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Contrast Sensitivity, First-Order Motion and Initial Ocular Following in Demyelinating Optic Neuropathy

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Abstract

The ocular following response (OFR) is a measure of motion vision elicited at ultra-short latencies by sudden movement of a large visual stimulus. We compared the OFR to vertical sinusoidal gratings (spatial frequency 0.153 cycles/° or 0.458 cycles/°) of each eye in a subject with evidence of left optic nerve demyelination due to multiple sclerosis (MS). The subject showed substantial differences in vision measured with stationary low-contrast Sloan letters (20/63 OD and 20/200 OS at 2.5% contrast) and the Lanthony Desaturated 15-hue color test (Color Confusion Index 1.11 OD and 2.14 OS). Compared with controls, all of the subject's OFR to increasing contrast showed a higher threshold. The OFR of each of the subject's eyes were similar for the 0.153 cycles/° stimulus, and psychophysical measurements of his ability to detect these moving gratings were also similar for each eye. However, with the 0.458 cycles/° stimulus, the subject's OFR was asymmetric and the affected eye showed decreased responses (smaller slope constant as estimated by the Naka-Rushton equation). These results suggest that, in this case, optic neuritis caused a selective deficit that affected parvocellular pathways mediating higher spatial frequencies, lower-contrast, and color vision, but spared the field-holding mechanism underlying the OFR to lower spatial frequencies. The OFR may provide a useful method to study motion vision in individuals with disorders affecting anterior visual pathways.

Keywords

optic neuritis; multiple sclerosis; saccades; pursuit

Introduction

Optic neuritis occurs in up to two-thirds of individuals suffering from multiple sclerosis (MS) at some point in their disease course [15,30]. It is characterized by painful, usually monocular, visual loss with decreased visual acuity, color vision, contrast sensitivity, and defects of the visual field. The latency to onset of visual evoked potentials in such individuals is increased in the affected eye by values ranging from 5 to 60 ms, reflecting the extent of demyelination [4].

Regardless of the severity of initial visual impairment, 95% of individuals have improvement of Snellen visual acuity to 20/40 or better at one year after the onset of symptoms [6].

Nonetheless, subjective visual complaints are common after optic neuritis, and most individuals have persistent visual deficits as judged by contrast sensitivity or persistent delays of visual evoked potentials[5,7,13,14,28]. At present, the most sensitive clinical method for detecting persistent visual impairment in individuals who have suffered optic neuritis appears to be Low Contrast Sloan charts [5]. Consistent with clinical findings, MRI studies indicate that persistent axonal damage may follow a single episode of optic neuritis [18,19], a view supported by measurements of the retinal nerve fiber layer using optical coherence tomography [11].

Although it is known that axonal damage persists after recovery from acute optic neuritis, the differential effect on axons subserving high spatial resolution via parvocellular visual pathways versus axons subserving high temporal resolution via magnocellular visual pathways is not settled. Several lines of evidence support the idea that the brain treats visual motion information separately from visual position information [10,22]. Visual motion can be measured using psychophysical tests, but a convenient and robust measure is the ocular following response (OFR), which can be elicited at ultra-short latencies by sudden motion of a large textured pattern. The OFR shows properties of a “first order” luminance-driven system, acting in a machine-like manner that is not sensitive to the subject's attentional state or the contrast of the stimulus. [8,16,26,32] Thus, the OFR offers advantages over psychophysical methods, which are more dependent on attentional and cognitive factors. We tested the hypothesis that the OFR would reveal properties of motion vision distinct from defects of spatial vision in individuals with optic neuritis, and present here preliminary results from one subject.

Subjects and Methods

Some of the techniques, such as those used for recording eye movements and for data analysis, were similar to those used previously [23,25,32,36], and, therefore, will only be described in brief here. Experimental protocols were approved by the Institutional Review Committee of the National Institutes of Health concerned with the use of human subjects.

