Orienting Gaze Shifts During Muscimol Inactivation of Caudal Fastigial Nucleus in the Cat. I. Gaze Dysmetria

LAURENT GOFFART AND DENIS PÉLisson
Espace et Action, Institut National de la Santé et de la Recherche Médicale U94, 69500 Bron, France

Goffart, Laurent and Denis Pélisson. Orienting gaze shifts during muscimol inactivation of caudal fastigial nucleus in the cat. I. Gaze dysmetria. J. Neurophysiol. 79: 1942–1958, 1998. The cerebellar control of orienting behavior toward visual targets was studied in the head-unrestrained cat by analyzing the deficits of saccadic gaze shifts after unilateral injection of muscimol in the caudal part of the fastigial nucleus (cFN). Gaze shifts are rendered strongly inaccurate by muscimol cFN inactivation. The characteristics of gaze dysmetria are specific to the direction of the movement with respect to the inactivated cFN. Gaze shifts directed toward the injected side are hypermetric. Irrespective of their starting position, all these ipsiversive gaze shifts overshoot the target by a constant horizontal error (or bias) to terminate at a “shifted goal” location. In particular, when gaze is directed initially at the future target’s location, a response with an amplitude corresponding to the bias moves gaze away from the actual target. Additionally, when gaze is initially in between the target and this shifted goal location, the response again is directed toward the latter. This deficit of ipsiversive gaze shifts is characterized by a consistent increase in the y intercept of the relationship between horizontal gaze amplitude and horizontal retinal error. Slight increases in the slope sometimes are observed as well. Contraversive gaze shifts are markedly hypometric and, in contrast to ipsiversive responses, they do not converge onto a shifted goal but rather underestimate target eccentricity in a proportional way. This is reflected by a decrease in the slope of the relationship between horizontal gaze amplitude and horizontal retinal error, with, for some experiments, a moderate change in the y intercept value. The same deficits are observed in a different setup, which permits the control of initial gaze position. Correction saccades rarely are observed when visual feedback is eliminated on initiation of the primary orienting response; instead, they occur frequently when the target remains visible. Like the primary contraversive saccades, they are hypometric and the ever-decreasing series of three to five correction saccades reduces the gaze fixation error but often does not completely eliminate it. We measured the position of gaze after the final correction saccade and found that fixation of a visible target is still shifted toward the inactivated cFN by 4.9 ± 2.4°. This fixation offset is correlated to, but on average 54% smaller than, the hypermetric bias of ipsiversive responses measured in the same experiments. In conclusion, the cFN contributes to the control of saccadic shifts of the visual axis toward a visual target. The hypometria of contraversive gaze shifts suggests a cFN role in adjusting a gain in the translation of retinal signals into gaze motor commands. On the basis of the convergence of ipsiversive gaze shifts onto a shifted goal, the straightforwardness of gaze trajectory during these responses and the production of misdirected or inappropriately initiated responses toward this shifted goal, we propose that the cFN influences the processes that specify the goal of ipsiversive gaze shifts.

INTRODUCTION

Shifting the direction of the eyes in space (= gaze) between relevant objects requires a transformation of sensory signals into appropriate motor commands for eye, head, and eventually body axis. Saccadic eye movements, usually studied in isolation by restraining the head, are by far the best understood component of this gaze-orienting behavior, both at the conceptual and neurophysiological levels. The original notions of a feedback burst generator and a neuronal integrator (Robinson 1975), involved in quickly displacing the eyes and holding them in a new position, respectively, are still common to all saccadic control models (see for review van Gisbergen and van Opstal 1989). Neuronal circuits responsible for the generation of these phasic and tonic motor signals have been identified in the brain stem (Fuchs et al. 1985; Moschovakis and Hightstein 1994). In addition, the contribution of structures projecting directly and/or indirectly to brain stem premotor neurons, like the superior colliculus (Sparks 1986), the frontal (Bruce 1990; Goldberg and Segraves 1989), and parietal (Andersen and Gnadt 1989) eye fields of the cerebral cortex, becomes progressively better understood (see also Schall 1991).

The cerebellum long ago was implicated in motor control in general and in oculomotor control in particular (for review see Ito 1984; Stein and Glickstein 1992; Thach et al. 1992). There is a large body of clinical and experimental data showing that cerebellar lesions severely interfere with saccade accuracy (reviews in Keller 1989; Leigh and Zee 1991; Lewis and Zee 1993). In conjunction with neurophysiological and anatomic studies, this lesion approach has led to progressively implicate vermian lobules VI–VII and the underlying caudal fastigial nucleus (cFN) as the core of the cerebellar regions involved in the control of saccadic eye movements in the monkey (Noda 1991). Indeed, any dysfunction of the fastigial nucleus (FN) leads to dysmetric saccades, whether in the case of permanent lesions (Optican and Robinson 1980; Ritchie 1976), reversible inactivations induced by cooling or inhibitory pharmacological drugs (Ohtsuka et al. 1994; Robinson et al. 1993; Vilis and Hore 1981), or electrical microstimulation (Ohtsuka and Noda 1991a). Saccade-related activities have been recorded in both vermian lobules VI–VII and cFN (cat: Gruart and Delgado-Garcia 1994; Harlay et al. 1974; Waterhouse and McElligott 1980; monkey: Fuchs et al. 1993; Hel McNen and Büttner 1995; Hel McNen et al. 1994; Hepp et al. 1982; Kase et al. 1990; Llinas and Wolfe 1977; Ohtsuka and Noda 1991b, 1995), and low-intensity electrical microstimulation of either lobules VI–VII or cFN evokes saccadic eye movements (Fujikado and Noda 1987; Noda and Fujikado 1987a,b; Noda et al. 1988).

