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Vernier acuity is selectively affected in infants and children with cortical visual impairment

Ann M Skoczenski* PhD, Eunice Kennedy Shriver Center, University of Massachusetts Medical School, Waltham, MA; **William V Good** MD, Smith-Kettlewell Eye Research Institute, San Francisco, CA, USA.

**Correspondence to first author at* Eunice Kennedy Shriver Center, University of Massachusetts Medical School, 200 Trapelo Road, Waltham, MA 02452, USA.
E-mail: ann.skoczenski@umassmed.edu

Cortical visual impairment (CVI) refers to bilateral impairment of vision that is usually due to damage occurring perinatally in the visual cortex and/or optic radiations. The most common cause of this damage is hypoxia, and other causes include encephalitis, meningitis, and trauma. Relatively little research has been done to quantify visual abilities in children with CVI. In the present study, we used an electrophysiological technique (visual evoked potentials) to measure two aspects of spatial vision in 35 infants and children with CVI (15 females, 20 males; mean age 3 years 6 months, SD 3 years 5 months; age range 4 months to 16 years). We measured each child's grating acuity (resolution for detecting high-contrast stripe patterns) and vernier acuity (resolution for localizing pattern elements). Performance on grating acuity and vernier acuity in individuals with CVI was compared with that of age-matched individuals with normal vision, and it was found that vernier acuity was relatively lower than grating acuity in children with CVI. Results support the theory that vernier acuity is cortically mediated, and suggest that vernier acuity is a more sensitive measure than grating acuity for quantifying vision deficits in patients with CVI.

Cortical visual impairment (CVI) is the leading cause of childhood vision impairment in Western countries (Johnson-Kuhn 1995, Steinkuller et al. 1999). It is a condition in which children have reduced visual acuity as a result of damage to posterior visual pathways (Good et al. 1994, 2001b). In most cases, the eyes of such children are 'structurally' normal, yet they have diminished visual capacity. The damage causing this reduction in vision may occur in white matter, as in periventricular leukomalacia, or in gray matter, as in perinatal hypoxia–ischemia. As cortical and subcortical damage can both potentially affect vision, the term cerebral visual impairment is preferred by some (Cioni et al. 1996, 1997).

Given that there is no precise medical treatment for CVI, management of CVI involves efforts at prevention, or rehabilitation once the neurological damage occurs. One problem hampering treatment efforts and treatment trials for CVI is difficulty quantifying the levels of residual vision in these children. Without reliable tools to measure vision in children with CVI, it is difficult to determine the appropriate level of visual stimulation for treatment and education, or to know whether treatment is having any effect.

Children with CVI are often non-verbal and cannot describe what they can see. Therefore, quantitative tools to measure vision in CVI must measure vision with behavioral or electrophysiological techniques that do not require verbal responses. Behavioral techniques rely on a specific response by the child (e.g. head/eye turn, or pointing) to indicate that a visual target has been seen (Dobson et al. 1978, McDonald et al. 1985). In the Teller Acuity Card procedure, for example, cards with grating lines are positioned in front of a child (Dobson et al. 1978). If the child sees the lines, they will make an eye and/or head movement towards the lines, with this movement interpreted as an ability to see the lines. Behavioral measures of vision in children virtually always use grating acuity for clinical purposes, and require head and eye movement control. Motor control is frequently diminished or lacking in children with CVI, making interpretation of behavioral tests problematic in some cases (Birch and Bane 1991).

The visual evoked potential (VEP) is a non-invasive electrophysiological measure of the component of the EEG elicited by a visual target (stimulus), obtained by positioning surface electrodes on the scalp over the region of the visual cortex. The stimulus can be presented as a single flash of light or briefly presented pattern to elicit a transient VEP, or continuously at a given temporal frequency to elicit a steady-state VEP. The rapid data collection that is possible with the steady-state VEP offers the opportunity to change the size or configuration of the target during a short presentation trial, in order to alter the visibility in the dimension of interest. For example, reducing the element size of the stimulus pattern during a presentation trial will cause a measurable reduction in the evoked response, and allows an estimate of detail vision with data obtained in six to ten 10-second trials. A threshold can be measured by extrapolating the signal into the background noise, as the stimulus parameter is 'swept' from above to below an individual's visual detection threshold.

Grating acuity thresholds have been measured successfully in children with CVI using both behavioral and electrophysiological techniques (Birch and Bane 1991, Good 2001). However, many studies have demonstrated that grating acuity does not always correlate precisely with optotype acuity; i.e. the ability to identify letters or other symbols (Cryotherapy for

Retinopathy of Prematurity Cooperative Group 1996, Dobson et al. 1999). Compared with grating acuity, optotype acuity is probably subserved by different cortical regions or mechanisms that support the discrimination of different spatial patterns (e.g. letters), rather than simple pattern detection. The ability to see and identify symbols is arguably more important than the ability to see finely spaced lines, so a vision test that reflects optotype acuity may be more valuable than a grating acuity measure alone.

