ANALYSIS OF CHANGES IN THE CORTICAL RESPONSES OF AN AMBLYOPIC PATIENT PRE-VISION THERAPY VERSUS POST-VISION THERAPY

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ABSTRACT

Pattern reversal visual evoked responses (PVER) are an objective way to measure cortical responses in a patient, to measure visual acuity, and to diagnose certain ocular conditions. Amblyopia, clinically defined as a decrease in best corrected visual acuity to less than 20/30 in one eye or a two line difference between the two eyes with no apparent pathological cause, is easily diagnosed by PVER. Amblyopic PVER's show decreased amplitudes and increased latencies compared to the normal eye. The amblyopic eye also shows a peak PVER response to a stimulus check size of 30 to 40 minutes of arc, as compared to the peak response of the normal eye to stimulus check sizes of 10 to 20 minutes of arc. Although these characteristic PVER responses have been demonstrated by many researchers, minimal attention has been paid to the effects of visual training on the PVER response. In this study, a 12 year old anisometropic-strabismic amblyope with visual acuities of 20/20 in the right eye and 20/40 in the left eye was recruited as a patient. Pre-visual training PVER's were performed on the patient. A sequential visual training program was initiated including full refractive correction, direct occlusion, and active visual training to remediate the amblyopia and strabismus. PVER's were monitored every one to two months during visual training. A final PVER was recorded two months after the cessation of visual training.

The initial PVER showed a decreased amplitude and increased latencies in the amblyopic eye compared to the normal eye. The amblyopic eye also showed a loss of response to stimulus check sizes below 15 minutes of arc in the amblyopic eye. PVER's during visual training showed a great deal of variability in the responses obtained. In general, the amplitude and latencies in the amblyopic eye remained the same despite the subjective increase in visual acuity to 20/30. However, the amplitude decreased in the normal eye as visual therapy progressed despite the fact that subjective visual remained unchanged. This finding could be due to the fact that during visual training, more attention is devoted to training the amblyopic eye to improve its vision which can result in decreased vision and decreased visual skills in the normal eye. The normal eye's vision and visual skills usually return to normal after the cessation of visual training. However, in our study, the amplitude did not return back to the baseline level two months post-visual training. The literature suggests that the decreased amplitude in the normal eye may be due to poor patient attention during the recordings of the

PVER or due to habituation. Habituation is a phenomenon seen in patients trained for the PVER or in patients very familiar with the procedure due to repeated testing. Habituation has been shown to produce a decreased PVER response. Although either of these two factors could have caused the decreased amplitudes observed, there is no way to test for these phenomenon to know for sure if they are the cause of the results seen.

The last PVER was recorded two months post-visual training. At this time, the patients visual acuities were 20/20 in the right eye, and 20/25 in the left eye. The patient dropped out of visual training before a full functional cure was established. The PVER's revealed the same asymmetry in amplitude between the two eyes noted initially, but the amplitude in the normal eye was decreased compared to the initial findings. The latencies between the two eyes began to equalize for the larger check sizes of 60, 30, and 15 minutes of arc. The normal eye did, however, lose its response to the smaller check sizes of 7.5 and 3.75 minutes of arc.

The results of this single patient study suggest that there is limited value in monitoring PVER's during amblyopia treatment as an index of visual training success because the responses obtained were too variable to draw conclusions from. Although the results of this study were contaminated by the patient dropping out of visual training prior to establishing a full functional cure, the data does suggest some differences in the PVER's measured pre and post visual training. Further investigation on a larger clinical population who will follow the visual training program to its completion, is warranted to see exactly how much change in cortical function is occurring as a result of visual training for amblyopia.

