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Clinical Implications of Vergence Adaptation

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ABSTRACT

Placement of a prism in front of an eye results in a change in the tonic position of the eyes, a shift in the forced fixation disparity curve, and a shift in fusional amplitudes. These changes remain in effect as long as motor fusion is maintained. Elimination of fusion by occlusion or by removal of the prism results in a slow movement of the eyes back to the preprism position. This phenomenon, known as prism adaptation or slow fusional vergence, has important clinical implications in maintaining binocular vision with anisometropic prescriptions, age-related physiological changes in the positions of the eyes, blinking, high phorias, etc. Vergence adaptation is useful in explaining previous discrepancies between alternate and unilateral cover test, pre- and postorthoptic ACA ratios, stimulus and response ACA ratios, changes in phorias after orthoptics, and the observation of patients "eating up prism." Vergence adaptation anomalies have been implicated in causing asthenopia. Adaptation has been shown to change after orthoptic therapy. This paper reviews the clinical findings associated with vergence adaptation.

Key Words: vergence adaptation, fusion, phoria, strabismus, asthenopia, orthoptics, vision training, fixation disparity, exotropia

REVIEW OF VERGENCE ADAPTATION

Maddox[1] in 1893 described four elements of convergence: tonic, accommodative, voluntary (which he assumed was based upon a knowledge of nearness), and fusional convergence. Though not specifically included in his model, Maddox made reference to a fifth component based upon the knowledge of nearness. Hofstetter[2] described this type of convergence, which results from the sensation of nearness, as proximal convergence. Maddox[1] assumed that these components were additive. Tonic vergence or the distance phoria, which is a result of persistent activity of the vergence system, brings the eyes in from the position of rest. Tonic vergence is supplemented by both accommodative vergence and proximal convergence to provide coarse tuning at any fixation distance other than infinity. According to Maddox, the remaining vergence disparity induces a fusional vergence movement to eliminate diplopia.

Though Maddox did not include a sixth type of vergence in his model, he described a phenomenon currently known as vergence adaptation. He reported that if he wore an 11$^{\Delta}$ base-out prism for 10 min, upon removal his tonic convergence increased by 5$^{\Delta}$. In other words, his phoria changed from ½$^{\Delta}$ eso to 5½$^{\Delta}$ eso. Upon removal of the prism, Maddox noted that it took a few minutes for him to reestablish his initial phoria of 1/2$^{\Delta}$ eso. A similar response occurred with plus lenses, in which tonic convergence decreased. Maddox reported that these changes were adaptive and served to relieve stress on the fusional vergence system.

Alpern[3] reported that measurements on standard clinical fusional amplitude tests before the measurement of phorias resulted in a change in tonic vergence, i.e., distance phoria. The shift in the lateral phoria was always in the direction of the prism duction. Base-out prism had a greater effect on altering the phoria as compared to base-in prism when both ductions were performed, resulting in a greater propensity for esophoria. A similar finding was reported by Ellerbrock,[4] who demonstrated a shift in the vertical phoria after wearing a vertical prism which persisted 30 min after its removal. These findings have led to the common clinical dictum that phorias should be measured before vergence amplitudes and divergence amplitudes should be measured and/or trained before convergence amplitudes.

Ogle and Prangen[5] measured vertical forced duction fixation disparity curves and found them to be a straight line. Then they had their subjects wear a 6$^{\Delta}$ vertical prism for approximately 2 h. They found that the vertical fixation disparity curve measured through the adapting prism was almost identical to

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that before the prisms were worn (see Fig. 1). The time course for adaptation was exponential, with most of the adaptation occurring within a few minutes of wearing the prism. Adaptation to horizontal prism has been reported to be logarithmic by Ogle et al.[6] The time course for adaptation is related to both the amount of prism and the duration of exposure to the adapting prism.

