saccade-vergence coordination and the neural mechanisms that may be involved. In this paper, we have attempted to study these aspects further by investigating the role of the rostral pole of the SC in the control of vergence eye movements. It is well-known that, by applying long-duration electrical stimulation to rostral sites in the monkey SC, saccadic responses can be prevented and interrupted. We have made use of these properties to extend this paradigm to eye movements that contain a substantial depth component. We found that electrical intervention in the rostral region also has a clear effect on vergence. For an eye movement to a near target, stimulation leads to a significant suppression and change in dynamics of the pure vergence response during the period of stimulation, but the depth component cannot be prevented entirely. When these paradigms are implemented for 3D refixations, the saccade is inactivated, as expected, while the vergence component is often suppressed more than in the case of the pure vergence. The data lead us to conclude that the rostral SC, presumably indirectly via connections with the pause neurons, can affect vergence control for both pure vergence and combined 3D responses. Suppression of the depth component is incomplete, in contrast to the directional movement, and is often different in magnitude for 3D refixations and pure vergence responses. The results are discussed in connection with current models for saccade-vergence interaction.

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Introduction

Binocular refixations in 3D space require coordinated saccade-vergence eye movements (Chaturvedi and Van Gisbergen 1998). While it has been widely reported that the dynamics of both vergence and version responses change during 3D refixations (Collewijn et al. 1995; Erkelens et al. 1989; Zee et al. 1992), it is not well understood how the two oculomotor subsystems interact and which neural mechanisms are involved. We have recently found evidence that the superior colliculus (SC), traditionally believed to be involved in the control of saccadic eye movements, may also code depth information (Chaturvedi and Van Gisbergen 1999). Furthermore, other recent studies have provided circumstantial evidence for a collicular involvement in 3D refixations (Bacon et al. 1998; Billitz and Mays 1997; Gnadt and Beyer 1998; Jiang et al. 1996; Mays 1996). Jiang et al. (1996) have reported that stimulation in the rostral region of the cat SC can induce disconjugate eye movements and that the activity of rostral neurons can be modulated by pure vergence.

It has recently been proposed that omnidirectional pause neurons, known to be actively involved in timing aspects of saccade control, are also involved in vergence control. According to one view (Zee et al. 1992), the fact that vergence velocity is enhanced in 3D refixations (facilitated vergence) is due to a distinct fast vergence subsystem which, like saccades, is switched on and off by the pause cells, while slow vergence is considered to be independent of this gating system. Actually, recent findings have suggested that the fast movements in depth that occur in 3D refixations may be due, at least partly, to the ability of the saccadic system to generate unequal saccades in the two eyes (Zhou and King 1998). An alternative view (Mays and Gamlin 1995) suggests that fast vergence occurs because midbrain vergence burst

cells (Mays et al. 1986) are relieved of a sustained weak suppression by the pause neurons during saccades. These models allow for the fact that a pure vergence response is possible even when the pause cells are actively firing, and a slow vergence response is typically observed to continue in 3D refixations after the saccade has been completed. Interestingly, while Mays and Gamlin (1995) have reported that pause-cell activity is not modulated during pure vergence, they did note that stimulation of this region during pure vergence dramatically slows the response down. These aspects do not seem to have been investigated for combined saccade-vergence eye movements. Our interest in this saccade-vergence interaction (see Chaturvedi and Van Gisbergen 1998, 1999) has motivated a study into how the directional and depth components of binocular 3D refixations are affected by electrical intervention at the rostral pole of the superior colliculus.

We have made use of the fact that electrical intervention in the rostral zone of the SC, in the cat and monkey, can be used to prevent conjugate eye movements and interrupt ongoing saccades (Munoz and Wurtz 1993; Paré and Guitton 1994). Since it has been shown that neurons in rostral SC project heavily to the pause-cell region (Buttner-Ennever and Horn 1994), the question arises how the vergence component might be affected during perturbations of the saccade trajectory.