INTRODUCTION

Amblyopia has a prevalence of two to three percent in the general population[1,9,10]. Clinically, amblyopia is defined as a decrease in best corrected visual acuity to less than 20/30 in one eye or a two line difference between the two eyes with no apparent ocular pathological cause[2]. There are many factors which can cause amblyopia, but the three most common causative factors are strabismus, uncorrected anisometropia, or a combination of strabismus and uncorrected anisometropia[3,11]. Although decreased visual acuity in one eye is often used for the initial diagnosis of amblyopia, the amblyopic patient often has a number of associated visual problems. The amblyopic patient may exhibit oculomotor and fixational problems, decreased accommodation ability in the amblyopic eye, decreased stereopsis, suppression, decreased contrast sensitivity, spatial uncertainty and distortion[2,11].

In the treatment of the amblyopic patient, the clinician must consider all aspects of the patient's visual system in order to prescribe a treatment regimen that will provide a functional cure for the patient's problems. Most clinicians agree that the goal of amblyopic visual training is to increase the visual acuity in the amblyopic eye and to establish good binocular vision[2]. There is much disagreement, however, over the age range that amblyopia treatment is successful. Many researchers and clinicians feel that it is of no use to attempt amblyopia treatment beyond the critical period of development during the first ten years of life[2,9,10]. Wick et al. studied the efficacy of the treatment of amblyopic patients beyond age ten who were assumed to have completed their critical period for ocular development. They found that amblyopic patients beyond the critical period could benefit from treatment. They felt that there was a "plastic period" during which the eye would respond to treatment, which extended into adulthood[2]. Caloroso agrees that amblyopia can be treated in patients beyond their critical period of development but it requires a great deal of patient motivation and a much longer course of treatment to achieve results[3].

In the last 25 years, researchers have been devoting a great deal of attention to the use of pattern reversal visual evoked responses (PVER) as a means of estimating visual acuity[4]. Clinically, however, PVER's have limited application due to a lack of standardized protocols for the recording of responses and the

analysis of data obtained, making it difficult to compare results between different clinics which are using PVER[4]. Despite the problems clinicians are encountering with the wide scale use of PVER as a standard clinical test of visual acuity, it has been shown to be very effective in detecting and aiding in the diagnosis of many vision problems, such as multiple sclerosis and functional amblyopia. It has been found that with multiple sclerosis, the PVER latencies are prolonged in one or both eyes[5]. In patients with functional amblyopia, the PVER in the amblyopic eye exhibits a decreased amplitude and increased latency compared to the normal eye[6,8,12,13,17]. It has also been noted that peak responses in the amblyopic eye occur at a stimulus check size of 30 to 40 minutes of arc[4,6,16]. Below this check size, the amplitude in the amblyopic eye is very decreased and often no recognizable response can be seen above the noise in the system[6]. For normal eyes, peak responses occur at check sizes of 10 to 20 minutes of arc[16].

Although the PVER has been used for the diagnosis of amblyopia in many patients, minimal literature has been found that compares the PVER of an amblyopic patient before and after visual therapy to see if the decrease amplitude and increased latency in the amblyopic eye change as subjective visual acuity improvement occurs. It is the focus of this paper to evaluate the use of PVER for monitoring the progress of visual therapy in the amblyopic patient and as a means of measuring change in cortical function as the amblyopia is remediated through traditional visual training programs. It is also the goal of this study to ascertain whether any cortical changes do occur as the amblyopia is resolved.

METHODS

A standard strabimus/amblyopia exam was performed on the subject to assess all areas of visual function and identify any areas that needed to be remediated through visual training. Visual acuities were assessed with a Snellen chart using letters for one eye and numbers for the other eye to prevent any memorization from contaminating the results. Visual acuities were also measured with the psychometric visual acuity tester as a means of comparing visual acuity without the contour interactions that can affect Snellen visual acuity[18]. A refraction was performed to establish best corrected visual acuity . A cover test was performed at distance and near to check for the presence of strabismus and to quantify any that was

present. Local and global stereopsis testing was performed. Suppression was checked for with the Worth Four Dot at distance and near. Monocular accommodation and oculomotor skills were assessed with $\pm$ 2.00 D flippers and a rotator, respectively. A visual training program was designed based on the results of the pre-visual training exam to address all problem areas. Visual training began with monocular occlusion four hours a day and monocular skills improvement activities. Once improvement was seen in monocular skills, antisuppression training was added to the treatment regimen. As suppression was remediated, binocular skills training was incorporated.