The gross anatomic connections of this medial cerebellar area also were well characterized. Both lobules VI–VII and
cFN receive projections from many brain stem structures providing visual, auditory, proprioceptive, vestibular, and oculomotor signals (cat: Batini 1979; Batini et al. 1978; Dietrichs and Walberg 1987; Gerrits and Voogd 1987; Gould 1980; Hoddevik et al. 1977; van Der Want et al. 1987; monkey: Carpenter and Batton 1982; Noda et al. 1990). In turn, Purkinje cells in vermal lobules VI and VII project to and monosynaptically inhibit cFN neurons (Courville and Diakiew 1976; Dietrichs 1983; Ito et al. 1970; Noda et al. 1990). Finally, cFN was shown to project to many oculomotor-related structures in the brain stem (Blanks 1988; Carpenter and Batton 1982; Noda et al. 1990), many of them projecting back to vermal lobules VI–VII and cFN (Batini et al. 1978; Gould 1980). The cFN also issues projections to the periculomotor area of the mesencephalon (Gruart and Delgado-Garcia 1994), to the superior colliculus (Hirai et al. 1982; Kawamura et al. 1982; May et al. 1990; Roldan and Reinoso-Suarez 1981; Sugimoto et al. 1982), and to the thalamic ventromedian (Jimenez-Castellanos and Reinoso-Suarez 1985; Kyuhou and Kawaguchi 1987; Nakano et al. 1980; Steriade 1995) and suprageniculate (Katoh and Deura 1993) nuclei.

Theoretically, the saccadic dysmetria observed after cerebellar dysfunction could result from impairments in guiding the eye toward the goal or in specifying the goal itself. Compatible with the first hypothesis are suggestions that dysmetria results from either an altered feedback control of the on-going eye trajectory toward its goal (Keller et al. 1983; Vilis and Hore 1981) or from impaired acceleration or deceleration phases (Dean 1995; Robinson et al. 1993). Alternatively, the possibility that dysmetria results from a more central deficit, i.e., from a disturbance in the processes that translate retinal signals into commands for the impending movement (Optican 1982; Pellowitz and Linas 1982), is supported by less direct evidence. In this perspective, neuronal activities related to visual and auditory information have been recorded in lobules VI and VII (Buchtel et al. 1972; Freeman 1970; Koella 1959; Snider and Stowell 1944) and in cFN (Kawamura et al. 1990), leading some authors to suggest a teleceptive function of these cerebellar areas (Altman et al. 1976; Donaldson and Hawthorne 1979; Fadiga and Puppili 1964; Wolfe 1972).

If dysmetria of saccades result from such a central deficit, one might expect that it affects orienting of the visual axis in general and not only the control of the eye plant. To our knowledge, there has been no systematic investigation of cerebellar gaze dysmetria under conditions that allowed the head to contribute to the orienting of the visual axis. In the present study, we characterized the deficits in visually triggered gaze shifts produced by the head-unrestrained cat after unilateral inactivation of cFN. The spatial aspects of gaze deficits presented in this paper suggest a participation of cFN in the processes that specify the goal of an impending gaze shift. Some of the results have been presented previously in abstract form (Goffart and Pelisson 1994a–c).

METHODS

Subjects

Five adult cats (E-I) were used. The animals were deprived of food overnight before experimental testing, after which they were allowed to eat to satiation. They were cared in accordance with the guidelines from the French Ministry of Agriculture (87/848) and from the European Community (86/609/EEC).

Surgery

Cats were prepared for the chronic recording of orienting movements and pharmacological local injections. A single surgical procedure was performed under general anesthesia [pentobarbital sodium (Nembutal) 30 mg/kg ip] and aseptic conditions. A three-dimensional (Nembutal) 30 mg/kg ip] and aseptic conditions. A three-

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for 2 min. Then the dura was cleaned and pierced with the tip of a cannula. A thin cannula (230 μm OD, bevelled tip) was lowered through the recording chamber and aimed at the injection site. The injection was made by a Hamilton syringe connected to the cannula. A saline solution of muscimol (Sigma, 0.3 μl, 1 μg/μl) was delivered progressively by small pulses of 0.05 μl during a total period of 5 min. Eye movements were monitored during the whole injection period as well as during periods of cannula positioning and withdrawal. At least 48 h separated two consecutive injections.

To allow histological reconstruction of the injection sites, electrolytical marks were made at the end of the experimental series by passing cathodal current through an electrode (30 μA, 20 s). The animal then was euthanized by an overdose of pentobarbital sodium and perfused transcardially with saline, followed by 10% formalin. Standard techniques were used to prepare 60-μm slices on a freezing microtome and to reconstruct electrolytical lesions and injection sites.

**Behavioral tests**

After withdrawing the cannula, spontaneous eye movements in light and in darkness were recorded for ~5 min. Then the animal’s head was freed and visually triggered gaze shifts were recorded with one of the two setups during a 20- to 120-min period after the onset of the injection. A recording session consisted of a series of 2-s trials, each of which was initiated (data acquisition started) when the animal looked roughly in the direction of the fixation stimulus. The effects on gaze shifts presented in the following text were stable during the entire recording period. Control behavioral responses were measured the day preceding each injection.

**Data recording and analysis**

Search coil signals were linearized and scaled on-line by a computer program, providing four signals proportional to the horizontal and vertical positions of gaze (eye-in-space) and head. The calibration of each coil was performed before implantation by measuring the output voltage while the coil achieved known angular positions. The computed parameters for the linearization algorithm (gain and offset) were checked in vivo and, if necessary, amended by presenting the animal an attractive target at different locations. The overall precision of gaze and head measurement was estimated to be ±0.5°. The same program controlled the infrared emitters fixed on the spoon, processed signals from two remote infrared sensors and delivered on-line signals proportional to horizontal and vertical positions of spoon target relative to the animal’s longitudinal body axis (Urquizar and Pélisson 1992).

Horizontal and vertical signals of gaze, head and target position were recorded on a second PC microcomputer (Experimenter’s WorkBench software from DataWave, sampling frequency = 500 Hz), displayed on-line and stored to disk for off-line analysis. Analyses were performed with PC software developed in our laboratory. Gaze and head position signals were filtered digitally (FIR filter, 70 Hz cutoff frequency) and differentiated. The onset and termination of gaze shifts and of head movements were detected based on a velocity threshold (30°/s). The results of this automatic process were checked by displaying each analyzed trial and corrected when required. The onset of the eye saccade corresponded to that of the gaze shift, and its termination was defined as the time of maximal orbital eye position. Spatial parameters of eye, head, and gaze movements then were extracted automatically from the detected gaze shifts and further processed by a spreadsheet program. The main parameters analyzed in the present paper are: initial and final position, displacement amplitude for eye, and head and gaze movements. Linear regression analyses and Student’s t statistical tests were performed by Statistica software (StatSoft).
RESULTS

General observations

The data presented in this and the companion paper (Goffart et al. 1998) result from the analysis of 13 experiments. For practical purposes each experiment was labeled according to cat (E to I) and cFN (e.g., E-L stands for left cFN inactivation in cat E). Postmortem histological reconstruction confirmed that the sites of muscimol injection were located inside the caudal part of the fastigial nucleus. Examples of electrolytic lesions and reconstructed injection sites are indicated on parasagittal sections of the cerebellum in cats G and H (Fig. 2).