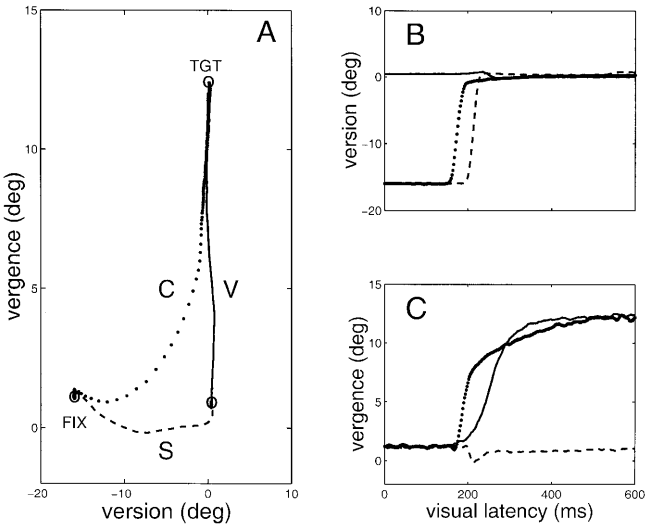

Fig. 1 **A** Typical control-response trajectories in the depth plane. Experimental paradigm was designed to elicit a pure saccade (*S*), a pure vergence response (*V*), and a combined saccade-vergence response (*C*). Note that the combined response to the near target had component amplitudes which were matched to the pure saccade and pure vergence responses that were elicited in isolation. The fixation-point (*FIX*) and visual-target (*TGT*) positions varied in space, depending on the type of response to be elicited. The fixation point for the pure vergence response was an LED in the frontal plane (a far target at roughly 2° vergence), and this point also served as a target for the pure saccade trials. While the combined response can be decomposed into a saccadic and vergence component, note that the 3D refixation was not just a linear addition of S and V. Both the saccade and the 3D response exhibited some degree of transient divergence. **B** Time-course profiles of the saccadic component of the three control responses. **C** Time-course profiles of the vergence component of the three control responses

Materials and Methods

Two adult male Rhesus monkeys (*Macaca mulatta*), trained to accurately fixate and follow visual targets presented in 3D visual space, were prepared for chronical neurophysiological experiments. All surgical procedures and experimental protocols were reviewed and approved by the university committee for the use of experimental animals. Binocular eye position was recorded with a sampling rate of 500 Hz using the double magnetic induction technique (Bour et al. 1984). Individual eye position signals were calibrated by requiring the monkey to binocularly fixate 85 real targets throughout the oculomotor range, up to eccentricities of 35° in the frontal plane. A neural network was trained to map the raw eye position signals onto the known associated target locations (see Melis and Van Gisbergen 1996). The animals were water-deprived before the experiment and received a small liquid reward after each correct trial. A trial was judged to be correct (stimulation and non-stimulation trials) when the monkey fixated the presented target within a specified time.

To record extracellular activity in the SC, a recording chamber was stereotaxically implanted over a trephine hole, such that both colliculi could be reached by microelectrode penetrations. During each session a glass-coated tungsten microelectrode (impedance 0.5–1.2 MΩ), placed inside a stainless steel guide tube, was moved downwards by a hydraulic stepping motor, mounted on the chamber. The deeper layers of the SC were localized based on a number of standard electrophysiological criteria, as described in Melis and Van Gisbergen (1996). While the neural activity observed at each location could be accounted for on the basis of the topographically arranged map of the SC (Robinson 1972), the rostral pole was further characterized by the fact that, upon low-intensity electrical stimulation, large saccades could be prevented or interrupted in midflight. Both monkeys were stimulated at a number of sites (n_I=4; n_{II}=8) in the right rostral SC.

Experiments were performed while the monkey sat, head-fixed, in a primate chair in complete darkness. Light emitting diodes (LEDs), positioned in 3D space, were used as real visual targets to guide eye movements. Target presentation and stimulus timing was computer-controlled. The experimental paradigm was designed to elicit a binocular refixation in direction, in depth or both (see Fig. 1). As shown in the figure, the control saccade (S) and the control vergence refixation (V) that we elicited matched the size of the components of the 3D refixation (C). In the experiments, the size of the saccades ranged between 10–20°, while the convergence responses ranged from roughly 10–15°. Note that, while the pure saccade and pure vergence responses were, respectively, fast and slow eye movements, the 3D refixation was composed of a fast intrasaccadic portion and a slower post-saccadic phase. The monkey (interocular distance about 3.5 cm) was presented with a fixation point (FIX), which had to be fixated for a random time (between 500 and 1500 ms). Directly after FIX offset, a visual target (TGT) was presented at a position nearby (located roughly 10–20 cm from the monkey), in 3D space, which required the monkey to make a visually guided binocular refixation. If a pure vergence response was required, both the FIX and the near target were aligned with the cyclopean eye in the horizontal plane of fixation (i.e., at eye-level). A 3D refixation, with roughly the same vergence component, was elicited by presenting FIX at a more peripheral location. In the experimental sessions, both near and far targets (the latter at roughly 125 cm from the monkey) were randomly interleaved together, although stimulation was only applied for convergence trials.

