Orthoptic treatment of vertical deviations

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Abstract: *Four patients with large vertical deviations were treated with a combination of prismatic glasses and orthoptics. The least amount of prism which eliminated diplopia, followed by horizontal fusional range extension, was prescribed. After vergences were normalized the prism was further reduced by two prism diopters and horizontal fusional range extension was repeated. This process was repeated until either a plateau was achieved or the prism was eliminated. All four patients completed therapy with almost total alleviation of symptoms and elimination of full-time prismatic correction.*

Key words: *hyperphoria, hypertropia, vertical deviation, prism, strabismus, orthoptics, vision training, vergence, fusion*

Vertical phorias are common binocular anomalies which occur in 7–25% of the population depending on the criteria of the study.[1-5] Scobee and Bennett[1] suggest that 9% of the population has symptomatic hyperphorias. The symptoms of a vertical deviation include headache, diplopia, loss of place while reading, drowsiness, fatigue, vertigo, nausea and motion sickness.[1,6]

Most authorities suggest that symptomatic hyperphorias should be corrected with vertical prism.[6] However, this may be difficult in the following cases: patients whose near and distance vertical deviations differ; patients who want to wear contact lenses; induced hyperphorias resulting from spectacle corrected anisometropia; patients with an additional torsional deviation; emmetropic patients who do not want to wear glasses; patients who require excessive prism; and patients with non-comitant deviations since the hyper deviation varies with position of gaze.

An alternate method of treatment is vision training or orthoptics. However, most authorities suggest that vertical fusional range extension is not effective.[7-9] Recently, Robinson and Kuhn[10] used orthoptics to treat three hyperphoric patients (range of deviation 1.5–4D of vertical phorias) by attempting to extend the vertical fusional ranges after improving ocular motilities and horizontal fusional ranges. They reported alleviation of symptoms in all three and an improvement in the opposing vertical range. Closer analysis of their data suggests that only one subject's vertical fusional ranges improved significantly, i.e., to 2.5D.

The differences between success in extending horizontal vs. vertical ranges may be explained by their respective motor characteristics. Schor has postulated that there are two components of the motor vergence system: a fast component which responds to vergence disparity, and is usually measured and easily trained; and a slow component which is adaptive.[11] The horizontal vergence system is thought to have both a large, fast component and a large, slow, adaptive component; the vertical system is thought to have a small, fast component (4Δ) and a larger, slow, adaptive component (10Δ).[12] The existence of only a small, fast vertical vergence system may explain previous failures to increase vertical fusion ranges with training. The larger, adaptive vertical fusional range suggests that treatment of a vertical deviation necessitates manipulation of the slow, adaptive system with a minimal regard for the fast component. Previous orthoptic attempts have ignored the adaptive vergence systems. We have utilized the adaptive system by prescribing the minimal vertical prism which relieves the initial symptoms (specifically all diplopia) followed by horizontal fusional range training. Upon normalization of horizontal ranges the vertical prism was reduced by two prism diopters followed by repeated horizontal fusional range training. The process was continued until either the prism was eliminated or the patient reached a plateau.

Case reports

Case 1

A 24-year-old female had a long history of intermittent left hypertropia. Her previous optometrist had increased the prism in her glasses every 1–2 years if she complained of diplopia. Prism had been first prescribed when she was 10 years of age. She presented wearing the following Rx: OD $-8.25 - 1.00 \times 85 = 4BU$; OS $-7.25 - 1.00 \times 85 = 4BD$. She would have preferred to wear contact lenses to eliminate frequent diplopia (which occurred 20% of the time), and desired better cosmesis with her glasses. She also complained of headaches and loss of concentration which usually occurred after 20 minutes of reading. She rejected surgery as a therapeutic option.

