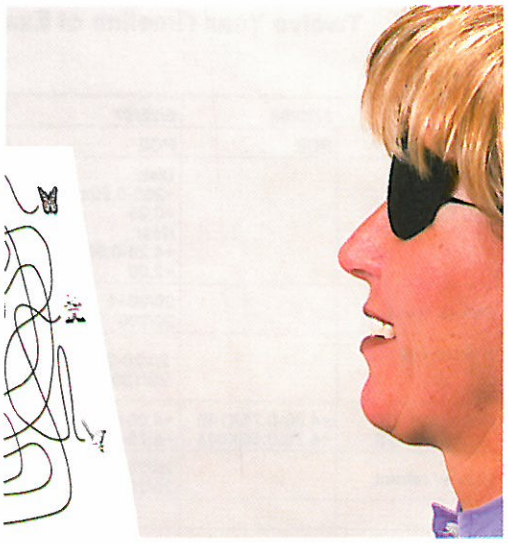
Active Vision Therapy on an Adult Strabismic Amblyope



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Abstract

Studies have shown that treatment of amblyopia after "visual maturity," which occurs around the age of 9, can improve not just visual acuity, but overall visual functioning. Nevertheless, many clinicians do not treat amblyopia if patients appear "too old." The case of a 45-year-old amblyope who was successfully treated is presented. The age of when treatment can be instituted is discussed, as well as the efficacy of different treatment modalities (i.e. occlusion and spectacle correction versus occlusion, spectacle correction, and active vision therapy).

Key Words

amblyopia, age, visual acuity, active vision therapy, passive vision therapy, occlusion, spectacle correction, critical period

Amblyopia is defined as a unilateral, and infrequently bilateral, condition in which the best corrected visual acuity is poorer than 20/20 in the absence of any obvious structural or pathologic anomalies.[1] One or more of the following accompanies the loss of vision: amblyogenic anisometropia or isometropia, amblyogenic unilateral or bilateral astigmatism, constant unilateral strabismus, or visual deprivation.[1] Amblyopia visually impairs roughly 3% of the general population. In the 45-year-old and younger population, amblyopia is responsible for more loss of vision than all ocular disease and trauma combined.[2] In addition to visual acuity, amblyopic eyes can manifest deficiencies in oculomotor[3] and fixation skills,[4] accommodation,[5] spatial uncertainty and distortion,[6] contrast sensitivity,[2] stereoacuity,[7] and suppression under binocular viewing.[8] It is unlikely that an amblyopic eye will spontaneously resolve these deficiencies without some form of intervention.

The case of an adult amblyope whose motivation compelled her to undergo active vision therapy is presented. Age as a function of amblyopia therapy, efficacy and permanence of amblyopia therapy, and of active vision therapy versus occlusion and spectacle correction alone as treatment modalities will be discussed.

CASE REPORT

History

A 45-year-old woman with amblyopia in the left eye was seen at the State University of New York, State College of Optometry University Optometric Center for an evaluation in June of 1998. She was evaluated previously at the clinic in July of 1987. Interestingly, she did not manifest a strabismus at either evaluation, but a variable small angle left strabismus was documented on each of three primary care examinations that occurred in the time between the strabismic evaluations. She showed a small angle left exotropia with a left hypertropia in 1989, a small angle left exotropia in 1993, and then a small angle left *eso*tropia in 1998. The ocular health and visual field confrontation evaluation during primary care exams in 1986, 1989, 1993, and 1998 were unremarkable. The distant visual acuities at each exam were consistently OD 20/25 and OS 20/200 with correction. The refractive status over the years remained generally stable: approximately OD +4.50-0.50x180, OS +4.75 DS.

The findings at each exam are listed in Table 1.

Her 1998 history revealed that a "problem" with the left eye was detected before first grade. A patch was worn over the right eye, but no improvement occurred with the left eye. The vision in her left eye had not changed since the first grade. She received her first pair of glasses around the age of 6 years. She could not recall whether she had an eyeturn at the evaluation in 1987, but reported having an alternating esotropia as a child at the evaluation in 1998. In 1987

Table 1
Twelve Year Timeline of Exams for Amblyopic Patient at the University Optometric Center

Date	5/29/86	5/28/87	7/15/87	5/31/89	4/19/93	6/4/98	6/17/98
Exam Type	PCE	PCE	STRAB WU	PCE	PCE	PCE	STRAB WU
Hab Rx: OD OS		Dist: +250-0.50x180 +0.25 Near: +4.25-0.50x180 +2.00		+4.50-0.50x180 +2.00	+4.00-0.75x180 +4.50	+4.75-0.75x172 +5.25	+4.25 +4.50 +1.75 add OU
DVA w/ hab rx		20/30+1 20/200	20/25 20/200	20/25-2 20/200	20/20-3 20/200	20/25 20/200-1	20/25 20/200-1
NVA w/ hab rx OD OS		20/20-2 20/120		Uncorrected: 20/50- 20/200-	20/30-1 20/200	20/50 20/320	20/30 20/200
Refraction: OD OS	+4.00-0.75X180 +4.75-0.50X005	+4.00-0.75X180 +4.75-0.50X180		+4.00-0.75x180 +4.50	+5.00-0.50x170 +4.75	+4.25-0.25x025 +6.00-0.75x170	
DVA w/ refract.		20/25 20/200		20/25 20/100-1	20/25+3 20/100	20/25 20/200-1	
PH				NI			NI
2.2x TS			NI				NI
Laser Inter			20/50 20/100				
DCT	Ortho		Ortho			LET	Ortho
NCT	Ortho		Ortho	LXT' w/ L Hyper	4Δ LXT'	LET	Ortho-LE(T)'
NPC	2"/5"				4"/6" (+)dipl	2"/8" (+)dipl	2" (-) dipl
Motilities	Jerky			Full	Slightly jerky	Slightly jerky	Slight V syndrome, subtle OS abduction deficit
Phorometry	N/A	N/A	N/A	N/A	N/A	N/A	Const OS suppression
Visuoscopy			OD: Central OS: 5-6Δ unsteady sup. EF				OD: Central OS: Grossly unsteady, no one point used for fixation
Haidinger Brush			poor appreciation				
Luster							Const OS suppression
Bagolini							NRC, OS central scotoma/suppression
Amblyoscope							<O= <S = ortho, OS target very blurred
Brock Posture							NRC, 8Δ eso w/ small scotoma/suppression zone
W4D							Const OS suppression
KVS							Const OS suppression
Vectos							Occasional diplopia, then immediate lapse into OS suppression
Brock String							Const OS suppression