We stimulated in the right SC and determined the threshold value (10–50 μA) required to suppress a visually guided ipsilateral saccade, using a 500 Hz biphasic pulse train (pulse duration 0.2 ms). In 20% of the trials, a long-duration stimulation pulse train (roughly 450 ms) was applied at different points in time after

target presentation, while the visual target remained on. To minimize predictive behavior, visual-catch trials (where the FIX was extinguished and no target was presented) and stimulation-catch trials (where no target was presented and stimulation was applied at FIX offset) were presented at random times. In the case of interruption, trial stimulation onset was generally triggered by the onset of the saccade.

Results

Long-duration electrical stimulation applied during or before ipsilateral frontal plane saccades was seen to, respectively, interrupt and prevent conjugate responses. The accompanying transient vergence components were observed not to be affected at all. This finding confirms earlier reports that rostral stimulation in monkey SC, during far fixation, does not lead to a vergence effect by itself (Billitz and Mays 1997). This result leads us to the more interesting question of how pure vergence movements and 3D refixations are affected during stimulation.

Figure 2 shows how rostral manipulation can affect the pure convergence response, for stimulation starting prior to movement onset (see prevention panels) and stimulation applied in midflight (see interruption panels). Control responses are shown for the sake of comparison. When stimulation onset occurred before normal visual latencies, the vergence response (in the lower left panel) appeared to be strongly, yet not completely, suppressed during the period of stimulation. After the pulse train ended, the normal response ensued. Note that even the normally present miniature saccadic components were completely suppressed. In the control responses, as well as during stimulation, both eyes moved slowly and symmetrically in opposite directions, rather than one eye staying fixed while the other eye moved (see monocular plots in the upper panels). The interruption panels show that stimulation during an ongoing pure vergence movement halted the movement completely.

In Fig. 3, we see typical examples from the same site for 3D refixations. As expected from frontal-plane studies, stimulation applied prior to the eye movement prevented the saccade from occurring. The vergence response was similarly inhibited, albeit not as completely as the conjugate component. After stimulation ended, the entire 3D refixation manifested itself prominently, with the fast vergence temporally linked to the saccade. When the saccade was interrupted in midflight, however, the vergence was suppressed in a comparable way to that

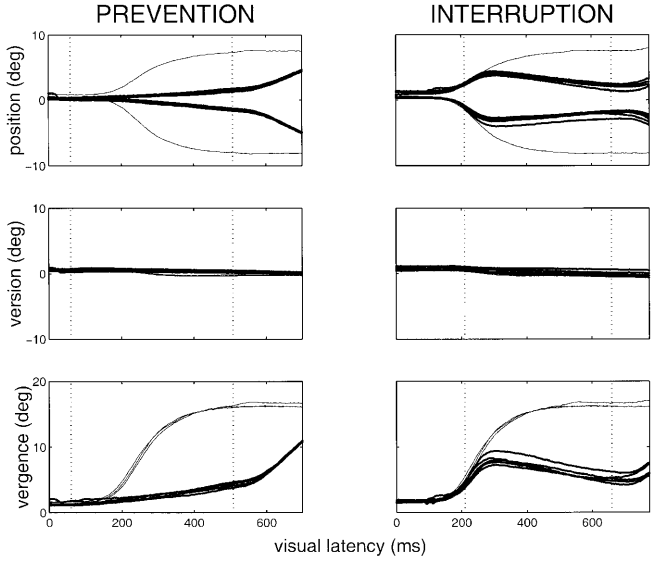

Fig. 2 Typical examples of long-duration stimulation applied to a pure vergence response. *Top panels* depict monocular responses, *middle panels* show version component, and *lower panels* show vergence components. Control responses (*thin lines*) are shown in each panel together with the stimulation-trial results (*bold lines*) from the same session. The *dotted vertical lines* denote stimulation onset and offset, respectively. *Left-hand columns* depict early stimulation starting before visual latency range. It is clear that the directional component was negligible and that the depth requirement was large. Rostral stimulation slowed the vergence response considerably in comparison to the controls. Approximately 50–80 ms after stimulation offset, the vergence component typically became faster again. The monocular plot shows each eye moving equally, but in opposite directions, during the controls. During stimulation, although both eyes moved much more slowly, the monocular contributions remained roughly equal. The visual target stayed on all the time. Data from site II-3

