TREATMENT APPROACHES TO: DIVERGENCE EXCESS INTERMITTENT EXOTROPIA

Intermittent exotropia is one of the most commonly encountered varieties of strabismus. Treatment is successful in approximately 90% of cases presenting for optometric vision therapy, according to well-documented studies using rigorous criteria.1-5 In this Viewpoints feature two authorities on divergence excess/intermittent exotropia describe their treatment approaches. Dr. Jeffrey Cooper, Clinical Professor at the State University of New York College of Optometry, has researched and published extensively on the subject. Dr. Nathan Flax, Professor Emeritus at SUNY College of Optometry and recipient of COVD's G.N. Getman and A.M. Skeffington awards, has published and lectured widely on optometric management of intermittent exotropia.

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Martin H. Birnbaum, O.D., FCOVD, FAAO
Contributing Editor
State College of New York, State College of Optometry

MANAGEMENT OF DIVERGENCE EXCESS INTERMITTENT EXOTROPIA

NATHAN FLAX, O.D.

Abstract

A method of treating divergence excess intermittent exotropia which differs from standard orthoptics is presented. Training is not done at the objective angle; ARC is ignored; diplopia awareness is not cultivated; stereoscopic targets are introduced before flat fusion or simultaneous perception targets; little emphasis is placed on developing base-out prism vergence ranges; monocular skills and accommodative facility are developed; and plus adds are utilized. The rationale for this approach is developed.

Key Words

exotropia, divergence excess, strabismus, orthoptics, vision therapy

For more than 30 years, I have been treating divergence excess intermittent exotropia in a highly successful way which produces excellent long-term control.[1,2] This approach differs from other methods in the following ways:

1. No attempt is made to make the patient aware of diplopia.
2. Training is not done at the angle of squint.
3. Anomalous retinal correspondence (ARC) is ignored.
4. Stereoscopic targets are utilized first, followed by second degree targets. Simultaneous perception targets are utilized last.
5. Relatively little emphasis is placed on increasing base-out prism vergence ability.
6. Monocular skills and accommodative facility are emphasized.
7. Near plus in bifocal form is utilized for long-term control.

The goal of my treatment is to achieve alignment without conscious effort by developing a postural vergence set for straight eyes. A basic premise of my approach is that tonic vergence, rather than fusional vergence to avoid diplopia, is the primary mechanism by which normal individuals maintain straight eyes. This differs from standard orthoptic methods which emphasize diplopia awareness and development of fusional vergence to overcome latent strabismus.[3,4] Diplopia awareness has little place in normal seeing. While it can be used as a tool to trigger fusion, this is not natural. Brock stated: "If the norm for any behavioral characteristic is determined by its frequency of occurrence, then it must be conceded that the awareness of physiologic diplopia is an entirely abnormal experience: ... The fact that you can 'teach' them to see double in a matter of minutes does not alter this fact."[5]

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A VIEWPOINT: INTERMITTENT EXOTROPIA OF THE DIVERGENCE EXCESS TYPE

JEFFREY COOPER, M.S., O.D.

Abstract

Intermittent exotropia of the divergence excess type is a unique strabismus with characteristic sensorimotor findings, symptoms and etiology. This paper reviews the most salient findings and incorporates these findings into a theory of etiology. Based upon etiology, a treatment regimen is presented which is consistent with clinical findings.

Key Words

exotropia, divergence excess, strabismus, orthoptics, vision therapy, anomalous retinal correspondence (ARC), vergence, fusion, stereopsis.

Classical teaching suggests that intermittent exotropia of the divergence excess type (DE) has the following characteristics: intermittency, normal stereopsis, rare amblyopia, good convergence amplitudes, and deep suppression during deviation. These patients align their eyes whenever stereoscopic information is placed in front of them. Some clinicians believe that the deviation seems to first appear at or around age 6 and that this deviation is a result of socially compulsive near tasks where there is an unchecked drive to release accommodation and thus convergence at distance. The result is divergent strabismus at distance. The etiology of DE, according to some clinicians, is an accommodative anomaly. However, this conclusion is untenable when one reviews the salient clinical findings. This is important since treatment should be consistent with both clinical and experimental findings. The following is a summary of a more comprehensive review on the subject which appeared in the Summer Edition of Binocular Vision and Eye Muscle Quarterly (1993).[1] This paper has 218 references encompassing most of the literature on DE, including a statistical analysis of the various treatment options.

