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Review Article

Visual Rehabilitation of Children with Visual Impairments

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ABSTRACT. Visual rehabilitation, consisting of visual stimulation and visual training, is a common practice in the education of children with visual impairments. Ferrell and Muir have stated that scientific research into the effects of visual stimulation and training is ambiguous and that therefore stimulation and training should be abandoned. The support for this statement is reviewed by describing the scientific relevance and plausibility of the aims and presuppositions of visual stimulation and training programs as well as the results of 10 empirical intervention studies. The review results are in strong agreement with the claim of Ferrell and Muir to abandon noncontingent visual stimulation. It is hypothesized that it is possibly counterproductive for the adaptive functioning of the brain to show strong visual stimuli in artificial surroundings, which are noncontingent on the behavior of the child. Training of visual functions seems fruitful whenever skills that are ecologically valid and adapted to the individual needs and task demands of the child are trained. However, the empirical evidence is still too sparse to draw convincing conclusions. There is an urgent need for good randomized controlled trials with dependent variables that are relevant to everyday life. *J Dev Behav Pediatr 27:493–506, 2006.* Index terms: *visual stimulation, visual training, blindness, visual impairment, rehabilitation, restoration of vision.*

After the first publications of Nathalie Barraga in the 1960s, vision rehabilitation of visual functioning became common practice in teaching and in early intervention programs for children with visual impairments. These rehabilitation programs replaced the prevailing philosophy of ‘‘sight saving,’’ because it became apparent that vision would not be further impaired by use.[1] Later, besides the efficient use of vision, vision itself also became the goal of training. However, in 1996, Ferrell and Muir[2] called for an end to vision stimulation as a goal in itself. Since Ferrell and Muir[2] found the results of scientific research on vision stimulation ambiguous, they considered vision stimulation only useful as a means of reaching other educational goals. However, the critique of Ferrell and Muir was problematic in several ways. First, they did not define what vision stimulation training exactly is. It is not clear whether they meant to stop all kinds of visual rehabilitation programs or just a few. It is also possible that they only criticized programs that performed poorly.[3,4] Second, no empirical studies were mentioned in their article to back up their argument of scientific ambiguity. Lastly, in contrast to visual stimulation, Ferrell and Muir seemed to accept visual training as being useful. But how strong is the case for visual training? In this review, visual rehabilitation will be defined, the available empirical evidence will be given, including that published after 1996, and the evidence for visual training will be examined.

What Ferrell and Muir should have done is to define what visual stimulation is. *Visual rehabilitation* entails all kinds of interventions aimed at recovery of visual abilities, improvement of visual functioning, and coping with visual disabilities. Common terms in visual rehabilitation are vision stimulation, visual stimulation, and visual training. These terms are used inconsistently and interchangeably in the literature. Two distinctions are important: the difference between vision and visual stimulation and the

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difference between stimulation and training. *Vision stimulation or training* is aimed at improving the visual system by enhancing and changing its anatomy and physiology. In contrast, *visual stimulation or training* is concerned with stimulating the development of children with visual impairments by means of visual material, with the goal of improving visual functioning. In the latter, the behavioral achievements of the children are evaluated, not the possible organic or physiological changes.

In stimulation programs, stimuli are presented noncontingently on the behavior of the child, whereas in training programs contingent presentation of stimuli is used. Noncontingent stimuli are presented without a direct link to the behavior of the child with regard to timing and intensity of the stimulation. In stimulation programs, typical stimuli are mobiles, lights, and brightly colored materials. These stimuli are often presented above the crib, bed, playpen, or in a ''snoezel'' room. The presumption is that visual stimulation is inherently reinforcing and will therefore elicit responses from the child, for instance, fixation. Visual stimulation is most often applied to children who hardly respond to normal visual impressions, whereas visual training is applied to children who show slightly more visual attention,[5] to teach them to make functional use of their sense of sight.[6] Behaviors aimed at these training programs are, for instance, efficient fixating, visual following of moving targets, and improving eye-hand coordination. Operant conditioning paradigms are often used to teach these behaviors. For instance, with the help of physical guidance or tactile or verbal prompts children are forced to watch to critical aspects of a task or an event for a certain amount of time to promote visual fixation or scanning. Subsequently, successful attempts are reinforced.

According to Ferrell and Muir,[2] the results of scientific research were too ambiguous to justify the continuation of vision stimulation or visual skills training programs. Furthermore, they stated that vision stimulation programs have a negative impact both on the normalization and integration of children with visual impairments in the mainstream society and on their self-esteem. This negative impact is the result of attempts to train the child to perform a task that he or she is not able to perform because of the visual impairment, along with the fact that the training hinders normal integrated functioning in society. Ferrell and Muir[2] considered the noncontingent exposure of strong visual stimuli, such as flashing lights, as counterproductive to normalization and integration, because children normally do not encounter such stimuli. Given the ambiguous scientific results and the negative impact on normalization and self-esteem, Ferrell and Muir[2] concluded that time should better be devoted to other educational goals than to noncontingent vision stimulation. Given the estimated prevalence of visual impairment in children younger than 15 years in the United States (1.2%) and in the world (2%),[7] this suggestion might affect the education and treatment of thousands of children.

However, when vision stimulation programs are effective, they could provide a strong argument against the presumed negative impact on normalization and self-esteem. One could state that the end justifies the means. Crucial in this regard is the effectiveness of vision stimulation, which ought to be supported by empirical research.

ENHANCING VISION IN CHILDREN

Professionals that are typically involved in these programs are, for example, teachers of the visually impaired, early interventionists, optometrists, psychologists, and occupational therapists. In the literature, different terms and approaches are used for rehabilitation of vision. To mention a few: visual or vision stimulation, visual training, visual therapy, visual skills programming, visual-motor training, optical aids adaptations, ergonomic interventions, instruction in the use of vision, visual environment management training, visual skill training, optometric vision training, and restoration of vision. Several other intervention strategies are used in vision rehabilitation programs. Some of these interventions are also called visual training. To avoid confusion, these related but distinct activities in vision rehabilitation will be described briefly.

Fitting Optical Devices

Optimal correction for refraction errors with glasses or contact lenses is a prerequisite for visual stimulation and training programs. A common finding in clinical practice is that advanced optical devices, such as magnifiers or electronic vision enhancement systems (eg, a closed-circuit television system), are usually not applicable to children. Mental and physical disabilities often interfere with appropriate use of these devices, and the usefulness of optical devices is rather task specific.[8] Moreover, many optical devices are designed for near-vision tasks. But one can argue about the number of near-vision tasks a young child has to perform for which accommodation is inadequate, and while optical devices would be helpful, shortening the viewing distance would not. For children, optical devices are often a nuisance, and they sometimes are rejected because of their appearances.[9] For the same reasons, children sometimes even refuse to wear corrective glasses and lenses. Surprisingly, the literature search revealed no empirical studies on teaching children to adapt to corrective glasses and lenses.

Ergonomic Interventions

Ergonomic interventions, for instance, by adapting the lighting, color, contrast, and movement of visual stimuli and also adapting the nature and intensity of the visual stimuli, constitute the second form of visual rehabilitation.[10,11] According to Corn[12] and Hall and Bailey[13] these ergonomic and adaptive changes to visual stimuli are also a kind of visual stimulation. After prescription of corrective glasses, it is often the second step in a visual stimulation program. In other programs, these changes comprise the total intervention program. An example of the latter is the intervention of Potenski,[14] in which black light and fluorescent materials were used to train deaf-blind children in putting together pieces of a puzzle.

Instruction to the Use of Visual Skills

This strategy entails instruction in the use of compensating strategies in case of uncorrectable diminished visual abilities.[15] This could be accomplished by teaching scanning techniques for persons with diminished fields of vision or teaching tactile approaches in situations in which vision is unreliable or inefficient.

