Effects of Lesions of the Oculomotor Vermis on Eye Movements in Primate: Saccades

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Takagi, Mineo, David S. Zee, and Rafael J. Tamargo. Effects of lesions of the oculomotor vermis on eye movements in primate: saccades. J. Neurophysiol. 80: 1911–1931, 1998. We studied the effects on saccades of ablation of the dorsal cerebellar vermis (lesions centered on lobules VI and VII) in three monkeys in which the deep cerebellar nuclei were spared. One animal, with a symmetrical lesion, showed bilateral hypometric horizontal saccades. Two animals, with asymmetrical lesions, showed hypometric ipsilateral saccades, and saccades to vertically positioned targets were misdirected, usually deviating away from the side to which horizontal saccades were hypometric. Postlesion, all animals showed an increase (2–to 5-fold) in trial-to-trial variability of saccade amplitude. They also showed a change in the ratio of the amplitudes of centripetal to centrifugal saccades (orbital-position effect); usually centrifugal saccades became smaller. In the two animals with asymmetrical lesions, for saccades in the hypometric direction, latencies were markedly increased (up to ~500 ms). There was also an absence of express and anticipatory saccades in the hypometric direction. When overall saccade latency was increased, centrifugal saccades became relatively more delayed than centripetal saccades. The dynamic characteristics of saccades were affected to some extent in all monkeys with changes in peak velocity, eye acceleration, and especially eye deceleration. There was relatively little effect of orbital position on saccade dynamics, however, with the exception of one animal that showed an orbital position effect for eye acceleration. In a double-step adaptation paradigm, animals showed an impaired ability to adaptively adjust saccade amplitude, though increased amplitude variability postlesion may have played a role in this deficit. During a single training session, however, the latency to corrective saccades—which had been increased postlesion—gradually decreased and so enabled the animal to reach the final position of the target more quickly. Overall, both in the early postlesion period and during recovery, changes in saccade amplitude and latency tended to vary together but not with changes in saccade dynamics or adaptive capability, both of which behaved relatively independently. These findings suggest that the cerebellum can adjust saccade amplitude and saccade dynamics independently. Our results implicate the cerebellar vermis directly in every aspect of the on-line control of saccades: initiation (latency), accuracy (amplitude and direction), and dynamics (velocity and acceleration) and also in the acquisition of adaptive ocular motor behavior.

INTRODUCTION

The dorsal cerebellar vermis (lobules VI-VII, the “oculomotor vermis”) has been long implicated in the control of saccades and pursuit based on single-unit recordings from Purkinje cells and mossy fibers (Baker et al. 1972; Helmchen and Büttner 1995; Kase et al. 1980; Llinas and Wolfe 1977; Ohtsuka and Noda 1992b, 1995; Sato and Noda 1992a; Suzuki and Keller 1988a,b), experimental electrical stimulation (Fujikado and Noda 1987; Keller et al. 1983; McElligott and Keller 1984; Noda and Fujikado 1987a,b; Ohtsuka and Noda 1991b; Ron and Robinson 1973), the effects of lesions (Keller 1988; Optican and Robinson 1980; Ritchie 1976; Sato and Noda 1992b; Straube et al. 1997b; Vahedi et al. 1995), and, more recently, in humans with transcranial magnetic stimulation and functional magnetic resonance imaging (Hashimoto and Ohtsuka 1995; Honda et al. 1997; Ohtsuka and Enoki 1998). In addition, axons of Purkinje cells from the oculomotor vermis impinge on cells in the most posterior extension of the fastigial nucleus, called the fastigial oculomotor region (FOR) (Noda and Fujikado 1987b; Noda et al. 1990; Yamada and Noda 1987), which also contains neurons that discharge in relation to saccades and pursuit. Furthermore, focal chemical lesions within the FOR also lead to specific deficits of saccades and pursuit (Goffart and Pélinson 1994, 1997, 1998; Goffart et al. 1998; Ohtsuka et al. 1994; Robinson et al. 1993, 1997).

The deficits in the control of eye movements that have been reported after lesions in the oculomotor vermis—dysmetric saccades and impaired pursuit during tracking of targets moving in a sinusoidal fashion—suggest a role for the vermis in the on-line control of the accuracy of both pursuit and saccades. But questions remain about the function of the oculomotor vermis. Most prior studies of vermal ablations also have involved the deep nuclei (Optican and Robinson 1980; Ritchie 1976). How do vermal lesions affect eye movements when the underlying FOR remains intact? Because the output of the cerebellar vermis is inhibitory, and solely to the underlying deep nuclei, one might expect vermal lesions to produce reciprocal defects to those after lesions in the FOR. Is this the case? And what might be the role of the cerebellar vermis in saccade initiation and saccade dynamics?

In addition to these “on-line” functions of the oculomotor vermis, more long-term, adaptive functions, which assure that the metrics of eye movements are accurate with respect to the stimuli that drive them, have been suggested for the cerebellum (Dean 1995; Dean et al. 1994; Houk et al. 1996; Ito 1993; Keller 1989; Optican and Robinson 1980; Schweighofer et al. 1996a,b). Such a learning capability is critical because during an eye movement, visual feedback about performance is unavoidably slow due to inherent delays in visual processing. So the initiation of smooth tracking (pursuit and vergence) and the generation of movements that are completed before there is time for visual feedback or that have dynamic characteristics that preclude visual
feedback during movement (saccades and the vestibulocu-
lar reflex) necessitate adaptive mechanisms for optimizing
visuomotor performance. The role of the oculomotor vermis
in such ocular motor learning is unknown. Accordingly, a
second purpose of the present study was to define the deficits
in ocular motor learning that appear after lesions that involve
the oculomotor vermis but spare the fastigial nuclei.

Here we report the effects of lesions of the oculomotor
vermis on saccades. Two future reports will deal with effects
of such lesions on pursuit and binocular eye movement con-
trol. Preliminary aspects of this work have been presented
in abstract form (Takagi et al. 1996a,b) at the Association
for Research in Vision and Ophthalmology and the Society
for Neuroscience.

