Article

Diagnosis of Normal and Abnormal Color Vision with Cone-Specific VEPs

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Purpose: Normal color vision depends on normal long wavelength (L), middle wavelength (M), and short wavelength sensitive (S) cones. Hereditary "red-green" color vision deficiency (CVD) is due to a shift in peak sensitivity or lack of L or M cones. Hereditary S cone CVD is rare but can be acquired as an early sign of disease. Current tests detect CVD but few diagnose type or severity, critical for linking performance to real-world demands. The anomaloscope and newer subjective tests quantify CVD but are not applicable to infants or cognitively impaired patients. Our purpose was to develop an objective test of CVD with sensitivity and specificity comparable to current tests.

Methods: A calibrated visual-evoked potential (VEP) display and Food and Drug Administration-approved system was used to record L, M, and S cone-specific pattern-onset VEPs from 18 color vision normals (CVNs) and 13 hereditary CVDs. VEP amplitudes and latencies were compared between groups to establish VEP sensitivity and specificity.

Results: Cone VEPs show 100% sensitivity for diagnosis of CVD and 94% specificity for confirming CVN. L cone (protan) CVDs showed a significant increase in L cone latency (53.1 msec, $P < 0.003$) and decreased amplitude (10.8 uV, $P < 0.0000005$) but normal M and S cone VEPs ($P > 0.31$). M cone (deutan) CVDs showed a significant increase in M cone latency (31.0 msec, $P < 0.000004$) and decreased amplitude (8.4 uV, $P < 0.006$) but normal L and S cone VEPs ($P > 0.29$).

Conclusions: Cone-specific VEPs offer a rapid, objective test to diagnose hereditary CVD and show potential for detecting acquired CVD in various diseases.

Translational Relevance: This paper describes the efficacy of cone-specific color VEPs for quantification of normal and abnormal color vision. The rapid, objective nature of this approach makes it suitable for detecting color sensitivity loss in infants and the cognitively impaired.

translational vision science & technology

Introduction

The ability to discriminate the myriad hues that surround us transcends species, confers survival value, and is essential for veridical object detection, discrimination, and recognition.[1,2] Normal color vision depends on a normal complement of long wavelength (L), middle wavelength (M), and short wavelength sensitive (S) cone photoreceptors. Hereditary red-green color vision deficiency (CVD; 8% of Caucasian males and 1 in 200 females) is an X-linked condition resulting in a shift in peak L or M cone absorption (protanomaly and deuteranomaly, respectively) or lack of either L (protanopia) or M (deuteranopia) cones.[3,4] The importance of normal color vision has

been identified for various occupations requiring accurate hue discrimination in cue-limited settings, including transportation (aviation, railway, and driving), military settings, as well as law enforcement.[5–8] Hence hereditary CVD is disqualifying for numerous occupations. Equally important, CVD can be acquired as an early clinical or subclinical sign of ocular, systemic, and neurologic disease offering potential for early diagnosis, monitoring, and treatment.[9–14] While numerous book tests of color vision are invaluable for detection of hereditary CVD, few offer qualitative (protan, deutan, or tritan) or quantitative (severity) metrics to correlate color ability with real-world demands. For hereditary red-green CVD, the anomaloscope remains a benchmark test, though newer

computer-based tests offer comparable sensitivity, specificity, and require less skill to administer and interpret.[15–20] Nevertheless, all test methods described thus far require subjective responses, which precludes testing of infants, nonverbal patients, as well as cognitively impaired patients with stroke, traumatic, or senescent brain disease. Hence our purpose in this initial proof-of-principle study was to develop and validate a rapid, objective, clinically expedient visual-evoked potential (VEP) measure of cone-specific color vision to diagnose hereditary and acquired CVD.

