REVIEW ARTICLE

Stereopsis From a Performance Perspective

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ABSTRACT: Purpose. A patient can demonstrate a poor stereoscopic test or task performance for reasons inherent within the test/task itself for reasons dependent on normal physiology common to all human subjects, and also for reasons that are outside of normal physiology and are unique or idiosyncratic to a particular person's visual system. This article reviews the literature for the first two reasons, but emphasizes the pathophysiology involved in the idiosyncratic and abnormal reasons. Results. Using control systems analysis, it is shown that deficits in stereoscopic performance can be explained by reference to the quantitative aspects of stereoscopic threshold and qualitative aspects such as speed of response, reliability–robustness, and strength of percept. The relationship between fixation disparity and stereopsis is seen to be central to this explanation. Conclusions. Proceeding from diagnosis to treatment, control systems analysis offers physiologically based explanations for the corrective procedures necessary to ameliorate abnormal conditions. Several topics for applied research in stereopsis are suggested. (Optom Vis Sci 2005;82:186–205)

Key Words: stereopsis tests, vision performance, fixation disparity, depth perception, binocular disparity

A mountain of theoretical knowledge exists on stereopsis but only a small part of that mountain has been carried over into the clinical world and made applicable to everyday tasks. A practitioner with a patient who has complaints of “poor depth perception” can often confirm the poor stereopsis but it is less likely to have a clear idea of how to correct that problem. This article seeks to provide a framework for the differential diagnosis of stereopsis problems and then to offer a related framework for solutions for those problems. What is it about a particular patient's visual system that degrades stereoscopic performance? We will see that there are general factors that degrade performance for everyone, and there are specific factors that affect only certain people. Although those general factors are mentioned, the emphasis is on those specific factors that are peculiar (idiomsyncratic) to a particular type of visual system or demand on that system. If the factors or problems can be pinpointed for a particular situation or person, a specific corrective or therapeutic approach is more accessible.

Stereopsis is a space sense that provides the relative z-axis or farther–nearer depth between two objects. Although it can be broadly equated with the overall visual perception of depth and so include the effects of “monocular” pictorial cues and motion parallax, I have chosen to define it more narrowly. For the purposes of this article and unless specifically stated, it is solely dependent on binocular disparity, which is the difference between the parallactic or binocular subtense angles, one for each of the objects. The parallactic or binocular subtense angle of an object is that angle formed with the apex at the object and subtended by the distance between the entrance pupils (Fig. 1). When speaking of stereopsis, the distance between the eyes, more specifically between the entrance pupils, is referred to as the eye base or stereo base. Stereopsis is said to be a relative rather than an absolute space sense, because the system does not calculate and specify the depth difference directly in units of absolute distance. Rather, the depth percept begins in internally coded angular binizational units recognized by the observer's internal physiology. The information is then subjected to higher-order processing, which converts that internal information to externally recognizable units of length. This conversion process requires that another object or distance of known size be in the same visual space to allow a comparison to be made. For example, if distances A and B are known in internally coded angular units, and distance A is also known in external absolute units (feet, inches, meters, and so on), then the distance B can be inferred. The fidelity of the percept depends on the correct knowledge of distance A. The smallest binocular disparity that can be detected a criterion proportion of the time under the specific testing conditions is the lower or minimum stereoscopic threshold. Similarly, the largest binocular disparity that can be observed is the upper or maximum threshold. This article is mainly concerned with how the stereoscopic space sense contends with parameters that put stress on the vision system between the lower and upper thresholds. For the purposes of this article, stress is any impending physical, physiological, or psychologic force that acts to handicap or distort and thereby prevent a particular system (here, the vision system) from performing at its optimum level of function. Stereoscopic testing is important in the clinical practice of optometry, because many of the adverse conditions that affect the...
function of the visual system will also affect stereopsis. Most frequently, stereopsis testing is not performed out of any concern for a given patient's binocular depth perception per se, although that may be important in individual instances. Instead, it is used because it is an excellent screening test for the detection of problems in much of the visual process. Stereopsis sits at the top of the food chain of vision and has been called the "barometer of binocularity." Schoen has stated that, "Low stereo-thresholds demonstrate a high fidelity of a binocular system that computes disparity, and they offer a way of directly examining the fidelity. For this reason stereo acuity is considered as a bench mark test for peak clinical performance of binocular vision." Uncorrected refractive error, decreased visual acuity, and sensory and motor fusion problems all have an effect on stereoscopic ability. Patients who have normal stereopsis are highly unlikely to have any serious problems with their refractive or oculomotor systems. It does not follow, however, that patients with abnormal results on stereopsis tests necessarily have a problem in their refractive or binocular/oculomotor systems. Some patients may be experiencing normal stereopsis on the test but for some reason do not respond to the examiner. There is another subset of people who lack the ability to process disparity information as a depth sensation, although other aspects of the refractive and binocular/oculomotor systems appear to be normal.

**TESTS OF STEREOPSIS**

How can stereopsis be tested and what is "normal"? For a clinician, stereopsis tests come in many different forms, and he or she must choose the one appropriate for the situation. Tests vary in practical aspects such as size and complexity of equipment required, ease of use, appropriateness given the age and/or abilities of the patient, and speed of application. They also vary in the psychophysical attributes of threshold, response task, and stimulus presentation form, all of which have a definite influence on how the test results are analyzed and interpreted.

There are two basic divisions of stereopsis, local and global, which share some aspects but differ in others. Local stereopsis depends on horizontal disparity from monocularly observable stimulus patterns. Global stereopsis is brought about by horizontal disparity patterns not monocularly observable the disparity patterns themselves cause binocularly observable figures and depth perceptions. Another division of stereopsis can be made between time-dependent (dynamic) and time-independent (static) manifestations. This article concentrates on the static aspects, because at the present time, dynamic stereopsis is not commonly measured in a clinical setting. (A need may exist for such a test.)

A review of four common static stereopsis tests will illustrate all of these factors. These four were chosen because they span the gamut of the variations in stereopsis tests and not because they are necessarily the "best" tests, even in their own category. A more complete description and explanation of the administration of these four representative tests is covered elsewhere. (See Cooper for a comprehensive listing and description of current stereopsis tests.) Stereopsis tests are among the more internally complicated tests that optometrists are called on to perform, and that very complex nature makes them and their results difficult to compare.

The Howard-Dolman peg test is the oldest of the standard stereopsis tests and serves as the "gold standard" against which all other stereopsis tests are compared. It is time-consuming, requires a relatively large piece of equipment, it is usually performed at a distance, and asks that the patient perform a complex motor task as a response indicator. Analysis requires a mathematical operation, either averaging or the computation of some measure of range and its variability. Different aspects of the test/task drive the resultant threshold in different directions. For instance, the Howard-Dolman test can be used with either a null task or a just noticeable difference (JND) task. Because the null task requires less decision effort of the observer and is less criterion-dependent and therefore more stable, the threshold tends to be lower than for the JND task.

From my personal experience with several thousand normal observers using the null task and a variance value of one standard deviation, a result of 14” of arc or better (smaller numeric value) is expected for an adult with a normal binocular system.
Stereopsis Performance—Saladin

The Wirt Rings portion of the Stereo Fly test is the most common test for stereopsis in the United States and is valued clinically because it is compact, performed at 40 cm, easy to store or carry, and quick to administer and score. This test does not establish a stereoscopic threshold; instead, it acts as a convenient screener for binocular/oculomotor or refractive problems at near. As such, for most people, it is a suprathreshold test and therefore is much easier for the patient on that point alone than the Howard-Dolman. On the other hand, the pattern observed is of several shades of gray and therefore is of reduced contrast. Although low contrast will tend to elevate the numeric amount of the threshold, most patients are not at threshold even on the ninth (40 s of arc) diamond, and therefore the low contrast may not affect performance. The low contrast would definitely have a deleterious effect if the patient were at or near threshold. Furthermore, the patient has only one chance to discriminate the correct circle at each disparity level; therefore, a 100% criterion is in place. Low contrast tends to make the task more difficult and the 100% criterion makes it more demanding but, working in the opposite direction, the four alternative forced choice psychophysical design eases the patient’s task. To further confound the results, monocular cues may be present. Clearly, one can see that different aspects of the test/task tend to make the task more or less difficult for the patient and will affect the practitioner’s interpretation of the test results. Forty to 50 s of arc is an acceptable score. Sixty seconds of arc is strongly suspect, but response time is a consideration. A fast (2 s of time or less per diamond) 90 s of arc may be comparable to a slow 40.

The Keystone Multi-Stereotest is mentioned mainly because an in-instrument stereopsis test should be included in any representative list of stereopsis tests, and because it measures stereoscopic ability under nearly ideal conditions. The task is very simple; it is to discriminate, in a forced-choice paradigm, which line of each pair of adjacent vertical lines is in front of the other while looking at a series of line-pairs of decreasingly less disparity. The vertical lines are presented on a card at optical infinity in a Brewster-type stereoscope, usually a Telebinocular (Keystone View Company, Reno, Nevada). Very little is required of the patient; he or she only needs to perceive which line is in front of an adjacent line for a split instant in time and to do so under the best possible conditions for observation. The expected stereo acuity on this test for a normal binocular system is 11 s of arc. This expected test result is numerically low because the test lines have a very high contrast, have a near-ideal spatial frequency pattern, and require very little decision-making by the patient.

Finally, representing the global types of stereopsis tests, the Random Dot E Stereotest serves as a screener for acuity and binocular/oculomotor imbalance/alignment problems, particularly in populations incapable of exacting response judgments or with limited communication abilities. Rosner suggested that the test be done at 150 cm and the patient be referred if he or she did not get four out of six successive presentations correct. The differences between global and local stereopsis have been reviewed frequently in the literature. There is reason to believe that, although local and global stereopsis share some aspects, considerable differences in physiology exist. As a rule, a person performing well on a global test will perform acceptably well on a local stereopsis test, but the reverse is not true.

No contemporary discussion of stereopsis tests should occur without mention of the Frisby Stereotest. It is my personal opinion that it is the most versatile stereopsis test currently available. It is a test that includes some of the best aspects of the four previously mentioned stereo tests. It is a true hybrid test, having some of the more desirable aspects of both global and local stereopsis tests. Properly presented, there are no monocularly observable patterns. It can be used for threshold testing like the Howard-Dolman or as a general purpose clinical screener like the Wirt Rings and has been used successfully on school screenings in place of the Random Dot E.