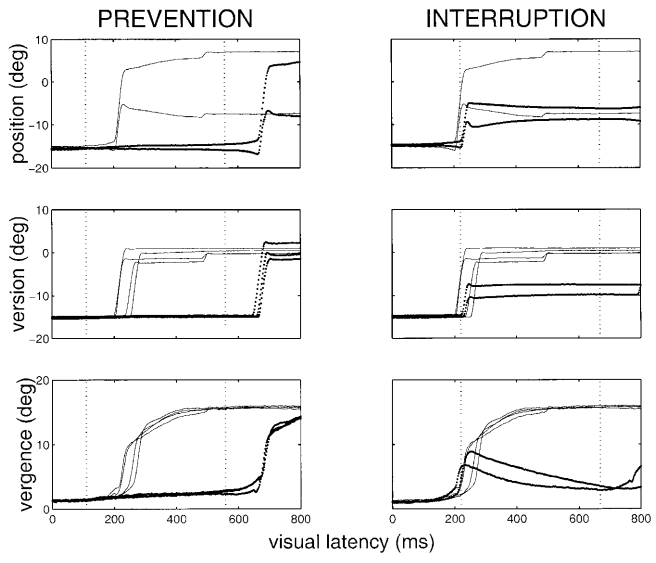

Fig. 3 Typical examples of long-duration stimulation applied to an ipsiversive 3D refixation presented as in Fig. 2. The required saccadic and vergence components are shown in the control traces. In the prevention trials, the saccade was delayed from occurring till after stimulation offset. The vergence was not, however, completely halted. A slow depth response could be observed. This is seen clearly in the *top monocular plots*, where one control trial and one stimulation trial is presented. It is apparent, during stimulation, that some slow vergence still occurred. In other words, rostral stimulation prevented the saccade, but could not completely inactivate the vergence. When, subsequently, the saccade headed to the target, the fast vergence also picked up. In the interruption trials, on the *right*, the saccade was interrupted for the duration of the stimulation train. After that, it continued to target. The fast vergence response was also interrupted. Here, we saw that, during saccade inactivity, it was strongly suppressed. When the saccade continued, so did the vergence. Data from site II-3

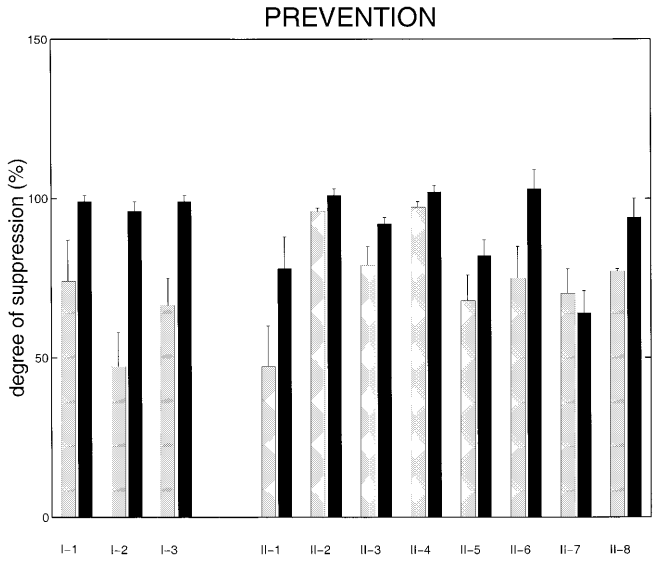

Fig. 4 The *gray columns* in the histogram depict the degree of pure vergence suppression found at each site (denoted in *abscissa*) during prevention trials. The *filled columns* in the histogram depict the degree of vergence suppression in 3D refixations. The average number of trials at a site was 15, with a range of 5–30. A test of significance (*t*-test) showed that the vergence component in the 3D prevention trials was significantly larger ($P<0.05$) than the pure vergence prevention trials for eight of the eleven sites. That some of the error bars of the 3D responses extend slightly beyond the 100% mark is due to the fact that the binocular point of fixation, instead of converging, sometimes diverged slightly from the initial vergence position during the stimulation in the prevention trials. In contrast to the situation in the interruption experiments (see Fig. 5), this effect appears to be rather rare and minute (a few percent). Accordingly, we believe that it probably reflects noisy variations rather than a genuine and systematic divergence effect