Sensorimotor Findings

Duane originally identified two distinct types of intermittent exotropia—convergence insufficiency and divergence excess (DE).[2] Each type has distinct sensorimotor findings and responds differently to treatment. Burian described a third type of intermittent exotropia in which he believed that a divergence excess acquired a convergence insufficiency.[3] He labeled the third variety as basic exotropia. Both basic and divergence excess types of intermittent exotropia have similar sensorimotor characteristics; therefore they may be thought of as variants of the same anomaly. Costenbader[4] provided an early, clinically accurate description of DE: exophoria or exotropia at distance, normal nearpoint of convergence, adequate convergence amplitude, intermittency, equal vision, good stereopsis, and anomalous retinal correspondence (ARC) when deviated. Costenbader reported that the deviation was first noted in the majority of DE before 18 months. Progression often occurred until about age 6, at which time the strabismus became more noticeable. Only 6% of DEs are first observed after 5 years of age. DE is found more commonly in women and blacks. In addition, this strabismus has a strong hereditary predisposition. The refractive distribution is similar to the normal population, not skewed towards myopia as originally suspected.[5]

It is often reported that the ACA ratio is high in DE. This is a logical conclusion when one calculates the ACA using distance and near measurements. The average deviation is approximately 30 prism diopters at distance and five at near. Thus, assuming a 60 mm PD, the calculated average ACA is 16 to 1.

$$ACA = 6 + \left(\frac{-5-(-30)}{2.5}\right)$$

This, by definition, results in a high ACA. However, there are too many clinical findings which mitigate against a high ACA ratio. For example, if DE patients really have a high ACA ratio, they should complain about blur whenever they move their eyes in from the deviated position to alignment via the CAC cross-link. Also, occlusion for 45 minutes in over 60% of the DE patients results in a substantial increase in the near deviation so that the angular measurement approximates the distant deviation. This would cause a significant reduction in the ACA ratio. Since occlusion does not effect accommodation it should not effect the ACA ratio. Studies by Ogle and Dyer, who used gradient fixa-

tion disparity methods to measure the ACA in DE, found them to be about 3.5/1, a relatively low ACA ratio.[6] Von Noorden found normal ACAs using traditional gradient measurements.[7] Lastly, if ACA ratios were really high, one would expect a consecutive esotropia to occur at near after surgical intervention if orthophoria was created at distance. Happily for the surgeon this usually does not occur.

The distance-near phoria ACA ratio measurements are tainted by proximal convergence, depth of field errors, blur interpretation, vergence aftereffects, and pupillary changes due to the near triad. To measure objective ACAs, Cooper, Ciuffreda and Kruger used an infrared optometer to accurately and instantaneously measure accommodation, and an infrared eye movement system to measure eye position.[8] They found that in both true and simulated DE the ACAs were normal and equal, i.e., 5.9/1. Kushner, on the basis of these findings, measured gradient ACA in 83 DE patients.[9] He reported that over 90% of the DE patients had normal ACAs and the small percentage that did have high ACAs resulted in surgical overcorrection at near.

As previously mentioned, over 60% of DE patients who are occluded for over 45 minutes show a dramatic increase in their near deviation and a small increase in the distance deviation.[3] This is termed simulated DE. Those who do not show this change in deviation with occlusion are termed true DE. The increase in the deviation after sustained occlusion demonstrates that occlusion does not immediately result in a loss of all vergence- related responses. This phenomena is not unique to the intermittent exotrope. It is also observed in normal individuals. For example, if a normal individual wears a 25 base-out prism for 45 minutes, and then is occluded, the occluded eye will become esotropic with a slow regress towards orthophoria. If the occluder and prism are removed before orthophoria is regained, the subject will often report diplopia and an esotropia which decreases slowly over time. However, if fusion occurs immediately after the removal of the prism and occluder, there is an immediate esophoria which rapidly decreases. This demonstrates the strong effect that fusion has on the apparent position of the eyes. This effect has been called vergence aftereffects, prism adaptation, or slow vergence.[10] If the subject goes to sleep with the prisms in place, and they are removed during sleep, the subject will wake up with an esotropia and a slow recovery of normal binocular vision. This vergence mechanism is responsible for orthophorization in normal individuals. In addition, it is probably responsible for the smaller exo deviation at near, which is held in check by strong stereo cues initiating binocular alignment in DEs. Thus, the slow vergence mechanism helps remove the load of sustained vergence (fast vergence) created by the large exophoria/tropia.[11] Thus, simulated DEs have strong vergence aftereffects while the true DEs have strong proximal convergence. Strong proximal convergence accounts for the discrepancy between distance-near phoria ACA and the gradient ACA in true DE.