Optometric and Orthoptic Interventions

Optometric and orthoptic interventions, also called vision therapy or optometric vision training, are designed for problems in acuity, fixation, accommodation, vergence, binocular vision, ocular motility, eye-hand coordination, strabismus, amblyopia, nystagmus, and myopia. The orthoptic techniques used are eye exercises and biofeedback. In a recent review, eye exercises were found to be useful in cases of convergence insufficiency and, to a lesser extent, in difficulties with stereoscopic skills.[16] No support was found for the use of eye exercises in the remainder of the areas reviewed. In biofeedback training, unconscious physiological functions are trained to submit to voluntary control by chaining them to auditory or visual stimuli. Biofeedback has been used effectively in the treatment of myopia and to enhance visual acuity, color vision, and contrast sensitivity in adults.[17–19] With the exception of treatment of amblyopia and strabismus, eye exercises and biofeedback are not applicable to or not effective with children who have severe visual impairments. Possibly as a result, the optometric and orthoptic literature is devoid of vision-training studies in children with severe visual impairment.

Orthoptic training procedures are also frequently used in the treatment of dyslexia and learning disorders in normally sighted children. With regard to dyslexia, reading problems are sometimes thought to be caused by eye movement disorders. However, eye movements reflect the cognitive processes associated with understanding the text and are not the cause of dyslexia.[20] For dyslexics, this could be problems in language comprehension or the processing of visual spatial information. Up until now, current research has failed to implicate vision as a source of dyslexia and learning disability.[16,21–25]

Teaching Visual Routines

The teaching of visual routines is done by persons who are closely related to or who take care of children with visual impairments.[26] Visual routines are behaviors that facilitate the intake of information by the child with visual impairments. These are routines for making contact and for sustaining interaction with visually impaired persons. Techniques in these routines concern timing, pace, viewing distance, spatial position when speaking to the child, making use of floodlights, avoiding back lighting, and so forth. In a way, these routines are ergonomic adaptations that promote the intake of information. At the same time, they also have important pedagogical and social implications.

Fitting Visual Prostheses

Visual prostheses are studied as an alternative way to restore useful vision to blind people. Visual prostheses are named according to their locations: cortical, optic nerve, subretinal, or epiretinal prosthesis. Implantation of visual prosthesis is mostly done on an experimental basis with animals and, on a small scale, with human adults. So far, children are not provided with these kinds of visual prostheses.[27–29] The benefits to the general population of visually impaired persons are also still limited.

Visual Restitution Training

Visual field disorders are treated by visual restitution training. In daily training sessions, luminous stimuli are presented at random locations in a previously defined training area on a dark computer screen. Visual field recovery was found with different methods of quantitative perimetry. Rehabilitation has also been described for color-vision deficits, space perception, visual agnosia, and central scotoma.[30–34] However, all these studies refer to otherwise healthy adults who acquired blindness, whereas visually impaired children often have congenital visual impairment or blindness. The results of visual restitution training seem to depend on the kind of perimetry used,[36] are often rather limited in degree,[34] and are task dependent. Generalization to everyday life is difficult.[37] There is also serious doubt about whether results from visual restitution training are artifacts resulting from a difficulty to control fixation.[38,39]

PRESUPPOSITIONS AND GOALS IN VISUAL REHABILITATION PROGRAMS

Everyday Experience Is Not Enough

The basic assumption underlying all visual rehabilitation programs is that everyday visual impressions are not sufficient for the child to see and look or to promote appropriate visual development.[40] But why is it that an artificial stimulus applied for an hour per day would be more effective than the incredibly rich repertoire of natural light patterns that stimulate the retina under normal everyday circumstances?[38] The answer, according to several authors, is that children with visual impairments do not learn to use their vision automatically and spontaneously.[41–44] As a result, ''Theoretically, the vision of severely visually impaired babies may remain suboptimal throughout the early months and their full potential for vision may never be achieved.''[26(p320)] Sonksen and Dale[45] hypothesized that the quality of visual input in infants with very low levels of vision is too poor to awaken interest in the environment and that active attention is essential for optimal visual development. Because everyday visual impressions are thought to be insufficient, stimuli in visual stimulation and training programs are often very salient to contrast them with the background.[13,46] From this suggestion, it followed that appropriate stimuli include high-contrast gratings with a large range of spatial frequencies and orientations.[47] However, according to Glass,[48] the use of strong stimuli, such as black and white patterns, is inappropriate. Its popular use stems from the well-known observations that neonates attend to black and white patterns in preference to gray nonpatterned surfaces. She continues with, ‘‘but an infant’s ability to respond to a type of stimulation does not necessarily mean that he or she should be stimulated in that matter (e.g., young infants will even stare at light bulbs).’’[48(p9)] According to Glass, increased attention for black and white stimuli is a less mature response for an immature brain that becomes increasingly responsive to specific characteristics of the visual environment.[48] It is also questionable whether strong visual stimuli, with which the children have no physical contact, will have any meaning for them. Showing visual stimuli to visually impaired children in a noncontingent way poses another problem. Noncontingent exposure of visual stimuli might lead to passive seeing (i.e., excitation of cells in the visual pathways and cortex without triggering the child to look).[26] This might even result in a kind of learned helplessness.[49,50]

Prevent Visual Deprivation

Visual rehabilitation programs are also justified by the fear that refraining from stimulating the child will lead to visual deprivation.[47,51,52] This fear is based on animal studies[11,26,47,51] and on studies of humans who recovered their vision after lengthy ocular blindness. Experience from humans deprived of vision at an early age indicates that recovery of vision after late correction of the abnormalities of the eye is rudimentary and often functionally not very useful.[53,54]

Prolonged deprivation, by occlusion of 1 or 2 eyes or by rearing the animal in atypical environments or in the dark, resulted in distorted visual development, amblyopia, or even blindness.[53] Rearing rats in impoverished environments resulted in decreased cortex thickness, whereas enriching the environmental conditions in which the animals were confined could alter both the chemistry and anatomy of the cerebral cortex and, in turn, improve the memory and learning ability of the animal.[55]

Intervene in the Sensitive Period

Analogous to eye occlusion as treatment of amblyopia, it is presumed that visual stimulation ought to start early to be effective.[56] The first reason for this lies in the way the brain develops. The first stage of brain development consists of relatively rapid change in which the brain is very receptive to extrinsic environmental experiences. To gather environmental information, sensory systems ought to function optimally. Normally the sensory systems become established and fully mature during the above-mentioned stage of rapid changes in brain development. Disturbance in development of the sensory systems will, therefore, also affect brain development.[57] A second reason comes from animal studies that have shown that there is a sensitive or critical period for visual development. Neuronal connections and cortical response properties are especially susceptible to visual experience during the sensitive period.[57–59] Eye closure in kittens during the sensitive period for as little as 3 to 4 days leads to a sharp decline in the number of brain cells that can be driven from both eyes, as well as an overall decline in the relative influence of the previously closed eye.[60] This sensitive period for visual development in humans coincides with the first years of life, with the fastest developments taking place between 0 and 1 year.[5,26,46] After this period, the consequences of visual deprivation are irreversible.[47] Results of visual rehabilitation studies after recovery of long lasting blindness in humans seem to confirm this conclusion. After recovery, the participants stayed behaviorally blind, although visual evoked potentials (VEP) measurements showed that the primary visual cortex was functioning. Training results were small and functionally not always very useful.[54,61] Long after operations, many patients complained about insecurity and often closed their eyes to be better able to perform daily activities.[62–64]

Powell[11] postulated the idea that the sensitive period for visual development might be prolonged for children without light perception (LP) and for adults with visual impairment and additional severe impairments. Neurological maturity rather than chronological age seems to be crucial in this respect. Studies on healthy premature infants confirm this conclusion. Extra visual experience does not seem to enhance visual competencies in premature infants. Neurological maturation rather than chronological age corresponds with visual development.[65]