QUALITATIVE FACTORS

Stereoscopic performance is usually described by stating the binocular horizontal angle attendant to a certain test or task. The smallest and largest disparities discerned as depth differences mark the extremes of this continuum of stereoscopically readable horizontal disparities. As mentioned previously in the discussion on stereoscopic tests, this numeric amount is affected by many stimulus and test variables. Whenever a disparity amount is used to describe stereoscopic performance, a quantitative estimate of that performance has been made. It could mean that the smallest (or largest) disparities discernible have been determined under those test/task conditions, or it could mean that the observer accomplished the test/task at some intermediate level of horizontal disparity. Any such single measurement of test/task performance does not adequately describe the stereoscopic performance characteristics of that system. Stereopsis has at least three qualitative aspects as well as two (smallest, largest disparities discernible) quantitative aspects. As put forth by Julesz, the qualitative aspects of stereoscopic ability relate to both the speed of the percept and the reliability—robustness or stability of the percept. To these first two aspects, I have added the strength or intensity of the percept. These three aspects are concerned with how the binocular system responds when the conditions are less than ideal and when disparity amounts are between the high and low thresholds for a given test/task. This between-threshold range of disparities is most often spoken of as suprathreshold (although a better term would be intrathreshold). In a very practical sense, people rarely operate under quantitative threshold conditions; they usually operate at intra/upper thresholds with many and varied constraints and influences. As mentioned previously, I have divided the qualitative considerations into those having to do with speed, reliability—robustness, and strength of percept.

By speed, I mean the time taken to detect the difference in three-dimensional relative depth for stationary and moving targets. As a very simple but common clinical example, consider again the Wirt Rings portion of the Stereo Fly test. Some patients correctly discriminate the appropriate circle in all nine diamonds very quickly, whereas others require more time to accomplish the same task. All patients would be given the same quantitative score (40 s of arc), but those who indicated the correct rings very quickly obviously have a more “efficient” stereoscopic system than those who require more time, all else being equal. The reason may not be so simple to explain by saying that the slow responders have a higher stereoscopic threshold and therefore are operating closer to...
threshold during the test. The time required for the perception of a random-dot stereogram (RDS) has been studied extensively. Julesz's speaks of the “jump out” quality of stereopsis. Such papers tend to dwell on the necessity of solving the “correspondence problem” as a contributor to the time required for perception of the RDS figure and also tend to speak in terms of milliseconds of time. The correspondence problem refers to the process of placing the images onto the two retinas by a motor movement (and perhaps a sensory shift) such that the optimum placement onto (or near to) corresponding points yields the minimum mean disparity. This placement process requires considerable processing to determine optimum vergence levels in both the horizontal and vertical planes. Contours or image points that “do not fit” are then read off as disparate. The speed that I speak of concerns that mentioned previously and more. It also includes the time (in terms of one or more seconds of time) required for full manifestation of the depth after the correspondence problem is solved in the case of RDS and the overall time required in the case of local stereoscopic patterns.

Reliability—reliability addresses the variation of stereoscopic ability over time as a result of external and internal stresses. External stresses are caused by factors that make the stereoscopic stimulus situation less than ideal. For the most part, these external factors are common to all observers and are addressed briefly in the next section. Internal factors are factors that are generated within or are peculiar (idiosyncratic) to the individual. Binocular/ocularmotor dysfunctions are internal factors, and discussion of the effects such dysfunctions have on stereopsis make up the bulk of this article. Inherent aspects of the physiology of the stereoscopic system (for example, the background noise resulting from spontaneous intravascular and muscular activity) may also be among the internal factors affecting stereopsis. The effect of such internal factors can be observed by listening to patients performing the Keystone Multi-Stere test. This test claims disparities down to the 4 s of arc range, which is close to the absolute stereo threshold. Here, reliability is a big factor. If such patients who are slow to respond but successful on the Multi-Stere are carefully questioned, one will find that the stereoscopic percept is “coming and going” on them. If they wait long enough, they will get a glimpse of the disparity difference and be able to report the correct answer to the examiner. Something within the anatomic or perceptual physiology of that person’s system is varying. To the extent that that “something” is varying, the system lacks reliability. A variation in the strength of a sensation near threshold is common and even expected given physiological noise, but the variation that I suggest here may be much stronger, particularly in subjects with clinically weak or abnormal binocular/ocularmotor systems. It can be sufficiently strong as to provoke native observers into volunteering a comment under relatively casual clinical conditions. Common physiological threshold variation is usually observed only with careful laboratory-controlled measurements in nominally normal subjects, perhaps with the subject coached to watch for such variation.

Strength of percept refers to the sensation of depth itself. If one carefully observes a stereogram, a certain order of perception occurs. First, there is no perception of stereoscopic depth; it looks flat. In many people, this first stage is over so fast that it is scarcely noticeable. The next stage, called the “jump out” stage, occurs as the depth sensation is becoming noticed. It, too, is usually over quickly and may be a function of “transient” stereopsis. In other individuals, this stage consumes a noticeable amount of time, even for local stereograms. However, once the stereoscopic sensation is achieved, the “sustained” depth just seems to become enhanced for quite a period of time. The strength of the percept builds. I have had the opportunity to question some very experienced stereophotogrammetrists (all had worked at least 30 years with the Defense Mapping Agency) about their experiences using stereopsis. These people are among the most experienced in the world at using stereopsis. I asked them, “Tell me what you see when you look into the stereoplotters. Talk about how you see stereoscopic depth.” Without my prompting or using any additional leading questions, several of them reported some variation of the same theme: after a week or more of vacation, it took several hours to several days before they “really saw depth” again in the instruments. They saw depth immediately when they looked into the stereoplotters after their vacations, but not the very strong sensation of depth that came to be after the vacation recovery period. A similar scenario occurred at the beginning of a shift during the normal workweek, but the depth sensation recovery period was very short (minutes) or almost nonexistent. Why or how does the depth percept build in strength? Is this an example of perceptual learning? The concept of quality of the depth percept is not new. Ogle recognized the concept of the strength of the stereoscopic percept when he noted that an initial strong sense of depth could fade immediately after a very large disparity was presented. Nelson spoke of the “solidity” of random-dot stereograms. Simmons and Kingdom noted a qualitative aspect of the stereoscopic percept: “This quality reduction may take the form of a reduced stereoscopic strength and contrast sensitivity.” Tyler, in his study of prisms and diastereopsia, noted that one (diastereopsia) was observed as two planes in space, whereas the other (diastereopsia) was observed as a volume of space. Here there is a qualitative distinction between the two percepts. Obviously, the stereoscopic percept has different degrees of strength and perceptual manifestation.

How might these three qualitative aspects of stereopsis be observed and, even better, quantified? One can measure how the thresholds, both upper and lower, change with different stimulus conditions. (The results of such studies are considered briefly in this article.) Speed-vs.-threshold experiments are common in the literature and need only be mentioned. The resistance of the upper and lower thresholds to change as the stimulus is degraded from the ideal is a measure of the robustness of the system; the effect of stimulus contrast on the threshold is an example. It is more difficult to find research on the strength of the percept at threshold perhaps because the strength of the percept is largely subjective and therefore more difficult to quantify. More to the interest for this article, however, is the measurement of intrathreshold stereoscopic ability. Some aspect of performance on an intrathreshold stereoscopic task is the measured or dependent variable. For instance, Sheedy and coworkers determined the time it took to put 26 wooden pointers into 28 drinking straws. The task was defined...
in their threshold. They could have used straws and pointers against a background of similar shade and varied the contrast; this would have yielded external stimulus parameters. However, they did not; they varied a parameter that was dependent on the internal characteristics of the subjects' binocular/oculomotor systems. The subjects were required to fuse through various amounts of base-in and base-out prism. The dependent variable was the time required to perform the task. This time is obviously a function of the speed of the percept—the faster the subject could fuse and detect the disparity, the faster they could respond. If one plots the task time vs. the prism amount/direction, one arrives at a measure of how robust the system is against disparity/vergence stress. The slope of the line is a measure of that robustness. One could also ask how the system is robust against other stresses, for instance, vertical disparity or even loud music in the background while the stimulus—horizontal prism—is varied. For the strength of the percept, in these last two instances, the subject could be asked to rate or compare the strength of the percept to the strength of some other percept, perhaps in a cross-modality experimental design. Remaining in the same modality, I have compared the strength of the percept under examination with the relative strengths of similar percepts in which the stereo base was varied.

THE CONTROL SYSTEM MODEL

Over the last few decades, engineering science has given physiologists a tool to investigate biologic systems. That tool is control systems analysis. With respect to oculomotor and binocular v processes, the control systems model provides a framework in which to build an understanding of how factors external to individual (stimulus variables) and internal to the individual (endogenous, idiosyncratic) affect stereoscopic performance. The model exists in several forms depending on the viewpoint necessary specific purpose. My descriptive model (Fig. 2) emphasizes which promotes the linking of physiology with the clinical w and is useful in both static and dynamic situations. It has an also correlation to most of its parts, and such a psychophysical model is desirable if clinicians are to readily relate to it. This sacrifices mathematical/engraining/discrimination and ease of view, but, I think, loss is warranted if it will bring the power of this engine to the clinical realm where it can bear on the actual, to-day problems of our patients.

The basic model (Fig. 2) with references is explained in the 1st edition of Borish's "Clinical Refraction," (See Schor's 1st formal version of this model, Hung and Ciuffo's 2nd ed. of the visual system in general, and Hung's 2nd for a complete description of the models of the accommodative and vergence systems.) The accommodative and disparity vergence systems are modeled similarly. Both have as input the difference between desired accommodative and/or vergence state and the actual accommodative and/or vergence state. Both begin the process w coarse reading of the accommodative and vergence stimulus brings the accommodative and vergence responses into app rate position. This coarse (transient) response is preprogram and not under feedback control. In a second or two, a fine

FIGURE 2.
The negative feedback model of the accommodative and disparity vergence systems. BLUR DET, blur detector; SAA, slow accommodative adapt; ACC. CONT, accommodative controller; CIL MUS., ciliary muscle; LENS, crystalline lens; Disp. DET, disparity detector; STC, coarse stereopsis; SFP, stereopsis; SVA, slow vergence adaptation; EOM, extraocular muscle; Conv. Acc., convergence accommodation; Acc. Conv., accommodative convergence. The positive disparity detector and positive slow vergence adaptation components signify disparity-driven convergence, whereas negative side in both components signifies disparity-driven divergence. The shaded areas of the blur and disparity detector boxes signify the constant or transient components, whereas the unshaded areas represent the fine (slow) or sustained components.