Table 1 Legend
dipl = Diplopia
DCT = Distant Cover Test
DVA = Distant Visual Acuity
EF = Eccentric fixation
KVS = Keystone Visual Skills
NCT = Near Cover Test
NI = No improvement
NPC = Nearpoint of Convergence
NRC = Normal Retinal Correspondence
NVA = Near Visual Acuity
PCE = Primary Care Exam
PH = Pinhole
STRAB WU = Strabismus
W4D = Worth Four Dot

she showed concern about the loss in vision in her left eye and wanted to know what could be done. She did not return to the Strabismic Clinic again until 1998. At this point, she was asymptomatic except for the significant left eye blur. She was still interested in seeking therapy for the left eye.

Assessment

She wore a spectacle correction of OD +4.25 DS and OS +4.50 DS, with a +1.75 add both eyes. This had been prescribed at the primary care exam on 6/4/98. She had corrected distant visual acuities of 20/25 OD, 20/200 OS, and near corrected acuities of 20/30 OD and 20/200 OS. No improvement was noted with a pinhole or 2.2x telescope. Unilateral cover testing revealed orthophoria in the distance, and a questionable very intermittent small angle left esotropia at near. Alternate cover testing at near revealed orthophoria. The patient had a nearpoint of convergence of two inches with no subjective diplopia. Motilities revealed a minimal V syndrome and a subtle left abduction deficit. She failed all stereoacuity testing; no randot stereopsis was exhibited and Wirt circles revealed OS suppression. Sensory testing (Worth-four-dot, red lens, lustre) demonstrated left eye suppression at all distances. Phorometry revealed constant left eye suppression.

With vectograms, she showed a left eye suppression. Additionally, by disparating the two vectograms we tested diplopia awareness and any suppression zone. She experienced occasional diplopia in the periphery, but then lapsed into left eye suppression, indicating a rather large and deep central suppression zone.

Correspondence testing (Bagolini Striated Lenses, Brock Posture Board,[a] and major amblyoscope,) suggested normal retinal correspondence. Both the Brock posture board and Bagolini lenses confirmed a left eye suppression or scotomatous zone. She postured 8Δ eso with the Brock posture board. Additionally, the major amblyoscope revealed no objective movement and subjective fusion at orthophoria with the left eye target being very blurred (i.e., Objective Angle = Subjective Angle = 0).

Assessing her binocular status with the Keystone Visual Skills[a] and Brock String revealed constant left eye suppression.

Visuoscopy indicated central and occasionally unsteady foveal fixation with the right eye, and grossly unsteady fixation with the left eye. The patient did not fixate with any one consistent retinal area with the left eye.

The patient was diagnosed with amblyopia of the left eye, most likely strabismic in origin. The deep suppression, lack of sensory fusion, and lack of diplopia awareness indicated a very poor prognosis for improvement with vision therapy. She was upset when told her prognosis. Nevertheless, she demonstrated extreme motivation and insistence with pursuing vision therapy. A trial of 10 weekly sessions was then implemented, with a guarded prognosis.

The progression of her vision therapy can be followed on Table 2.

Therapy

Since the fixation ability of the left eye was quite poor, the initial sessions stressed developing this skill.

At the first session, she was unable to place a peg into a slowly rotating pegboard. She then attempted to place the peg into a *stationary* pegboard. She under- and overshot, then eventually succeeded in placing the peg into the hole by feeling for it. She could not distinguish the letters of the large Michigan Tracking[b] (5-6 mm in height). For home activities, she was to patch the right eye for two to four hours while eating and engaging in other home-based activities. In addition, she was to color in quarter-sized circles, place pencil dots inside quarter-sized circles (develop accurate fixation with tactile reinforcement), and fill in letters of very large newspaper print. These tasks were very difficult for her to perform.

At the second visit, the patient exhibited a surprising, yet questionable increase of visual acuity to 20/60. With a +5.00 add, she was able to appreciate the Haidinger brush and actually place the brush onto a 20/20 letter that she was fixating. She was to continue the previous home activities. Instead of filling in letters of large newspaper print, Michigan Tracking letters were enlarged on a photocopier to approximately 15mm in height.

She showed a distance visual acuity of 20/70 and a near acuity of 20/100 with her current +1.75 add at the third visit. With a +5.00 add, she achieved $20/30^{-2}$. With foveal tagging procedures, she demonstrated the ability to place an afterimage on a designated target, thus enforcing central fixation. However, she had a very difficult time placing the afterimage on a rotating letter and sustaining the image on the moving target. Home activities included the previous activities and Groffman Tracing.[c]

At the fourth session, she reported difficulty with the Groffman traces because the lines appeared to move. Her letter fill-ins for homework exhibited eccentric fixation and past-pointing: the area in which she thought she was filling in the letter was actually shifted slightly up and to the right. This indicated that she required more intensive work with accurate fixation. At this session she demonstrated the ability to place a peg into a *rotating* pegboard. However, she still fixated inaccurately and undershot. Monocular tracing exhibited eccentric fixation, but she improved and increased in accuracy with repetition and auditory feedback (i.e., she was told when not tracing on the line). With fastpointing techniques, she tended to past-point but improved accuracy with repetition. Homework included the previous activities (letters for fill-ins now 5-6 mm in height). Additionally, she was to use a toothpick to "spear" raisins.