METHODS

General experimental procedures

Experiments were conducted on three male rhesus monkeys
weighing from 5 to 6 kg. All surgical and experimental protocols
were approved by The Johns Hopkins University committee on
animal experimentation, and all aspects of their care complied with
the guidelines for veterinary care of The Johns Hopkins University
School of Medicine and of the National Institutes of Health Guide
for the Care and the Use of Animals including appropriate analgesia
after surgical procedures. During the initial period of training, mon-
keys learned to come out of their cages and to sit quietly in a
primate chair for 2–3 h. Then the animals were prepared for
chronic experiments. Under pentobarbital anesthesia using aseptic
techniques, a head plate was implanted on the skull. Small holes
for screws were made with a drill, and the heads of the screws
were placed between the inner table of the skull and the dura mater.
The head plate was fixed with dental acrylic. The head plate had
small holes for pins to hold an opaque lucite patch in front of the
eye. In a second operation, a search coil made of three turns of
Telfon-coated stainless steel wire was implanted and sutured on
the sclera of each eye in front of the insertions of the extraocular
muscles. After recovery from the second surgery, the monkey was
trained to fix on and follow targets using a water reward. During
a recording session, the monkey was seated in a primate chair with
its head fixed to the frame of the chair.

Measurement of eye movements

Eye movements were measured by the magnetic field search
coil method (Robinson 1963). The output signal from the phase
detectors was filtered with a bandwidth of 0–90 Hz, sampled by
a digital computer at 500 Hz with 12-bit resolution, and then stored
to disk for later off-line analysis. System noise limited resolution
to ~0.05°. The coil signal was calibrated by requiring the animal
to fix successively on targets at 2.5° intervals over a range of ±25°
horizontal and ±22.5° vertical. Calibration data were obtained with
one eye viewing and the other occluded. In the later off-line analy-
sis, a calibration index was obtained using a third-order polynomial
curve fit.

With this method of calibration, we cannot exclude that the
animal fixed a position slightly eccentric from that of the target
(fixation offset), but prior work suggests that such fixation offsets
are small, on the order of only a few degrees (Robinson et al.
1993).

Stimulus presentation

The target was a red square, 0.3 × 0.3°, presented on the video
monitor placed at a distance of 33 cm in front of the animal’s
eye. Animals viewed with both eyes except during calibration. For
saccades, two main paradigms were used. In the first, the target
jumped with horizontal or vertical amplitudes of 10, 20, and 30°
across the center. The target changed amplitude after every fourth
displacement. In the second paradigm, the target jumped with an
amplitude of 10°, sequentially, beginning at 20° eccentric on one
side and finishing at 20° eccentric on the other, and then going
back in the opposite direction. For vertical saccades, the target
moved up and down over the same 40° excursion. The target
jumped to its new position ~1.9 s after the eye reached the previous
target position.

Short-term adaptation of the amplitude of saccades was tested
using a double-step paradigm in which the target was displaced by
10°, and then while the monkey was making a saccade toward it,
the target was displaced backward by 3° (Melis and Van Gisbergen
1996; Straube et al. 1997a). Other variations of the target stimuli
are detailed in RESULTS.

Data collection and analysis

The amplitude, latency, and dynamic characteristics of saccades
always were determined from data sets collected on the same day.
Individual trials were displayed on a video monitor. A computer
algorithm detected the onset, peak velocity, and end of saccades
using velocity criteria after the position data were filtered digitally
(3 dB point at 112 Hz). The onset of saccades was chosen when
the eye velocity exceeded 40°/s, and the end of the rapid pulse
portion of the saccade when saccade velocity dropped to <45°/s.
The correctness of these points was verified by the experimenter.
Trials were rejected if the saccade was in the wrong direction, if
the saccade started before the target jump, or if the record could
not be marked because of blinks or other artifacts. To evaluate
dynamic properties of saccades, the position data of individual
trials was loaded into Matlab and further processed to produce
velocity and acceleration data by differentiating and filtering (3
dB point at 70 Hz). For statistical analysis, the distributions of
prelesion and early and late postlesion data were compared using
the Kruskal-Wallis one-way analysis of variance on ranks. When
there was a significant difference each distribution was compared
using Dunn’s method.

For the saccade adaptation data, we first used the least squares
method, fitting an exponential curve to the values of saccade ampli-
tude as a function of trial number during the training period. Then
the initial value and final value at 200 trials were used to calculate
the percent change during the training period. Although systematic
differences were seen in the adaptation data, the relatively small
changes and the large amount of variability made a standard statisti-
cal approach difficult. Therefore a nonparametric procedure was
used to compare regression lines (Connover 1980). Because the
form of the adaptation curve is not known (although often assumed
to be a decaying exponential), we wished to make the assumption
only that it is monotonic (i.e., the saccade gain increased or de-
creased steadily with each trial). Under this assumption, a regres-
sion was performed on the ranks of the data. A r-test then was
performed to test for differences between slopes of the regression
lines before and after adaptation (Glantz 1992).

Cerebellar lesions

The cerebellum was lesioned under pentobarbital anesthesia us-
ing aseptic techniques. The midline of the dorsal cerebellum was
exposed through a suboccipital approach, and a lesion centering
around lobules VI–VII was made by cauterization and aspiration.
Corticosteroids and antibiotics were administrated for 1 wk after
the surgery. Monkeys showed no defects in their general neurologi-
cal performance, and recording of eye movements was initiated
within the first week after surgery.
**Histological confirmation**

After the experiments were completed, the animals were killed with an overdose of pentobarbital and perfused with 0.1 M phosphate-buffered saline containing 1% sodium nitrite as wash, followed by 4% paraformaldehyde. The brains were hardened for 1 wk and then examined grossly to determine the approximate location of the lesions. Subsequently, the tissue was sectioned every 25 μm and every fifth section stained with cresyl violet. The sections were compared with those of a normal brain. We used the atlas of Madigan and Carpenter (1971) as a guide for the interpretation of the extent of lesions.

**RESULTS**

**Extent of lesion: functional-anatomic correlations**

Figure 1 shows the reconstruction of the lesions based on histological examination, with the sagittal extent of the lesions being referenced to Fig. 1, bottom. The black areas reflect either absence of tissue or areas in which neurons appeared destroyed or white matter disrupted. For monkey 1 (M1), the lesion was nearly symmetrical, being deepest in the midline where lobules VII and VIII were lesioned completely. Lobules VI and IX were partially involved. The lesion extended laterally to section 3 on both sides, just encroaching on the simple lobule. The lateral component of the lesion on the left side was slightly more extensive (compare left and right sides in sections L3 and R3). For monkey 2 (M2), near the midline lobules VI–VIII were lesioned on both sides though the lesion was deeper on the right at the R1 level. The lesion, however, extended slightly more laterally on the left side. For monkey 3 (M3), on the midline lobule VII was completely lesioned but there was mild sparing in lobule VI and moderate sparing in lobule VIII. Both near the midline and more laterally, the lesion was more extensive on the right side and reached the paravermis. In all three monkeys, the cerebellar deep nuclei were spared including the posterior portion of the fastigial nucleus. Only in monkey 1 was there some gliosis at the very posterior tip of the fastigial nucleus though the neurons appeared intact.

