Fastigial Oculomotor Region and the Control of Foveation During Fixation

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Guerrasio L, Quinet J, Büttner U, Goffart L. Fastigial oculomotor region and the control of foveation during fixation. J Neurophysiol 103: 1988–2001, 2010. First published February 3, 2010; doi:10.1152/jn.00771.2009. When primates maintain their gaze directed toward a visual target (visual fixation), their eyes display a combination of miniature fast and slow movements. An involvement of the cerebellum in visual fixation is indicated by the severe gaze instabilities observed in patients suffering from cerebellar lesions. Recent studies in non-human primates have identified a cerebellar structure, the fastigial oculomotor region (FOR), as a major cerebellar output nucleus with projections toward oculomotor regions in the brain stem. Unilateral inactivation of the FOR leads to dysmetric visually guided saccades and to an offset in gaze direction when the animal fixates a visual target. However, the nature of this fixation offset is not fully understood. In the present work, we analyze the inactivation-induced effects on fixation. A novel technique is adopted to describe the generation of saccades when a target is being fixated (fixational saccades). We show that the offset is the result of a combination of impaired saccade accuracy and an altered encoding of the foveal target position. Because they are independent, we propose that these two impairments are mediated by the different projections of the FOR to the brain stem, in particular to the deep superior colliculus and the pontomedullary reticular formation. Our study demonstrates that the oculomotor cerebellum, through the activity in the FOR, regulates both the amplitude of fixational saccades and the position toward which the eyes must be directed, suggesting an involvement in the acquisition of visual information from the fovea.

INTRODUCTION

Foveate animals explore the world with saccadic eye movements (Yarbus 1967). Even when looking at small targets, periods of relative stability of the eyes are interrupted by sudden jerk-like movements, known as microsaccades (Fig. 1A) (for recent reviews, see Collewijn and Kowler 2008; Martinez-Conde et al. 2009). Several factors influence microsaccades, e.g., the visual properties of the fixated target (Martinez-Conde et al. 2004; St Cyr and Fender 1969; Steinman 1965) and the illumination conditions (Goffart et al. 2006; Snodderly 1987). Previous studies have stressed the corrective nature of these miniature saccades generated during fixation (Carpenter 1988; Cornsweet 1956; Nachmias 1959; St Cyr and Fender 1969); their probability of being triggered and directed toward the target increases with the target eccentricity (see also Young 1981). Because of these movements, the fovea scans an area that can be estimated by the spatial distribution of dwell times, i.e., the most used eye positions during the fixation time (Fig. 2A).

Microsaccades and larger visually guided saccades display a monotonic relationship between their amplitude and peak velocity (Becker 1989; Zuber and Stark 1965). Since the work of Zuber and Stark, it has been proposed that a common neuronal substrate is involved in generating saccades and microsaccades, and an increasing number of behavioral studies support this hypothesis (e.g., Otero-Millan et al. 2008; Rolfs et al. 2008). A common neuronal substrate is also supported by the observation of saccade-related neurons in the pontine reticular formation that burst for saccades whose amplitudes include those of microsaccades (van Gisbergen et al. 1981). More recently, Hafed and coworkers showed that microsaccades modulate the activity of saccade-related neurons in the rostral superior colliculus (SC) and that local injection of muscimol impairs their generation (Hafed et al. 2009).

Among the several brain regions involved in gaze orientation, the fastigial oculomotor region (FOR) has been shown to play a major role in the control of gaze accuracy (Pélisson et al. 2003; Robinson and Fuchs 2001). Located in the caudal part of the medial deep cerebellar nuclei (Yamada and Noda 1987), the two FORs house neurons that display a sustained baseline activity and bursts of spikes during every saccade (Fuchs et al. 1993; Kleine et al. 2003; Ohtsuka and Noda 1991). Clinical evidence also suggests an involvement of the cerebellum in visual fixation (Hotson 1982; Leigh and Zee 2006b; Salman et al. 2009; Selhorst et al. 1976). The FOR represents one of the main cerebellar outputs for the control of eye movements. Its participation in controlling saccade accuracy is demonstrated by the dysmetria observed after its pharmacological inactivation (Goffart et al. 2004; Robinson et al. 1993). In particular, after local and unilateral injection of muscimol, the horizontal component is hypermetric (too large) for ipsilesional saccades and hypometric (too small) for contralesional ones. In addition, vertical saccades are horizontally deviated toward the inactivated side with a magnitude that increases with saccade size. Interestingly, similar saccade disorders are observed in patients suffering from Wallenberg syndrome (Straube et al. 1994). Unilateral FOR inactivation also impairs the ability to fixate small targets: when compared with the positions taken before inactivation, the gaze is directed toward positions that are shifted by −1° toward the side of the injection, whether the head is restrained (Goffart et al. 2004; Robinson et al. 1993) or unrestrained (Quinet and Goffart 2005). The shifted spatial distribution of dwell times in Fig. 2B illustrates this change in gaze orientation, a disorder that has been called “fixation offset.”

Two impairments, non-mutually exclusive, could account for the fixation offset. On the one hand, the offset could be due
to an altered encoding of target position, i.e., the position toward which fixational saccades are directed. This possibility is supported by the observation of saccades after FOR inactivation that move gaze away from the target location rather than toward it (see blue colored movement labeled s3 in Fig. 1B). On the other hand, the offset could be the consequence of dysmetric microsaccades and, in particular, could be attributable to an asymmetrical impairment in the control of the amplitude of contra- and ipsilesional movements. According to this second possibility, the eye would approach the target with a series of hypometric contralesional microsaccades, while a single ipsilesional hypermetric microsaccade would create a larger fixation error. Due to this pattern of dysmetric microsaccades, the gaze would be more often directed ipsilaterally relative to the target, resulting in a distortion of the spatial distribution of dwell times (see Fig. 2B). To test these two possibilities, we analyzed the effects of unilateral FOR inactivation on the direction and amplitude of saccades occurring during the fixation of a central target (located straight ahead) and their relation with the spatial distribution of dwell times.

Our results show that the unbalanced activity between the two fastigial oculomotor regions affects the execution of saccades generated during fixation. They also demonstrate that the fixation offset is the result of both a shift of the goal position and of an impaired amplitude control of saccades. Part of the data reported here was published in abstract form (Guerrasio et al. 2008).