Methods

A fully calibrated Diagnosys, LLC visual electro-diagnostic system (Lowell, MA) was used to record L, M, and S cone-specific VEPs in pattern-onset mode with colored checkerboards exchanged for a gray background. This approach is comparable to previous studies of chromatic VEPs showing pattern onset to be more effective than pattern reversal for isolating chromatic mechanisms.[21–25] The checkerboard appeared two times per second, with each presentation lasting 100 msec followed by the gray field lasting 400 msec. The VEP was recorded for 300 msec at the onset of each checkerboard presentation. Each VEP signal was amplified 8X, band-pass filtered (1 – 30 Hz), and the system computed the average VEP response to 75 pattern onsets, which was repeated twice for right eye, left eye, and both eyes of each subject. The display size was 30 degrees and viewed at 57 cm in an otherwise dark room. Subjects wore their habitual correction with added positive power as needed for patients 40 years and older. L and M cone check sizes were 1 degree, while S cone check sizes were 2 degrees to compensate for the lower resolution of the S cone pathway and based on preliminary measures that showed comparable amplitudes for these check sizes in color vision normal (CVN) subjects. After cleaning the scalp and earlobes with alcohol and abrasive cleaner, the VEP gold cup active electrode was filled with conductive paste and taped 1 cm above the inion with reference and ground electrodes affixed to the earlobes with electrode clips. The subject wore an elastic headband to secure the active electrode in place and electrode impedance was maintained at $\leq$ 5 Kilo-ohms. Each subject adapted to the display gray background (127 cd/m^2; $x,y = 0.303, 0.323$) for 6 minutes during electrode application. Prior to L, M, and S cone VEP recordings, each subject viewed the gray display with the right eye at zero contrast for the duration of one recording epoch to determine the noise level of the system yielding a median peak to trough amplitude of 2 μV, which was 5X smaller than normative amplitudes for L, M, and S cone VEPs but within the range of some severe CVD subjects. However, as specified in the Results, VEP amplitude ratios and latency differences allowed for definitive distinction of decreased amplitudes as well as delayed waveforms from artefactual noise.

To ensure that stimulation was specific for L, M, or S cones, display luminance and CIE chromaticities were measured with a Spyder 4 colorimeter (Data-color, Lawrenceville, NJ) using a custom program to transform the measured values to cone excitations based on Smith and Pokorny cone fundamental sensitivities and equations specified by Wyszecki and Stiles and Cole and Hine[19,20,26–28]:

$$\text{L excitation} = \text{Luminance}$$
$$\times \left[0.15514 \frac{x}{y} + 0.54312 - 0.03286 \frac{1 - x - y}{y} \right]$$

$$\text{M excitation} = \text{Luminance}$$
$$\times \left[-0.15514 \frac{x}{y} + 0.45684 - 0.03286 \frac{1 - x - y}{y} \right]$$

$$\text{S excitation} = \text{Luminance}$$
$$\times \left[0.00801 \times \frac{1 - x - y}{y} \right]$$

Percent cone contrast was computed as Weber contrasts: difference between cone excitation in colored checks and gray background divided by excitation in background multiplied by 100. Figure 1 shows L, M, and S cone-specific VEP displays based in part on the Cone Contrast Test (CCT; Innova Systems, Inc.), which presents letters of decreasing L, M, and S cone Weber contrast to determine the threshold for L, M, and S cone letter recognition. The CCT, used US Air Force-wide, diagnoses type and severity of hereditary CVD[19,20] and can also reveal acquired CVD in various diseases.[12,14] Hence the VEP stimulus used herein approximates a supra-threshold version of the CCT. By stimulating the targeted cone type well above threshold (color coded for each cone type in Fig. 1) but keeping stimulation to the other two cone types near or below threshold (white print in Fig. 1), isolation of a single cone mechanism can be achieved comparable to the time-honored technique of silent substitution. While stimulation of each targeted cone type with the VEP stimuli was above threshold for CVD subjects, the possibility of luminance artifacts or inadvertent stimulation of
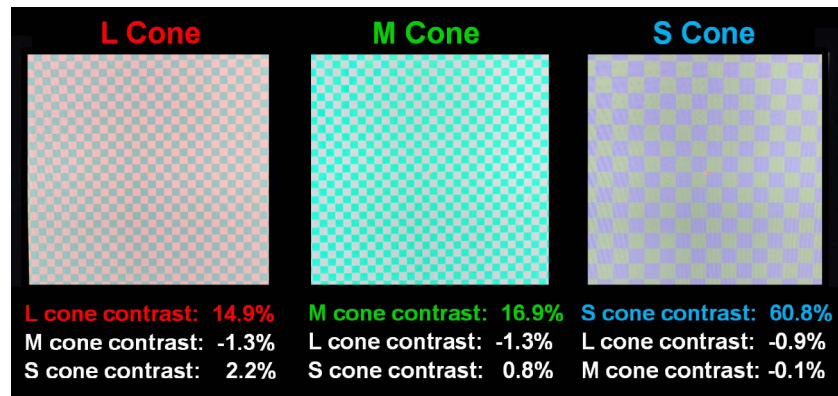
Figure 1. L, M, and S cone-specific checkerboard patterns used for pattern-onset VEPs. The cone-specific colored checks appeared as increments to the gray background, which appears different on each display due to color induction effects and photographic differences. The Weber contrast values are color coded for each cone type, while the *white text* represents contrast to nontargeted cone types, which is near or below threshold for detection.