Subjects

The subject studied was a 42-year-old African American man with a history of relapsing-remitting MS and left optic neuritis. His initial symptom was an episode of diplopia in 1992, followed shortly by an episode of facial numbness, at which time a brain MRI revealed multiple periventricular T2 hyperintensities. In 2002, he experienced visual loss in the left eye with pain on eye movement. The nadir of his visual acuity was his ability to count fingers at six feet. At that time, he had a large central scotoma, a left afferent pupillary defect to light stimuli, and a normal-appearing optic nerve. Retrobulbar optic neuritis was diagnosed and he was treated with intravenous methylprednisolone. Subsequently, visual acuity recovered slowly and optic nerve pallor developed. Despite good recovery of high contrast visual acuity, he continued to experience subjective difficulty with vision in the left eye. He denied ever having visual symptoms in his right eye.

Current neurologic examination revealed minimal disability, with an Expanded Disability Status Scale [21] score of 3.5. He was able to walk normally, but had the following deficits: decreased sensation in the left foot, slightly decreased left visual acuity (detailed below), and very mild cognitive dysfunction. His MS was treated with immunomodulatory therapy, starting with interferon beta-1A from 1998 until 2001, followed by glatiramer acetate until the present. Brain MRI in 2005 showed signals indicating a moderate number of demyelinating plaques in the subcortical white matter and mild cerebral atrophy.

Neuro-ophthalmologic examination revealed best corrected monocular high contrast visual acuity of 20/20 OD and 20/40 OS (Early Treatment Diabetic Retinopathy Study acuity chart).

Monocular low contrast visual acuity was measured with Low-Contrast Sloan Letter charts (Sloan charts; Precision Vision; LaSalle, IL). At 10% contrast, acuity was 20/32 OD and 20/200 OS. At 2.5% contrast, acuity was 20/63 OD and 20/200 OS. At 1.25% contrast, acuity was 20/125 OD but no letters could be seen with his left eye. Color vision was tested in two ways. With Ishihara color plates, his responses were normal OD, but only 1 of 14 color plates were correctly identified OS. With the Lanthony Desaturate 15-hue color test, the Color Confusion Index was 1.11 OD and 2.14 OS; this indicated mild, insignificant errors OD and moderate dysfunction of color vision OS. Additional examination findings included a left afferent pupillary defect, left optic nerve pallor, and a small temporal visual field defect OS (Humphrey Automated Sita-Standard 24-2 Perimetry). The right optic nerve and visual field were normal. Visual evoked potentials to pattern reversal stimulation showed slightly increased latency OD (P100 102-112 ms) and a minimally identifiable response OS (P100 130-168 ms), indicating very subtle demyelination of the right optic nerve and severe demyelination of the left optic nerve.

During a preliminary study of his eye movements (binocular magnetic search-coil technique) in April 2004, there were two findings of note. The first was frequent, small, to-and-fro horizontal, conjugate, saccadic intrusions (square-wave jerks, SWJ) [1]. The frequency of SWJ was 2.15/s with his right eye viewing and 1.85/s with his left eye viewing. There was no difference in the size of the SWJ while viewing with the right or left eyes (median 0.35 degree for both conditions). The second finding was that during horizontal smooth pursuit of a red laser spot moving sinusoidally at 0.33 Hz, gain (eye velocity/target velocity) was 0.87 with his right eye viewing and 0.77 with his left eye viewing. He commented that although he could clearly see the laser spot with his left eye, it appeared white, not red. Saccades showed normal dynamic properties and there was no evidence of internuclear ophthalmoplegia. Convergence eye movements were normal.

Results from the patient were compared with three healthy control subjects (ages 42, 64, and 64 years old) who have been previously studied using the same methods [32].

Visual Stimuli

The subject sat in a dark room with his head positioned by means of adjustable rests for the forehead and chin, and held in place with a headband. Visual stimuli were presented on a computer monitor located straight ahead at 45.7 cm from the corneal vertex; details of instrumentation and setup have been described previously [32]. The visual images consisted of one-dimensional vertical grating patterns that could have one of two horizontal luminance profiles which were run in separate sessions: 1) session 1: a sine-wave with a spatial frequency of 0.153 cycles/° (wavelength, 6.55°, which was 264 pixels), termed “the *1f* stimulus”, 2) session 2: a sine-wave with a spatial frequency of 0.458 cycles/° (wavelength, 2.183°, which was 88 pixels), termed “the *3f* stimulus”. A previous study [32] indicated that pure sine-wave stimuli with the spatial frequencies of these *1f* and *3f* stimuli elicit robust OFRs of similar amplitude in normal subjects. Each image extended 257 mm horizontally (31.4°; 1280 pixels) and 206 mm vertically (25.4°; 1024 pixels) and had a mean luminance of 42.6 cd/m². The initial phase of a given grating was randomized from trial to trial at intervals of ¼-wavelength.