**METHODS**

**Animal preparation**

For this study, two Rhesus monkeys (B: 8.8 kg and E: 5.7 kg) were prepared for chronic head-restrained eye recording (search coil technique), single unit recording, electrical microstimulation, and muscimol injection, as described elsewhere (Quinet and Goffart 2007, 2009). All surgical procedures and experiments were performed in accordance with the guidelines from the French Ministry of Agriculture (87/848) and from the European Community (86/609/EEC).
Moreover, the first saccades frequently terminated outside the acceptance window around the central target; therefore the radius of the window was increased after the injection (radius of 10° around the central target). Before each muscimol injection session, two to five sessions were performed to gather control data.

**Localization of the FOR and muscimol injection**

Before the injection of muscimol, the location of the saccade-related region of the fastigial nucleus was identified after several experimental sessions using electrophysiological recording and electrical microstimulation in the head-restrained and unrestrained conditions as described in detail elsewhere (Quinet and Goffart 2007, 2009). For the muscimol injections, a thin cannula (230 μm OD, beveled tip) and polyethylene tubing were filled with a solution of muscimol (2 μg/μl) and connected to a Hamilton syringe. The cannula was lowered to the location previously identified as the FOR. After a delay of ~3 min, a small amount of the solution (0.5–1.1 μl) was injected by small pulses (0.1 μl every 2–3 min), until the muscimol-induced effect on saccades became clearly visible. Recordings started after the withdrawal of the cannula (2–5 min after the last pulse). The successful inactivation of the FOR was confirmed by its effects on visually guided saccades (ipsilesional hypermetria, contralesional hypometria, and ipsipulsion of vertical saccades) (Goffart et al. 2004; Robinson et al. 1993). Moreover, histological analysis performed in one animal (monkey B) confirmed that the injections were performed in the deep medial cerebellum (see Fig. 2 in Quinet and Goffart 2007).

**Eye movement analysis**

The horizontal and vertical eye position signals were sampled with the search coil technique at 500 Hz (SD of the noise = 0.004 and 0.006° on the horizontal and vertical position, respectively). For this study, we only analyzed the eye positions recorded while the monkey was fixating the central target. The eye position signals were manually calibrated at the beginning of each experimental session (6 muscimol sessions and 19 control sessions for each monkey). This calibration was further refined by a second off-line automated calibration that consisted of averaging eye positions during the last 750 ms of the fixation interval (before the fixation target was turned off). Postinjection data were calibrated from trials made before lowering the cannula for muscimol injection. Eye position signals were then filtered with a low-pass zero-phase Gaussian filter (3 dB attenuation at 30 Hz, 30/3 dB shape factor = 3.2). Eye velocity (SD of noise = 0.37°/s) and acceleration (SD of noise = 99.6°/s²) were derived from the eye position signal. Saccades were automatically detected when the velocity (horizontal or vertical) and acceleration exceeded a threshold of 5°/s and 1500°/s², respectively. To avoid labeling the small return saccade that sometimes happens immediately after a microsaccade, a second velocity threshold (3°/s) was used to define the start and end times of each detected saccade. These two consecutive movements without intersaccadic delay were considered as a single movement. Saccade onset was defined when either the horizontal or vertical velocity exceeded 3°/s. Saccade end was labeled when both horizontal and vertical velocities fell below 3°/s and did not exceed this threshold in the next two samples.

The first saccade produced after the onset of the central LED target was excluded from our analysis. Moreover, the eyes sometimes displayed rapid back and forth movements to similar positions during fixation (also called double saccadic pulses) (Abadi and Gowen 2004). We decided not to include these movements in the analysis because strongly curved and straight saccades (the large majority) may have different etiologies (Gowen et al. 2005). Saccades were considered as curved when the arc length exceeded the cord length by 0.5°. The 0.5° threshold was chosen after examination of the relation between saccade length and amplitude. It was restrictive enough to remove all
saccades lying out of the main sequence (Fig. 3). Note that these curved saccades occurred in similar proportion before and after the muscimol injection.

Because we were interested in the effect of FOR inactivation on fixation, we avoided using a restrictive amplitude criterion to analyze the monkeys' behavior. Our study is not restricted to microsaccades with amplitude <20 minarc (Collewijn and Kowler 2008) but also includes fixational saccades, defined as saccades occurring during fixation (Engbert and Kliegl 2003; Hafed and Clark 2002). It is noteworthy that the median amplitude of fixational saccades described in our study (~0.9 and 0.6°, see Table 1) is comparable with those described in other studies using non-human primates (Bair and O'Keefe 1998; Skavenski et al. 1975; Snodderly et al. 2001).

Measures of fixation

Fixation has been traditionally described by the scatter of eye positions or the spatial distribution of dwell times. In the former case, the eye position is sampled at regular time intervals or relative to particular events (i.e., saccades), and the distribution of the horizontal and vertical positions is statistically analyzed to infer the stability of the eyes during fixation (see for instance Steinman 1965). The second measure, also defined as the density of fixation (Bennet-Clark 1964; Møller et al. 2006a), computes the most used eye positions, in a temporal sense. Both measures, when obtained from repeated trials, can provide an estimate of the portion of the retina that is preferred (i.e., used more often and for longer times) when foveating a target, the latter adding the information that the triggering of saccades depends on eye position (Boyce 1967; Kalesnykas 1994; Young 1981). However, these two estimates have the disadvantage of assuming a spatial matching between the preferred retinal locus for fixation and the position of the goal. Thus any anisotropy (pathological or not) in the mechanisms triggering and executing fixational saccades renders both the scatter of eye positions and the spatial distribution of dwell times inaccurate to define the goal of fixation. While we use the spatial distribution of dwell times to describe the visual area explored during fixation (see Calculation of the spatial distribution of dwell times), we propose a novel method to describe fixation (see Analysis of the direction of fixational saccades), which is not affected by the subject’s oculomotor performance. This alternative method is inspired by the work of Cornsweet (1956), who defined as “on-target,” the