nontargeted cone types is a potential source of error addressed more fully in the Results and Discussion sections. The likelihood of rod receptor stimulation with the VEP stimuli used herein is essentially nonexistent since the VEP display luminance was > 100 cd/m^2 well above rod saturation.[29]

Subjects were recruited from the students, faculty, staff, and patients at the University of the Incarnate Word Rosenberg School of Optometry (UIWRSO). The study protocol was approved by the UIWRSO Institutional Review Board, and all subjects were briefed on the protocol and provided written informed consent prior to participation in the study in accord with the Declaration of Helsinki. All subjects had visual acuity of at least 20/20 in each eye and no history of ocular disease. Subjects included 18 CVN subjects (mean age ± SD = 29 ± 11 years, 11 females) and 13 hereditary protan or deutan CVDs (mean age ± SD = 34 ± 12 years, 11 males, two females) confirmed to be CVN or CVD on a battery of tests including the CCT, Ishihara pseudo-isochromatic plates, and Oculus HMC Anomaloscope (Oculus, Inc., Arlington, WA). All CVN subjects reported no history of CVD and passed the Ishihara test (at least 12 of 14 plates correct) and the majority took the CCT achieving passing scores of 95 or higher on a scale of 100; < 75 is a failing score indicative of hereditary CVD. All CVD subjects failed the Ishihara test, CCT (mean ± SD on the cone CCT corresponding to their CVD = 38 ± 14), and Anomaloscope testing (mean deviation from normal matching midpoint ± SD = 16 ± 3; mean matching range ± SD = 18 ± 17). CVDs included four protanomalous subjects, confirmed to be protanomalous on the

Anomaloscope and CCT, eight deuteranomalous confirmed on Anomaloscope and CCT, and one subject who tested as deuteranopic in one eye and had an extensive matching range in the other eye on the Anomaloscope, with severe deutan deficiency on the CCT. There was no discrepancy between type of CVD (protan versus deutan) diagnosed by the HMC Anomaloscope and CCT, as reported previously.[19,20]

Results

Figure 2 shows the typical waveform for the pattern-onset cone VEP consisting of an initial negative trough followed by a positive peak. Consistent with previous studies of chromatic VEPs[21–25] latency was measured in milliseconds from pattern onset to the negative trough, and amplitude was measured in microvolts (µV) as the amplitude at the negative trough to amplitude at the subsequent positive peak (Fig. 2). In addition, data analysis showed that latency delays to the negative component was a better predictor of CVD than latency to the positive peak. Evaluation of CVNs showed no significant differences between right and left eyes for amplitude ($P > 0.9$) or latency ($P > 0.2$); hence average values from right and left eyes were used for analyses.

Figure 3 shows results for a normal, deutan, and protan subject on cone-specific VEPs. Compared to the normal subject, the protan subject shows a selective decrease in VEP amplitude and increase in latency on the L cone VEP, while the deutan subject shows a selective decrease in amplitude and increase in latency on the M cone VEP. S cone VEPs yielded