Eye-Movement Recording

Viewing was always monocular with the horizontal and vertical positions of the viewing eye, left or right, being recorded with an electromagnetic induction technique using a scleral search coil embedded in a silastic annulus [9,35]. At the beginning of each trial, a grating pattern appeared (randomly selected from a lookup table) together with a central target spot (diameter, 0.25°) that the subject was instructed to fixate. After the subject's recorded eye had been positioned within 2° of the fixation target for a randomized period of 900 to 1200 ms the fixation

target disappeared and the apparent-motion stimulus began. The motion lasted for 200 ms, at which point the screen became a uniform gray (luminance, 42.6 cd/m²) marking the end of the trial. After an inter-trial interval of 500 ms a new grating pattern appeared together with a fixation point, commencing a new trial. The subject was asked to refrain from blinking or making any saccades except during the inter-trial intervals but was given no instructions relating to the motion stimuli. If no saccades were detected during the period of the trial (using an eye velocity threshold of 12°/s), then the data were stored on a hard disk; otherwise, the trial was aborted and subsequently repeated.

At the beginning of the first session (*1f* stimulus) a coil was placed over subject's right ("good") eye, his left ("bad") eye was covered, and data were collected for 30 minutes. The coil was then removed and subject had a ~10 minute rest. After that period the coil was placed over subject's left ("bad") eye, his right eye was covered, and another 30 seconds of data collection followed. The next day, the second session (*3f* stimulus) was conducted, in which the order of the eye being recorded first was reversed: right followed the left. In each session, for each eye, each condition was repeated 23-58 times. In each session there were 14 different stimulus conditions (7 contrasts of grating pattern, 2 directions of motion).

A separate pure psychophysics session was also conducted, in which the subject was presented with the same *1f* stimulus set of motion stimuli described in detail earlier. Eye movements were not recorded in this session; instead the subject was required to report the perceived direction of motion of the grating by a button press (two-alternative forced choice). Twenty blocks of trials were run with the subject observing the display monocularly: 10 with the right eye covered, followed by 10 with the left eye covered.

Data Analysis

The horizontal and vertical eye position data obtained during the calibration procedure were each linearized and filtered as previously described [32]. Trials with saccadic intrusions (that had failed to reach the eye-velocity threshold of 12°/s used during the experiment) were deleted. The OFRs to rightward and leftward were pooled to improve the signal-to-noise by subtracting the mean response to each leftward motion stimulus from the mean response to the corresponding rightward motion stimulus: the "R-L position responses". The onset latency of responses was derived from the velocity data (termed "mean R-L velocity response profiles"), which were estimated at successive 1-ms intervals by computing the two-point (20 ms apart) central difference between the symmetric weight moving averages (20 points) of the position signal. The onset latency was considered to commence when the amplitude of the velocity response exceeded 3 times the standard deviation (SD) of the noise during the pre-response period (based on the 40-ms period starting 20 ms after the onset of the motion stimulus). The initial horizontal OFR were quantified by measuring the changes in R-L position responses over the 100-ms time periods commencing 60 ms after the onset of the motion stimuli: "the R-L response measures". The minimum latency of onset was ~80 ms (see Fig. 1) so that these response measures were restricted to the period prior to the closure of the visual feedback loop (i.e., twice the reaction time): initial open-loop responses.

