Compensation for Gaze Perturbation During Inactivation of the Caudal Fastigial Nucleus in the Head-Unrestrained Cat

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Goffart, Laurent, Alain Guillaume, and Denis Pélisson. Compensation for gaze perturbation during inactivation of the caudal fastigial nucleus in the head-unrestrained cat. J. Neurophysiol. 80: 1552–1557, 1998. Muscimol injection in the caudal part of the fastigial nucleus (cFN) leads, in the head-unrestrained cat, to a characteristic dysmetria of saccadic gaze shifts toward visual targets. The goal of the current study was to test whether this pharmacological cFN inactivation impaired the ability to compensate for unexpected perturbations in gaze position during the latency period of the saccadic response. Such perturbations consisted of moving gaze away from the target by a transient electrical microstimulation in the deep layers of the superior colliculus simultaneously with extinction of the visual target. After injection of muscimol in the cFN, targets located in the contralateral hemifield elicited gaze shifts that fell short of the target in both “perturbed” and “unperturbed” trials. The amplitude of the compensatory contraversive gaze shifts in perturbed trials coincided with the predicted amplitude of unperturbed responses starting from the same position. Targets located in the opposite hemifield elicited hypermetric gaze shifts in both trial types, and the error of compensatory responses was not statistically different from that of unperturbed gaze shifts. These results indicate that inactivation of the cFN does not interfere with the ability of the head-unrestrained cat to compensate for ipsiversive or contraversive perturbations in gaze position. Thus the gaze-related feedback signals that are used to compute a reference signal of desired gaze displacement are not impaired by cFN inactivation.

METHODS

INTRODUCTION

It was proposed that saccadic shifts of the visual axis are controlled by a feedback mechanism. First formulated for head-restrained saccadic eye movements (Jürgens et al. 1981; Robinson 1975), this hypothesis was later extended to gaze shifts resulting from combined eye and head movements (for review see Guitton 1992). The displacement of the line of sight would be driven by a reference signal specifying the gaze displacement required to foveate the target (desired gaze displacement command). The basic idea of this feedback control hypothesis is that gaze accuracy is preserved regardless of how the movement is executed, as long as the feedback mechanisms accurately inform about the actual performance. Compatible with this prediction is the demonstration in behaving animals of a preserved accuracy of eye or gaze saccades despite perturbations induced by intracerebral electrical microstimulation (e.g., Keller et al. 1996; Pélisson et al. 1989, 1995; Sparks and Mays 1983).

The contribution of the medioposterior cerebellum to the control of gaze-shift accuracy is well established (Leigh and Zee 1991), but its specific role in the context of the feedback control hypothesis is still unclear. The loss of gaze accuracy after inactivation of the caudal fastigial nucleus (cFN) (head-fixed monkey: Ohtsuka et al. 1994; Robinson et al. 1993; head-free cat: Goffart and Pélisson 1998) could result from a mismatch in the actual movement and its internal representation or from an impaired specification of the reference signal of desired gaze displacement.