Optometry and Vision Science, Vol. 82, No. 3, March 2005
tained, more exacting reading of the remaining accommodative and vergence stimulus is obtained through the negative feedback control mechanisms, and the systems are guided to an initial response level. The innervation to support that initial response level comes from the detector/controller portions of both the accommodative and disparity vergence sections of the model. In the case of disparity vergence in a normal, healthy system, further innervation is provided in 10 to 15 s of time by the slow vergence adaptation (SVA) mechanism. This contribution allows the negative feedback system to further reduce the input (now fixation disparity) to the disparity vergence system. Similarly, but requiring several minutes, the slow accommodative adaptation mechanism (SAA) begins contributing innervation to the accommodative system, allowing further reduction in the accommodative input (now the lag or lead of accommodation). While the whole process is going on, the accommodative and disparity vergence systems are interacting through the accommodative convergence and convergence accommodation linkages. When the entire system is operating properly and a final steady-state has been attained, both the accommodative system and the disparity vergence system are principally responsible for their own fine adjustments.25, 26

Careful study of the model leads to some interesting clinical ramifications. Note that the inputs to the two systems under steady-state conditions are vergence error (fixation disparity) and accommodative error (accommodative lag-lead). The systems interact with each other in a complicated manner, and eventually the system integrates the interactions and presents the clinician with the “bottom line”: If fixation disparity and accommodative lag are within acceptable limits, the system must be operating comfortably, no matter the stresses being put upon that system. Once again, stress is here defined as any physical, physiological, or psychologic force that acts to impede a particular system from its own optimum level of function. Applying this principle clinically, we are not particularly concerned with the nature and strength of the stresses (such as the various heterophorias) if the system has sufficient compensatory mechanisms and abilities to manage them.

Proper disparity detector function can be tracked through observation of fixation disparity.4 For its simplest clinical analytic application, fixation disparity should be <10 min of arc27 and should be steady (no more than 2° fluctuation).28 In general, the smaller and steadier, the better. Clinically, I have observed that if the patient finds it necessary to volunteer the information that the nonius lines are fluctuating during the measurement of fixation disparity, then the movement is more than normal and the fixation disparity is unstable. Usually, the patient is uncomfortable with the nonius lines in motion. Proper accommodative system function can be observed directly with dynamic retinoscopy. At near distances, the lag of accommodation should be 0.50 ± 0.25 D and should be steady to within a retinoscopically observable 0.25 D. In actual practice, the clinician must be sensitive to fatigue and facility factors that may cause a degradation with time. These factors may not

be obvious with one look at fixation disparity and accommodative lag.

If fixation disparity and accommodative lag are such supreme indicators of the well-being of the oculomotor system, where does stereopsis fit in? Blakemore34 showed that as pedestal disparity (an experimentally imposed fixation disparity) increased, stereocuity decreased. Since that time, many other researchers35–38 have verified this close association between fixation disparity and stereocuity and have found that it holds down to even 1 min of arc. Therefore, although a small amount of fixation disparity may be “normal,” it exerts a price from the system in accordance with its amount and, as explained later, its stability. Ultimately, this stability is even affected by irreducible noise in the system.39 An improper accommodative state affects stereopsis directly through the effect of blur on the disparity detectors and indirectly through the interactive linkages between the accommodative and disparity vergence systems. Keep in mind that stereoscopic ability can be affected by problems independent of fixation disparity or by problems that operate in parallel with fixation disparity on stereopsis. All challenges to stereopsis are not necessarily mediated through or by fixation disparity, but where fixation disparity is affected, stereopsis is affected.

Fixation disparity is here defined as a misalignment of the eyes that prevents images of a fixated object from falling onto exactly corresponding retinal points but does allow them to fall within Panum’s area. A major contention of this article is that fixation disparity affects stereopsis in a very direct and exacting way. This contention rests on research that is, for the most part, centered on fixation disparity measured subjectively over seconds of time and with head and eyes held relatively steady. The straightforward explanation is that if stereopsis is optimum for stimuli around the horoptor, and the individual fixates onto a point that is just in front of or just in back of the object of regard (which is therefore off the horoptor), discrimination ability for other objects about that object of regard for relative disparity differences must decrease. Certainly, the fusional mechanism that maintains eye alignment is narrowly tuned (within 30° of arc) and requires stimuli near the horoptor.40 I believe that this view is the mainstream view and is supported by the preponderance of evidence, theoretical or clinical, but there is evidence that the complete picture may not be so simple, especially when vergence and the head are not held stable.41 Under these conditions, “the discrepancies between the sensitivity of stereoscopic vision and the slippiness of oculomotor control mean that oculomotor stability is at least one order of magnitude less precise than measured stereocuity.”42 Howard and Rogers43 offer the explanation that because fixation disparity causes a constant vergence disparity across the entire visual field, and stereopsis is deduced from a first-order spatial derivative, fixation disparity would not affect stereopsis. This explanation leaves me wondering just how this could be supported neurophysiologically; the disparities processed would still involve areas off the horoptor, and there are fewer cortical cells to serve these areas. I think that the researchers44 holding this view must contend with a vast amount of established research to the contrary. They must also contend with the overall picture being developed in this present article: the relationships between fixation

A strict interpretation of the Sheedy-Saladin95, 96 data would lead to the conclusion that up to 10 min of arc fixation disparity statistically promised comfortable vision for stereopsis, but any eso fixation disparity at all was unacceptable, no matter the direction of the phoria. Other considerations from that same and later research along with clinical experience97–100 have led to 4-min eso and 6-min eso limits for acceptable clinical fixation disparity measurements given a ±5-deg fusion break.
disparity and stereopsis garnered from clinically obtained data just fit together too neatly to be ignored. Attacking the question directly, Ullswater and coworkers.23,24 published convincing evidence that a vergence constant error (fixation disparity), simulated as a pedal disparity, impaired stereothresholds when the error exceeded 1.5 min of arc.

**STIMULUS VARIABLES**

If stimulus conditions are varied, a person’s ability to discriminate depth differences will vary in accordance with normal physiology. These variations should affect binocular/oakal motor normals and is summarized in many texts. Of more interest is the series on “Vision and Visual Dysfunction” (and more specifically volumes 9 and 10 of that series) and the book “Binocular Vision and Stereopsis” by Howard and Regens.44

- Spinal frequency, size contrast and contrast sign, surround configuration, temporal frequency and presentation times, spatial crowding, color, target motion, and various differences between the stimuli presented to the two eyes provide a partial list. The reader is directed to such texts as given here for the study of stimulus parameters.

This article is more concerned with the individual differences in response to those stimulus characteristics, and even more specifically to the differences in the visual systems in which individual differences are more pronounced. Because our patients are idiopathic stereopsis situations to contend with, a few comments are warranted.

What advice can we give to our patients to maximize their stereopsis for particular tasks? Training for the stereoscopic task itself is a real possibility and is covered later in this article. Manipulation of stimulus variables to enhance stereopsis is possible in many cases and is certainly worthy of a study of its own. The list of examples given here is far from exhaustive, but it will serve to make the point.

Stereopsis depends on the use of horizontal disparity; therefore, the presence of vertical contour is desirable. Here, rotation of the field of view to enhance the number of vertical contours should prove beneficial. A dentist may simply change his or her direction of view into the working area. Similarly, the direction of lighting onto the field may be changed to enhance vertical contours. Simply putting more light coming in from different directions (using a light source with a large source or using multiple sources) should prove beneficial.

A portion of the effect may be the result of changing fixation disparity amounts. Pickwell et al.45 reported decreasing illumination increased fixation disparity; therefore, stereoscopic ability would be expected to decrease. Magnification can control the horizontal spatial frequency of the relevant contours. Better stereopsis is achieved around three to six cycles per degree of spatial frequency.46 Too much or too little magnification alike can be counterproductive. A surgeon operating through a microscope may find that one magnification level produces a better depth effect. Perhaps the stereoscopic scene itself can be manipulated. For instance, viewing the contour of a golf course from stereoscopic photographs taken from an airplane is very difficult, because it is composed of very high spatial frequencies (the grass) and very low spatial frequencies (the gently rolling terrain over most of it). If the photograph is taken when many people and objects are on the golf course, the number of vertical contours is increased in the spatial frequency range most conducive to achieving better stereopsis.

Finally, for those persons who may be working with a VD stereo display, the rules**7 that apply for best computer stereo viewing remain relevant.
and testing of the accommodative and/or the disparity vergence system. Because both of these systems, at least in the steady-state in which stereopsis is most commonly measured, are self-governing negative feedback systems, their inputs should reflect their normality. If the inputs are within a normal range, then the systems are operating in a manageable way. Conversely, if the inputs are abnormal, the systems are operating under a relatively unmanageable handicap—either some component of the system is too weak or the environmental demand (load) is too large, or perhaps a combination of the two. Control systems analysis offers a way to understand how the human visual system may be optimized or handicapped by the functioning of the accommodative and disparity vergence systems.

