Effect of Distance Fixation Disparity on Distance Stereopsis as Compared Among Individuals with Superior Visual Systems.

Phillip J. Mackinder
Optometric Extern, Michigan College of Optometry, Ferris State University, Big Rapids, MI
J. James Saladin, PhD., OD
Faculty, Michigan College of Optometry, Ferris State University, Big Rapids, MI

ABSTRACT

Stereopsis and fixation disparity (FD) were measured at a distance of 6 meters on 10 patients who exhibited very small amounts of vertical and horizontal heterophoria as well as excellent visual acuity (20/20). The amount of FD was then increased by stressing the visual system with base-in (BI) prism insertion. As FD increased for each individual patient, their ability to appreciate stereopsis at 6 meters was reduced. By selecting patients with normal systems, we isolated the results to only FD and its effect on stereopsis. This study proved that as FD increased, stereo acuity decreased and suggests that FD rather than heterophoria poses the threat to stereopsis.

Key Words: BVAT, Fixation Disparity, Heterophoria, Stereopsis

There are many factors that influence an individual's binocular vision and the oculomotor balance that supports binocular vision. While not totally eliminating the effects of accommodation and vergence dysfunction, naturally occurring heterophoric imbalance, and subtle challenges to sensory fusion, the present study does minimize their effects by selecting subjects with no complaints that relate to their vision and who demonstrate clinical orthophoria. The study asks whether degradation in stereopsis is better related to induced heterophoric amount (and direction) or induced fixation disparity amount (and direction). More specifically, it investigates the effects of a base-in prism stimulus that induces an esophoria and a relative eso fixation disparity on a measure of stereoscopic ability.

Previous studies have called stereopsis the "barometer of binocularity". (Griffin 1982) The implication is that a good stereopsis test score means that the oculomotor system supporting binocular vision is operating properly. Fry and Kent (1944) showed that heterophoric amount and direction was not simply related to stereoscopic performance. Saladin (1995) showed that exophoria up to approximately seven prism diopters had scant effect on stereopsis and the same could be said for smaller amounts of esophoria (up to 2 or 3 prism diopters). If heterophoria stresses the oculomotor system, why does it not degrade stereopsis in a fairly predictable fashion over these amounts of heterophoria and why is the critical amount smaller in esophores than exophores? Perhaps fixation disparity is the measure that more closely correlates with stereoscopic ability.

Many of a person's visual tasks are conducted at distances greater than the 40 cm distance for which the common stereopsis tests are designed. This study took advantage of the BVAT which is designed to be used at optical infinity or 6 meters. The BVAT has the capability of producing a measure of stereoscopic ability and a measure of fixation disparity under very similar test conditions.

If fixation disparity, not heterophoria, has the more direct effect on stereoscopic performance, then it seems reasonable that oculomotor dysfunction and/or oculomotor well-being is better related to fixation disparity than heterophoria. In turn, vision therapy (VT) and prism prescription should be directed at modifying fixation disparity rather than some direct or indirect measure of heterophoria. Similarly, our diagnostic procedures should emphasize fixation disparity aspects rather than heterophoria.

METHODS

There are a number of ways to degrade stereopsis. These include blur, decreased contrast and visual acuity, suppression, amblyopia, anisophoria, aniseikonia, as well as strabismus (Peters 1969, Legge and Gu 1989, Wood 1983, Heckman and Schor 1989, Simpson 1991, Schor 1991, Reading and Tanlamai 1980). This study reduced the effect of these elements by employing subjects who possessed superior visual systems and by maintaining constant contrast. The ten subjects who were selected from the Michigan College of Optometry at Ferris State University to participate in this preliminary study were required to meet the following criteria:

1. Best Visual Acuity (BVA) 20/20 or better in each eye as measured by a BVAT with maximum contrast at 6 meters.
2. Horizontal Phoria*: 1Δ eso to 1Δ exo
3. Vertical Phoria*: 0.5Δ right hyper to 0.5Δ left hyper

* measured by Modified Thorrington at 6 meters

All tests were performed at a 6 meter distance with the patient seated and the tests at eye level. The illumination level coming from above the front of the subject was 4 foot-candles. At the end of the testing lane, the overhead lights produced 16 foot-candles. The BVAT produced an averaged luminance of approximately 80 foot Lamberts.