Results

Fig. 1 shows sample mean R-L velocity response profiles over time elicited by successive 1/4-wavelength shifts applied to *1f* and *3f* stimuli of different contrast. Top row graphs depict responses recorded during the right-eye-viewing sessions (REV; Fig. 1A, B), while middle row graphs – responses from the left-eye-viewing sessions (LEV; Fig. 1C, D); left column graphs depict responses to *1f* stimulus (Fig. 1A, C), right column graphs – responses to *3f* stimulus (Fig. 1B, D). The initial OFR in panels A-D of Fig. 1 are characterized by several common features: 1) virtually no response at 2% contrast (threshold contrast), 2) monotonic rise of the

peak velocity with increasing contrast (contrast of the pattern is shown by encircled numbers superimposed on traces) with 3) notable saturation of the response at highest contrasts, and 4) short latencies (~80-100 ms).

The bottom plots in Fig. 1 depict onset latencies of the OFRs obtained with the *1f* (Fig. 1E) and *3f* stimuli (Fig. 1F) sessions. With both *1f* and *3f* stimuli the latencies in LEV and REV sessions were statistically indistinguishable ($p > 0.05$), although at least with *1f* stimulus responses latencies in the LEV session do appear to be slightly longer (2% contrast data were not included into this statistical analysis, because there was virtually no response at this contrast: e.g. Fig. 1C, D).

The quantitative dependence on contrast, based on the mean R-L response measures, is presented in panels A (*1f* stimulus) and B (*3f* stimulus) of Fig. 2, with LEV and REV responses being shown by closed and open circles, respectively. These data were fitted with the following expression:

$$R_{\max} \frac{c^n}{c^n + c_{50}^n}; \quad (1)$$

where $R_{\max}$ is the maximum attainable response, c is the contrast, c_{50} is the semi-saturation contrast (at which the response has half its maximum value), and n is the exponent that sets the steepness of the curves. This expression is based on the Naka-Rushton equation [27], and various studies have shown that it provides a good fit to the contrast dependence curves of neurons in the LGN, V1 and MT areas of monkeys [2,3,17,31], as well as to the human contrast dependence curves for the OFR to moving sine-wave gratings and unikinetic plaid patterns [24,32]. The continuous smooth curves in Fig. 2 are the best fit curves using expression (1) and are excellent approximations to the data with r^2 values of 0.97 or greater in all cases. The parameters, c_{50} and n , for these various fits are printed beside the curves in Fig. 2. For both stimuli (though much more prominent with the higher spatial frequency stimulus, i.e. the *3f* stimulus) the best-fit curves for LEV data are always less steep and reach 50% maximum at a higher contrast than those for the REV data (*1f* stimulus: n - 2.08 vs. 2.54, c_{50} - 9.2% vs. 7.2%; *3f* stimulus: n - 1.24 vs. 3.44, c_{50} - 15.4% vs. 8.3%).

In Fig. 3 we compare the subject's OFR dependence on contrast with that of three normal subjects that we have previously reported using the same stimuli [32]. Several observations can be made. First, the subject's OFR generally show higher thresholds than controls. This is reflected in higher c_{50} values for both the REV and LEV data for our patient than for controls (*1f* stimulus: mean 3.9%, range 3-5%; *3f* stimulus: mean 5.7%, range 4.4-7.8%). Second, when the higher threshold is taken into account, the OFR curves of both eyes for the *1f* stimulus show similarities to those of the controls. Third, with the *3f* stimulus the data obtained with the subject's left eye showed a more gradual rise than controls (smaller slope constant, n , as estimated by Naka-Rushton equation) whereas the data obtained with the right eye showed a less gradual rise than controls (larger slope constant, n).

Perceptual judgments of motion direction with the *1f* stimulus were close to 100% for all but the lowest contrasts with either eye viewing the stimulus, reinforcing the notion that this subject has slightly elevated contrast thresholds for both the OFR as well as motion perception: see Fig. 4.