PVER's were recorded monocularly and binocularly pre-visual training, every one to two months during training, and two months post-visual training. The PVER apparatus consisted of a Venus Model 1020 stimulus generator by Neuro Scientific, an AST 286 computer with color monitor for analyzing recorded PVER responses, a Grass PS22 Amplifier, and a 16 inch Mitsubishi HL6615 TK color monitor for stimulus checkerboard pattern presentation. The stimulus used was a square wave checkerboard pattern of varying sizes ranging from 120 minutes of arc to 3.75 minutes of arc in one octave steps. Refer to Figure 1 for the parameters of each stimulus tested and its equivalent Snellen visual acuity.

The patient was placed 1.5 meters from the color monitor. The forehead, midpoint of the skull superior, and a point 3 cm above the external occipital protruberance were cleaned with alcohol and abraded with Nuprep to aid in conductivity and to decrease background noise during the recordings. Three Grass gold cup scalp electrodes were filled with EEG paste and applied to the skull in the three abraded positions previously described. The forehead scalp electrode served as a ground, the midpoint of the skull electrode served as the reference electrode and the electrode 3 cm above the external occipital protruberence served as the recording or active electrode. The resistance of each electrode was tested with a Grass EZN5B impedance meter. The resistances of each electrode were kept under 10 ohms. The PVER's were recorded in the dark with the patient fully corrected for any ametropia. A standard black eye patch was used for occlusion during monocular readings. All recordings were performed from largest check size to smallest check size.

PATIENT INFORMATION

The subject used was a 12 year old white male with a previous history of "lazy eye" four years prior to our initial exam. He had previously tried patching but his mother reported that he refused to wear the patch due to teasing he received from siblings, classmates, and friends. The patient was initially seen at the Ferris State University Optometry Clinic in August 1992. At that time, he was a 10$^{\Delta}$ intermittent alternating esotrope. His refraction and best corrected visual acuities were OD +1.75 -1.00 x 180 20/25 OS +3.00 -2.00 x 180 20/50. Suppression was noted at distance and near. Visual training was declined at that time.

The patient was seen again in September 1994 for an annual exam. At this time, he was a 15$^{\Delta}$ intermittent left esotrope at distance and 10$^{\Delta}$ intermittent left esotrope at near. No stereopsis was present. Refraction and best corrected visual acuities were OD +1.50 -0.75 x 160 20/20 OS +2.00 -1.50 x 05 20/40 . Psychometric visual acuity showed OD 20/20; OS 20/60. The patient did not want spectacles and the mother reported a past history of poor compliance with spectacle wear. Due to these factors, it was decided to fit the patient with contact lenses. He was fit with a Cibasoft 8.6 +1.25 13.8 lens in his right eye with 20/20 visual acuity. He was fit with a B&L Optima Toric 8.6 +2.00 -1.50 x 25 in his left eye with 20/40 visual acuity. Visual training was discussed with the patient and his parents offering the alternative of a blur contact lens to the traditional patch that the patient refused to wear. The parents and the patient expressed a desire to try visual training.

The pre-visual training exam in February 1995 showed similar findings to the comprehensive exam in September 1994. Additional tests were performed for determination of visual training goals and treatment options. Suppression testing with a Worth Four Dot showed alternating suppression at distance and superimposition at near (first degree fusion). No anomalous retinal correspondence or eccentric fixation were present on testing with Haidingers brushes. Monocular accommodative skills were decreased in the left eye especially with -2.00 D flippers. Monocular oculomotor skills were very unsteady with the left eye. Occlusion with a blur contact of +6.00 D for the right eye was initiated four hours a day.