Tests for fixation disparity and stereoscopic ability were performed in random order through 2, 4, and 6 prism diopters of base-in prism. The random character of the presentation was assured by making a grid of fixation disparity and stereoscopic performance using 60, 30, and 15 second of arc presentations at the various prism amounts (See **Figure 1**). The grid was then cut into squares, each square containing prism amount and seconds of stereopsis or prism amounts and fixation disparity. Finally, the squares were placed in a box and drawn at random.

Figure 1: Grid for Random Measurement of Stereopsis and FD vs. Prism Amount

2Δ BI 60"	2Δ BI 30"	2Δ BI 15"	2Δ BI FD	2Δ BI 60"	2Δ BI 30"	2Δ BI 15'	2Δ BI FD
4Δ BI 60"	4Δ BI 30"	4Δ BI 15"	4Δ BI FD	4Δ BI 60"	4Δ BI 30"	4Δ BI 15"	4Δ BI FD
6Δ BI 60"	6Δ BI 30"	6Δ BI 15"	6Δ BI FD	6Δ BI 60"	6Δ BI 30"	6Δ BI 15"	6Δ BI FD

The stereoscopic test was a standard BVAT test based on the diastereo principle (Pardon 1962). The task of the subject was to select which of the four rings "floated" out from the BVAT monitor after the random amounts of prism were inserted. If unable to tell, they guessed. The prism was removed before 10 seconds elapsed to prevent adaptation and the patient was given 3 subsequent presentations of diastereo rings with the same prism amount (Schor 1979). The data was recorded number correct over 4 and converted into decimal form.

Additionally, fixation disparity was randomly measured by the BVAT without central fusion locks after inserting random amounts of prism. Patients looked away from the screen and the peg was moved off center. The subject was directed toward the screen immediately after prism insertion and asked which way the peg need to go to be aligned properly. The subject then signaled when the peg was aligned. Once again, the patient was required to make decisions before 10 seconds elapsed to prevent adaptation. Two sessions were required for the gathering of data to reduce patient fatigue. For each possible combination of prism amount and stereopsis or prism amount and FD, 4 measurements were taken at 4 different times. See **Figure 2.** Note, baseline information was recorded without prism at the onset of the experiment to determine the subject's actual fixation disparity and stereopsis.

Figure 2: Recording Form

Name: **Clinician:**
Session 1 Date:
Session 2 Date:

Visual Acuity(c or s): **Fixation Disparity:**
Horizontal Phoria: **Stereopsis:**
Vertical Phoria:

Data:

		Session 1					Session 2				
	Stereopsis:	60sec.	30sec.	15sec.	FD		60sec.	30sec.	15sec.	FD	
P	2BI										
r	4BI										
i	6BI										
s	2BI										
m	4BI										
	6BI										

RESULTS

Number Cruncher Statistical Package

Utilizing "The Number Cruncher Statistical Package", we were able to break down the data into readable results. We first noted whether BI prism (0, 2, 4, and 6 prism diopters) degraded the overall stereopsis of our patients for each level of stereopsis. The data demonstrates that as BI prism is added, stereopsis decreases in a predictable manner with good correlation.