![Fig. 3. Main sequence of fixational saccades in monkeys B (A) and E (B). Relation between amplitude and peak velocity of fixational saccades before (top) and after (bottom) unilateral inactivation of the FOR. O, each corresponds to a detected saccade; saccades excluded from the analysis are superimposed. They were excluded because of their curved trajectory (×) or because they were considered as 1st (primary) saccades toward the central target (∗).](http://jn.physiology.org/)

**TABLE 1.** Number and amplitude of fixational saccades used in this study

<table>
<thead>
<tr>
<th>Monkey B</th>
<th>Monkey E</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control (n = 6)</strong></td>
<td><strong>Muscimol (n = 6)</strong></td>
</tr>
<tr>
<td>Detected saccades</td>
<td>4110 ± 2010</td>
</tr>
<tr>
<td>Used saccades</td>
<td>3450 ± 1700</td>
</tr>
<tr>
<td>Median amplitude of used saccades</td>
<td>0.88 ± 0.15°</td>
</tr>
</tbody>
</table>

After detecting saccades, some of them were excluded from the study because they were curved or executed too shortly after the onset of the fixation target (see METHODS for details). The average median amplitude of saccades recorded during the control experiments is in agreement with previously reported data. Note the larger size of saccades after fastigial oculomotor region (FOR) inactivation, reflecting the impaired fixation. The average median values remain well below the size of the area of acceptance (radius = 10°), indicating that the monkeys were actually attempting fixation.
position from where the eyes are “equally likely to move to the left or to the right.” This way of describing fixation only relies on the corrective nature of fixational eye movements and does not depend on mechanisms regulating their timing or amplitude. However, Cornsweet’s definition has the limitation of only considering one-dimensional eye movements. Because of that, it cannot differentiate between two horizontal opposite movements and two vertical movements with small opposite horizontal components (but identical, larger vertical components). To overcome this limitation, we used directional statistics to identify those eye positions characterized by a high directional variability. Finally, our analysis provides a motor description of the foveating behavior that takes into account its dynamic nature.

Calculation of the spatial distribution of dwell times

To compute the positions where the eyes dwell longer, we used a modified version of the method described by Cummings et al. (1985). We summed the durations of the intersaccadic intervals (from the end of a saccade to the onset of the next one) for all saccades landing in a square window of eye position (0.2° horizontally and vertically). This summation was made in an iterative manner by moving the window in steps of 0.05°. After scanning the complete range of eye positions, the values of the resulting matrix were normalized, and a Gaussian two-dimensional (2D) filter (sigma = 1) was applied. Then, a second iterative process calculated the isoline levels of the spatial distribution that contained 95, 68, and 5% of the total dwell time (maximum error 0.5%).

Analysis of the direction of fixational saccades

In the following sections, we describe the computation of the variability of saccade directions as a function of eye position and the area of highest directional variability (A). Then, we illustrate the algorithm used to classify a population of directions according to its number of modes to graphically visualize it (B).

A: ALGORITHM FOR COMPUTING THE DIRECTIONAL VARIABILITY. Directions are described by the angle between a reference and the measured direction. The reference direction (0 rad) is defined here as the horizontal movement to the right. The algebra that applies to linear measures is not suitable for angles (for example, the difference done by multiplying every direction by different values of \( \alpha \) from uniformity \( [\text{critical values for rejecting the null hypothesis of unimodal variability area.}] \) can also be used to test the deviation from uniformity \([\text{critical values for rejecting the null hypothesis of uniform distribution can be found in Holmqquist and Sandberg (1991)]\).
The procedure for classifying the distribution can be summarized as follows. The Rayleigh test was first used to test uniformity. When it rejected the null hypothesis of uniform distribution ($P < 0.05$), the BAT (with $\text{ANG}_{\text{mean}}$ and $\text{ANG}_{\text{max}} + \pi$ rad as reference angles) was used to classify the underlying distribution as unimodal ($\alpha_{\text{max}} = 1$ with $\text{ANG}_{\text{mean}} + \pi$ rad as reference angle) or bimodal. When it failed to reject the null hypothesis, then uniformity was further tested with the broken axis technique: if the BAT found a significant deviation from uniformity, $\alpha_{\text{max}}$ was used to classify the distribution as bimodal or multimodal ($\alpha_{\text{max}} > 2.5$).

The power of our algorithm was tested with directions randomly selected from uniform and uni-, bi-, and multimodal distributions (Table 2).

After the classification, the sampled directions were graphically represented. When the distribution was classified as unimodal or bimodal, the visual representation was informative of the statistical properties of the distribution. In the unimodal case, the distribution was represented by a vector having the median direction and a pair of vectors including 68% of direction values. To compute the pair of vectors representing the 16th and 84th percentiles ($P_{16}$ and $P_{84}$), successive values of $P_i$ were assigned to directions adjacent to the median

$$P_i = d_{\text{median}} \pm 100 \cdot i/N$$

where $i$ is the number of directions below or above the median direction $d_{\text{median}}$, and $N$ is the total number of angles in the sample. Intermediate percentiles were obtained by linear interpolation between each successive $P_i$.

When the distributions were classified as coming from a bimodal distribution, the visualization was different depending on the number of samples inside each mode. When the mode contained <30 samples, no graphical representation of the directional dispersion was given; the population was represented with only two vectors, each directed according to the modes identified with the BAT. The length of each vector was proportional to the proportion of directions neighboring the represented modes. When the mode contained >30 directions, the algorithm computed the angles of the two modes and the dispersion around them by fitting data with a bimodal von Mises distribution, defined by

$$P(d|p, d_1, d_2, k_1, k_2) = p \cdot \left[ e^{i (d_1 - d)} / \{2 \pi I_0(k_1) \} \right] + (1 - p) \cdot \left[ e^{i (d_2 - d)} / \{2 \pi I_0(k_2) \} \right]$$

where $p$ is the mixture probability of two von Mises distributions having $k_1$ and $k_2$ as concentration parameters, $d_1$ and $d_2$ as location parameters (mean directions), and $I_0(k)$ is the modified Bessel function of order zero at point $k$. The concentration parameter describes how the distribution is packed around each mean direction. The five parameters were estimated with the maximum likelihood methods. The starting values of the location parameters were the directions of the two principal modes computed with the BAT. To avoid local minimum, several combinations of starting values of $k_1$, $k_2$, and $p$ were tested while the values of the location parameters were kept fixed. More specifically, four values for each concentration parameter (linearly spaced between 1 and 50) and two values of $p$ (0.5 and 0.75), making a total of $4 \times 4 \times 2 = 32$ combinations, were used to compute the logarithmic likelihood. The best combination of parameters was then used as starting value set for the estimation of the five parameters. At the end of the estimation, the circular variance (CV) of each of the two distributions was calculated from the concentration parameters by

$$\text{CV} = \left[ I_1(k) \right]^2 / \left[ I_0(k) \right]^2$$

From the circular variance, a circular SD (CSD) can be computed (Fisher 1993)

$$\text{CSD} = \sqrt{2 \cdot \ln (1 - \text{CV})}$$

Thus when >30 directions were considered to be sampled from a bimodal distribution, the directional properties of saccades were summarized and graphically visualized with two vectors directed as $d_1$ and $d_2$, and two couples of vectors representing the dispersion of each mode, each including one CSD.