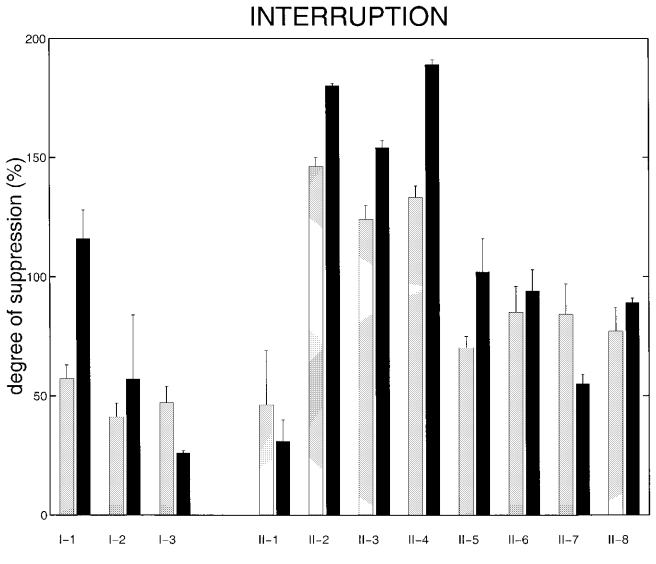

Fig. 5 The *gray columns* in the histogram depict the degree of pure vergence suppression found at each site (denoted in *abscissa*) during interruption trials. The *filled columns* in the histogram depict the degree of vergence suppression in 3D refixations. The average number of trials at a site was 12, with a range of 5–25. A test of significance (*t*-test) suggested that the vergence component in the 3D interruption trials was significantly larger ($P<0.05$) than the pure vergence interruption trials for roughly half the sites. As follows from our definition (see text), suppression values in excess of 100% reflect reversal of the interrupted convergence response as seen, for example, in the lower right-hand panels of Figs. 2 and 3

seen for the pure vergence response in Fig. 2. Note that during this entire time-period, when the vergence response was brought to a standstill and then started to decrease, the saccadic system remained inactive. Once the saccade started, after stimulation ceased, the fast vergence picked up again towards the visual target. In the example shown, the vergence response reverted to its initial starting point. At other sites, we saw the vergence just stop completely or, in some cases, increase slowly, during the period of stimulation.

The histogram in Fig. 4 depicts the degree of suppression seen during prevention, observed for pure vergence responses (gray columns) and for the vergence component of 3D refixations (black columns) for all sites. The vergence change was calculated between stimulation onset and offset. Suppression was defined with respect to the mean vergence change in the control experiments. In other words, 100% suppression would indicate no residual vergence, while 0% suppression would signify that the responses look like the controls. While suppression was found at all sites in the case of pure vergence, its magnitude in the prevention trials varied. It is clear that these graded phenomena stand in stark contrast to the full-blown prevention described above for the saccadic system. Given this contrast, it now becomes interesting to look at the results obtained with combined saccade-vergence responses in 3D space and the effect of stimulation on the vergence component. The 3D prevention data are represented as black bars, next to the pure vergence "prevention" for each respective site. It is clear that, while suppression was complete (roughly 100%) in a number of sites, there were cases where suppression was less. Note, however, that stimulation inhibited the 3D refixation more than in the pure vergence case at the same site, except for site II-7.

The histogram in Fig. 5 similarly depicts the degree of suppression during the interruption paradigm observed for pure vergence responses (gray columns) and 3D refixations (black columns), seen for all sites. The amount of suppression was calculated here as a ratio of what the vergence did in the time-window between 50 ms after stimulation onset until stimulation offset versus how much the response still needed (to reach target) after the stimulation effect began. During this time the saccade was always halted. In the example of Figure 2, we see that the vergence needed to still complete around half of the required amplitude when it got broken off. Instead, due to the stimulation-induced inhibition, it actually reversed itself. In this case, 100% suppression would have indicated that it was laid still (like the saccade) and it therefore still needed to cover half of the response. Actually, it was suppressed more than 100%, as can be seen in the gray column at site II-3. The suppression for the 3D refixation at the same site was even larger. For the in-

terruption paradigm, the degree of suppression was found to vary a lot among sites. Recall that sites with inhibition less than 100% were just slowed down. More than 100% suppression meant that responses were reversed in the direction of divergence (see Figs. 2 and 3, lower righthand panels). While most sites showed larger suppression effects for 3D refixations than pure vergence, sites I-3, II-1, and II-7 showed the opposite effect.

In both sets of histograms, data from site I-4 is not shown since only short-duration stimulation (roughly 50 ms) was applied here. Our qualitative observation here was that a short-duration intervention did not cause as large a suppressing effect as long-duration stimulation did. At sites where the saccade was briefly halted, fast vergence stopped too while the slow phase just continued towards the visual target.

Discussion

Refixations in 3D space often require the synergy of a head movement and a combined saccade-vergence eye movement. In coordinating these various motor systems, the brain needs to ensure that all systems are directed towards a common goal (Chaturvedi and Van Gisbergen 1998). In earlier work (Chaturvedi and Van Gisbergen 1999), we have found evidence that the superior colliculus may be involved in coding both fast vergence and saccadic eye movements. If the view of a 3D colliculus is valid, it becomes easier to see how coordinating these movements might come about as the natural consequence of a joint central representation of the required direction and depth movements. Recently, a neurophysiological study in the cat found evidence for near-response related activity in the rostral pole of the SC (Jiang et al. 1996). This report states that rostral neurons modulate their activity in response to pure vergence eye movements and that electrical stimulation in this region can induce disconjugate responses. These findings suggest that the classical view of the SC as a 2D oculomotor structure may be too limited. The present paper has further pursued this line of investigation by performing stimulation experiments in the rostral pole of the monkey SC for eye movements requiring a large depth component.