Carter[7] showed that prolonged wearing of prisms not only altered the phoria position and shifted the fixation disparity curve but also shifted the fusional reserves for their subjects. His subjects completely adapted to horizontal prisms as large as 10$^{\Delta}$ base-in and 32$^{\Delta}$ base-out in 15 min. Most of the adaptation occurred within the first few minutes. After adaptation, fixation disparity curves, fusional amplitudes (including distance divergence amplitudes which are extremely reliable), and phoria measurements taken through the prisms were similar to the measurements before wearing the prism. These findings imply that wearing prisms causes an actual shift in the oculomotor position of the eyes. These adaptive changes remained stable for as long as the prisms were worn.

Removal of the adapted prism, when it is of a large magnitude, rarely results in immediate motor fusion because the aftereffects of adaptation decay over time. The recovery after adaptation upon removal of the prism generally takes from 15 min to 8 h if fusion is not allowed, i.e., dark, sleep, or diplopia. Carter[7] noted that esophoria, which was induced by wearing a base-out prism, was still evident after 8 h of sleep. "However, the esophoria disappeared after only 20 min of single binocular vision." Thus, sensory fusion with a compensatory motor movement is necessary for adaption or vergence aftereffects to occur. Carter has suggested that the previously described changes in tonicity

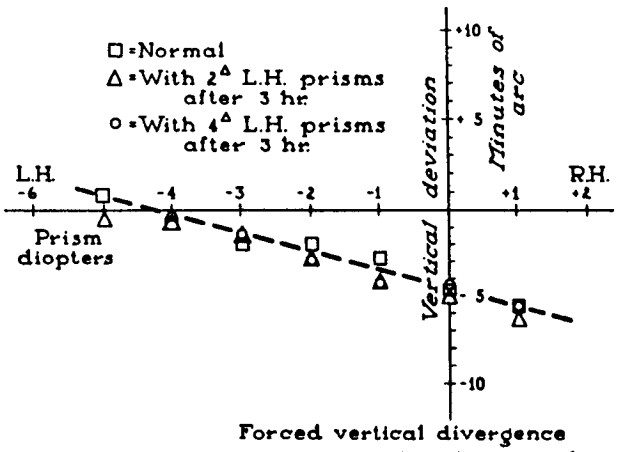

Figure 1. Forced vertical fixation disparity curves for a hyperphoric subject without prism and after wearing 2, 4, and 6$^{\Delta}$ of vertical prism. While wearing this prism, the subject demonstrates a forced fixation disparity curve similar to that found before wearing prisms. The subject has shifted his forced fixation disparity curve by adapting to the prism. (Reprinted by permission of the publisher from Ogle KN, Prangen A. Observations of vertical divergence and hyperphorias. Arch Ophthalmol 49:325. Copyright 1953, Am Med Assn.)

from adaptation serve to relieve the stress on the fusional or disparity vergence system. Carter stated that his subjects who demonstrated significant adaptation did not report discomfort from prolonged vergence. Carter concluded from these findings that patients with good sensory fusion adapt, whereas those with poor sensory fusion do not adapt and show symptomatic heterophoria.

Schor[8] incorporated the results of the previously described studies and his research findings into a model which explained the interactions of the various components of both the accommodative and vergence system. Specifically, Schor[9] described two types of vergence: a fast, reflex fusion system driven by retinal disparity vergence and a slow, adaptive system which received its input from the fast fusional disparity vergence system. According to this model the phoria is a vergence error which is corrected by fusional vergence. The slow vergence system or adaptor "reduces the stress or load placed on the vergence system by the heterophoria under binocular conditions." The sum of the slow and fast systems equals the total fusional vergence. According to Schor, fixation disparity is the result of incomplete prism adaptation and represents a purposeful error necessary to sustain vergence.

Schor[8,9] has suggested that prism adaptation varies from individual to individual, varies with time of adapting prism, and varies with the direction of the adapting prism. Rapid prism adaptation, according to Schor,[10] is highly correlated with the shape of the fixation disparity curve. The steeper the curve the poorer the prism adaptation. According to Schor's model the output from the fast fusion system decays similar to a leaky neural integrator over a 10- to 15-s period of time. On the other hand, the slow disparity vergence system increases its output to relieve the fast fusion mechanism which maintains a constant total output (see Fig. 2).