Vernier acuity is a form of hyperacuity that measures the ability to discriminate the relative position of different components of a visual pattern (Westheimer 1975). In this task, the individual is required to discriminate lines that are perfectly aligned, from lines that are offset. The ability to identify fine offsets exceeds the visual resolution predicted by photoreceptor cell spacing. The designation of vernier acuity as a

Table I: Descriptive information for 35 patients tested

Patient	*Test age (y)*	*Etiology*	*Seizure*	*Other eye anomalies*	*Medications/ treatments*	*Grating acuity ratio*	*Vernier acuity ratio*
1	0.3	HIE	–	–	–	0.607	0.147
2	0.9	HIE	+	–	Phenobarbital Clonazepam	0.152	
3	5	HIE	+	–	Phenobarbital Ketogenic diet	0.109	0.063
4	4	Partial complex mitochondrial disorder	+	Mild optic atrophy	Clonazepam	0.144	0.174
5	0.31	HIE	–	–	–	0.890	0.119
6	2.7	Unknown	–	–	–	0.279	0.094
7	0.5	HIE	–	Nystagmus	–	0.336	
8	6.8	HIE	+	–	Valproic acid Clonazepam	0.305	0.231
9	2.2	HIE	+	–	Valproic acid Clonazepam	0.094	
10	4.7	Encephalitis	–	–	–	0.512	0.991
11	0.87	HIE	+	–	–	0.371	0.193
12	3.3	Stroke, 0.4y age	+	–	Carbamazepine Clonazepam	0.079	
13	2.8	HIE	+	–	–	0.196	0.234
14	4.5	Porencephalic cyst	–	–	–	0.636	0.418
15	2.4	HIE	+	–	Clonazepam Valproic acid Topiramate	0.170	0.071
16	16	Near drowning, 1.3y age	–	–		0.337	0.072
17	4.9	HIE	+	–	Clonazepam	0.386	0.127
18	1.49	HIE	+	–	Carbamazepine Valproic acid	0.486	
19	6	Meningitis, 0.3y age	+	–	Lamotrigine	0.280	0.069
20	1.9	Trauma	+	–	Valproic acid	0.438	
21	1.75	HIE	–	–	–	0.588	0.188
22	2.3	Unknown	–	–	–	0.164	0.109
23	1.9	HIE	+	–	Lamotrigine Phenobarbital	0.331	
24	1.37	HIE	+	–	Lamotrigine	0.486	0.153
25	1.81	Hydrocephaly	–	–	–	0.497	0.201
26	0.34	HIE	–	–	–	0.414	
27	3.1	HIE	+	–	–	0.307	0.101
28	2.9	Cardiac arrest	+	–	Phenobarbital	0.273	0.147
29	0.31	HIE	+	–	Phenobarbital	0.529	0.153
30	0.5	HIE	–	–	–	0.457	0.113
31	5.9	Encephalitis	–	–	–	0.453	0.256
32	9.9	Cerebellar atrophy	–	–	–	0.561	0.165
33	1	HIE	–	Esotropia	–	0.264	0.152
34	6.1	Encephalitis	+	–	Phenobarbital	0.714	0.231
35	11.1	HIE	+	–	Valproic acid Corticotropin	0.149	0.026

HIE, hypoxic– ischemic encephalopathy.

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hyperacuity comes from the fact that, in adults with normal vision, vernier acuity is superior to grating acuity, the latter of which is predicted by photoreceptor spacing (Westheimer 1975). Vernier acuity probably requires cortical interaction and processing. Levi and others have shown that vernier acuity probably more closely approximates optotype acuity than does grating acuity, making vernier acuity tasks attractive stimuli with which to attempt to measure vision in children with CVI (Levi and Klein 1982, Levi et al. 1983). During normal development, vernier acuity develops significantly later than grating acuity, suggesting that the two functions are limited by different visual mechanisms (Zanker et al. 1992; Carkeet et al. 1997; Skoczenski and Norcia 1999, 2002). Moreover, there is some evidence that vernier acuity is a 'hypo-acuity' (worse than grating acuity) during early infancy in children with normal visual experience (Shimojo and Held 1987, Skoczenski and Norcia 2002). Several investigators have attempted using behavioral techniques to measure vernier acuity in a number of childhood visual disturbances (Holmes and Coates 1994, Holmes 1996, Birch et al. 2000). We have studied VEP vernier acuities in amblyopia, and compared these acuity thresholds with VEP grating acuity and Snellen (optotype) acuity in adults (Good et al. 2001a). Vernier acuity is a better approximation of Snellen acuity than is grating acuity in this subject group. In the present investigation we measured VEP vernier and grating acuity in children with CVI. Our goal was to determine the utility of each measure in identifying vision loss in patients with CVI. We hypothesized that if vernier acuity is, in fact, critically dependent on cortical processing, it may be selectively lower, compared with grating acuity, in children with CVI.