It is also noteworthy that most DE and basic exotropes are intermittent even though they both tend to deviate more upon distance fixation. The degree of intermittency is not dependent on the angle of deviation. Whereas 20' is assumed to be infinity in most ophthalmic examinations, this is not the case with DE. The deviation increases significantly in both amount and duration when fixation changes from 20' to 200'. When in bright sunlight most intermittent exotropes usually close one eye or deviate. Some clinicians believe that the closing of one eye is pathognomonic of a DE. When DEs have amblyopia it is usually mild and related to an anisometropic error.[1] Fusional amplitudes may be measured with a prism bar. A prism bar measurement allows the clinician to view the patient's eyes and check for suppression or anomalous responses. Surprisingly, DEs have normal convergence fusional amplitudes; on the other hand the divergence amplitude is usually less than the phoria measurement. This mitigates against the common perception that DE is the result of an inability to sustain fusional convergence or a result of a latent deviation becoming manifest. Fusion can be initiated by the presentation of stereoscopic detail, while deviation can be initiated by the presentation of simultaneous perception targets. Thus, the position of the eyes at any specific time is stimulus dependent. Said another way, DEs become binocular when there is an advantage, i.e., in the presence of stereoscopic information, and they deviate when there is no advantage—in the absence of stereoscopic information. Somehow DEs know apriori that a stimulus contains retinal disparity information.

When DE patients deviate, a variety of things can happen: They can see double with normal correspondence (NRC), or they can change their retinal correspondence to one of anomalous correspondence (ARC). Cooper and Feldman investigated how the DE "sees" during deviation.[12] They used a translucent hemisphere to present peripheral and foveal visual stimuli and monitored eye position with an EOG during both alignment and deviation. They found that during deviation DEs had an extension of the binocular field known as panoramic viewing. There was also a shift in retinal projection which matched the spatial world—harmonious anomalous retinal correspondence (HARC). In addition, they found that neither fovea suppressed during deviation. The foveas had different retinomotor values during deviation, but had the same retinomotor values during alignment. Thus, patients with DE have dual retinal correspondence. If the stimulus conditions are changed, ARC may be eliminated to produce NRC with or without suppression.[13] Therefore, retinal correspondence is not "wired in" but variable.

This phenomenon cannot be explained by Burian's classical adaptation theory.[3] Burian believed that ARC was a learned phenomena related to the age when the strabismus was first noted. Morgan[14] explained the phenomenon with changes in egocentric localization of afterimages with changes in oculomotor position. For example, if one puts an afterimage on one eye and if that person makes a version movement, the image moves with the eye, resulting in a change in egocentric localization of the afterimage, i.e., the afterimage moves to the side with eye movement (see Figure 1). On the other hand, if one makes a vergence movement, there is no change in egocentric localization, i.e., the afterimage appears straight ahead but smaller in size. In other words, if the deviation is initiated by a version, there is a change in egocentric localization which would match the objective angle resulting in HARC. Realignment, as predicted by Morgan, results in normal retinal correspondence (NRC).

What Causes DE?

Bielchowsky[15] thought that DE was a result of anatomical divergence of the

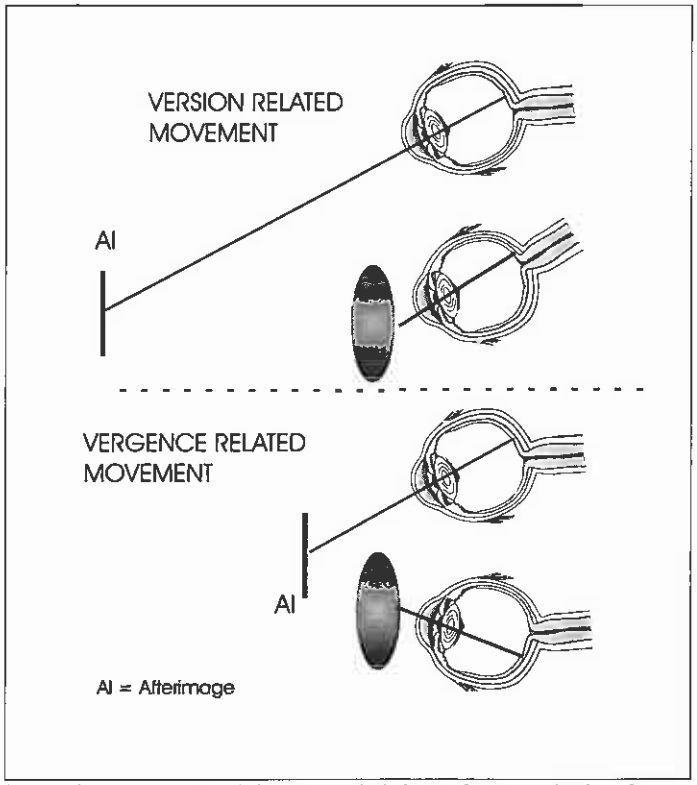

Figure 1. Top panel. The left eye is occluded. An afterimage is placed on the fovea of the right eye. The afterimage appears straight ahead. A levo version is then made. The afterimage appears to move to the left and appears off to the side of the patient. The movement is predicted by the angular movement of the eye. Thus, there is a change in egocentric localization but no change in oculocentric localization. In the bottom panel, an afterimage is placed on the fovea as in the first panel. With the eyes straight, the image also appears straight ahead. As soon as the eyes make a convergence movement, the image appears to become smaller and closer, but still straight ahead. There is no change in egocentric localization. In both cases the right eye made a levo movement, but in one there is change in egocentric localization while in the second case there is no change in egocentric localization.