The phenomenon of vergence adaptation also occurs with nonconcomitant vergence disparity. Cusick and Hawn[11] and Pascal[12] measured the phoria in both primary and in depressed gaze in spectacle-corrected anisometropes. They found that the induced hyperphoria was eliminated by adaptation. Ellerbrock and Fry[13] measured vertical phorias in 42 anisometropes in both primary and depressed gaze. Orthophoria was measured regardless of the induced prismatic error created by the anisometropia. They postulated that there was a continuous adaptive process which occurred over the entire oculomotor field. Similar findings have been reported by Allen[14] and Henson and Dharamshi.[15]

Henson and Dharamshi[15] measured the time course of adaptation with 1.00, 3.00, and 4.00 D of induced anisometropia. They reported that adaptation rates were similar for both concomitant vs. noncomitant stimuli and spread to areas where visual experience was not allowed. Interestingly, adaptation was more complete in downgaze (where there was more visual experience) than in upgaze.

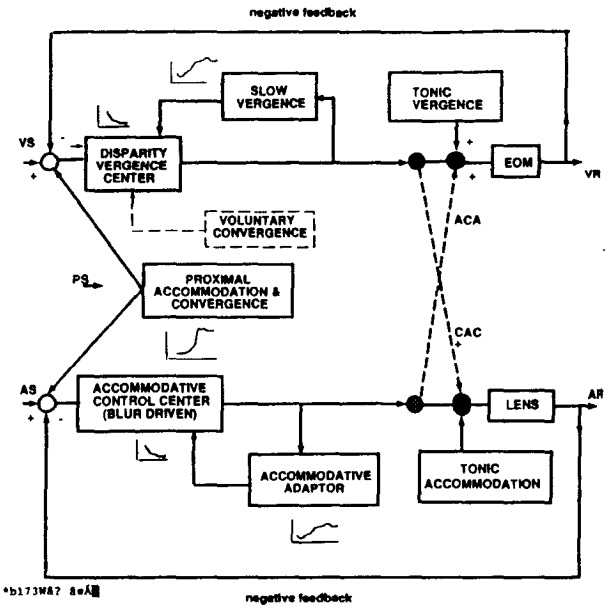

Figure 2. Analysis of closed-loop and individual elements of vergence responses to step stimuli created by a prism. Fast or disparity vergence supplies most of the initial response. As the disparity vergence response decays, slow vergence makes up the lost difference. Removal of prism eliminates the remaining fast disparity vergence signal. Because slow vergence or adaptation has a longer time constant, its demise is much longer. The final endpoint of decay is equal to the level of tonic vergence.

Henson and Dharamshi postulated that a cortical motor memory map developed with each point influenced by its neighboring points. Sethi and North[16] showed that adaptation was improved immensely if the prismatic steps were small.

Schor's[8] model, incorporating a slow vergence system with very long decay time constant (greater than 30 s) into Maddox's model, is very useful in explaining a multitude of clinical observations. It also has implications in diagnosis and treatment of various oculomotor anomalies. Fig. 3 presents a block diagram to depict the vergence feedback based upon the findings of Schor,[17] Ciuffreda,[18] and Ciuffreda and Hung.[19]

CLINICAL IMPLICATIONS OF VERGENCE ADAPTATION

Cover Test

The most commonly performed measurement of oculomotor integrity is the cover test. Testing is usually done while the patient fixates an accommodative target at both 6 m and 40 cm. It is often noted that the initial measurement of the angle of deviation is not stable. With repeated alternate occlusion the angle of deviation often increases. The increase in angular measurement seems to be dependent on: (1) the size of the initial latent deviation; (2) the duration of occlusion; and (3) the strength of vergence adaptation. The increase in the angle is a result of a rapid decay of fast fusional vergence by occlusion followed by a longer decay of the slow fusional vergence response.