Eye movements recorded in the head-restrained animal after a unilateral injection of muscimol in the caudal part of the FN did not reveal any ocular nystagmus or gaze holding deficit, neither in light nor in darkness. Spontaneous saccadic eye movements in darkness did not develop any significant directional preference after muscimol injection (Fig. 3A).

Ipsilateral deviations of the visual axis

In both the head-restrained and -unrestrained conditions, the direction of the visual axis was deviated toward the inactivated cFN. Figure 3B shows that in the head-restrained animal, the mean orbital eye position was deviated slightly toward the injected side when tested with ambient lights (−5.9 ± 7.4 (mean ± SD) vs. −2.3 ± 6.7 for muscimol and control data respectively, Student’s $t$ (184) = 3.16, $P < 0.01$; Fig. 3B). The magnitude of this deviation increased in darkness (−7.5 ± 8.7 vs. 0.5 ± 8.1, Student’s $t$ (183) = 6.33, $P < 0.001$). When the head was unrestrained and the animal waited for target presentation, both gaze and head positions were deviated toward the injected side. Although systematically observed, these deviations could not be quantified as the animals had not been trained through reinforcement to align gaze or head with the fixation point.

The issue of target fixation by gaze will be further documented later in the fixation offset section. Nevertheless, the deviation of the eye in the orbit observed in the head-restrained condition disappeared in the head-unrestrained condition as illustrated by the relationship between eye position in the orbit (Eh) and head position (Hh; Fig. 4). The data reported in Fig. 4 were collected with the hemicylindrical setup in a left cFN experiment (injection I-L2 in cat I), by attracting gaze to different initial positions before target presentation (see METHODS). Eye deviation in the orbit increased with head deviation according to a similar linear relationship in the control condition (Eh = 0.20 · Hh − 1.24; $r^2 = 0.85; P < 0.001$) and in the muscimol condition (Eh = 0.21 · Hh − 1.26; $r^2 = 0.79; P < 0.001$).

A striking observation that confirmed the ipsilateral head deviation was systematically made after each muscimol injection in the cFN. When the food target (i.e., the spoon) was offered to the animal to reward it, the cat turned its head away from the food toward the injected side. This ipsilateral deviation of the head always was accompanied with masticatory jaw movements beside the spoon and, in the most severe cases, prevented the animal from getting the reward unless being helped by the experimenter. This typical deficit remained constant during the whole recording session. The animal also was observed when walking around in the laboratory after completion of the recordings after each injection. A crab-like walking pattern and a very weak tendency to fall on the ipsilesional side were sometimes noted, as if an external force pulled the animal laterally. No circling behavior ever was seen. Interestingly, when the animal walked toward an attractive goal (food target) or spontaneously toward its cage, we noticed for several experiments that the walking path was systematically and strongly curved toward the ipsilesional side. A videographic illustration of these head and body orienting deficits has been presented in a previous communication (Goffart and Pélisson 1994a).
A

**Light**

- Polar plots of the direction of spontaneous saccades recorded in the light (left) or in darkness (right).
- Proportion of saccades (scale bar = 10%) is plotted along the direction (30° bins).

**Dark**

- Control
- Muscimol (left cFN)

B

**Light**

- Control
- Muscimol (left cFN)

**Dark**

- Control
- Muscimol (left cFN)

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**FIG. 3.** Characteristics of spontaneous eye movements recorded in the head-restrained cat (left cFN inactivation, experiment I-L2). A: polar plots of the direction of spontaneous saccades recorded in the light (left) or in darkness (right). Proportion of saccades (scale bar = 10%) is plotted along the direction (30° bins). Control data shown by open area delimited by heavy line, pharmacological data shown by shaded area. B: orbital eye position at the beginning of each spontaneous saccade recorded during periods of ~5 min in the light (left) or in complete darkness (right).

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**Qualitative description of gaze dysmetria**

Figure 5 shows the horizontal trajectories of sample gaze shifts elicited in *cat I* by a target presented straight ahead, i.e., along the body sagittal axis (hemicylindrical setup). For the sake of demonstration, we selected responses starting from different initial positions. During the control session (Fig. 5A), gaze moved accurately toward the target or, when it initially was directed toward the target (initial position = 0°), remained stable. After muscimol injection in the left cFN (Fig. 5B), leftward gaze shifts were hypermetric and overshot the target by ~10°. Remarkably, this error was constant irrespective of initial gaze position. Even when gaze initially was directed at the future target location (0° initial gaze position), the cat moved its gaze away from the target toward the same final position. Furthermore, when gaze initially was aligned with this location where all ipsiversive responses ended (shifted goal), the cat did not produce any response at all. Beyond that position, rightward responses were generated that fell short of the target. But contrary to ipsiversive responses, it was clear that these contraversive gaze shifts did not converge onto a common final position. Instead, hypometric gaze error gradually increased as a function of initial target eccentricity relative to gaze.

To further illustrate the remarkable deficit of visually triggered gaze shifts directed toward the inactivation side (ipsiversive movements), we show in Fig. 6 the spatial trajectory of some sample movements recorded in another cat (*cat F*), in this case after inactivation of the right cFN (injection F-R). These responses were elicited by presenting the visual target at an eccentricity of 19° in the right visual field (screen setup) and were selected for this illustration because of their widely differing starting positions. For comparison with normal performance, the mean and variability (SD) of gaze end
Gaze dysmetria during CFN inactivation

We occasionally tested gaze shifts in response to a visual target presented above the center of the screen (screen setup). Again, a shift of vertical gaze saccade endpoints was observed in this situation, as shown in Fig. 7 by responses recorded from cat H before and after inactivation of the left CFN. Figure 7A illustrates the final positions reached by “postinactivation” gaze shifts (dashed ellipse: mean ± SD) evoked, from various initial positions ( ), by a target located 27° up (*). These upward gaze shifts were directed toward a final position that was shifted by ~10° leftward (−9.95 ± 2.42, n = 20) with respect to the target and to the final position of control responses as well (dashed ellipse). The time course of horizontal and vertical components, shown in Fig. 7B for four selected responses, indicates that initial movement direction already was aligned with this shifted position. For example, although starting from the same azimuth as the visual target, response labeled c had a large leftward component synchronized with the vertical one; and conversely, responses a and d that started from the same azimuth as the shifted goal showed almost no horizontal deviation. A significant shift can be noted on the vertical component as well, but it is smaller than the horizontal one.