Extraocular muscle movements (Parks 3-step method) revealed the presence of a moderate left superior oblique paresis increasing in superior dextroversion. Cover testing with refractive correction in

place revealed a 12D intermittent left hypertropia (LH(T) with 3D of exophoria (X) at distance; and a 12D intermittent left hypertropia (LH(T′) with 6D of exophoria (X′) at near. Deviation with diplopia occurred approximately 30% of the time at distance and near. Near point of convergence was 4/10″. Distance base-in ranges were 4/2 and base-out ranges were 2/6/3 with her vertical phoria corrected. Distance left supra and infra ductions were 14/12 and −6/−8 respectively. Near base-in ranges (BI) were 6/10/4 and near base-out ranges (BO) were 4/6/8. Near left supra and infra ranges were 14/12 and −6/−8. Stereo acuity on the Titmus test at 40cm was 40 sec arc. A random dot stereogram (RDS) was appreciated indicating bifoveal fixation.[13] Accommodative amplitude determined by minus lens to blur was low (4.00D) as was her PRA (positive relative accommodation) (−1.25). Dilated funduscopic examination and slit lamp examination were negative.

The patient was given the following prescription: OD −8.25 − 1.00×83 4 1/2 BU 20/20; OS −7.25 − 1.00×83 4 1/2 BD 20/20 to eliminate diplopia and to reinforce binocular vision. The prism prescribed was the least amount of prism which eliminated any complaints of diplopia for 1 hour in primary gaze.

The patient was advised that the vision training prognosis was poor-fair to achieve the desired results, i.e., elimination of asthenopia, diplopia, and wearing of contact lenses. Training began with vectograms to extend both convergence and divergence fusional ranges while wearing the new prescription. The patient was encourgaged to use vergence cues, i.e., size and localization to provide feedback (Silo). Gross suppression was monitored by having the patient maintain stereoscopic depth and to note changes in perception of the target associated with changes in vergence, i.e., smaller for convergence, larger for divergence (Silo). Fusional ranges were also improved with Keystone stereograms™ and flat fusion synoptophore type stimuli presented in a stereoscope. Vertical range extension was attempted but failed to result in change in vertical vergence. Once adequate horizontal ranges were developed, i.e., BO = 40△, BI = 20△, which could be sustained for 30 minutes without fatigue, movement was added. The patient was encouraged to maintain fusion with both head and body movements. First the movements were slow but then were gradually increased. Lastly, targets were made smaller to reduce the role of reflex fusional movements.

The second stage incorporated traditional jump vergence training. Again, treatment began with vectograms, i.e., one which was disparited in a BO direction and the other BI direction. Training began with small jump stimuli and progressed until the patient could make rapid jumps from 25△ base-out to 15△ base-in and back again. Vectographic jump vergence techniques were supplemented with prism and stereoscope jump duction techniques. Home therapy using Brock string, loose prisms, and vectograms was given to reinforce office-learned skills.

The prism in the glasses was reduced by 2△ and training continued using the previously described techniques to improve smooth vergence and jump vergence skills. Once criteria were achieved, the prism was again reduced. Reduction occurred until either the desired results or a plateau was reached. In this case the prism was reduced to 2△ in her glasses and 2△ in a toric contact lens OS. The patient was prescribed a second pair of glasses with 4△ to be used in case of fatigue. The total process took 9 months of weekly sessions. At the end of therapy an alternate cover test revealed a small hyperphoria (2△) while wearing her contact lenses. However, with either prolonged cover testing or with total dissociation found during phoria testing, the original deviation could be elicited.

Three years after initial therapy, the patient noticed a partial recurrence of the initial symptoms of diplopia and headaches. Therapy was resumed for 3 months. Again the patient was dismissed almost asymptomatic with rare diplopia and no headaches. The patient was seen for a 4-year follow-up without a recurrence of symptoms. Cover testing revealed a 2△ left hyperphoria while wearing contact lenses. Phorometric testing indicated an 8△ left hyperphoria.

Case 2

A 27-year-old female had consulted an ophthalmologist with a complaint of diplopia of recent onset. The ophthalmologist changed her prescription from +2.25 OU to +3.00 OU to correct her latent hyperopia. She returned to him complaining that glasses were uncomfortable and did not eliminate the diplopia which was vertical in nature. Three months after her last examination, she consulted me for a second opinion. There were no other medical signs or symptoms.

