Acute Superior Oblique Palsy in Monkeys: II. Changes in Dynamic Properties during Vertical Saccades

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PURPOSE. To investigate vertical and torsional eye motion during and immediately after vertical saccades with acute acquired superior oblique palsy (SOP) in monkeys.

METHODS. The trochlear nerve was severed intracranially in two rhesus monkeys. After surgery, the paretic eye was patched for 6 to 9 days, and then binocular viewing was allowed. Three-axis eye movements (horizontal, vertical, and torsion) were measured with binocular, dual search coils. Eye movements were recorded before surgery and then beginning 2 to 3 days after surgery during 20° vertical saccades over a ±20° horizontal and vertical range.

RESULTS. The main findings were: (1) Saccade amplitude in the paretic eye (PE) was smaller than that of the normal eye (NE), especially for downward saccades with the PE in adduction; (2) vertical drift was backward after upward saccades with the PE in adduction or abduction, onward after downward saccades with the PE in adduction, but backward for downward saccades with the PE in abduction, drift time constants averaged 35 ms; (3) peak dynamic blip intrasaccadic torsion increased (relative extorsion), the most for upward saccades with the PE in abduction; (4) postsaccadic torsional drift increased (relative intorsion), the most for downward saccades with the PE in adduction; and (5) the peak velocity–amplitude relationship in vertical saccades was little affected, but the ratio between the peak velocity of the two eyes was a consistent indicator of the palsy.

CONCLUSIONS. Rhesus monkeys with acute acquired SOP show characteristic changes in vertical and torsional movements during and immediately after vertical saccades that help define the ocular motor signature of denervation of the SO muscle. These dynamic changes were largely unrelated to the changes in static alignment over time, suggesting that static and dynamic disturbances in SOP are influenced by separate central mechanisms. (*Invest Ophthalmol Vis Sci.* 2007;48:2612–2620) DOI:10.1167/iovs.06-1318

Most studies of the changes in eye movements that occur with superior oblique palsy (SOP) have focused on changes in alignment during fixation with the head upright or tilted. The effects of SOP on changes in eye *movements* have received less attention, with the most emphasis on saccade velocity.[1–5] With a few exceptions,[6,7] other characteristics such as the drift of the eye after vertical saccades and the patterns of torsion associated with vertical saccades have not been reported. In the preceding paper we quantified the changes in static vertical and torsional alignment induced by acute SOP in monkeys, as well as the influence of disparity cues on those changes.[8] We do the same in this paper for the dynamic properties of saccades, to define further the ocular motor signature of acute SOP and to investigate the evolution of these deficits over time. Furthermore, the data will provide additional quantitative information that can form a frame of reference for analyzing the clinical presentation of SOP in humans.

METHODS

General Experimental Procedure and Eye Movement Recording

Two female juvenile rhesus monkeys (M1 and M2) weighing from 4 to 6 kg were studied. Three dimensional (3-D) eye movements were measured with the magnetic field search coil method. Two eye coils made from Teflon-coated stainless steel wire were implanted in each eye. A directional coil of 14.5 to 15 mm in diameter was sutured on the sclera under the conjunctiva just in front of the insertion of the rectus muscles, and a torsional coil with a diameter of 7.5 to 8 mm was implanted superotemporally between the lateral and superior rectus muscles and centered on the equator of the globe. The coils were sutured to the sclera with several small sutures. SOP was induced by an intracranial sectioning of the trochlear nerve in monkeys under gas anesthesia. Control recordings were obtained before surgery. A period of monocular viewing was also tested with the to-be-paretic eye (the left eye in M1, the right eye in M2) covered with an opaque acrylic patch attached to the head plate for 4 (M1) and 7 (M2) days. Then binocular viewing was allowed. Immediately after the trochlear nerve surgery, the paretic eye was patched for 6 days in the case of M1 and 9 days in M2. Then habitual binocular viewing was allowed again. Further details of the general experimental procedure and the eye movement recording are described in the preceding paper.[8] The protocol was approved by the Institutional Animal Care and Use Committee (IACUC) of the Johns Hopkins University and adhered to the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research.

Stimulus Presentation

The target stimulus, a 0.3° × 0.3° red square, was under computer control and rear projected on a tangent screen 66 cm in front of the monkey. The experiment was performed in an otherwise dark room. Twenty-degree vertical saccades were elicited from three different horizontal positions (left 20°, midline, and right 20°), and between the following pairs of elevations: down 20° and 0°, down 10° and up 10°, and 0° and up 20°. The target jumped to its new position, which was predictable, after the animal had kept its eye within a fixation window

of 4° for at least 250 ms. For each pair of targets, we recorded 10 trials in each session and in most cases more than 5 trials were acceptable for analysis (i.e., the monkey performed the task adequately by looking directly to the next target). Viewing was always monocular during the recording session, even after habitual binocular viewing was allowed.

Data Analysis

Data were analyzed offline with commercial software (MatLab; The MathWorks, Natick, MA). The 3-D position of each eye was recorded and analyzed with rotation vectors. The reference position for each eye was taken with monocular viewing at straight ahead (0°, 0°). To calculate the angular velocity trajectories, we differentiated the data using a fifth-order REMEZ (MatLab; The MathWorks) FIR filter, designed to differentiate up to 40 Hz and to low-pass filter above 80 Hz. An interactive program was used to mark automatically the onset, the peak velocity, and the end of saccades. The onset of the saccade was defined when the eye velocity first exceeded 30°/sec and the end of the rapid-pulse portion of the saccade was taken when the eye velocity first dropped below 30°/sec. The amplitude of the saccadic pulse was determined by subtracting the vertical eye position at the onset from that at the end of the saccade. The step position of the eye after the saccade was taken at 80 ms after the end of the pulse. This time point was chosen to ensure that there was no time for visual feedback to alter the pattern of any drift after the saccade. The step amplitude of the saccade was calculated by subtracting the vertical eye position at the onset of the saccade from the step position after the saccade. Trials were rejected if the initial position of the saccade was incorrect, if the saccade was not directed toward the target (e.g., in the opposite direction), if the amplitude was less than 60% of the amplitude of the target displacement, or if the trial was contaminated by blinks. Corrective saccades were also excluded from the quantitative analysis.