The patient returned one month later to start visual training. Monocular visual acuity improvement, monocular accommodative skills, and monocular oculomotor skills were initially worked on. By the end of three months of visual training, visual acuity in the left eye had increase to 20/30 and the patient was able to progress to antisuppression visual training. After another three months of visual training, visual acuity was still 20/30 but suppression was shallowing. Binocular visual training was initiated using tasks with antisuppression checks. The patient dropped out of visual training ten months after the initiation of treatment. See Figure 2 for examples of visual training tasks performed.

A post visual training exam was performed in February 1996. At this time, a new refraction was performed and Snellen visual acuity through the new prescription was OD +1.25 -0.25 x 90 20/20 OS +1.50 -1.50 x 10 20/25. Psychometric visual acuity showed OD 20/30 and OS 20/60. Cover testing revealed 6$^{\Delta}$ alternating esotropia at distance and near. Suppression testing with Worth Four Dot revealed flat fusion at distance and near. Refer to Figure 3 for a comparison of exam findings pre-visual training versus post-visual training.

RESULTS

Visual training progress was monitored at monthly visits. At each followup visit the skills being worked on by the patient at home were demonstrated to the examiner. If the patient could perform the task correctly and effortlessly with each eye, the patient was advanced to the next stage of therapy.

PVER's were recorded before visual training, every one to two months during visual training and two months after the completion of visual training. The patient was tested monocularly and binocularly at all stimulus check size patterns. The computer produced an average waveform of all of the sample readings collected. Each waveform was analyzed in three ways. First, it was decided whether there was a recognizable waveform present. Secondly, the amplitude of the response from N1, the first negative polarity change, to P2, the first positive polarity change, was measured. Thirdly, the latency, time from onset of stimulus until the peak of the P2 wave, was measured. The values for amplitude and latency were compared to the tables of normals compiled by Jeffrey M. Chadwick on the Ferris State

University PVER equipment. The values were also compared between each eye and with both eyes. Refer to Figure 4 for an example of the waveforms obtained and the analysis procedure performed on all PVER data. Refer to Figure 5 and 6 for a table of the results of the PVER for this study.

The results of the analysis of the PVER for larger check sizes of 120 and 60 minutes of arc showed decreased amplitudes in the amblyopic eye and slightly increased to approximately equal latencies compared to the normal eye. For check sizes of intermediate spatial frequencies of 30 and 15 minutes of arc, the amblyopic eye showed decreased amplitude and increased latencies compared to the normal eye. For the check sizes of 7.5 and 3.75 minutes of arc, no response was recorded for the amblyopic eye. Initially, a response was seen in the normal eye, but as treatment progressed, this response disappeared.

The PVER's monitored over the course of visual training showed a large amount of variability in amplitude and latencies each time a recording was taken. In comparing the pre-visual training results with the post visual training results, the general trend showed an end PVER which was decreased in amplitude from the initial PVER's recorded. The asymmetry in amplitudes was retained between the two eyes with the amblyopic eye having the lower amplitude. The latency in each eye increased compared to the initial PVER recordings but the latency for the two eyes were closer to being equal than they were initially. Refer to Figure 6 and 7 for a comparison of all the PVER recording pre and post visual training.

PVER's can also be used to estimate visual acuity. In the literature three methods are described for obtaining visual acuity from PVER recordings. First, visual acuity can be estimated from the smallest check size producing a recordable and reproducible pattern[4,7]. Secondly, the amplitude at different spatial frequency can be measured and compared to identify a peak response location which corresponds to the patient's visual acuity[4]. Thirdly, a regression line of an amplitude check size curve can be analyzed to the zero point. The check size correlating to the zero point equals the visual acuity[7]. In this study we employed the first method of estimating visual acuity from the smallest check size responded to. The second technique was not chosen because there was too much variability in peak responses. The third technique was not chosen because it has been shown not to be effective in estimating visual acuity with amblyopia[7]. Initially our study showed PVER visual

acuities of 20/80 in the right eye (3.75 minutes of arc), 20/300 in the left eye (15 minutes of arc) and 20/80 with both eyes. At the end of visual training, the PVER visual acuities were 20/300 in the right eye, left eye, and in both eyes.