orbits. Others have maintained that DE is a result of abnormal insertions of the extraocular muscles. Surgical exploration has not provided physiological support for mechanical theories. In addition, exotropia cannot be surgically induced in monkeys even if the medial rectus is disinserted. After disinsertion, the medial rectus often spontaneously reinserts with the development of normal concomitant movements.[16]

Duane thought that the deviation in DE was a result of active divergence.[2] There is strong evidence supporting Duane's hypothesis. Blodi and Van Allen[17] have shown that during deviation the lateral rectus increases its rate of firing with a concomitant decrease in the firing rate of the medial rectus. Also, the deviation is greater than the maximum fusible divergence amplitude (phoria is larger than the divergence amplitude). This strongly suggests that the position of the eyes during the tropia phase is not due to a relaxation of convergence or an inability to converge but rather to active divergence.

Posner[18] suggested that DE was a result of phylogenic development. Exotropia occurs with decerebralization of the cortex and a return to a lower level of binocularity (cerebellar function). Contrary to Posner, ocular placement of the eyes, straight ahead or laterally, is not related to phylogeny but to function. Animals which hunt have frontal position of their eyes for accurate depth location of their prey, while non-carnivorous animals, the hunted, have lateral position of their eyes to increase the peripheral field to detect danger.

I believe that the current information suggests that DE is a genetic anomaly that actually creates a purposeful deviation of the eyes.[19] Chavase noted that during the course of evolution from vertebrates to primates the eyes began to move frontally, with the final placement being related either to aggression or protection.[20] Protective mechanisms were dependent on the largest possible field of vision, while predation was based upon exact localization of the prey. Hunting animals such as man, monkey, wolves, tigers, cats, lions, etc. have frontal position of the eyes, while hunted animals such as rabbits, cows, horses, etc. have lateral placement of their eyes.

DE seems to be a functional compromise of these two visual systems. DEs have both stereoscopic vision when there is an advantage and panoramic viewing when there are few stereoscopic cues. Panoramic viewing increases the teleological sensing system to a full 300 degrees and serves to expand the motion detection system of the eyes. During near viewing when stereoscopic cues are plentiful, the eyes are usually aligned. An experienced clinician will attest to the fact that intermittent exotropes rarely deviate during stereoscopic testing. They demonstrate normal stereopsis on both contour targets and on random dot stereograms. Thus, DEs have normal binocular disparity detectors in the cortex. The deviation is reduced by a feedback loop from the fast disparity vergence system to the slow vergence system. Sustained utilization of slow vergence reduces the load on fast system and decreases the apparent phoria. At distance, where stereo cues are less abundant, deviation occurs, which decreases the output to the slow vergence system, resulting in an apparent increase in the angle of deviation. When deviation occurs, there is a shift in retinomotor values as predicted by Morgan's motor theory and ARC ensues.[14] I believe that this chameleon-like theory of being binocular, with resultant stereopsis when advantageous, and deviating at distance viewing or when stereoscopic information is lacking, is the true cause of DE.

Some clinicians have advocated that the basic etiology of DE is accommodative in origin. However, as previously noted, DEs have a normal ACA, relatively normal accommodative dynamics, and are first noted well before any socially compulsive near tasks occur. All of the abnormal findings reported are vergence related. Thus, it is difficult for me to accept an accommodative etiology.

Treatment

I have found that treatment which is not consistent with the physiological findings has not been very successful. For example, treatment to build fusional amplitudes in the DE who has normal fusional amplitudes does not result in a lasting cure. Treatment to eliminate the deviation by surgery does not result in a lasting cure. Surgical treatment should be the most successful when it is directed at creating a significant overcorrection so that the drive to redevelop original divergence excess deviation becomes mechanically impossible. Unfortunately the surgeon cannot create a predictable overcorrection.

Treatment consistent with my theory employs behavior modification whereby the visual system is initially stimulated with targets which elicit binocular alignment (stereoscopic targets). The left side of Figure 2 depicts in flow chart form the treatment of DE. After therapy, using stereoscopic targets, the cues which stimu-