**BLUR, VISUAL ACUITY, AND REFRACTIVE FACTORS**

Blur and visual acuity factors affect stereopsis with blur being best thought of for the purposes of this article as having two divisions, that of being equal and unequal between the two eyes. Blur is the subjective sensation produced by a reduction of retinal illuminance gradients when the depth of field is exceeded. Such a physical degradation of the image is more apparent at higher spatial frequencies. Lege and Gu showed that the stereoscopic threshold had an approximately inverse square root dependence on contrast that was affected by the spatial frequency and that unequal contrast had an even greater detrimental effect. Thresholds were lowest at three cycles per degree. Heckmanns and Schoot noted that stroes sensitivity was strongly dependent on particular properties of edge information found that luminance gradient and spatial phase of the edges remained dependent on contrast and spatial frequency for their effect. Once again, decreased contrast and, even more strongly, unequal contrast caused a decrease in stereocuity. Lowvink and Smykawa imposed anisometropic conditions on 50 normal subjects and found that monocular defocus was very effective at reducing stereopsis. One-half disper of monocular defocus showed an effect with the effect increasing as the defocus increased. Interestingly, the effect was more noticeable when testing the left (Wert) Ring rather than global (Randot) stereopsis. Perhaps this difference relates to the spatial frequency compositions of the two tests. Lam et al. studied naturally occurring visual acuity differences between the two eyes and found that as little as one line difference in visual acuity had an effect on stereocuity. Goodwin and Rama found that similar amounts of monocular defocus caused different degrees of stereoscopic reduction in different people. Some subjects were affected little, others more, but all were definitely adversely affected when visual acuity was <20/40. The significant intersubject variation implies that the effect of stresses from other binocular ocular sources can become manifest when blur occurs. In a somewhat different vein, Wenzers et al. found that 1.5 D of monocular defocus can have a lasting effect on the disparity sensitivity in young monkeys, particularly at the higher spatial frequencies. This suggests that in young humans, uncorrected anisometropia can make somewhat permanent changes to that person’s ability to detect and process binocular disparities for depth discrimination tasks. Saladin reviewed over 2000 vision screening records from the Vision Enhancement Program of the Defense Mapping Agency (DMA), a division of the U.S. Defense Department. Of that number, 1539 were found to meet that agency’s criteria for stereop heterotropia. Those criteria required distance visual acuity of 20/20 or better in each eye, distance and near-horizontal phoria between ±5° eso and ±5° eso, vertical phorias <2Δ, and Howard-Dolman stereopsis scores (one standard deviation, null threshold) of 14.1 s of arc or better. The same 1539 data have been reanalyzed for visual acuity effects. Referring to Table 1, subjects with 20/15 in both eyes had better stereopsis than those with 20/20 in both eyes (unpaired Student’s t-test with p < 0.01). Comparing those with 20/15 in both eyes with those with one eye at 20/15 and the other at 20/20, a similar reduction in stereoscopic acuity was seen (p < 0.01). When those with 20/20 in both eyes were compared with those with 20/20 in one eye and 20/15 in the other eye, no statistically acceptable (p < 0.05) significant difference was found, although average Howard-Dolman scores were numerically larger in the group with unequal visual acuities. This research demonstrated the extreme sensitivity that small amounts of normally occurring reduced visual acuity have on stereoscopic thresholds when most of the other detrimental influences on stereopsis have been eliminated or controlled. No attempt was made in this research to separate those with small amounts of uncorrected refractive error from those with nonrefractive visual acuity differences.

There are differences in visual acuity that cannot be attributed to uncorrected refractive error, for instance, the amblyopias that frequently accompany strabismus and those that accompany anisometropia. According to Levy, such differences can be regarded as abnormalities in contrast sensitivity for high spatial frequencies for both types of amblyopia, although the two types of functional amblyopia do not share a common etiology.

Table 2 presents well-established criteria for the allowable limits of refractive error before correction is necessary if the goal is to maximally enhance binocular coupling or stereoscopic performance. Recently, Dwyer and Welch refined those criteria. With the sensitivity of stereopsis to differences in visual acuity demonstrated earlier in this article, the values in Table 2 for myopia, hyperopia, and anisometropia seem reasonable, and if anything, a bit too lenient. Assume that the small differences in visual acuity found in this study are the result of refractive defocus (for that small amounts of refractive defocus and small differences in best-corrected visual acuity have the same effect on stereopsis). If defocus blur and visual acuity

### TABLE 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Visual Acuity</th>
<th>Mean</th>
<th>S.D.</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>20/15 both eyes</td>
<td>8.60</td>
<td>2.86</td>
<td>928</td>
</tr>
<tr>
<td>B</td>
<td>20/20 both eyes</td>
<td>9.20</td>
<td>2.76</td>
<td>203</td>
</tr>
<tr>
<td>C</td>
<td>20/20 one eye, 20/15 other eye</td>
<td>9.40</td>
<td>2.66</td>
<td>208</td>
</tr>
</tbody>
</table>

*Optometry and Vision Science, Vol. 82, No. 3, March 2005*
TABLE 2.
The permissible limits of uncorrected refractive error if noticeable consequences of stress due to that refractive error on the accommodative and disparity vergence systems are to be avoided.

<table>
<thead>
<tr>
<th>Refractive Error</th>
<th>Permissible limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myopia</td>
<td>0.50 D</td>
</tr>
<tr>
<td>Hyperopia</td>
<td>1.00 D</td>
</tr>
<tr>
<td>Anisometropia</td>
<td>0.50 D</td>
</tr>
<tr>
<td>ATR Astigmatism</td>
<td>0.50 D</td>
</tr>
<tr>
<td>WTR Astigmatism</td>
<td>0.75 D</td>
</tr>
</tbody>
</table>

WTR = With the rule astigmatism; ATR = Against the rule astigmatism

Line differences from the DMA data are translated to dioptic values; these criteria could be made more stringent by at least 0.25 D. Such a translation is nonlinear but every practicing optometrist is accustomed to making it. The reason to be drawn is that refractive error should be corrected to within these limits or it has the potential to act as a handicap to proper function of the oculomotorbinocular system. Refractive error and visual acuity differences are clinically significant different blocks that act to handicap the input to the efferent and sensor-integrative systems. Certainly, they should be corrected before any modification (vision therapy) of those systems is attempted.

How can the effect of blur on stereopsis be explained? First, equal blur or decreased visual acuities will decrease the disparity detectors' ability to detect and process the horizontal disparity. Disparity detector gain will decrease (or the latency and/or time constant will increase), and fixation disparity will increase to maintain the same vergence controller output that is necessary to hold that particular vergence level. Interestingly, blurring one image tends to "unbalance fixation disparity" in that most of the misalignment is manifested in the blurred eye. Second, unequal blur affects one eye more than the other and thereby enables an active suppression process to be initiated. Once again, fixation disparity must increase to obtain the same output from the disparity vergence controller. When Simpson looked at the dynamic development of suppression with unispecic defects, he found that suppression did not become manifest over a range of ±0.50 D, but greater amounts of blur caused by the defective produced successively larger suppression scotomas: the more detailed the target pattern, the greater the effect.

The 0.50-D limit for myopia and anisometropia is supported by the DMA data (see Table 1). Because there was no control for hypopria in the DMA screening process, the DMA data cannot be used to support the 1-D allowable limit given in Table 2, the support must rest on other evidence. Obviously, age and ACA ratio may make a difference in this criterion value for the individual patient. However, as a general rule, leaving more than 1 D of hypertonic uncorrected in a vision therapy patient or in a person expected to operate at his or her peak vision performance level is unwise.

Booth mentioned that 0.50 D of against-the-rule (ATR) astigmatism can cause as much complaint as 1.00 D of with-the-rule (WTR), in approximate agreement with Table 2. Dyer and Wick found that refractive correction alone was better able to correct a vergence dysfunction in against-the-rule astigmatics than WTR astigmatics. There is a reasonable explanation for these differences between the two axis types of astigmatism. Freeman found that the accommodative system tends to focus the lines closest to the retina in compound myopic astigmatics. Compound myopic against-the-rule astigmatism blurs vertical lines more than horizontal lines, and it is the vertical lines (or the vector equivalents) that set up the physical arrangement for the horizontal disparity stimulus necessary for both horizontal fixation disparity and stereopsis. In mixed astigmatism in which one meridian is hyperopic, Freeman found that the hyperopic meridian is favored, once again blurring the vertical lines in against-the-rule astigmatics. The only exception to the pattern was for compound hypertonic astigmatics in which the lines near the retina were preferred, but even then, all hyperopic astigmatics did not share this preference. Therefore, for myopic and mixed astigmatics and some of the hyperopic astigmatics, Freeman's data are consistent with the idea that against-the-rule astigmatism is more problematic than WTR, because it blurs vertical lines more than horizontal. If the vertical lines are blunted, the horizontal disparity system is impaired both for control of vergence and for stereopsis.

SUPPRESSION

Suppression has a negative effect on stereopsis. Binocular inhibitory interactions effectively produce a lower contrast image (by reducing sensitivity) in the suppressed eye and a relatively higher contrast image in the nonsuppressed eye. Suppression is an active physiological process that is dependent on the stimulus situation; it causes a fall-off in stereoscopic ability that is well documented. This fall-off has at least two interrelated causes. First, a suppression area, no matter how small, always includes the central retina, and it is that central retina that is responsible for the very best of stereoscopic acuity. Second, suppression tends to decrease the gain of the disparity detector/vergence controller and to increase fixation disparity, thereby decreasing stereoscopic ability. Support for this statement rests on the work of Saladin and Carr. They found that fixation disparity curves of symptomatic subjects steepened as the diameter of the fusion lock was increased. Suppression acts to force the governing fusion lock radially (albeit asymmetrically) from the fovea and thereby increases the diameter of the governing fusion lock. Because the curves tended to pivot about the x-axis intercepts, the y-axis intercepts—fixation disparity with no prism stimulus—also tended to increase. Given that gain is a measure of how well the system converts fixation disparity into innervation signal, the greater the amount of fixation disparity necessary for a...
given innervational output, the less the gain of the vergence controller. In the Saladin-Carr research, the decrease in gain should be largely attributed to the disparity detector/vergence controller and not to the slow vergence adaptation mechanism because the forced vergence fixation disparity curves were determined with a 10- to 15-s prism exposure time. The curves are very largely a function of the sustained (as opposed to the transient) action of the vergence controller, particularly in symptomatic individuals. If such individuals had a slow vergence adaptation mechanism of sufficient gain and speed to reduce the dependence on the vergence controller, they would probably not be symptomatic. Ramifications of the eccentricity effect discussed by McCormack provide additional support for the concept that suppression decreases the gain of the vergence controller, but the relationship may not be straightforward and without exception.

Common clinical tests (Worth Four Dot, Keystone Visual Skills series) for suppression are usually not sensitive enough to detect the presence of the small amounts of suppression that will begin to affect stereopsis adversely. One must resort to the Pola-mirror or Vii-6iv tests, or other high-spatial frequency and/or low-contrast tests. Clinically, the opposite approach is more often taken: stereoptic tests are used as indicators that suppression is or is not present.