Gaze displacement amplitude

The pattern of ipsilateral hypermetria/contralateral hypometria induced by muscimol injection was analyzed quantitatively as illustrated in Fig. 8 for two experiments. The horizontal component of each recorded gaze shift was plotted as a function of target eccentricity relative to gaze along the azimuth (horizontal retinal error). Horizontal retinal error is by convention negative, positive or null when the target azimuth or elevation. This ipsilateral shift is representative of all other injections with respect to its amplitude and its predominantly horizontal direction.

FIG. 4. Relationship between orbital eye position and head position at the onset of orienting gaze responses (left CFN inactivation, experiment I-L2). These data have been collected when the head-unrestrained animal, in the hemicylindrical setup, was fixating various locations along the azimuth both before ( , – – –) and after CFN inactivation ( , – – –). Overlap of data points and the similarity of the regression lines (equations in text), suggest similar eye to head position relationships in both conditions.

FIG. 5. Temporal trajectories of horizontal component of gaze shifts (left CFN inactivation, experiment I-L2). These responses were elicited by presenting the visual target at the central location (along the animal’s sagittal plane) in the hemicylindrical setup while the animal was fixating at various locations along the horizontal meridian. In the control condition (A), gaze responses converge onto the target. However, after left CFN inactivation (B), leftward responses are hypermetric and converge onto a position that is shifted ~10° to the left of target location ( – – –). Note that gaze reaches a similar location even when its initial position corresponds to that of the target. Rightward gaze shifts are hypometric with an error that increases with initial gaze location.
is presented, in the left hemifield, in the right hemifield, or along the vertical meridian, respectively. Negative and positive horizontal amplitudes correspond to leftward and rightward gaze shifts, respectively. Each data point represents a gaze response recorded either before (○) or after (●) muscimol injection (Fig. 8, A and B; injection G-L in the left cFN; Fig. 8, C and D; injection F-R in the right cFN). Linear regression analyses of the relationship between horizontal retinal error (x) and horizontal gaze amplitude (y) were performed separately for leftward (Fig. 8, A and C) and rightward gaze shifts (Fig. 8, B and D). After muscimol injection in the left cFN of cat G, leftward gaze shifts (Fig. 8A) were hypermetric by a constant error. As reported qualitatively above, this bias is illustrated clearly by the presence of inappropriate movements (0° abscissa), misdirected gaze shifts elicited by a visual target presented to the right (abscissa in the 0°–15° range) and by the absence of an horizontal component for responses toward a target presented at.
FIG. 8. Relationships between horizontal retinal error and horizontal gaze amplitude illustrated for 2 experiments: left cFN inactivation, experiment G-L (A and B) and right cFN inactivation, experiment F-R (C and D). In each panel, the amplitude of horizontal gaze displacement is plotted vs. horizontal retinal error (i.e., actual vs. desired amplitude of horizontal gaze shift component); pre- and postinactivation data are shown by ○ and ●, respectively. Leftward (negative values) and rightward (positive values) movement directions are shown in separate panels (A and C and B and D, respectively). Equations of regression lines drawn on the plots are indicated in Table 1. Note that cFN inactivation resulted in changes in the intercept for ipsiversive movements (A and D) and in the slope for contraversive movements (B and C).

The shifted-goal location (abscissa in the 15°–20° range). This leftward bias of gaze final position relative to the control condition was confirmed by a 19° downward shift of the regression line (y intercept negative) (see regression equations in Table 1). A similar, although smaller (10°), change in the regression line y intercept was observed for rightward gaze responses recorded in cat F after muscimol injection in the right cFN (Fig. 8D). The slopes of the regression lines for ipsiversive responses were, as compared with the y intercept, much less (cat G) or not at all (cat F) affected. Note also an increase in the scatter of the data points around the regression lines in the muscimol condition. For contraversive gaze shifts, the deficit was clearly different and an opposite pattern was found. As previously shown qualitatively in Fig. 5, error increased with larger target eccentricities. This is demonstrated here by a change in the slope of the relationship between horizontal retinal error and horizontal gaze amplitude: the slope decreased by 37% for cat G (Fig. 8B) and by 53% for cat F (Fig. 8C). The hypometria of contraversive gaze shifts also can be related to a change in the regression line y intercept (−4.24 vs. −0.40° for cat G), but this effect accounts for a small part of gaze inaccuracy. Finally, the scatter of the data points around the regression lines was much larger in the muscimol condition than in the control one; and this increase of variability seems larger than for ipsiversive movements.
The equations of the relationships between horizontal retinal error and horizontal gaze amplitude for 13 experiments performed on five cats are summarized in Table 1. Overall, y-intercept and slope values confirm, in a very consistent manner, the pattern illustrated above.

**Correction saccades**

For all responses presented so far, movement onset triggered the offset of ambient light so that the gaze shifts were performed in complete darkness (see METHODS). In this condition, we observed gaze correction saccades only occasionally. To investigate whether correction saccades compensate for the dysmetria described above, we included some trials where the ambient light remained on. Figure 9 shows two such trials recorded after injection in the left cFN (injection G–L), with the visual target presented at 35° either in the left (Fig. 9A) or in the right (Fig. 9B) visual hemifield. Although visually guided correction saccades were observed in both cases, it appeared that static alignment of gaze with the target was abnormal after correction for the ipsilateral hypermetria: the example in Fig. 9A shows that a single and small correction saccade was made in the contraversive direction and that a large gaze fixation error was left uncorrected for a period of >500 ms. In other cases (not shown), despite the large hypermetria of ipsiversive gaze shifts, no correction saccade was ever produced. For contraversive hypometric movements, staircases saccades were observed most often. In some cases, like the example illustrated in Fig. 9B, these correction saccades eventually brought gaze very close to the target, but in other cases they failed and a significant fixation error remained. Note that the direction of correction saccades is always contraversive.

