Investigative Ophthalmology & Visual Science, Vol. 29, No. 8, August 1988

Saccadic Latency as a Measure of Afferent Visual Conduction

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Latency to initiate a saccadic eye movement to a visual target, and visual evoked potential, were measured in seven patients with resolved unilateral optic neuritis. Saccades were delayed when the target was presented to the clinically involved eye, but were normal when the contralateral eye was tested. With binocular target presentation, saccades were symmetric between eyes and normal in latency. In two patients with pituitary adenoma and low-grade bitemporal field defects, saccades were delayed when targets were presented in the temporal field, but were within normal limits when presented in the nasal field. These results cannot be attributed to lesions in the motor pathways. It is concluded that saccadic latency to visual targets is a valid measure of afferent conduction. If the robust delays found in this study prove to have test-retest reliability, saccadic latency may provide a measure of afferent function which is sensitive to the demyelination that preceeds neuronal degeneration and sensitivity loss in patients suspected of having optic neuropathy. Invest Ophthalmol Vis Sci 29:1331–1338, 1988

Previous research has shown that the quantitative study of eye movements provides a technique for documenting clinically silent lesions in the visual pathways of suspected multiple sclerosis (MS) patients.[1–5] The purpose of these studies has been to detect lesions in the ocular motor pathways. Among the frequent ocular motor abnormalities observed in MS patients are slowing of the adducting eye, associated with a lesion in the medial longitudinal fasiculus; nystagmus and impaired saccade accuracy, suggesting a cerebellar lesion; and low-gain pursuit, probably indicating involvement of the supranuclear ocular motor pathways.

Most of these studies have reported increased saccadic latency (SL) in some MS patients.[1–4] This result has generally been attributed to a lesion in the supranuclear efferent pathway.[2,4] Delays in afferent conduction of the optic nerve is a common finding in patients with MS and could conceivably contribute to observed SL delays.

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Portions of this paper were presented at the March, 1986 topical meeting of the Optical Society of America on Noninvasive Assessment of the Visual System, in Monterey, California.

Submitted for publication: October 20, 1987; accepted March 17, 1988.

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The current study is intended to demonstrate that saccadic reaction time to the displacement of a visual target can be used as a measure of afferent visual function. The potential of this method in detecting subclinical dysfunction of the optic nerve and chiasm is shown, and the relationship between clinical findings, visual evoked potential (VEP), and SL results is discussed.

Materials and Methods

Subjects

Controls: Nine visually normal controls participated in the SL test. This group ranged in age from 21 to 39 years, and had visual acuities correctable to 20/20 without history of neurologic or ophthalmologic abnormality. Forty similar individuals were tested as control subjects for the VEP. They ranged from 18 to 43 years of age.

Patients: Table 1 summarizes the diagnostic categories and visual findings of the 14 patients in this study. Seven patients had a clinical history of unilateral optic neuritis. At the time of testing all had Snellen acuity of 20/25 or better. Goldmann visual fields were normal in all but patient 7 who had a 5° central field defect to the I-2e target. Afferent pupillary responses were normal in all but patients 2 and 7 who had persistent left afferent defects despite relatively normal visual acuity. Patient 6 had a right internuclear ophthalmoplegia (INO) and patient 4 had upbeating nystagmus in upgaze. All other optic neuritis patients had no clinically apparent abnormalities of eye movement.