At the fifth visit, she manifested an esotropia on unilateral cover test, a finding that had not been seen from the time of the most recent strabismus evaluation until that day. Distance visual acuity was $20/60^{-2}$, and near acuity was $20/100^{+1}$ (with +1.75 add) and $20/50^{-2}$ (with +3.00 add).

Fixation was still very unsteady, but not as random as before, when assessed with visuoscopy at the sixth visit. She had increased significantly in her accuracy with letter fill-ins. With the quoit vectogram, she demonstrated the ability to appreciate diplopia in the periphery.

At the seventh session, she experienced physiologic diplopia with the Brock string. In addition, she could converge and diverge her eyes appropriately with 5Δ flips and maintain physiologic diplopia most of the time with the Brock string. If she momentarily lost the left-eye image, she could voluntarily break her suppression and see the image again. The ability for her to make appropriate eye vergence movements, maintain physiologic diplopia, and not suppress in the presence of a stressor stimulus (5Δ) indicated encouraging and positive prognostic signs.

At the eighth session, she exhibited a sensory fusion response with lustre testing. She now demonstrated signs of both sensory and motor fusion, unlike the initial evaluation where she exhibited neither. Her fixation had significantly improved as evidenced by the increase in her visual acuity and in her accuracy of letter fill-ins. Additionally, the depth of her suppression had significantly lessened as evidenced by her sensory fusion responses and physiologic diplopia awareness. That day she was able to sustain an afterimage on a rotating target with her left eye. At this point, the smallest print size letters being sent home was 3.5 mm in height. She could complete the task, but encountered much difficulty. She also reported that she could work more easily at closer distances with the Brock string.

The patient reported a subjective improvement with her left eye at the ninth visit. She felt that she saw better and that she possessed more control of that eye. A new spectacle prescription was written, changing the current +1.75 add to a +3.00 add in both eyes. She had shown consistent near visual acuity of 20/30 with a +3.00 add over the previous few visits. Improving her near visual acuity would help increase her binocularity at this

Table 2
Vision Therapy Timeline

Week	1	2-Shows ability to foveate	3	4	5	6-Shows periph diplopia awareness	7-Shows physiol diplopia	8-Shows sensory fusion	9	10	11-Shows ARC
DVA OS		20/60-2	20/70		20/60-2				20/60-2		20/50-1
NVA OS +1.75 add		20/200	20/100 +5.00 add: 20/30		20/100 +1 +3.00 add: 20/50-2				20/40-2 ; +3.00 add: 20/30-2	20/30	20/30-2
DCT									Very slight LE(T)		2LET w LHyper
NCT					Very small LET' uncovered				Ortho —> LE(T)'		Ortho
Visuoscopy						Fixation less random, but still very unsteady					
Amblyo-scope											<O=7BO <S=8 BI ARC
Bagolini											ARC
Rotating Pegboard	Stationary board; feels for hole			Rotating board-still over and undershoots							
Mich Track	Large (6mm Height)-unable to see	Enlarged to 15mm in height						Letter size 3.5mm in height	Very difficult Pastpoints when cross out letters	Same	
Haidinger Brush		With +5.00 add, can place brush on smallest letters								Work on sustaining foveation	
Afterimage			Can place AI on letter; Cannot sustain AI on rotating letter					Can sustain AI on moving target			
Groff Trace			Difficult								
Monoc. Cheiro Trace				Traces off to side of lines, improves w/repetition		Traces off lines, improves w/repetition					
Telebinocular				Tends to past point; ↑accuracy w/repetition & concen-tration							
Vectos						Diplopia awareness					
Brock String							Physiologic diplopia, Vergence mvnts w/ 5Δ prism flips	Same		Same	
Home VT	Patch 2 hrs; dot circles, fill in letters, color circles	Cont same	Cont same HVT, using smaller letters, Groff Trace	Cont same, spear the raisin,	Cont same, # Search	Showing increasing accuracy with letter fill-in; cont same HVT	Cont same, Brock String	Tracing more accurate, NFHC; cont same	Cont same; Mich Tracking	Continue same	Cont same; lost to f/u

distance, thereby facilitating her binocular training. Although she could fill in specified letters accurately at this point, she experienced significant difficulty when asked to cross out letters of the alphabet in sequence, which were contained in a background of other letters. This task demands much more visual discrimination, organization, and figure-ground skills when compared to just filling in specified letters. Now she had to look for different letters (not solely "o"s or "e"s) and find the letters in sequence. She would overshoot and cross out the letter next to the one she intended to cross out. She also noticed a preference to fixate the right visual field with her left eye. She encountered more difficulty fixating letters on the left side of the Hart chart. For example, in order to read the first letter in each row, she would first fixate a letter in the middle of the row and read backwards until she arrived at the first letter.

At the tenth visit, we continued working with foveation skills with the Haidinger brush, fixation skills in an increased resolution background (Michigan Tracking, Hart Chart), and physiologic diplopia and vergence movements with the Brock string. By the eleventh visit, she had achieved an acuity of $20/50^{-1}$.