Dynamic or blip intrasaccadic torsion was defined as the peak torsion during the vertical saccade above the torsion predicted from the change in static torsion associated with fixation 50 ms before the onset and 200 ms after the end of the vertical saccade.[9] Two hundred milliseconds was chosen as the time for the steady state value of post-saccadic torsional drift because after surgery (when there was more obvious drift than before surgery) the time constants of torsional drift were small, ranging between 12 and 77 ms (mean, 38 ± 19.2 ms [SD], in both monkeys; up and down saccades in adduction and abduction). Postsaccadic torsional drift was quantified by subtracting the torsional eye position at the end of the pulse portion of the vertical saccade from the torsional eye position at 80 ms after the end of the saccade. The time course of vertical and torsional drift was approximated by a single exponential fit ($y = a + be^{(-cx)}$) to the first 80 ms of postsaccadic drift and a time constant ($T = 1/c$) was determined. Statistical analysis was performed with three-way ANOVA, with time (before and after surgery) and horizontal and vertical positions from which the saccades were made as factors. All data presented are for 20° vertical saccades with the normal eye viewing.

RESULTS

Qualitative Features

Figure 1 shows representative single trials of the paretic eye for vertical saccades, with the normal eye viewing and the paretic eye in adduction or abduction, before and after lesioning in monkey 1 (M1). Before surgery, saccades were recorded after 4 days of monocular viewing (to-be-paretic eye patched) and postlesion saccades were recorded 4 days after SOP in M1 and 3 days in M2 (the surgical eye was continuously patched after the surgery). In the left-hand panels, vertical and torsional eye positions are plotted against time, and in the right-hand panels, torsion is plotted against vertical eye position. The main features of acute SOP, which were shown by both monkeys, were as follows: (1) The amplitude of the pulse of the vertical saccade made by the paretic eye relative to the normal eye was

diminished, more in adduction than in abduction. (2) In downward saccades with the paretic eye in adduction, onward vertical postsaccadic drift developed (Fig. 1A, top). For downward saccades with the paretic eye in abduction, backward vertical postsaccadic drift developed (Fig. 1A, bottom). For upward saccades, backward vertical postsaccadic drift developed (Fig. 1B). (3) The change in static torsion (gray solid traces) between before and after a vertical saccade increased after the lesion, toward extorsion (positive) for downward saccades and toward intorsion (negative) for upward saccades. This reflects the gradient of static torsional deviation (considerable extorsion in down gaze, small extorsion in up gaze) associated with the SOP. (4) The peak dynamic, or blip torsion (see the Methods section) was altered by the lesion. The change was particularly obvious for downward saccades in adduction (Fig. 1A, top right; note increase in peak dynamic torsion after surgery with a maximum at the vertical arrow) and for upward saccades in abduction (Fig. 1B, bottom right; note reversal in direction of peak dynamic torsion after surgery with a maximum at the vertical arrow). In both cases there was more relative extorsion during the saccade. (5) After the lesion there was often a change in the direction of post-saccade drift toward intorsion. For example, in Figure 1, right, note the torsional (*y*-axis component) and vertical (*x*-axis component) components of post-saccade drift. We found no significant effects of the SOP on horizontal saccade dynamics or on torsion associated with horizontal saccades; accordingly, horizontal saccades were not examined further.

Quantitative Features

Pulse-Pulse Ratios. Figure 2 shows a comparison of the pulse-pulse ratios before surgery (after 4 days of wearing the patch on the to-be-paretic eye) and 3 to 4 days after the lesion (while the eye was still patched). The pulse–pulse ratio was defined as the amplitude of the high-speed (pulse) part of the movement made by the paretic eye (PE) divided by the amplitude of the pulse made by the normal eye (NE; see the Methods section). Both monkeys showed a similar pattern of disconjugacy, with smaller pulses by the paretic eye. Not surprisingly, in both animals the deficit was greater for downward than for upward saccades and with the paretic eye in adduction than in abduction. The change in pulse–pulse ratio was slightly greater for downward saccades in M2 than in M1 and slightly greater for upward saccades in M1 than in M2. In each monkey, statistical analysis of the pulse–pulse ratios in up and down vertical saccades with a three-way ANOVA with three factors (time before versus after surgery, horizontal eye position, and vertical eye position) showed significant differences (at least $P < 0.05$) for before and after pulse–pulse ratios for up and downward saccades. In addition, after surgery, significant differences emerged in the effect of vertical position on pulse–pulse ratios for up and downward saccades in both monkeys, in the effect of horizontal position on upward and downward saccades in M1 and in the effect of horizontal position on downward saccades in M2.

The change over time in the pulse–pulse ratio in 20° vertical saccades is shown in Figure 3. In both monkeys, this parameter changed significantly as a result of the SOP, but was stable before and after surgery. Even after habitual binocular viewing was allowed, changes in the pulse–pulse ratio were minimal, with the exception of a further *decrease* in the ratio for upward saccades (Fig. 3, arrow) in M2 (who preferred to fix with her paretic eye).