The above 2 presuppositions were partly based on animal studies. However, extrapolations from animal studies to human children are problematic. First, because most of these animals were blinded or visually deprived after birth, often by lid closure, animal studies are probably somewhat more comparable to studies of humans who recovered their vision after long lasting ocular blindness, than to studies of human children with prenatal or congenital blindness or visual impairment. In human children, visual impairment as a result of damaged or malfunctioning brain cells is nearly always accompanied by neurological abnormalities that might also affect other body functions.[66] These children are incomparable with animals in which visual impairment resulted because of postnatal deprivation or an artificially induced trauma to altogether normal eyes and brain cells and not because of prenatal damage. Up until now, there are no animal studies known in which the animals were deprived of vision prenatally. Second, pathogenesis and recovery from deprivation follows different tracts in different species. For instance, visual deprivation appears to affect more strongly the behavior of monkeys than that of cats.[53] Third, comparisons between brain development and behavior of man and animal are fraught with difficulty. Not only is it not possible to control all of the experimental variables at work in humans, but the diversity and complexity of human experience militates against designing experiences comparable to those used with lower animals.[63] Fourth, techniques used in animals are often invasive or terminal and therefore not used in humans. As a result, much of what we know about the human brain is based on inference or extrapolation from studies of other species.[67] However, species show analogies and differences in development. As a result, there is large variation. The nature of this variation is not one of degree from a common type but one of substantial difference.[67] The sharing of features between species does not mean, however, that they are identical or that extrapolations can be made more easily. Homologies with cortical areas in monkeys or other animals, therefore, remain tentative.[68]

Brain Plasticity

The next presumption is that the anatomy, physiology, and neural base of the visual system changes as a result of visual stimulation.[26,40,47] This improvement does not consist of origination of new cells but of increased branching of axons and dendrites and an increase in response properties of surviving nerve cells. These processes are thought to stimulate the origination of alternative neurological tracts[13,69] and to restructure brain processes. Also, cells will be used for vision while they were predestined to fulfill other functions.[47,57]

There is not much empirical evidence for the presupposition that visual stimulation results in anatomical and physiological changes. Only Werth and Seelos[70] found changes in a functional magnetic resonance imaging study on vision restoration in 2 children with cerebral blindness. In other studies, the anatomical and physiological changes were mostly not assessed directly, but changes were only presumed to be present based on the fact that some behavioral changes were noted as a result of visual stimulation. As a result, it is indeterminate whether anatomical and physiological changes truly occur in humans. With regard to brain plasticity, it may even be harmful to try to change the visual system of the brain, because visual stimulation might interfere with brain reorganization and specialization. Several studies, including functional magnetic resonance imaging studies, have shown that the visual cortex is more modifiable than previously considered. In congenitally blind persons occipital activation was found during tasks on verbal memory, verb generation, reading Braille, and sensory discrimination of tactile or auditory stimuli.[71–76] In one study, this shift was only found in subjects who lost their sight before the age of 16 years and not in subjects who either lost sight after the age of 16 years or who were sighted.[77] These results implicate that visual rehabilitation with young children might conflict with spontaneous adaptations to blindness, which is cross-modal plasticity of the primary visual cortex. However, up until now, no studies are known that have addressed this issue.

Change Visual Behavior and Attention

Some authors explicitly stress that behavior instead of brain functions change after visual rehabilitation.[12,26] Accordingly, behavioral change should be the topic of visual rehabilitation. They mention improving visual functions, optimizing residual vision, especially visual acuity,[78] and improving visual-motor functions.[40] According to Sonksen et al[26] ''looking'' and not ''seeing'' should be stimulated. Seeing (i.e., light sensation and transportation to the brain) is not affected by visual stimulation or training. In contrast looking, that is paying attention to what is seen, is stimulated or trained. Looking is an acquired skill based on previous experiences and, as such, looking can also be stimulated and trained.[79,80] From neurophysiological studies, it is clear that processing within the extrastriate areas in the cortex (i.e., the primary areas responsible for vision) is strongly modulated by selective attention.[68] Some authors therefore see attention as the crucial factor in looking behavior. Visual rehabilitation might affect visual attention more than visual functioning itself.[81,82] The role of attention is quite explicit in the VAP-CAP program of Blanksby.[83] According to her, visual functioning is dependent on visual capacities, visual processing, and visual attention. Increased awareness of visual stimuli enables the visually impaired child to use the stimuli for acting, reacting, and responding.[15] As a consequence, cognitive and personal functioning is also affected.[13] Examples are improvement in sound localization, cognitive development, and motor skills.[26]

Follow Normal Visual Development

With regard to the content of visual rehabilitation programs, most program developers presuppose that the development of visual functioning of children with visual impairment follows the same hierarchical order as the visual development of typically developing children.[1,12,26,42,84–86] According to Corn,[12] this assumption is taken as a starting point, because of a lack of alternative points of view. Most authors, however, state that persons with visual impairment might have deviations from the normal order of visual development.

INTERVENTION PROGRAMS

Best Practice Programs in Visual Rehabilitation

Examples of programs on visual stimulation and training that are widely used by early interventionists working with children with visual impairments are *Look and Think*,[87] *Look at Me*,[43] *Bright Sights: Learning to See*,[41] *Learning to Look*,[80] *Preschool Vision Stimulation: It's More Than a Flashlight*,[88] vision program from *The INSITE Model*,[89] *Vision for Doing*,[90] ''Cortical Visual Impairment, Presentation, Assessment, and Management,''[52] *VAP-CAP*,[91,92] and *Low Vision*.[93] They can also be found in descriptive articles about visual and early intervention programs[10,11,15,51,69,94–99] and in unpublished documents and nonrefereed forums. A common characteristic of the above-mentioned programs is their lack of empirical data on reliability, validity, efficiency, and effectiveness. Consequently, this occurs at the moment their status is that of best practice.

Empirical Stimulation Programs

A search was performed of the following databases: Index Medicus (MEDLINE), Psychological Abstracts (PsycInfo), Education Resources Information Center (ERIC), Cumulative Index to Nursing and Allied Health Literature (CINAHL), Cochrane Database of Systematic Reviews, and Cochrane Register of Controlled Trials. This

Table 1. Empirical Visual Stimulation and Training Programs

Authors and Year	Sample Characteristics	Design	Dependent Variables	Rehabilitation Procedures	Treatment Results	Remarks
Stimulation programs						
Leguire et al, 1992	n = 15 experimental, n = 14 controls, n = 50 normal sighted; age, younger than 13 months at the start	Pretest-posttest with experimental and 2 control groups (Level 2)	Latencies of pattern and flash VER p1, amplitudes were not recorded reliably, BSID MDI	During 1-year presentation of slides for 12 minutes a day, 5 days a week, slides consisted of square wave gratings, checkerboards, geometric patterns, facial drawings.	Initial differences in latencies between the visually impaired and normal population were the following: pattern VER p1 = 28 msec, flash VER p1 14 msec. Experimental group exhibited a systematic decrease (improvement) in latencies as shown by significant regression lines. Experimental group showed improved and control group decreased BSID MDI.	Concrete differences among experimental, normal and control groups were not mentioned. The clinical relevance of small improvements in VER p1 latencies were not mentioned either. What is the impact of improved latency for everyday visual functioning?
Mamer, 1999	n = 10; ages 9–21 years, multiply handicapped; visual acuity ≤ LP	Single-group repeated-measure design, 2 baseline, 4 to 5 interventions, and 2 follow-up measurements (Level 4)	Visual acuity with Teller acuity cards and scoring of number of eye blinks, duration of visual fixations, number of gaze shifts, turning away, and reaching toward object during 5-minute observation	15–20 minutes of passive exposure in a 3-sided carrel with 30 × 20-in sheets with black and white patterns for 4 days per week during 8 weeks. A different set of sheets was used each week.	Increase in visual acuities, no changes in the visual behaviors	Owing to a very short baseline in the absence of a control group, the design is not very strong. Absolute increases in Teller scores were not mentioned, and initial acuity scores were very low (acuity ≤ LP). The clinical relevance of increased Teller scores can be questioned when the visual behaviors do not change.
Training programs						
Barraga, 1964	n = 10 experimental, n = 10 control blind, n = 10 control low vision; age 6–13 years; visual acuity, <3% experimental group, <10% control groups	Pretest-posttest with experimental and control groups (Level 2)	Score on VDT, reliability of VDT is good, validity insufficient	8 weeks of training for 45 min a day in couples.	No change in visual acuity 9 out of 10 experimental, low-vision children showed an increase from pretest to posttest; 8 out of 10 experimental children improved significantly more than their matched controls; control children improved their VDT scores from pretest to posttest.	VDT and the training are more cognitive and perceptual than visual. Blind control children recruited at another school than that of the experimental children.