DISCUSSION

This case helps to dispel the notion of an upper age limit for amblyopia treatment and of a decreased prognosis for treatment after the age of 10. The concept of a "sensitive" or "critical" period plays a major role in assuming an upper age limit. Worth[9] first proposed the idea of a critical period in 1903, which he believed lasted until the age of 6. Hubel and Wiesel[10] further investigated the concept of a critical period. They defined the critical period for the development of the visual system as the time period in which vision can be *permanently* affected by abnormal visual experience; abnormal visual experience beyond this time period will not affect vision. Hardman Lea, et al,[11] defined the critical period more precisely as "that passage of time during which the development of the immature visual system may be altered by change in the quality, quantity, or balance of the visual input via the two eyes."

Clinicians and prominent writers in the field of amblyopia have related this concept of a sensitive period to clinical practice. Von Noorden[12] stated that "the sensitive period during which recovery is possible is less well defined although there seems to be a general agreement that it ends with the 8th year of life." Harwerth, et al,[13] agreed. Campos[14] said that the "…concept of a sensitive period is extremely relevant to clinical practice, as it indicates that anti-amblyopia therapy after the sensitive period is not effective." According to Shapero,[15] "prognosis becomes progressively poorer as treatment is delayed beyond age of 5 or 6" and "hope of achieving normal and complete development, with 20/20 vision, becomes progressively reduced with advancing age." Wright[16] believed that "amblyopia tends to be reversible when treated with the appropriate visual stimulation during early childhood." He further said that "the prognosis for amblyopia depends upon the age of the patient, severity of amblyopia, and type of amblyopia. The earlier the amblyopia occurs, and the longer it remains untreated, the worse the prognosis… Each case must be evaluated individually as to whether or not the child is too old to undergo amblyopia therapy. The general rule of thumb is every child under 9 years of age should undergo a trial of amblyopia therapy." He associates amblyopia therapy only with children, and makes no inferences to older patients.

Although there may well be a critical period up to the age of 8 or 9 in which amblyopia can develop, there may not be such a temporal limitation to treatment and visual recovery. Studies show that neural plasticity lasts well into adulthood.[17-21] Therefore, we must differentiate between a sensitive period for when amblyopia can occur, when functional vision is recoverable, and when amblyopia can recur once treated or cured.

Age as a Function of Therapy

In considering treatment for amblyopia, many clinicians view the patient's age as the prevailing factor. This may be influenced by the idea of a sensitive period. In addition, it has been shown that occlusion alone without visual therapy for treating amblyopic patients 10 years and older gave unsuccessful results.[22] Therefore, many clinicians who utilize occlusion as the only form of treatment may conclude that treating amblyopic patients 10 years or older is ineffective. However, age alone does not determine the eligibility of a person for amblyopia therapy. The following studies indicate that increased age does not preclude older people from visual rehabilitation.

Birnbaum, et al,[23] showed that older patients exhibited the same success rates as younger patients. They analyzed 23 published amblyopia studies to determine the success rates of therapy as a function of age. These studies were divided into two large age groups of under 7 years and over 7 years. They performed a separate analysis and further divided 17 of the studies into three age groups: 7 to 10, 11 to 15, and 16 and older. Two criteria for success were used: 1) improvement of four or more lines in visual acuity, and 2) achievement of 20/30 acuity or better. These findings indicated that the different age groups showed almost identical success rates for each criterion. Hardman Lea, et al,[11] agreed that the final vision achieved does not depend on the age at which treatment is initiated.

Other studies have investigated whether age at the onset of treatment influences post-treatment regression. Levartovsky, et al,[24] gave evidence that children older than 9 years of age improved in visual acuity and that visual acuity did not deteriorate any more than in their younger patients. In fact, their numbers indicate that the older children had less deterioration after therapy. Another study found that older children may indeed have more stable outcomes. Oster, et al, [25] found that older children at the start of treatment (mean age of 33 months) and at the end (mean age of 40 months) did not require maintenance occlusion, whereas the younger children in the beginning (mean age of 26 months) and at the end (mean age of 31 months) did require maintenance occlusion. Thus, the older child samples appeared to have more stable visual acuities after treatment. This finding supports that of Levartovsky, et al.[24]

Several studies investigated improvement of vision specifically in adults. Kasser and Feldman[26] found improvements with adults. In addition, they noted that compliant subjects achieved better visual acuity than non-compliant. A study conducted by Saulles [27] showed that treatment with spectacle correction, part-time occlusion, and visual therapy increased visual acuity and functioning of adult amblyopes ages 21-39.

Wick, et al, [22] also implemented a similar treatment protocol of spectacle correction, part-time occlusion, and active vision therapy. They treated 19 anisometropic amblyopes, ages 6 to 49, with the same sequential protocol for each. They determined that adults could achieve successful visual acuity. They used the Amblyopia Success Index (ASI) of Meyer, et al,[28] to record results:

$$\text{ASI} = \frac{\text{Initial VA} - \text{Final VA}}{\text{Initial VA} - \text{Test Dist}} \times 100$$

Snellen acuity must be converted from feet to meters (i.e. 20/60 = 6/18). For example, my patient began with an initial acuity of 20/200 and ended with a final acuity of 20/50. She showed an 80% improvement:

Initial VA = 20/200 = 6/60

Final VA = 20/50 = 6/18

$$\text{ASI} = \frac{60 - 18}{60 - 6} \times 100 = 80\% \text{ improvement}$$

The ASI's for Wick, et al's patients averaged 92.1 % (+/- 8.1%) improvement; some 42% of the patients achieved 100% improvement. They found no statistically significant relationship between treatment duration and patient's age. Additionally, the gained visual acuity remained stable when followed at least one year after treatment.