ANISEIKONIA

As little as 1% difference in magnification between the two eyes can decrease stereopsis, and the effect increases with aniseikonic amount. Concentrating on the more directly relevant horizontal magnification differences, what are some possible explanations for this loss? First, aniseikonia presents the visual system with similar contours that fall on noncorresponding points, producing the ideal arrangement for suppression. Here, the degradation of stereopsis follows the path of suppression's effect on stereopsis as just described. The interocular size difference causes a loss in stereoscopic acuity more from differences in edge positions than differences in spatial frequencies across the two images. Second, the rotation of the longitudinal horoptor around a vertical axis that occurs with aniseikonia in the horizontal meridian has at least two possible consequences, the effects of which would be small, especially in the foveal area where stereopsis is most sensitive. First, an observer suffers a decrease in ability to stereoscopically localize objects in the objective frontoparallel plane because objects to the left and right of fixation fall off the horoptor. Even small amounts of angular distance off the horoptor have an effect on stereopsis. Second, an observer is also presented with differing amounts of, or even oppositely signed, disparity on either side of the fixation point. These effects would be small but should result in some detrimental effects—perhaps a loss of sensitivity—on the vergence disparity system and a consequent loss (or at least some destabilization) in stereoscopic ability. If disparity vergence is influenced by slanted surfaces, how much aniseikonia would be needed to mimic the effect?

ANISOPHORIA

Clinical experience has taught that aniseikonia resulting from the spectacle correction of anisometropic refractive error is less responsible for the often accompanying asthenopia than is the simultaneously induced anisophoria. This unequal induced prism at various directions of gaze encountered by the two eyes poses problems for stereopsis. Although the ocular motor system has an amazing ability to adapt to these anisophoric demands, some patients simply lack an adequate ability to adapt. Even with the adaptive ability present, that ability may require considerable time to build to a useful amount. Just how this adaptation occurs is currently under research, but such an adaptation would imply that the system had somehow "learned" to vary the interocular pattern to the extraocular muscles with direction of gaze. Although a demand for asymmetric vergence occurs in real life even without lenses before the eyes, the direction and amount of the adaptation must be adjusted for the induced prism pattern appropriate for the corrective lenses. Without such an adaptation, the disparity detector/vergence controller output must change constantly as gaze angle changes. This constant change of disparity detector/vergence controller output should lead to discomfort and presumably fatigue, a decrease in disparity/vergence controller gain or sensitivity, an increase in fixation disparity amount and instability, and a commensurate drop in stereoscopic performance.

I am not aware of any research that has specifically addressed the effect of repeated vergence stimulation on the gains and time constant of the vergence controller and of the slow vergence adaptation mechanism, although an effort of fatigue—decrease in velocity—on the vergence system has been investigated. On the other hand, with such an adaptation, whether a change in fixation disparity pattern with gaze angle occurs depends on whether in the innervational sequence the adaptation is located. If the adaptation is preprogrammed into the vergence movements, the fixation disparity pattern would be symmetric with gaze angle, and the cascade of events leading to a drop in stereoscopic performance should not occur.

HETEROPHORIA

The relationship between heterophoria and stereopsis has been studied, but such investigation as often as not has led to considerable frustration. The relationship is not direct. In the early 1940s, Pay and Kent determined that induced phoria bore no predictable relationship to stereoscopic acuity. Looking at naturally occurring phorias, Casten et al. found no correlation with observed stereoscopic acuity. On the other hand, in the applied, stereopsis-demanding world of stereophotogrammetry, it has long been known that some relationship must exist. Saltzman, Dwyer, and Moore and Bryan forcefully state that esophoria makes the best stereocompilers. I used the data from the DMA vision screenings to investigate the relationship that horizontal and vertical heterophoria had with stereoscopic test scores. This database (described earlier) is distinguished, because it had been carefully filtered to remove most of the common motor and sensory dysfunctions and has the very large sample size of 1339. Fig. 3 is a plot of small amounts of horizontal heterophoria (ΔH eso to 4Δ H eso) and the corresponding average Howard-Dolman scores. Assuming that correlation here implies some effect, Fig. 3 suggests that relatively small amounts (arguably any esophoria at all, but with a definite effect by 3Δ) of esophoria will adversely affect stereopsis, but the ability of the system to overcome the effects of esophoria is con-
FIGURE 3.
The average Howard-Dolman scores are plotted against the various horizontal heterophoria amounts with the bars representing plus or minus one standard deviation of the mean. The number of subjects at each phoric amount is given at the bottom (32).

considerably more robust. Taking into consideration the height of the standard error bars, one can see that a given exophoric amount has less impact than esophoria of a similar amount. This graphic observation was supported by statistical analysis.32 An asymmetry definitely exists between the esophoric and exophoric results. Compared with exophores, esophores as a group tend to have reduced stereopsis, and the greater the esophoria, the greater the effect over the 9Δ exo to 4Δ eso range used in the DMA research. Moore and Bryan33 noted a similar difference between exophores and esophores on an actual stereoscopic task, namely stereophotogrammetric contouring. During contouring, the photogrammist must maintain a constant Z-axis elevation while moving in the flat X-Y plane (for example, going around a hill maintaining the same altitude). Saladin et al.34 noted a similar difference between exophores and esophores on the stereophotogrammetric profiling task. During profiling, the photogrammist must maintain movement in a constant X or Y direction and manipulate the Z-axis elevation (for example, going straight south and determining the relative altitude of the terrain at each point).

One possible explanation for the asymmetry between the esophoric and exophoric stereopsis scores lies with the fixation disparity—heterophoria relationship. Fig. 4 is an idealized curve adapted from Ogle35 and further supported by data from Janoplosky et al.36 The direction and amount of the heterophoria is compared with the accompanying amount of fixation disparity. There is good reason to believe that this function represents the average heterophoria–fixation disparity relationship from acceptably normal binocular oculomotor systems.84,85 In other words, this function represents the expected variation in fixation disparity given the direction and amount of the horizontal heterophoria in a normal system. Inspection of Fig. 4 shows that the fixation disparity amount is not linearly dependent on the exophoric amount; indeed, amounts of heterophoria up to seven or eight prism dipters seem to be accompanied by almost the same relatively small amounts of exo fixation disparity. In contrast, for esophores, the relationship is much more linear over the range tested, approaching an easy-to-remember 1Δ of esophoria for each 1 min of arc fixation disparity. Sheedy and Saladin37 found that esophores tended to have much smaller fixation disparities for a given amount of heterophoria and could tolerate without symptoms much larger amounts of phoria than could exophores. In comparison, esophores tend to have more fixation disparity and more symptoms at even a small (3Δ) diptriometric amount of heterophoria. Although small amounts of fixation disparity (<10 min of arc)38 are acceptable and even expected with a normally operating disparity vergence system, even 1 min of fixation disparity causes a penalty in a reduction of stereopsis.34,35 In the clinical situation, fixation disparities exceeding 6 min of arc eso and 4 min of arc exo are considered to be of diagnostic significance.

The control system model (Fig. 2) of the oculomotor system provides a platform for further understanding of the physiological basis for this asymmetry. In the steady-state situation in which sufficient time has elapsed to enable sustained accommodative and disparity vergence mechanisms to be ascendant, esophoria shows a much less linear relationship with stereopsis than does esophoria because esophores depend on the (positive) slow vergence adaptation mechanism to overcome the heterophoric stress much more than exophores depend on the oppositely directed (negative) slow vergence adaptation mechanism. The positive slow vergence adap-
stereoscopic mechanism has a greater potential gain and more effective dynamics than has the negative slow vergence adaptation mechanism.\textsuperscript{46–48} Hung et al.\textsuperscript{49} provided an extensive defense of the concept that convergence and divergence dynamics are different with the convergence system definitely more robust. Of special note is the work of Patel et al.,\textsuperscript{50} who used long (5- to 90-s) vergence exposure times and found that the divergence and convergence mechanisms had different adaptation characteristics. Five- to 90-s adaptation times make this research by the Patel-Jiang group directly relevant to clinical situations.

Finally, looking at the asymmetry between the stereopsis scores for esophoric and exophoric, esophoric can augment reflexive convergence with voluntary convergence; voluntary convergence does not exist in the same manner. Such voluntary convergence can augment the initial vergence movement when the more reflexive coarse disparity vergence mechanism is inadequate to provide innervation for a large disparity stimulus. Thus, large esophories can use volition to put the vergence level within range of the fine disparity vergence system. Exophories can become very proficient at this.\textsuperscript{51} Esophories do not have this option; they must rely on the more reflexive coarse (phasic) and fine (sustained) mechanisms of the vergence controller.

From a clinical standpoint, the approximate correlate to the gain of the vergence adaptation mechanism is fusion vergence amplitude, and, in general, negative or divergent disparity (fusional) vergences seem inherently less robust than the accompanying positive disparity vergences. Therefore, because the innervation to provide the divergence has to come from somewhere in an esophoric system, and the negative slow vergence subsystem cannot be relied on to provide that innervation, the fine or sustained disparity detector system (Fig. 2) must provide that innervation. Although a normal disparity vergence system under steady-state conditions has a relatively high gain factor (150 to 200),\textsuperscript{52} that alone is insufficient; fixation disparity must be increased much more than if the adaptation mechanisms were available to contribute to the necessary innervation. Disparity detector\textsuperscript{53} vergence controllers are also prone to saturation effects (fatigue, addressed later in this article), leading to even more fixation disparity. Once again, an increase in fixation disparity will lead to a decrease in stereoscopic performance.

There are consequences of increasing fixation disparity. A large heterophoria will cause little problem if the slow vergence adaptation mechanism\textsuperscript{54} is able to contribute the greater part of the innervation and therefore permit a decreased demand on the vergence controller (sustained portion) system. If the slow vergence adaptation mechanism is not sufficient for the task, a larger portion of the innervational demand will be put on the sustained vergence controller system and the fixation disparity will increase. If fixation disparity increases past the limits of central Panum's area (given the stimulus situation\textsuperscript{55}), then central suppression must develop to avoid diplopia. The cure for inadequate innervation—more fixation disparity—now begins adding to the problem, because the central retina provides a better fusion lock than peripheral retina, and it is that very same central retina that must now be suppressed.

Of course, the finest of stereopsis requires the most central retinal elements.

Addressing phorias in other than the horizontal direction, a small amount of vertical phoria can cause a relatively large amount of fixation disparity, because vertical adaptation mechanisms are not well developed. Poor vertical adaptation ability leads to a steep vertical fixation disparity curve.\textsuperscript{6} Fixation disparity will affect stereopsis regardless of whether the fixation disparity is vertical or horizontal, and the effect seems to compound when fixation disparities exist in both directions. London and Wide\textsuperscript{41} found that the slope of the horizontal fixation disparity curve was decreased if a naturally existing vertical fixation disparity was corrected with vertical prism. Inspection of their data shows that the amount of the horizontal fixation disparity at the vertical axis intercept was also reduced. Remole\textsuperscript{56} used fixation eccentricity and the border enhancement phenomenon to indirectly measure a fixation miscalignment, which correlated to fixation disparity. Given that such a third-order effect is truly reflective of fixation disparity, Remole found that a vergence stress or imbalance in one meridian will amplify the disruptive effects of a stress or imbalance in another meridian. Therefore, both horizontal and vertical fixation disparities will act to decrease stereoscopic detection and recognition. Let it be remembered, however, that horizontal fixation disparity directly impacts the essence of the stereoscopic stimulus, namely horizontal disparity, whereas vertical fixation disparity will have an indirect effect. Thinking of the problem at the corneal level, with vertical fixation disparity in place, vertically disparate horizontal elements must read the horizontal disparity.

