

**Monocular and Binocular Vision With
Asymmetrical Visual Pathology**

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ABSTRACT

Monocular and Binocular Vision with
Asymmetrical Visual Pathology

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The effects of asymmetrical visual pathology on binocular vision were examined in four diagnostic categories: amblyopia, cataract, optic neuritis, and macular degeneration. Suppression, summation, and averaging, as adaptive binocular mechanisms, were assessed in six experiments which evaluated standard visual acuity, contrast sensitivity, temporal resolution, stereopsis, colour vision, and reading performance. It was expected that binocular performance on all the tasks would be impaired in all of the diagnostic groups except amblyopia.

Five observers in each diagnostic category and five normally sighted subjects participated in this study and their monocular and binocular vision was assessed in each of the six experiments. The results of the acuity test showed that all observers were able to suppress the weaker eye during binocular viewing. Equivalent results were obtained on the reading test and led to the conclusion that targets with high contrast and high spatial frequency allowed suppression to occur during binocular viewing. The tests of spatial and temporal resolution indicated a tendency for averaging of monocular inputs to occur when macular degeneration was the cause of asymmetrical vision. The other diagnostic groups tended to use suppression or summation on these tasks. Only

those observers with optic neuritis and the normally sighted individuals showed evidence of good stereoacuity which requires cooperative interaction of the two eyes. Finally, tests of colour vision produced data that suggested the use of averaging by the groups with optic neuritis and macular degeneration. The results of the six experiments suggest that the choice of binocular strategy may depend primarily on the locus of the pathology and secondly on the visual task.

An additional aim of this study was to examine currently prescribed occluders for patients with asymmetrical visual impairment. Opaque occluders and frosted lenses were used in the monocular viewing conditions in three of the experiments. The data indicated no significant difference in visual performance when either occluder was used.

The results of this study indicate that the binocular processing of visual information may be contingent on the locus of pathology as well as the type of task performed. The findings are discussed in the context of current theoretical formulations.

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MONOCULAR AND BINOCULAR VISION
WITH ASYMMETRICAL VISUAL PATHOLOGY

Statement of the Problem

Research dealing with various forms of visual pathology has examined the changes which are observable in monocular and binocular vision. Since many diseases which affect the visual system are bilateral and symmetrical, much of the present knowledge is based on these disorders. Although many of the questions that have been addressed by researchers in this area stem from pragmatic considerations and thus are of practical importance, they do not allow much novel speculation or deduction concerning binocular visual processing.

Unilateral or asymmetrical pathology provides an opportunity to study the adaptive capacity of the visual system. This adverse effect on binocular vision, that is caused by unequal responding of the two eyes, has been of great interest to medical investigators. For example, the case of amblyopia has led to much speculation concerning the affected perceptual processes. The currently accepted theory states that the brain suppresses or ignores the information transmitted by an eye which is weaker due to a strabismic deviation or an uncorrected asymmetrical refractive error. The latter condition, which is called anisometropia, is particularly interesting since there is no organic defect in the weak eye (Vaughan & Asbury, 1977). The study of amblyopia has provided much information about the underlying processes of binocular vision. However, more extensive investigation of asymmetrical visual pathology is necessary in order to further the understanding of

these mechanisms.

Three major issues must be addressed if one is to comprehend the response of the perceptual system to pathologic or traumatic intervention. The first issue concerns the process which is responsible for the final perception of a visual stimulus when there exists a significant difference between the input from the two eyes, and when this difference is due to more than geometrical, optical factors. In this context it has been proposed that "situations where the visual input is not only different in each eye but also of poor quality would tend to cause a reliance upon the physiologically superior eye" (Porac, 1974, p. 71). In other words, through suppression or inhibition of some visual input, the information being channeled from the weaker eye becomes unavailable to conscious perception. Perry and Childers (1972) had previously noted a dominance-suppression mechanism to be operating in both amblyopes and normally sighted individuals. On the other hand, it has been suggested that, for certain types of unequal visual input, an averaging process based on the information from the two optic channels is activated. (Hurvich & Jameson, 1967; Levelt, 1965; Overbury & Bross, 1978). Finally, there are the classical fusion theories of Kepler, Helmholtz, and Fechner which suggest a summation of input (Kaufman, 1974).