In addition to the above, other studies and case reports support amblyopia therapy into adulthood.[19,21,29,30] Obviously, adult amblyopes could benefit significantly from therapy.

Efficacy and Permanence of Amblyopia Therapy Results

Efficacy studies have shown that a high percentage of patients with functional amblyopia show visual acuity improvement with amblyopia treatment. Most studies utilized direct occlusion of the non-amblyopic eye as treatment. Scott and Dickey[31] followed strabismic amblyopic patients who underwent full-time occlusion, through at least 9 years of age. They found that 72% had attained a visual acuity of 20/20 and 20% had achieved between 20/25 to 20/40. Rutstein and Fuhr[32] followed patients aged 2 to 53 years with strabismus and/or anisometropia who underwent direct occlusion. A log unit method to follow acuity improvement was used. They determined that an improvement of 0.3 log units was equivalent to a doubling effect of visual acuity. Ninety percent of patients 7 years or younger showed acuity gain, with 67% achieving at least a doubling of acuity and 54% obtaining 20/40 or better. Seventy-seven percent of their patients 8 years or older showed acuity gain, with 37% achieving at least a doubling of acuity and 27% obtaining 20/40 or better. Thus, patients with functional amblyopia can benefit from treatment.

Although treatment may increase visual acuity, the lasting effects of amblyopia treatment have been questioned. Rutstein and Fuhr[32] followed 64 amblyopes. They divided their subjects into two age groups. Group one consisted of subjects 7 years and younger, whose final visual acuities were taken on average 11.8 months after discontinuing treatment. Group two consisted of subjects 8 years and older, whose final visual acuities were taken on average 13.1 months after discontinuing treatment. They found that 75% of their strabismic and/or anisometropic amblyopic patients in Group one showed some VA reduction, but overall showed a net acuity gain. Sixty-seven percent of their patients in Group two showed some acuity reduction. Gregerson and Rindziunski[33] found a similar percentage. They followed 53 patients with strabismic amblyopia 10 years after stopping therapy. About 25% maintained the gained visual acuity; this agrees with findings from the study of Rutstein and Fuhr. The average age when first seen and when last evaluated was 4.3 and 16.9 years, respectively. Fifty-three percent showed a one-two line decrease in acuity. On average, the patients preserved about 50% of the visual improvement.

In contrast to the above 75% of subjects who showed regression, Scott and Dickey[31] found that 75% of their strabismic amblyopic patients *retained* their gained acuity. The subjects underwent full-time occlusion and were followed until 9 years of age. They were re-evaluated at an average age of 15.9 years. Seventeen percent showed a one to two line decrease in acuity, and 8% showed a decrease of more than two lines.

Other studies have shown more mid-range percentages of subjects retaining their gained visual acuity. Sparrow and Flynn[34] followed 30 children with amblyopia who were 12 years or younger at the onset of therapy and had initial acuity of 20/100 or worse (21 with strabismus, eight with strabismus and anisometropia, one with anisometropia). The majority were treated with full-time direct occlusion. Their final visual acuity was taken at least three years following discontinuing therapy. Forty percent of their patients maintained the gained acuity. Those who showed regression from the gained visual acuity regressed less than two lines. Ching, et al,[35] followed 116 strabismic children with amblyopia who began direct occlusion therapy by the age of 5. Occlusion was discontinued at a maximum age of 9 years. Visual acuities of patients were reassessed at least one year after occlusion therapy ended. Their study found that 52% of the patients lost some acuity, and 48% retained visual acuity gained. The mean visual acuity lost was less than one Snellen line. Oster, et al,[25] followed 188 patients with amblyopia at least one year after discontinuing treatment. They found that 47% did not require maintenance patching. This agrees with findings from Ching, et al. In addition, their findings also suggest that older children may be more stable than younger.

Levartovsky, et al,[24] investigated the influence of age at the onset and cessation of amblyopia therapy on long-term stability of visual acuity. They examined 104 children who were treated for amblyopia by direct occlusion 6.4 years, on average, after discontinuing therapy. All children were younger than 12 years of age at the start of occlusion therapy, and 90% received evaluations up to the age of 9 years. Fifty-five percent of patients showed acuity deterioration. The age at which occlusion therapy began did not significantly affect the final visual acuity outcome after cessation of occlusion, provided the acuity was monitored to the age of 9 and maintenance patching implemented if a decrease in acuity was noted. The small number of patients whose occlusion treatment was discontinued before the age of 9 years did not achieve as good acuity, in addition to showing a higher deterioration rate when compared to those who were followed to 9 years of age. The average visual improvement was 5.5 lines. The average acuity loss was 1.2 lines.

Levartovsky, et al,[36] investigated the influence of initial visual acuity and amblyopia type on the long-term stability of visual acuity following completion of occlusion therapy. The acuities of 94 children were followed to the age of 9 years and examined 6.4 years, on average, after the end of treatment. Patients were divided into two groups based on their initial acuities: 1) 20/60 to 20/100, 2) 20/100 or worse. Group one showed that 42% of the patients regressed 0.58 lines on average in acuity. Group two showed that 63% regressed by 1.54 lines on average. The patients also were analyzed by their type of amblyopia: strabismic, strabismic-anisometropic, anisometropic. Deterioration of acuity occurred in 46%, 79%, and 36% of patients with an average deterioration of 0.70, 2.04, and 0.64 lines, respectively. They concluded that low initial visual acuity and strabismus-anisometropia were risk factors for losing gained acuity over time after the end of occlusion therapy.