Table 1. Visual symptoms of patients at time of testing

Pt. #	Age	DX	Acuity OD	Acuity OS	HX O.N. OD	HX O.N. OS	Field	APD	Oc. Motor
I. Optic neuritis									
1	27	Def. MS	20/15	20/25	−	+	Full	No	NML
2	31	Def. MS	20/15	20/15	−	+	Full	Yes	NML
3	26	Pos. MS	20/15	20/15	−	+	Full	No	NML
4	35	Pos. MS	20/15	20/15	+	−	Full	No	Nystagmus
5	33	Pos. MS	20/20	20/20	−	+	Full	No	NML
6	44	Prob. MS	20/15	20/15	+	−	Full	No	INO-R
7	47	Pos. MS	20/20	20/25	−	+	Cent. scot	Yes	NML
II. Rule out MS									
8	39	Pos. MS	20/20	20/20	−	−	Full	No	INO-L&R
9	38	Pos. MS	20/20	20/15	−	−	Full	No	INO-R
10	24	Pos. MS	20/15	20/25	−	−	Full	No	NML
11	31	Pos. MS	20/15	20/15	−	−	Full	No	NML
III. Chiasmal defect									
12	33	Pit. adeno.	20/15	20/15	−	−	Bitemp.	No	NML
13	24	Pit. adeno.	20/20	20/25	−	−	Bitemp.	No	NML
14	37	Chiasmal O.N.	20/15	20/25	−	+	Bitemp.	No	NML

Pos. = possible; Prob. = probable; Def. = definite; O.N. = optic neuritis; APD = afferent pupillary defect; Cent. scot = Central scotoma; NML = normal; MS = multiple sclerosis; Pit adeno = pituitary adenoma; DX = diagnosis; INO = internuclear ophthalmoplegia; L = left; R = right; OC = ocular.

A group of four patients with possible or probable MS (McAlpine criteria) but no clinical history of optic neuritis was also tested. These patients were sent to our laboratory for clinical VEP evaluation and were chosen for inclusion in this study to demonstrate various aspects of SL testing. One of these patients had bilateral INO (patient 8), and another had a right INO (patient 9).

The third group of patients had low-grade bitemporal visual field defects. Patients 12 and 13 had pituitary adenoma with suprasellar extension. Patient 14 had optic neuritis with chiasmal involvement (anterior notch syndrome).

Following complete description of the procedures used in the study, informed consent was obtained from all subjects.

Eye Movement Recording

A microcomputer (model IIe, Apple Computer, Cupertino, CA) was used to generate saccade targets on a video monitor. The target, a white square subtending 30′ of visual angle with a luminance of 2.42 log cd/m^2 on a 1.63 log cd/m^2 grey background, was presented randomly 8° to the left or right of a fixation spot that was constantly present. Patients were instructed to look toward the peripheral target as quickly as possible and to return their gaze to center following target foveation. Anticipatory saccades were discouraged. The time interval between successive targets was varied randomly between 1.0 and 2.5 seconds.

Horizontal eye position was measured using infrared reflection spectacles (model 200, Applied Science Laboratories, Waltham, MA). The resolution of this technique varied from 10′–30′ of visual angle depending on distance of the sensors from the eye. For patients tested earlier in this study analog signals were digitized at a rate of 250 Hz on a signal averager (model 4800, Dagan Corp, Minneapolis, MN), and down-loaded to the microcomputer for later analysis. Because of the limited memory of the signal averager only 16 saccades were typically recorded from each eye to each target position. The equipment was subsequently modified to allow the microcomputer to simultaneously present targets and record data at 500 Hz using an interrupt driven system. This modification made it possible to obtain 40 saccades to each location for each eye.

Visual Evoked Potentials

Visual evoked potentials were recorded from scalp electrodes placed on the midline occiput (Oz) and 5 cm laterally over the left and right occipital areas (O1 and O2) referenced to the vertex (Cz). A forehead electrode (Fpz) was used as ground. EEG amplifiers (series 7, Grass Instruments, Quincy, MA) were used to amplify signals by 100K. Low- and high-frequency filters reduced signal amplitude to one-half at 1 and 300 Hz, respectively. A 60 Hz notch filter was used to minimize AC interference. Analog signals were digitized at a rate of 1.25 kHz (0.8 msec dwell time) for a sweep duration of 410 msec. Sixty-four sweeps were

Table 2. Normal latency values (msec)

	Pattern VEP		*Flash VEP*		*Saccadic latency*			
	OS	*OD*	*OS*	*OD*	*OS-L*	*OS-R*	*OD-L*	*OD-R*
$\bar{x}$	99	101	113	113	238	247	249	249
SD	7.3	7.5	12.3	12.0	27.8	32.9	27.7	35.9
$\bar{x}$ + 2SD	114	116	138	137	294	313	304	320