Vertical Saccade Velocity. As expected, postlesion saccades by the paretic eye were both smaller and slower, so that a typical main sequence plot (peak velocity versus saccade amplitude) did not reveal changes in saccade velocity, per se. Instead, we computed the peak-velocity ratio between the
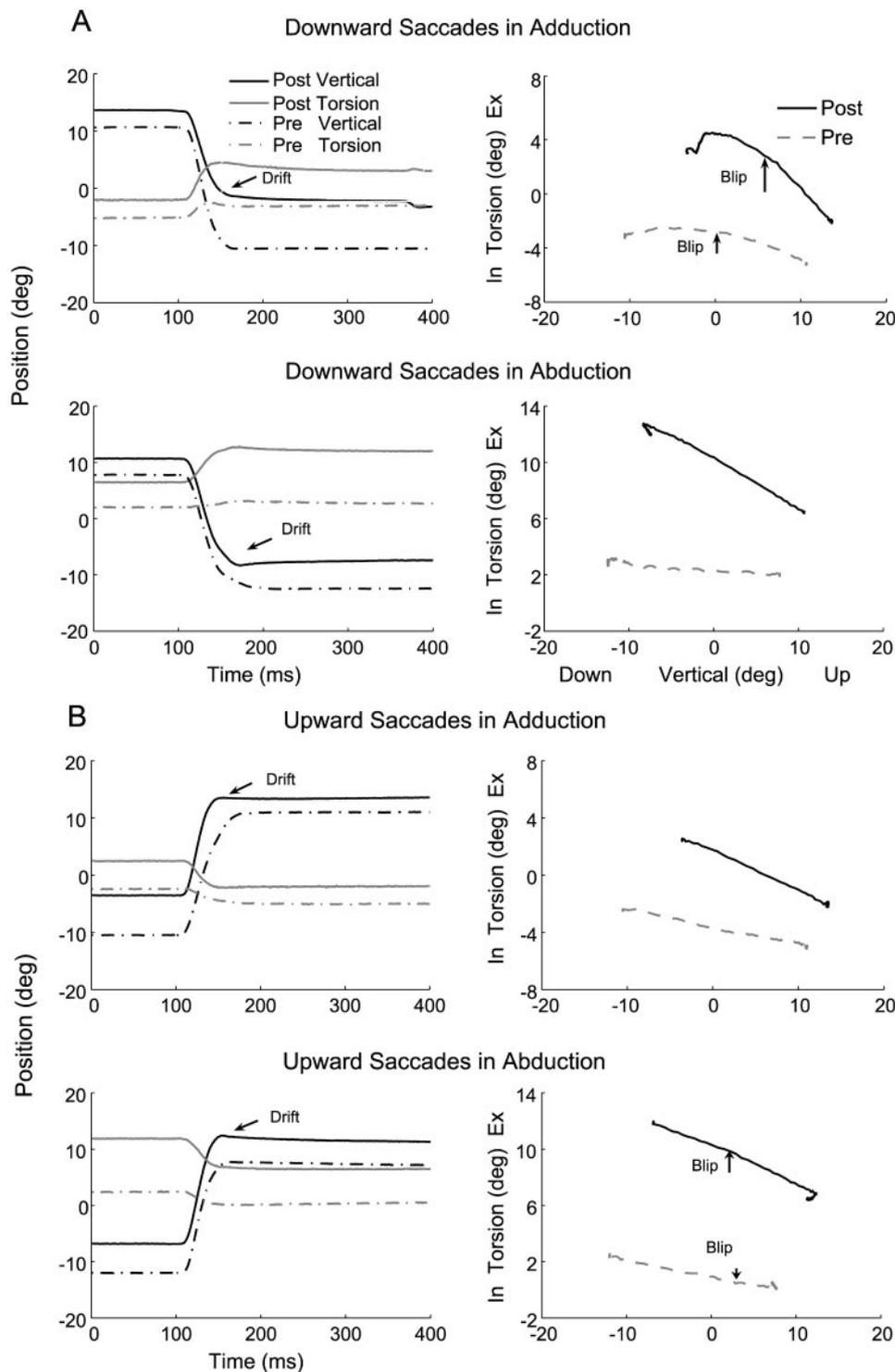
FIGURE 1. Downward (**A**) and upward (**B**) saccades of the paretic eye (PE) in M1. Representative individual saccade trials with time trajectories on the left and position plots of the same data on the right. Data are normal eye viewing and were taken both before and after (3–4 days) surgery during habitual monocular viewing. *Vertical arrows*: point of maximum intrasaccadic blip torsion.

paretic eye and the normal eye (Fig. 4), which allowed us to examine the effect on velocity without contamination from variability in saccade amplitudes and to control for any spontaneous fluctuations in the central commands that might influence the velocity of saccades made by both eyes. The results were similar to the changes in pulse–pulse ratios (Fig. 2), with the effects of the SOP being greatest for downward saccades made with the paretic eye in adduction. In each monkey, statistical analysis of the peak-velocity ratios during up and down vertical saccades using a three-way ANOVA as before, showed significant differences (at least $P < 0.05$) in pre- and

post-peak-velocity ratios in up and downward saccades. In addition, after surgery significant differences emerged in the effect of vertical and horizontal position on peak-velocity ratios for upward saccades in both monkeys and for downward saccades in both monkeys except in M2, in the vertical position.

The change over time of the peak-velocity ratios for 20° saccades is shown in Figure 5. Clearly, in both monkeys the peak-velocity ratio decreased immediately after surgery, more so for downward saccades than for upward saccades and, in downward saccades, more so with the paretic eye in adduc-
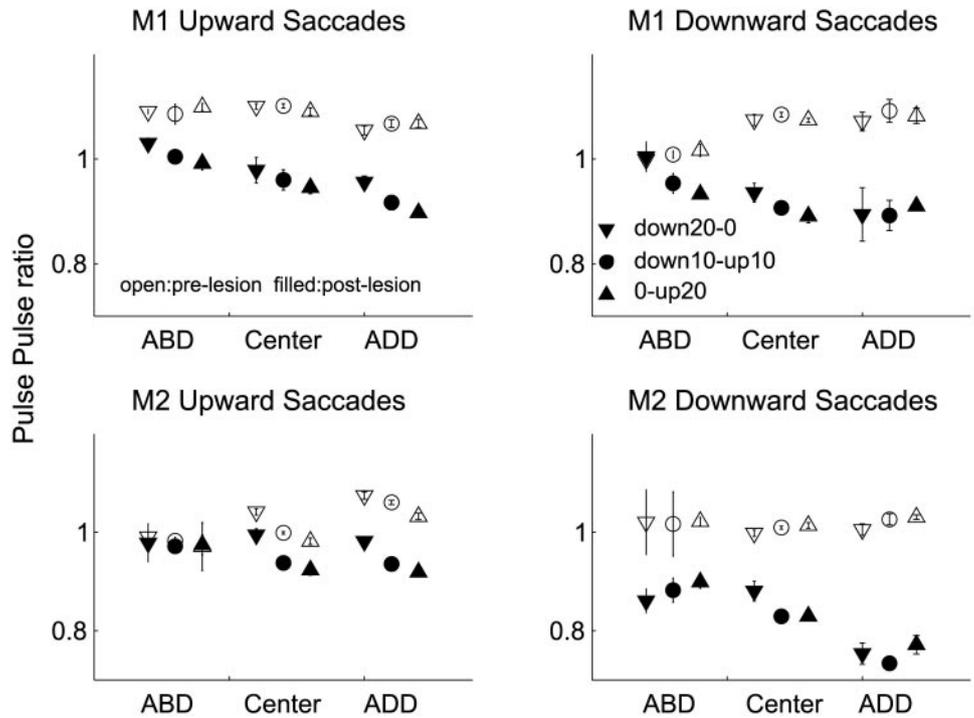
FIGURE 2. Pulse–pulse ratios (PE/NE). ADD (adduction) and ABD (abduction) refer to the paretic eye position. Data are the mean ± SD. Twenty-degree saccades are shown from different starting and ending points. Note the greatest change between before and after surgery occurred for down saccades with the paretic eye in adduction.

tion. Otherwise, this parameter was remarkably stable in both animals, even after habitual binocular viewing was allowed. Note that in M2 when habitual both eyes viewing was first allowed after surgery, there was a considerable decrease in the pulse–pulse ratio during upward saccades (Fig. 3, arrow), but with a lesser decline in the peak-velocity ratio (Fig. 5, arrow).