Method

PARTICIPANTS

We tested 35 children with CVI (15 females, 20 males; mean age 3 years 6 months, SD 3 years 5 months; age range 4 months to 16 years). The experimental protocol was approved by the Institutional Review Board of the California Pacific Medical Center, and informed consent was obtained from parents or legal guardians. Participants were referred by pediatric ophthalmologists or by the Blind Babies Foundation of Northern California, a non-profit service organization. All participants had substantial vision impairment. Each patient had an eye examination to rule out problems in the anterior visual path-

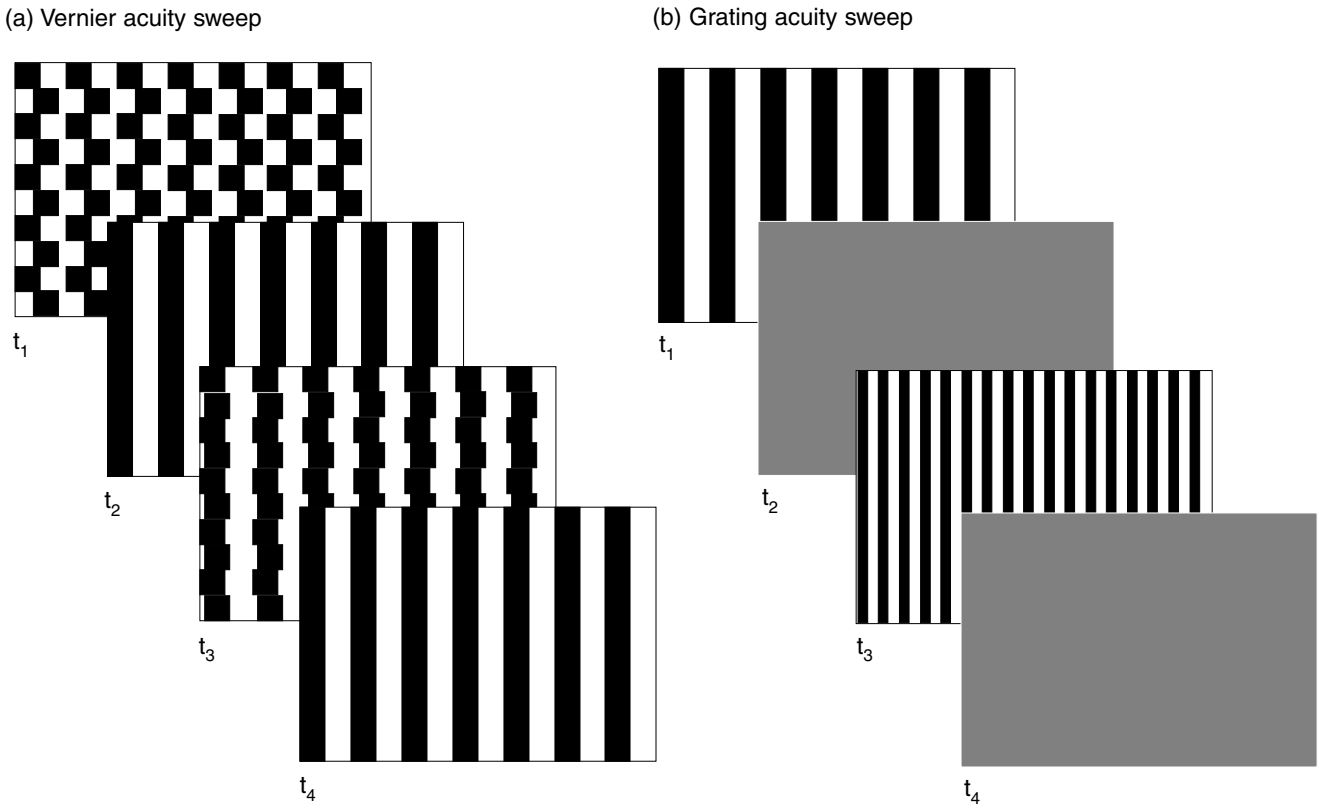

Figure 1: *Schematic drawings of visual stimuli used to measure vernier acuity and grating acuity. (a) Vernier acuity stimulus consisted of a squarewave grating (spatial frequency = 1 cycle per degree) in which vernier offsets appeared and disappeared at a rate of 5Hz. Each offset appearance or disappearance phase (labeled t_1, t_2, etc.) lasted six video frames. Over a 10-second trial duration, size of vernier offset was systematically decreased in 10 equal logarithmic steps. (b) Grating acuity stimulus consisted of a sinewave grating that alternated with a space-average luminance-matched blank screen at a rate of 5Hz. Each phase of grating or blank field (labeled t_1, t_2, etc.) lasted six video frames. During each 10-second trial, spatial frequency of grating was systematically increased (i.e. size of bars decreased) in a series of 10 equal linear steps.*

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way. CVI was diagnosed when vision was clearly subnormal from a clinical standpoint, in the context of a normal, or nearly normal eye examination. This clinical diagnosis of reduced visual acuity was never subtle in this group of children with CVI. Pupillary reactions were normal. In three cases, children showed reduced vision and also had signs of anterior visual pathway disease (Table I). These children were included in the study because the level of vision loss greatly exceeded that which would have been expected solely from the eye findings. In all children, there was a clear and plausible etiology for a diagnosis of CVI.