	Interocular Difference			
	PVEP	*FVEP*	*SL-L*	*SL-R*
$\bar{x}$	2.1	3.2	12.7	13.3
SD	2.7	4.5	8.8	8.1
$\bar{x}$ + 2SD	7.9	12.2	30.3	29.5

L = left; R = right.

averaged for each evoked potential. The pattern stimulus was a checkerboard generated on a video monitor by an Apple computer. Each check subtended 15′ of visual angle on a side. Contrast of the checkerboard was 85% and the mean luminance of the screen was 75 cd/m^2. The checkerboard pattern subtended 8° of visual angle at the viewing distance of 2 m. It was phase alternated twice per second. Flash stimulus was produced by a xenon arc photostimulator (model P-22, Grass Instruments) set at I = 4. Flashes were presented at a rate of 1 per second.

Both eye movements and VEPs were tested monocularly. Eye movement recordings were obtained from the stimulated eye only in monocular conditions, as the nonstimulated eye was occluded. If the second eye tested showed a relative latency delay, measurements were repeated on the first eye to ensure that this delay was not due to fatigue. Saccadic latencies were also measured with binocular stimulation in selected patients. During binocular testing, eye movements were recorded from both eyes simultaneously. Eye movement recording was generally completed within 45 min and duration of VEP testing was approximately 1 hr. In some cases, VEP and SL were tested on consecutive clinic visits.

Results

Normative Data

Normative VEP and SL data are presented in Table 2. VEP latencies are reported for the Oz-Cz recording. The criterion for abnormality was two standard deviations above the mean. For the pattern VEP, P-100 latencies above 114 msec OS, 116 msec OD and interocular latency delays of greater than 8 msec were abnormal. The upper limit of normal for flash VEP latency was 138 msec with an abnormal interocular latency difference of greater than 12.2 msec. For SL, an interocular difference of greater than 30 msec was considered abnormal. Limits of normal SL varied somewhat with position in the visual field with a tendency for latencies to be shorter in the left visual field. However, analysis of variance revealed that SL was not significantly affected by position in the visual field (left vs. right, $F(1,8) = 1.33$, $P > 0.05$), eye tested ($F(1,8) = 2.59$, $P > 0.05$), or nasal vs. temporal field (eye by position interaction, $F(1,8) = 2.52$, $P > 0.05$).

Unilateral Optic Neuritis

All patients in the optic neuritis group had subacute onset of unilateral decrease in visual acuity, central scotoma, and an afferent pupillary defect. In all cases, visual acuity had resolved to 20/25 or better at the time of testing.

Saccadic latency: Saccadic eye movement recordings from patient 7 are shown in Figure 1. Each tracing begins when the saccade target appeared in the peripheral field. SL is represented by the horizontal distance between the beginning of the tracing (time = 0) and the initiation of the saccade. Latency to initiate a saccade was longer when OS was stimulated than when OD was stimulated, especially for targets presented in the left visual field (leftward saccades). Velocity and accuracy of all saccades were within normal limits. The magnitude of the delay in saccadic initiation is evident in the frequency histograms presented in Figure 2. With stimulation of OS, mean SL of 451 msec and 332 msec for leftward and rightward saccades, respectively, were well beyond normal limits. Saccades were initiated at normal latency with stimulation of OD. The interocular latency difference of 165 msec, with stimulation of the left visual field, is significantly larger than normal. It is also substantially larger than the 60 msec delay in the pattern VEP OS relative to OD. Right visual field stimulation did not show a significant interocular SL delay (27 msec).