To distinguish between these two possibilities, we tested the ability of the cFN-inactivated cat to compensate for a perturbation in gaze position while orienting toward a visual target. Gaze perturbation was induced by electrically microstimulating the deep layers of superior colliculus (SC) during the reaction time of a gaze shift toward a flashed target (Pélisson et al. 1989; Sparks and Mays 1983). To test the effectiveness of compensations independently from the dysmetria related to cFN inactivation, we compared the compensatory gaze shift of such perturbed trials to the primary gaze shift of unperturbed trials with a similar desired gaze displacement. The predictions are as follows. If the dysmetria induced by cFN inactivation results from inadequate feedback signals, gaze perturbations may be micoded and compensation failures are expected. On the other hand, if the dysmetria results from a change in the reference signal, full compensations for gaze perturbations are expected.
Compensatory gaze shifts were still observed after muscinol injection in the left cFN of cat \( L \) (experiment \( L/cFN/ISC \)), as illustrated in Fig. 1A, showing representative responses elicited by a target located \( 27^\circ \) to the right. The temporal courses of horizontal gaze and head position are plotted for two unperturbed (Fig. 1A, left panel) and one perturbed trials (Fig. 1A, right panel). Stimulation of the right SC shifted gaze to the left ("perturbation") and was followed by a rightward gaze shift ("compensation") toward the remembered target location. Note that because of the cFN inactivation, all three target-elicited gaze shifts markedly undershot the target location. Note further that the compensatory gaze shift was more hypometric than the unperturbed gaze shift that started from a gaze position corresponding to the preperturbation position (Fig. 1A, trajectory a). Nevertheless, the amount of hypometria (error = \(-15.7^\circ\)) was similar to that of the other unperturbed gaze shift (error = \(-15.6^\circ\)), which started from a position comparable with the gaze position reached at the end of the perturbation (Fig. 1A, trajectory b). For a given target position when gaze starts from the same position, the similarity between gaze-shift amplitude in the perturbed and unperturbed trials suggests that the gaze perturbation was adequately taken into account in the production of the compensatory gaze shift. To evaluate the consistency of this observation, the amplitude of each unperturbed and compensatory gaze shift recorded during cFN muscinol inactivation is plotted as a function of the amplitude of the desired horizontal gaze displacement (Fig. 1B). For unperturbed trials (\( \bigcirc \)) the desired gaze displacement corresponds to the distance between target and gaze during the target presentation time, and for perturbed trials (\( \blacktriangle \)) it represents the new distance between target and gaze after the electrically induced change in gaze position. The regression analysis that was performed for unperturbed gaze shifts confirmed the typical gain reduction of contraversive gaze shifts (regression equation in legend). It is remarkable that the amplitude values of the compensatory gaze shifts (\( \blacktriangle \)) are distributed along the regression line fitting the unperturbed responses. This observation was analyzed further by computing for each perturbed trial the inaccuracy of compensation as defined relative to comparable unperturbed responses. This value of compensation error was obtained by subtracting from the compensatory gaze-shift amplitude the amplitude of the predicted unperturbed gaze response to the same target (i.e., distance between each perturbed data point and the regression line in Fig. 1B). A positive value corresponds to an overcompensation and a negative one to an undercompensation. As seen in Fig. 1C, the compensation error did not depend on the perturbation size (—, regression); in addition, it was not statistically different from zero (\(-0.7 \pm 2.9^\circ\), mean \( \pm \) SD; \( t (df = 17) = 1.0, P > 0.05 \)). The values of compensation error differed clearly from the theoretical values predicted from the hypothesis of no compensation (— — —).

Target in the ipsilesional visual hemisphere

Figure 2 shows the data obtained after muscinol injection in the left cFN of cat \( L \) (experiment \( L/cFN/ISC \)). The time course of the horizontal component of gaze and head movements toward a target located 19° to the left are shown in Fig. 2A. All gaze shifts were hypermetric and ended at the same position beyond the target. In the perturbed trial, the left SC stimulation quickly shifted gaze to the right (perturbation). This perturbation was shortly followed by a leftrightward gaze shift (compensation) toward the remembered target location. This compensatory gaze shift was markedly hypermetric and overshoot the target location by 13.5°, an error relative to the target virtually identical to that of the two unperturbed gaze shifts. The amplitude of each unperturbed and compensatory gaze shift was plotted as a function of the amplitude of the desired horizontal gaze displacement (Fig. 2B). First, note that the “unperturbed relationship” is displaced toward negative values of actual gaze displacement (see equation of regression in Fig. 2 legend). This result illustrates the characteristic hypermetria of gaze shifts directed toward the inactivated cFN, which is largely related
to a bias of the response end points relative to the target. Second, the overlap between the perturbed data points and the unperturbed ones indicates that the relationship between the amplitude of compensatory gaze shifts and the desired gaze displacement is similar to that of unperturbed responses. The average compensation error was not statistically different from zero [0.73 ± 3.92°, t (15) = 0.74, P > 0.05]. In addition, as shown in Fig. 2C, there was no correlation between compensation error and perturbation size. In the same cat we injected muscimol in the right cFN, and compensatory responses to a target presented in the ipsilesional hemifield (experiment L/rcFN/rSC) provided the same results. Table 1 shows that the average value of compensation error and its correlation with perturbation size did not reach a significant level in any experiment.