Stated another way, a measurement which increases with repeated alternate occlusion represents an initial elimination of fast fusional vergence followed by a subsequent elimination of slow fusional vergence. Conversely, removal of an occluder during a unilateral cover test permits fusion to reoccur. This results in stimulation of the fast fusional vergence system which feeds into the slow fusional vergence system. Repeat occlusion with unilateral cover testing results in the elimination of fast fusional vergence signals with minimal effect on slow fusional vergence signals because slow fusional vergence has a long time constant. Therefore, the deviation measured with the alternate cover test is

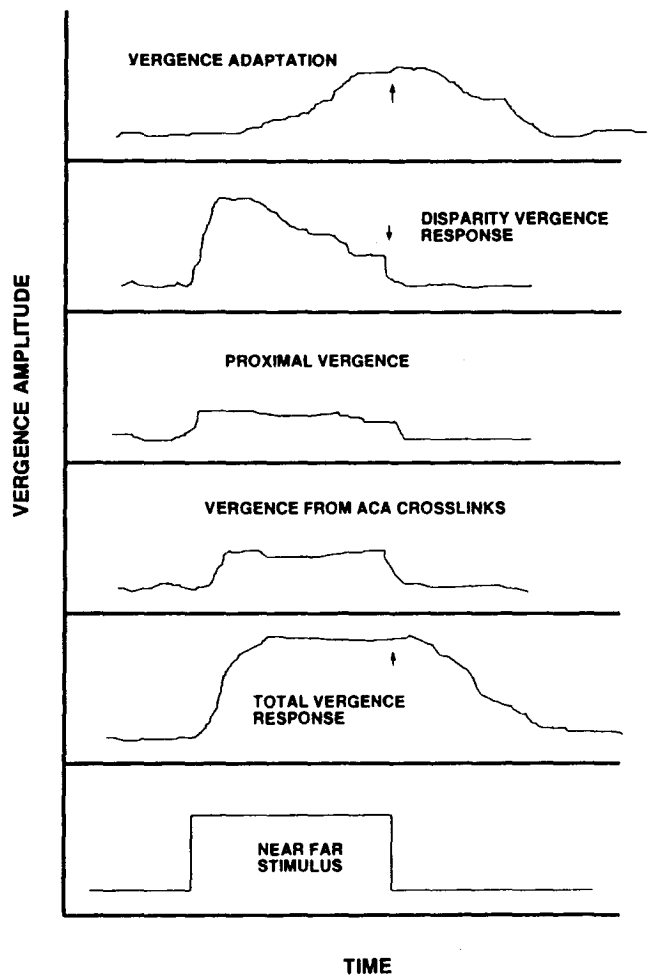

Figure 3. Block diagram depicting the interaction of the various components of accommodation and vergence to create a closed-loop negative feedback system. Disparity vergence has a rapid rise and slower decay. As disparity vergence decays, slow fusional vergence increases its output and sustains the vergence response. The slow fusional vergence system is driven by a signal created by the difference between the signal to the fast fusion system and the output from the fast fusion system. The vergence response is also supplemented by proximal vergence and the cross-links from the ACA ratio. Accommodation has a similar servomechanism driving it.

usually larger than the amount measured with the unilateral cover test. The former is a result of the sum of the fast and slow fusional vergence system, whereas the latter is a measurement of the fast fusion system. Patients who show strong vergence adaptation and have high latent phorias will increase the angle of deviation with prolonged, repeated alternate cover testing; patients with large phorias and weak slow vergence systems will not. Thus, patients with robust vergence adaption are less likely to be symptomatic, inasmuch as the slow fusional vergence system eliminates the constant demand on the fast fusional vergence system.

Burian and Smith[20] noted that both intermittent exotropes and normals often show significant increase in the measurement of their deviations when fixation changes from 6 to 30 m (20 to 100 ft). At greater viewing distances there are fewer stereoscopic cues and less fusible detail, thus decreasing reflex fusional vergence and subsequent slow fusional vergence. This predictably results in a larger deviation at a farther distance.