Because of the known variability in the location of the oculomotor vermis (usually taken as lobules VI and VII in monkeys [Noda and Fujikado 1987b]), it is difficult, without recording from or stimulating neurons in the vermis, to know exactly where the oculomotor vermis was in each of our animals. Furthermore, portions of the paravermis were variably lesioned in our animals, and part of lobule VIII was lesioned in every animal. Lobule VIII is not usually considered to be part of the oculomotor vermis, but it may be that some of our findings might be attributed to involvement of this structure. At any rate, for the purposes of placing the somewhat variable results among animals in context, we propose the following scheme. Based on the pattern of asymmetry of saccadic dysmetria and the trajectories of attempted vertical saccades, monkey 1 behaved as if it had a bilateral lesion and monkeys 2 and 3 behaved as if each had a predominantly right-sided lesion.

**Fixation**

Animals could hold fixation after surgery without spontaneous or gaze-evoked nystagmus. In complete darkness without a fixation target, monkeys 2 and 3 tended to keep the position of their eyes in the upper-left field of gaze, ~20° from straight ahead. With a fixation target straight-ahead, they appeared to be able to hold their eyes on target, though often when a blink occurred, the eyes would deviate slowly a few degrees to the left of the target followed by a corrective saccade back to the target.

**Saccade metrics**

Saccade metrics will be discussed in terms of accuracy, latency, velocity, and acceleration. These data were obtained from the same set of saccades and hence permit comparison and correlations among various saccade parameters at a given time following the lesion.

**SACCADE ACCURACY.** A uniform finding was a change in saccade accuracy. Figure 2 shows 10 consecutive traces of horizontal saccades from monkey 1 before and 14 days after the cerebellar lesion. The target displacement was between right and left 10° on the horizontal meridian. Before the lesion, the gain (amplitude of the initial saccade/target displacement) was 0.97. The variability in amplitude from trial to trial was small (25–75% range for saccade amplitude was 2.5°). After the lesion, the gain was reduced to 0.51. The variability in amplitude was larger (25–75% range, 4.8°).

A quantitative analysis of saccade dysmetria for all three monkeys is shown in Fig. 3. For horizontal saccades, two data sets were obtained before the lesion (□), and the results from each were similar. Early postlesion data (■) were obtained at 14, 12, and 3 days after surgery for horizontal saccades and 9, 13, and 12 days after surgery for vertical saccades for monkeys 1–3, respectively.

For all animals, the effect of the lesion was greater on the accuracy of horizontal than on vertical saccades. For monkey 1, horizontal saccades (Fig. 3) became hypometria in both horizontal directions though the degree of hypometria was slightly greater for leftward saccades to the 30° target displacement. The gain was less for larger target displacements so that the range of amplitudes of the primary saccades to the three different target displacements was small (range of ~3.3° for leftward saccades and 6.2° for rightward saccades). In monkey 1, vertical saccades also became hypometric; for upward saccades, the gain was relatively lower for larger target displacements and for downward saccades, relatively lower for the smaller target displacements.

Monkey 2 showed hypometria to the right and hypermetria to the left, with the gain change greater for the larger target displacements in both horizontal directions. Monkey 2 showed only small changes in the accuracy of vertical saccades; upward saccades to smaller target displacements became slightly hypermetric.

Monkey 3 developed hypometric saccades to the right, with lower gains for the larger target displacements. There was also slight hypometria to the left for the smaller target displacements. Vertical saccades essentially were unchanged.

Variability often increased after the lesion as reflected in the increase in size of the error bars in Fig. 3. The increase in variability tended to increase with the degree of hypometria, and the increase was less when saccades became hypermetric. For example, when saccade gains were as low as...
FIG. 1. Extent of lesion in monkey 1 (M1), monkey 2 (M2), and monkey 3 (M3). Sagittal sections of vermis and paravermis (Nissl stain). Extent of the lesion is indicated by the black area on the template of normal anatomy. Lateral extent of the sections is depicted on the dorsal view (Fig. 1, bottom). Middle far left section: on the midline; top sections: on the left side; bottom sections: on the right side. VI, VII, VIII are vermis lobules. FN, fastigial nucleus; AIN, anterior interposed nucleus; PIN, posterior interposed nucleus; SL, simple lobule; PML, paramedian lobule. Distribution of the lesions is discussed in the text.
0.5, the 25–75% range of values could increase by as much as four to five times. Even when saccade accuracy for a given target displacement was unchanged, however, there was still an increase in trial-to-trial variability (on average about twofold).

Figure 3 also shows data collected in the late postlesion period (82–114 days). All animals showed some recovery in saccade amplitude, though dysmetria was particularly enduring for rightward saccades in monkeys 1 and 3.

We next examined the effect of the starting and ending positions of the eye in the orbit on the amplitude of saccades made in the same direction with a particular emphasis on centripetal-centrifugal differences using eccentric target displacements between 10 and 20°. Before surgery, animals showed either no or only a minimal orbital-position effect; usually centrifugal saccades were slightly smaller than centripetal saccades. For all animals, the centripetal (cp)–centrifugal (cf) ratios for horizontal saccades ranged between 0.93 and 0.97 (median = 0.95) and for vertical saccades between 0.89 and 1.02 (median = 0.96).

After surgery, there were variable changes in the orbital-position effects on saccade size. Monkey 1 developed a relative centrifugal hypometria for both horizontal and vertical saccades (e.g., for horizontal saccades, the cp/cf ratio dropped from 0.94 to 0.85). Monkey 3 developed a relative centrifugal hypometria for its rightward (hypometric) saccades (cp/cf ratio dropped from 0.96 to 0.79). In contrast, monkey 2 showed a diminution of its prelesion orbital position effect.