Unaided visual acuity was OD 20/40, OS 20/40. A cycloplegic refraction revealed OD +3.50 and OS +4.00 while a dry manifest refraction revealed OD +2.75 20/25 and OS +3.25 20/25. Cover testing with her current prescription indicated a 6△ right intermittent hypertropia occurring 40% of the time with 2△ eso at distance, and 9△ right, intermittent hypertropia occurring approximately 50% of the time at near. Photometric testing revealed a distance phoria of 9 right hyper and a near phoria of 12 right hyper. Base-out ranges with the vertical deviation corrected were x/15/3 and base-in ranges with the vertical corrected were ×/10/4. Supraductions and infraductions were 13/10 and −7/−9, respectively. The Parks 3-step method indicated the presence of a mild right superior oblique paresis. Stereopsis was not appreciated on a random dot stereogram but was found to be 40 sec arc on a Titmus

stereo test. NRA was +2.00 and PRA was −1.50. Monocular accommodative amplitudes were reduced (5D).

The patient disliked her original glasses perceptually and cosmetically. She would only accept surgery as a last resort. The patient was advised of the need of a temporary hyperopic prismatic correction. Our goal was to prescribe contact lenses for daily wear and prescribe hyperopic prismatic glasses to be used in the evening.

Again the least amount of prism which eliminated diplopia for 1 hour was given which was as follows: OD +2.25 = 2BD and OS +2.50 = 2BU. Slit lamp and dilated funduscopic examinations were negative. A full medical evaluation which included a 5-hour glucose tolerance test, T_3, T_4 and TSH tests, was performed. All were negative. Since the supra and infraduction were asymmetric, and photographic review demonstrated a classic head tilt, a diagnosis of decompensated hyperphoria was made. The patient also had a secondary accommodative insufficiency as evidenced by the reduced visual acuity without glasses, low amplitude of accommodation, and reduced PRA. Therefore, orthoptic (vision training) treatment was advised.

Therapy again began with vectograms. A new instrument called Computer Orthoptics®[11] was used to build fusional ranges. Initially, the vertical was corrected while horizontal fusional ranges were increased using large targets (16°). An "auto" program was used to build sustaining ability by automatically separating the targets between established BO and BI vergence limits at a given speed. The advantage of this program was that the speed of vergence separation could be increased requiring faster, reflex fusional movements. Also, the targets could be made smaller requiring less disparity-induced fusional vergence or more voluntary fusion. In addition, a combination vergence and rotation program was used requiring more complex oculomotor movements. All other vergence training was similar to Case 1. The Computer Orthopter was also used to provide nonpredictive jump vergence training, i.e., vergence demand was randomly picked by the computer between parameters established by the doctor/therapist. The same methodology of decreasing the prismatic correction was used, i.e., the prescription was changed to OD +2.75 = 1.5 BD OS +2.50 = 1.5 BU.

The patient was fitted with contact lenses of the following prescription OD +3.25, OS +3.50. Final glasses were prescribed with the following prescription OD +3.00 = 1 BD, OS +3.25 = 1 BU. The patient was instructed to wear her contact lenses for 8 hours and her glasses in the evening. At the end of 7 months of therapy, she reported infrequent periods of diplopia occurring once or twice weekly at the end of the day, which disappeared after blinking.

A re-evaluation performed 9 months after therapy demonstrated a minimal right hyperphoria during a rapid cover test. Upon slow, prolonged cover testing, the deviation increased to 5 R hyper at distance and 8 right hyper at near. Phorometric findings were 10△ right hyper at distance and 12△ right hyper at near.