Prolonged Occlusion

Marlow[21] popularized a technique of prolonged occlusion to uncover a latent heterophoria. He used prolonged occlusion when traditional methods of refractive or prismatic correction failed to provide relief of asthenopia. The test consisted of prolonged occlusion followed by measurement of a phoria with a Maddox rod. Care was taken not to allow any opportunity for fusion during testing.

Beisbarth[22] studied 29 patients with prolonged occlusion (1 h to 9 days). Thirteen of 16 eyes with preocclusion hyperdeviation showed a significant increase in their deviation, 5 of 8 orthophoric patients demonstrated a postocclusion hyperphoria; and the final 5 showed minimal changes with occlusion. Beisbarth suggested that prismatic correction based upon prolonged occlusion would result in overcorrection of most vertical deviations. Prolonged occlusion eliminates vergence aftereffects, resulting in an increase in both the number of patients with a vertical deviation and in the amount. However, robust vergence aftereffects eliminate the load on the fast fusion system and reduce asthenopia and diplopia. There is controversy whether prolonged occlusion produces a true deviation or just an exaggeration of Bell's phenomenon. For a comprehensive review of this topic see Amos and Rutstein.[23]

The important point to remember is that the cover test does not measure the "true phoria." Elimination of all fusion-related impulses requires prolonged occlusion. The "latent deviation" identified may in some cases be the elusive cause of asthenopia in patients. Thus, prolonged occlusion may be used to identify binocular-induced symptoms by the elimination of binocular fusion impulses and to measure vergence aftereffects.

In 1952 Scobee[24] occluded intermittent exotropes of the divergence excess type for 1 h; he reported that a significant percentage of them increased their angular measurement at distance and near after occlusion. Burian,[25] based upon the results of occlusion, classified the divergence excess type of intermittent exotropia (DE) into two groups. One group, which he called simulated DE, responded to occlusion by increasing the angle of deviation at near so that it approximated the distance deviation (this represented 60% of the cases). The other 40%, which were not affected by occlusion, were called true DE.

ACA Ratio

Burian[25] suggested that differentiation between simulated and true DE was important because each required a different surgical procedure. This finding was confirmed by von Noorden[26] though it has been subsequently denied by other surgeons. This finding with occlusion is important in understanding the physiological mechanisms responsible for sensory-motor functioning in DE. Before this finding, most authorities reported that DE was associated with a high ACA ratio. Using the distance near formula, the minimum calculated ACA for a DE patient, where the distance deviation is at least 10 Δ greater than the near deviation, has to be at least 10/1.[27] However, Scobee's[24] and Burian's[25] observations with occlusion suggested that occlusion decreased the ACA ratio to approximately 6/1. Occlusion should not have altered the true response ACA ratio.

Cooper et al.,[28] using an infrared measurement system, measured accommodation and vergence simultaneously to determine objective, response ACA's. They demonstrated that response ACA's in patients with intermittent exotropia for both simulated and true DE were normal (mean = 4.9) and did not change with occlusion. It should be remembered that all response ACA measurements, by the nature of testing, use prolonged occlusion and therefore eliminate vergence aftereffects as opposed to clinical stimulus ACA measurements.

Cooper et al.[28] postulated that the difference in ACA ratios between objective, response ACA's and subjective, stimulus, distance-near ACA ratios in patients with DE was due to the additive effects of both vergence adaptation (slow fusional vergence) and proximal convergence findings. They postulated that most patients with simulated DE have a robust slow vergence system, whereas most true DE patients use excessive proximal vergence. Obviously, some DE patients have mixed systems.

Kushner[29] substantiated the findings of Cooper et al. by studying gradient ACA's in intermittent exotropes. He reported that the majority (93%) had normal gradient ACA's. Only 7% of all DE patients had a true high ACA. In those few DE patients who had both high gradient and distance-near ACA ratios, Kushner reported that surgical correction resulted in a near esotropia. Thus gradient ACA's, near-far ACA's, and occlusion testing should be done on intermittent exotropes to determine the

following respective components of the deviation: accommodative-vergence, proximal vergence, and vergence aftereffects. DE patients with high gradient ACA's who show minimal effects with occlusion should not have as a surgical goal alignment or overcorrection. Surgery, if used, should undercorrect the deviation followed by vision training to compensate for the residual deviation. The goal should be to create strong vergence adaptation coupled with reflex fusional vergence.