The DMA data were analyzed for the effect of vertical heterophoria on the Howard-Dolman stereopsis scores (Table 3). The difference between the stereopsis scores of those who manifested vertical orthophoria and those with greater than 6 but <2Δ was found to be significant (unpaired t-test, t = 2.7, p = 0.006 one tail). Thus, as a group, the subjects with the slight vertical phoria permitted by the screening criteria had a small but statistically significant reduction in stereoscopic test performance. Feidman et al.\textsuperscript{57} varied retinal disparity over the 90 deg between horizontal and vertical and noted that the stereoscopic effect was sinusoidally related with the maximum stereopsis at horizontal and the least (none) at vertical.

Once the asymmetry between the esophoric and exophoric stereoscopic sensitivities is understood, the explanation for the strong dependence of stereoscopic threshold on vertical phoria is apparent. Vertical vergence systems are even less able to rely on slow vergence adaptation mechanisms at the response times needed for practical everyday tasks. As stated before, visual fixation disparity curves are characteristically steep and usually do not exhibit the relative flattening in the middle suggestive of a significant slow vergence adaptation mechanism with a time constant comparable

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\textsuperscript{4}In the actual situation, it is difficult to separate the adaptation processes of the sustained portion of the vergence controller and that of the slow vergence adaptation mechanism. Schor\textsuperscript{46} understood this dilemma over 20 years ago, when he addressed an effect of orthophoria,\textsuperscript{47} orthophoria to increase the rate and magnitude of slow fusional vergence and perhaps the gain or velocity of the fast fusional vergence mechanism.\textsuperscript{48} In a similar line of thought, Hung\textsuperscript{49} combined the effect of a long-term adaptation mechanism with that of a shorter-term mechanism in the controller, using the output of the latter to be the input of the former.

\textsuperscript{5}A device such as a Steddy Dipimeter or a Saladin Near Point Balance Card that permits an actual measurement of the angular amount of fixation disparity is necessary. The Dipimeter is no longer available at the time of this writing, but the Saladin Near Point Balance Card is available from Vision Training Products Inc. (Bernei).
TABLE 3.
The vertical phoria and stereocuity measurements of those with essentially zero vertical phoria and those with a slight vertical phoria are compared. The difference between the average Howard-Dolman scores (8.7 and 9.3 seconds of arc) of the two groups was small but statistically significant (unpaired t test, p = 0.004, one-tailed).

<table>
<thead>
<tr>
<th>Vertical Phoria and Stereocuity</th>
<th>Mean</th>
<th>S.D.</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.5 Δ</td>
<td>8.7</td>
<td>2.8</td>
<td>1125</td>
</tr>
<tr>
<td>≥0.5 Δ but &lt;2 Δ</td>
<td>9.3</td>
<td>2.7</td>
<td>214</td>
</tr>
</tbody>
</table>

to that of the horizontal slow vergence adaptation time constants. Vertical vergence adaptation mechanisms are very slow (6 minutes). Therefore, the only recourse: the vertical system has is to increase vertical fixation disparity to increase vertical disparity detector/controller output. This increase in fixation disparity will cause a definite degradation of stereoscopic performance. Anecdotally, I have seen patients with large vertical phorias who had very good stereopsis and no real complaints. When questioned, these patients tended to have or "always had" a vertical imbalance at least as long as they could recall. I suspect that the vertical slow vergence adaptation mechanism can be made to perform as strongly as the horizontal if the vertical imbalance is successfully confronted at a very early age. This would imply that a time period of plasticity may exist in the cortical/brainstem elements controlling vertical vergences in which cells may be allowed to the task of providing the innervational support for vertical disparity vergence movement and maintenance. If so, this time of plasticity concept has implications for the case management of young, vertical heterophores.

Ogle and Ellerbrock found stereoscopic abilities to be impeded by cyclophoria. Research in this area is difficult because the same stimulus pattern that will cause a cyclophoria movement can also be perceived as a tilt. Depending on the stimulus pattern, either or both a vertical and a horizontal fixation disparity will need to occur to produce the innervational signal to maintain eye position in the presence of a cyclophoria. The resultant fixation disparity will produce a decrease in stereoscopic abilities. Although majority opinion at the present time is that the cyclophoria stimulus is positional (horizontal with some vectored vertical effect) disparity rather than some torsional disparity, the physiology remains unclear. The response has been reported to be a stimulus-dependent mixture of actual motor motion about an anterior-posterior axis and an apparent sensory shift that was interpreted as a change in correspondence. Recently, Taylor et al. found that the torsional system operates as though there is a separate cyclophoria adaptation mechanism that has similarities with the vertical and horizontal adaptation mechanisms. It is interesting to note that a person need not have a cyclophoria to be presented with a cyclophoria-inducing stimulus pattern. Such patterns naturally occur when one looks at inclined contours and/or at contours located in the tertiary gaze position.

AC/A RATIO

An abnormal AC/A ratio can have an effect on the stereoscopic process. If the near and far horizontal phorias are not equal, the disparity vergence loop must adjust for a new innervational pattern every time the person changes fixation distance. If the task requires a fast, repetitive response, the innervational change must be supplied by the disparity detector/vergence controller system components (corneal and lens) because the slow vergence adaptation systems cannot follow rapid alternations. When the slow vergence adaptation mechanism is abnormal (again too low or too horizontal), the innervational demand on the disparity detector/vergence controllers is increased even more. Thus, abnormal AC/A ratios can create increased demand on the disparity/vergence system components, leading to greater amounts of fixation disparity. Hung and Cuffrech described the extreme sensitivity of the accommodation and vergence systems to the AC/A and CA/C crosstalks. Of course, in the steady-state condition, an abnormal AC/A ratio can lead to a large horizontal heterophoria and the attendant abnormal motor demand on the system.

AGE

In general, stereoscopic test results improve with age until midlife approaches, remain stable for a few decades, and then begin a seemingly inexorable decline. The effect is far from linear. Jain using the Dextero test, reported that stereoscopic ability increased through the teen years, remained stable until the 40s, and then decreased. In one of the best studies to date on older people, Wright and Wormald used the Fruity Stereotest to study the effect of aging on stereopsis, controlling for the effect of visual acuity. Fifty-nine of 120 65 to 69 year olds had scores of 55 s of arc or better (numerically less), whereas only 16 of 68 of those over 80 years of age had scores of 55 s of arc or better. Although the methodology in the article is difficult to interpret, Ospina reported testing the stereoscopic thresholds of 1810 subjects. He found that the thresholds of children ages 4 to 6 years averaged 80 s of arc, children ages 16 to 20 years averaged 55 s, young adults from 21 to 40 years showed a 45-s average, and then the threshold began to rise again, reaching 60 s of arc by age 60 years. Table 4 compares age with Howard-Dolman performance using the DMA database (n = 1339). The conclusions from this database are important, because the study itself is well controlled for extreme factors. The scores of those employees in their 20's

TABLE 4.
Age and stereocuity. The Howard-Dolman scores of the various age groups are compared. The scores of those employees in their twenties were statistically less than those in their thirties (unpaired t-test, p = 0.009). Those employees in their thirties, forties, and fifties were statistically the same. Those employees in their sixties had scores of greater numerical value than those in their fifties but only at the p = 0.09 level.

<table>
<thead>
<tr>
<th>Age</th>
<th>Mean</th>
<th>S.D.</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>22-29</td>
<td>8.6</td>
<td>2.7</td>
<td>483</td>
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<tr>
<td>30-39</td>
<td>9.0</td>
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<td>461</td>
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<tr>
<td>40-49</td>
<td>8.9</td>
<td>2.9</td>
<td>234</td>
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<tr>
<td>50-59</td>
<td>9.0</td>
<td>2.7</td>
<td>138</td>
</tr>
<tr>
<td>60-68</td>
<td>10.7</td>
<td>2.2</td>
<td>23</td>
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Optometry and Vision Science, Vol. 82, No. 3, March 2005
were numerically less than those in their 30’s (unpaired t-test, \( t = 2.4, \alpha = 0.009 \)). There was no statistical difference between the scores of those in their 30’s, 40’s, and 50’s. If the scores of those in their 50’s are compared with those in their 60’s, a trend for a difference probably exists (unpaired t-test, \( t = 1.35, \alpha = 0.09 \) one tail). The overall result of comparing age with stereoscopic ability is that those stereophotogrammeters in their 20’s had a small advantage; those ages 30 to 30’s were on an equal footing; and those over 60 may be at a disadvantage if taken as a group. When the DMA data results are compared with the results of the other studies reported here, there is good agreement.

What could cause the age-related changes in stereoscopic precision? The increase in stereoscopic ability in the early years may come from some form of learning to interpret the sensation. It may also come from the stabilization of the accommodative and disparity vergence interaction that comes about from the early school years through the mid-20’s. Schor\(^{107}\) described the manner in which the two systems are able to reach a stable innervational interaction in that younger age range when the CA/c and ACA are evenly matched. It is in these years that the CA/C ratio is at its highest, but (presumably) it decreases\(^*\) with age, and therefore the whole system becomes progressively governed by the much more age-stable ACA ratio. This should lend stability to the overall system. Hypothetically, it may not be the stereoscopic potential (threshold) that improves over the younger age groups, but the stereoscopic stability—a qualitative aspect. Research could be
designed to test whether the early (ages 5 to 25) increase of stereopsis test scores is the result of qualitative or quantitative factors. Of course, maturation/perceptual and testing challenges may be powerful factors, particularly at the younger age.