### Table 2. Efficiency of the method for classifying the number of modes of a distribution

<table>
<thead>
<tr>
<th>$P(\alpha)$</th>
<th>$n$</th>
<th>Uni-modal</th>
<th>Bi-modal</th>
<th>Multi-modal</th>
<th>Uniform</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unimodal $[1 + \cos(\alpha)]/2\pi$</td>
<td>12</td>
<td>57.00</td>
<td>9.30</td>
<td>0.84</td>
<td>32.86</td>
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<tr>
<td></td>
<td>24</td>
<td>82.74</td>
<td>11.79</td>
<td>0.04</td>
<td>5.43</td>
</tr>
<tr>
<td></td>
<td>36</td>
<td>87.40</td>
<td>12.04</td>
<td>0.01</td>
<td>0.56</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>88.75</td>
<td>11.24</td>
<td>0</td>
<td>0.01</td>
</tr>
<tr>
<td>Bimodal $[1 + \cos(2\alpha)]/2\pi$</td>
<td>12</td>
<td>1.28</td>
<td>24.60</td>
<td>2.16</td>
<td>71.96</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>0.25</td>
<td>54.42</td>
<td>0.97</td>
<td>44.36</td>
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<tr>
<td></td>
<td>36</td>
<td>0</td>
<td>81.49</td>
<td>0.44</td>
<td>18.07</td>
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<tr>
<td></td>
<td>48</td>
<td>0.01</td>
<td>96.18</td>
<td>0.25</td>
<td>3.56</td>
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<tr>
<td>Trinodal $[1 + \cos(3\alpha)]/2\pi$</td>
<td>12</td>
<td>0.07</td>
<td>4.60</td>
<td>21.16</td>
<td>74.17</td>
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<tr>
<td></td>
<td>24</td>
<td>0.07</td>
<td>3.37</td>
<td>43.92</td>
<td>52.64</td>
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<tr>
<td></td>
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<td>3.34</td>
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<td>0.11</td>
<td>3.58</td>
<td>90.33</td>
<td>5.98</td>
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<td>Quadrinodal $[1 + \cos(4\alpha)]/2\pi$</td>
<td>12</td>
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<td>0.35</td>
<td>22.64</td>
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<td></td>
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<td>0.06</td>
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<td>78.10</td>
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<td>0.02</td>
<td>93.73</td>
<td>6.25</td>
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<tr>
<td>Uniform 1/2π</td>
<td>12</td>
<td>4.45</td>
<td>2.86</td>
<td>2.01</td>
<td>90.68</td>
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<tr>
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<td>2.34</td>
<td>0.68</td>
<td>93.23</td>
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<td>0.77</td>
<td>92.21</td>
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<tr>
<td></td>
<td>48</td>
<td>3.46</td>
<td>4.54</td>
<td>1.03</td>
<td>90.97</td>
</tr>
</tbody>
</table>

A number $n$ of directions was randomly extracted from known distributions with probability density function $P(\alpha)$, and the described method for classification was applied. Results indicate both the percentage of correct classification (bold characters) and, in case of error, how the distribution was labeled. Note that the result “uniform” indicates that the null hypothesis of uniform distribution could not be rejected. Not shown, results with $n = 96$, where all distribution where correctly detected with an error rate <5%. Number of simulations for each distribution and for each $n$: 10000.
Analysis of the amplitude of fixational saccades

To describe how inactivation of the FOR affects the amplitude of saccades, the change in the saccade amplitude/target eccentricity ratio (also called gain) has been used in past studies (Robinson et al. 1993). This calculation raises problems when analyzing fixational saccades for the two following reasons. The first problem is logical: this ratio uses as a denominator a value (target eccentricity) that is not directly measured but inferred from recordings of eye positions; in turn, eye positions are influenced by the very same impairment (fixation offset and dysmetria) that our analysis wants to describe. The second problem is numerical: fixational saccades can be produced even when the monkey “fixates” the target. In that case, target eccentricity is “zero” and gain values tend toward “infinite” values. To avoid these problems, we used a technique that exploits the corrective nature of fixational saccades (as confirmed in our analyses by the high correlation between eye positions and saccade amplitudes; see RESULTS, Fig. 5). Because unilateral inactivation of the FOR differently alters ipsilesional and contralesional saccades (Goffart et al. 2004; Robinson et al. 1993), we hypothesized different constants of proportionality (gains) for the relations between initial eye position and the amplitude of leftward and rightward saccades. The gains were computed by fitting a broken line with distinct slopes for contra- and ipsilesional saccades ($G_l$ and $G_r$, respectively), and passing through the intersection point ($x_0,0$). The intersection point was the third free parameter of the fit because the null position of the oculomotor system is unknown. The three parameters of the fitting were calculated by minimizing the square error in the orthogonal sense. The orthogonal distance regression (also known as model-2 regression) was chosen instead of the ordinary least square regression (or model-1 regression) because of the uncertainty existing on the nature of the input driving fixational saccades. Moreover, this model-2 regression is less affected by data non-normalities, and our data showed that both positions and amplitudes were strongly leptokurtic. The fitting was also constrained to continuity, i.e., for any small increase in eye position, there was a small change in eye amplitude. This solution was preferred instead of fitting two different lines (with 2 different intercepts) for leftward and rightward movements (Goffart and Pélinson 1998). A negative gain means that when the eye was to the left of the intersection point, it moved to the right and vice versa. Robust fitting was used to reduce the influence of outliers on the calculated regression. The algorithm iteratively reweighted residuals with a sigmoid function. More specifically, the normal cumulative function with $\mu = 0.5$ and $\sigma = 0.15$ was used on the normalized residuals to generate weights after removing the 5% largest residuals. A small (10%) contribution to the weighting function was attributed to the relative sample size (percentage of saccades in one direction) as well as to the correlation values between eye positions and saccade amplitudes for each direction. These two additional weights attenuated the biases that the largest dataset or poorly correlated data could exert on the fitting.