These studies investigating the stability of amblyopia therapy after discontinuing therapy showed a range of 25%-75% of patients who retained their gained visual acuity. This difference may be attrib-

uted to the variability in research design and methodology. Overall, the results are positive. Findings in some of the studies within this group were in close agreement: 40%,[34] 45%,[24] 47%,[25] 48%.[35] In addition, the remaining percentage of subjects who regressed in visual acuity showed a regression of less than one line to no more than two lines. Levartovsky, et al,[24] found an average gain of 5.5 lines. Thus, although slight regression may occur, the overall visual acuity gained surpasses the acuity prior to treatment.

Long-term stability of improved visual acuity has also been associated with binocular function.[1] Ham, et al,[37] treated 205 strabismic amblyopes ages 3-16 years. In some cases acuity did not regress but improved over time, after the cessation of therapy. Ham, et al, attributed the improvement and stability of VA to binocular function made possible by surgery and/or glasses. Other clinicians propose that the decrease in visual acuity in the amblyopic eye after cessation of treatment can be related to the level of binocularity the patient has at the time of discontinuation.[1, 33, 38] Therefore, treatment of amblyopia should not be considered complete or successful until the visual function of the amblyopic eye is integrated into and stabilized under binocular viewing conditions.[39]

In addition to exploring the level of binocularity as a factor in post-therapy visual acuity stability, investigators have researched other possible factors, such as age at the onset and cessation of therapy, depth and type of amblyopia, and compliance. Levartovsky, et al,[24] found no correlation between the age of onset or cessation of therapy with the amount of deterioration in visual acuity. Findings from this study and that of Oster, et al,[25] suggest that older subjects may possess more long-term stability with visual acuity. In a separate study[35] investigating depth and type of amblyopia, Levartovsky and colleagues found that a poorer visual acuity or a strabismic-anisometropia may yield a worse prognosis. Non-compliance in children[40] and adults[26] resulted in significantly poorer visual outcomes.

Wick, et al,[22] treated adult amblyopes, who later showed visual acuity retention and no regression of at least one year post-treatment. They implemented active vision training in their treatment protocol, in addition to spectacle correction and part-time occlusion. This suggests that the addition of active vision therapy can help stabilize long-term visual acuity better than with passive treatment alone (i.e., spectacles and occlusion).

Active Vision Learning Vs. Passive Occlusion

Most of these studies discussed investigating the stability of amblyopia treatment over time utilized occlusion as the main, if not only, form of treatment. These studies investigated the stability of visual acuity that was passively and "spontaneously" regained by spectacle correction and occlusion. Amblyopic eyes generally show deficiencies in oculomotor[3] and fixational[4] skills, accommodative skills,[5] spatial distortion,[6] stereopsis,[7] and suppression under binocular viewing conditions.[8] It seems reasonable that to rehabilitate a patient with such visual deficiencies, spectacle correction and occlusion should be used in conjunction with active vision therapy. This modality affords the opportunity for the patient to learn how to enhance the underdeveloped visual skills of the amblyopic eye, expediting visual recovery and stabilizing the visual system.

"Vision therapy" should be called "vision learning." Active vision therapy goes beyond passive therapy (i.e., spectacle correction and occlusion) by providing appropriate stimuli and feedback. Feedback allows for a conscious awareness that helps us to monitor internal and external stimuli, which in turn helps us control our body actions. Humans and animals learn through feedback. Most theorists agree that learning 1) is a change in behavior that is non-genetic and results from environmental interaction, 2) is dependent on action and are effects from practice, and 3) results in a relatively permanent change in behavior that will modify future behavior.[41] Polarizing lenses, red-green filters, lenses, and prisms provide useful feedback information for patients about what they visually feel and see and what they must do to change the situation. The ultimate goal of vision therapy is to improve underdeveloped visual skills and help transform them into habits.

Kirschen and Flom[42] demonstrated that amblyopic eyes had a maximum visual acuity at the fovea. They and other clinicians "taught" patients to foveate to achieve maximum visual acuity. Kirschen and Flom used auditory feedback to control unsteady and eccentric fixators. Another clinician, Kupfer,[43] believed that central fixation was required to achieve best acuity. He stated that the chance of central fixation being learned spontaneously is decreased with the presence of central inhibition and eccentric fixation. *"If the visual acuity is to be improved, patching of the good eye alone may not be successful. It is essential for these patients to acquire or to be taught central fixation before any significant improvement in visual acuity can occur."*[43]

To further emphasize the importance of incorporating active vision therapy in amblyopia treatment, occlusion and spectacles alone oftentimes do not improve visual acuity and function, as in the patient discussed in this case report. This may be attributed to the lack of awareness and feedback from the dysfunctional visual system; the amblyopic eye has not learned to function properly. Studies and case reports have shown that active vision therapy reduces the total amount of therapy time to achieve best acuity[44, 45] and that it can recover visual loss that passive treatment can not.[29, 46-54] Additionally, it has been shown that active therapy combined with passive therapy was more effective than passive treatment alone. Wick, et al,[22] implemented a sequential treatment protocol of spectacle correction, part-time occlusion, and active vision therapy. They compared their ASI findings to those of Meyer, et al,[28] who used only passive treatment. The treatment protocol instituted by Wick, et al, produced higher ASI scores compared to the passive therapy used by Meyer, et al. Additionally, success rates were significantly decreased in patients older than 10 with Meyer's study. Subjects older than 10 years from Wick's study showed similar success rates compared to their younger ones. They concluded that occlusion alone was not as effective in patients older than 10, and that active vision therapy needs to be implemented for an effective therapy program.