Base In Prism Insertion (0,2,4,6) vs. Effect on 60 Seconds of Stereopsis:

x intercept (a) = 1.08
Slope (b) = -0.066
Correlation Coefficient (r) = -0.66
T value, B = 0: 4.82
Tail Probability = 0.00

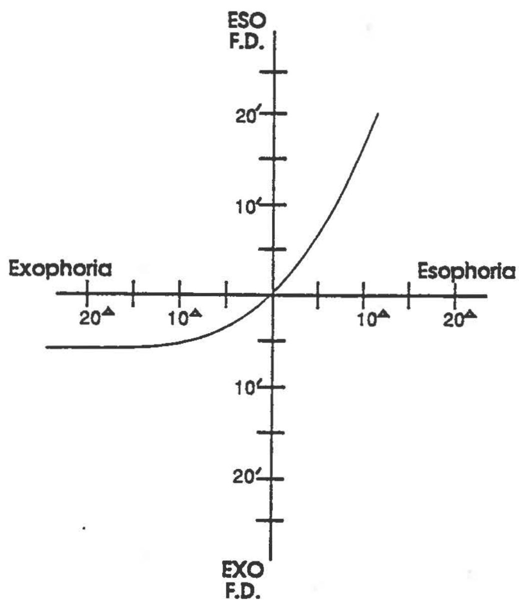
Base In Prism Insertion (0,2,4,6) vs. Effect on 30 Seconds of Stereopsis:
a = 1.01
b = -0.067
r = -0.61
T = 4.26
Tail = 0.00

Base In Prism Insertion (0.2,4,6) vs. Effect on 15 Seconds of Stereopsis:
a - 0.84
b = -0.063
r = -0.63
T = 4.48
Tail = 0.00

Next, we noted from the data that as the prism amounts increased, the amount of FD increased. This is not new information. It quite simply is the BI side of a FD curve and reiterates what we already know about prism and its effect on FD. In essence, what we are doing is creating an esophoria in functionally orthophoric individuals. The corresponding increase in fixation disparity is justified by **Figure 3** which was adapted from Ogle (1964) and Jompolsky et al (1957)

Base In Prism Insertion vs. Effect on Fixation Disparity
a = 0.0075
b = 1.62
r = 0.67
T = 4.91
Tail = 0.00

Figure 3: "The angular amount of FD proposed as the expected for a given direction and amount of horizontal heterophoria in a properly function oculomotor system. (Copied from Saladin 1995)



The data becomes more interesting when we compare the amount of FD for each level of stereopsis. We were able to prove with good correlation that as FD increased, stereopsis decreased. The equal or slightly better correlation for each level of stereopsis for fixation disparity over the induced esophoria implies that a predictable pattern exists for FD that may be useful in assigning a diagnostic value to FD amount in clinical practice.

Fixation Disparity vs. Effect on 60 Seconds of Stereopsis
a = 1.00
b = -2.71E-2
r = -0.66
T= 4.83
Tail = 0.00

Fixation Disparity vs. Effect on 30 Seconds Stereopsis
a = 0.95
b = -2.87E-2
r = -0.64
T = 4.59