**DISCUSSION**

These results provide some clues regarding the origin of the head-unrestrained gaze dysmetria observed after muscimol inactivation of the cFN (Goffart and Pélisson 1994, 1998; Goffart et al. 1998). It is shown that inactivation of the cFN did not interfere with the ability of the head-unrestrained cat to compensate for collicular-induced pertur-
Compensatory Gaze Shifts During cFN Inactivation

A

Unperturbed

Perturbed

20 R (Contra)

20 L (Ipsi)

Target

Target

Target on

Target on

Time

200 ms

FIG. 2. Compensatory responses toward a target located in the ipsilesional visual hemifield (experiment L/cFN/ISC). Same conventions as in Fig. 1. A: time course of horizontal gaze and head position during representative responses elicited by a target briefly presented at 19° to the left and recorded during inactivation of the left cFN. B: relationship between actual and desired amplitude of horizontal gaze displacement achieved during the primary gaze shift of unperturbed trials (○) or during the compensatory gaze shift of perturbed trials (▲). Equation of regression: \( y = 1.24x - 10.36 \) (\( R^2 = 0.89 \)). C: relationship between gaze compensation horizontal error and perturbation size [— — , equation: \( y = 0.06x \), slope not statistically different from 0, \( t (15) = 0.8, P > 0.05 \)]. B and C, arrow: compensatory gaze shift shown in A.

B

C

Table 1. Perturbation size and compensation horizontal error computed for each experiment

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Perturbation Size (°)</th>
<th>Compensatory Error (°)</th>
<th>Theoretical Slope</th>
<th>Actual Slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>H/cFN/lSC</td>
<td>9.4 ± 3.7</td>
<td>0.7 ± 2.9 NS</td>
<td>-0.65</td>
<td>-0.12 NS</td>
</tr>
<tr>
<td>L/cFN/ISC</td>
<td>11.5 ± 4.6</td>
<td>-0.7 ± 3.9 NS</td>
<td>-1.24</td>
<td>0.06 NS</td>
</tr>
<tr>
<td>L/cFN/lSC</td>
<td>10.7 ± 3.6</td>
<td>-0.7 ± 5.5 NS</td>
<td>-1.30</td>
<td>-0.09 NS</td>
</tr>
</tbody>
</table>

Values are means ± SD. Number of responses is 18, 16, and 24 for rows 1, 2, and 3, respectively. Last two columns are slopes of regressions (intercept set to zero) between the following parameters: 1) theoretical relationship predicted from the hypothesis of no compensation and 2) actual relationship. NS, differences not statistically significant from zero (Student’s t-test, \( P > 0.05 \)).

bations moving gaze away from the target of an impending gaze shift (Pélisson et al. 1989). Irrespective of whether the perturbation was directed toward or away from the inactivated cFN, the amplitude of the compensatory gaze shifts to a given target was inaccurate but nevertheless comparable with the amplitude of unperturbed gaze shifts starting from similar positions. In both cases, the compensation error was not related to the size of the perturbation within the tested range, and its average value was virtually zero. These findings indicate that the gaze perturbation is taken into account to update the command specifying the required gaze displacement for foveating a visual target. This result in turn suggests that neither the feedback mechanism informing about the gaze position perturbation nor the combination of this gaze perturbation with the memorized retinal signals of initial gaze error, to update the desired gaze displacement command, are affected by the muscimol cFN inactivation.

On the basis of the present findings, the origin of the gaze dysmetria induced by cFN inactivation can be discussed further in relation to two possible feedback control systems for gaze shift. On the one hand, if the feedback signals discussed above (hEGD for estimated horizontal gaze displacement) are used for the dynamic control of the gaze shift (Fig. 3A), gaze dysmetria cannot result from an inadequate dynamic feedback control. Instead, gaze dysmetria would result from a modification of the desired horizontal gaze displacement command (hJDG), which serves as a reference signal to the dynamic feedback mechanisms. This hypothesis was proposed previously to account for the bias of ipsiversive gaze shifts and the ensuing fixation offset (Goffart and Pélisson 1994, 1998) and for the unchanged dynamics of contraversive gaze shifts (Goffart et al. 1998). On the other hand, if distinct gaze-related feedback signals are used for updating the reference signal and for the dynamic
control of gaze shift (Fig. 3B), the current findings have some implication regarding the hypometria of contraversive gaze shifts during cFN inactivation. Indeed, the correct updating of the desired gaze displacement after gaze perturbation (see long feedback pathway in Fig. 3B) implies that the gain reduction of contraversive gaze shifts takes place downstream from this updating. In addition, the unchanged dynamics of contraversive gaze shifts (Goffart et al. 1998) would again place this gain reduction upstream from the dynamic feedback loop.

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