The second issue, which may be independent of whether suppression, averaging, or summation occurs, concerns the locus of impairment which would engage the appropriate process. Four major locations of unilateral or asymmetrical disorders are the ocular media, the retina, the optic nerve, and central sites. It is important to ascertain whether a process such as suppression, fusion, or averaging would be

activated with an early interference in visual input that could be caused by a media opacity or whether retinal or optic nerve components would be necessarily involved before the process could be observed.

The final issue deals with the prescription of spectacles in cases of unilateral and asymmetrical visual input which presently is not based on any outlined criteria. Specifically, whereas the practitioner carefully prescribes the optimal correction for the "stronger" eye, the fate of the "weaker" eye needs investigation, due to its potential impact on binocular processing. Currently, three alternatives are available to the practitioner. A clear "balance lens" which contains a refractive correction equal to that for the stronger eye may be placed in front of the weaker eye. This procedure, naturally, allows complete visual input to the eye, regardless of the distortion which the individual may experience. Secondly, one can place a "frosted lens" in front of the weaker eye, this being the clinical equivalent of a monocular Ganzfeld which allows unpatterned light to enter the eye. Finally, one can suggest that an opaque occluder be worn over the eye, thus allowing the stronger eye to work alone. The importance of this issue lies in the possibility that any or all of these procedures may be affecting a natural adaptive process such as averaging, suppression, or fusion. On the other hand, depending on which pathological condition evokes any of these natural processes, the above mentioned clinical procedures may be necessary in specific diagnostic categories. In either case, a comparative investigation of the three techniques is necessary to ensure optimal visual functioning.

This study was based on the assumption that, in the face of asymmet-

rical visual input; the mechanism employed to efficiently process the available information may be determined by the locus of impairment. Although one process may be objectively superior, it may be the case that with certain pathologies the system is incapable of employing an optimal strategy or that a mature visual system is not flexible enough to shift from summation or averaging to suppression. Three major issues on this topic are examined in this study: the first issue is directed at the determination of the adaptive strategies which are employed with the occurrence of a major asymmetrical pathology. The second issue dealt with the localization of impairment which evokes a particular adaptive strategy, and the final issue addresses the problem of clinical management in order to optimize the quality of the visual information which is available to the impaired system.

Introduction

The examination of the processes underlying binocular vision has a long and diverse history. Braunstein (1976) suggests that many current studies have their roots in the writings of philosophers of the seventeenth and early eighteenth centuries. Julesz (1971) mentions that in the nineteenth century Helmholtz "coined the term "cyclopean eye", a hypothetical construct which Hering used to explain identical binocular directions. The current consensus among workers in the area of perception is to accept some form of "fusion" theory to explain binocular vision (Kaufman, 1974). Moreover, ophthalmologists now assume that under normal viewing conditions, when the two eyes can focus on one object and send highly similar messages to the cortex, the system fuses the two images into one (Newell, 1978; Vaughan & Asbury, 1977). When unequal inputs are received, however, the brain suppresses the information from the weaker channel. This conclusion has been based on years of ophthalmological and perceptual research dealing with the problem of amblyopia.

The difficulty with the current state of affairs rests on the inability to generalize these results beyond the case of amblyopia. Other unilateral or asymmetrical visual disorders are quite common and have been studied for diverse clinical reasons but their effect on binocular perception has yet to be empirically examined. One could argue that sufficient information in this regard has been accumulated from the amblyopia research, but this contention is weakened by two confounding variables. The first problem, that of generalization, is caused by age differences between the typical onset of amblyopia in

contrast to other asymmetrical visual pathology. It may well be that the visual system in the developmental stage is capable of adopting a suppression strategy to deal with unequal input from the eyes but that it loses this adaptive capability when it reaches maturity or when degeneration begins in the later years. The second confounding variable arises from the onset and duration patterns of different pathologies. Amblyopes, who have been studied extensively both during the developmental and mature stages, exhibit long-term and typically permanent difficulties. Here too, one can speculate that even if a mature visual system maintains its adaptive capacities, these may require an extensive time period in order to become noticeably functional.