The metrical characteristics of these correction saccades were further analyzed for the two experiments presented above (cats F and G tested in the screen setup, see also Fig. 8). Figure 10 represents the amplitude of all contraversive gaze responses (primary and correction saccades) observed in cat G after muscimol injection in the left cFN (Fig. 10A) and in cat F after injection in the right cFN (Fig. 10B). First, the amplitude of correction saccades was smaller than the retinal error, indicating that saccadic hypometria during cFN inactivation also affected correction saccades, as already illustrated in Fig. 9. Second, the degree of hypometria depended according to whether correction saccades were generated after an ipsiversive primary saccade and directed toward the body sagittal plane (Fig. 9A; Fig. 10, ▲: centripetal saccades) or generated after a contraversive primary saccade and directed away from the body sagittal plane (Fig. 9B; Fig. 10, △: centrifugal saccades). This is reflected by
GAZE DYSMETRIA DURING CFN INACTIVATION

FIG. 9. Temporal trajectories of orienting movements recorded in "permanent target" trials after left cFN inactivation (experiment G-L). A: horizontal trajectories of eye, head, and gaze movements (top) and corresponding gaze and head velocity profiles (bottom), generated after the presentation of the target at 35° to the left (— — —). Note that the large overshoot of the primary ipsiversive saccade is left essentially uncorrected after the contraversive correction saccade. B: responses toward a target located at 35° to the right are shown with the same conventions as in A. In this trial, the large hypometria, typical of contraversive primary gaze shifts, is suppressed almost completely after termination of a series of 3 hypometric correction saccades.

FIG. 10. Relationships between horizontal retinal error and horizontal gaze amplitude for primary and correction gaze shifts in the contraversive direction. A: left cFN inactivation, experiment G-L. B: right cFN inactivation, injection F-R. Primary and correction gaze shifts are represented by ◊ and ▲ and ◇ and ▲, respectively. Both primary and correction gaze shifts are distinguished according to whether they are directed away from (centrifugal responses) or toward (centripetal responses) the body sagittal axis.

A statistically significant lower saccadic gain (amplitude of correction saccade to residual retinal error ratio) for centripetal correction saccades than for centrifugal ones: 0.13 ± 0.06 versus 0.64 ± 0.06 [Student’s t(41) = 10.6, P < 0.001] for cat G and 0.30 ± 0.17 versus 0.54 ± 0.15 [Student’s t(28) = 3.4, P < 0.001] for cat F. It is noteworthy that the same analysis applied to primary contraversive saccades of cat G revealed a similar difference between centrifugal and centripetal saccades. These two types of primary contraversive gaze shifts were obtained, respectively, by having the animal initially gazing close to the center of the screen or gazing more eccentrically than the target toward the ipsilateral side. The gain of centripetal primary saccades (Fig. 10, ◊) was statistically significantly lower than the gain of centrifugal primary saccades (●): 0.13 ± 0.06 versus 0.50 ± 0.09 [Student’s t(148) = 12.6, P < 0.001]. Third, centrifugal correction saccades show a similar degree of hypometria as centrifugal primary saccades (cat G: 0.64 ± 0.17 vs. 0.50 ± 0.09; cat F: 0.54 ± 0.15 vs. 0.54 ± 0.10).

Fixation offset

As mentioned above, when the cat waited for target presentation, gaze, and head were deviated toward the injected side as compared with the preinjection condition. Because our animals were not specifically reinforced for accurate looking at a fixation point before the visual target is presented (see METHODS) but for orienting toward the target, we investigated this tonic gaze deviation by measuring the position reached after all correction saccades (final gaze position) toward a permanently visible target. This analysis of target fixation by gaze was made for all control and musci-
mol experiments. The horizontal fixation offset was calculated for each postinactivation trial by the difference between final gaze position and the corresponding mean value obtained in the control condition. This offset always was directed toward the inactivation side (grand mean computed on 13 experiments $\pm$ SD: $4.9 \pm 2.4^\circ$; range: $1.0 - 9.4^\circ$). Then the fixation offset for targets presented in the ipsilesional hemispace was compared with the horizontal constant error of ipsiversive gaze shifts ($y$ intercept of gaze displacement amplitude against retinal error relationship). The absolute value of the fixation offset was correlated with the absolute value of the $y$ intercept (Pearson correlation coefficient $r = 0.61, P < 0.001$). However, these values did not match as the fixation offset was significantly smaller than the ipsilateral constant error [5.5 $\pm$ 2.8$^\circ$ vs. 10.9 $\pm$ 4.7$^\circ$. Student’s $t(24) = -3.49, P < 0.01$]. On average, the constant error of the primary ipsiversive saccades was corrected by a factor of $45 \pm 29\%$ (range: $-5 - 81\%$). The correction exceeded 50% in 9 of 13 cases (range: $50 - 81\%$). Altogether, these results suggest that the visual feedback provides information that help to compensate for the hypermetria of the primary ipsiversive gaze shifts.

**DISCUSSION**

We have shown in this paper that unilateral muscimol injections in the caudal part of the fastigial nucleus lead to marked and consistent impairments in the accuracy of goal-directed saccadic gaze shifts. Like the dysmetria of saccadic eye movements recorded in the head-restrained monkey (Ohtsuka et al. 1994; Robinson et al. 1993), this gaze dysmetria depends on the direction of the movement with respect to the injected side: ipsiversive gaze shifts are hypermetric, whereas contraversive ones are hypometric. Our observations therefore illustrate the key role of this cerebellar area in the control of feline saccadic gaze shifts and extend the dysmetria observed in the head-restrained monkey (Ohtsuka et al. 1994; Robinson et al. 1993; Vilis and Hore 1981) to the head-unrestrained condition.

Histological reconstruction verified that the injection sites were within the caudal part of FN. Also, we believe that neurons were inactivated within 1 mm of our injection sites. Indeed, a previous work on muscimol diffusion in the rat brain revealed a diffusion radius of 1.7 mm for an injected volume of 1 $\mu$l (Martin 1991), three times larger than the volume used in the present experiments. In addition, different deficits were observed when injections were made 2 mm apart: for example, in an investigation of the rostral part of FN (which will be reported in a subsequent paper), we showed that injecting muscimol 2 mm more rostral than in the present study did not induce any bias in ipsiversive gaze shifts (Goffart 1996).