CONCLUSION

Considering all things discussed and given the speed and magnitude of improvement of the patient discussed earlier, this case argues that there exists no true upper age limit for the treatment of amblyopia and that patching alone, without attacking the underlying etiology, will not resolve the problem. Older age, having amblyopia for a long period of time,

showing severe visual acuity reduction, and being strabismic have been implicated as poor prognostic factors. This woman who has lived with 45 years of severely reduced vision resulting from a strabismus that could not be detected due to the gross disuse of that eye, achieved an 80% improvement (using the Amblyopia Success Index[28]) in a period of three months. She is 36 years beyond the "critical period." In addition, she demonstrated deep suppression and a lack of motor or sensory fusion at the initial evaluation. Nevertheless, in a relatively short time, she achieved a visual acuity of 20/50- with both motor and sensory fusion. Clearly, there exists no upper age limit for the treatment of amblyopia.

Future studies regarding vision therapy and amblyopic adults may further confirm that age does not preclude adults from therapy. Amblyopia visually impairs 3% of the general population. We can therefore conclude that a significant number of visually impaired people live in our world today. Consequently, there exists no reason why these people should be told that nothing could be done to improve their vision. Rather than dismissing amblyopia in an adult patient as a foregone conclusion, both passive and active vision therapy should be considered as a viable option.

References

1. Griffin JR, Grisham JD. Binocular anomalies: diagnosis and vision therapy. Boston: Butterworth-Heineman, 3rd Ed., 1995.
2. Ciuffreda KJ, Levi DM, Selenow A. Amblyopia: basic and clinical Aspects. Boston: Butterworth-Heineman, 1991:1, 3.
3. Stark LW, Ciuffreda KJ. Kenyon RV. Abnormal eye movements in strabismus and amblyopia. In: Lennerstrand G, Zee DS, Keller EL. eds. Functional basis of ocular motility disorders. New York: Pergamon Press, 1982:71-82.
4. Schor CM. A motor theory for monocular eccentric fixation of amblyopic eyes. Am J Optom Physiol Opt 1978; 55:183-6.
5. Abraham SV. Accommodation in the amblyopic eye. Am J Ophthalmol 1961; 52:197-200.
6. Flom MC, Bedell HE, Bareito R. Spatial mechanisms for visual acuity deficits in strabismic and anisometropic amblyopia: developmental failure or adaptation? In: Keller EL, Zee, DS, eds. Adaptive processes in visual and oculomotor systems. New York: Pergamon Press, 1986 45-51.
7. Holopigian K, Blake R, Greenwald MJ. Selective losses in binocular vision in anisometropic amblyopes. Vis Res 1986;26:621-30.
8. Holopigian K, Blake R, Greenwald MJ. Clinical suppression and amblyopia. Invest Ophthalmol Vis Sci 1988;29:444-51.
9. Worth CA. Squint: its causes, pathology, and treatment. Philadelphia: F. Blakiston's Son and Co., 1903.
10. Hubel DH. Wiesel DN. The period of susceptibility to physiological effects of unilateral eye closure in kittens. J Physiol 1970;206:419-36.
11. Hardman Lea SJ, Loades J, Rubinstein MP. The sensitive period for anisometropic amblyopia. Eye 1989;3: 783-90.
12. von Noorden GK. Binocular vision and ocular motility theory and management of strabismus. 5th Ed. St. Louis: Mosby Co., 1996.
13. Harwerth RS, Smith, III EL, Crawford MLJ, von Noorden GK. Multiple sensitive periods in the development of the primate system. Science 1986;232:2358.
14. Campos E. Amblyopia: major review. Surv Ophthalmol 1995;40:23-9.
15. Shapero M. Amblyopia. Philadelphia: Chilton Book Co., 1971:172.
16. Wright KW. Pediatric ophthamology and strabismus. St. Louis: Mosby Co., 1995.
17. Zohary E, Celebrini S, Britten KH, Newsome WT. Neuronal plasticity that underlies improvement in perceptual performance. Science 1994;263:1289-92.
18. Levi DM, Polat U. Neural plasticity in adults with amblyopia. Proc Natl Acad Sci 1996;93:6830-4.
19. Selenow A, Ciuffreda KJ. Vision function recovery during orthoptic therapy in an adult esotropic amblyope. J Am Opt Assoc 1986;57(2):132-40.
20. Ciuffreda KJ. Visual system plasticity in human amblyopia. In: Hilfer SR, Sheffiel JB, eds. Development of order in the visual system. New York: Springer-Verlag (in press).
21. Selenow A, Ciuffreda KJ. Visual system plasticity in older amblyopes. Poster #30, Annual meeting of the American Academy of Optometry, Atlanta, 1985.
22. Wick B, Wingard M, Cotter S, Scheiman M. Anisometropic amblyopia: Is the patient ever too old to treat? Opt Vis Sci 1992;69(11):866-78.
23. Birnbaum MH, Koslow K, Sanet R. Success in amblyopia therapy as a function of age: a literature survey. Am J Optom Physiol Optics 1977;54(5): 269-75.
24. Levartovsky S, Gottesman N, Shimshoni M, Oliver, M. Factors affecting long-term results of successfully treated amblyopia: age at beginning of treatment and age at cessation of monitoring. J Ped Opthalmol Strab 1992;29: 219-23.
25. Oster JG, Simon JW, Jenkins P. When is it safe to stop patching? Br J Ophthalmol 1990;74: 709-11.
26. Kasser MD, Feldman JB. Amblyopia in adults. Am J Ophthalmol 1953;36(10): 1443-6.
27. Saulles H. Treatment of refractive amblyopia in adults. J Am Opt Ass 1987;58(12):959-60.
28. Meyer E, Mizrahi E, Perlman I. Amblyopia success index: A new method of quantitative assessment of treatment efficacy; applications in a study of 473 anisometropic amblyopic patients. Binoc Vis Q 1991;6:75-82.
29. Verma A, Singh, D. Active vision therapy for pseudopakic amblyopia. J Cataract Refract Surg 1997;23:1089-94.
30. Dalziel CC, Woodruff ME. Case report: Strabismic and anisometropic amblyopia. Am J Optom Physiol Optics 1981;58:603-5.
31. Scott WE, Dickey CF. Stability of visual acuity in amblyopic patients after visual maturity. Graefe's Arch Clin Exp Ophthalmol 1988;226:154-57.
32. Rutstein RP, Fuhr PS. Efficacy and stability of amblyopia therapy. Opt Vis Sci 1992;69(10):747-54.
33. Gregersen E, Rindziunski E. "Conventional" occlusion in the treatment of squint amblyopia. ACTA Ophthalmologica 1965;43:462-74.
34. Sparrow JC, Flynn JT. Amblyopia: A long-term follow-up. J Ped Ophthalmol 1977;14(6):333-6.
35. Ching FC, Parks MM, Friendly DS. Practical management of amblyopia. J Ped Ophthalmol 1986;23(1):12-16.
36. Levartovsky S, Oliver M, Gottesman N, Shimshoni M. Factors affecting long-term results of successfully treated amblyopia: Initial visual acuity and type of amblyopia. Br J Ophthalmol 1995;79:225-8.
37. Ham O, Claramunt M, Diaz T. Strabismic amblyopia: Final results of occlusion treatment in 205 Cases. Bin Vis 1985;1(4):195-202.
38. Parks M. After the eyes are straightened what is the ophthalmologist's responsibility? Ophthalmol 1986;93:1020-22.
39. Garzia R. Efficacy of vision therapy in amblyopia: A literature review. Am J Opt Physiol Optics 1987;64(6):393-404.
40. Oliver M, Neumann R, Chaimovitch Y, Gottesman N, Shimshoni M. Compliance and results of treatment for amblyopia in children more than 8 years old. Am J Ophthalmol 1986;102:340-5.
41. Forrest E. Feedback and the visual process. J Am Opt Ass 1981;52(9): 717-24.
42. Kirschen DG, Flom MC. Visual acuity at different retinal loci of eccentrically fixating functional amblyopes. Am J Opt Physiol Optics 1978;55(3):144-50.
43. Kupfer C. Treatment of amblyopia ex anopsia in adults. Am J Ophthalmol 1957;43:918-22.
44. Francois J, James M. Comparative study of amblyopic treatment. Am Orthopt J 1955;5:61-4.
45. Callahan WP, Berry D. The value of visual stimulation during contact and direct occlusion. Am Orthop J 1968;18: 73-4.
46. Wick B. Amblyopia—a case report. Am J Optom Arch Am Acad Optom 1973;50:727-30.
47. Wanter BS. Pleoptic therapy in amblyopes. Am Orthop J 1980; 30:77-82.
48. von Noorden GK, Lipsius RMC. Experiences with pleoptics in 58 patients with strabismic amblyopia. Am J Ophthalmol 1964;58:41-51
49. Maywey S, Massie HH; Amblyopia ex anopsia. A preliminary report of the more recent methods of treatment of amblyopia, especially when associated with eccentric fixation in cases of strabismus. Br J Ophthalmol 1958;42:257-69.
50. Tomlinson E, Jablinsky M. Results of modified pleoptic therapy in eccentric fixation. Am Orthopt J 1973;23:60-4.
51. Girard LJ, Fletcher MC, Tomlinson E, Smith B. Results of pleoptic treatment of suppression amblyopia by conventional patching, intensive hospital pleoptics, and intermittent office pleoptics. Am Orthop J 1969;19:40-7.