Table I lists participants' medical history and the etiology of the CVI diagnosis. The suspected cause of CVI was hypoxic–ischemic encephalopathy in 21 of the 35 children. Other

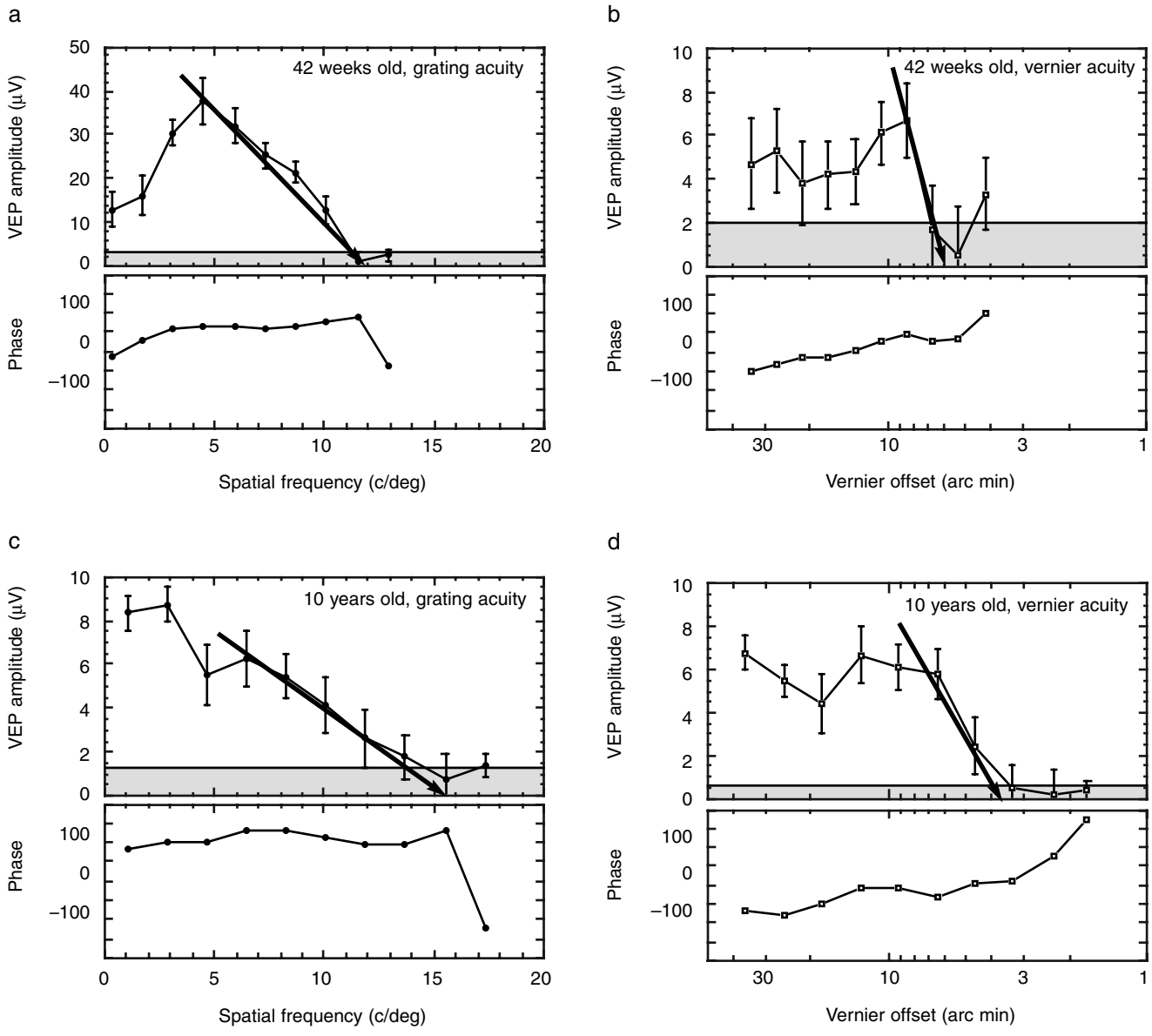

Figure 2: *Sample visual evoked potential (VEP) datasets from two participants. In each of four pairings of graphs, the top portion (a &b) shows amplitude (in microvolts) of 5Hz visually evoked response as a function of swept stimulus parameter (spatial frequency or offset size). Bottom (c &d) shows phase of evoked response as a function of each stimulus parameter. Grey-shaded area at bottom of each amplitude graph shows level of background (non-visual) EEG, estimated as the average of amplitude at 4Hz and 6Hz. Each amplitude function is significantly greater than the background EEG at beginning of each trial, when the varied spatial parameter is most visible. As the spatial parameter is swept into a less visible range, VEP amplitude decreases until by end of trial it has fallen to level of the non-visual background EEG. Threshold is estimated by extrapolating along falling portion of amplitude function (arrows) to zero microvolts. Phase graphs (lower sections) provide information about temporal synchronization between visual stimulus and evoked response. In portions of data collection when a significant evoked response is present, phase is relatively flat, whereas the lack of an evoked response is indicated by random phase.*