A 1-year follow-up indicated the following: slight hyper with an alternate cover test increasing in amount with prolongation of cover testing; phoria with her distance refractive error corrected was 8E with 13RH at distance and 10E with 14RH at near; phorometric findings with glasses were 3 eso with 9 right hyper. Base-out ranges through her current glasses at near were ×/24/16 and base-in were ×/14/6. Supra and infra findings were 18/15 and 6/−13. Extraocular movements revealed a mild right superior oblique paresis. Pre-and postorthoptic head tilt were similar. Preorthoptic and 1-year postorthoptic therapy motor fields were essentially the same as measured on the Computer Orthopter. She reported infrequent diplopia with her contact lenses at the end of the day. Diplopia could always be relieved with her glasses.

Case 3

A 34-year-old female was referred by a contact lens practitioner for an orthoptic evaluation. The patient stated that she had experienced intermittent diplopia since she was a child. She had been given push-up exercises which she stated were of no help. Since wearing contact lenses she had experienced a significant increase in diplopia associated with severe headaches. All other medical history was within normal limits.

Best corrected vision with her current prescription (OD −1.50 − 1.50 × 98 = 2BU and OS −1.50 − 1.50 × 90 = 2BD) was 20/20 OD and 20/20 OS, respectively. Cover testing revealed a 12 LH = 8× at distance and a 6 LH at near. Near point of convergence was 4/6. Extraocular muscle movements were full and concomitant with a mild V syndrome (6△ difference between up and down gaze). Phorometric testing revealed the following: distance 3× = 9LH; BI 6/3; BO 6/14/5; and near phoria 4×′ = 9LH′; BO′ 6/10/5, BI′ 6/10/6, PRA = −1.50 NRA +1.50. Vectographic testing resulted in normal fusional response, i.e. Silo, float, localization and parallax. Stereopsis was 60 sec on the Titmus stereo test and 40 sec on the Randot stereo test.

Therapy was directed to eliminate asthenopic and diplopic symptoms. The following glasses prescription was given: OD −1.50 − 1.50 × 98 = 3.5 BU and OS −1.50 − 1.50 × 90 = 3.5 BD. Prismatic determination was as previously described. Ocular health was within normal limits.

Again, therapy was directed toward the development of large, horizontal fusional reserves. Since the patient appreciated a random dot stereogram (RDS), vergence train-

ing incorporated these stimuli with the Computer Orthopter. Correct responses to RDSs provided feedback to the patient and to the therapist that bifoveal fixation was being maintained during vergence activities. Both tonic (ramp) and jump vergence (step) techniques were generated with a RDS. Jump vergence skills resulted in jumps from 50 BO to 20 BI. All previously described techniques were incorporated.

The patient terminated training with almost total alleviation of symptoms, i.e., diplopia occurring rarely in the evening after a stressful day. Either contact lenses or glasses could be worn depending on the patient's desires. During therapy, vertical range extension was attempted with only limited success. As in the other cases, rapid postth-erapeutic cover testing indicated the presence of a minimal deviation with a rapid motor recovery. Only on prolonged dissociation could the original deviation be elicited. Upon blinking, sensory fusion was regained.

Case 4

A 27-year-old had a partially accommodative, non-comitant, alternating left hyper esotropia. The turn was first noticed at birth and was confirmed by photo review. Previous treatment included patching at 3 years of age and spectacle correction. The patient was cosmetically bothered by the spectacle and esotropic appearance.

The initial examination revealed the following prescription: OD +4.50 − 1.75 × 65 20/25 and OS +5.75 − 1.75 − 15 20/25. Cover testing with the prescription on demonstrated a 45Δ alternating constant esotropia (ET) with 6Δ of left hypertropia at distance, and 60Δ of constant esotropia with 6Δ of left hypertropia at near. Distance/near ACA was calculated to be 12/1. A +3.00 add reduced the near deviation to 35Δ (gradient ACA = 8.51). Extraocular movements revealed a moderate, bilateral overaction of the inferior oblique. Suppression was noted on both vectograms, and large synoptophore first degree targets. Hering Bielchowsky and Bagolini testing revealed alternate suppression. Computer Orthoptics evaluation of subjective angle with a large target indicated harmonious ARC.

Due to the size of the deviation, the noncomitancy of the deviation, and the depth of suppression, surgery was advised. A left medial rectus recession and a left lateral rectus resection was performed. Two months postsurgically another orthoptic evaluation was carried out.