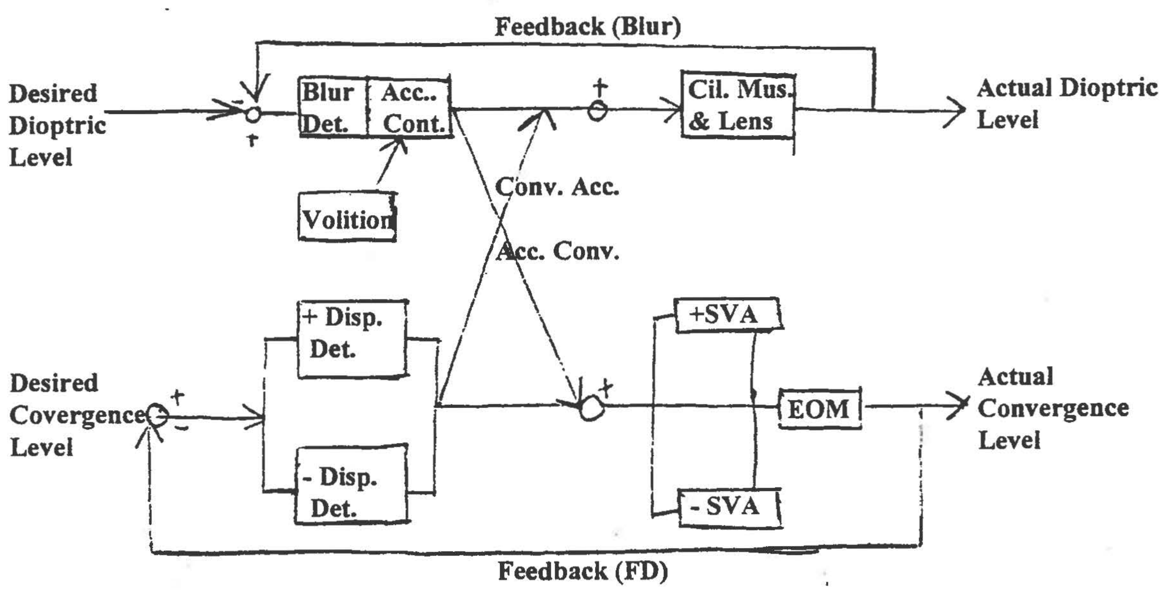
Tail = 0.00

Fixation Disparity vs. Effect on 15 Seconds Stereopsis
a = 0.75
b = -2.63E-2
r = -0.64
T = 4.60
Tail = 0.00

DISCUSSION

To better interpret the results and explain the relation of FD to binocularity, a modest understanding of control systems theory is necessary. **Figure 4** shows the "Negative Feedback Model of the Accommodative and Disparity Vergence System" (Saladin in Borish). This control system demonstrates a link between all the components of the binocular system and their interrelation. The model depicts the disparity detectors as a major component of the fusion mechanism. After a coarse fixation movement in response to a large disparity stimulus, the disparity detectors react to crossed or uncrossed disparity with either convergence or divergence respectively to fine tune the alignment of the eyes. Some maintenance level of innervation must be left if the object of regard is not at the phoric position (i.e. phoria = ortho). Keep in mind that phoria is a product of 1. Anatomical strength, innervation and insertion of extra ocular muscles, 2. Relation between the AC/A and CA/C ratios and 3. Interpupillary distance (pd).

Figure 4: Negative Feedback Model of the Accommodative and Disparity Vergence System (Copied from Saladin, 1995)



Blur Det = Blur Detectors Acc Cont. = Accommodative Controller Cil. Mus. & Lens = Ciliary Muscle and Lens Disp. Det = Disparity Detectors SVA = Slow Vergence Adaptation EOM = Extraocular Muscles Conv. Acc. = Convergence Accommodation Acc. Conv. = Accommodative Convergence FD = Fixation Disparity

It used to be thought that FD wass the residual amount from the phoria after the disparity detectors aligned the eyes. The eyes did not need to be perfect because Panum's central area permitted some "slop" in the system. Therefore, the eyes, not wanting to be overworked, turned in (or out) just enough for fusion to occur in Panum's area. On the contrary, scientists now believe that the residual FD provides the tiny amount of negative feedback to the disparity detectors to maintain fusion. If the feedback loop enabled the FD to go to zero, the disparity detectors would shut down, the eyes would return to their phoric position and fusion would be lost.

The disparity detectors do align the eyes but they are not efficient. They react fast to disparity but tire quickly. Another element is necessary to amplify the gain of the disparity detectors and decrease their workload. This system is known as the Slow Vergence Adaptation Mechanism (SVA). The SVA is slow to act as the name implies and requires about ten seconds to initiate (Schor 1979). Additionally, it offers insight as to how individuals "adapt" to high phoric postures with little or no binocular complaints.

As already stated, the SVA amplifies signals from the disparity detectors. Thus, if the SVA is strong, it will require less signal from the disparity detectors which ultimately get their feedback from FD. Therefore, smaller amounts of FD are required for the eyes to be held in a position where fusion may occur.

The strength of the SVA varies from patient to patient. For instance, if we compared two exophores:

Patient A: 12 exo, strong SVA requiring 2 minutes arc exo FD to maintain fusion.