Although little improvement was seen and there was actually a decrease in visual acuity in the right eye on the PVER, Snellen visual acuity did increase two lines in the amblyopic eye and stayed the same in the right eye. Many researchers have found that in amblyopic patients, Snellen visual acuity is generally better than PVER visual acuity[4,8]. Young-Hoon et al. concluded that at small check sizes, the amblyopic PVER had very decreased amplitudes and very irregular patterns[4]. We found this to be true in our study also. Snellen visual acuity was consistently better in both eyes than viusal acuity estimated with PVER and the amblyopic eye showed no recordable pattern with check sizes of 7.5 minutes of arc or smaller.

The one puzzling factor our study uncovered was the decrease in amplitude and loss of response to smaller check sizes in the normal eye, although subjective visual acuity did not change. In the literature, there have been reports of decreased amplitude of the PVER due to poor patient attention during test recordings[12,14]. Although the patients fixation was monitored during each recording, there was no direct way to assess the patients attention state, so the decrease in the PVER amplitude and the loss of response to smaller check sizes could be due to this. One other possible explanation for the results observed could be due to habituation. Habituation is seen in subjects who are trained for the PVER or who have performed this task a number of times and have become very familiar with the procedure for the PVER. Perry et al. have reported that habituation in trained subjects can cause decreased PVER responses with repetitive stimulation[17]. Although this is another possible explanation for the decreased amplitude seen in the PVER's recorded as visual therapy progressed, there is no way to know for sure if this phenomenon was occuring.

One possible explanation for the loss of the response at the 3.75 minutes of arc check size could be due to the property of the stimulus used. Numerous studies have shown that stimulus check sizes smaller than 5 minutes of arc are not square waveforms and their resolution on the monitor is disrupted by the raster lines of the computer and interaction between the square wave harmonics of the pattern,

causing the eye's response to the pattern to become very variable, making consistent recordable responses difficult to obtain[4,7,8]. The initial response to the 3.75 minutes of arc check size may have been a fluke.

DISCUSSION

The treatment of amblyopia with occlusion and active visual training is a well established standard of clinical care[11,15]. We employed a sequential plan of treatment suggested by Wick et al.[2]. First of all, the patient was given his full prescription to ensure clear images for each eye. Secondly, we initiated direct occlusion four hours a day. Thirdly, we employed active visual training to increase monocular visual acuity and improve binocular visual function[2]. There is much disagreement over the relevancy of treating a patient who is considered to be beyond his "critical period" for development. Wick et al. also have shown that amblyopia treatment can be successful past the age of ten because the visual system has a "plastic period" extending into adulthood during which treatment can work. We did exhibit increased visual acuity and visual performance in the amblyopic eye with treatment. However, since the patient dropped out of therapy before it was completed, it cannot be concluded whether a full functional cure could be established in a patient beyond the critical first ten years. There is also some question as to whether the increased visual acuity and improvements in visual performance will last without maintenance visual training and the establishment of solid binocular vision. It has been shown in previous studies that 25% to 52% of patients require maintenance visual training to maintain visual acuity[2]. It has also been shown that normal binocularity is required for a functional cure[2,15].

The PVER results show the diagnostic differences in decreased amplitudes and increased latency in the amblyopic eye. This study's goal was to ascertain whether PVER would be useful for monitoring the success of visual training and to see whether any cortical changes took place as vision returned to normal in the amblyopic eye. PVER's were run every one to two months during visual training. The results were very erratic and showed no clear patterns of behavior. These erratic results could be due to the changes going on in the visual system during visual training making the ocular system less consistent and repeatable. These results suggest that the PVER is of little therapeutic use in monitoring visual

training progress in the amblyopic patient.