Postsaccadic Drift. Pulse-step ratios, which quantify postsaccade vertical drift, are shown for the paretic eye in Figure 6. They were calculated from the position of the eye just before, at the end and 80 ms after the end of the saccade (see the Methods section). Both monkeys behaved similarly after sur-

gery. For downward saccades, pulse-step ratios decreased, implying onward or less backward (compared with before surgery) drift with the paretic eye in adduction, and increased (implying backward drift) with the paretic eye in abduction. For upward saccades, pulse-step ratios always increased, implying more backward or less onward (compared with before surgery) drift. Again using the three-way ANOVA as before, there was a statistically significant difference (at least $P < 0.05$) between pre- and postlesion pulse-step ratios for upward but not for downward saccades. Position effects (horizontal or vertical versus pulse-step ratio), however, were all significant.
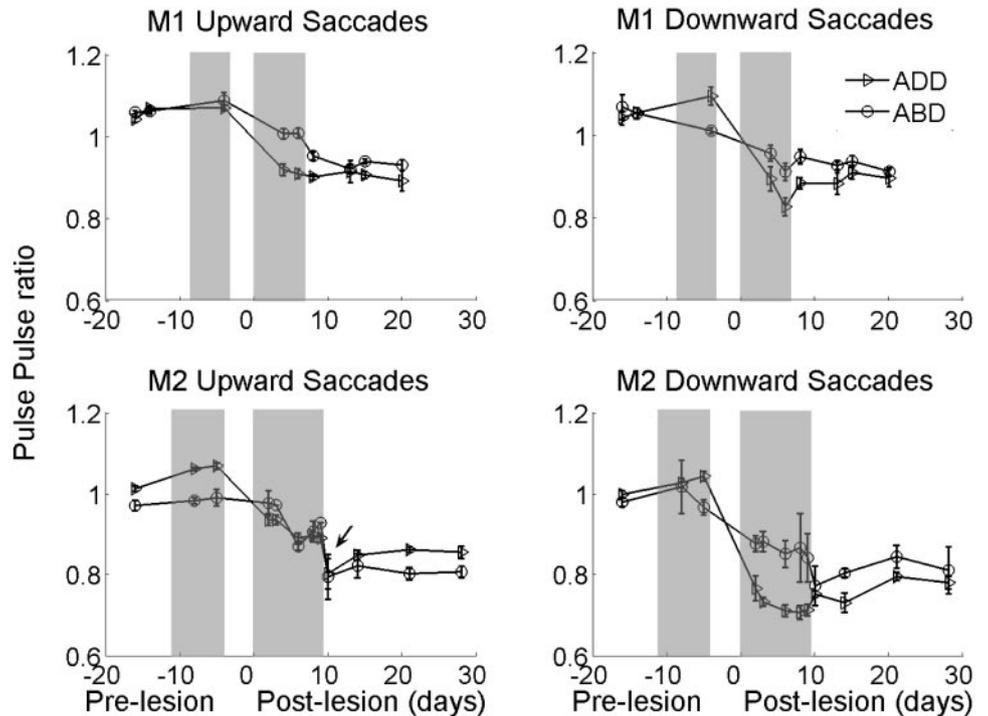

FIGURE 3. Time course of change in pulse–pulse ratios (PE/NE). *Shaded areas*: time when the paretic eye (or to-be-paretic eye, prelesion) was patched. Data are the mean ± SD. *Arrow:* relatively abrupt decrease in pulse–pulse ratio, 24 hours after habitual binocular viewing was allowed in M2.

M1 Upward Saccades

M1 Downward Saccades

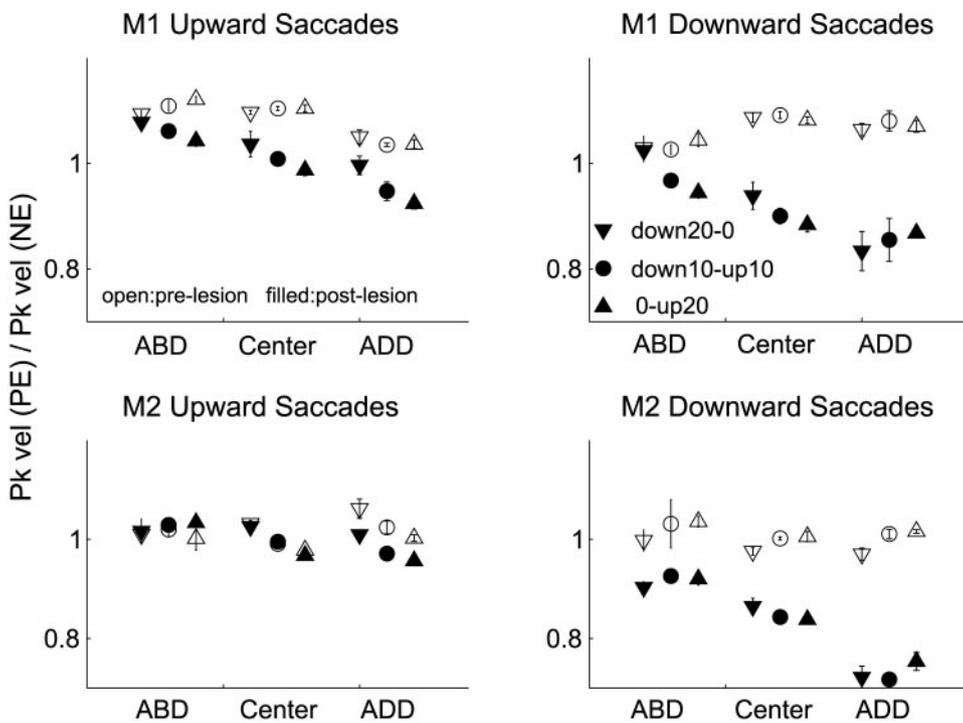## M2 Upward Saccades

M2 Downward Saccades

FIGURE 4. Peak-velocity ratios (PE/ NE). Data are the mean ± SD. Twenty-degree saccades are shown from different starting and ending points. Note the greatest change between before and after surgery is for downward saccades with the paretic eye in adduction.