RESULTS

Using the novel technique detailed in METHODS, we were able to visualize the relationship between the position of the eye and the direction of fixational saccades starting from neighboring positions. Figure 4 shows this relationship for saccades recorded before and after one injection of muscimol in the right FOR of monkey E. During the control (preinjection) conditions, two primary observations were made. First the direction of fixational saccades depended on the initial position of the eye, and it was generally oriented toward the area from where saccades did not display any preferential direction (Fig. 4A). Second, the center of this zone of high directional variability (red-colored area) acted like a “watershed” (or “divide”) for saccade directions: when the eye was, e.g., below and right with respect to it, fixational saccades had a higher probability to be directed up and leftward. After FOR inactivation, the high directional variability area was shifted toward the injected side (Fig. 4B). This ipsilesional shift was statistically significant in both monkeys (Wilcoxon 1-tailed signed rank tests, $P < 0.05$, mean ± SD magnitude $= 0.87 ± 0.44$ and $0.25 ± 0.27°$ for monkeys B and E, respectively). The shift was also characterized by a vertical component (see Fig. 4B): in monkey E, the vertical shift was always downward ($−0.46 ± 0.39°$, $n = 5$), whereas it was upward in all experiments but one in the other monkey ($0.55 ± 0.65°$, $n = 6$). Interestingly, also after FOR

![Figure 4](http://jn.physiology.org/)

FIG. 4. Effect of inactivating the right FOR on the relationship between the eye position and the direction of fixational saccades. The black arrows represent the median direction of saccades starting from eye positions within a squared 0.25° area around the tail of the vectors. Pairs of white vectors enclose the 68% directional variability area. In both monkeys (Wilcoxon 1-tailed signed rank tests, $P < 0.05$), the center of this zone of high directional variability (red-colored area) acted like a “watershed” (or “divide”) for saccade directions: when the eye was, e.g., below and right with respect to it, fixational saccades had a higher probability to be directed up and leftward. After FOR inactivation, the high directional variability area was shifted toward the injected side (Fig. 4B). This ipsilesional shift was statistically significant in both monkeys (Wilcoxon 1-tailed signed rank tests, $P < 0.05$, mean ± SD magnitude $= 0.87 ± 0.44$ and $0.25 ± 0.27°$ for monkeys B and E, respectively). The shift was also characterized by a vertical component (see Fig. 4B): in monkey E, the vertical shift was always downward ($−0.46 ± 0.39°$, $n = 5$), whereas it was upward in all experiments but one in the other monkey ($0.55 ± 0.65°$, $n = 6$). Interestingly, also after FOR inactivation, the location of this area is shifted, and fixational saccades starting from similar positions display different directions.
unilateral inactivation, saccades generated from eye positions characterized by a low directional variability, aimed coherently at the (new) area of higher variability. In other terms, when compared with control saccades, saccades initiated from similar positions were directed differently after muscimol injection.

Muscimol injection in the FOR also changed the relationship between the horizontal initial eye position and the horizontal amplitude of fixational saccades. During the control sessions (Fig. 5, A and C), a significant negative correlation was found between eye position and saccade amplitude (mean of Spearman correlation coefficients for the horizontal component: $R = -0.75 \pm 0.02$ and $-0.74 \pm 0.07$, vertical component: $R = -0.80 \pm 0.03$ and $-0.81 \pm 0.04$ for monkeys $B$ and $E$, respectively, $n = 6$, all 1-tailed $P < 0.001$). Such a correlation illustrates the corrective nature of fixational saccades: leftward saccades were generated from rightmost positions and vice versa. From central positions, both right- and leftward saccades were produced. After muscimol injection (Fig. 5 B and D), the horizontal component of fixational saccades was altered: it was hypermetric for ipsilesional saccades and hypometric for contralesional ones. Because of these asymmetrical changes, the hypermetric for ipsilesional saccades and hypometric for contralesional ones. In most cases, the correlation between eye positions and saccade amplitude was highly significant ($P < 0.001$), both for the horizontal and vertical components. In some cases (3 of 12), however, it did not reach statistical significance, and the gain could not be calculated for ipsilesional saccades (see for instance the dashed line in Fig. 5D; leftward saccades do not display any relation between their horizontal amplitude and the starting eye position). Interestingly, while ipsilesional saccades were characterized by smaller and sometimes non-significant correlation between amplitude and eye position ($R = -0.17 \pm 0.26$ and $-0.59 \pm 0.04$ in monkeys $B$ and $E$, respectively), Spearman correlation coefficients were always very high for contralesional saccades ($monkey B$, $-0.82 \pm 0.06$, monkey $E$, $-0.87 \pm 0.04$, all $n = 6$). In other words, unilateral FOR inactivation did not affect the corrective nature of contralesional movements (in spite of their reduced gain) while it weakened the relationship between the eye position and the eye amplitude of ipsilesional saccades. This weakening ($\chi$, estimated with the percent decrease in the correlation coefficients) was found to be correlated with the shift of the high directional variability area ($y$), even after removing the influence ($z$) related to changes in the number of ipsilateral saccades [$R(x, y/z) = 0.79, P < 0.01$; see also Fig. 7B]. No correlation was found between the shift and the increased correlation between eye position and amplitude for contralesional saccades ($R = 0.03, P > 0.9$). Finally, it is noteworthy that muscimol injection in the FOR did not significantly affect the vertical component of fixational saccades (Fig. 6B). Indeed the correlation between the vertical amplitude of saccades and the vertical initial eye position was not affected in a consistent manner.