Treatment of DE patients with normal ACA's also requires improvement of both fusional vergence amplitudes and slow vergence to reduce the load on the fusional vergence mechanism. A strong slow vergence system results in an apparent change in the tonic position of the eyes, i.e., orthophorization, and a decrease in the distance-near ACA ratio measurement. Surgical correction appears to be most successful when both fusional and adaptive vergence components are eliminated.

After orthoptic therapy, rapid alternate cover testing often shows a reduction in any deviation. This change in tonic position has often been taken as evidence of a change in the neurological gain of the ACA cross-link system (increase in the ACA ratio). However, because prolonged occlusion eliminates this effect, alteration of the slow vergence system must be involved in the change. Thus, strong fusional vergence training in the direction opposite to the phoria should result in a reduction in the apparent phoria (orthophorization) and a reduction in the load on phasic disparity-driven fusional vergence.[30]

Adaptation to Prescribed Prism

Carter[31] described a patient who was prescribed compensating prisms for a moderate exophoria. After a short period of time the patient exhibited the original phoria while wearing the prism. According to Carter there was a "shift in the fusion free position to maintain the same demand on fusional convergence that existed prior to wearing the prism." This phenomenon of adaptation has been described as "eating up the prism" and occurs for horizontal and vertical phorias. Eating up the prism is a direct result of elimination of fusional disparity by prism with a subsequent increase in adaptation.

Carter described another patient given a prescription for an asymptomatic 1^{Δ} left hyperphoria. Upon reevaluation 1 month later, the patient measured 1^{Δ} left hyperphoria through the prismatic prescription that he was wearing. A new prescription was given incorporating the additional prism. A subsequent recheck 3 weeks later again revealed 1.5^{Δ} left hyperphoria. The spectacles already had 2^{Δ} of vertical prism; thus the patient was really manifesting 3.5^{Δ} of left hyperphoria. Carter removed the prism. Retesting 1 week later revealed 1.5^{Δ} hyperphoria without any symptoms.

Patients similar to the one described by Carter, who appear to be eating up the prism, are actually adapting to the prism. Clinically, these patients may be identified by having them wear the prism for 20 min in a trial frame. If the patients adapt, relieving prisms are contraindicated. Thus, short-term evaluations are valid because the process of adaptation does not disappear with long-term wearing of prisms. Carter suggested that patients with good adaptation are usually asymptomatic, whereas those with poor sensory fusion are usually symptomatic due to poor vergence adaptation. Carter suggested that patients who are symptomatic and demonstrate poor vergence adaptation would benefit from prismatic correction. North and Henson[32] have confirmed Carter's findings that symptomatic patients demonstrate poor vergence adaptation, i.e., slow fusional vergence.

Orthophorization

Adaptation is probably responsible for the leptokurtic distribution of distance phorias. Morgan[33] noted that 76% of all patients have a distance phoria between 1^{Δ} of esophoria and 3^{Δ} of exophoria. Adaptation serves to eliminate the apparent phoria through the feedback mechanism from the disparity vergence system. After initial motor fusion subsequent to disparity vergence, slow fusional vergence with its long-time decay maintains the eyes in alignment over a period of time. The resulting orthophorization may be disrupted by prolonged occlusion. If fusional vergence and vergence adaptation are improved with orthoptics, there should be a reduction of the apparent phoria or orthophorization.

Slow fusional vergence is probably responsible for maintaining ocular alignment during blinking. Short or long blinks disrupt sensory fusion, but have little or no effect on the slow fusional vergence signal. Therefore, upon opening the eyes after blinking or sleep, one would expect alignment of the eyes even in the presence of a high phoria. One may conclude that the fast sytem is responsible for initiation of sensory fusion, whereas the slow fusional vergence system is responsible for maintaining oculomotor position over time.