We now will compare our findings with previous data and discuss the cerebellar-dependent neuronal processes that have been altered by unilateral cFN inactivation. Because each demonstrates a different dysmetria and because the premotor centers that control gaze shifts have a lateralized organization, ipsiversive and contraversive movements will first be considered separately.

**Ipsiversive movements**

After muscimol injection in the cFN, gaze shifts toward the injected side are hypermetric by a constant horizontal error. This striking deficit is illustrated clearly by the convergence of gaze spatial trajectories onto a location that is shifted horizontally from the actual target (shifted goal). The nonzero $y$ intercept in the relationship between horizontal retinal error and horizontal gaze amplitude further indicates that this constant error is present for all gaze shifts, irrespective of target eccentricity. In addition, the horizontal error observed in the gaze shifts having a purely vertical direction and the similarity of this error with that of ipsiversive gaze shifts cannot be accounted for by a deficient braking of the horizontal trajectory of gaze. Moreover, gaze shifts during cFN inactivation all had normally straight trajectories and gaze moved in the direction of the shifted goal from the beginning of the movement. The observation of straight gaze shifts confirms previous reports on saccadic eye movements in fastigial-inactivated monkeys (Ohtsuka et al. 1994; Vilis and Hore 1981; but see Robinson et al. 1993) and in cerebellar patients (Ranalli and Sharpe 1986). Altogether, these observations suggest that muscimol injection in cFN interferes with the elaboration and/or the maintenance of control signals generated upstream from or at the level(s) where the motor commands, leading to horizontal and vertical motions, interact.

The fact that when gaze initially is directed at the target location (null retinal error), gaze moves away from the target suggests that cFN inactivation has impaired processes that specify the goal before movement initiation. Just as meaningful are the misdirected ipsiversive responses produced when the target is presented in the contralateral hemifield, at a retinal eccentricity smaller than the constant error. All these inappropriately triggered and misdirected movements have dynamic (velocity-duration) properties and eye/head coordination pattern resembling those of other visually triggered saccadic gaze shifts (Goffart et al. 1998). Finally, the fact that, for a given target, these inappropriately triggered and misdirected gaze shifts end at the same final position as the final location of hypermetric ipsiversive gaze shifts leads us to propose that a common target specification process was affected in all these ipsiversive gaze shifts.

In conclusion, the deficits in ipsiversive movements, as well as the gaze fixation offset discussed in the following text, are not consistent with the hypothesis of an impairment of the processes controlling exclusively movement execution. In contrast, they suggest a dysfunction in the processes that specify the spatial dimensions of the goal (desired displacement—or final position—of gaze) during movement preparation.

This conclusion about ipsiversive movement dysmetria differs from the hypothesis that was proposed previously to account for inactivation-induced dysmetria in the primate (Ohtsuka et al. 1994; Robinson et al. 1993; Vilis and Hore 1981). Indeed, it was proposed that the FN controls the trajectory of the on-going saccade: hypermetria would result either from an impaired feedback control of the saccade (Vilis and Hore 1981; see also Keller 1989 for a similar interpretation of cerebellar role in the control of contraversive saccades) or from a flawed control of its deceleration.
phase (Ohtsuka et al. 1994; Robinson et al. 1993). In the first case, hypermetria would be the consequence of the internal feedback signal of current eye position underestimating actual eye position, leading to an incorrect updating of dynamic motor error. In the second case, saccadic hypermetria would result from the absence of the “late” neuronal burst of fastigial activity that normally is produced during ipsiversive saccades (Fuchs et al. 1993; Helmchen et al. 1994; Ohtsuka and Noda 1991b) and that is supposed to help decelerate the eyes.

Because the previous inactivation studies were performed in the head-restrained monkey, differences in species or in testing condition (head-restrained vs. unrestrained) could be invoked. However, we believe that other methodological differences could explain these divergent views. Vilis and Hore (1981) observed that cooling the fastigial nucleus essentially results in a hypermetria of saccadic eye movements in all directions. This deficit, which resembles the generalized hypermetria described after bilateral inactivation of cFN by muscimol (Robinson et al. 1993), can be explained by simultaneous inactivation of neuronal bodies in the ipsilateral FN and of fibers originating from the contralateral FN (Sugita and Noda 1991). In addition, because the cooling probe was located between the fastigial and interposed nuclei, the inactivation area was larger than in pharmacological experiments and most likely not confined to the cFN. Robinson et al. (1993) used the same method as ours to specifically inactivate cFN and found a similar ipsiversive hypermetria/contraversive hypometria pattern. However, they described the hypermetria of ipsiversive saccades as an increase in gain (ratio of actual to desired eye displacement). We wonder whether this analysis captured the essential features of their results because computation of a gain value could not test the existence of a constant error in saccadic hypermetria. It is remarkable that in their two animals (see their Fig. 3), the computed value of saccadic gain after cFN inactivation was larger for responses to a 10° than for those to a 20° target displacement (1.31 vs. 1.15 in monkey M1 and 1.58 vs. 1.19 in monkey M2), just as can be expected if the hypermetria of ipsiversive responses comprised a constant term. Such a constant error in monkey ipsiversive saccades can be found in a brief report on saccadic dysmetria induced by muscimol inactivation of the cFN (Ohtsuka et al. 1994). It was shown clearly that the tested monkey made saccades toward a terminal location that was shifted horizontally by ∼7° from the location of the flashed target (see their Fig. 2H). Altogether, these observations in the monkey do not rule out the existence of a saccadic ipsiversive constant error and suggest that the deficits described in the present paper are not specific to the cat behaving in a head-unrestrained condition. Conversely, our proposal that cFN inactivation impairs the specification of the metrics of the impending gaze shift does not exclude the possibility of additional deficits affecting gaze shift dynamics. Indeed, in some experiments, the slope of the relationship between horizontal retinal error and horizontal gaze amplitude increased for ipsiversive responses. This gain increase tendency could, as previously suggested (Robinson et al. 1993), result from a flawed control of the movement deceleration phase (see companion paper).