An additional problem that requires investigation concerns the locus of pathology within the visual system. Since no definitive data exist on asymmetrical disorders, other than amblyopia, there is no evidence that only one adaptive mechanism is employed regardless of the type of disorder. For instance, if visual input is degraded by a unilateral opacity of the ocular media, the binocular processing of that information may differ from the situation where retinal or optic nerve damage has occurred. Alternatively, the choice of strategy utilized by the visual system may be determined not by the locus of pathology but, rather, by the type of task which must be performed. Thus, colour sorting, temporal resolution of flashing light, and reading may not all require the same adaptive process under conditions of asymmetrical visual input.

Regardless of the outcome, the investigation of asymmetrical visual pathology and its effect on binocular vision holds potential benefit for

both theoretical and clinical advancement. From a purely investigative perspective, binocular vision is still not completely understood although it has been extensively studied for over a century. By examining binocular vision that has been pathologically undermined, it may be possible to shed some light on the underlying mechanisms that contribute to this complex process. From a clinical perspective a reasonable assumption is that any unilateral insult to the visual system is highly detrimental and that, when it occurs in later years, it destroys any possibility of acceptable binocular interaction. This assumption is based on numerous cases of patients who either report that they consciously appreciate a decrease in their vision when both eyes are open or who unconsciously favor one eye during clinical testing to the point of closing or covering the pathological eye. Empirical investigation may allow categorization of individuals who may or may not be able to efficiently use both eyes simultaneously. Furthermore, if only the strong eye is functional for a viewer, it is imperative to ascertain the optimal prescription strategy that would allow that eye to work effectively.

During the past century many theories have arisen to explain the complementary functioning of the eyes (Helmholtz, 1867/1925; Hering, 1868/1977; Sherrington, 1904/05; Sperling, 1970). Concurrently, a variety of experiments have been conducted in order to support or dispute the theoretical formulations. These have ranged from McDougall's (1904/05) demonstration of "nerve-path competition" to explain Fechner's paradox to a recent intriguing research series conducted by Blake and his colleagues which examines the underlying mechanisms of binocular

perception in the normal visual system and which has supported the binocular summation viewpoint (Blake, Martens, Garrett, & Westendorf, 1980; Blake & Rush, 1980; Blake, Westendorf, & Overton, 1980).

Additionally, a great amount of clinical research has been devoted to the investigation of the degraded visual input caused by visual pathology. As mentioned above, this work has emphasized bilateral involvement, however, the results of these studies provide a basis from which a model of asymmetrically impaired binocular vision may be constructed. The clinical studies, apart from elucidating the visual experiences of affected patients, also emphasize the necessity of valid and reliable measures of visual functioning.

In the past twenty years, there has developed a slow merger between the experimental and clinical investigations of binocular vision. Perhaps the best example of this process is evident in research that have been directed at the study of ocular dominance in normally sighted individuals and the comparison of these results with those of research concerning amblyopia (Crovitz, 1961; Porac, 1974; Porac & Coren, 1975; Shapero, 1971; Sokol, 1976). Another instance of combined effort is found in the endeavour to improve upon the standard evaluation techniques used to measure the level of visual functioning through the use of psychophysical techniques, and in the clinical attempt to find reliable diagnostic tools for the assessment of many forms of physical impairment (Brussel, White, Bross, Mustillo, & Borenstein, 1981/82; Galvin, Regan, & Heron, 1976; Regan, Silver, & Murray, 1977; Sjostrand, 1979; Wanger & Nilsson, 1978).