SACCADE LATENCY. Data for saccade latencies are shown in Fig. 4. For monkey 1, with bilateral hypometria there...
was little change in saccade latency. On the other hand, for monkeys 2 and 3 with an asymmetrical hypometria, the latencies of rightward horizontal saccades (the same direction as their hypometria) were considerably prolonged and also more variable. There were no consistent changes in the latency for vertical saccades. Monkey 2 showed some recovery in saccade latency in the late postlesion period, but the values were still much greater than prelesion. For example, performing a linear regression on the data in Fig. 4, the latency for saccades to 30° target displacements was 240 ms before the lesion, 524 ms in the early postlesion period, and 328 ms in the late postlesion period. For monkey 3 there was no recovery in saccade latency. The latency for saccades to 30° target displacements was 237 ms before the lesion, 478 ms in the early postlesion period, and 493 ms in the late postlesion period. In monkey 2, the recovery of saccade latency roughly paralleled the recovery of saccade amplitude. In monkey 3 there was little recovery in either. There were several other features of the changes in latency shown by monkeys 2 and 3. First, after the lesion there was an exaggeration of a prelesion trend for saccade latency to increase with saccade amplitude. Second, saccade latency was better correlated with target displacement than with saccade gain. The latency–target displacement correlation for monkey 2, rightward saccades, was \( r = 0.62, P < 0.01 \) and for monkey 3, rightward saccades, \( r = 0.48, P < 0.01 \). In contrast, when the latency–gain correlation was calculated for each target displacement, it was significant only for monkey 3, and then only for saccades to a 30° target displacement (\( r = -0.44, P < 0.05 \)). Finally, there was an orbital-position effect for saccade latency. For both monkeys 2 and 3, for rightward saccades, centrifugal saccades were more delayed than centripetal saccades. The difference in median latency between 10–20° centrifugal saccades and 20–10° centripetal saccades increased from 31 ms prelesion to 85 ms postlesion for monkey 2 and from 16 ms prelesion to 144 ms postlesion for monkey 3. The change was due to an increase in latency for centrifugally directed saccades.

For monkeys 2 and 3, we evaluated the ability to make express saccades (saccades appearing with a latency in the range of 100–120 ms) and anticipatory saccades (saccades appearing with a latency of <80 ms in a predictive paradigm; Fig. 5). Using a gap (200 ms) between fixation target offset and peripheral target onset, with a nonpredictive target direction (right or left) and amplitude (10 or 20°), we found that express saccades (peaking at \( \sim 110 \) ms) only occurred in the leftward (relatively normal) direction for both monkeys. Likewise, in a predictive paradigm (0.5 Hz, 200-ms gap, 20° amplitude), we found that anticipatory saccades (latency <80 ms) and express saccades only occurred in the leftward direction.

SACCade DynAmics. Figure 6 presents the peak velocity–amplitude relationship (main sequence) (A) and duration–amplitude relationship (B) for horizontal saccades. Using the least-squares method, the data usually could be fit to an exponential using the following formula, \( V_{pk} = K[1 + \exp(Amp/Le)] \) (where \( V_{pk} = \) peak velocity, \( Amp = \) saccadic amplitude, \( Le = \) angle constant, \( K = \) constant). In monkeys 1 and 2, horizontal saccades became slow bilaterally but the speed of vertical saccades was slightly increased (monkey 1) or not affected (monkey 2). For monkey 3, smaller rightward saccades became slightly slow but there was no change in the peak velocity for leftward or vertical saccades. Note that even postlesion, the data points cluster tightly around the exponential fit, implying no increase in variability of the relationship between the peak velocity and the amplitude of saccades. Changes in the amplitude-duration relationship for horizontal saccades are shown in Fig. 6B; there was an increase in the duration of saccades for monkeys 1 and 2. As for peak velocity, however, there was no increase in the variability of the duration of saccades for a given amplitude. This finding contrasts with the increase in variability of saccade amplitude and latency.

We found no orbital-position effect for the relationship between saccade size and either duration or peak velocity. There was, however, a mild orbital-position effect for saccade acceleration. For monkey 2, prelesion, peak acceleration for 10° centrifugal saccades was 80% of that for 10° centripetal saccades, while postlesion it was only 66%. For monkey 3, prelesion the value was 90% and postlesion 62%. In neither animal could this effect be attributed simply to a
difference in amplitude between centripetal and centrifugal saccades because the changes in acceleration were relatively so much larger than changes in amplitude.

In the late postlesion period, monkey 1 showed complete recovery of peak velocity for rightward saccades and a 50% recovery for leftward saccades. Monkey 2 showed no recovery for rightward saccades and ~50% recovery for leftward saccades. Monkey 3 showed no recovery in peak velocities for rightward saccades. Just as there was a dissociation between the immediate effects of the lesion on the accuracy of saccades and on their speed, the patterns of recovery of peak velocity of saccades were quite different from those of saccade accuracy.

To analyze further the changes in saccade dynamics, we plotted average traces for position, velocity, and acceleration as a function of time after saccade onset for 10 horizontal saccades of the same size, independent of target amplitude (Fig. 7). These are the same saccades from which changes in saccade amplitude were calculated (Fig. 3). For monkey 1, the acceleration of the eye was affected both in its maximum amplitude and in its initial rate of rise ("jerk"), though the duration of the acceleration period was unchanged. The amplitude of the deceleration of the eye toward the end of the saccade especially was diminished, resulting in an increase in the duration of saccades with a long tail in the velocity profile. For vertical saccades, there was little change in saccade dynamics in spite of the changes in saccade accuracy.

For monkey 2, saccade acceleration and deceleration also were decreased in both horizontal directions even though in this case rightward saccades had become hypometric and leftward saccades hypermetric. The very initial portion of the saccade (1st 10–20 ms) had a normal rate of rise in acceleration (jerk), though both the duration and the maxi-
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FIG. 4. Horizontal saccade latency (ordinate) as a function of saccade amplitude (abscissa). Positive amplitudes are for rightward and negative for leftward saccades. Prelesion and early postlesion results are shown. Data also are sorted by the size of the target displacement as shown in the legend on the figure. Regression lines are drawn through the pre- and postlesion data. Panels are for monkeys 1–3 (M1–M3). See text.

maximum amplitude of eye acceleration were decreased. Again, the relatively larger effect on both the duration and amplitude of deceleration was associated with saccades with an increased duration and a long tail in their velocity profile. There were no changes in vertical saccade dynamics.

Monkey 3 had a slight increase in the maximum amplitude of eye acceleration and eye deceleration for leftward 20° horizontal saccades. There was a slight decrease in the duration of the acceleration period. There was no change in vertical saccade dynamics.

To summarize, for horizontal saccades, all monkeys showed some change in saccade dynamics after the lesion in the cerebellar vermis. The effect was most striking in monkeys 1 and 2 with a decrease in both the acceleration at the beginning of saccades and the deceleration at the end of saccades. There were differences among monkeys in the effects of the lesion on saccade acceleration, variously altering the rate of rise of acceleration (jerk), the value for peak acceleration, and the duration of the acceleration period. The dynamics of vertical saccades were largely unaffected by the lesions.