Contraversive movements

The metrics of contraversive gaze shifts reveal two features that distinguish these movements from ipsiversive ones: they undershoot the target and this hypometric error markedly increases as a function of target eccentricity. These characteristics are reflected in the reduced slope of the relationship between horizontal retinal error and horizontal gaze amplitude after cFN inactivation. This type of hypometria is consistent with a reduction of an overall gain in the visuomotor transformation mechanisms, in the line of the proposed cerebellar role in tuning the gain of saccadic system (Dean 1995; Keller 1989; Keller et al. 1983; Optican and Robinson 1980; Robinson et al. 1993; Schweighofer et al. 1996; Selhorst et al. 1976; Vilis and Hore 1981).

Our data on contraversive movements are compatible with the effects of electrical stimulation of the vermal cortex described in the monkey (Keller et al. 1983; Ohtsuka and Noda 1991a). In the first study (Keller et al. 1983), a subthreshold stimulation (i.e., with an intensity insufficient to evoke any saccade) was applied to lobules V and VI during on-going visually triggered saccades. Stimulations delivered during contraversive saccades resulted in a shortening of eye displacement that was proportional to the intended saccade size. Ohtsuka and Noda (1991a) have extended these stimulation tests of visually directed saccades to stimulations of the oculomotor vermis (lobules VIc–VII) during the latency period of contraversive saccades. They showed a saccade shortening associated with a truncation of the presaccadic burst of cFN neurons. Noteworthy, a similar, although more subtle, saccadic amplitude modification recently was reported in man by applying transcranial magnetic stimulation over the cerebellar vermis (Hashimoto and Ohtsuka 1995). It has been proposed that the “early burst” of cFN neurons help the initiation of simian contraversive saccades by excitation of burst neurons and inhibition of omnipause neurons of the reticular formation (Fuchs et al. 1993; Ohtsuka and Noda 1995). This hypothesis is compatible with the increased latency of contraversive movements during cFN inactivation in the cat (Goffart and Pélisson 1997). However, although suprathreshold stimulation of these vermal lobules also evokes ipsiversive saccades in the cat (Cohen et al. 1965; Gauthier and Stark 1979), no data are available about the effect of transient vermal stimulation on visually triggered saccades, and the presence of an early burst associated with contraversive saccades is not well established. Indeed, in a systematic study of oculomotor properties of deep cerebellar nuclei neurons in the cat, Gruart and Delgado-Garcia (1994) show that, among antidromically identified output neurons located in the caudal part of FN, the majority burst only after the initiation of contraversive saccades (type I eye position velocity neurons). Comparatively, they found only a few saccadic neurons that started their phasic discharge before contraversive saccade onset. Based on the similarity of gain reduction for both monkey saccades and cat gaze shifts after cFN inactivation, it can be hypothesized that future unit recording and stimulation studies in the cat will provide more compelling evidence for an early neuronal burst contributing to the acceleration of contraversive saccades.
Fixation offset

The deficits of gaze shift accuracy during muscimol injection always were associated with a tonic, horizontal deviation of gaze toward the inactivated cFN when the animal attempted to fixate the center of the screen. Note that no spontaneous nystagmus was observed in light or in darkness, in agreement with previous cFN inactivation studies (Kurzan et al. 1993; Ohtsuka et al. 1994; Robinson et al. 1993). A fixation offset of gaze also was observed after completion of the total orienting response. A similar, although smaller, ipsilesional offset in target fixation was reported after unilateral cFN inactivation in the head-fixed monkey (Ohtsuka et al. 1994; Robinson et al. 1993) and a contralesional fixation offset was described after permanent (Aschoff and Cohen 1971) or reversible decortication of lobules VI–VII in monkey (Sato and Noda 1992). This deficit seemingly results from the suppression (during cFN inactivation) or the disinhibition (during decortication) of fastigial tonic discharges that have been recorded in both cat (Gruart and Delgado-Garcia 1994) and monkey (Noda et al. 1988; Ohtsuka and Noda 1991b). Finally, we systematically observed an ipsilesional shift in head positioning when the animal approached its mouth toward the spoon to get its reward; and for several experiments, the path followed by the animal when walking toward a goal was curved in the ipsilesional direction (Goffart and Pélisson 1994a).

Based on the following observations, we propose that the fixation offset is distinct from the constant error of ipsiversive gaze shifts. First, a large fixation offset was not consistently observed in every trial as the animal frequently could look ultimately at the strongly attractive food targets after successive corrective saccades. This is confirmed by our quantitative analysis showing a mean offset smaller than the mean constant error. Second, considering the data collected with the screen setup, it is clear that the hypermetria of ipsiversive gaze shifts is the same irrespective of initial gaze position (gaze directed toward the plastic bolt or offset relative to it toward the contralesional or ipsilesional side). This observation indicates that the constant error of ipsiversive gaze shifts is not related to the initial gaze deviation relative to the fixation point.

Possible neurophysiological substrate

Throughout this paper, we have maintained a clear distinction between movements according to their direction because it soon appeared that the corresponding deficits were qualitatively different (Goffart and Pélisson 1994c, 1997). This dichotomy must be related somehow to the pattern of efferent projections from cFN. On the one hand, the cFN issues efferent projections toward oculomotor areas of the contralateral pontine and mesencephalic reticular formations (Carpenter and Batton 1982; Gruart and Delgado-Garcia 1994; Homma et al. 1995; Noda et al. 1990) and to the contralateral nucleus prepositus hypoglossi (McCrea and Baker 1985). These crossed projections could mediate cFN inactivation-related deficits of contraversive gaze shifts. On the other hand, the cFN also sends bilateral projections toward both superior colliculi (Hirai et al. 1982; Kawamura et al. 1982; May et al. 1990; Roldan and Reinoso-Suarez 1981; Sugimoto et al. 1982; but see Noda et al. 1990) and thalamic nuclei (Jimenez-Castellanos and Reinoso-Suarez 1985; Kyuhou and Kawaguchi 1987; Katoh and Deura 1993; Nakano et al. 1980; Steriade 1995), providing a possible substrate both for the ipsiversive gaze shift deficits and for a component of the contraversive gaze shift deficits as well. We discuss in the following text the possibilities of the cFN acting at the collicular level or downstream of the superior colliculus (SC).