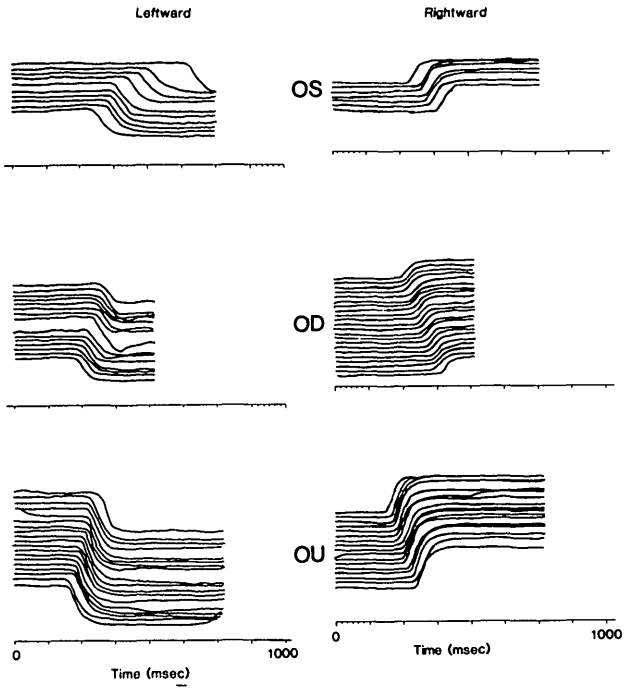

Fig. 1. Data from patient 7. Saccade records obtained with stimulation of OS, OD, and OU. Each tracing begins when the target appeared on the screen. Rightward movements are represented by upward deflections. Tracings have been ordered by latency and vertically displaced for illustrative purposes.

SL data from all optic neuritis patients are summarized in the upper portion of Table 3. Abnormal values are printed in italics. SL was abnormal for at least one target position in six of seven eyes with previous optic neuritis, but was normal in all clinically unaffected eyes. When interocular difference in latency is considered, the affected eye was significantly slower in seven out of seven patients for SL. For some patients the magnitude of interocular SL delay was quite different for right and left target locations (eg, 272 msec vs. 139 msec for patient 1). For patients 2 and 7 interocular delay was only abnormal for one of the target locations.

SL abnormalities for optic neuritis patients are presented graphically in Figure 3. An ellipse has been drawn to represent the boundaries of normality. The length of this ellipse was determined by ±2 SD from the mean latency for each eye, and its width was determined by ±2 SD from the mean difference between eyes. Mean rightward and leftward SL are connected by a line for each subject. Data from each optic neuritis patient fall either above or below the normal ellipse, indicating significant delay of the left or right eye, respectively.

If monocular delays in SL are representative of demyelination of the optic nerve, then one might expect that this delay would disappear with binocular stimulation. This hypothesis is based on the assumption that the normally conducting optic nerve can provide sufficient information on target location for saccade initiation. However, if the SL delay is a result of a lesion in the motor pathways, then one would expect that the monocular delay would persist with binocular stimulation.

Results from patient 7 are shown in the lower panels of Figures 1 and 2. With binocular stimulation, saccades were conjugate and the mean latency of 248 msec recorded from OS was 50 msec shorter than that obtained with monocular stimulation of the unaffected right eye. A similar pattern of SL results was found in patient 10. Despite normal VEPs, no history of optic neuritis and a normal visual examination, this patient had a significant SL delay OS to targets in both hemifields. With binocular viewing, the mean SL of 222 msec was 12% shorter than that obtained with the faster eye alone.

Visual evoked potential: For all optic neuritis patients, stimulation of the affected eye resulted in a pattern VEP P100 component that was delayed in latency with respect to normal, and with respect to