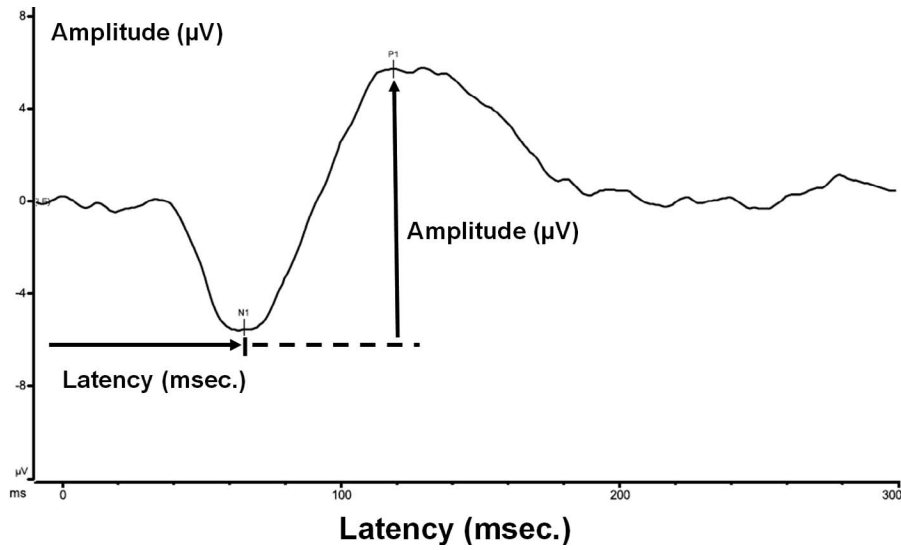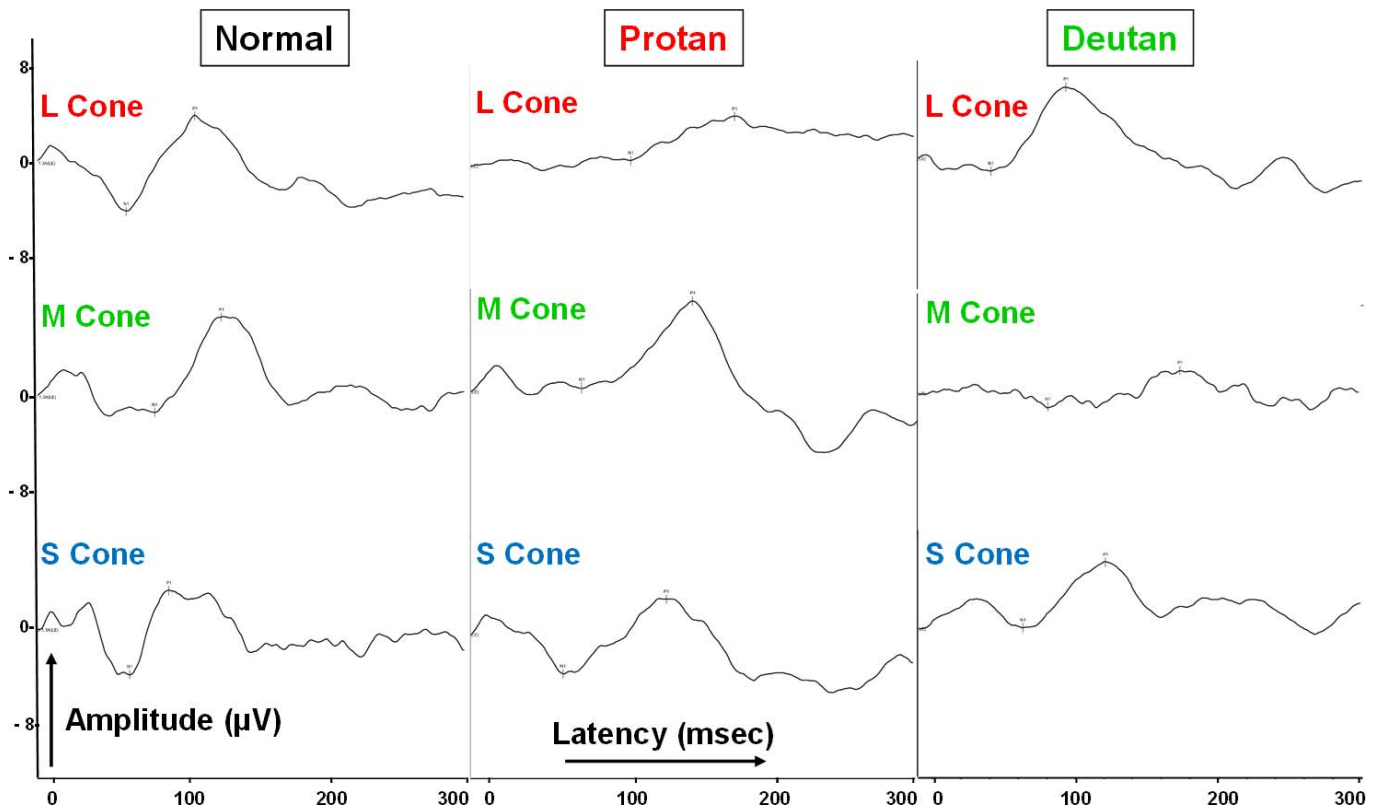**Figure 2.** Normal cone-specific pattern-onset VEP showing VEP latency and amplitude.