Table 1. (Continued)

Authors and Year	Sample Characteristics	Design	Dependent Variables	Rehabilitation Procedures	Treatment Results	Remarks
Training programs						
Poland and Doebler, 1980	n = 4, 6, and 7 years	Repeated measurement design, counterbalanced for lighting conditions (Level 4)	Phase 1: Eye-to-object contact; Phase 2: eye-to-object contact 2–10 sec; Phase 3: visually following 180°	40 sessions with 20 trials per session, 1 session per day. Children had to look at a penlight. The cue given is "child's name," 2-sec pause, "look." Advancement to the next phase required 4 successful out of 7 sessions.	All subjects performed better under black light than under white light. This effect overcame the expected practice effect when the black light training occurred before the white light training.	The ecological validity of the black light condition is questionable. But because all participants had made little progress prior to the eye contact training, this training is a useful first step before more ecologically valid training conditions are undertaken.
Potenski, 1983	n = 7 experimental, n = 6 controls; all participants were deaf-blind, legally blind with some residual vision, IQ < 20	Pretest-posttest with experimental and control group (Level 2)	Increase in number of correctly placed pieces of puzzle	20 minutes training for 3 days under black light (experimental group) or normal light (control group). Task: puzzle with 4 geometric forms.	Significantly better performance experimental group. Under black light, there was longer attention, more exploration, and following of objects in the experimental group.	6 subjects in experimental and 5 subjects in control group dropped out because of ceiling and floor effects.
Goetz and Gee, 1987	n = 1; age 3 years; aphakia and glaucoma with delayed development	Multiple baseline, multiple probe design across responses (6) (Level 2)	Visual attention to the task and percent of tasks performed correctly	60 trials of 30 minutes. First task training then verbal and acoustic prompting of visual fixation. Tasks: puzzles, stack rings, put lid on pot, stack glasses, insert coin, hang cup on hook.	Visual attention training necessary with first 2 tasks. Spontaneous generalization with other tasks	Prompting at the critical spot triggers fixation but also suggests the correct manipulation that has to be performed. Child has to be able to understand verbal requests (e.g., "watch and do this").
Sonksen et al, 1991	n = 35 experimental , n = 23 control group; younger than 13 months at the start	Repeated (4×) measures design with experimental and control group (Level 2)	Ordinal scale measuring: near vision, distance vision, following horizontal, vertical, and convergence, distance tracking across and "to and fro," sphere of attention: inanimate and animate	Intervention for 1 year consisting of suggestions for visual activities at 6 levels adapted to the potential of the child. Control children received a general, early intervention program. Activities were carried out by the parents.	The experimental children improved on all 9 scales of visual functioning. Greatest improvements in the first 4 months. Important predictors for success were initial visual level, and developmental level. No improvement found for children with Leber congenital amaurosis (n = 3) and Norrie disease (n = 2).	Program aimed at looking, not seeing. Reliability and validity of measurement instrument is not given.

Table 1. (Continued)

Authors and Year	Sample Characteristics	Design	Dependent Variables	Rehabilitation Procedures	Treatment Results	Remarks
Hall Lueck et al, 1999	n = 1; age 14 months; child with cortical visual impairment	ABA design for 2 tasks: (1) choosing a favorite toy out of 2; (2) visual following of large translucent beads on a string (Level 3)	Touch, reach, fixation only, touch, follows	Training was discontinued several times because of illness of participant. Training lasted 36 days, 108 trials task 1, 84 trials task 2. The training was based on the model of Hall and Bailey (1989) and consisted of visual environment management, visual skills training, and visual dependent task training	Increase of 38% for touching preferred toy; increase of 65% for following beads on a string; anecdotal information that the child generalized learned behavior spontaneously.	The training failed with another child, especially because this child did not accept the effects of visual environment management.
Werth and Moehrenschlager, 1999	Experimental group, n = 22: 16 children (mean age, 45 months), blind due to ischemic cerebral lesions following perinatal asphyxia plus 6 children (mean age, 78 months), blind due to cerebral trauma. Control group, n = 43: 31 children with ischemic lesions (mean age, 43 months) and 12 children with traumatic lesions (mean age, 78 months). All participants had cerebral visual impairment and no ocular pathology for at least 1 year before treatment. Lesions were verified by MRI scans.	Repeated measurement design with randomized experimental and 2 control groups. Number of measurements in the experimental group is not clear. Control groups had a pretest and posttest. Level 2 (no complete randomization)	Expansion of visual field in degrees measured by eye movements (EOG recordings) toward a light stimulus (diameter, 2.5°; luminance, 5 cd/m^2) or during blank trials on an arc perimeter. After nonresponse, the luminance of the target was increased. Functional luminance difference threshold measured with the arc perimeter and systematic variation of the target luminance. Visual acuity with forced-choice preferential looking technique.	Blind visual field of the experimental group was stimulated by moving a light (velocity, ~3°/sec; luminance, 26,000 cd/m^2; diameter, 5 mm) on a dark background along the horizontal meridian from the periphery toward the center of the visual field. The training for the control group was no training (n = 13) and for 30 children identical to the experimental group except that the light stimulus was replaced by objects between 5 and 10 cm in diameter (luminance, between 8 and 15 cd/m^2; background of about 35 cd/m^2. Training lasted 4 weeks to 3 months with daily half-hour sessions.	Of the 22 experimental children, 15 children recovered from cerebral blindness within 3 months. In all but 1 child, the visual fields did not shrink within 2 years after the end of visual field training. In 5 of these 15 children, the functional luminance difference thresholds were still elevated and in 10 they were normal. The placebo and no-treatment group showed no recovery of visual fields. Eye movement latencies were measured in 4 children. In 3 of them, eye movements proved to be longer for stimuli presented in the recovered visual fields then in the normal visual fields.	Unknown why stimulation failed to enlarge visual fields in 7 children. Elevated functional luminance difference thresholds make it unlikely that visual performance generalized to everyday visual functioning in 5 children. Conscious perception or visually guided behavior in everyday life was not studied. Results restricted to children with cerebral visual impairment who can hold their heads stable and had no severe eye movement disorders

Table 1. (Continued)