The initial PVER's and the final PVER's were compared for differences that would suggest changes in cortical functioning as visual acuity increased. The results showed that the normal eye's amplitude responses became worse as visual therapy progressed and the asymmetry in amplitudes between the two eyes was maintained. The latencies did show a trend towards equalization at larger check sizes. Because the patient dropped out of visual training prior to establishing a "functional cure" it is uncertain whether any cortical changes did occur. Our findings suggest no, but they are contaminated by a lack of a fully "cured" patient. Further studies in this area would be required to state for certain whether visual training alters cortical functioning in the amblyopic patient. Our results show some cortical changes are occuring and further investigation on a larger clinical population is warranted to establish definite conclusions.

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ADDENDUM 1: FIGURES

Stimulus Check Size	Minutes of Arc	Visual Acuity
128 check	3.75	20/80
64 check	7.50	20/150
32 check	15.00	20/300
16 check	30.00	20/600
8 check	60.00	20/1200
4 check	120.00	20/2400

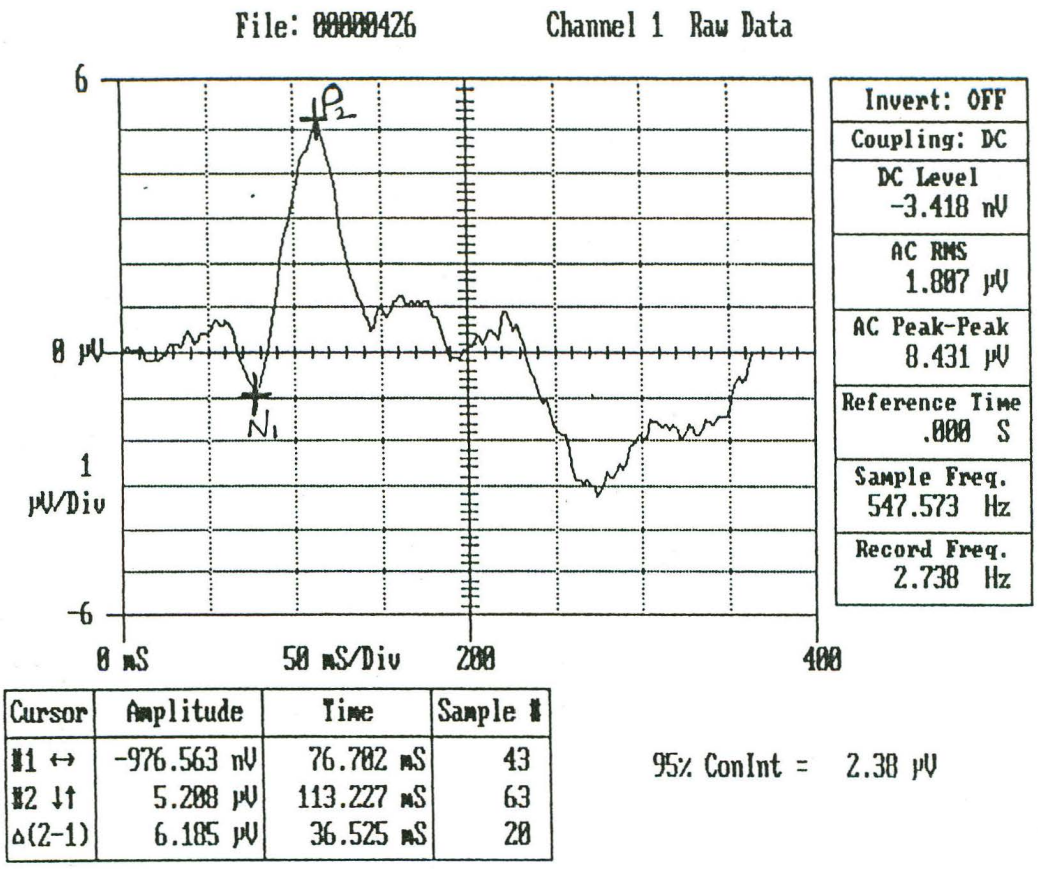
Figure 1 Stimulus check size parameters for the pattern reversal visual evoked potential.