Figure 3. VEP waveforms from a normal subject (*left*), protan (L cone deficient, *middle*), and deutan (M cone deficient, *right*). VEP amplitude is shown on the y-axis (μV) and latency on the x-axis (msec). The L cone deficient (protan) shows decreased VEP amplitude and increased latency on the L cone VEP, and the M cone deficient (deutan) shows decreased amplitude and increased latency on the M cone VEP.
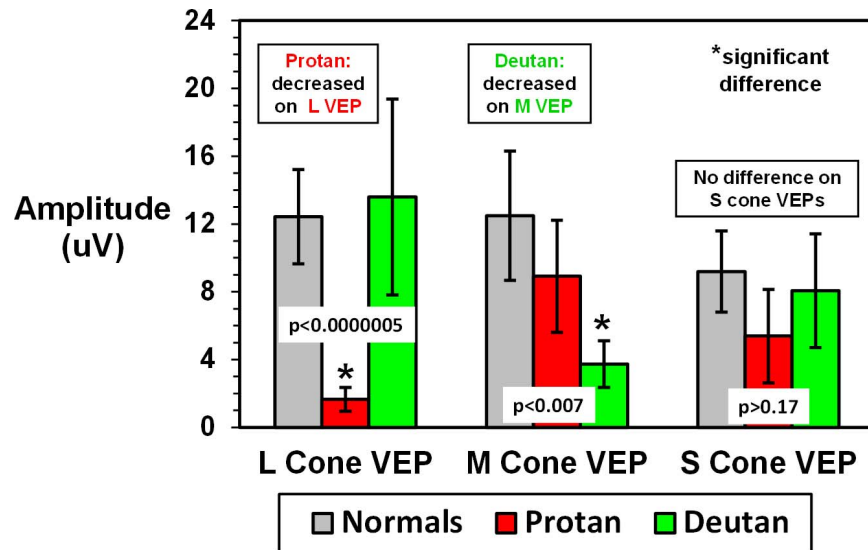
Figure 4. Mean VEP amplitudes (±2 standard errors) are shown for CVN, protan, and deutan groups. Compared to CVNs, two-tailed *t*-tests showed a significant decrease in VEP amplitude for protans on the L cone VEP ($P < 0.0000005$; "*") and for deutans on the M cone VEP ($P < 0.007$; "*"). There was no difference between groups on the S cone VEP ($P > 0.37$).

comparable results from all three subjects. These findings exemplify results obtained from 18 CVNs and 13 CVDs described in the results which follow.

Figure 4 shows mean (±2 standard errors) VEP amplitudes from CVN and CVD groups for L, M, and S cone VEPs. One-way analysis of variance (ANOVA) showed a significant difference between groups in L cone VEP amplitudes ($F = 6.0$, $P < 0.007$), but posthoc two-tailed *t*-tests showed that the only significant difference was the decrease in VEP amplitude in protan CVDs ($P < 0.0000005$; mean decrease 10.8 µV). Similarly, ANOVA showed a significant difference between groups in M cone VEP amplitudes ($F = 5.1$, $P < 0.02$) but *t*-tests showed that the only significant difference was the amplitude decrease in deutan CVDs ($P < 0.007$; mean decrease 8.4 µV). In contrast to these findings, there was no significant difference between CVD and CVN groups in S cone VEP mean amplitudes ($F = 1.0$, $P > 0.37$).

Figure 5 shows comparable results for VEP latency. There was a significant difference between groups in L cone VEP latency ($F = 16.9$, $P < 0.00002$) but *t*-tests showed that this difference was limited to increased VEP latency in protan CVDs ($P < 0.003$; mean increase 53.1 msec). Similarly, there was a significant difference between groups in M cone VEP latency ($F = 19.1$, $P < 0.000006$) due solely to increased latency in deutan CVDs ($P < 0.000004$; mean increase 31.0 msec). As with VEP amplitude,

there was no difference between groups in S cone VEP latency ($F = 0.6$, $P > 0.5$).

Both VEP latency and amplitude were utilized to establish initial criteria for cone VEP sensitivity for detecting CVD and specificity for confirming CVN status. Based on CVN 85th percentiles for L cone VEP latency (69 msec) and M cone VEP latency (86 msec), 100% of protan and deutan subjects were identified as CVD (i.e., 100% sensitivity) while specificity in CVN subjects was 83%. Based on CVN 15th percentiles for L cone VEP amplitude (8.4 µV) and M cone VEP amplitude (6.0 µV), 100% of protan and 89% (8/9) of deutan subjects were identified as CVD while specificity in CVN subjects was 83%. If VEP latency and amplitude are combined (i.e., both abnormal) to identify VEPs outside normal limits, then sensitivity increases to 100% for all CVDs and specificity increases to 94% for CVNs.