Authors and Year	Sample Characteristics	Design	Dependent Variables	Rehabilitation Procedures	Treatment Results	Remarks
Werth and Seelos, 2005	n = 17 experimental (mean age 26 months), n = 20 control, pseudotraining (mean age 20 months), n = 17 control, no treatment (mean age 22 months); all participants had cerebral visual impairment and no ocular pathology due to perinatal asphyxia and blind visual fields for at least 1 year before treatment. Lesions were verified by MRI scans.	Repeated measurement design with randomized experimental and 2 control groups (Level 1)	Expansion of visual field in degrees measured by the difference between eye movements (EOG recordings) toward and away from a light stimulus (luminance, <50 cd/m^2 on an arc perimeter). Functional luminance difference threshold measured with the arc perimeter and systematic variation of the target luminance. Latency of eye movements toward a stimulus at 30° eccentricity in 6 patients. Visual acuity with Teller acuity cards. fMRI in 2 recovered children and in 2 children who showed no improvement after training.	Blind visual field of the experimental group was stimulated by moving a light (velocity, ~3°/sec; luminance, 26,000 cd/m^2; diameter, 5 mm) on a dark background along the horizontal meridian from the periphery toward the center of the visual field. The training for the control group was identical except that the light stimulus was replaced by an object about 5 cm in diameter (luminance, ~25 cd/m^2; background, ~35 cd/m^2). Training lasted 3 months with daily half-hour sessions.	11 out 17 experimental children showed enlarged visual fields of 30°-90 within 3 months. In 4 of these 11 children, the functional luminance difference thresholds were still elevated, and in 7, they were normal. The placebo and no-treatment group showed no recovery of visual fields. 7 of the 11 children had normal acuities, and 5 elevated Teller acuities after treatment. In only 1 of 6 children, eye movement latency was longer for the recovered vs the unaffected visual field. fMRI showed cortical activation corresponding to the recovered visual fields. In the 2 children who did not recover, there was no activation in the occipital lobe contralateral to the hemianopic visual hemifield.	Experiments were done to check for the influence of light scatter as an alternative explanation of the results. Two additional experimental children with hypoxic ischemic lesions did not recover visual fields. Unknown why stimulation failed to enlarge visual fields in 8 children. After training stopped, the visual fields in some children became blind again. Eye movement latencies were not convincing because 3 children were blind. Latencies should have been compared with a norm group instead of comparing the affected and unaffected visual fields. Conscious visual perception tested in only 3 children by asking them to point to the location of a target shown for 200 msec. Visually guided behavior in everyday life was presumed but not tested.

BSID MDI indicates Bayley Mental Scale of Infant Development Mental Development Index; EOG, electro-oculogram; LP, light perception; fMRI, functional magnetic resonance imaging; VDT, Visual Discrimination Test.

Table 2. Levels of Evidence for Grading Research According to Siebes et al,[103] Based on Sackett,[100] Butler et al,[101] and Butler and Darrah[102]

Level	Group Research	Single Individual Research
1	Randomized controlled trial	n = 1 randomized controlled trial
2	Nonrandomized controlled trials	ABABA designs
	Prospective cohort studies with concurrent control group	Alternating treatments (eg, ABACA)
	Multiple baseline across participants	
3	Case studies with control participants	ABA designs
	Cohort studies with historical control group	
4	Case series without control participants	AB designs
	Case reports	Case reports
5	Nonempirical methods	Nonempirical methods

search yielded 10 published empirical studies on visual rehabilitation (see Table 1). The heading ''design'' in Table 1 also includes the level of evidence of the research design. The first author ranked the levels of evidence according to the modified classification system of Sackett.[100–103] Information on the level of evidence is straightforward and could be easily derived from the methods section of the articles. Given the unambiguous nature of these data, no interrater reliability was determined for these data (compare the work of Siebes et al[103]). An explanation of this system is given in Table 2. The modifications were applied to evaluate research in developmental disabilities. The level of evidence classification system was based on a hierarchy of research designs that range from the greatest to the least, according to the ability to identify causal relations and to reduce bias, combined with a means of assessing the thoroughness with which the particular study was conducted. According to Butler and Darrah,[102] Level 1 studies produce the most credible evidence and, thus, yield the most definitive results. Level 2 studies, based on less convincing evidence, produce tentative conclusions. Levels 3 and 4 reflect still less persuasive evidence and merely suggest causation. Level 5 evidence does not lead to conclusions about treatment efficacy.

Table 1 shows the results of the literature search on visual rehabilitation programs. Only 2 of the studies were ''stimulation'' programs: the Columbus Children's Hospital vision stimulation program[40] and an intervention study by Mamer.[78] The rest were training programs.[14,26,70,81,84,104–106] The levels of evidence ranged from 1 to 4, with 6 studies producing tentative conclusions regarding treatment efficacy. Only the study of Wert and Seelos[70] produced credible evidence of treatment effects.

GENERAL DISCUSSION

Owing to the small number of empirical studies, a meta-analysis was impossible to perform on the question whether the 1996 statement of Ferrell and Muir, to call for an end to vision stimulation as an end in itself, is still valid. To answer this question, we have defined what visual rehabilitation is, looked at the presuppositions and goals of visual rehabilitation programs, and studied the results of 10 empirical intervention programs.

Empirical Programs: Visual Stimulation

Both visual stimulation programs[40,78] that used noncontingent visual stimulation did find an effect of the stimulation on visual functioning. However, the clinical relevance of both these studies is rather weak. Although, in the Leguire et al study,[40] the latencies improved for the experimental group of children with visual impairment, it is unclear whether the initially significant difference in flash and pattern latency between the experimental group and the sighted control group also changed significantly. Owing to fact that the exact differences in latency times at the last measurement point were not given by Leguire et al, it is not possible to conclude whether the improvement in VER latencies are clinically very relevant. Based on the small differences in the average latencies of pattern and flash VERs between the low vision and sighted children during the 12-month intervention period, 28 and 14 milliseconds respectively, an observable behavioral change in visual functioning is not to be expected. In the study of Mamer,[78] the participants' visual acuity scores increased significantly, but the visual behaviors did not change. Unfortunately, Mamer did not mention the absolute increase in Teller acuity scores. A floor effect could be a likely explanation of the increase in visual acuity, because all participants had light perception or less visual ability at the start of the intervention. Children with light perception are, at most, only able to fixate the Teller card with largest gratings. Fixating 1 extra card (Teller cards increase with half an octave; an octave is a halving or doubling of spatial frequency) at the posttest measurement could lead to a significant statistical improvement in Teller acuity. However, the clinical relevance is limited, because the 95% confidence interval for acuity cards is about 1 octave.[107] Fixating 1 extra card at follow-up could easily be the result of chance or measurement error. Moreover, given the design used by Mamer (i.e., a single group repeated measurement design), one cannot be sure that the intervention caused the effect. Learning effects of repeated testing or maturation could also explain these effects. Mamer gave a likely explanation why visual acuity increased,[78(p367)] ''Since both the grating in the Teller Acuity Cards and the sheets used in the intervention are black and white, the time spent with the intervention might have better prepared the students for the presentation of the Teller Acuity Cards.''

Conclusion. At the moment, the evidence in support of performing vision or visual stimulation is frail and not convincing, and it might even be counterproductive for the adaptive functioning of the brain, as discussed in the section on brain plasticity. We tend to disregard the claim of Powell that the sensitive period for visual development might be prolonged for children without light perception and for adults with visual impairment and additional severe impairments, because studies with preterm infants showed that extra visual input did not affect visual functioning. Delayed neurological maturation, not extra visual input, might be responsible for increased visual behavior with age. Therefore, we agree with Ferrell and Muir to end vision stimulation training and that one should refrain from visual stimulation programs aimed at improving the visual system. But how strong is the case of Ferrell and Muir for visual training?

Empirical Programs: Visual Training

In training programs, ''looking'' (i.e., paying attention to what is seen), and not ''seeing'' (i.e. light sensation and transportation to the brain), is trained. Consequently, the most common goal in visual training programs is to change visual behavior, instead of brain functioning.