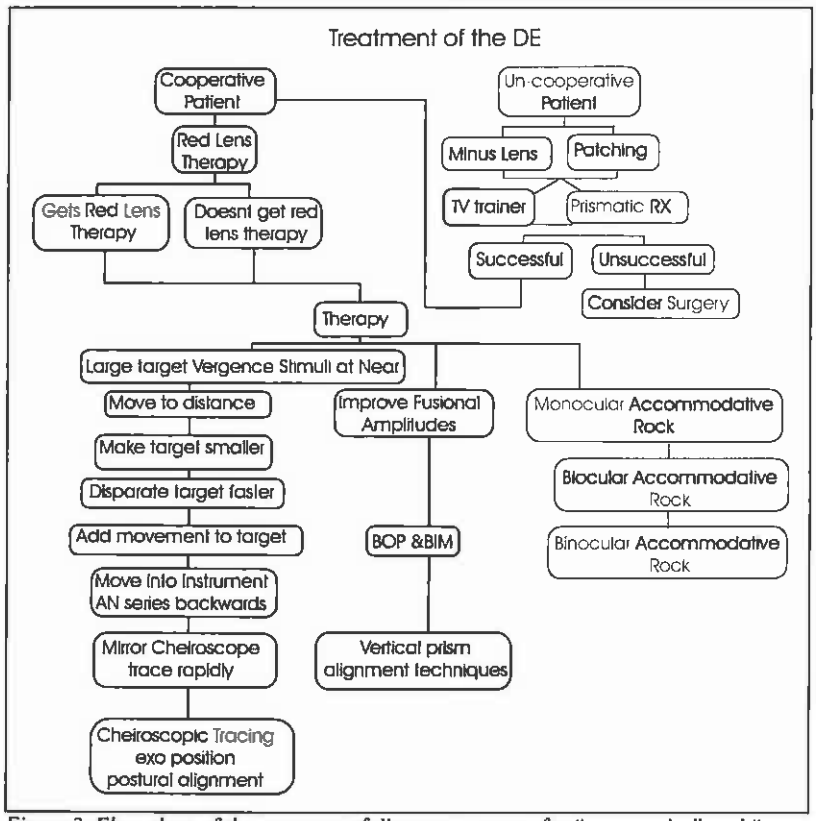

Figure 2. Flow chart of the treatment of divergence excess for "cooperative" and "uncooperative" patients.

late binocular alignment (disparity and similarity) are sequentially faded out while providing reinforcement. The end result is alignment in the absence of any fusion stimulus. Brock[22] was the first to advocate the use of large, peripheral stereoscopic stimuli to initiate alignment. He suggested that therapy begin at near viewing and then slowly move to distance where the stimuli become smaller, the retinal disparity induced by the stimuli decreases, and the patient is more likely to be exotropic. Before one moves from distance to near, the targets should be made progressively smaller. Cooper, et al[23] have experimentally shown that as the stimulus becomes smaller, fusion amplitudes and the ability to maintain binocularity decreases. When the patient demonstrates a consistent binocular alignment response, the disparity cues are decreased, i.e., flat fusion targets are used. The last step of treatment uses simultaneous perception targets which are devoid of vergence cues. They are initially presented in free space at 40 cm and then moved to distance viewing. There is always reinforcement for alignment. Some of the techniques I employ use a vertical prism for dissociation to obtain ocular alignment. Some other examples of simultaneous perception targets include: Cheiroscopic and Amblyopia Series with the Liquid Crystal Version of the Computer Orthopter,[a] mirror transfer techniques, and right view of the Clown Vectogram[b] with the left view of the Spirangle.[b] The last phase of treatment reinforces alignment in instrument in the absence of any cues for fusion. For some reason, I have found that binocular alignment without suppression is more difficult when targets are presented in a stereoscope.

The hardest task for the typical DE patient is cheiroscopic tracing. For some reason it is easier for the DE patient to initially eliminate suppression while using a mirror cheiroscope vs. a Brewster stereoscope. Initially, cheiroscopic tracings are performed in the exo position, the goals being no suppression and normal projection. The ultimate goal is to maintain an ortho or aligned position while performing a cheiroscopic tracing. One of the most effective techniques utilizes the liquid crystal version of the Computer Orthopter.[a] The butterfly and the box are used in Auto Vergence, Random Pattern modes. This superimposition is employed to disrupt suppression mechanisms. The patient's goal is to match the stimulus position with his/her eyes, i.e., using voluntary vergence to keep the butterfly in the box as the vergence demand changes. The final goal of treatment is the maintenance of binocular alignment in the absence of any visual cues, i.e., a blank visual field.

In terms of vergence training, I believe that if one can obtain divergence fusional amplitudes greater than the measured objective angle, that the deviation will probably disappear. This is because you will have eliminated all ARC and suppression responses. In any case, if convergence therapy is employed it must be balanced by divergence therapy. Personally, I do not advocate the use of plus lenses at near for the typical DE. I have not seen a difference in the long term success between those patients prescribed glasses and those not.