**FIG. 5.** Relation between the eye position and the amplitude of fixational saccades before (A and C) and after (B and D) unilateral inactivation of the FOR. B: injection in the right FOR of monkey $E$ (same experiment as in Fig. 1). D: injection in the left FOR of monkey $B$. The values of the fitting (see METHODS) are displayed in the top–right corner of each plot. The injection of muscimol causes a shift of the relation toward the injected site and an asymmetrical change in slope; the amplitude is increased for ipsilesional saccades and decreased for contralesional ones. • • •, amplitude and initial eye position were not significantly correlated.
in the aiming position should affect the offset of the relationship. Interestingly, the magnitude of the shift in the intersection point was strongly correlated with the displacement of the zone of high directional variability (Fig. 7A, Spearman $R = 0.95$ and $R = 0.99$ for the horizontal and vertical component, respectively, $P < 0.001, n = 11$). The shift in the aiming zone was neither correlated with the gain decrease of contralesional movements ($R = 0.17, n = 11$) nor with the increased gain of ipsilesional ones ($R = -0.24, n = 8$, Fig. 7B). Moreover, the partial correlations between the ipsilesional gain increase $(\chi)$, contralesional gain decrease $(\gamma)$, and the shift of the area of maximal variability $(\zeta)$ were not statistically significant [$R(x, yz) = -0.27$ with $P > 0.55$; $R(x, zy) = 0.15$ with $P > 0.75$ and $R(y, z/x) = 0.46$ with $P > 0.29$].

Another interesting observation is that the eyes spent more time in positions that were more deviated than the aiming zone or “on target” position (i.e., the position where saccades are likely to move in opposite directions) (Cornsweet 1956). The spatial distribution of dwell times was stretched toward the injected side in such a manner that its center was shifted further than the center of higher variability (difference $= 0.20 \pm 0.19^\circ$, $n = 11$; compare Fig. 2B with 4B). One possible explanation for the observation that the eye dwells longer toward the ipsilesional side is that a hypermetric ipsilesional saccade moved the eyes further away from the position of high directional variability while more hypometric contralesional saccades were required to bring the eyes back to the on-target position. To test this hypothesis, we calculated the correlation between the gain changes and the stretching effect (measured by the horizontal distance between the center of the dwell time distribution and the center of the higher variability area). A significant correlation ($R = 0.71, P < 0.05, n = 11$) was found between the contralesional gains and the shifts of the dwell time distributions (Fig. 8). The lack of significant correlation between the ipsilesional gain changes and the stretching effect ($R = 0.25$) suggests that the hypermetria of ipsilesional saccades barely contributed to the stretching effect.

Inactivation of the FOR did not change the frequency of fixational saccades. After muscimol injection, the frequency (median: 2.51 and 1.31 saccades/s in monkeys B and E, respectively) was not significantly different from the frequency observed during the control sessions (monkeys B and E: 2.45 and 1 saccades/s, Wilcoxon rank-sum test, 2-tailed $P > 0.05, n = 6$). Because of the postlesional changes in amplitude, saccades occurring in the early part of the fixation period were usually corrective, i.e., their horizontal component was significantly different from zero (Wilcoxon sign rank test $P < 0.05$ for saccades occurring in the 1st 250 ms). However, after a time that was relatively variable [$1.18 \pm 0.29$ and $1.3 \pm 0.48$ s, monkeys B and E], the average horizontal component of saccades occurring became indistinguishable from 0 (Wilcoxon sign rank test $P > 0.95$, Fig. 9).

To test whether monkeys’ fixational saccades have a preferred direction as proposed in humans (see Fig. 5a in Engbert 2006), we analyzed the 48 directions of saccades initiated around the point of maximal variability (Fig. 10). If saccades have a preferred direction, they should be distributed around two opposite modes. In such a case, $\alpha_{\text{max}}$ should have a value near 2; the tighter the directions are clustered around the two modes, the larger the value of AMP$^{\text{mean}}$($\alpha_{\text{max}}$) is. In general, both before and after muscimol injection in the FOR, fixational

**FIG. 6.** Effect of the unilateral FOR inactivation on the accuracy of the horizontal (A) and vertical (B) component of fixational saccades. Each square represents the value of the fitting for all saccades in 1 direction before (black squares) and after (gray squares) the inactivation. For each data set, the gain values were computed only if eye positions and saccade amplitudes were significantly correlated. Dotted lines link the mean value for different directions before and after (black and gray line, respectively) the inactivation. Bars indicate confidence intervals. Before the injection, fixational saccades displayed a slight hypermetria (mean $\pm$ SD of gains: $-1.15 \pm 0.17$ and $-1.6 \pm 0.4$ for horizontal and vertical component). The unilateral inactivation caused a reduction ($27 \pm 15\%$ decrease) in the gain of contralesional saccades (mean $\pm$ SD of gains: $-0.81 \pm 0.12$) and an increase ($90 \pm 60\%$ increase) in the gain of ipsilesional saccades (mean $\pm$ SD $-2.7 \pm 0.40$). The overall effect is an asymmetrical change in the horizontal control of fixational saccades (A). The lesion does not have any effect on the accuracy of vertical saccades but increasing the variability of the gain (B).

In addition to the slope changes in the relationship between the horizontal starting eye position and the horizontal amplitude of fixational saccades, muscimol injection in the FOR also shifted the intersection point ($X_{\text{int}}$) toward the injected side, an observation that is consistent with the change in the zone toward which saccades were directed (Fig. 4). Note that the data were fitted without constraining the relation to a certain null position; the point of intersection was one of the free parameters of the fit (see Methods). If fixational saccades still display a corrective nature after muscimol injection, a change...
saccades displayed a symmetrical bimodal distribution (mean \( \alpha_{\text{max}} \pm \text{SD} = 1.79 \pm 0.13 \) and 2.0 \( \pm 0.19 \) before and after the injection, respectively, \( n = 11 \)). The preferred directions were upright (73.5 \( \pm 12.6^\circ \) in control condition; 72.9 \( \pm 27.8^\circ \) after FOR inactivation) and down-left (before and after the inactivation: 262.5 \( \pm 9.2^\circ \) and 242.4 \( \pm 30.1^\circ \)). The statistic \( \text{AMP}_{\text{mean}}(\alpha_{\text{max}}) \) was significantly decreased after muscimol injection (mean \( \pm \text{SD} = 0.69 \pm 0.05 \) before and 0.56 \( \pm 0.11 \) after injection). This decrease was not due to the fact that the area required for gathering the same number of directions (48) was larger for the postinjection data (radius = 0.22 \( \pm 0.06^\circ \)) than for the control data (radius = 0.07 \( \pm 0.02^\circ \)). Indeed, the decrease was still significant when the same area were used for control and postinjection data [\( \text{AMP}_{\text{mean}}(\alpha_{\text{max}}) = 0.67 \pm 0.05 \)]. The reduction of \( \text{AMP}_{\text{mean}}(\alpha_{\text{max}}) \) indicates that the variability of saccade directions was higher after unilateral FOR inactivation. However, it is important to stress that both before and after FOR inactivation, the statistic \( \text{AMP}_{\text{mean}}(\alpha_{\text{max}}) \) was always larger than the 95% of the simulations made with the same amount of directions randomly selected from a uniform distribution.