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causes were: encephalitis (n=3), trauma (n=1), partial complex mitochondrial disorder (n=1), postnatal stroke (n=1), near drowning (n=1), porencephalic cyst (n=1), meningitis (n=1), hydrocephaly (n=1), cardiac arrest (n=1), cerebellar atrophy (n=1), and two cases were of unknown origin. Twenty participants were taking seizure medications, and three had secondary signs of anterior visual pathway disease.

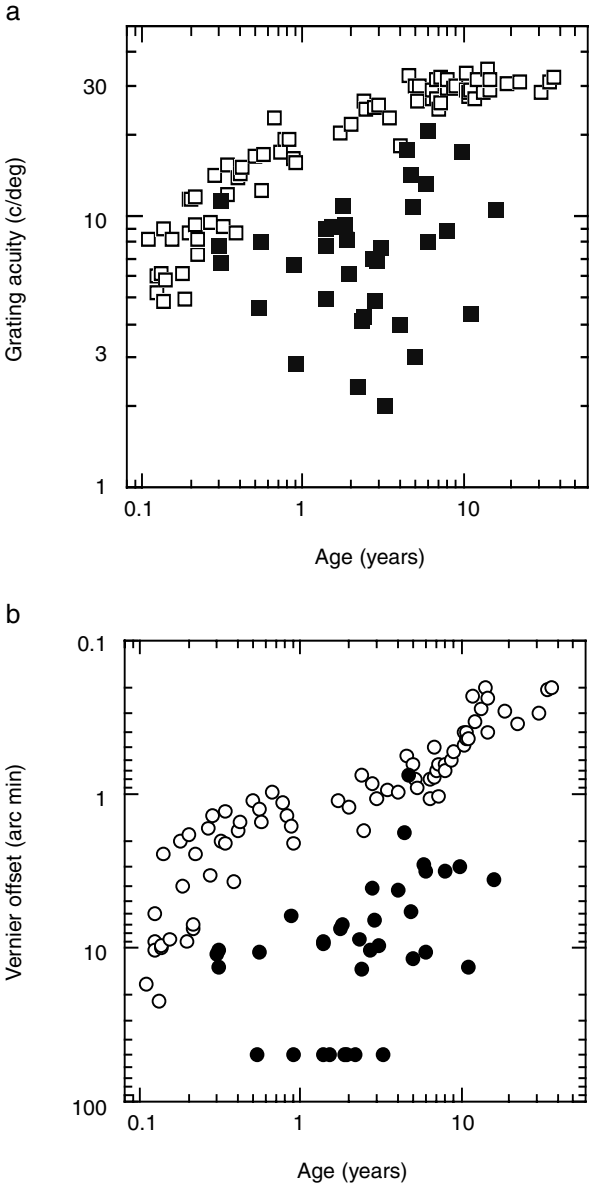

Figure 3: *Scatterplots of (a) grating acuity and (b) vernier acuity as a function of age in individuals with and without cortical visual impairment. Open symbols show data from participants with normal vision (from Skoczenski and Norcia 2002), and filled symbols show data from participants with cortical visual impairment. In (b) vernier acuity graph, filled symbols along bottom of graph represent those participants in whom an evoked response to vernier offset appearance/disappearance could not be measured.*

TESTING PROCEDURE AND DATA ACQUISITION

Gold cup electrodes (Astromed-Grass Instruments, West Warwick, Rhode Island, USA) were positioned on the scalp according to the International 10-20 system, at O_1, O_z, O_2: each referenced to C_z (Regan 1989). Each electrode site was prepared with Omni Prep, and electrodes were affixed with water-soluble 10-20 conductive paste and a soft headband.

Each participant was tested on both grating acuity and vernier acuity. Schematic drawings of the visual stimuli are shown in Figure 1. To measure grating acuity, we presented sinewave gratings that appeared and disappeared at a fixed temporal frequency of 5Hz, while spatial frequency changed in linear steps from low to high during 10-second trial presentations. To measure vernier acuity, square wave gratings were used, with vernier offsets appearing and disappearing within each grating bar at a fixed temporal frequency of 5Hz. Vernier offset size changed from large to small in logarithmic steps over each 10-second trial presentation. Both stimulus types had a Michelson contrast of 80%, and a mean luminance of 100cd/m^2.