Binocular Analysis

These findings show that Maddox analysis or graphical analysis needs to include both proximal and the important slow (adaptation) vergence controller system. The above findings suggest that the ability of the binocular system to maintain comfortable binocular vision at a given fixation distance is related to the strength of the slow vergence system.

Schor[34] believes that the shape of the fixation disparity forced vergence curve is reciprocally related to vergence adaptation; i.e., the flatter the curve the better the vergence adaptation. Large associated phorias would represent a steady-state error for the fast vergence system. If adaptation is weak, the position of the fixation disparity curve may be altered with prism or surgery. Orthoptics

causes the slope of the fixation disparity curve to flatten and eliminates asthenopia.[35-37] If orthoptics flattens the curve enough and sustained vergence skills (isometric) are developed, the curve may also be shifted.

Wick and London[38] have developed a system of analysis based on measurements made under binocular testing. They take into account not only open loop findings (phoria, vergence amplitudes, ACA ratio, CAC ratio, and proximal vergence), but also binocular closed negative feedback loop findings (binocular proximal convergence, accommodative lag, accommodative facility testing, and slow vergence adaptive changes).

It should be remembered that all the methods for binocular vision analysis previously described ignore certain components of the patient's behavior which must be accounted for. For example, accommodative and vergence testing often create discomfort irrespective of their measured amplitude. The effort to sustain binocular vision during testing is not easily quantified and so is usually ignored. Clinically, the patient who displays asthenopia after measurements of accommodation and vergence amplitudes is most often the patient who benefits from orthoptic therapy.

Other commonly ignored factors include: (1) individual pain thresholds, (2) total amount of time spent at near tasks, (3) total amount of time spent at near tasks without a break, (4) amount of visual detail, and (5) cognitive demand. Last but not least, most of the vergence studies cited assume that open loop data can be applied to closed loop accommodative-vergence functioning. Though a more comprehensive accommodative-vergence analysis, as proposed by Wick and London, is more accurate, it is not complete because the above described variables have not been accounted for.

Vergence Adaptation with Physiological Change

Concomitant-type vergence adaptation effects are important in maintaining binocular alignment changes that occur over time. Sethi and Henson[39] have shown that adaptation increases when the prism is introduced in small steps. Each small step reaches a larger proportional amount of adaptation which is additive to the preceding step. Sethi[40] postulated that small step adaptation may account for gradual physiological changes in ocular position that occur with both age-related and slowly advancing pathological processes. For example, with advancing presbyopia, vergence as a direct result of accommodation decreases, thus causing the near phoria to become more divergent or exophoric. This increases the demand on the fusional convergence system which results in constant output from the slow vergence adaptor. Thus, adaptation serves to eliminate the increased demand on fusional vergence which results from a loss of accommodative vergence. Clinically, this may be seen in patients who present with orthophoria or a small exophoria on an initial cover test but who, with prolonged repeated occlusion, demonstrate an increasing divergent deviation. Prolonged sustained vergence maintains vergence adaptation, whereas prolonged repeated cover testing eliminates fusion and, consequently, vergence adaptation with its long time course.

Similar adaptive findings would be expected to occur with enophthalmos, which increases with age, tumor-generated proptosis, and slowly increasing exophthalmos secondary to Graves' disease. Rapidly occurring paresis secondary to trauma, vascular accidents, etc., results in diplopia and lacks slow vergence adaptation.

Often, patients with congenital deviations such as a congenital superior oblique paresis or congenital facial asymmetries (resulting in one orbit being higher than the other) are relatively orthophoric or demonstrate significantly less vertical deviation than expected. The binocular system of these patients may decompensate in later years. They must be differentiated from the person with a newly acquired superior oblique palsy who has no adaptation. Patients with congenital superior oblique paresis often show, with repeated occlusion, a slowly increasing vertical deviation. Their opposing infraduction is usually much greater than the expected findings, i.e., 4^{Δ}. Also, the amount of prism needed to eliminate the diplopia is much less than expected.