Insofar as VEP amplitude varies considerably between subjects, we sought an alternative metric to quantify hereditary CVD based on amplitude. We computed the ratios of L/M cone VEP amplitudes in CVN subjects (mean ratio ± SD = 1.15 ± 0.48) and compared these values to the ratio of: normal amplitude/affected cone amplitude in CVD subjects (mean protan ratio = 6.27, range: 2.95 to 12.22; mean deutan ratio = 3.80, range: 1.90 to 9.07). This approach detected all protan subjects and eight/nine deutan subjects (92% sensitivity) and yielded 89% specificity in normals. Sensitivity improved to 100%
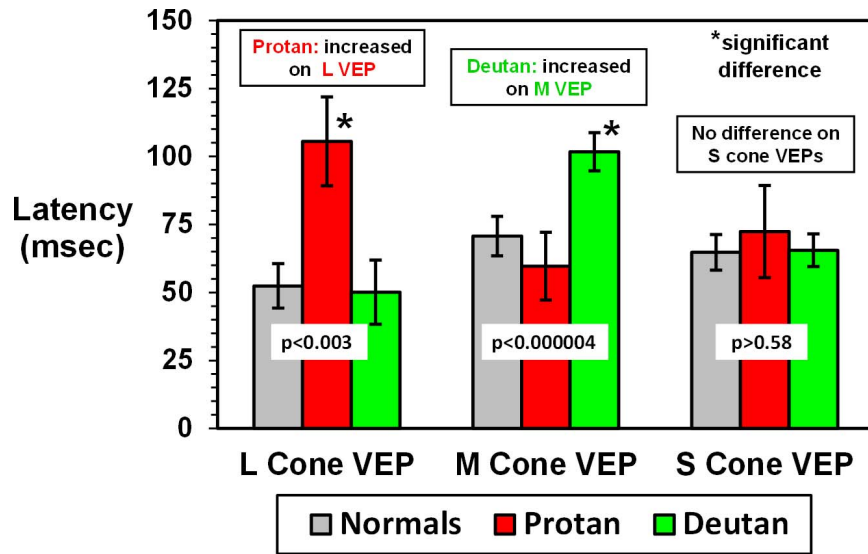
Figure 5.   Mean VEP latencies (±2 standard errors) are shown for CVN and protan, deutan, and CVD groups. Compared to CVNs, two-tailed *t*-tests showed a significant increase in VEP latency for protans on the L cone VEP ($P < 0.003$; "*") and for deutans on the M cone VEP ($P < 0.000004$; "*"). There was no difference between groups on the S cone VEP ($P > 0.58$).

and specificity to 94% when both latency and amplitude ratio are considered.

To establish normative values for S cone VEPs based on the 18 CVN subjects, the 85th percentile for latency (78 msec) and 15th percentile for amplitude (4.8 μV) were applied yielding 89% specificity for latency, 83% for amplitude, and specificity remained at 89% when both amplitude and latency were combined. Prior studies of S cone VEPs have argued that using large field sizes exceeding the area of the macular pigment introduces luminance artifacts since display calibration to selectively stimulate the S cones assumes absorption by the macular pigment.[21,30] To investigate this possibility, we recorded S cone VEPs on a CVN subject using our standard field size (30 degrees) as well as decreasing field sizes from 30 to 5 degrees. VEP waveforms are shown in Figure 6 for each field size. While VEP amplitude clearly decreases with decreasing field size and latency increases somewhat for small field sizes, waveform shape
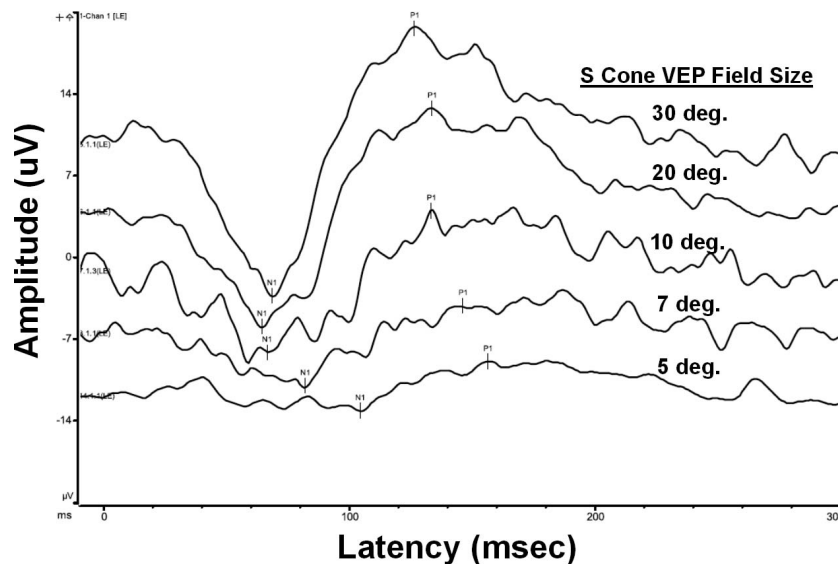
Figure 6.   S cone VEPs are shown for a CVN subject at field sizes ranging from the standard 30-degree field down to 5 degrees. Please see text for further details.