SACCADE TRAJECTORIES. In addition to changes in saccade dynamics there were also striking changes in saccade trajectories. Before the lesion, saccades made between vertically displaced targets were accurate (Fig. 8, top), though there were high-frequency oscillations in horizontal velocity during purely vertical saccades. Postlesion both monkeys 2 and 3 developed an inappropriate horizontal component to the
**A** \textbf{RANDOM PARADIGM}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{random_paradigm}
\caption{Latency for horizontal saccades from monkey 3 (M3), postlesion, in response to random 20° target jumps, random 20° target jumps with a gap (200 ms), and predictive 20° target jumps with a gap (200 ms). Abscissa, saccade latency; left ordinate, saccadic amplitude corresponding to the data points; right ordinate, frequency corresponding to the histogram. Note the difference between right and left saccades with express saccades and anticipatory saccades only occurring for leftward target displacements.}
\end{figure}

left (Fig. 8, bottom). A horizontal corrective saccade occurred at a latency of 200–400 ms.

**Saccade adaptation**

Figure 9 shows the response of monkey 2 in the decreasing saccade adaptation paradigm. The target jumped from right 3.5° to left 6.5° (leftward 10°) and then jumped back by 3° during the initial saccade to the target. In Fig. 9, left, are the early (top) and late (bottom) responses during the training period. Prelesion, after training, the size of the primary saccade had diminished. When adaptation was tested 22 days postlesion, there was an increase in variability of response...
and an increase in latency to the secondary, corrective saccades. During training there was no decrease in primary saccade amplitude (Fig. 9, right). The relatively increased latency for corrective saccades, however, decreased during training. Figure 10 (monkey 2) shows the progressive change in saccade amplitude during the entire adaptation period for rightward primary saccades (target initially displaced to the right and then jumped back to the left; top) as well as for leftward primary saccades (bottom, same data set as in Fig. 9). The size of the primary saccade at the onset of training was similar in the pre- and postlesion data sets (see initial value at the bottom of each figure). Prelesion there was about a 10% drop in primary saccade amplitude during training. In the early postlesion data (post 1), there was an increased variability of saccade size and a lack of a decrease in the amplitude of the primary saccade with training (percent changes are shown at the bottom of each figure, compare pre- and postlesion). In the late postlesion test (51 days postlesion, post 2), the amplitude of the primary saccade again decreased with training to about the same degree as prelesion (~10%), implying a recovery of adaptive capability. The increased variability in the amplitude of the primary saccade, however, was still present.

Data on saccade adaptation for all three animals are
shown in Fig. 11. All three monkeys had a deficit in adaptive capability for horizontal saccades. It is worth noting that the adaptation defect could be present even when the amplitude defect had recovered. This was the case, for example, for *monkey 1*, leftward saccades. The initial amplitude during adaptation training prelesion was 9.37° and postlesion was 9.42°, yet there was a clear difference in adaptive capability. Conversely, some adaptation could take place even when the initial saccade was hypometric. This was the case, for example, for *monkey 3*, leftward saccades. The initial amplitude during adaptation training prelesion was 9.42° and postlesion was 8.33°. In *monkey 3*, we also could test vertical saccade adaptation; it was impaired for downward saccades.

To assess the significance of these changes in adaptive capability, especially in view of the considerable variability of response and the relatively small changes in amplitude, we applied a nonparametric procedure to compare the slopes of regression lines fit to the data (see METHODS). There were no significant differences between the two prelesion data sets for horizontal saccades in any monkey. An asterisk next to the early (post1) postoperative data bar indicates a statistically significant difference (*P* < 0.05) between the slopes from the pre- (pre2) and postoperative (post1) data. Not
unexpectedly, the percent change in saccade amplitude after training (as determined from the exponential fit) did not always accord with the statistical measure of the difference in slopes because the choice of an exponential fit to describe the time course of adaptation was arbitrary and subject to the degree of variability. Nevertheless, both methods show some degree of change in adaptive capability in each monkey after the vermal lesion.

Data on the changes in the latency of corrective saccades during saccade adaptation in the early postlesion state are shown in Fig. 12. Monkey 2, with prolonged latencies of corrective saccades in both horizontal directions at the beginning of the adaptation period, showed a considerable decrease in latency by the end of the adaptation period. This occurred in spite of a lack of adaptation of saccade amplitude. Monkeys 1 and 3 showed a similar but smaller effect.

**DISCUSSION**

The main finding of this study is that lesions restricted to the dorsal vermis of the cerebellum (‘‘the oculomotor vermis’’), lead not only to changes in the accuracy of saccades, as has been noted previously, but also to changes in the latency and dynamics of saccades, findings that were relatively unsuspected based on previous work. These effects were independent of any structural damage to the deep cerebellar nuclei, which implicates the cerebellar cortex directly in almost every aspect of the control of saccades. First, we will compare our results to those of previous studies of cerebellar lesions or inactivation. Then we will interpret our findings in light of the current hypotheses about the role of the cerebellum in the control of saccades. Finally, we will discuss issues related to saccade adaptation and the cerebellar vermis.

**Comparison of the present findings with those from previous studies of the effects of lesions of the cerebellar vermis and the fastigial nuclei**

**SACCADE AMPLITUDE.** The effects of our lesions on saccade accuracy are in basic agreement with previous studies (Aschoff and Cohen 1971; Sato and Noda 1992b; Vahedi et al. 1995; Waespe and Müller-Meissner 1996), if one assumes that the lesion in monkey 1 was relatively symmetrical.
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FIG. 7. (continued)

and in monkeys 2 and 3 asymmetrical and larger on the right side. Based on histological examination, this appeared to be the case. Hypometria is the main finding with vermal lesions, usually most prominent for centrifugal saccades. This pattern contradicts the report of Ritchie (1976), who found various patterns of saccadic hypermetria and hypometria in monkeys with dorsal vermis lesions. Most of the data shown in his paper, however, are from a monkey in which there was also extensive involvement of both fastigial nuclei. Hence, attributing functions to the oculomotor vermis using the results of Ritchie may be incorrect.

Our results are also compatible with the idea that lesions in the oculomotor vermis produce disturbances of saccade amplitude that are the reciprocal of those defects with lesions in the underlying fastigial nuclei (Büttner et al. 1994; Goffart and Pélisson 1994; Goldberg et al. 1993; Ohtsuka et al. 1994; Optican and Robinson 1980; Robinson et al. 1993; Vilis and Hore 1981). This is not surprising because the saccade-related activity of vermal Purkinje cells (which is inhibitory) and the results of experimental stimulation are the reciprocal to those of the neurons in the underlying FOR (Fuchs et al. 1993; Fujikado and Noda 1987; Hashimoto and Ohtsuka 1995; Helmchen and Büttner 1995; Helmchen et al. 1994; Keller et al. 1983; McElligott and Keller 1984; Noda et al. 1988; Ohtsuka and Noda 1991a, b, 1992a, 1995; Ohtsuka et al. 1994).