Type of visual training	Tasks
Monocular Oculomotor Skills	Rotator with and without stressors
	Mazes
	Dot to Dots
	Underline and Circle
Monocular Accommodative Skills	± 2.00 D Flippers
	Hart Chart Accommodative Rock
Monocular Visual Acuity	Blur Contact Lens
	Tweezers and Rice
	Toothpicks and straws
	Clothes pins in a can
Antisuppression Skills	Red-Green T.V. trainer
	Red-Green Black Jack
	Red-Green Mazes and Dot to Dots
	Red-Green Bar Reader
	Franzblau
Binocular Skills	Brock String
	(Patient dropped out of therapy at this point)

Figure 2 List of visual training techniques used on the patient.

Exam Date	Visual acuity		Refraction	Cover Test	Worth 4 Dot
02-13-95 (Pre-VT)	OD	20/20	OD +1.50 -0.75 x 160	10$^{\Delta}$intermittent OS Esotrope at distance	Alternating Suppression at distance
	OS	20/40	OS +2.00 -1.50 x 05	10$^{\Delta}$intermittent OS Esotrope at near	Super-imposition at near
02-13-96 (Post-VT)	OD	20/20	OD +1.25 -0.25 x 90	6$^{\Delta}$ alternating Esotrope at distance	Flat Fusion at distance
	OS	20/25	OS +1.50 -1.50 x 10	6$^{\Delta}$alternating Esotrope at near	Flat Fusion at near

Figure 3 Comparison of exam findings pre-visual training (pre-VT) and post-visual training (post-VT).



Cursor	Amplitude	Time	Sample #
#1 ↔	-976.563 nV	76.782 mS	43
#2 ↓↑	5.288 µV	113.227 mS	63
Δ(2-1)	6.185 µV	36.525 mS	20

95% ConInt = 2.38 µV

Figure 4 An example of a PVER average waveform obtained as data. The major peaks are labeled. N1 represents the first negative polarity change and P2 represents the first positive polarity change. The amplitude is measured from the trough of N1 to the peak of P2 and is represented by the box (2-1) Amplitude. The The latency is measured from the time of stimulus onset to the peak of P2 and is represented by the box #2 time. For this example, the amplitude is 6.185 uV and the latency is 113.227 ms.

Date	Eye	4 check	8 check	16 check	32 check	64 check	128 check
	OD	7.045 uV	6.185 uV	6.366 uV	5.839 uV	2.131 uV	2.279 uV
02-13-95	OS	4.639 uV	5.052 uV	4.150 uV	No Resp.	No Resp.	No Resp.
	OU	6.177 uV	5.524 uV	6.648 uV	4.691 uV	1.160 uV	1.872 uV
	OD	6.699 uV	4.929 uV	5.127 uV	2.482 uV	No Resp	No Resp
04-18-95	OS	3.362 uV	2.797 uV	4.447 uV	2.476 uV	No Resp	No Resp
	OU	No Readings taken ou					
	OD	2.930 uV	6.290 uV	4.761 uV	3.209 uV	1.933 uV	No Resp
05-21-95	OS	3.681 uV	3.196 uV	2.413 uV	3.113 uV	No Resp	No Resp
	OU	5.397 uV	4.541 uV	5.266 uV	3.526 uV	No Resp	No Resp
	OD	4.224 uV	6.547 uV	3.304 uV	1.600 uV	1.563 uV	No Resp
07-31-95	OS	3.812 uV	4.272 uV	2.594 uV	1.031 uV	No Resp	No Resp
	OU	4.069 uV	3.723 uV	4.293 uV	2.974 uV	No Resp	No Resp
	OD	Not Run	4.594 uV	5.571 uV	1.831 uV	No Resp	No Resp
10-02-95	OS	1.709 uV	2.950 uV	No Resp	No Resp	No Resp	No Resp
	OU	4.395 uV	4.468 uV	4.297 uV	No Resp	No Resp	No Resp
	OD	4.986 uV	3.391 uV	2.563 uV	2.097 uV	No Resp	No Resp
02-13-96	OS	2.482 uV	2.014 uV	2.380 uV	2.752 uV	No Resp	No Resp
	OU	4.069 uV	5.821 uV	4.708 uV	3.482 uV	No Resp	No Resp

Figure 5 Data on amplitudes of PVER's recorded.