One of the problems that I encountered in my early career was that after treating patients with the previously described regimen, it was not unusual to have infrequent reports of the exotropia occurring during periods of fatigue, illness, or prolonged non-visual attention. The most vivid one I recall was a parient with DE on whom I had just completed a vision therapy reevaluation. The parent was very pleased because the child's eyes no longer deviated. Cover testing revealed orthophoria at distance and near. Prolonged cover testing did not elicit a deviation. There was no suppression on cheiroscope, etc. While I was patting myself on the back and completing the record, I noticed the patient from the corner of my eye. He was deviating without knowing it. I then tried to teach pathological diplopia to provide him/her with an additional cue that he/she was exotropic. This failed because I could not elicit a tropia during therapy.

Subsequently, I have changed my treatment regimen. I now employ pathological diplopia, i.e., diplopia awareness upon deviation, at the onset of therapy. The technique involves darkening a room, putting a red lens in front of the fixating eye, and then occluding until deviation occurs. I then interpose a muscle light or penlight to obtain diplopia. If this doesn't work, I oscillate the penlight or use vertical prism to elicit diplopia. If I can't obtain diplopia at this point I probably never will, and the prognosis for a cure decreases somewhat. (A cure occurs when the examiner can't elicit a deviation

with occlusion and the parent/patient states that the deviation rarely or never occurs.) If I can elicit diplopia, then I proceed to do the following, in order: decrease the oscillation, increase the room illumination, increase the transmission of the filter, move from near to far, and, lastly, dim the brightness of the transilluminator. When this procedure is finished, most of these patients will experience pathological diplopia. I always warn both the patient and parent that during this phase of therapy the patient may complain of diplopia. We will eliminate it later. Also, during this phase of therapy, no fusional therapy should be given, since we want deviation and diplopia. Once this is accomplished, I then begin therapy as previously described, stressing postural alignment. Although I do improve fusional amplitudes by utilizing voluntary vergence and cross-linking of both ACA and CAC with binocular accommodative rock therapy and accommodative rock with variable prism demands, the thrust of this therapy is postural alignment in the absence of cues.

Treatment initially results in the reduction in the amount of time of deviation. Secondarily, this presumably strengthens the slow vergence system to hold the eyes in alignment through a feedback loop. Demer has recently discovered that within Tenon's capsule there is a fine muscular circular pulley system through which the extraocular muscles pass through.[24] This pulley system is controlled by numerous smooth muscles. This may provide the mechanism for vergence adaptation. Over time, there are probably changes in the length of the extraocular muscles. The lateral rectus lengthens and the medial rectus muscle shortens. There is strong histological evidence for this. Goldspink and his co-workers have shown that striated mammalian muscles in both adults and children can adapt by changing to a new length.[25] This occurs by the addition or subtraction of sarcomeres at the tendon-myofibril junction. Thus, continuous vergence adaptation or slow vergence may cause a progressive change in the number of sarcomeres with a change in muscle length. The final result is permanent orthophorization. Lack of treatment should result in either no change in the deviation over time or a decreased ability to maintain sensory fusion over time. If the deviation occurs more frequently, current theories would predict a decrease in vergence adaptation with secondary muscular changes due to elimination or addition of sarcomeres. Sarcomere changes can occur within 7-15 days. The result would be an increase in both the size and frequency of the deviation. Thus, sensory fusion changes ultimately affect both neural and muscular function.

Non-cooperative or Unsuccessful Patient

Though I believe the regimen described has the greatest chance of success in treating DE, I will employ any other technique which I think might help (see Figure 2, Right Side). That includes patching, minus lenses, prismatic correction, and/or surgery. Young children, non-communicative patients, non-responsive patients, and patients who refuse vision therapy are provided these passive interventions. Usually I begin by alternately patching each eye for four-five hours per day for three months. I warn the patient that the deviation might increase with this procedure, but not to worry. If patching decreases either the apparent angle or the amount of time of deviation, I continue patching for another three months. Surprisingly, this has a positive effect in over 60% of the cases. I may also arbitrarily prescribe -2.00 OU over the patient's distance prescription. When over-minusing works, it is apparently independent of the ACA ratio. Rutstein and London[26] have shown that over-minusing probably does not result in an increase in myopia. If there is a residual defect, I sometimes add prism to eliminate the deviation by slowly tapering the prism over time. Lastly, if both these passive interventions and active vision therapy are unsuccessful, I believe surgery should be performed to eliminate the motor deviation. Optimally, the surgeon strives for a 10 diopter over-correction as long as the gradient ACA is within normal limits. Both you and the surgeon must be aware that if the gradient ACA is high, over-correction techniques are contraindicated. Over-correction techniques are also contraindicated in adults.