The first 80 ms of drift was fit with a single exponential. This was satisfactory for all saccades, before and after surgery, with the exception of downward saccades in adduction in M2, in which the drift seemed more linear after surgery. There were no statistically significant differences between pre- and postlesion time constants of drift. Combining both monkeys, the mean prelesion time constant was 38.5 ±14.7 ms and the mean postlesion time constant was 34.3 ± 10.8 ms. The average difference (absolute—in some cases there was an increase, in others a decrease) between before and after surgery was small, 10.8 ± 7.6 ms.

The change over time in the pulse-step ratios for 20° vertical saccades is shown in Figure 7. During the prelesion period of patching, changes were small without a consistent pattern. During the immediate postlesion period, the only consistent change was for downward saccades in M2. There was a gradual increase in the pulse–step ratio in abduction and a gradual decrease in adduction in the paretic eye. There were no corresponding changes in the pulse–step ratio in the normal eye. Once habitual binocular viewing was allowed, for M2 the change in pulse-step ratio of the PE soon reversed direction, with the ratio moving toward 1.0. Postlesion, pulse-step ratios
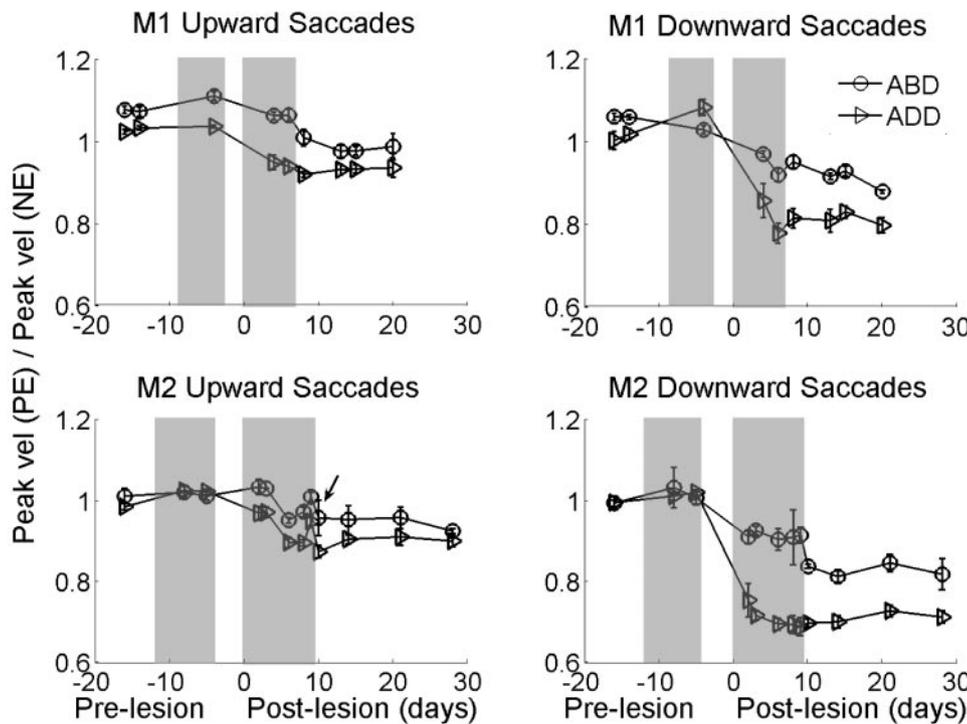
M1 Upward Saccades

M1 Downward Saccades

M2 Upward Saccades

M2 Downward Saccades

FIGURE 5. Time course of change in peak-velocity ratios (PE/ NE) for 20° made from up 10° to down 10°. *Shaded areas*: time when the paretic eye (or to-be-paretic eye, before surgery) was patched. Data are the mean ± SD. *Arrow*: the data point at which there was a much larger change in the pulse-pulse ratio (compare with Fig. 3).
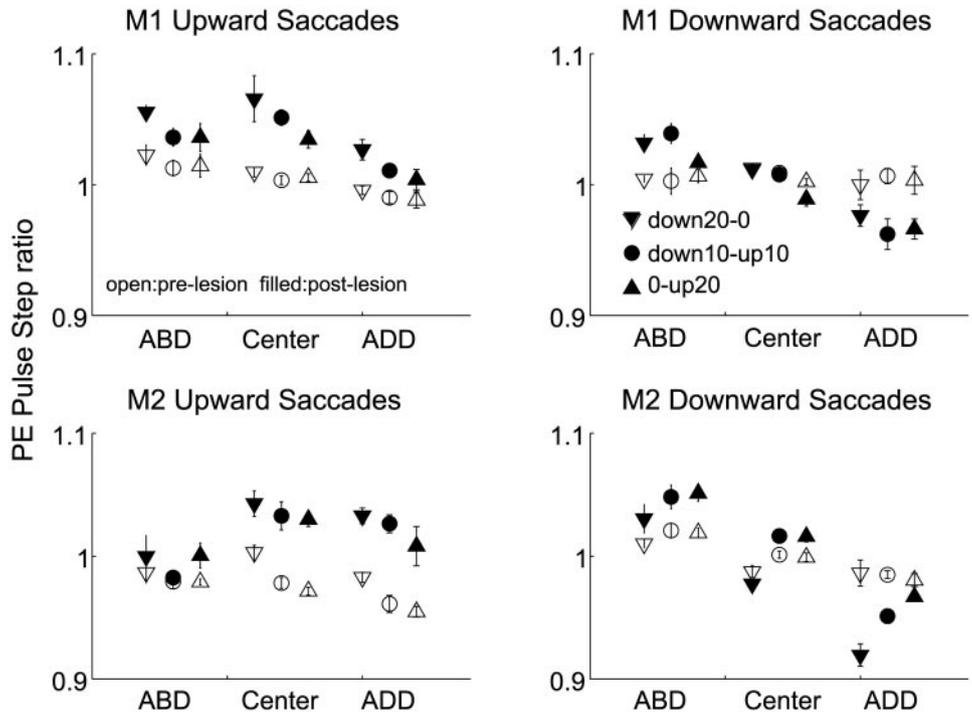
FIGURE 6. Pulse–step ratios for PE (paretic eye) at 80 ms after the pulse (mean ± SD). Twenty-degree saccades are shown from three different vertical starting and ending points.

in the normal eye changed little over time except for M2 during downward saccades (Fig. 7, bottom right) after binocular viewing was allowed. Note the oppositely directed changes in the pulse–step ratio in the normal compared with the paretic eyes (Fig. 7, dash-dot arrows).