The possibility that cFN activity influences the superior colliculus (SC) (collicular hypothesis) is relevant for three reasons. First, the deep layers of the SC, to which the cFN projects, classically are assigned a key role in encoding the metrics of impending eye (or gaze) saccades within an eye (or gaze) frame of reference (Freedman and Sparks 1997; Freedman et al. 1996; Munoz and Guittion 1991; Sparks and Mays 1990). Second, these collicular layers also are involved strongly in the control of head and body movements that contribute to shifting gaze in space (Guittion 1991; Sparks 1986; Stein and Meredith 1993). Third, the deep SC has been involved in the target selection process and in keeping in memory information about the selected goal until initiation of the saccadic response (Glimcher and Sparks 1992, 1993; van Opstal and van GISBERGEN 1990). Through the direct or indirect tectal projections, discussed in the following text, the cFN is in a position to modify the activity of SC neurons during the “preparation phase” of the orienting saccade. Indeed, although previous unit recording studies have emphasized the late burst of cFN neurons occurring just before the end of ipsiversive saccades, a strong tonic activity also is observed in the cFN in both cat (Gruart and Delgado-Garcia 1994; unpublished observations) and monkey (Fuchs et al. 1993; Ohtsuka and Noda 1991b). Suppression of this tonic activity by muscimol could alter the collicular mechanisms that keep accurate information about the saccade goal in memory, resulting in the bias of ipsiversive gaze shifts and in the fixation offset. Besides the direct fastigio-collicular projections, other indirect pathways can be involved. One such possible pathway involving the
thalamocortical ventromedian nucleus and the frontal eye field (FEF) has been demonstrated in the cat (Kyuhou and Kawa-
guchi 1987). Another indirect pathway involves a crossed
cFN projection to the contralateral nucleus prepositus hypo-
glossi that, in turn, bilaterally projects to the SC (Hardy and
Corvisier 1996; Stechison et al. 1985). Also, there are some
indications in the literature that fixation offset could result
from alteration of the SC and/or thalamus. Indeed, a tonic
ipsilateral head deviation and severe head-orienting deficit
have been reported in the cat after a SC lesion (Isa et al.
1992). In the monkey, a moderate fixation offset was ob-
served after lesions of the SC (Keating and Gooley 1988)
and of FEF (Dias et al. 1995; Latto and Cowey 1971). A
much larger fixation error appeared when the SC lesion was
combined with a lesion of either the dorsomedial thalamic
area (Albano and Wurtz 1982) or the FEF (Keating and
Gooley 1988). In these cases, however, the fixation error
depended on target position relative to the head (Albano and
Wurtz 1982; Keating and Gooley 1988; Optican 1982). In
any case, irrespective of the routes that functionally connect
the cFN to the SC, this collicular hypothesis of cerebellar
dysmetria leads to specific predictions regarding the pattern
of collicular activity in cFN-inactivated animals. For a given
target eccentricity, the population of active presaccadic neu-
rons should not be located on the same site of the collicular
custom as the one encoding accurate gaze shifts during the
control behavior.

As an alternative or a complement to this ‘collicular’
hypothesis, muscimol inactivation of cFN may have altered
structures located downstream of the SC. Hence, contralat-
eral hypometria could be the consequence of a decreased
activity in the fastigio-reipient areas of the contralateral
pontine reticular formation devoted to the control of hori-
zontal orienting responses. In particular, as suggested in the
monkey (Fuchs et al. 1993; Ohtsuka and Noda 1995), the
activity of cFN neurons could normally help the initiation
of contraversive saccades by excitation of burst neurons and
inhibition of omnipause neurons. This hypothesis, which im-
plcitly requires some perturbation in the dynamic feedback
control (Jürgens et al. 1981; Zee et al. 1976), has some
predictions on saccade dynamics that will be tested in the
companion paper. Note that the straightness of dysmetric
gaze shifts already suggests that cFN inactivation does not
basically affect neurons that exclusively drive the horizontal
motoneurons pool. Regarding fixation offset, the fastigio-
pointo-medullary projections that terminate in the contralat-
eral medial vestibular nuclei or prepositus hypoglossi nuclei
(cat: Carleton and Carpenter 1983; McCrea and Baker 1985;
squirrel monkey: Belknap and McCrea 1988; see, however,
Noda et al. 1990 for macaque monkey) also could be in-
volved, as suggested by Ohtsuka and Noda (1995). This
offset indeed was attributed to an unbalanced tonic bilateral
activation of abducens neurons by prepositus hypoglossi
neurons. Note further that a cFN projection toward vestibulo-
nuclear neurons in the contralateral median vestibular nuclei
might account for the deviation of the head we observed in
the cat. However, beside the arguments mentioned above in
favor of an alternative, collicular hypothesis, this ‘medul-
lar’ hypothesis of fixation offset requires some adjustments.
First, the absence of a postsaccadic glissade suggests that
the cFN does not influence only the output of the neural
integrator but also contributes to the pulse of activity that
drives the eyes during the saccade. In other words, the un-
changed pulse-step matching suggests a cFN influence up-
stream from the neural integrator, at a level where pulse and
step commands have not been dissociated yet. In any case,
if the dysmetria observed after muscimol cFN inactivation
is the consequence of changes (through various pathways)
downstream of the SC, a pronounced modification of the
movement fields of tectosensory and tentorietalospinal neu-
rons should be observed. Moreover, saccades evoked by
electrical microstimulation of the SC should have different
amplitude and direction properties after cFN inactivation.

Conclusions

Saccadic dysmetria after cFN inactivation previously has
been interpreted as the result of suppression of the ‘early’
and ‘late’ saccade-related bursts recorded in primate cFN.
Our observations about ipsiversive movement hypermetria
and fixation offset more directly stress the possible role of
cFN tonic discharges. These two hypotheses predict different
effects on movement dynamics. Indeed, changes restricted
to the control of acceleration or deceleration phases likely
would be accompanied by changes in gaze dynamics. In
contrast, no modification in movement dynamics is expected
if, as suggested here, gaze shift dysmetria results from
changes in the desired displacement signal driving eye and
head movement generators. Furthermore, analysis of eye/
head coordination might indicate whether cFN acts at the
level of the eye in the orbit or at the level of both eye
and head components. These analyses are presented in the
companion paper.

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Present address of L. Goffart: Division of Neuroscience, Baylor College
of Medicine, One Baylor Plaza, Houston, TX 77025.

Address for reprint requests: D. Pélinos, Espace Et Action, INSERM
U94, 16 Ave. Doyen Lépine, 69500 Bron, France.

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