Cooper[41] has described a treatment regimen for decompensated vertical deviations. He prescribes the minimal amount of vertical prism to eliminate diplopia by stimulating fusional vergence. This is followed by orthoptic therapy to improve horizontal fusional amplitudes. Upon normalization of horizontal fusion amplitudes the prism is reduced in 2^{Δ} steps to encourage adaptation followed by subsequent orthoptic treatment. Often the prism can be eliminated entirely. The full amount of vertical prism measured is prescribed in cases of recently acquired deviations in order to eliminate diplopia; then the clinician should attempt to decrease the prism slowly and encourage adaptation.

Cooper believes that vergence therapy should include: (1) slow ramp vergence training to improve fusional vergence; (2) continuous ramp therapy to improve the vergence reflex which eliminates constantly changing disparity vergence signals; (3) jump or step vergence to improve recovery once sensory fusion is lost; and (4) sustained vergence training (isometric) to improve adaptation.[42] Slow, continuous, and step vergence therapy must be practiced with various binocular stimuli until these skills become an effortless, automated reflex.

Noncomitant Vergence Adaptation

As mentioned previously, Henson and Dharamshi[43] and Sethi and Henson[39] have shown that noncomitant disparity induced by anisometropia results in adaptation across the entire oculomotor field. Adaptation is maximal where binocular experience usually occurs (primary position and

downgaze) and spreads from one position to another. Sethi and Henson[39] suggested that there is a neurological memory map to maintain a compensated stable "orthophoric" position. This memory map accounts for different amounts of adaptation at different distances contributing to the distance and near phoria measurements.

These findings explain why some patients are asymptomatic with anisometropic prescriptions. The induced prismatic effects of an anisometropic prescription are compensated for by a robust adaptation system. Patients with a weak adaptation system require prism to decrease the demand on fusional disparity vergence, which subsequently decreases vergence adaptation. The result is an apparent increase in the deviation and the need for a stronger prism. The patient appears to eat up the prism. Thus, patients with a poor ability to adapt to prism should have judicious application of prism. They should be advised of the necessity for subsequent orthoptics to improve both fusional and adaptive vergence.

With large anisometropic corrections, such as for unilateral aphakia, the prismatic deviation may be too large to be overcome by fusional vergence. Diplopia occurs without allowing for adaptation. This author believes that the large, noncomitant, vertical prism induced by the anisometropia is the deterrent to single binocular vision rather than the aniseikonia. Patients with smaller anisometropic prescriptions rely on fusional vergence to eliminate the diplopia. If adaptation is robust, the induced error does not produce symptoms. If either fusional vergence or slow fusional vergence adaptation is weak, diplopia or asthenopia will ensue. Correction will require a slab-off prism.

A similar situation occurs with an incorrectly centered ophthalmic lens. The higher the prescription the higher the induced prism. The incorrectly incorporated prism becomes adapted to. The "correction of the problem" often results in recentering of the lenses to match one's interpupillary distance. Unfortunately, a new prismatic demand has been created which needs to be adapted to. Asymptomatic patients with prism induced secondary to incorrect optical alignment should be slowly weaned off the inappropriate optical centers. Symptomatic patients should have their optical centers properly aligned.

CONCLUSION

Vergence aftereffects are a distinct type of vergence which receives its input from fast fusional vergence, and has a long time constant. Clinically it is important in explaining changes in phoria position with sustained occlusion such as Marlow occlusion, changes in ACA ratio, decompensation of hyperphoria, changes in prismatic correction in patients who eat up prism or adapt to spectacle-induced prism, and adaptation or nonadaptation to anisometropically induced prism. Therapeutically, the adaptation mechanism can be manipulated to overcome large horizontal and vertical deviations by judicious application of prism followed by active orthoptic therapy to build compensating fusional amplitudes followed by the reduction of prism and further therapy. When adaptation is weak, prism may be used to eliminate the demand on disparity-driven vergence. Orthoptics, on the other hand, will result in an improvement in the quality of vergence adaptation and subsequently reduce the demand on the fast fusional vergence system at a given fixation distance.

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