Eight empirical visual training programs were reviewed. All of them showed significant improvement of visual behavior after intervention. However, from a methodological point of view, these results are not always very convincing. First of all, despite the critique one can have on ranking levels of evidence,[108] there was only one Level 1 study, the randomized controlled trial of Werth and Seelos.[70] For 5 of the 8 studies, the levels of evidence for treatment efficacy produce only tentative results (Level 2). The treatment effects in the study of Poland and Doebler[104] and Hall Lueck et al[105] merely suggest causation. Second, a serious setback in the studies of Goetz and Gee[81] and Hall Lueck et al[105] is that in single subject studies, it is hard to generalize the results to other children, tasks, or situations. Moreover, whereas Goetz and Gee used a relatively strong single subject design, namely the multiple baseline, multiple probe design (Level 2), Hall Lueck et al used the weaker, with regard to internal validity, ABA design (Level 3). The failure to implement the intervention in 1 child in the Hall Lueck study and in 11 children in the Potenski study[14] limits the applicability of these interventions to the larger population of children with visual impairments, with or without additional impairments. Success and failure of the above-mentioned interventions seems to be, at least in part, dependent on individual child characteristics. An extensive description of these characteristics is necessary, but not given in the reviewed studies. For instance, characteristics of the 11 children excluded from the Potenski study could be very helpful for other clinicians who would like to use black light conditions for training task performance in severely or profoundly retarded children with multiple visual impairments.

Regardless of the enormous impact the Barraga study[84] had on the field of teaching visually impaired children, 3 critical remarks must be made about this study. First, Barraga claimed to have improved visual functioning, whereas if one looks at the content of the training and the Visual Discrimination Test, it seems that cognitive and perceptual abilities were dealt with as much as visual functioning. Second, Barraga constructed her own assessment instrument for visual functioning. Reliability and validity of this instrument are not given, so that it remains uncertain whether this instrument is capable of detecting significant behavioral change. Lastly, the blind children in the control group came from a different school than the children in the experimental group. Theoretically, group differences could be the result of factors related to the school and not to factors related to the intervention itself. Comparable comments can be made about the study of Sonksen et al.[26] First, the homemade ordinal scales to measure visual functioning were not psychometrically studied. As a result, the reliability and validity of the scales are not known. Second, the interventions were based on the results of the visual assessments and were also very similar to the visual assessments. The test and intervention goals were related, which could have been to the advantage of the experimental children during the visual development test, because they had more practice in comparable tasks than the children in the control group. A placebo program could have prevented this design flaw. However, according to the authors, performing a placebo visual program was not a realistic proposition from an ethical point of view, and was therefore omitted.

With the exception of the study of Werth and Seelos,[70] a general setback in the reviewed programs is the lack of decent follow-up data. Given the claim of Powell[11] that the sensitive period for visual development might be prolonged for children without light perception and for adults with visual impairment and additional severe impairments, it is strange that follow-up measurements were not undertaken. As a result, the possible invalidating effects of history and maturation are not considered.[95] Both the studies of Werth and colleagues[70,106] were aimed at improving visual field size in children with cerebral visual impairment without ocular pathology. Although Werth and colleagues called them both training programs, they were in part stimulation programs because stimuli were given noncontingent upon the behavior of the child. The results of both these studies seemed quite spectacular because the visual fields extended 30° to 90° and the intervention effects were controlled for light scatter. Whether fixation control was arranged is less clear, because the participants were unable to give verbal or motor responses to check central fixation. Also unknown is why in both studies stimulation failed to enlarge visual fields in 7 and 8 children, respectively. The visual fields in some children became blind again after the training stopped. Although this result is evidence of treatment effects, it is not very promising with regard to generalization of visual stimulation results to everyday life. Conscious visually guided behavior was another problem, because it made presumptions but only tested 3 of the 17 children by asking them to point to the location of a target shown for 200 milliseconds.

Owing to all the aforementioned methodological problems and the rather small number of empirical studies, Ferrell and Muir[2] are right in stating that the scientific results on interventions effects are ambiguous.

Research Implications

The complaint of Ferrell and Muir that visual stimulation and training hinders normalization and integration of children with visual impairments could be overcome if training results are not only statistically significant but also ecologically valid, which is the extent to which findings can be generalized to the ''real world.'' The ecological validity of the studies of Poland and Doebler,[104] Potenski,[14] and Werth and colleagues[70,106] is dubious. Black and bright lights are hardly ever used in normal daily living conditions but mainly in ''snoezel'' rooms. Consequently, training programs that use black light or light stimuli should have a generalization phase to establish transfer of the learned responses to daily life under normal lightening conditions. Unfortunately, the aforementioned training programs did not have generalization training. A way to improve ecological validity is to have an individual program design, because the successful outcome of the studies of Potenski,[14] Sonksen et al,[26] Goetz and Gee,[81], and Hall Lueck et al,[105] might be the result of the individual adaptations made in these programs. Individual programs take into account individual differences and the heterogeneity of the population of children with visual impairment. Especially these 2 factors make it very hard to design uniform and general visual training programs for all children with visual impairment.

Clinical Implications for Developmental and Behavioral Pediatrics

The results of the empirical studies have 2 important implications for clinical practice. First, visual training should be performed by the people that have the most frequent contact with the child with visual impairment. In most cases, this will be the parents. One of the reasons that the Sonksen program might have been successful is that the training was carried out by the parents. As a result, many more training opportunities were available than in interventions that are performed by professionals, who normally meet the children no more than once or twice a week. Moreover, the interventions are incorporated in the daily interactions with the child. As a result, not only the visual performance of the children is affected but also parental competence in skills regarding play, attachment, language, and interaction. Second, visual training should be adapted to individual needs and task demands. In the studies of Potenski,[14] Sonksen et al,[26] Goetz and Gee,[81] and Hall Lueck et al,[105] the dependent variables were both visual functioning as well as correct task performance. In these studies, visual training was not a goal in itself but a means to achieve other goals. In task training, child development training covers other domains than just visual ones, such as cognition, attention, and fine motor development. Given the current state of affairs, we prefer visual training programs that also improve these skills above programs that only aim at visual functioning, because they seem to be more effective. By itself, task training is ecologically valid; otherwise, one should not have chosen this particular task.

Our conclusion is that visual stimulation or training programs are not very effective or ecologically valid whenever task demands and individual differences are not taken into account. For the moment, this should urge teachers, trainers, and clinicians to refrain from non-contingent visual stimulation programs and from training general visual functions independently from certain tasks. The 1996 statement of Ferrell and Muir, to call for an end to vision stimulation as an end in itself, is still valid, but visual training adapted to the individual needs of child and related to certain task demands is certainly promising and should be the subject of future research.

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REFERENCES

1. Moore MW. Visual efficiency training with low vision children. *Am Orthopt J*. 1972;22:68–70.
2. Ferrell KA, Muir DW. A call to end vision stimulation training. *J Vis Impair Blind*. 1996;9:364–366.
3. Hall Lueck A, Bailey IL. Letter to the editor. *J Vis Impair Blind*. 1997;9:101–103.
4. Mckenna KE. Letter to the editor. *J Vis Impair Blind*. 1997;91:103–105.
5. Tavernier GGF. Slechtziendheid: perspectieven op visuele stimulatie en training. *Psychol Belg*. 1991;3:23–51.
6. Lundervold D, Lewin LM, Irvin LK. Rehabilitation of visual impairments: a critical review. *Clin Psychol Rev*. 1987;7:169–185.
7. Resnikoff S, Pascolini D, Etya'ale D, et al. Global data on visual impairment in the year 2002. *Bull World Health Organ*. 2004;8:844–851.
8. Peterson RC, Wolffsohn JS, Rubinstein M, et al. Benefits of electronic vision enhancement systems (EVES) for the visually impaired. *Am J Ophthalmol*. 2003;1:1129–1135.
9. Sterns GK, Hyvärinen L. Addressing pediatric issues. In: Fletcher DC ed. *Low Vision Rehabilitation*. San Francisco, CA: American Academy of Ophthalmology; 1999:107–119.
10. Gellhaus MM, Olson MR. Using color and contrast to modify the educational environment of visually impaired students with multiple disabilities. *J Vis Impair Blind*. 1993;8:19–20.
11. Powell SA. Neural-based visual stimulation with infants with cortical impairment. *J Vis Impair Blind*. 1996;9:445–450.
12. Corn AL. Low vision and visual efficiency. In: Scholl GT ed. *Foundations of Education for Blind and Visually Handicapped Children and Youth: Theory and Practice*. New York: American Foundation for the Blind; 1986:99–117.
13. Hall A, Bailey IL. A model for training vision functioning. *J Vis Impair Blind*. 1989;8:390–396.
14. Potenski DH. Use of black light in training retarded, multiply handicapped, deaf-blind children. *J Vis Impair Blind*. 1983;7:347–348.
15. Corn AL. Instruction in the use of vision for children and adults with low vision: a proposed program model. *Review*. 1989; 2:26–38.
16. Rawstron JA, Burley CD, Elder MJ. A systematic review of the applicability and efficacy of eye exercises. *J Pediatr Ophthalmol Strabismus*. 2005;42:82–88.
17. Birnbaum MH. The power of visual training. *J Am Optom Assoc*. 1984;4:257–260.