Discussion

We have applied a test of motion vision, the ocular following response (OFR), in an individual with MS who had previously suffered an attack of acute left optic neuritis and made a partial

recovery. His persisting defects in the involved eye affected vision of low contrast Sloan letters and color. These defects could be attributed to residual optic neuropathy, evident electrophysiologically by increased latency and decreased amplitude of visual evoked responses. Compared with controls, for all stimuli, the subject required an increased level of contrast for him to develop OFR similar to normals. However, when this threshold effect was factored out, two findings emerged. First, for the lower spatial frequency used (0.153 cycles/°) the curve describing OFR in response to increasing contrast was similar in both of his eyes and also showed similarities to controls. Consistent with this, his subjective ability to detect these moving gratings was similar in each eye. The story was different for OFR in response to 0.458 cycles/° moving gratings: for his affected eye, the curve describing OFR in response to increasing contrast had a smaller slope (as estimated by n of the Naka-Rushton equation) than the better eye, which was more similar to the curve for controls (Fig. 3). These findings raise two questions: First, what do these findings tell us about the disturbance of vision in our subject? Second, could the OFR be useful for testing visual system disorders?

The color and low-contrast sensitivity visual defects in our patient's left eye indicated predominant involvement of the parvocellular visual pathway and foveal (macular) vision. Thus, the impaired OFR of this eye to the stimulus with higher spatial frequency (Fig. 3) might also have been caused by disease affecting parvocellular pathways. Conversely, for stimuli with lower spatial frequencies, his OFR and perception of motion were similar in each eye, and the latency of the OFR was only slightly increased (Fig. 1E and F), suggesting preservation of the magnocellular visual function. This interpretation – that the affected eye of our patient showed a deficit of parvocellular pathway function, with relative sparing of magnocellular pathway function – is in agreement with some prior studies [12,29,34].

One possible implication of these findings is that identification of abnormalities of ocular following eye movements are more likely to be manifest during tracking of higher spatial frequencies. Thus, his ability to smoothly pursue a moving laser spot was more impaired with his affected eye. A second possible implication is that visual fixation following saccades – a function that probably depends on the OFR [20] – should be normal in our subject. In fact, he did show frequent small, saccadic intrusions (square-wave jerks), but there were no slow drifts of his eyes (*i.e.*, no nystagmus). It is difficult to know if these saccadic intrusions were due to MS, optic neuropathy, or represent a normal phenomenon, since they were slightly more common when he viewed with his better eye, and they are reported to occur with a frequency of up to 60/minute in some normal subjects [1].

Finally, the OFR provides a quantitative method for evaluating defects of motion vision that is less dependent on subject attention than psychophysical methods. In this study, we have shown how the OFR may be applied to investigate disturbance of motion vision in a disorder affecting the optic nerve. Our results are preliminary and should be interpreted with caution, but we suggest that the OFR could be usefully employed to study a range of disorders of the visual system and would supplement electrophysiological and psychophysical approaches [33].

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Figure 1.

Sample mean R-L velocity OFR profiles over time elicited in the subject by successive $\frac{1}{4}$ -wavelength shifts applied to $1f$ stimuli (left column) and $3f$ stimuli (right column) of different contrast. A and B: right-eye-viewing sessions (REV). C and D: left-eye-viewing sessions (LEV). E and F: onset latencies of the OFR to stimuli of different contrast. See text for details.

Figure 2.

Quantitative dependence of the subject's OFR on contrast, based on mean R-L response measures. A: 1f stimulus; B: 3f stimulus. Data were fitted with the Naka-Rushton equation [27], and the parameters, c_{50} and n , for these various fits are printed beside the curves.

Figure 3.

The initial OFR: dependence on contrast (normalized R-L response measures); comparison of the subject with controls. Plots show the horizontal OFR to pure sine-wave gratings of $1f$ (open symbols) or $3f$ (filled symbols) spatial frequency. For clarity responses to the pure $1f$ and $3f$ sine waves are shown on opposite sides from zero. Circles and smooth grey lines – 3-subject means and best-fit Naka-Rushton functions, respectively, for controls (3 subjects; error bars are SD's of the means; c_{50} and n parameters for Naka-Rushton functions are also shown); squares and smooth continuous lines – patient's right eye data and best fitting curves; diamonds and smooth dashed lines – patient's left eye data and best fitting curves.

Figure 4.

The subject's perceptual judgments of motion direction with the *If* stimulus. There were close to 100% for all but the lowest contrasts with either eye viewing the stimulus. See text for details.