Goffart and Pélisson (1994, 1998) studied the effect of unilateral muscimol inactivation of the caudal fastigial nucleus on relatively large gaze (eye plus head) shifts in the head-unrestrained cat. As is the case for saccades in the usually most prominent for centrifugal saccades. This pattern contradicts the report of Ritchie (1976), who found various patterns of saccadic hypermetria and hypometria in monkeys with the head fixed, they found that contralateral gaze shifts became hypometric and ipsilateral gaze shifts became hypermetric. The pattern of dysmetria, however, was different for the two directions. The change in gain for contralateral, hypometric shifts in gaze was independent of the positions of the gaze in space and the target relative to the head, suggesting that the shifts in gaze were encoded in a retinal or motor-error frame of reference. On the other hand, the change in amplitude for ipsilateral, hypermetric gaze shifts depended on the positions of the gaze in space and the target relative to the head and could be attributed to a bias in the amplitude of the gaze shift. This finding suggested that the ipsilateral shifts in gaze were goal directed, albeit to an erroneous one. Whether or not a similar process occurs in the monkey with FOR inactivation is not yet settled. Nevertheless experimental stimulation studies have suggested such a goal-directed function for the cerebellum (McElligott and Keller 1984; Ron and Robinson 1973).

As in many previous studies of cerebellar disease in patients (Versino et al. 1996) and in many of the experimental studies described in the preceding text, our lesioned
FIG. 8. Saccades to vertically displaced targets are shown for up and downward movements, prelesion and early postlesion for monkey 3. Ten to 20 saccades are shown. Horizontal and vertical eye positions are plotted against each other. Velocity trace also is shown and aligned with saccade onset. Vertical arrow, correct direction of the saccade. Note that prelesion there are some horizontal oscillations at a high frequency during the pure vertical saccades (best seen on the horizontal velocity traces). Postlesion, saccades are biased to the left for upward saccades (bottom left) especially toward the end of the saccade. Downward saccades show a deviation to the left with the initial part of the saccade. Any corrective saccades needed to bring the eye to the target are reflected in the velocity traces late in the trial. H and V capped with filled dots indicate horizontal and vertical velocity.

monkeys showed a change in the effect of the position of the eye in the orbit on saccade amplitude. The usual postlesion pattern for a target displacement of a given amplitude was for centrifugally directed saccades to become relatively (compared with prelesion performance) smaller than centripetally directed saccades, but in monkey 2 the opposite occurred. With respect to the findings of Goffart and Pélisson (1994, 1998a), it is difficult to use our results either to support or refute the hypothesis that the cerebellum is involved with programming goal-directed saccades. The lesions in our monkeys usually led to hypometric saccades, whereas Goffart and Pélisson found that only hypometric saccades appeared goal directed. The fact that the lesions in our animals were chronic, bilateral (albeit asymmetrical) and involved vermal and possibly some paravermal cortex precludes using our data to draw definitive conclusions about a role for the oculomotor vermis in producing goal-directed saccades.

SACCade Dynamics AND Trajectories. Our animals showed changes in saccade dynamics, which in two animals were striking. Not only were peak velocity–amplitude relationships affected but even more prominent changes were seen in the acceleration of the eyes at the beginning of saccades and, especially, in the deceleration of the eyes at the end of saccades. In the two animals with the more asymmetrical lesions, we also found distorted saccade trajectories with vertical saccades deviating away from the side toward which horizontal saccades had become hypometric. We did not, however, find an orbital-position effect for saccade peak velocity or duration, though there was an orbital-position effect for saccade peak acceleration. The pattern of changes in saccade dynamics and trajectories is similar to that reported for animals with chemical inactivation of the FOR, though, of course, the direction of hypometria and deviation are opposite relative to the side of the lesion (Goffart et al. 1998; Robinson et al. 1993).
SACCADE LATENCY. Changes in saccade latency were a prominent finding in the two animals (monkeys 2 and 3) that developed asymmetrical dysmetria. There was also a pronounced orbital-position effect for the change in saccade latency; centrifugal saccades became more delayed. The animals also showed defects in generating express and predictive saccades. Until recently (Goffart and Pélisson 1997; Waespe and Müller-Meisser 1996), defects in the initiation of saccades were not considered part of the oculomotor syndrome of the cerebellum. In the work of Robinson et al. (1993), no consistent changes in saccade latency were found. There are hints, however, of disturbances in saccade latencies in the work of Sato and Noda (1992b) in which they described animals having difficulty initiating saccades in the hypometric direction after functional ablation of the oculomotor vermis. Goffart and Pélisson (1997) reported that after unilateral inactivation of the FOR, contralateral, hypometric gaze shifts were delayed with the increase in latency proportional to the decrease in gain. Ipsilateral gaze shifts were initiated earlier than normal, but the decrease in latency was independent of the increase in gain. Why similar changes in saccade latency have not been reported with FOR inactivation in monkeys is unexplained. Perhaps, the degree of stimulus predictability may play a role. Our results, however, clearly indicate that a lesion in the oculomotor vermis of the monkey can influence the time to initiate saccades, if the saccade is hypometric and if the dysmetria is asymmetrical. It is worth noting again that although the changes in saccade latency in our monkeys roughly paralleled those in saccade amplitude, there was no relationship between changes in saccade latency (or amplitude) and saccade dynamics.

How does the cerebellum influence the generation of saccades?

Our results suggest that the oculomotor vermis can influence saccade latency, accuracy, trajectory, and dynamics; in other words, every key aspect of the generation of saccades. It appears to affect these various properties relatively independently; only the changes in saccade initiation and accuracy seemed to vary together, and then only when the hypometria was asymmetrical because there was no change in latency associated with a bilateral lesion that produced bilateral hypometria. Because the influence of the oculomotor vermis on the generation of saccades is almost certainly mediated by its sole projection site and conduit, the FOR, the question becomes how does the FOR influence the mechanisms within the brain stem and superior colliculus that produce the saccadic pulse. These issues have been considered by others (Dean 1995; Fuchs et al. 1993; Goffart and Pélisson 1998; Goffart et al. 1998; Keller 1989; Ohtsuka and Noda 1995; Quaia et al. 1996; Robinson et al. 1993); here we will only review features relevant to the findings in the present study.