One goal of the sweep VEP is to observe changes in the amplitude of the evoked response as the visual stimulus changes from above to below the sensory threshold, as in Figure 2. We attempted to choose sweep ranges that bracketed visual thresholds for each type of acuity in each child. The exact range of the swept parameter (spatial frequency for grating acuity; offset size for vernier acuity) was determined by taking into account information such as the child's age, and any existing information

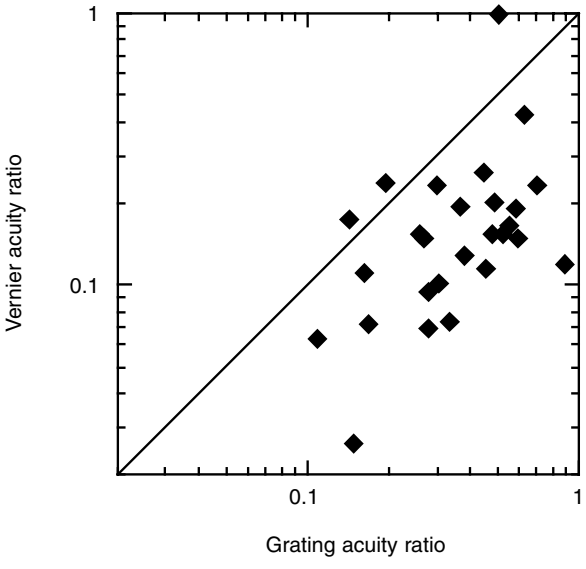

Figure 4: *Relation between vernier acuity ratio and grating acuity ratio for 27 individuals with cortical visual impairment (CVI). Each acuity ratio shows an individual's acuity relative to mean of age-matched participants with normal vision, therefore a ratio of 1 would indicate normal acuity. If vernier acuity and grating acuity were equally degraded in those with CVI, acuity ratios in this graph would tend to fall along the diagonal line. Actual spread of acuity ratios indicates that vernier acuity is more degraded (i.e. has lower ratios) compared to grating acuity.*

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about the child's visual abilities. If significant VEP responses to the smallest offset or highest spatial frequency were shown, the condition was retested with a more difficult sweep range (two children). Retesting with a new sweep range also was performed if there was no significant evoked response across the original sweep range (nine children).

Participants sat on a parent's lap or in their own wheelchair during testing. An experimenter initiated each trial, observed the child's fixation, and used singing and small bells dangled in front of the monitor to maintain the child's attention. If the child looked away from the stimulus monitor, the trial was interrupted, and was resumed when the child's gaze returned to the monitor.

The EEG was digitized to an effective 14 bits at 397Hz over an amplifier pass-band of 1 to 100Hz (–6db points) using a Spectral Innovations SIAD-8C acquisition card (Spectral Innovations, Santa Clara, CA, USA) and a Spectral Innovations M/BA digital signal processor hosted by an Apple Macintosh Quadra 800 computer. We recorded amplitude and phase of the VEP as a function of spatial frequency or offset size. Figure 2 shows amplitude and phase data from two participants. Thresholds were estimated by extrapolating the first harmonic amplitude functions to zero microvolts (see Fig. 2), taking into account phase and error statistics. Each threshold was based on a vector average of 5 to 15 trials.

Results

We obtained measures of both types of acuity (vernier and grating acuity) in 27 of 35 patients. In the remaining eight patients, grating acuity was measurable, but the evoked potential to vernier offset stimulation was either undetectable, or the evoked signal strength was insufficient for a threshold extrapolation. Therefore, no vernier acuity estimate was obtained in these eight participants.

Figure 3 shows threshold data for grating acuity (3a) and vernier acuity (3b) as a function of age in CVI patients, and in children with normal vision. Data from participants with normal vision are taken from Skoczenski and Norcia (2002), and were collected with identical test parameters on the same apparatus. Across all ages tested, both vernier and grating acuity were lower in patients with CVI compared with children with normal visual experience. In Figure 3b, data for those patients with CVI with no vernier acuity estimate are shown as symbols along the bottom of the graph, at the appropriate age value on the *x*-axis.

In order to quantify each child's spatial vision deficits, we calculated two acuity ratios, one for grating acuity and the other for vernier acuity. Each acuity ratio was calculated by dividing the acuity of a patient with CVI, with the mean acuity from age-matched children with normal vision, using data from Skoczenski and Norcia (2002). An acuity ratio of 1 would indicate normal vision. Figure 4 plots vernier acuity ratio as a function of grating acuity ratio, and shows a within-participant comparison of the two ratios for the 27 participants who had measures on both acuity types. If vernier and grating acuity were equally low in those with CVI, the symbols on this graph would fall along the diagonal. Instead, the majority fall in the lower portion of the graph, illustrating that grating acuity ratios were closer to normal (i.e. 1) than vernier acuity ratios, and that vernier acuity was more severely degraded than grating acuity in most children with CVI. Individual acuity ratios are listed in Table I.

The bar graph in Figure 5 summarizes the acuity ratio data from Figure 4. The mean vernier acuity ratio in these patients with CVI was 0.196, while the mean grating acuity ratio was 0.395. This difference is statistically significant (t=3.99; df=26; p=0.0006).