Success Rates

There is controversy over which is the best method to cure the intermittent exotrope. This probably means that there are many ways to correct DE, all of which are effective, or no one single treatment is always effective. Review of the literature suggests that orthoptics/vision therapy has the success rate of between 60-90%, while surgery has a 45-80% success.[1] It should be remembered that neither surgery nor vision therapy have ever undergone a rigorous double-blind, clinical trial to determine efficacy of treatment. Also, it is possible that the vision therapy and surgical population differ in magnitude of deviation and intermittency. I personally believe the best way to treat DE is to use all methods to increase the chances of success. I sometimes begin with patching, minus lenses and/or prism therapy before initiating active vision therapy. I then begin intense red lens therapy, followed by the therapeutic regimen that I have previously described. If we do not meet the patient's expectations, I will employ the skills of a well-trained strabismus surgeon. Immediately after surgery, we will resume a short-term vision therapy program. I believe this provides the best care for the patient.

Remember, no one obtains 100% success rate of anything. Also, remember the DE patient is relatively asymptomatic other than cosmesis. His/her binocular system may be functionally better than the non-DE individual in certain ways. Treatment eliminates certain binocular advantages, specifically an increased field of view during deviation. Treatment should be undertaken only when cosmesis or asthenopia are of concern to the patient or parent. I advise that treatment takes approximately nine months of once-a-week therapy with home therapy as a supplement. In my experience strictly home-based therapy for any exo deviation including convergence insufficiency rarely works. One needs to monitor, reinforce, and alter therapy on the basis of performance. There is no control with home-based therapy programs.

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a. R.C. Instruments, 10 Brinton St., Cicero, IL 46034 (800) 984-9661
b. Bernell, 750 Lincolnway East, South Bend, IN 46634-4637 (800) 348-2225

Corresponding author:
Jeffrey Cooper, M.S., O.D.
SUNY State College of Optometry
100 East 24th Street
New York, NY 10010
Date accepted for publication:
February 29, 1996

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Intermittent exotropes of the divergence excess type usually can demonstrate fusion, including high level stereopsis before therapy is initiated. Their binocular vision is closer to that of normals than to constant strabismics since they can demonstrate sensory fusion and bifoveal motor alignment at the same time, a condition that constant strabismics cannot meet. Their problem is not lack of sensory fusion, but rather an inability to maintain alignment on a consistent basis. My approach emphasizes reinforcement of straight eye posture rather than elimination of suppression or ARC when the eyes are turned. Suppression and ARC are considered to be adaptations that will fade when they are no longer useful to the patient. If the turn is intermittent, it is not even necessary to test for ARC. A guiding principle is that the sensory status when deviated becomes moot if the deviation itself is eliminated by developing tonic postural vergence. Training at the angle of turn is avoided and the problem of ARC is finessed. If present, ARC disappears as the patient gains motor control over the deviation.

My training regimen reverses the usual order of presentation of binocular stimuli. The usual schema of classifying fusion stimuli as first degree simultaneous binocular awareness, second degree flat fusion, and third degree stereopsis implies a hierarchical relationship which does not exist in the real world. Simultaneous awareness of dissimilar foveally viewed targets is not encountered under normal circumstances. It can occur only if the patient is strabismic or if a carefully structured artificial environment is created by use of a stereoscope, vectographs, anaglyphs, or a distorting device such as a Maddox rod. First degree binocular awareness is not likely to be a stage in the development of normal binocular vision. Flat fusion is also rarely encountered in the real world. There are almost no conditions in which the two eyes do not receive disparity cues. Even when looking at a flat page or CRT screen, the overall scene contains stereoscopic cues. Almost all natural stimulus conditions are essentially stereosacopic. It requires special manipulation of the visual inputs to achieve flat fusion.

Intermittent exotropes are unconsciously selective in the manner in which they utilize binocular vision, aligning the eyes when binocular cues are useful and allowing an eye to deviate when there is little benefit from binocular vision. The turn often occurs when "daydreaming." Parents of young children note that the child's eyes straighten whenever there is attention to the task at hand. For those patients whose turn is present at near as well as at distance, the push-up nearpoint of convergence may be receded. However, when the same target is utilized with instructions to localize it (as in the stick in straw test), convergence improves. These patients find it easiest to fuse stereoscopic targets. They find it more difficult to fuse and maintain alignment when there is no intrinsic benefit to binocular vision. This is the reason for my reversal of the standard order of presentation of stimuli. I begin with stereoscopic targets and gradually introduce second degree and finally simultaneous perception targets. The goal of treatment is to have the patient maintain straight eyes even when stereopsis is not useful or available.