remains constant suggesting that a common mechanism is responsible for each S cone VEP. Hence, despite the probable existence of peripheral luminance artifacts with large fields, the high S cone contrast utilized for S cone VEPs in the present study apparently exceeds significant artifacts that would likely alter waveform shape and time course of the VEP, a finding which is consistent with recent research.[31,32]

In CVD subjects, there was no was no significant correlation between cone VEP parameters and anomaloscope findings. However, regression analysis between VEP amplitude and CVD CCT score approached significance ($P = 0.067$) indicating that CVD performance on the CCT is predictive of cone-specific VEP amplitude. Additional data are needed to substantiate this trend supporting the use of cone VEPs as a metric of hereditary CVD.

Discussion

The results of this study demonstrate that cone-specific VEPs can detect and diagnose hereditary CVD with sensitivity and specificity comparable to benchmark tests of color vision. The VEP test shows promise to confirm diagnosis in occupational settings wherein exact diagnosis is in question due to applicant familiarity or preparedness for test procedures. VEP amplitude and latency can be combined to enhance diagnostic accuracy, as well as amplitude ratios, which decreases the impact of individual differences in amplitude and potentially between different VEP systems. Additional advantages of cone-specific VEPs include objectivity (no subjective response from patient), rapidity (<10 minutes to administer), and access to diagnostic quantitative data at readily visible, supra-threshold levels of stimulation applicable to patients with decreased vision who are unable to respond to conventional tests including young patients, and both elderly and cognitively challenged patients from brain injury, stroke, or disease.

Limitations of this study include the relatively low number of CVN and CVD subjects, though differences were highly significant for diagnosis of hereditary CVD. In addition, the unique method of VEP stimulation limits its immediate clinical applicability and the cone contrasts utilized were limited by the color fidelity of the system. Efforts are underway to make this approach widely available and to expand the available levels of cone stimulation.

It is possible that artefactual stimulation of nontargeted cones (e.g., −1.3% contrast to M cones on L cone VEP, −1.3% contrast to L cones on M cone VEP) contributed variability to the results. However, in CVN subjects L cone VEP latency was significantly shorter than M cone latency ($P < 0.001$) indicating unique responses for each cone relatively free of contributions from nontargeted cones. In addition, the degree of artefactual stimulation is much less than the targeted supra-threshold stimulation of specific cone types and sensitivity and specificity results do not indicate that these small artifacts impede accurate diagnosis of hereditary CVD. As noted earlier, the 30-degree VEP field substantially exceeded the area of macular pigment such that luminance artifacts may have influenced S cone VEPs. However, our control recordings (Fig. 6) demonstrate that S cone VEP waveform remains relatively constant with changes in field size, consistent with the contention that S cone input dominates the large field VEP response as reported recently by Crognale and colleagues.[31,32]

In summary, this proof-of-principle study demonstrates the efficacy of cone-specific VEPs for diagnosis of hereditary CVD and shows potential for detection and monitoring of acquired CVD in ocular, systemic neurologic diseases. The pathway specificity, robust response to supra-threshold stimulation, and objective nature of cone-specific VEPs may further elucidate the pathophysiology of senescent and acquired brain disease enhancing earlier detection and fostering expeditious treatment.

Acknowledgments

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