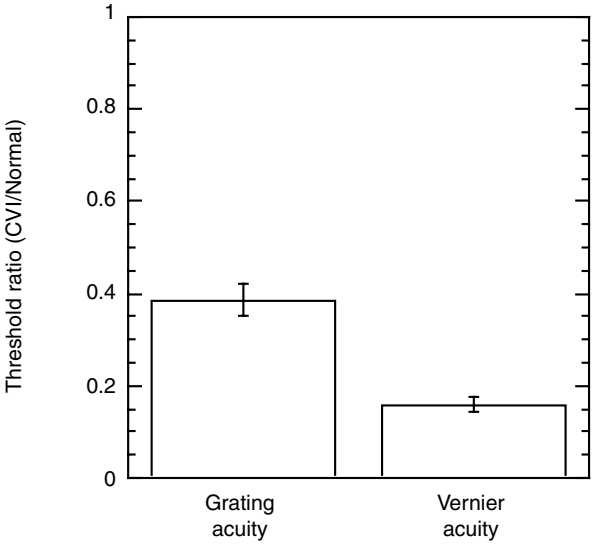

Figure 5: *Mean of individual acuity ratios that were plotted in Figure 4. Grating acuity ratio of 0.395 is significantly higher than vernier acuity ratio of 0.196. CVI, cortical visual impairment.*

Discussion

Many of the leading causes of vision impairment in children can be treated, with the results of treatment monitored. In retinopathy of prematurity, for example, significant, favorable advances in the outcome of advanced disease have occurred because retinal anatomy and topography can be observed after experimental intervention (Cryotherapy for Retinopathy of Prematurity Cooperative Group 1988a,b). The same is true for congenital cataracts, formerly a leading cause of vision impairment but now uncommonly a cause of blindness in children in Western countries. In cataracts, intervention and treatment effects can be studied and directly observed. But research on prevention, treatment, and rehabilitation of CVI has been hampered, in part because there is no way to directly visualize or accurately measure the effects of intervention on the cerebral visual pathways.

The children in this study are likely to represent more severe cases of CVI. They were referred by pediatric ophthalmologists and/or the Blind Babies Foundation of Northern California. The diagnosis of CVI was confirmed by one of the authors (WVG) and was based on clinical signs of reduced visual acuity, normal pupil reactions, and confirmatory history of central nervous system disease or injury.

Children with CVI in this study showed diminished grating and vernier acuity compared with children with normal vision, but the vernier acuity threshold was disproportionately affected. Specifically, vernier acuity was lower, on average,

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by a factor of 6.5, while grating acuity was lower, on average, by a significantly lower factor of 2.5 compared with age-matched participants with normal vision. If vernier and grating acuity were limited by the same mechanisms in the visual pathway, we would expect them to be degraded in equal amounts in these patients. The selective deficit for vernier acuity in these patients with cortical damage suggests that vernier acuity is more strongly limited by cortical processing than is grating acuity. This finding converges with studies of normal adults, in whom vernier acuity is a form of hyperacuity (Westheimer 1975). Hyperacuity may be attained by the reconstruction of the visual image in later stages of visual processing, e.g. cortical stages.

The vernier selective deficits that we observed suggest that vernier acuity may be more accurate than grating acuity in quantifying visual deficits in individuals with CVI. This finding is consistent with studies of adults with amblyopia, another visual deficit where cortical processes play an etiologic role: vernier acuity is a better predictor, compared with grating acuity, of optotype acuity in amblyopic eyes (Levi and Klein 1982, 1985; McKee et al. 2003). The current data also converge with behavioral studies of infants with developmental delay (Holmes 1996) and intrauterine growth retardation (Stanley et al. 1989) who also may have selective deficits in vernier acuity.

The sensitivity of the vernier acuity measure suggests that it will be valuable in determining appropriate levels of visual stimulation for young patients with CVI, and in evaluating the effects of their treatment and education. A behavioral measure might have been more difficult with most of the patients with CVI tested in the present study, as a majority of them were pre- or non-verbal and had compromised motor responses. The lack of verbal or motor abilities makes behavioral measures of vision in these children difficult. The VEP technique may be the most accurate and time-efficient way to measure functional vision in people with CVI, and may be the only response measure that can be used with infants and children with multiple disabilities.

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References

Birch EE, Bane MC. (1991) Forced-choice preferential looking acuity of children with cortical visual impairment. *Dev Med Child Neurol* **33:** 722–729.

Birch EE, Swanson WH, Wang YZ. (2000) Infant hyperacuity for radial deformation. *Invest Ophthalmol Vis Sci* **41:** 3410–3414.

Carkeet A, Levi DM, Manny RE. (1997) Development of vernier acuity in childhood. *Optom Vis Sci* **74:** 741–750.

Cioni G, Fazzi B, Coluccini M, Bartalena L, Boldrini A, van Hof-van Duin J. (1997) Cerebral visual impairment in preterm infants with periventricular leukomalacia. *Pediatr Neurol* **17:** 331–338.