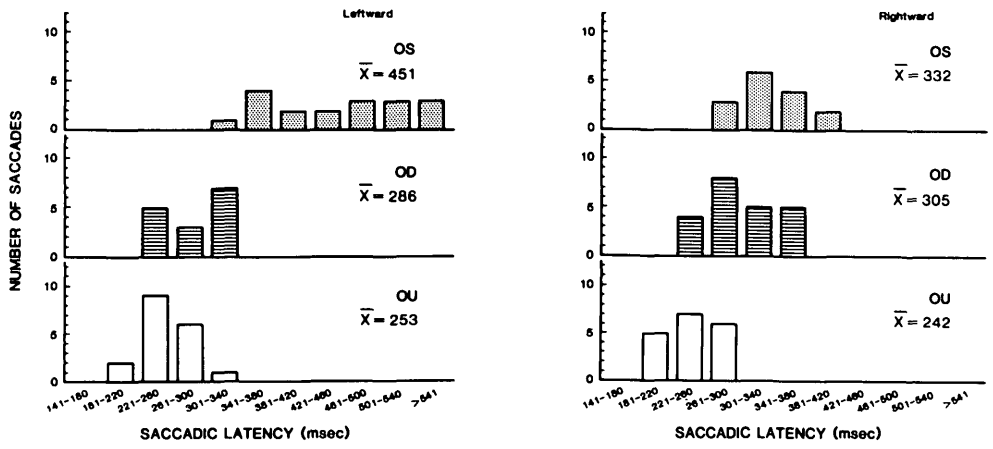

Fig. 2. Frequency histograms of saccadic latency for data shown in Figure 1. Numbers represent mean latency for each condition.

Table 3. Saccadic and VEP latency (in msec) from unilateral optic neuritis and rule out MS groups (values in italics are abnormal)

	PVEP		*FVEP*		*Saccade latency*				*Interocular diff.*			
Pt. #	*OS*	*OD*	*OS*	*OD*	*OS-L*	*OS-R*	*OD-L*	*OD-R*	*PVEP*	*FVEP*	*SL-L*	*SL-R*
I. Optic neuritis												
1	*131**	99	82	70	*533*	*432*	261	293	*32*	12	*272*	*139*
2	*125**	100	120	115	293	305	262	304	*25*	5	*31*	1
3	*133**	99	114	90	*332*	294	284	238	*34*	*24*	*48*	*56*
4	103	*136**	115	129	278	281	*331*	*391*	*−33*	*−14*	*−53*	*−110*
5	*130**	105	131	123	*307*	277	188	196	*25*	8	*119*	*81*
6	106	*130**	118	124	232	230	*373*	318	*−24*	−8	*−141*	*−88*
7	*185**	114	*151*	127	*451*	*332*	286	305	*61*	*24*	*165*	27
				$\bar{x}$ =	346	307	284	292	33†	14	120	72
II. Rule out MS												
8	108	115	104	106	*415*	*421*	*307*	*335*	−7	−2	*107*	*86*
9	108	109	121	121	276	283	301	*342*	−1	0	−25	*−59*
10	90	89	85	87	290	298	248	253	1	−2	*42*	*45*
11	86 *149*	88 *153*	110	11	220	227	232	218	−2/−4	0	−12	9
				$\bar{x}$ =	326	307	272	287	3	1	46	50

* Eye with clinical history of optic neuritis.
† Means calculated on absolute values.

L = left; R = right; SL = saccade latency; PVEP = pattern VEP; FVEP = flash VEP; DIFF = difference.

the unaffected eye (interocular latency difference). Interocular delay was significantly larger when measured by SL than when measured by pattern VEP (t (6) = 2.68, $P < 0.05$), and no systematic relation between size of the VEP latency delay and size of the saccadic delay was observed ($r = 0.06$, t (6) < 1). The flash VEP was abnormal in latency in only one affected eye, and interocular delay was abnormally large in three of seven cases.