**DISCUSSION**

In this study, we analyzed the effects of unbalanced activity in the fastigial oculomotor regions on the fixation of a small field of view.
visual target. A novel technique was adopted to describe the direction of fixational saccades generated from various eye positions while attempting to look at a visual target located straight ahead. Our results show that the fixational saccades move the eye toward a zone that acts like an “attractor.” However, their size is variable and often too large to place the eye in the center of this zone. When the eye falls within it, the direction of saccades becomes unpredictable and equally distributed between two opposite directions (Fig. 10). They also show that this aiming zone is changed after muscimol injection in one FOR: it is shifted toward the inactivated side (Fig. 4). Such an ipsilesional shift does not result from different numbers of leftward and rightward saccades because our analytical technique is not influenced by this factor. The shift is due to the fact that saccades starting from similar positions are generated with different directions after FOR unilateral inactivation. In addition to this effect on the direction of saccades, unilateral FOR inactivation also causes asymmetrical changes in the amplitude of fixational saccades: the horizontal amplitude is hypermetric for ipsilesional saccades and hypometric for contralesional ones (Figs. 5 and 6A). These asymmetrical changes can be described by two gain values whose computation is also independent of the relationship between eye position and saccade direction.

Our study clarifies how the change in horizontal amplitude and the shift of location of the aiming zone contribute to the fixation offset observed after FOR inactivation. On the one hand, if FOR inactivation only shifted the aiming zone, the gains of ipsilesional and contralesional saccades should not change, and a spatial translation of the distribution of dwell times would be the only observed effect. On the other hand, if the impairment only concerned the amplitude of fixational saccades, the location of the aiming zone should not change after unilateral FOR inactivation: being pushed toward ipsilesional positions by hypermetric fixational saccades, more contralesional saccades would be required to fixate back the target because of their hypometria. Thus the hypometria of contralesional fixational saccades would cause the gaze to spend more time in ipsilateral positions, resulting in a stretching of the distribution of dwell times toward the injected side. Our results show that a change in horizontal amplitude and a shift of the aiming zone both affect fixational saccades after FOR inactivation. Two observations suggest that these two impairments are independent. First the two effects are not correlated (Fig. 7B). Second, the shift of the area of maximal directional variability affects both the horizontal and the vertical component, while the changes in amplitude only concern the horizontal component (Fig. 6B).

**Hypothetical roles for the fastigio-tectal and -reticular pathways**

Independent impairments, as a result of FOR inactivation, could be explained by the different targets of FOR projections to the brainstem. The shift of the aiming zone could result from a functional perturbation of the fastigio-tectal pathway, whereas the change in horizontal amplitude could result from a dysfunction of the fastigio-recticular pathway. Indeed in the rhesus monkey, the fastigial projections terminate bilaterally in the rostral end of the intermediate gray layer of the deep superior colliculus (May et al. 1990). Their perturbation could change the topography of active neurons in the rostral superior colliculi. Thus an unbalanced FOR activity would change the activity of neurons in the rostral superior colliculi, which in turn, would affect the encoding of the position of foveal targets and lead to a shift of the aiming zone of fixational saccades. The fixation offsets recently described after local injection of muscimol in the rostral superior colliculus (Hafed et al. 2008) are compatible with this scenario. The vertical component of
the fixation offsets described in our study is compatible with a change in collicular activity during fixation, rather than with a direct effect on the vertical burst generator, because the fastigial projections to the mesodiencephalic reticular formation are quite modest (Sato and Noda 1991). Moreover the similar magnitude of the ipsilesional offsets observed in the head restrained [range = 0.7°–1.6° in Robinson et al. (1993); mean = 1.1° in Goffart et al. (2004)] and head unrestrained monkey [mean = 1.1° in Quinet and Goffart (2005)] suggests a disorder that is related to the orientation of gaze (i.e., a behavioral parameter that takes eye and head orientations into account) during fixation. Given the well-established gaze-related function of the deep superior colliculus (Sparks 2004), this similarity is also compatible with the hypothesis of a fastigio-tectal perturbation. The larger offsets (~5°), observed in head unrestrained cats after muscimol injection in the caudal fastigial nucleus (Goffart and Pélisson 1998), could result from the larger extent of fastigial projections to the deep superior colliculus in the feline species (Hirai et al. 1982; Sugimoto et al. 1982). Thus the sustained firing rate displayed bilaterally by FOR neurons during intersaccadic intervals (Fuchs et al. 1993; Kleine et al. 2003; Ohtsuka and Noda 1991) would participate in the control of fixation by balancing the activity between the left and right rostral SC. With respect to the dysmetria that affects the horizontal component of fixational saccades, a perturbation of the fastigial influence on saccade-related neurons in the contralateral pontomedullary reticular formation (Noda et al. 1990; Scudder et al. 2000) would be the most parsimonious explanation. This reticular region is indeed known to control the horizontal component of saccades (Barton et al. 2003; Cohen et al. 1968), and the perturbation of the fastigio-recticular pathway has already been proposed to account for the horizontal dysmetria of saccades observed in the head restrained and head unrestrained monkey after FOR inactivation (Goffart et al. 2004; Quinet and Goffart 2007; Robinson and Fuchs 2001; see also Kojima et al. 2008). Finally, the oculomotor function of the fastigio-recticular pathway, which is suggested by microstimulation studies in the head unrestrained monkey (Quinet and Goffart 2009), is also consistent with the independency observed in the present study, between the asymmetrical gain changes that affect ipsilesional and contralesional fixational saccades and the change in their aiming zone.