Intervention in rostral colliculus affects vergence components

The data presented in this paper show that electrical intervention in the rostral pole of the monkey SC affected not only the saccadic response, as shown in classical "prevention" and "interruption" paradigms applied by Munoz and Wurtz (1993), but also the vergence movement. During pure vergence trials, we have observed that, when stimulation was applied before the range of typical visual latencies, pure vergence responses could still be initiated, although they were much smaller and slower than those seen during control sessions. Stimulation, applied in midflight, was seen to suppress the pure vergence response by dramatically slowing down the ongoing eye movement. In a number of cases, suppression was so intense that the trajectory of the converging response was completely stopped or even reversed for the duration of the stimulation. Similar effects were seen during interruption of the 3D refixations, where suppressed vergence responses could occur when the saccadic system had been briefly prevented or halted.

Dissociation of initiation mechanisms

Behavioral experiments have not yet led to agreement on how well the version and vergence components of 3D binocular refixations are synchronized (for a review, see Enright 1998), but it is clear that there may be a small vergence movement before the saccade (Collewijn et al. 1995; Takagi et al. 1995). The present monkey experiments have shown that the vergence system can start and continue a movement when the saccadic system is completely inactivated by rostral SC stimulation. This shows, therefore, that there can be a degree of dissociation between the initiation and execution mechanisms of these two oculomotor subsystems. A further result is that the pure vergence response, in the prevention paradigm, was somewhat less affected by the stimulation-induced inhibition than the vergence component in a 3D refixation (see Fig. 4). Nevertheless, it is clear that the emergent vergence response was dramatically slowed by the stimulation, in comparison to its normal velocity profile observed during control responses. To provide some background for a discussion on possible mechanisms, we will first briefly review some current ideas on gating of oculomotor subsystems.

Gating of oculomotor subsystems

If we are to understand the processes that govern the cyclical transition from fixation to refixation, a major question is which systems should be engaged or disengaged, to what extent, and at which moment in time when gaze is shifted to a new point in 3D space. The situation for saccades is clear: in this system, pause-cell gating has an all-or-nothing character so that it is either fully engaged (during rapid eye movements) or switched off completely (during fixation). A more complex situation, involving graded suppression effects, is found in the vestibulo-ocular reflex (VOR). This system is active during fixation and is suppressed during large gaze shifts, but becomes active again in the final phase of the head movement. This orchestration allows the VOR to do its job of stabilization when this is useful and suppresses the system at a time when its action would be counterproductive. This behavior of the saccadic system and the VOR is well established and has been incorporated in models of these two subsystems (Guitton and Volle 1987). By contrast,

there is no agreement on how the vergence activation/inactivation process proceeeds and how this is related to the saccade gating process. We shall now attempt to discuss how our results can be related to existing ideas and models on saccade-vergence interactions.

Saccade-vergence models

All-or-nothing gating

Zee et al. (1992) have proposed two types of vergence: saccade-related vergence and slow vergence. In this scheme, both vergence channels have an all-or-nothing gating. Like saccades, saccade-related vergence in the model is gated by omnipause neurons. This distinction between saccade bursters and saccade-related vergence bursters was made at a time when it was thought that burst cells in the pontine reticular formation (Van Gisbergen et al. 1981) code conjugate saccades. Recent work by Zhou and King (1998) has shown, instead, that these cells code monocular saccades, apparently irrespective of whether the refixation occurs in the frontal plane or whether it concerns a rapid refixation in 3D that comes about by combining unequal right eye and left eye saccades. So, in view of these new results, there is now indirect evidence for a pause-cell gated fast depth-movement system. To account for pure vergence and post-saccadic vergence, Zee et al. (1992) assumed the existence of an additional slow-vergence channel. According to their proposal, slow vergence has a separate gating system whose neural basis has not been specified.

In terms of our results, this slow system would be held responsible for the residual slow-vergence response that emerges during attempted 3D refixations that are "prevented" from occurring. Similarly, when rostral-SC stimulation has stopped the saccadic component in midflight completely, there is still a slow vergence response. Thus, while the unequal saccade theory may explain fast vergence, at least partially, one has to assume that the slow-vergence system also plays a role. If the same slow-vergence system generates the pure vergence response, then the Zee et al. (1992) model would have to assume that this channel is also under some degree of omnipause cell control. This idea is a key feature of the model by Mays and Gamlin (1995), which will now be discussed.