To determine whether the disparity in magnitude

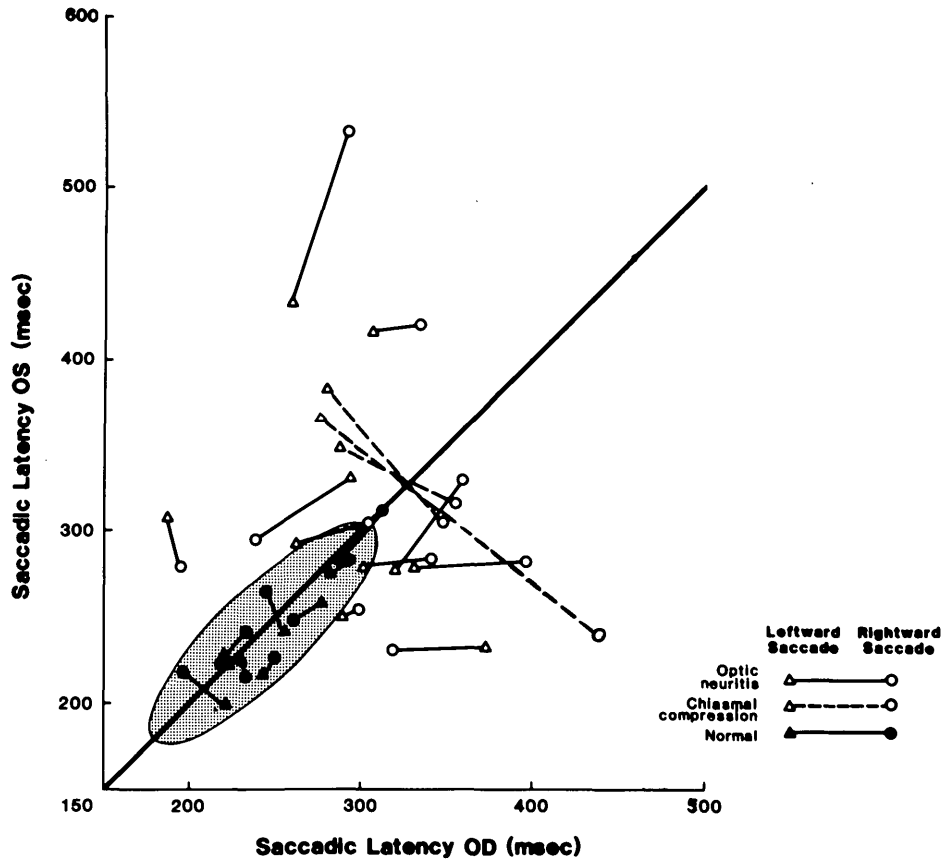

Fig. 3. Saccadic latency OD as a function of saccadic latency OS for normals (filled symbols), patients with optic neuritis (unfilled symbols—solid lines), and patients with chiasmal compression (unfilled symbols—dashed lines). Triangles represent means from leftward saccades and circles represent means from rightward saccades. Stippled area represents normal limits.

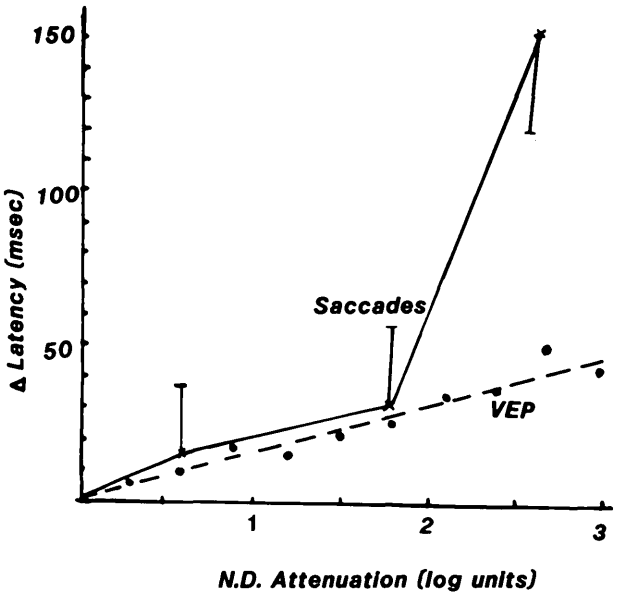

Fig. 4. The effect of attenuating mean target luminance (using neutral density filters) on saccade latency (solid line) and VEP latency (dashed line) for one normal observer. Each point on the VEP line represents the mean of two measures. Each point on the saccade line represents the mean of eight to ten measures. Vertical lines at each saccadic latency point represent one standard error of the mean.

between SL and VEP delays might be attributable to an afferent factor, we artificially induced a delay in the afferent system of a normal observer by placing a neutral density (ND) filter in front of the eye. Results are shown in Figure 4 where the dashed line connects the points representing induced delay in VEP latency as a function of ND and the solid line connects points representing induced SL delay as a function of ND. Comparable effects of ND were obtained on the two measures for ND values of 1.75 log units or less. When luminance was reduced by 2.5 log units, SL was increased to a much greater extent than was VEP latency. The saccade target was well above threshold at this luminance level, but detection was probably mediated by the scotopic system.[6] Thus, saccadic and VEP latency delays can be dissociated by a manipulation of target luminance which affects afferent conduction time but does not directly influence the ocular motor pathways.