**Foveation and the generation of fixational saccades**

More generally, our study provides additional evidence that looking at a target does not consist of bringing the eyes to a particular position and leaving them there. Indeed there was no specific eye position where saccadic eye movements were prevented. Our results show that fixational saccades help the fovea to scan an area that is centered on the zone of higher directional variability. We call this scanning process *foveation*, the oculomotor behavior that consists in orienting the fovea toward a target. In spite of the large size of acceptance windows used in our study (see Methods) and a relatively extended fovea (Perry and Cowey 1985; Wickler et al. 1990), the area of “foveal exploration” is rather limited (Table 1 and Fig. 2). Our study shows that this area is influenced by corrective mechanisms under the control of the oculomotor cerebellum. These cerebellar-dependent mechanisms would center the area of foveal exploration on the aiming zone (via the fastigio-tectal connections) and regulate the amplitude of saccades (via the fastigio-reticular connections). Other mechanisms are also suggested by previous studies. Indeed, both humans (Steinman et al. 1973) and monkeys (Skavenski et al. 1975) can suppress the generation of fixational saccades. In the monkey, this result can be obtained after a special training, while a verbal instruction to humans suffices to prevent the generation of microsaccades (Kowler and Steinman 1980). Moreover, the eyes can also efficiently maintain fixation without saccades by means of slow eye movements (Steinman et al. 1973). Our study shows that inactivation of the FOR did not impair this slow eye movement control, which presumably is influenced by other cerebellar structures like the flocculus ( Büttner and Büttner-Ennever 2006).

Cornsweet (1956) have hypothesized three functional systems for the control of saccades. These systems would control the direction, the magnitude of fixational saccades, and their triggering. Our study shows that the fastigial oculomotor region participates in the first two systems but not in the third. Indeed the saccade rate was unaffected by FOR inactivation. The absence of changes in the saccade triggering mechanisms is consistent with the lack of significant changes in latencies after FOR inactivation in the monkey [Quinet and Goffart 2007; note that changes in latencies happen after muscimol injection in the feline caudal fastigial nucleus (Goffart and Pélisson 1997)]. Previous studies reported a dependency of triggering mechanisms on attentional factors (Barlow 1952; Engbert and Kliegl 2003; Hafed and Clark 2002). Our results are also consistent with the observation that while several cortical areas (corresponding in the monkey to the FEF, SEF, and LIP-7a) are active during both oculomotor and attentional tasks, the medial cerebellum is activated only during oculomotor tasks (Corbetta et al. 1998). Accordingly, the observed shifts of fixation after unilateral FOR inactivation have a magnitude that is not comparable with the gaze deviations observed in patients suffering from neglect after right hemispheric lesions (Leigh and Zee 2006a).

It has been shown that the subcortical network for the generation of fixational saccades involves burst neurons in the paramedian pontine reticular formation (van Gisbergen et al. 1981) and in the rostral SC (Hafed et al. 2009). When the target image falls on the fovea, the activity in both rostral SC would be roughly at equilibrium, and fixational saccades would result from fluctuations in the equilibrium of this bilateral activity (Hafed et al. 2008, 2009). The variability in the direction of fixational saccades, outlined by our analytical technique, would result from these fluctuations when a visual target is being foveated. When the activity in the SC is unbalanced because of a distance between the actual position of the eye and the optimal position for foveation, saccades are generated to reduce this distance and to restore the balance in bilateral collicular activity. Accordingly, when the gaze is in the direction that permits the optimal retinal stimulation, the activity in the rostral SC would be roughly at equilibrium, and saccades are generated with an unpredictable direction. After FOR inactivation, saccades move the eyes toward an aiming zone that has shifted. This shift does not reflect an inability to correct for a small residual retinal error but presumably a new encoding of the foveal target position. Indeed we showed that after a certain time, the average effect of fixational saccades on eye position cancel each other out (Fig. 9) after the gaze has reached the target position.
Finally, we showed that in monkeys fixational saccades initiated from the aiming zone have a preferred direction (Fig. 10). When looking at the target, our monkeys preferentially move their eyes along a vertical axis, whereas humans seem to prefer a rather horizontal direction (Engbert 2006). Although balanced fastigial activity is important for modulating the horizontal component of saccades (Goffart et al. 2004), its pharmacological perturbation does not affect the bimodality of fixational saccades. Further studies are required to clarify the origin of preferred directions.

Conclusion

Our work provides experimental results supporting the suggestion that fixation impairments in cerebellar patients can be due to a defective control of fixational saccades (Helmchen et al. 2003; Ramat et al. 2007; Selhorst et al. 1976; Tsutsumi et al. 2009). It would be very interesting to test whether patients suffering from Wallenberg syndrome show deviations of gaze during fixation similar to those described here because these patients have saccade disorders similar to those observed in the monkey after FOR inactivation (Straube et al. 1994). In any case, by taking a novel quantitative approach, we showed that the oculomotor cerebellum regulates the amplitude of fixational saccades and adjusts the position toward which gaze is directed. The presence of a control system for fixational saccades would support the hypothesis, arising from neuronal recordings in the visual cortex (Leopold and Logothetis 1998; Martinez-Conde et al. 2002; Snodderly et al. 2001), of a physiological role of fixational saccades in vision (Martinez-Conde et al. 2004). Moreover, it has been shown that the retinal portion used for foveating a target is not hardwired with the photoreceptor distribution (Putnam et al. 2005). Given the general role of the cerebellum in motor learning (Ito 1984), the FORs could play a specific role in the learning phase of the foveating behavior, compensating for idiosyncratic differences in neuro-muscular morphology and anisotropies in the mechanisms triggering and executing fixational saccades. Such an involvement in optimizing the acquisition of visual information from the fovea would support a recent theory on the function of the cerebellum (Bower 1997). Future ontogenetical and phylogenetical studies will link the development of this part of the medio-posterior cerebellum to the development of foveal fixation in the newborn baby (Slater and Brenner 1989) and across species (Martinez-Conde and Macknik 2008).

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