Incomplete suppression

One way to interpret the fact that vergence may make a modest start in a 3D refixation task, before saccade onset (Collewijn et al. 1997) or in our prevention paradigm (Fig. 2), is to assume that pause-cell activity blocks the saccadic system completely, but causes only partial suppression of the vergence system (Mays and Gamlin 1995). Recently, Mays and Gamlin (1995) made the observation that pure vergence is slowed down considerably by pause-cell stimulation. They propose a single-channel vergence system, embodied by midbrain vergence burst neurons (Mays et al. 1986), whose gain is under graded pause cell control. Unlike the situation in the saccadic system, the vergence gain varies between full-blown when the pause cells are not firing (during saccades) and a low, but non-zero, default gain when the pause cells are active (fixation, pure vergence). In the sense that the vergence system would not simply be on or off, but could operate at an intermediate gain value, the Mays and Gamlin proposal for the vergence system has some similarity to the operation mode of the VOR (see above). To interpret such an arrangement from a functional point of view, one may note that, if vergence is only partially suppressed during fixation, this will allow the system to correct for small body movements in the depth dimension, just like the VOR can correct for directional disturbances. Since the gain is low, the resulting movements would be slow, allowing the system the potential benefit of sensory feedback. When a refixation in 3D is called for, the saccadic system is enabled when the pause cells cease firing. The resulting release from suppression would unleash the vergence system which, given the very high retinal image velocities during a saccade, could no longer have access to reliable sensory feedback anyway. At the end of the saccade, the pause cell system again switches the vergence system back to a low gain state, thereby allowing the system to operate in closed loop once again in the post-saccadic vergence phase. If pause cells cause partial vergence suppression, then this may also help to explain the phenomenon of vergence-velocity enhancement that occurs when vergence occurs in combination with a saccade as part of a 3D refixation. Our 3D refixation experiments have shown that the rapid vergence response is lost when the rostral pole of the SC is stimulated in midflight. While this model appears to be able to explain how a slow vergence response may emerge during rostral stimulation, it would have a hard time interpreting the Zhou and King (1998) results, which suggest that saccades and fast depth movements are intertwined.

Possible superior colliculus involvement

In earlier work (Chaturvedi and Van Gisbergen 1998, 1999), we have proposed that the SC may participate in the coding of fast vergence responses during saccades. This work did not allow any conclusions to be drawn on the possibility that pure vergence may also be coded at this level (Jiang et al. 1996). Clearly, only single-unit recordings can resolve these issues.

The present study shows that the initiation of vergence and saccades has a certain degree of independence. This is not necessarily an argument against the idea that the SC may be involved in specifying the metrics of both systems. A case in point is the generation of combined eye-head movements. Although it is now generally agreed that the SC codes a desired gaze (eye and head) displacement (Freedman and Sparks 1997), it is

also clear that eye and head do not necessarily start their movements simultaneously (Freedman et al. 1996; Goossens and Van Opstal 1997).

Several behavioral studies of the pure vergence response have suggested a model of vergence control based on a dual-mode concept (see Horng et al. 1998). This work has shown that the initial component in the vergence response to a step in disparity is preprogrammed. This can be demonstrated with the disappearing-step paradigm, where the step stimulus is briefly presented (e.g. 50 ms) and then disappears before the response starts. Under these circumstances, the initial response is quite similar to normal step responses. Since it occurred in total darkness, it must have been open-loop. The late component in the model is a slow, visually guided response. So far, little is known about the neural basis of this preprogrammed response. It would be interesting, in future work, to use the disappearing-step paradigm in the monkey to check whether rostral stimulation can prevent the preprogrammed response completely. If this is the case, it could be argued that the residual slow vergence movement that we saw in prevention and interruption trials for pure vergence may be related to the late visually guided signals.

Conclusion

The present work has shown that rostral SC stimulation, strong enough to block saccades completely, also has effects on pure and saccade-related vergence. Comparison of our results with those of Mays and Gamlin (1995) suggests that our effects were probably mediated by indirect activation of omnipause neurons in the brainstem. Our data provide indirect support, obtained with a different set of paradigms, for their idea that the omnipause neuron gating system for saccades has a graded gain-modulation effect on vergence.