Date	Eye	4 check	8 check	16 check	32 check	64 check	128 check
	OD	107.748 ms	113.227 ms	111.401 ms	122.358 ms	136.968 ms	131.489 ms
02-13-95	OS	109.574 ms	113.227 ms	116.879 ms	No Resp	No Resp	No Resp
	OU	113.227 ms	109.574 ms	111.401 ms	116.879 ms	133.316 ms	147.925 ms
	OD	113.227 ms	111.401 ms	111.401 ms	118.706 ms	No Resp	No Resp
04-18-95	OS	115.053 ms	111.401 ms	118.706 ms	No Resp	No Resp	No Resp
	OU	No PVER's were run ou					
	OD	109.574 ms	116.879 ms	116.879 ms	131.489 ms	168.014 ms	No Resp
05-21-95	OS	116.879 ms	109.574 ms	124.184 ms	131.489 ms	No Resp	No Resp
	OU	109.574 ms	116.879 ms	116.879 ms	124.184 ms	No Resp	No Resp
	OD	116.879 ms	116.879 ms	116.879 ms	138.794 ms	153.404 ms	No Resp
07-31-95	OS	116.879 ms	124.184 ms	131.489 ms	131.489 ms	No Resp	No Resp
	OU	116.879 ms	124.184 ms	124.184 ms	131.489 ms	No Resp	No Resp
	OD	Not Run	109.574 ms	116.879 ms	116.879 ms	No Resp	No Resp
10-02-95	OS	124.184 ms	124.184 ms	No Resp	No Resp	No Resp	No Resp
	OU	124.184 ms	116.879 ms	116.879 ms	No Resp	No Resp	No Resp
	OD	109.574 ms	116.879 ms	116.879 ms	124.184 ms	No Resp	No Resp
02-13-96	OS	124.184 ms	116.879 ms	124.184 ms	124.184 ms	No Resp	No Resp
	OU	116.879 ms	109.574 ms	116.879 ms	124.184 ms	No Resp	No Resp

Figure 6 Data on PVER's latencies.

Date	Eye	4 Check	8 Check	16 Check	32 Check	64 Check	128 Chec
	OD	7.045 uV	6.185 uV	6.366 uV	5.839 uV	2.131 uV	2.279 uV
02-13-95	OS	4.639 uV	5.025 uV	4.150 uV	No Resp	No Resp	No Resp
	OU	6.177 uV	5.524 uV	6.648 uV	4.691 uV	1.160 uV	1.872 uV
	OD	4.986 uV	3.391 uV	2.563 uV	2.097 uV	No Resp	No Resp
02-13-96	OS	2.482 uV	2.014 uV	2.380 uV	2.752 uV	No Resp	No Resp
	OU	4.069 uV	5.821 uV	4.708 uV	3.482 uV	No Resp	No Resp

Figure 7 Comparison of PVER amplitudes pre and post visual therapy.

Date	Eye	4 Check	8 Check	16 Check	32 Check	64 Check	128 Chec
	OD	107.748 ms	113.227 ms	111.401 ms	122.358 ms	136.968 ms	131.489
02-13-95	OS	109.574 ms	113.227 ms	116.879 ms	No Resp	No Resp	No Resp
	OU	113.227 ms	109.574 ms	111.401 ms	116.879 ms	133.316 ms	147.925
	OD	109.574 ms	116.879 ms	116.879 ms	124.184 ms	No Resp	No Resp
02-13-96	OS	124.184 ms	116.879 ms	124.184 ms	124.184 ms	No Resp	No Resp
	OU	116.879 ms	109.574 ms	116.879 ms	124.184 ms	No Resp	No Resp

Figure 8 Comparison of PVER latencies pre and post visual therapy.