Patient B: 6 exo, weak SVA requiring 6 minutes arc exo FD to maintain fusion.

Which patient has the better oculomotor balance? Patient A is better off because he/she can adapt to the phoric posture more readily. Patient B may suffer from convergence insufficiency, leading to asthenopia, and may also have decreased stereopsis because increasing FD stretches the limits of Panum's area. Thus, even though the phoric amount differs in each patient, it is not the crucial feature in this instance. This also offers insight for the lack of correlation between phoria and its effect on stereopsis.

Studies have illustrated this when comparing esophores to exophores to see which was more detrimental to stereopsis. First, compared to -SVA, +SVA seems to be better at amplification (Saladin 1995). Morgan's expected vergence ranges also demonstrate that humans are better at converging than diverging (Morgan 1940). This may offer insight as to why exophores tend to fair better than esophores. Saladin (1995) shows in **Figure 5** how exophores up to 7 prism diopters had no effect on stereopsis whereas esophores of the same amount tended to reduce stereopsis slightly. However, Dr. Saladin believes the explanation became evident when you compared the SVA's. Because - SVA is not as strong (Sethi and North, 1987), increased amounts of FD are needed to signal the disparity detectors. The weak - SVA requires this increase in feedback to the disparity detectors so there is more innervation from the disparity detectors to be amplified for continuous ocular alignment and fusion. The resultant increase in FD, not the phoria, leads to decreased stereopsis. Note from **Figure 6** that similar amounts of FD up to 10 minutes of arc whether eso or exo caused similar deterioration of stereopsis providing further support to the data from this experiment. Thus, one may conclude that while the minimal effects of phoria tend to vary, stereopsis is reduced by FD in a predictable manner.

Figure 5: Comparison of Heterophoria and Effect on Stereopsis (Copied from Saladin 1995)

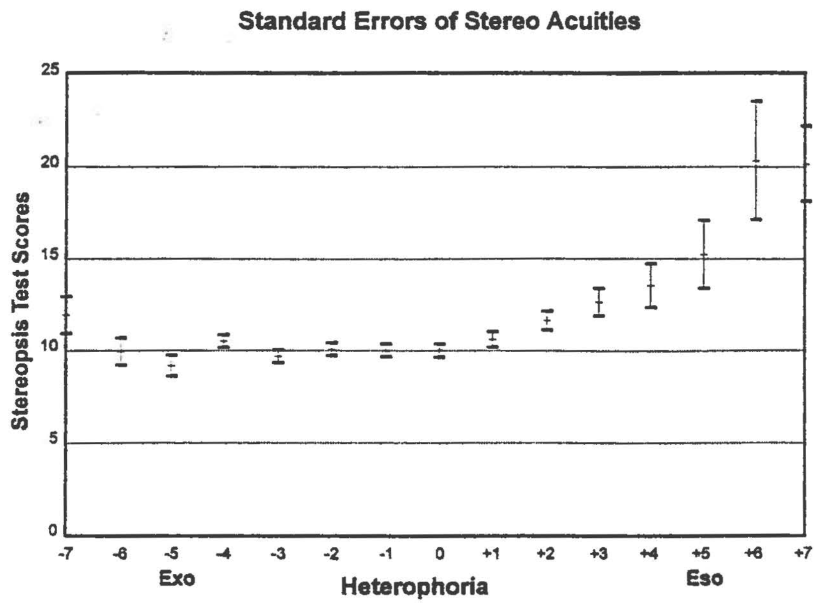
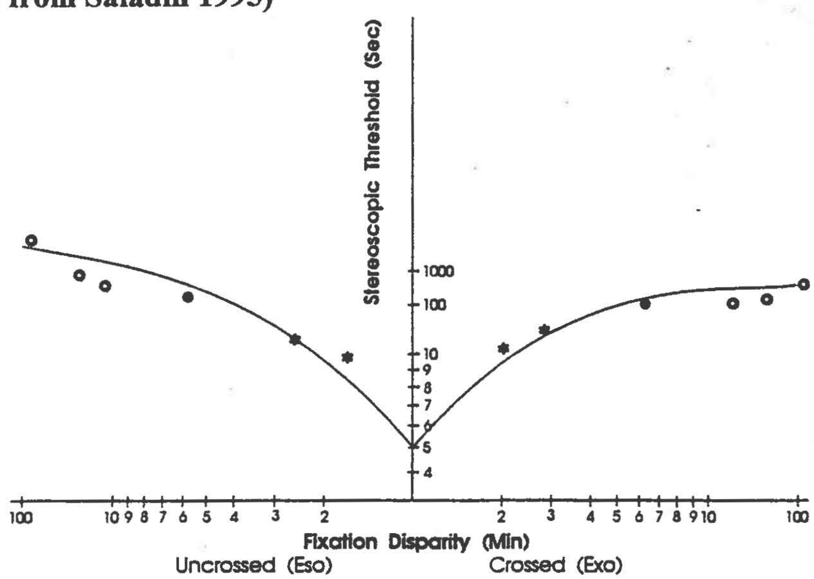