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52. Kageyama CJ, Loomis SA. Central fixation amblyopia: A case report. Optom Man 1980;71:33336.
53. Wesson MD. Use of light intensity reduction for amblyopia therapy. Am J Optom Physiol Opt 1983;60:112-7.
54. Selenow A, Ciuffreda KJ. Vision function recovery during orthoptic therapy in an exotropic amblyope with high unilateral myopia. Am J Optom Physiol Opt 1983;60:659-66.
55. von Noorden GK, Leffler MB. Visual acuity of amblyopic eyes in strabismic amblyopia under monocular and binocular conditions. Arch Ophthalmol 1966;76:172-7.
56. von Noorden GK, Leffler MB. Visual acuity of amblyopic eyes under monocular and binocular conditions: further observations. J Pediatr Ophthalmol 1972;9:8-13.
57. Cohen AH. Monocular fixation in a binocular field. J Am Optom Assoc 1981;52:801-6.
58. Campos E. Update on strabismus and amblyopia. ACTA Ophthalmologica Scan Supp 1995;214:17-25.
59. Daw NW. Critical periods and amblyopia. Arch Ophthalmol, 1998 Apr;116:502-5.
60. Keech, RV; Kutsche, PJ. Upper age limit for the development of amblyopia. J Ped Ophthalmol Strab 1995; 32: 89-93.
61. Kivlin JD, Flynn JT. Therapy of anisometropic amblyopia. J Ped Ophthalmol Strab 1981;18(5):47-56.
62. Attebo K, Mitchell P, Cumming R, Smith W, Jolly N, Sparkes R. Prevalence and causes of amblyopia in an adult population. Ophthalmol, 1998;105:154-9.
63. Flom MC, Kerr KE. Amblyopia: A hidden threat? J Am Opt Assoc 1965;36(10):906-12.
64. von Noorden GK, Crawford MLJ. The sensitive period. Trans Ophthalmol Soc UK 1979;99(3):442-6.

Sources

a. Bernell, Box 4637, South Bend, IN 46634-4637
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