Note also that in M1, at approximately 15 days after surgery, the pulse–step ratio increased toward 1.0 in downward saccades with the paretic eye in adduction, without any corresponding change in the pulse–step ratio of the normal eye (Fig. 7, top right, solid arrow). In M2, during upward saccades with the paretic eye in adduction and abduction, between 15 and 22 days after surgery, the pulse–step ratio in the paretic eye

dropped to approximately 1.0, with little change in the ratio in the normal eye (Fig. 7, bottom left, solid arrow). Likewise, during the same period, during downward saccades with the paretic eye in adduction, there was an increase in the pulse–step ratio in the paretic eye toward 1.0 without a corresponding change in the pulse–step ratio in the normal eye (Fig. 7, bottom right, solid arrow).

Peak Dynamic Torsion. Peak dynamic (blip) torsion measures the amount of torsion during a vertical saccade above that expected from the change in static torsional eye orientation between the two steady state torsion positions before and after the saccade (see the Methods section). The effects of the
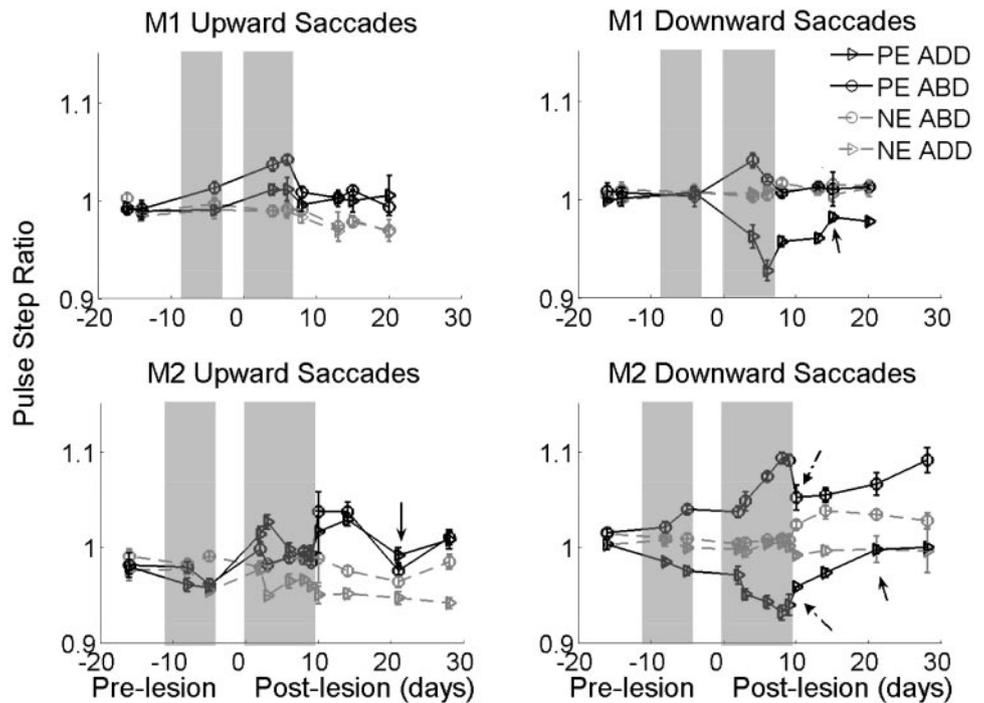

FIGURE 7. Time course of change in pulse–step ratios in the normal eye (NE) and paretic eye (PE). ADD (adduction) and ABD (abduction) refer to the position of the specified eye. Shaded areas are the time when the paretic eye (or to-be-paretic eye, before surgery) was patched. Data are the mean ± SD. *Dash-dot arrows:* suggestive of conjugate adaptation; *solid arrows:* disconjugate adaptation.

lesion on the peak values of dynamic torsion during vertical saccades are shown in Figure 8. Results were similar in both monkeys. In both animals, extorsion before surgery was associated with all downward saccades, with the exception of M2 for downward saccades (between the 0° and down 20° positions) made with the to-be-paretic eye in abduction (Fig. 8, bottom right, downward-oriented triangles). There was a gradient in peak dynamic torsion for downward saccades such that peaks were higher (relatively more extorsion) during saccades made in adduction. Before surgery, during upward saccades there was more dynamic intorsion than during downward saccades, except in M2 during saccades made with the to-be-paretic eye in abduction (Fig. 8, left, downward-oriented triangles; between the 0° and down 20° positions). After surgery, there was a small increase in relative extorsion during vertical saccades in both directions, though the effect was greatest for upward saccades, especially in abduction. Three-way ANOVA showed a significant difference (at least $P < 0.05$) between pre- and postlesion peak dynamic torsion in both animals. There was a significant effect of horizontal position on postlesion saccades except for upward saccades in M2. There was no consistent pattern of change in peak dynamic torsion over time—neither during the period of monocular viewing before and after surgery nor after habitual binocular viewing was allowed. Likewise, there was no correlation between the changes in peak dynamic torsion and the horizontal phoria at the beginning position before the vertical saccade.