Cover testing revealed a cosmetically noticeable constant 12 LET = 10 LHT at distance and a variable constant 25 LET = 10 LHT at near. Hering Bielchowsky, after image testing, revealed unharmonious ARC and Bagolini testing revealed OS suppression. Troposcope testing measured 3Δ of eso combined with 5 LH with second degree sensory fusion and no movement on a Douse target test indicated NRC.[14] Computer Orthoptics (CO) subjective angle testing revealed 4Δ LET = 4LH. A cover test performed while the patient subjectively aligned the target demonstrated a left eso flick indicating ARC. Fusional range testing measured from the objective angle demonstrated both anomalous sensory and motor fusion responses. No stereopsis or fusion could be demonstrated with vectograms or Titmus stereo test. The following prescription was given: OD + 4.25 − 1.50 × 165 = 1 BU. OS + 5.50 − 1.75 × 15 = 1BO. The prism prescribed was equal to the opposing recovery value. The prescription of an add was deferred since the patient wanted to wear contact lenses.

Therapy again began with a prismatic correction and horizontal fusion training. Large targets (16°) on the CO were initially used to extend fusional ranges. Again the goal was to improve horizontal fusion ranges and to slowly eliminate the vertical prism. Vergence with rotations using the CO were trained; the goal was 15 BO and 10 BI. The targets were rotated at a speed of three cycles per minute. After 4 months of therapy, stereo acuity improved to 100 sec on the Titmus stereo test (Animal portion); normal stereoscopic appreciation was elicited on Topper vectogram; NRC fusion (no movement on a cover test) was found on the Worth 4-dot from 3″ to 10′. Therapy continued using vectograms, a Rotoscope, Keystone AN series, Worth 4-dot, and Keystone Stepping Stones cards, all building convergence and divergence fusional ranges.

After 8 months of therapy, a partial re-evaluation indicated a mild bilateral overaction of the inferior oblique; an intermittent subtle eso flick or alignment on unilateral cover test; and 8Δ esophoria with 4Δ of left hyper at distance and near with an alternate cover test. The patient was, therefore, fitted with toric soft lenses. The contact lenses resulted in a dissociation of binocularity. Again, vision training was reinstituted until the deviation was controlled. The patient was dismissed after 12 months of therapy with 100 sec of stereo and minimal overaction of the inferior oblique. The patient demonstrated bifoveal alignment approximately 80% of the time. When deviated, the patient was cosmetically straight. A 6-month postorthoptic therapy re-evaluation revealed ocular alignment with sensory fusion. The deviation was noted 20% of the time and only after prolonged cover test dissociation.

Discussion

The four cases presented represent a continuum of large hyper deviations. Case 1 demonstrated a continuously increasing hyperphoria which had been corrected with ever-increasing prism. Case 2 was either a recent onset intermittent hypertrope or a decompensated hyperphoria which seemed to stabilize. Case 3 was a patient with a long-term, stable hyperphoria with dip-

lopia which had been symptomatic without treatment for years. Case 4 was a congenital constant esotropic hypertrope. All had large hyper deviations which were controlled with orthroptic treatment.

Three of four were motivated to undertake therapeutic intervention by the desire to wear contact lenses. Therefore, they could not be corrected with vertical prisms. Thus, the only two options available to alleviate the patient's symptoms were surgery or orthoptics. Orthoptics, the safer, non-invasive procedure, was used.

Previous attempts to alleviate diplopia or asthenopia via orthoptics have been generally unsuccessful. Robertson and Kuhn[10] have recently suggested that vertical deviations could be treated with vertical range extension. They found that orthophoric patients could not increase their vertical fusional ranges with training while patients with vertical deviations could. Analysis of their data showed that only one of the three significantly improved their vertical range after vertical range extension techniques. The magnitude was small (2.5Δ).