Suspected MS Cases

The lower section of Table 3 contains SL data from four patients (8–11) suspected of having MS without a clinical history of optic neuritis and with normal or questionable VEPs. Patient 8 is a 39-year-old woman with a 5-year history of intermittent ataxia and right facial weakness. At the time of testing, her eye movements were characterized by bilateral INO. Absolute pattern and flash VEP latencies, as well as interocular latency differences were within normal limits. Saccade testing revealed abnormal latencies with stimulation of either eye and a significant delay of OS relative to OD for both target locations. Despite the presence of decreased velocity of the adducting eye, there was no evidence of selective delay in the initiation of adducting saccades. A similar dissociation of SL and velocity was found in patient 9, who had a right INO, and yet adducting SL was shorter than abducting SL OD.

Patient 11, a 31-year-old woman, was suspected to have MS due to a recent history of bilateral ataxia of the lower extremities and decreased bowel and bladder control. She also complained of intermittent retrobulbar pain OS over a 5-month period with onset 7 months prior to testing, and was sent for VEP testing to rule out optic nerve demyelination. Pattern VEP results were ambiguous with stimulation of either eye, in that two positive peaks were observed, one within normal latency limits and one abnormally delayed. Flash VEPs were normal. Saccadic latencies were normal with stimulation of either eye. It was concluded that afferent visual conduction was normal based on normal SL and the presence of a positive peak within normal latency limits in the VEP. A subsequent myelogram revealed syringomyelia which accounted for her clinical symptoms.

Chiasmal Dysfunction

Patients 12 and 13 each had a prolactin-secreting pituitary adenoma which compressed the optic chiasm, resulting in low-grade (I-1e isopter) bitemporal visual field defects. Full field pattern VEPs were not sensitive to these low-grade field defects in that they were symmetric between left and right occipital electrodes. Stimulation of the nasal and temporal fields separately produced subtle, if any, amplitude or latency asymmetries, with the exception of a substantial conduction delay observed with stimulation of the right visual field of the right eye of patient 13.

Patient 14 had definite MS and presented with a chiasmal optic neuritis. One month prior to testing she noted blurred vision in her left eye. Visual acuity had decreased to 20/60 with a patchy central scotoma. At the time of testing, visual acuity had resolved to 20/25 OS with no afferent pupillary defect. Her visual fields showed an upper temporal field defect OD and a temporal hemianopia OS detectable only by the I-1e target. Pattern VEPs showed delayed latency OS as well as a significant interocular delay of 44 msec.

SL data for patients 12, 13 and 14 are represented in Figure 3 as open symbols connected by dashed

lines. There was a marked delay in saccades to targets presented in the temporal field for all six eyes. All latencies for targets presented in the nasal fields were within normal limits, whereas the shortest mean latency for temporal targets of 349 msec was greater than two standard deviations above the normal mean. SL was longer to targets presented in the temporal field than to nasal targets by 63, 155 and 50 msec for patients 12, 13 and 14, respectively. In Figure 3 this pattern of delays, limited to the temporal field of each eye, results in lines connecting leftward and rightward saccades that are roughly orthogonal to the 45° ocular symmetry line.