Changes in Torsion: Postsaccade Torsional Drift. Postsaccade torsional drift was based on the difference in torsion between the end of the vertical saccade and 80 ms later, when vertical eye position had largely become steady. The effects of the lesion on torsional drift after vertical saccades are shown in Figure 9. In both animals, the prelesion postsaccade torsional drift was relatively small; most fell between −0.25 and 0.25° (Fig. 9, open symbols). After surgery, in saccades of all types in both directions in both animals, there was a relative increase toward more intorsion (i.e., at the end of the pulse, the eye is more extorted than at the end of the step). This effect was greatest for downward saccades with the paretic eye in adduction. The three-way ANOVA showed a statistically significant

difference (at least $P < 0.05$) between pre- and postlesion postsaccadic torsional drift. Horizontal position effects were also significant in both animals. There was no consistent pattern of change in torsional drift during the period of monocular viewing before and after surgery or after habitual binocular viewing was allowed after surgery. Likewise, there was no correlation between the changes in torsional drift and the horizontal phoria at the beginning position before the vertical saccade. The time constants of postsaccade torsional drift after surgery were in the same range (12–77 ms; mean 38 ± 19.4 ms [SD], in both monkeys, up and down 20° saccades in adduction and abduction) as the time constants of vertical postsaccadic drift.

DISCUSSION

There are few studies of the dynamic properties of saccades in humans or monkeys with vertical paralytic strabismus. With a few exceptions in humans[5-7] and in monkeys[10,11] the focus has been solely on saccade velocity. Furthermore, prior studies of acquired paralytic strabismus in monkeys have primarily studied the effects on the *horizontal* rectus muscles, using botulinum injections or recessions or tenotomies, which are procedures in which the ocular muscles are directly manipulated.[12-14] An exception is the reports of Lewis et al.[10,11] who studied the effect of proprioceptive deafferentation on disconjugate adaptation in monkeys with vertical strabismus (trochlear nerve palsies). Furthermore, except for Wong et al.,[6] torsion during vertical saccades has not been measured in patients with acquired vertical paralytic strabismus, and no one has reported measures of torsion in monkeys with acquired strabismus. In the present report, we have described in detail the dynamic deficits associated with SOP, both acutely and over time, thus defining further the ocular motor signature of SOP palsy in monkeys.

Saccade Amplitude

After surgery, vertical saccades in the paretic eye became smaller, as reflected by the changes in pulse–pulse ratios (a measure of vertical intrasaccadic disconjugacy). The greatest
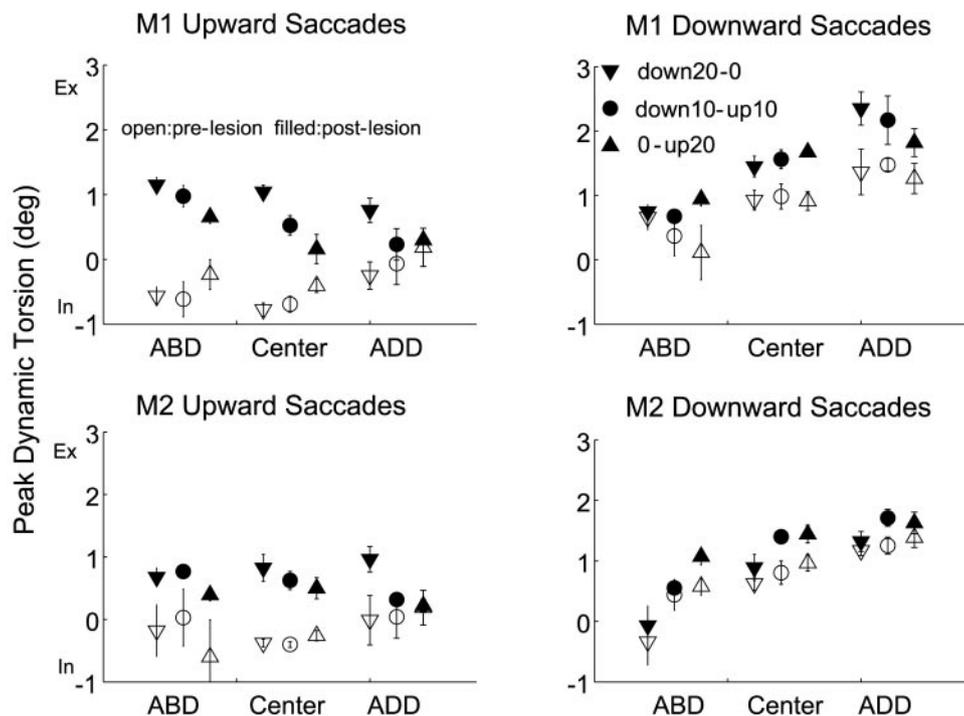

FIGURE 8. Peak dynamic (blip) torsion for the PE (paretic eye). Data are the mean ± SD. Twenty-degree saccades are shown from three different vertical starting and ending points.
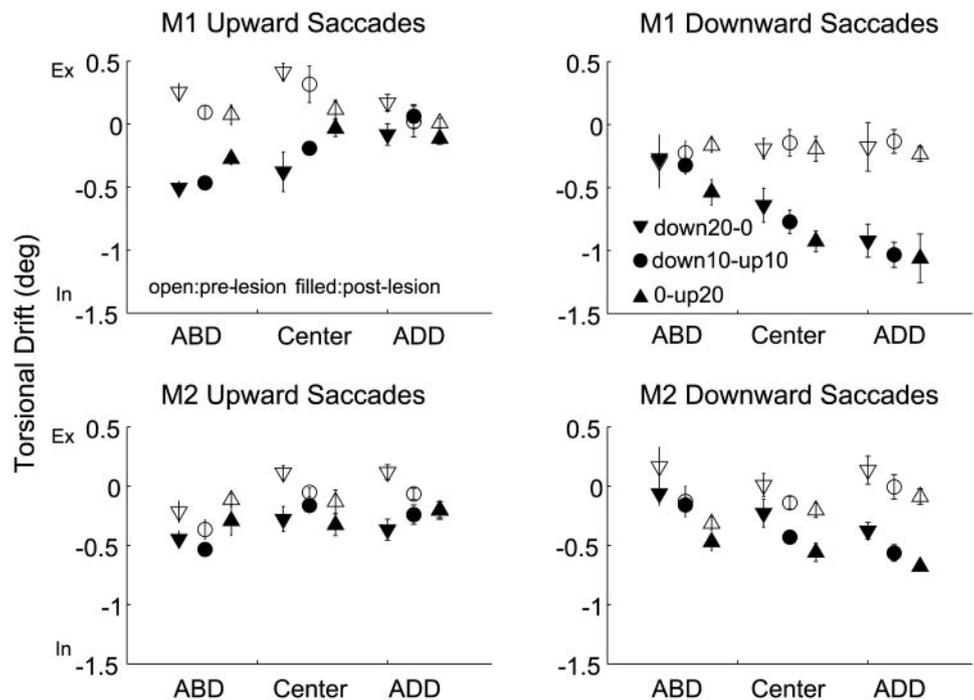
FIGURE 9. Postsaccade torsional drift in the PE (paretic eye). Data are the mean ± SD. Twenty-degree saccades are shown from three different vertical starting and ending points.