18. Greenburg RM. What is visual training: a patient education handout. *J Am Optom Assoc*. 1985;5:57–58.
19. Contestabile MT, Recupero SM, Palladino D, et al. A new method of biofeedback in the management of low vision. *Eye*. 2002;1:472–480.
20. Pirozzolo FJ, Rayner K. Dyslexia: the role of the eye movements in developmental reading disabilities. In: Johnston CW, Pirozzolo FJ, eds. *Neuropsychology of Eye Movements*. Hillsdale, NJ: Lawrence Erlbaum Associates; 1988.
21. Getman GN. A commentary on vision training. *J Learn Disabil*. 1985;18:505–512.
22. Beauchamp GR. Optometric vision training. *Pediatrics*. 1986;7: 121–124.
23. Hoyt CS. Visual training and reading. *Am Orthopt J*. 1999;49: 23–25.
24. Olitsky SE. Dyslexia. *Am Orthopt J*. 1999;49:17–22.
25. Skottun BC. Is dyslexia caused by a visual deficit? *Vision Res*. 2001;4:3069–3070.
26. Sonksen PM, Petrie A, Drew KJ. Promotion of visual development of severely visually impaired babies: evaluation of a developmentally based program. *Dev Med Child Neurol*. 1991;3:320–335.
27. Suaning G, Lovell NH, Schindhelm K, et al. The bionic eye (electronic visual prosthesis): a review. *Aust N Z J Ophthalmol*. 1998;2:195–202.
28. Maynard EM. Visual prostheses. *Annu Rev Biomed Eng*. 2001;3:145–168.
29. Margalit E, Maia M, Weiland JD, et al. Retinal prosthesis for the blind. *Surv Ophthalmol*. 2002;4:335–356.
30. Kasten E, Wüst S, Behrens-Baumann W, et al. Computer based training for the treatment of partial blindness. *Nat Med*. 1998;4:1083–1087.
31. Kasten E, Müller-Oehring E, Sabel BA. Stability of visual field enlargements following computer-based restitution training, results of a follow-up. *J Clin Exp Neuropsychol*. 2001;23:297–305.
32. Poggel DA, Kasten E, Müller-Oehring EM, et al. Unusual spontaneous and training induced visual field recovery in a patient with a gunshot lesion. *J Neurol Neurosurg Psychiatry*. 2001;70:236–239.
33. Wüst S, Kasten E, Sabel BA. Visuelles Restitutionstraining nach Schadigung des Nervus opticus. *Z Med Psychol*. 2004;13:131–141.
34. Reinhard J, Schreiber A, Schiefer U, et al. Does visual restitution training change absolute homonymous visual field defects? A fundus controlled study. *Br J Ophthalmol*. 2005;8:30–35.
35. Deleted in proof.
36. Sabel BA, Kenkel S, Kasten E. Vision restoration therapy. *Br J Ophthalmol*. 2005;8:522–524.
37. Zihl J. *Rehabilitation of Visual Disorders After Brain Injury*. Hove, UK: Psychology Press; 2000.
38. Horton JC. Disappointing results from Nova Visions's visual restoration therapy. *Br J Ophthalmol*. 2005;8:1–2.
39. Plant GT. A work out for hemianopia. *Br J Ophthalmol*. 2005;89:2.
40. Leguire LE, Fellows RR, Rogers GL, et al. The CCH Vision stimulation program for infants with low vision: preliminary results. *J Vis Impair Blind*. 1992;8:33–37.
41. Moore S, Bensinger S, Frere S, et al. *Bright Sights: Learning to See*. Louisville, KY: American Printing House for the Blind; 1984.
42. Barraga NC, Collins ME. Development of efficiency in visual functioning: rationale for a comprehensive program. *J Vis Impair Blind*. 1979;7:121–126.
43. Smith AJ, Cote KS. *Look at Me, A Resource Manual for the Development of Residual Vision in Multiply Impaired Children*. Philadelphia: Pennsylvania College of Optometry Press; 1982.
44. Jones CJ. *Evaluation and Educational Programming of Deaf-Blind/Severely Multihandicapped Students: Sensorimotor Stage*. Springfield, IL: Charles C. Thomas; 1988.
45. Sonksen PM, Dale N. Visual impairment in infancy: impact on neurodevelopmental and neurobiological processes. *Dev Med Child Neurol*. 2002;4:782–791.
46. Tavernier GGF. The improvement of vision by vision stimulation and training: a review of the literature. *J Vis Impair Blind*. 1993;8:143–148.
47. Fellows RR, Leguire LE, Rogers GL, et al. A theoretical approach to vision stimulation. *J Vis Impair Blind*. 1986;8:907–909.
48. Glass P. Development of the visual system and implications for early intervention. *Infants Young Child*. 2002;15:1–10.
49. Utley B, Duncan D, Strain P, et al. Effects of contingent and noncontingent vision stimulation on visual fixation in multiply handicapped children. *J Assoc Sever Handic*. 1983;8:29–42.
50. Chen D, Haney M. An early intervention model for infants who are deaf-blind. *J Vis Impair Blind*. 1995;8:213–221.
51. Hyvärinen L. Early stimulation of visually impaired infants. *Ophthalmic Paediatr Gen*. 1983;2:129–133.
52. Grossman H. Cortical visual impairment, presentation, assessment, and management. *The Royal New South Wales Institute for Deaf and Blind Children Monographs Series.* No. 3. North Rocks, Australia: North Rocks Press; 1992.
53. Hyvärinen J, Hyvärinen L, Linnankoski I. Modification of parietal association cortex and functional blindness after binocular deprivation in young monkeys. *Exp Brain Res*. 1980;4:1–8.
54. Carlson S, Hyvärinen L. Visual rehabilitation after long lasting early blindness. *Acta Ophthalmol*. 1983;6:701–713.
55. Diamond MC. Brain: response to enrichment. In: Smelser NJ, Baltes PB, eds. *International Encyclopedia of the Social and Behavioral Sciences*. Amsterdam: Elsevier; 2001:1352–1358.
56. Erin JN, Paul B. Functional vision assessment and instruction of children and youths in academic programs. In: Corn AL, Koenig AJ, eds. *Foundations of Low Vision: Clinical and Functional Perspectives*. New York: American Foundation for the Blind; 1996:185–220.
57. Anastasiow NJ. Implications of the neurobiological model for early intervention. In: Meisels SJ, Shonkoff JP, eds. *Handbook of Early Childhood Intervention*. New York: Cambridge University Press; 1990:196–216.
58. Movshon JA, Van Sluyters RC. Visual neural development. *Annu Rev Psychol*. 1981;3:477–522.
59. Schmidt KE. Neural plasticity in visual cortex. In: Smelser NJ, Baltes PB, eds. *International Encyclopedia of the Social and Behavioral Sciences*. Amsterdam: Elsevier; 2001:10549–10553.
60. Hubel DH, Wiesel TN. The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *J Physiol*. 1970;206:419–436.
61. Carlson S, Hyvärinen L, Raninen A. Persistent behavioral blindness after early visual deprivation and active visual rehabilitation: a case report. *Br J Ophthalmol*. 1986;7:607–611.
62. Gregory RL, Wallace JG, Recovery from early blindness, a case study. *Experimental Psychology Society Monograph.* No. 2; 1963.
63. Gregory RL. Seeing after blindness. *Nat Neurosci*. 2003; 6:909–910.
64. Fine I, Wade AR, Brewer AA, et al. Long-term deprivation affects visual perception and cortex. *Nat Neurosci*. 2003;6:915–916.
65. Atkinson J. *The Developing Visual Brain*. Oxford: Oxford University Press; 2000.
66. Huo R, Burden SK, Hoyt CS, et al. Chronic cortical visual impairment in children: aetiology, prognosis, and associated neurological deficits. *Br J Ophthalmol*. 1999;8:670–675.
67. Preuss TM. The argument from animals to humans in cognitive neuroscience. In: Cazzaniga MS, ed. *Cognitive Neuroscience*. Oxford, UK: Blackwell Publishers; 2000:483–502.
68. Ungerleider LG, Haxby JV. ''What'' and ''where'' in the human brain. *Curr Opin Neurobiol*. 1994;4:157–165.
69. Bell J. An approach to the stimulation of vision in the profoundly handicapped, visually handicapped child. *Br J Visual Impair*. 1986;4:46–48.