SACCADE ACCURACY AND THE CEREBELLM. It is worth remembering that the “default” value of saccade accuracy after total cerebellectomy and bilateral FOR inactivation (Robinson et al. 1993) is extreme hypermetria (Büttner and
FIG. 10. Time course of adaptation for monkey 2 in gain-reduction double-step paradigm for rightward initial target jumps with a backward target jump (top) and for leftward initial target jumps (bottom, same data as Fig. 9). Note that the increased variability of response, as reflected in the scatter of amplitude values, in the early postlesion period (post 1), persists even in the late postlesion period (post 2). On the other hand, the deficit in adaptation of saccade amplitude in the early postlesion period recovered in the late postlesion testing period. Initial value and percentage change in saccade amplitude after training are based on an exponential fit and are shown at the bottom of each figure. See text.

Straube 1995; Optican and Robinson 1980). Hence, a clear function of the FOR must be to overcome the inherent hypermetria of the brain stem saccadic pulse generator. Presumably the FOR acts to stop the saccade when it reaches its desired position and thus prevents hypermetria. It has been suggested that the FOR computes the instantaneous position of the eye during the saccade by a mathematical integration of a velocity command, presumably coming from pons. Using this estimate of where the eye is and also a knowledge of the desired eye position derived from the superior colliculus via the nucleus reticularis tegmenti pontis, the FOR can apply a “brake” or a “choke” to stop the eye when it reaches the desired position. This inhibition could be mediated by virtue of a projection of FOR to inhibitory burst neurons within the brain stem saccadic pulse generator (Quaia et al. 1996; Van Gisbergen et al. 1981) or by projec-
FIG. 11. Percentage change in saccade amplitude after training in gain-reduction double-step paradigm for all 3 monkeys (M1–M3) in the early and late postlesion period. The initial and final values (at the end of training) were based on exponential fits to the data as shown in Fig. 10. For horizontal saccades, note the bilateral changes in monkeys 1 and 2 and the change for rightward saccades in monkey 3. Vertical adaptation also was studied in monkey 3 (M3) and was found to be impaired in the downward direction. *Statistically significant difference (P < 0.05) in the slopes of the regression lines fit to the pre2 and post1 data. Data for early postlesion adaptation were taken within the 1st month after surgery (except monkey 3, vertical at 41 days) and for late postlesion adaptation, in the late 2nd to 3rd month after surgery.

saccades and the cerebellar oculomotor vermis

In addition to saccade hypometria, we also found that the trial-to-trial variability of saccade gain was increased after the vermal lesion. It has been suggested that the cerebellar cortex plays a role in synchronizing activity of FOR neurons so that saccades are consistently accurate (Ohtsuka and Noda 1995). One might then expect—and this is what we found—that the animals with more marked hypometria would show larger effects of the loss of the influence of Purkinje cells on activity in the FOR and hence more trial-to-trial variability in saccade amplitude.

saccade dynamics and the cerebellum. How might the cerebellum influence saccade dynamics. The timing of the discharge of neurons in both the FOR and oculomotor vermis is such that they are poised to influence the acceleration of saccades in one direction and the deceleration of saccades in the other (Fuchs et al. 1994; Helmchen and Büttner 1995; Helmchen et al. 1994; Ohtsuka and Noda 1991a, 1995; Ohtsuka et al. 1994). In both cases, however, the effect would likely be on the same set of burst neurons. Defects in this cerebellar input to brain stem burst neurons could lead to a decreased acceleration, decreased deceleration, and increased duration of saccades, which is what we found. Based on the dissociation of the findings in our animals—that changes in saccade dynamics and saccade accuracy can be independent—the dorsal ver-
mis may be able to influence the output of the FOR so that the calculation of when (during the saccade) the FOR should act to stop the movement, which would determine saccade accuracy, could be separate from the effect on the total amount of FOR activity being projected to the brain stem, which would determine saccade dynamics. Critical to confirming such a scheme will be the anatomic localization of the "resettable" mathematical integrator, which provides the signal needed for determining when the eye has reached its desired position. The cerebellum, brain stem, and superior colliculus have all been suggested as possible loci (Handel and Glimcher 1997; Kaneko 1996, 1997; Kokkoroyannis et al. 1996; Kustov and Robinson 1995; Nichols and Sparks 1995; Quaia et al. 1996; Waitzman et al. 1996).

The changes in horizontal position associated with saccades to vertically displaced targets also can be considered in light of activity of horizontal excitatory burst neurons (EBN) during vertical saccades. Behavioral evidence for activity of horizontal EBN during vertical saccades is the horizontal oscillations that occurred during pure vertical saccades in our monkeys prelesion. Such a finding also has been reported in normal humans. After the lesion in the vermis, two of our animals (monkeys 2 and 3) developed a horizontal bias that was associated with vertical saccades. The bias was in part dynamic, leading to curved saccades, and also directional, leading to horizontally inaccurate saccades. This dynamic change during the saccade presumably reflects the exaggerated and asymmetric effect of imbalanced FOR inputs—both in the acceleration and deceleration phases of the saccade—on the brain stem burst neurons during vertical saccades. This pattern of oblique saccade dysmetria also reflects a disturbance in the directional accuracy of saccades, which could be dissociated from the effects of the cerebellar lesion on saccade dynamics.

**SACCADe INITIATION AND THE CEREBELLUM.** A striking finding in our study was the increase in latency for saccades in the hypometric direction in the animals that had an asymmetrical dysmetria. Imbalanced tonic FOR inputs to pause cells might sustain pause cell activity inappropriately and so delay saccade initiation. Or when pause cell inhibition is lifted to initiate the saccade, imbalanced FOR outputs would gain access to the brain stem burst neurons and so bias these neurons to facilitate initiation of saccades in one direction and/or retard it in the other. Another possibility would be that changes in saccade latency and defects in production of express saccades might be related to cerebellar projections to the superior colliculus, which has cells that encode activity related to the initiation of saccades including the generation
of express saccades (Dorris et al. 1997). The change in the orbital-position effect for latency also might be related to imbalanced cerebellar outputs, perhaps to the superior colliculus, which also has position-related activity though primarily in its most rostral portion (Van Opstal et al. 1995).

It is clear from these considerations that the cerebellum could influence saccade accuracy, dynamics, and initiation in multiple ways at multiple sites. Furthermore, the cerebellum could act on brain stem burst or pause neurons in a feedforward pathway, in parallel with descending saccade-related signals from higher centers including the superior colliculus. Or the cerebellum could feedback onto the superior colliculus and so influence descending signals from the superior colliculus that are destined for brain stem burst and pause neurons. Or the cerebellum could do both, for example, influencing the dynamics of saccade by virtue of direct projections to the brain stem and influencing the initiation and accuracy of saccades via projections to the superior colliculus. Clearly, to understand the rather striking changes in saccade latency, accuracy, and dynamics that occur after lesions of the cerebellar vermis, the specific anatomic connections and the electrophysiology of the cerebellar projections to the brain stem pulse generator and the superior colliculus need to be elucidated.