changes (up to 20%) were during downward saccades, especially when the paretic eye was in the adducted position (Fig. 2) which is where the superior oblique muscle contributes more to vertical eye rotation. The changes were stable over time, especially so after binocular viewing was allowed (Fig. 3). (One exception was M2 who had a relatively large *decrease* in the pulse–pulse ratios, i.e., an *increased* disconjugacy, primarily for upward saccades just after binocular viewing was allowed.) One contributing factor to the increasing dynamic misalignment at this time could be that M2 preferred to fix with her paretic eye—which would act to induce conjugate saccade adaptation to optimize saccade metrics for the paretic eye.[14–16]

Saccade Velocity

After surgery we observed a conspicuous (20%–30%) decrease in the peak velocity (paretic eye)-to- peak velocity (normal eye) ratio for downward saccades in adduction, where the contribution of the SO to vertical velocity is normally strongest (Fig. 4). This difference was consistent in both monkeys and remained virtually unchanged throughout our observation period (Fig. 5), despite considerable changes in static eye alignment. In the past, Tian and Lennerstrand[5] also quantified peak saccade velocity in SOP. They suggested that an elevated ratio of peak velocity in up to down vertical saccades in a given eye in the adducted position may have diagnostic significance for acquired SOP, but their data still showed considerable overlap among normal subjects, patients with congenital SOP, and patients with acquired SOP.

Postsaccadic Drift

Whereas the effects of a muscle palsy on the amplitude and the speed of a movement are, at least qualitatively, straightforward, it is conceptually harder to predict the effect of a palsy on the matching of the pulse and step of innervation. The direction of postsaccadic drift is determined by whether the palsy impairs more the ability to overcome viscosity to rotate the eye rapidly, or the ability to overcome elasticity to hold the eye in position. If both these abilities are equally affected, no drift is observed.

Indeed, compared with the changes in saccade amplitude and velocity, postsaccadic vertical drift showed a more complex dependence on orbital position and direction. Drift was backward after upward saccades, but in downward saccades it was *onward* after saccades made with the paretic eye in adduction and *backward* after saccades made with the paretic eye in abduction (Fig. 6). Note that this asymmetry during downward saccades occurs despite the fact that the innervational force to the superior oblique muscle is relatively independent of horizontal eye position.[17]

The adaptive changes that developed in postsaccadic drift over time were also complex. During the immediate postlesion period while the paretic eye was patched, M2 showed an increasing pulse-step mismatch (deviating further from 1.0) for downward saccades made by the paretic eye. Once habitual binocular viewing was allowed, this trend immediately reversed (moving back toward 1.0), possibly related, at least in part, to M2's habitually fixing with the paretic eye (Fig. 7, dot-dash arrows). Thus, to minimize postsaccadic drift of the paretic eye, M2 may have adjusted innervation *conjugately* to both eyes, which would be at the expense of increasing postsaccadic drift (albeit in the opposite direction) of the normal eye.[14,15] In both animals, however, there were later *disconjugate*, almost monocular changes in postsaccadic drift that minimized the difference in pulse-step ratios between the two eyes (Fig. 7, solid arrows). This capability of disconjugate adaptation of postsaccadic drift has been described previously in monkeys with paralytic strabismus[7,10,12] and seems to depend on the presence of disparity cues but not on fusion.[10]

Comparison with Previous Reports

A decrease in vertical saccade velocity has been reported in some[1,2,5] but not other studies of SOP,[3,4] though there was considerable heterogeneity in the patient groups studied. Wong et al.[6] reported an unusual pattern of change in saccade dynamics in their study of paralytic SOP in humans. In two patients with acute SOP (duration of weeks), saccades made by the paretic eye were *hypermetric* with a rapid drift *backward* drift, lasting 175 to 400 ms (based on their Fig. 4, Ref. 6) and

leading to large pulse–step ratios of between ~1.3 and ~1.67. Although the exact lesions in these patients are unknown (and might have involved the brain stem), and the patients were not examined immediately after the onset of their palsy, the striking discrepancy between their findings, and ours points to a need for examining saccade dynamics further in humans and monkeys with paralytic strabismus.

The duration of vertical saccade drift in our monkeys was brief; when approximated as an exponential, the time constant was ~35 ms. This value is close to that described elsewhere in experimental horizontal paralytic strabismus in monkeys[12,13] but lower than in reports of experimental SOP.[10] Our values are also close to those after experimental stimulation of an ocular motor nerve.[18] These values are much less than predicted from the mechanical properties of the orbit and the behavior of ocular motor neurons during saccades and fixation (~200 ms)[19]; the mechanism is unclear but probably relates to unmasking of nonlinear properties in the orbital plant when a single muscle is paralyzed.

Clinical Implications

One prominent finding in our monkeys was the relative dissociation between the effects of SOP on static alignment (our preceding paper[8]) and on the dynamic properties of saccades (described here). Over time, there were large changes in vertical misalignment (a decrease during the initial period of monocular viewing after surgery and an increase once habitual binocular viewing was allowed) that were not paralleled by changes in dynamic behavior. It thus seems that the changes in static vertical alignment that occur after SOP mostly reflect the actions of neural mechanisms distinct from those that control the dynamic properties of saccades.

Potentially, then, dynamic measures may carry more information about the underlying cause of vertical misalignment. In both monkeys several patterns of change consistently emerged, including changes in pulse–step ratios and intrasaccadic peak torsion that were sometimes larger in upward than in downward saccades. Particularly appealing however, is the peak-velocity ratio, since in our two monkeys it was remarkably robust despite presumed adaptive processes. Our results suggest that a peak-velocity ratio that is lower in downward than in upward saccades and more so in adduction than in abduction, may be a reliable indicator of SOP. Of course, the diagnostic usefulness of these parameters should be evaluated by careful quantification of eye movements in both clinical and experimental settings.

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