Figure 6: Composite Curve Showing the Relation Between FD and Stereoscopic Threshold; Blakemore (1970) Data o, Saladin (1990) Data •, Westheimer (1979) Data * (Copied from Saladin 1995)



Given our understanding of the control system theory, consider the case of a patient who requires a large amount of FD to give enough feedback ultimately to the

SVA to maintain fusion. One may assume their stereopsis would be degraded because after increasing FD to meet the demands of fusion, they come closer and closer to the limits of Panum's central area. To avoid diplopia, central suppression may develop further decreasing stereopsis (Tyler 1991). Additionally, the central fusion locks are better than the remaining peripheral fusion locks so fusion becomes harder and harder to maintain (Cooper et al 1992). The compounding of these problems sets off an internal conflict between all the components of the control system model and leads to the somewhat vague symptom known as asthenopia. At this time, the patient becomes symptomatic.

As discussed previously, the subjects where not allowed to "adapt" to the prism amount by removing it before 10 seconds elapsed. Once the SVA initiated, it would decrease the load on the disparity detectors and would push FD closer to zero. By limiting time, we were able to force the subjects to rely solely on their disparity detectors. This gave us increased amounts of FD to work with. We were then able to demonstrate that increasing FD does, indeed, degrade stereopsis. Also, if we factor in our current understanding of the control system theory, we can clearly identify the importance of FD clinically from the binocular standpoint. In addition, we must recognize that increased FD is not the cause of poor binocularity but rather the result which leads to poor stereopsis. Thus, if this is true, we can utilize FD as a clinical tool for assessing good or poor binocularity rather than conventional phoria measures. However, improvements must be made with this study to draw more reliable data and predictable results. Until then, stereopsis remains the standard for screening patients for binocular anomalies.

CONCLUSION

1. Increasing FD on patients with exceptional visual systems caused a decrease in stereopsis. This implies that stereopsis is effected by FD not phoria because previous studies have not linked poor stereoscopic performance to heterophoria.
2. Patients who exhibit poor stereopsis and increased levels of FD may have a poorly functioning SVA.
3. Since this study is serving as a prototype to a more detailed experiment, it could be improved in the following ways:
    a. Increase room illumination to increase contrast and acuity.
    b. Patients must exhibit 15 seconds of stereopsis at start.
    c. Utilize 2, 3, and 4 prism diopters rather than 2, 4, and 6 prism diopters at 6 meters.
    d. Increase the number of subjects to improve statistical analysis.
    e. Compare ability of esophores to exophores to adapt to stress from BI prism insertion.
    f. Measure FD after patients adapted and compare resultant stereopsis.
    g. Fatigue patients to see response. Most patients who present with symptoms are not tired so the visual system at the time of testing appears normal.

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