Since the goal of treatment is to develop a postural set for straight eyes, no activities are done which would reward or reinforce the patient when the eyes are in the deviated position. Training is not done at the angle of turn. Binocular activities are introduced at the ortho setting utilizing stereoscopic targets since these are more likely to permit the patient to hold alignment. Straight eye posture is rewarded and the stimuli are gradually modified from stereoscopic to second degree to simultaneous perception with continual emphasis on holding posture. When the patient cannot hold alignment, the activity is modified by providing a stronger stimulus to fusion rather than calling attention to diplopia. Quality of fusion is emphasized, including attention to spatial localization and SILO when using vectographic and anaglyphic targets. Peripheral stereopsis using projected targets is the usual starting point of binocular treatment. Most of the early binocular training does not involve closed stereoscope or amblyoscope procedures because the patient usually cannot succeed in fusing at ortho in these instruments. Closed instrument tasks are introduced when the patient can perform at ortho. Third degree targets are used initially and then followed by second de-

It is generally not necessary to emphasize development of base-out vergence ranges since fusional convergence tends to restore spontaneously as the patient responds to the treatment approach. The notion of increasing fusional convergence assumes that the fundamental problem is structural exophoria. My approach is based on a different premise regarding the etiology of divergence excess. Early in my career I noted inconsistencies in the behavior of many divergence excess patients which could not be reconciled with a standard graphical analysis model of binocular vision. The positive relative accommodation-negative relative accommodation (PRA-NRA) relationship often was at variance with what might be expected for an exophore, with the negative relative amplitude high and the positive relative amplitude low. Some showed near esophoria which was not consistent with the distance deviation. During treatment, the near lateral phoria of some patients changed abruptly in the direction of esophoria despite the fact that the treatment had not been heavily involved with development of near convergence. The blur, break, and recovery of the prism vergence measurements were not always consistent, nor did they respond to treatment in the same way.

These behaviors are not readily explained by graphical analysis, but they are by the OEP-Skeffington model.[6] The B case type of Skeffington and divergence excess bear striking similarities.[7] The OEP model predicts development of farpoint exophoria as a response to an inability to sustain attention at near. At risk of oversimplification, the scenario is briefly as follows: The effort needed to function at demanding near tasks creates a drive to converge at a plane closer than accommodation. The patient becomes esophoric at near and must inhibit fusional convergence to maintain single vision. The habituated behavioral pattern of inhibition of convergence (or active divergence relative to accommodation) is an appropriate compensation to permit performance at near. This behavior pattern is then carried over to distance activities resulting in myopia or divergence excess. Such a patient would show near esophoria, reduced accommodative facility, a low PRA, and high NRA. The near base-out vergence finding would show a high blur since base-out prism permits convergence at a closer plane than accommodation. Once the break point has been reached, the recovery to fusion measurement would be low since the patient has been inhibiting fusional convergence. Each component of the prism vergence measurements has a different basis in this model and need not covary.

Skeffington[6] explains this sequence of adaptive behaviors as a response to the intense near demands of our society. Utilization of plus lenses at near satisfies the drive to converge closer than accommodate and permits the patient to maintain clear, single binocular vision without developing compensatory behaviors which then become inappropriate at distance. Application of near plus is expected to impact distance measurements by inhibiting the development of farpoint exophoria and/or myopia. Divergence excess strabismus usually begins long before school age and could not be due to the social demands of our culture in most cases. An alternative possibility is that a slightly high ACA ratio produces excessive accommodative convergence, requiring the patient to utilize fusional divergence at near to maintain single vision, setting a scenario similar to that postulated by Skeffington. Regardless of etiology, the measurement profile of the divergence excess patient is quite similar to that of the OEP B case. This similarity led me to treat divergence excess patients as if they were in fact OEP B cases. This has been my strategy for long-term management of these patients.

Most patients with divergence excess whom I have encountered demonstrate poor oculomotor control on a monocular basis and accommodative inefficiency along with their binocular problem, still further resembling the OEP B type case. These skills are trained along with fusion. Divergence excess patients treated in this manner follow the pattern predicted by the Skeffington model. Almost invariably, the measurements resemble a B case as alignment is achieved. At this point a near add is called for based upon OEP precepts, the near phoria is eso and the add normalizes both near prism vergence blur findings and the PRA-NRA relationship. The divergence prism vergence measures may actually be lower than the convergence range. Vergence range extension training may be undertaken at this juncture, but the purpose is to normalize binocular function rather than to develop compensation to overcome exophoria. Both divergence and convergence are developed along with voluntary convergence on non-fusable simultaneous perception targets. Near plus is prescribed in bifocal form to satisfy the drive to center (converge) closer than the plane of identification (accommodation). This has a salutary effect on the long-term stability of the condition. With the need to adapt at near relieved, farpoint exophoria (which was induced by the near adaptation) often reduces. Those patients who continue to utilize their reading lenses after formal training is completed often show gradual reduction of far exophoria over a period of years.

This approach to treating intermittent strabismus of the divergence excess type has been very successful. Patients respond rapidly and show little or no regression after completion of formal training. In addition to resolution of the cosmetic problem, improvement in scholastic and/or athletic performance is almost invariably reported.

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Corresponding author:
Nathan Flax, O.D.
585 Stewart Avenue
Garden City, NY 11530-4701

Date accepted for publication:
November 23, 1995