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References

Bacon BA, Villemagne J, Bergeron A, Lepore F, Guillemot J-P (1998) Spatial disparity coding in the superior colliculus of the cat. Exp Brain Res 119:333–344

Billitz MS, Mays LE (1997) Effects of microstimulation of the superior colliculus on vergence and accommodation. Invest Opthalmol Vis Sci 38:S984

Bour LJ, Van Gisbergen JAM, Bruijns J, Ottes FP (1984) The double magnetic induction method for measuring eye movements: results in monkey and man. IEEE Trans Biomed Eng 31:419–427

Buttner-Ennever JA, Horn AKE (1994) Neuroanatomy of saccadic omnipause neurons in nucleus raphe interpositus. In: Fuchs AF, Brandt T, Buttner U, Zee DS (eds) Contemporary ocular motor and vestibular research: a tribute to David A Robinson. Thieme, Stuttgart pp 488–495

Chaturvedi V, Van Gisbergen JAM (1998) Shared target selection for combined version-vergence eye movements. J Neurophysiol 80:849–862

Chaturvedi V, Van Gisbergen JAM (1999) Perturbation of combined saccade-vergence movements by microstimulation in monkey superior colliculus. J Neurophysiol 81:2279–2296

Collewijn H, Erkelens CJ, Steinman RM (1995) Voluntary binocular gaze-shifts in the plane of regard: dynamics of version and vergence. Vision Res 35:3335–3358

Collewijn H, Erkelens CJ, Steinman RM (1997) Trajectories of the human binocular fixation point during conjugate and non-conjugate gaze-shifts. Vision Res 37:1049–1069

Enright JT (1998) Monocularly programmed human saccades during vergence changes? J Physiol 512:235–250

Erkelens CJ, Steinman RM, Collewijn H (1989) Ocular vergence under natural conditions. II. Gaze shifts between real targets differing in distance and direction. Proc R Soc Lond B Biol Sci 236:441–465

Freedman EG, Sparks DL (1997) Activity of cells in the deeper layers of the superior colliculus of the rhesus monkey: evidence for a gaze displacement command. J Neurophysiol 78:1669–1690

Freedman EG, Stanford TR, Sparks DL (1996) Combined eye-head gaze-shifts produced by electrical stimulation of the superior colliculus in rhesus monkeys. J Neurophysiol 76:927–952

Gnadt JW, Beyer J (1998) Eye movements in depth: what does the monkey's parietal cortex tell the superior colliculus. Neuroreport 9:233–238

Goossens HHLM, Van Opstal AJ (1997) Human eye-head coordination in two dimensions under different sensorimotor conditions. Exp Brain Res 114:542–560

Guitton D, Volle M (1987) Gaze control in humans: eye-head coordination during orienting movements to targets within and beyond the oculomotor range. J Neurophysiol 58:427–459

Horng J-L, Semmlow JL, Hung GK, Ciuffreda KJ (1998) Initial component control in disparity vergence: a model-based study. IEEE Trans Biomed Eng 45:249–257

Jiang H, Guitton D, Cullen KE (1996) Near-response-related neural activity in the rostral superior colliculus of the cat. Soc Neurosci Abstr 22:662

Mays LE (1996) Activity of superior colliculus burst neurons during saccade-vergence interactions. Soc Neurosci Abstr 22: 664

Mays LE, Gamlin PDR (1995) A neural mechanism subserving saccade-vergence interactions. In: Findlay J, Walker R, Kentridge RW (eds) Eye movement research: mechanisms, processes and applications. Elsevier, Amsterdam, pp 215–223

Mays LE, Porter JD, Gamlin PDR, Tello CA (1986) Neural control of vergence eye movements: neurons encoding vergence velocity. J Neurophysiol 56:1007–1021

Melis BJM, Van Gisbergen JAM (1996) Short-term adaptation of electrically induced saccades in monkey superior colliculus. J Neurophysiol 76:1744–1758

Munoz DP, Wurtz RH (1993) Fixation cells in monkey superior colliculus. II. Reversible activation and deactivation. J Neurophysiol 70:576–589

Paré M, Guitton D (1994) The fixation area of the cat superior colliculus: effects of electrical stimulation and direct connection with brainstem omnipause neurons. Exp Brain Res 101:109–122

Robinson DA (1972) Eye movements evoked by collicular stimulation in the alert monkey. Vision Res 12:1795–1808

Takagi M, Frohman EM, Zee DS (1995) Gap-overlap effects on latencies of saccades, vergence and combined vergence-saccades in humans. Vision Res 35:3373–3388

Van Gisbergen JAM, Robinson DA, Gielen S (1981) A quantitative analysis of generation of saccadic eye movements by burst neurons. J Neurophysiol 45:417–442

Zee DS, Fitzgibbon EJ, Optican LM (1992) Saccade-vergence interactions in humans. J Neurophysiol 68:1624–1641

Zhou W, King WM (1998) Premotor commands encode monocular eye movements. Nature 393:692–695