Cioni G, Fazzi B, Ipata AE, Canapicchi R, van Hof-van Duin J. (1996) Correlation between cerebral visual impairment and magnetic resonance imaging in children with neonatal encephalopathy. *Dev Med Child Neurol* **38:** 120–132.

Cryotherapy for Retinopathy of Prematurity Cooperative Group. (1988a) Multicenter trial of cryotherapy for retinopathy of prematurity: preliminary results. *Pediatrics* **81:** 697–706.

Cryotherapy for Retinopathy of Prematurity Cooperative Group. (1988b) Multicenter trial of cryotherapy for retinopathy of prematurity. Preliminary results. *Arch Ophthalmol* **106:** 471–479.

Cryotherapy for Retinopathy of Prematurity Cooperative Group. (1996) Multicenter trial of cryotherapy for retinopathy of prematurity. Snellen visual acuity and structural outcome at 5½ years after randomization. *Arch Ophthalmol* **114:** 417–424.

Dobson V, Quinn GE, Siatkowski RM, Baker JD, Hardy RJ, Reynolds JD, Trese MT, Tung B. (1999) Agreement between grating acuity at age 1 year and Snellen acuity at age 5.5 years in the preterm child. Cryotherapy for Retinopathy of Prematurity Cooperative Group. *Invest Ophthalmol Vis Sci* **40:** 496–503.

Dobson V, Teller DY, Lee CP, Wade B. (1978) A behavioral method for efficient screening of visual acuity in young infants. I. Preliminary laboratory development. *Invest Ophthalmol Vis Sci* **17:** 1142–1150.

Good WV, Hou C, Norcia AM. (2001a) Interocular differences for sweep VEP vernier and grating acuity in amblyopia. *Invest Ophthalmol Vis Sci* **42:** s3945.

Good WV, Jan JE, Burden SK, Skoczenski A, Candy R. (2001b) Recent advances in cortical visual impairment. *Dev Med Child Neurol* **43:** 56–60.

Good WV. (2001) Development of a quantitative method to measure vision in children with chronic cortical visual impairment. *Trans Am Ophthalmol Soc* **99:** 253–269.

Good WV, Jan JE, DeSa L, Barkovich AJ, Groenveld M, Hoyt CS. (1994) Cortical visual impairment in children. *Surv Ophthalmol* **38:** 351–364.

Holmes JM. (1996) Comparison of grating and Vernier acuity in infants with developmental delay. *J Pediatr Ophthalmol Strabismus* **33:** 31–34.

Holmes JM, Coates CM. (1994) Assessment of visual acuity in children with trisomy 18. *Ophthalmic Genet* **15:** 115–120.

Johnson-Kuhn J. (1995). The Blind Babies Foundation: registry of early childhood visual impairment in central and northern California. In: Bernas-Pierce J, editor. *The Hoyt-Akeson Selected Readings in Pediatric Ophthalmology*. San Francisco: Blind Babies Foundation.

Levi DM, Klein SA. (1982) Hyperacuity and amblyopia. *Nature* **298:** 268–270.

Levi DM, Klein SA. (1985) Vernier acuity, crowding and amblyopia. *Vision Res* **25:** 979–991.

Levi DM, Manny RE, Klein SA, Steinman SB. (1983) Electrophysiological correlates of hyperacuity in the human visual cortex. *Nature* **306:** 468–470.

McDonald MA, Dobson V, Sebris SL, Baitch L, Varner D, Teller DY. (1985) The acuity card procedure: a rapid test of infant acuity. *Invest Ophthalmol Vis Sci* **26:** 1158–1162.

McKee SP, Levi DM, Movshon JA. (2003) The pattern of visual deficits in amblyopia. *J Vision* **3:** 380–405.

Regan D. (1989) *Human Brain Electrophysiology: Evoked Potentials and Evoked Magnetic Fields in Science and Medicine*. New York: Elsevier.

Shimojo S, Held R. (1987) Vernier acuity is less than grating acuity in 2- and 3-month-olds. *Vision Res* **27:** 77–86.

Skoczenski AM, Norcia AM. (1999) Development of VEP Vernier acuity and grating acuity in human infants. *Invest Ophthalmol Vis Sci* **40:** 2411–2417.

Skoczenski AM, Norcia AM. (2002) Late maturation of visual hyperacuity. *Psychol Sci* **13:** 537–541.

Stanley OH, Fleming PJ, Morgan MH. (1989) Abnormal development of visual function following intrauterine growth retardation. *Early Hum Dev* **19:** 87–101.

Steinkuller PG, Du L, Gilbert C, Foster A, Collins ML, Coats DK. (1999) Childhood blindness. *J AAPOS* **3:** 26–32.

Westheimer G. (1975) Visual acuity and hyperacuity. *Invest Ophthalmol* **14:** 570–572.

Zanker J, Mohn G, Weber U, Zeitler-Driess K, Fahle M. (1992) The development of vernier acuity in infants. *Vision Res* **32:** 1557–1564.

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