Attempts to improve vertical ranges in three of the four patients presented in this paper resulted in failure, while one (Case 3) showed minimal improvement. The range of improvement was too small to handle the larger deviations presented in this paper. This concurs with previous findings that the vertical fast fusional mechanism is small and resistant to training.[12]

On the other hand, the slow, adaptive mechanism seems to be fairly large in the vertical direction. Ogle and Prager[12] demonstrated that by slowly adding prism to normal patients, most patients could adapt up to 9Δ of vertical prism within 30 minutes. Adaptation is defined as no diplopia or asthenopia with normal vertical fixation curves while wearing vertical prism. Both Ogle and Prager[12] and Carter[14] have found that after removal of the prism, the oculomotor system slowly returns to its status prior to wearing the prism. The rate of recovery is dependent on the total time the prism is worn. As a matter of fact, Carter[14] demonstrated that if vertical prisms are worn all day and are removed just before going to sleep, upon waking the tonic oculomotor position (phoria) corresponds to whatever position occurred before sleep. Thus, the adaptation is time-dependent, has a memory not altered by sleep, and is probably responsible for the maintenance of binocularity in patients reported to having hyperphorias as great as 20Δ as reported by Von Noorden.[8]

Using Schor's[11] model we postulate that the initial vertical prism prescribed removes diplopia by stimulating the fast fusion system to reduce vergence disparity to near zero. The output of the fast fusion mechanism is then fed into the slow vergence mechanism further reducing the disparity vergence to near zero. The longer the closed loop system maintains fusion, the stronger the slow vergence response is in decreasing the need for vergence disparity. The smaller the phoria, the easier it is for the fast vergence system to reduce the remaining vergence disparity to zero or near zero. The maintenance of the slow vergence system output (adaptation) after sleep may be important in explaining long-term orthoptic and prismatic therapy.

The treatment described in this paper improved the slow adaptative system by eliminating diplopia, stimulating fusion and reducing the need for vergence disparity through the feedback loop. This treatment required the prescription of the least amount of prism, which eliminated diplopia, followed by improvement in horizontal fusion ranges. This was followed by repeated prism reduction and horizontal vergence training.

We hypothesize that horizontal range extension was effective in reducing the motor defect in four ways. First, horizontal range extension resulted in reinforcement of both the motor and sensory fusion reflexes by reinforcing a rapid fusional movement in the presence of diplopia. Second, removal of disparity vergence in a closed loop system increases the adaptative response. Third, convergence training may have resulted in a subtle improvement in vertical ranges by moving the eyes into the field of action of the oblique muscles. Divergence moves the eyes into the field of action of the vertical recti muscles. Since most vertical deviations are secondary to (superior) oblique dysfunction,[15] convergence would stimulate contraction of the superior obliques with a resultant vertical fusional movement.

Lastly, Peter[16] and Scobee and Bennett[1] have noted that elimination of either the horizontal or vertical component often results in an elimination of the other component. Vector analysis elimination of the horizontal component may reduce the total muscle effort, thus leaving only the vertical component to be compensated for — a level for which the ocular motor system can more easily compensate.

Horizontal disparity vergence training seems to have improved the quality of the small vertical fast fusional systems since posttherapy cover test findings demonstrate a rapid redress toward fusion. Surprisingly, our patients with large vertical deviations often needed minimal amounts of vertical prism to maintain binocular vision. This phenomena has been previously reported by Von Noorden,[8] who describes a patient who decompensated into 20Δ hypertropia but only needed a minimal vertical prismatic prescription to regain binocularly. As previously stated we believe this response indicates a large adaptive response.

The methodology suggested here for treatment of vertical deviations need not be limited to patients with hyperphorias desiring nonprismatic correction or reduced prismatic correction, but also to small angle hyperesotropic and

other hyperphoric patients who tend to show yearly increases in vertical prism needed to maintain binocularity.

In summary, I have presented a novel method of treating hyper deviations by first correcting the vertical deviation with prism followed by vision training and then stepwise reduction of the vertical prism. ■■

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Footnote

a. Computer Orthoptics, 8200 Bay Parkway, Brooklyn, NY 11214.

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