Past research from both the experimental and clinical fields must,

then, be considered if one is to adequately explain the possibility of adaptation to asymmetrically impaired vision since this provides a base of knowledge concerning the capabilities of the visual system before the onset of the pathology. Secondly, it is important to take into account the functional impact of differentially located damage in the visual system, and finally, it is necessary to test different levels of visual functioning before drawing conclusions about adaptive binocular processes.

Theoretical Formulations

The classical theories of binocular vision state that binocular perception is a result of the fusion of the monocular signals. Helmholtz (1867/1925) believed that "...the explanation of single binocular vision is that, when the eyes are used in the natural, normal way, the object at which we are gazing is imaged in the fovea centralis of each eye at the same time; and we know, by touching the object that there is really only one thing there" (p. 404). He further asserted that each eye could be moved independently and that the two eyes came to be used together only by habit in the interest of single clear vision. Hering (1868/1977), on the other hand, believed that "the two eyes should not ordinarily be seen as two separate organs steered by the same commands, but so to say as two halves of a single organ" (p. 16). Although both men believed that fusion of the monocular inputs produced the binocular percept, Hering formulated a law which stated that the two eyes share a common innervation, whereas Helmholtz maintained that fusion is purely a psychic act and is not produced by means of organic

mechanisms. Sherrington (1904-05) also concluded that "...each unocular mechanism develops independently a sensual image of considerable completeness. The singleness of binocular perception results from the combining of these elaborated unocular sensations" (p. 60).

The early theorists acknowledged the existence of the suppression mechanism only when the input to the eyes was greatly different, such as is the case in strabismus. However, fairly recent formulations of a suppression theory emphasize that, even in a normal visual system, the single percept results from the inhibition of one monocular signal by its partner (Burian & Boeder, 1955; Fox & McIntyre, 1967; Perry & Childers, 1972; Porac, 1974; Porac & Coren, 1975). The overwhelming tendency, it seems, is still to explain normal binocular functioning in terms of fusion and to relegate suppression to situations where retinal stimuli are not in correspondence (Schapero, 1971; Sperling, 1970). Porac and Coren (1976) summarize current speculation by noting that "sensory dominance represented a condition in which there is a sustained discrepancy in the input to the two eyes. The inputs are non-fusable and alternate in consciousness. Such stimulus conditions are seldom found in normal viewing but seem confined to pathological conditions such as strabismus or anisometropia. It may be the case that this mechanism does not express itself until the binocular coordinations are in a state of malfunction, and thus it may not be important for visually normal observers" (p. 885).

Normal Binocular Processing

Studies of binocular vision have been traditionally devoted to attempts at explaining ocular dominance, cooperative mechanisms, central

and peripheral contributions to single vision, stereopsis, and binocular rivalry. Researchers have also examined a variety of interesting binocular phenomena ranging from Fechner's paradox to random-dot stereograms.

Brightness One aspect of binocular vision which has intrigued investigators of perception is that of Fechner's brightness paradox. It refers to the visual experience which occurs when the intensity of a luminous target is markedly different for the two eyes (Curtis & Rule, 1980). For instance, if a white area is first presented to one eye and then a grey area is presented to the other eye, the perception of the binocular brightness is not increased by the additional grey stimulus but is diminished by it. Attempts to explain this paradox have been numerous. McDougall (1904-05) suggested that "...the fatigue of the cortical paths, induced by the preliminary fixation of the brighter field and the consequent increase of the resistance of those paths, enables the paths excited by the darker field to draw off a part of the energy of the excitation process from the former, diminishing the brightness of the sensation-elements contributed by them" (p. 115).

More recently, it has been suggested that, contrary to Fechner's expectation that summation would occur, the paradox is explainable in terms of an averaging process (Hurvich & Jameson, 1967; Levelt, 1965, 1966). Levelt, for instance, believes that the eyes work in a complementary fashion in binocular brightness perception. Specifically, one eye's increased contribution causes a decrease on the part of the other eye, particularly in the absence of contours associated with a given stimulus. This compromise, therefore, causes the brightness of

the dichoptically viewed target, to fall between the brightness of the two monocular views.