70. Werth R, Seelos K. Restitution of visual functions in cerebrally blind children. *Neuropsychologia*. 2005;4:2011–2023.
71. Chugani HT, Müller R-A, Chugani DC. Functional brain reorganization in children. *Brain Dev*. 1996;1:347–356.
72. Röder B, Rösler F, Neville H. Event-related potentials during auditory language processing in congenitally blind and sighted people. *Neuropsychologia*. 2000;3:1482–1502.
73. Röder B, Stock O, Bien S, et al. Speech processing activates visual cortex in congenitally blind humans. *Eur J Neurosci*. 2002;16: 930–936.
74. Amedi A, Raz N, Pianka P, et al. Early ''visual'' cortex activation correlates with superior verbal memory performance in the blind. *Nat Neurosci*. 2003;6:758–766.
75. Sathian K. Visual cortical activity during tactile perception in the sighted and the visually deprived. *Dev Psychobiol*. 2005;4: 279–286.
76. Burton H. Visual cortex activity in early versus late blind people. *J Neurosci*. 2003;23:4005–4011.
77. Sadato N, Okada T, Honda M, et al. Critical period for cross-modal plasticity in blind humans: a functional MRI study. *Neuroimage*. 2002;1:389–400.
78. Mamer L. Visual development in students with visual and additional impairments. *J Vis Impair Blind*. 1999;9:360–369.
79. Greenburg RM. What is visual training: a patient education handout. *J Am Optom Assoc*. 1985;5:57–58.
80. Heiner D. *Learning to Look, A Handbook for Parents of Low Vision Infants and Young Children*. East Lansing, MI: Blind Children's Fund; 1986.
81. Goetz L, Gee K. Teaching visual attention in functional contexts: acquisition and generalization of complex visual motor skills. *J Vis Impair Blind*. 1987;8:115–117.
82. Blanksby D. The use of video to facilitate visual attention in preschool children. *J Vis Impair Blind*. 1992;8:72–73.
83. Blanksby D. Visual therapy: a theoretically based intervention program. *J Vis Impair Blind*. 1992;8:291–294.
84. Barraga N. *Increased Visual Behavior in Low Vision Children*. New York: American Foundation for the Blind; 1964.
85. Barraga NC, Collins ME, Hollis J. Development of efficiency in visual functioning: a literature analysis. *J Vis Impair Blind*. 1977; 7:387–391.
86. Corn AL, Koenig AJ. Perspectives on low vision. In: Corn AL, Koenig AJ eds. *Foundations of Low Vision: Clinical and Functional Perspectives*. New York: American Foundation for the Blind; 1996:3–25.
87. Chapman EJ, Tobin MJ, Tooze FH, et al. *Look and Think: A Handbook for Teachers*. 2nd ed rev. London: Royal National Institute for the Blind; 1979.
88. Harrell L, Akeson N. *Preschool Vision Stimulation: It's More Than a Flashlight*. New York: American Foundation for the Blind; 1987.
89. Watkins S. *The INSITE Model, A Model of Home Intervention for Infant, Toddler, and Preschool Aged Multihandicapped Sensory Impaired Children. SKI-HI Institute*. North Logan; UT: HOPE, 1989.
90. Aitken S, Buultjens M. *Vision for Doing, Assessing Functional Vision for Learners Who Are Multiply Disabled*. Edinburgh: Moray House Publications; 1992.
91. Blanksby DC. *Visual Assessment and Programming, The VAP-CAP Handbook*. Burwood, Australia: Royal Victorian Institute for the Blind; 1992c.
92. Blanksby DC, Langford PE. VAP-CAP: a procedure to assess the visual functioning of young visually impaired children. *J Vis Impair Blind*. 1993;8:46–49.
93. Levack N. *Low Vision, A Resource Guide with Adaptations for Students with Visual Impairments*. Austin, TX: Texas School for the Blind and Visually Impaired; 1998.
94. Bozic NM, Hill EW, Tobin MJ. Pre-school visually impaired children: visual stimulation and micro-computers. *Child Care Health Dev*. 1993;1:25–35.
95. Goetz L, Gee K. Functional vision programming. In: Goetz L, Guess D, Stremel-Campbell K, eds. *Innovative Program Design for Individuals with Dual Sensory Impairments*. Baltimore, MD: Paul H. Brooks; 1987:77–97.
96. Lanners J, Goergen E. Functional evaluation and rehabilitation of severely visually impaired children (0–4 years). *Vis Impair Res*. 2001;3:1–5.
97. Utley BL, Roman C, Nelson G. Functional vision. In: Sacks SZ, Silberman RK, eds. *Educating Students Who Have Visual Impairment With Other Disabilities*. Baltimore, MD: Paul H. Brooks; 1988:371–412.
98. Hall Lueck A. *Functional Vision, A Practitioner's Guide to Evaluation and Intervention*. New York: AFB Press; 2004.
99. Barraga NC, Morris JE. *Program to Develop Efficiency in Visual Functioning*. Louisville, KY: American Printing House for the Blind; 1983.
100. Sackett DL. Rules of evidence and clinical recommendations on the use of antithrombotic agents. *Chest*. 1989;9:2s–4s.
101. Butler C, Chambers H, Goldstein M, et al. Evaluating research in developmental disabilities: a conceptual framework for reviewing treatment outcomes. *Dev Med Child Neurol*. 1999;4: 55–59.
102. Butler C, Darrah J. Effects of neurodevelopmental treatment (NDT) for cerebral palsy: an AACPDM evidence report. *Dev Med Child Neurol*. 2001;4:778–790.
103. Siebes RC, Wijnroks L, Vermeer A. Qualitative analysis of therapeutic motor intervention programmes for children with cerebral palsy: an update. *Dev Med Child Neurol*. 2002;4:593–603.
104. Poland DJ, Doebler LK. Effects of a blacklight visual field on eye-contact training of spastic cerebral palsied children. *Percept Mot Skills*. 1980;5:335–338.
105. Hall Lueck A, Dornbusch H, Hart J. The effect of training on a young child with cortical visual impairment: an exploratory study. *J Vis Impair Blind*. 1999;9:778–793.
106. Werth R, Moehrenschlager M. The development of visual functions in cerebrally blind children during a systematic visual field training. *Restor Neurol Neurosci*. 1999;1:229–241.
107. Thompson C. Assessment of child vision and refractive error. In: Buckingham T, ed. *Visual Problems in Childhood*. Oxford: Butterworth-Heinemann, 1993:159–210.
108. Dunst CJ, Trivette CM, Cutspec PA. Toward an operational definition of evidence-based practices. *Centerscope*. 2002;1:1–10.