The decrease in stereoscopic acuity with advancing age has been discussed in the literature much more than the aforementioned increase at earlier ages. Heckenmnan and Schor\(^{107}\) showed how very dependent stereopsis is on contrast sensitivity. Greene and Madden\(^{105}\) noted that the loss of stereoscopic acuity with age was correlated with loss of contrast sensitivity. Adams et al.\(^{109}\) studied the loss of light transmission through the ocular media that occurs with age and suggested that this loss could decrease the contrast sensitivity. Schneck et al.\(^{110}\) using the Frisby Stereotest at 40 cm on some 900 individuals between the ages of 58 and 102 years, attributed a large decline in coarse stereopsis mostly to light scatter and retinal problems. This is to be contrasted to the study of Wright and Wormald\(^{108}\) who thought the decline in stereopsis was the result of more centrally located factors. Wright and Wormald\(^{108}\) speculated on the neural effects of aging and reported that defective stereopsis occurs with Alzheimer’s disease regardless of age. Cortical disease/lesions can also cause disturbances in spatial vision.\(^{111}\) Habak and Fabert\(^{112}\) thought that aging tended to affect the higher-order stimuli that require considerable processing and speculated that it is the processing changes that affect performance. One cannot help but wonder if the lack of short-term memory that is a mark of such degenerative changes as occur in Alzheimer’s disease may be preventing a perceptual buildup and organization of visual space. In still another direction, Winn et al.\(^{113}\) offered evidence that vergence adaptation decreases with age. As explained earlier, if vergence adaptation decreases, then fixation disparity must increase, negatively affecting stereopsis.\(^{4}\) As Bender\(^{115}\) pointed out, “the reticular activating system and the oculomotor pathway are located in the same zone of the brain stem tegmentum.” From an anatomic viewpoint, Mays et al.\(^{116}\) found cells in or near this reticular formation that behave as if they might contribute to the slow vergence adaptation mechanism. Finch and Schneider\(^{117}\) mentioned that age affects activity in this reticular activating system as it does in other portions of the brain. Spear\(^{118}\) although not speaking of stereopsis specifically, noted the “mental and visual attention” effects on hyperacuities with age. Clinicians are also very cognizant how vergence ranges collapse if wakefulness/alertness decreases during a vision examination. Finally, looking in a different direction for a possible explanation for the decrease in stereopsis with later age, significant differences between normal and glaucomatous subjects were found using stereoscopic visual-evoked potentials (VEP).\(^{119}\) Perhaps some subtle retinal problem such as low-tension glaucoma\(^{120}\) could be making itself felt. That same kovale region that is necessary for the finest of stereopsis is also necessary for proper VEP signal.\(^{121}\)  

**FATIGUE**

When the topic of fatigue is addressed, one must immediately ask: what is fatigue and exactly what is being fatigued? Fatigue occurs when some power or capacity to respond is lost because of excessive activity.\(^{122}\) Taking a very broad view, Bartley\(^{123}\) defined visual fatigue as a personal state, not a restricted tissue condition or a localized discomfort, that results in a general inability or aversion to a visual task. This view combines the effects of the physical, physiological, and psychologic. While recognizing that Bartley’s view has the widest clinical application, for the purposes of this article, I consider fatigue in the more objectively quantifiable sense—a measurable loss of ability to perform a task. Furthermore, whatever entity or process that is fatigued should be at least partially dependent on the vision process. For instance, although a thought-occupying task such as reading\(^{124}\) may be a contributor to fatigue, it is not the sole contributor. In other words, some part of the fatigue should be local to the visual system.

Thinking of fatigue as a measurable decrease in task performance, Yuan and Semmlow\(^{127}\) exposed four subjects to 500 sacadic changes, 100-step vergence changes, and 100 sinusoidal vergence changes to induce fatigue. They found that the peak velocity of the eye movement decreased after the 100-step vergence changes

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\(^{4}\) know of no research that specifically addresses how fixation disparity might change with age at distance. Shockey and Saladin\(^{114}\) addressed the possible change at near. In this research, vision was thought to confound the picture; therefore, any such research should be conducted at distance to reduce the number of possible variables.
but not after the 160 sinusoidal vergence changes. Furthermore, it took a much larger number of saccadic changes to cause a similar effect on the dynamics of the required eye movement. They also found that there were more instances of the total step vergence movement breaking down into two smaller vergence movements and concluded that repetitive vergence eye movements induce fatigue in the neural processes that produce the pulse command signal. Finally, they summarized by saying that the fatigue comes from the modification of brainstem neural processes rather than the modification of neuromuscular efficiency. It would appear that the fatigue—performance loss demonstrated in the Yuan and Semmlow vergence experiments are somewhat attributed to the plastic or fast process in the vergence controller rather than the sustained-Adaptive process further downstream in the model of the disparity vergence system.

Defining fatigue as a deterioration of performance on a task with repetition, Schor and Tsutakawa found that with 4-min ramp-tracking exercises, the accommodative and the disparity vergence systems can both be selectively fatigued and thereby change the ACA and/or CAC values and interactions. Quoting Schor and Tsutakawa, "The site of fatigue caused by ramp-tracking exercises is not likely to be at the accommodative plant or the extracocular muscles since the plants for accommodation and vergence do not fatigue easily. Rather, fatigue is likely to occur centrally, at sites where adaptable tonic accommodation and tonic vergence are controlled and perhaps also at sites where phasic responses of optical reflex accommodation and disparity are controlled."

With regard to the model (Fig. 2), both of the just-mentioned studies indicate the disparity vergence controller and perhaps the SVA as a locus for fatigue effects. The inputs to the extracocular muscles and to the ciliary muscle must be maintained if clear focus and acceptable binocular alignment are to occur. Therefore, inefficiencies anywhere in the system must be compensated for in some manner. Referring to the model (Fig. 2), the input of the disparity vergence plant is the integrated output of tonic, proximal, accommodative, and disparity vergence interactions, the various adaptation mechanisms, and the disparity detectors. To make up a deficiency, one or more of these entities must change its output to maintain the same input to the extracocular muscles. Barring the possibility of some complex adaptation process, the most likely candidate to change its output is the disparity detector/vergence controller, and more specifically, disparity detector output, which would require an increase in fixation disparity. Once again, increased fixation disparity will lead to a decrease in stereoscopic ability.

Does fixation disparity change with fatigue? García and Dyer altered fixation disparity curves on asymptomatic participants, but not on asymptomatic patients, with a 25-min reading task. The implication was that something changed after the reading task in the symptomatics that affected fixation disparity, and that change was interpreted as an indication of fatigue. A concentrated reading task does not require repetitive changes in vergence, so whatever is fatigued here is not closely related to the fatigue effects of Yuan-Semmlow and Schor-Tsutakawa. "Putting it all together, I must ask the question, "Would fixation disparity change after jump vergence repetition?"" Taking the opposite tack, Pickwell et al. showed that rest decreased fixation disparity. In a related but separate vein, Schumer and Ganz suggested that the stereoscopic system as a whole may be subject to fatigue, perhaps indicating that the higher interactive aspects of stereopsis were somehow involved.

**PERCEPTUAL CHALLENGES**

Looking to the more perceptual aspects of stereopsis, some patients have difficulty with certain stereopsis tests because their hierarchy of depth cues is not optimal for those tests. For instance, the DC Aviator Test and the Verhoef Stereoptikon both require that the observer ignore size cue and depend on the disparity cue as the main arbiter for the percept. In these two tests, the larger of the comparison objects may be in relative uncrossed disparity and therefore should be seen farther away. If the observer's perceptual system gives the size cue greater weight relative to other cues, the observer will see the larger of the targets as nearer, although it is in uncrossed disparity compared with the reference target. McKee et al. found that stereopsis is very good at breaking camouflage for static targets but is less effective in that role for moving targets. Just how good depends on the observer's depth hierarchy and the stimulus pattern. During World War II, there were many instances of stereopsis being used to "see through" camouflage. In one specific instance, camouflage netting had been used to conceal a series of buildings being used to build German jet fighters. A very experienced stereophototomographer looked into a stereoscope and could see the buildings that had eluded the less experienced photographic interpreters. Presumably, the experienced interpreter had "trained" her perceptual system (or she knew what to look for) to perceive the disparity-delineated three-dimensional structures through the netting. This example shows the power of a predetermined depth hierarchy and that the hierarchy may be overcome if the salient disparity characteristic is emphasized. Acknowledging that stereopsis can be used to group parts of the scene together, Morgan stated that common depth is at least as powerful as common fate (color, form, and so on) and is useful in overcoming camouflage. The message to clinicians is that they must make sure that the patient really understands the task for a stereoscopic test. A poor performance on the stereopsis test may be the result of the patient using the wrong cue to determine the percept.

**CORRECTIVE PROCEDURES**

If stereoscopic ability is found to be deficient, the corrective process for binocular/oculomotor dysfunction is sequential. Any attempt to better a patient’s stereoscopic performance must begin with ensuring that clear images are on the retina and that they are being faithfully relayed to the cortex. In other words, the refractive error must be corrected and acceptable visual acuities demonstrated. Table 2 shows time-tested criteria for refractive correction. Clear and balanced images present the disparity detectors with the ideal situation to accomplish their intended task: detecting disparity. Given appropriate monocular fixation abilities, the next step is vision therapy for accommodative problems, because accommodation is essentially a monocular process and does not depend strongly on binocular aspects. In other words, one can treat accommodative problems independently of disparity vergence problems, but the reverse, although possible, is clinically much
more difficult. Proper accommodation is necessary for proper focus so that disparity may be detected and processed at all distances.

Vision therapy for problems of binocular/ocularmotor problems can be monitored through fixation disparity and accommodative lag. At 6 meters, the accommodative state should have 0.25 to 0.50 D of lead, that is, relative hypoaopia. At 40 cm and all reasonable near working distances, 0.25 to 0.75 D of lag (relative hyperopia) should be in place, and the steady-state accommodative response should be stable to within 0.25 D. Lead/lag of accommodation is perceptually acceptable because of the depth of field but is physiologically demanded for proper control of the accommodative system. Fixation disparity should be minimized (4-min eso to 6-min exo) and stabilized (maximum variation of 2 min of arc) at a level appropriate for that phoric condition (Fig. 4) with the understanding that the smaller and the more stable, the better. Fixation disparity is much more sensitive to oculomotor dysfunction than the accommodative system, because it controls a system that is a disparity vergence system, which requires control to much greater precision and has a very high gain. The various aspects of the forced vergence fixation disparity curve are often used to follow the progress of vision therapy. Here, the ability of the system to withstand vergence stress and remain under acceptable reflex control is monitored.