Saccade adaptation

For more than 25 years, the cerebellum has been implicated in various types of ocular motor learning including saccadic eye movements (Dean 1995; Dean et al. 1994; Goldberg et al. 1993; Houk et al. 1996; Optican and Robinson 1980; Optican et al. 1986; Raymond et al. 1996; Schweighofer et al. 1996a,b; Straube et al. 1995; Waespe 1995; Waespe and Müller-Meissner 1996). One contentious issue has been the relative roles of the cerebellar cortex and the underlying deep nuclei in ocular motor learning. One part of the cerebellar cortex, the flocculus/paraflocculus, has been shown to have a role in the adaptive control of the amplitude of the vestibulocular reflex (Lisberger et al. 1984) and of the pulse-step match of innervation that prevents post-saccadic drift (Optican et al. 1986). This adaptation is probably mediated via projections from the flocculus to the brain stem. Our study addresses the issue of differential function in saccade adaptation of the oculomotor vermis and the underlying fastigial nucleus.

ADAPTATION OF SACCADe AMPUTUcE. Goldberg et al. (1993) reported in a single monkey with a large bilateral lesion of the deep nuclei that adaptation of saccade amplitude in a double-step paradigm was impaired. Waespe (1995) studied saccade adaptation with a similar paradigm in patients with Wallenberg’s syndrome who had lesions in the lateral medulla. He found a loss of the capability for saccade adaptation. In these patients, the climbing fiber input to the cerebellum is interrupted. This is thought to result in an increase in the simple-spike activity of Purkinje cells, resulting in a functional ablation of the ipsilateral fastigial nucleus. Waespe attributed the defect in adaptation to a decrease in the activity in the fastigial nucleus and to the loss of the climbing fiber input from the inferior olive, which presumably provides an error signal necessary for adaptation (Raymond et al. 1996). With respect to the cerebellar cortex, Waespe and Müller-Meissner (1996) reported a decreased adaptive capability in a patient with a lesion that presumably involved the dorsal cerebellar vermis. The defect in adaptation of saccade amplitude in our lesioned monkeys also indicates that the cerebellar vermis plays a role in ocular motor learning.

One possible explanation for the impaired learning capability in our monkeys is that saccade amplitude became so variable after the lesion that whatever structure mediates saccade adaptation no longer had access to a consistent error signal on which it could program the correct adaptive response. In other words, the loss of adaptation could be an epiphenomenon, secondary to the increased variability of saccade size after the lesion. Although the increase in variability of saccade amplitude could be a factor in the decreased adaptive capability, we found a clear dissociation in the variability of the amplitude of saccades and adaptive capability in monkeys 2 and 3 (an example is illustrated in Fig. 10, in which adaptation recovers but the scatter in saccade amplitudes is unchanged). This finding argues that increased variability is not the sole explanation for the adaptation defect.

Two of our monkeys showed another type of adaptation to saccade dysmetria. Although the amplitude of the primary saccade was unchanged during the double-step learning paradigm, the latency to the subsequent corrective saccade, and there is some variability in the location of the oculomotor vermis. The defect in adaptation of saccade amplitude in our monkey who had lesions that presumably involved the dorsal cerebellar vermis (Waespe and Müller-Meissner 1996).

VARIABILITY OF EFFECTS OF LESIONS AMONG MONKEYS. The considerable differences in the types of deficits and the patterns of recovery shown by each monkey and the dissociation in the effects of the lesions on the various parameters of saccade generation (only the effects on saccade latency and amplitude were roughly comparable and then only when the lesions were asymmetrical) must reflect, at least in part, differences in the extent and location of lesions. Portions of lobules V and VIII, were inconstantly lesioned, and there is some variability in the location of the oculomotor vermis itself (Noda and Fujikado 1987b). In monkeys 1 and 3, the vermal lesions extended far enough laterally to involve paravermal cortex, which overlap and probably projects to the posterior interpositus nucleus. Lesions in this nucleus also lead to disturbance of saccade accuracy, although vertical saccades seem to be more affected (Robinson et al. 1996). There also may be a compartmentalization within the oculomotor vermis of the various types of influences that the cerebellum can have on saccades.

Another factor contributing to the variability, however, may be related to a more fundamental role of the cerebellum in compensating for imperfections in performance that will be idiosyncratic from individual to individual depending on their inherent physical makeup and their life history of exposure to disease, trauma, and other environmental factors. The oculomotor vermis could provide the crucial information necessary for this long-term maintenance of saccade accuracy. Our results, of course, do not settle the issue as to the location of the synapses—be they neurons in the brain stem, in the fastigial nucleus, or in the Purkinje cells themselves—on which learned behaviors are stored. It may well be that there are multiple locations for saccade learning, as has been
suggested for the vestibulocular reflex (Lisberger et al. 1994; Partsalis and Hightstein 1996; Raymond et al. 1996).

ORBITAL POSITION EFFECTS AND CONTEXTUAL LEARNING. There are other ways in which a learning function of the oculomotor vermis may become apparent. By virtue of its multiplicity of sensory inputs (Yamada and Noda 1987) and its access to efferent copies of motor commands (Ohtsuka and Noda 1987b), the dorsal vermis is poised optimally to learn to recognize contexts in which a movement is to occur and then to provide any immediate adjustment in innervation needed for that particular context (Houk et al. 1996). The position of the eye in the orbit would be one such context for which the vermis could provide the correction. The discharge of some Purkinje cells in the oculomotor vermis does vary with orbital position during saccades (McElligott and Keller 1982); and the vermis also has access to proprioceptive signals that could be used in the long term for adjusting orbital-position-dependent innervation and so help assure saccade conjugacy and prevent ocular misalignment (Baker et al. 1972; Lewis et al. 1994). Other “higher-level” contexts, e.g., saccades generated to the appearance of a visual target versus saccades generated volitionally without a novel target, also may depend on the cerebellum for their proper elaboration (Houk et al. 1996; Melis and Van Gisbergen 1996; Straube et al. 1995).

In conclusion, our findings suggest an intimate relationship between the cerebellum and the saccade-generating circuits in the brain stem and superior colliculus in all aspects of saccades: initiation (latency) and termination, accuracy (including amplitude, direction and trajectory), and dynamic properties (speed and acceleration). In addition, the oculomotor vermis plays a role in oculomotor learning, assuring the accuracy of saccades to optimize visuomotor performance.

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