Discussion

The results of this study indicate that the latency of saccades to a visual target can be used as a measure of afferent visual function. Delays in saccadic initiation were found only to targets presented to the affected eye in patients with resolved unilateral optic neuritis, and only to targets presented to the affected visual field of patients with chiasmal compression and subtle bitemporal field defects. With binocular target viewing, saccadic latencies reverted to normal, a result that may be parsimoniously explained if the monocular delay is attributed to an afferent source. The afferent signal from the intact optic nerve should be sufficient to initiate a saccade at a normal latency with binocular stimulation. Our results suggest that SL is actually shorter with binocular stimulation than with stimulation of either eye alone. This binocular facilitation of SL is considered in more detail by Brigell and Peachey.[7]

Previous studies[2,4] have discounted a purely afferent etiology to SL delays in MS patients based upon longer delays in SL than in the VEP and the absence of significant correlation of SL with VEP latency, results that are replicated in the current study. Reulen et al[4] attribute the SL delays that they observed in MS patients to supranuclear lesions in the cerebellum or basal ganglia. Although it has been shown that lesions in the supranuclear ocular motor pathways can effect latency of all saccades,[8-10] it has not been shown that lesions in these areas can affect SL monocularly. Thus, the absence of SL delays in unilateral optic neuritis patients while viewing targets either with the unaffected eye monocularly, or under binocular viewing conditions, is not compatible with a supranuclear lesion in the ocular motor pathway. A lesion in the motor pathways could not delay adducting and abducting saccades monocularly, without other signs of third and sixth nerve involvement. With the exception of those patients with INO, saccadic waveform, velocity and accuracy were normal in all patients, suggesting the absence of ocular motor nerve dysfunction. Two patients with INO did not show a correlation between saccadic velocity and latency, suggesting that the lesion in the motor pathway responsible for decreased saccadic velocity was not responsible for increased SL.

The lack of correlation between VEP and SL measures of conduction delays and the greater magnitude of the SL interocular delay do not exclude a purely afferent etiology of abnormalities in both measures because a different contingent of afferent neurons are probably involved in the two tasks. The pattern VEP is generated primarily by the central 5° of the visual field,[11,12] whereas targets for saccades are presented to more peripheral areas of the visual field. Thus, since demyelination of the optic nerve is patchy in optic neuritis,[13,14] disparate results between tests in an individual patient could be obtained without indicating involvement of the ocular motor pathways. Similar factors probably underlie the low correlation between VEP latency and other measures of afferent visual function, including contrast sensitivity threshold[15,16] and pupil cycle time,[17] as well as the dissociation between SL and VEP latency induced by manipulating stimulus luminance in the current study.

The sensitivity of VEP and SL to lesions of the optic nerve in patients with normal Snellen acuity and visual fields can be compared in results of the current study. Both pattern VEP and SL were quite sensitive to optic nerve demyelination in that all optic neuritis patients showed significant interocular latency delays on both tests. The flash VEP was less sensitive to the effects of optic neuritis, a result consistent with previous reports.[18,19] The pattern VEP was a more sensitive measure of absolute latency delay than was SL. Although the pattern VEP is sensitive to lesions involving the central visual field, it has proven unsatisfactory in the diagnosis of chiasmal and retrochiasmal lesions in the visual pathway.[20-22] Data from the present study confirm the insensitivity of the VEP to low-grade peripheral field defects, and show a distinct advantage of SL measurement in detection of these defects. As these field defects were subtle, it is suggested that SL measurement may be of value in the detection of subclinical visual field abnormalities and in monitoring the progression of these abnormalities.

The SL test offers an advantage over the VEP in that, like perimetry, delay campimetry,[23] and multiflash campimetry,[24] it allows for testing of individual points in the visual field. Thus, if instead of only testing two points in the visual field, latency from a central fixation point to various points in the visual field are measured, a delay field showing areas of impaired optic nerve conduction could be plotted.

Delay fields have previously been plotted using delay campimetry and multiflash campimetry.[23,24] One might speculate that latency delay would be a precursor to sensitivity loss in compressive optic nerve lesions on the basis of histologic evidence of local demyelination preceding axonal damage in experimentally compressed optic nerve.[25] Our results suggest that SL is a valid measure of afferent conduction. If the measure proves to have high test-retest reliability, SL testing may prove to be an effective method of monitoring afferent visual dysfunction.

Key words: saccadic eye movement, visual evoked potential, optic neuritis, chiasmal compression, optic nerve conduction delay

Acknowledgments

The authors are grateful to Drs. Tulay Kansu, Neal Peachey, and V. Leo Towle for their helpful comments on earlier versions of this manuscript.

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