Any factor that increases fixation disparity amount or variability will have an adverse effect on stereopsis (and, by implication, binocular/ocularmotor function). An increase in the amount of fixation disparity will increase the stereoscopic threshold (acuity) and so worsen stereopsis. An increase in the variability of fixation disparity will lead to a decrease in some aspect of the quality of the stereoscopic perception, because reliability over time and differing conditions are adversely affected. If we set as the goal of vision therapy to normalize and stabilize fixation disparity, we will be forced to attend to all of the other factors of oculomotor and binocular input. Stereopsis is at the top of the food chain of binocularity, and fixation disparity is its nearest and most direct controlling factor. All of the other factors input either in parallel with fixation disparity or in series further down the chain and through fixation disparity.

How does vision therapy normalize and stabilize fixation disparity and therefore improve stereopsis? Think of the control system model (Fig. 2). Given good monocular function and a vision therapy establishment better reflex automatic control of the accommodative system. During steady-state binocular operation, the accommodative system should be controlled by the blur provided by the accommodative lag with some minor input from the disparity vergence system via convergence accommodation. It is a negative feedback system and therefore acts to control itself. Given reflexive correction, the main opponent to this reflex automatic control is voluntary control, which cannot melt out the interaction with the precision needed by the system. Reflex control will provide a smooth flow of interaction both to the accommodative system and to the disparity vergence loop via accommodative convergence. As far as the disparity vergence system is concerned, vision therapy decreases suppression and therefore increases the gain and response speed of the disparity detector/vergence controller. It also makes the slow vergence adaptation mechanism more rigid by increasing its gain and velocity. Both the decrease of suppression and the enhancement of the vergence adaptation mechanisms act to decrease the effective demand on the disparity detector output and, therefore, to decrease fixation disparity. A decrease in steady-state fixation disparity will permit a lower stereoscopic threshold to manifest itself, thereby improving the quantitative aspect of stereoscopic ability. The reflex-controlled disparity vergence system can now precisely and smoothly control itself. At the end of the vision therapy process, both the accommodative system and the disparity vergence system should be under smooth and efficient reflexive control, leading to the optimum level in reliability and thereby improving stereoscopic quality.

VISION THERAPY AND STEREOPSIS

What is the proof that vision therapy will improve stereopsis, and what is the proof that fixation disparity is so tightly linked to that improvement? Wittenberg et al. 137 attempted to train disparity sensitivity itself with a Bauch and Lomb M-2 trainer (Bauch and Lomb, Rochester, NY), a modified Bremner stereoscope. They reported an improvement, but because no feedback was provided, the improvement could have been attributed to either or both a sharpening of the disparity detectors’ ability and/or to some improvement in the person’s ability to intellectually interpret that disparity. 138 One is left with the question of what was improved.

Saladin and Rich 139 made no attempt to modify stereoscopic skills themselves, but instead concentrated on maximizing all of the supporting visual skills. They trained monocular eye movements, performed antisuppression exercises, exercised the accommodative skills, and improved jump and ramp vergence abilities. They used 13 control and 13 experimental subjects matched according to binocular system characteristics. None of the subjects had significant refractive or binocular problems. They found a significant effect of vision training on two of the three tests for stereopsis used. Those two, the Howard-Dolman and a similar test using a stereotester, required a great consistency of stereoscopic ability. Interestingly, the Howard-Dolman and stereotester scores were highly correlated (r = 0.63; p = 0.04), but only in the experimental group after vision therapy. The third test, the Keystone Multi-Stereo Test, was a test for stereoscopic threshold with very few qualitative aspects; great reliability was not required. The conclusion was that enhancement of the visual oculomotor and binocular skills tended to help the qualitative aspects of stereopsis more than the threshold-determined quantitative aspects in normal oculomotor systems. The two test/tasks with large qualitative components were the ones most affected by the vision therapy procedures. I emphasize that the subjects all had clinically normal visual systems before the experiment began, and so all started with an acceptable level of stereoscopic performance. If subjects with binocularly abnormal visual systems had been included, the results should have been more dramatic with the quantitative measurements showing a definite improvement. Referring back to the observation that the Howard-Dolman and stereotester scores were highly correlated, one wonders if local and global stereopsis scores might be better correlated after vision therapy, and, if so, exactly what kind of therapy.

Saladin et al. 9 looked at the effect that maximizing the supporting visual skills had on a standard stereophotogrammetric task using 38 experienced stereotest technicians, 19 experimental
and 19 control. All subjects were employees of the DMA. Once again, none of the subjects had any significant refractive or binocular problems; they all passed the DMA vision requirements. As explained before, during this task (profiling), the operator must adjust the vertical height of a floating point as it travels at a constant speed in a straight line across a stereoscopically viewed terrain. He must "keep it on the ground" on the way up the profile and again back down the same profile and, for this research, with 622 points visited and recorded on a round trip. Because there was no opportunity for bracketing at each of the points, the photogrammetry fixed on the floating point and kept the ground at the edge of his or her stereoscopic (sensory) dead space. An observer with eso fixation disparity would keep the ground on the far or uncrossed disparity edge of this dead space, and one with eso fixation disparity would keep the ground on the nearer or crossed disparity edge of this dead space. The average offset from actual ground level was a task-specific manifestation of fixation disparity and the stereoscopic sensitivity of the observer, which, as we have seen, are dependent. From the view of the mapmakers, this offset was quite a nuisance because the final map was composed from contributions from many different stereogrammetrists, each of whom was known to have different habitual offsets. Careful record was kept of each person's offset from many points and used to compute an average offset (called a personal index) for that worker. All of their work was adjusted by that personal index so that it would fit into the work of their fellow stereogrammetrists. Obviously, the offset was much less of a problem if it was small and constant for each contributor. Vision therapy was shown to reduce that offset (p = 0.03) and improve reliability (p = 0.05) in the experimental as compared with the control group. Thus, the two experiments by Saladin and coworkers clearly demonstrated that optimizing supporting visual skills will improve stereoscopic test (Howard-Dolman) results and task (profiling) performance and will also reduce fixation disparity. The Saladin et al. experiment also shows, very compellingly, that fixation disparity in a real-life task situation and over a period of time has a direct connection to stereoscopic ability.

FUTURE DIRECTIONS

There are several areas in need of applied research in stereopsis. In general, needs are developing in the study of virtual reality and the effect of age on binocularity. The development of computer technology is driving one, and the aging of the population along with that same technological rampage is driving the other. More specifically, I have four additional areas that I see as needs.

The first area concerns the "build-up" or enhancement of the stereoscopic percept with time. Many, if not most, people get the strong impression that the depth in a stereoscopic presentation increases with observation time. Is this a strictly perceptual phenomenon or can it be linked to a physically measurable change? We know that fixation disparity decreases with time as the slow vergence adaptation mechanism shoulders more of the interocular demand. Is the observer seeing the effect of successively less fixation disparity? If so, fixation disparity can be monitored as the percept builds. On the other hand, it could be that the depth hierarchy process is progressively accentuating the parallaxic cue. Maybe part of the reason is that more clinical and some undiagnosed abnormality is present; could it be a slow reduction in the amount of suppression or some similar change toward more normal sensory fusion? On the other hand, is it a sharpening of retinal correspondence or a recruitment of binocular cells so more and more are devoted to the task at hand. Spirin et al. used rhesus monkeys and a matching-to-sample task and noted that "Behavioral and neurophysiological results indicate that increasing the amount of effort needed to perform a perceptual task can affect how information is processed within the visual system." The same thing might happen with processing for retinal disparity; the absolute threshold might not be lowered but the quality and consistency of the percept might be enhanced. Could it be, as van Ee and Erkelens suggested, that "integration of either non-binocular cues or extra disparity signals (for instance vertical disparity) enabled a build-up of the percept. Might there be some observable physical change, perhaps measurable with thermal, magnetic resonance, or some similar imaging technique? In still another way to investigate the perceptual build-up for local stereopsis, one might look for similar processes from that which is known about the time taken to see a random-dot stereogram.

A second area deserving research attention looks directly at the perceptual aspects of stereopsis. Can the stereoscopic percept be somehow enhanced through feedback? Instructors in photogrammetry coach their students verbally. Photogrammetrists have used dual binocular stereoscopes on one stereoscopic model in much the same way we optometrists have used teaching tubes on slit lamps. I developed a task on a stereoplotter that worked like a Perceptuo Motor Pen used for fixation/amblyopia therapy. Instead of the observer hearing a sound when he drifted off the line on a piece of paper, the observer heard a sound if he lost the floating point get too high or too low off the ground level in the photogrammetric model. The sound varied in pitch in the person was too high or too low. Exactly what is being trained in these instances? Are we retraining the motor or sensory situation like in the last paragraph? Is this a perceptual sharpening? Could the same techniques be applied in optometry schools to enable the students to better see the stereoscopic depth in binocular indirect opthalmoscopes, slit lamps, and fundus lenses? There are some very practical reasons to pursue this research.

The third area concerns past research. Much of the research in stereopsis has used subjects with presumably normal oculomotor systems, so determined very simplisticly, often with a Telebinocular/Visual Skills (Keystone View Company, Reno, Nevada) screening. The knowledge so gained has been generalized to be accepted normal physiology. Close reading of many of the papers makes it clear that much of that normal physiology could be explained by the person-specific individual binocular eccentricities of the subjects, i.e., idiosyncratic receiver variables. A need exists for a careful review of the basic papers in binocular vision to see if the

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There are two dead spaces to consider, one sensory (stereopsis) and one motor (fixation disparity). Both are in operation here. The arguments that Huang puts forth to support the necessity of a motor dead space with a directional effect apply to the necessity for a sensory dead space and a directional offset.

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The Perceptuo Motor Pen is a product of Wayne Engineering Products. It is available through Vision Training Products, 4106 N. Homo St., Mishawaka, Indiana 46545.
findings warrant generalization in light of our present understanding of the pathophysiology of the ocularmotor and binocular systems.

Finally, the fourth area is a plea for a specific area of research as it is a plea to channel some of our efforts into applying the knowledge that already exists. From’s 1, 41 addressed the same problem from a larger perspective in a discussion on basic and applied research. As I stated at the beginning of this article, a mountain of information exists about stereopsis: the problem is that application of that knowledge lags. Funding/support is available for basic research and the workers—vision scientists—are many. As a result, the mountain is growing. Funding/support is needed for those who would apply this knowledge for the improvement of patient care.

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204 Stereopsis Performance—Saladin
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Stereoscopic Performance—Saladin 205


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