Blake, Breitmeyer and Green (1980) conclude, on the basis of earlier research, that the luminance of a homogeneous field viewed by one eye can influence the performance of the contralateral eye. They cite studies with human amblyopes that have demonstrated an enhancement of visual acuity in the amblyopic eye by luminance reduction of the other eye's visual field. Nevertheless, current research demonstrates that this aspect of binocular vision is still not completely understood (Curtis & Rule, 1980). It seems that a nonmonotonic relation may exist in perceived binocular brightness. At low intensities, the data indicate possible averaging while there is evidence for partial summation at the higher luminances. Furthermore, the locus of this type of binocular interaction has yet to be identified.

Stereopsis Another topic of research concerns the interpretation of binocular disparity. Investigators have examined the perception of depth in a textureless optical array (Natsoulas, 1963), with stimuli that present no familiarity cues (Julesz, 1964, 1971), and in situations of stimulus uncertainty (Staller, Lappin, & Fox, 1980).

The research of Julesz, in particular, has provided a great deal of information regarding the underlying mechanisms of stereopsis. He determined, for instance, that even with computer generated random-dot images, the quality of stereopsis is excellent. In other words, the time required for the perception, the stability of the fused image, and the amount of retinal rivalry compares favorably with the experiences produced by familiar stimuli. Julesz claims that the random-dot images

may even be easier to perceive in depth.

From his results, Julesz has inferred that stereopsis is the result of central visual processing. He bases this inference on the observation that, although the random-dot patterns are vastly different when viewed monocularly, they can nevertheless be perceived in depth binocularly. This suggests that the processing of the information occurs after binocular combination of the images has occurred. Julesz further claims that this assertion is in agreement with the neurophysiological work of Hubel and Wiesel (1962).

The development of stereoacuity has also been examined in order to explain the underlying mechanisms that are responsible for the process. Romano, Romano and Puklin (1975) conclude from their review of the literature that there is little agreement about the age at which human stereopsis becomes fully established. The suggestions that have been offered range from three to nine years of age. There has also been some evidence that a decrease in stereoacuity may begin in the fifth decade of life.

Recent studies of stereopsis have attempted to elucidate the process by studying the effects produced by intentional disruption of the system. Staller et al. (1980), for example, increased their subjects' uncertainty about the visual stimuli which they were viewing by decreasing the discriminability of the contours and by allowing a multiple-choice response in identifying the stimuli. Letters of the alphabet were presented either as random-dot stereograms or as two-dimensional physical contours. The results suggested that the uncertainty about the shape of the target did not impair global stereoscopic

resolution.

Another team of researchers has examined the effects of REM deprivation and awake-state visual deprivation on binocular depth perception (Herman, Roffwarg, Rosenmann, & Tauber, 1980). They hypothesized that REM sleep activity in the visuomotor pathways and dream imagery constitute a replication of perceptual aspects of normal binocular viewing. Therefore, they expected that these processes would prevent impairment of stereoacuity due to disuse which would be observed after awake-state visual deprivation. Their results, in fact, showed significantly better stereoacuity following REM sleep deprivation than following a normal night of sleep. Furthermore, they found that stereoacuity may be significantly impaired by monocular as opposed to binocular patching in the awake state. They propose that the reason for this difference is that "...single eye vision causes nonsymmetrical activation of visual input to neural centers regulating disparity detection, whereas input is limited but still symmetrical with both eyes patched. This situation apparently does not disrupt the normal balance of binocular innervation." They go on to postulate that "a monocular patch situation (misuse of the visual system) is much more disturbing to stereoacuity than is binocular patching (disuse)" (p. 241).

In the present context, these results are particularly interesting in relation to the examination of the clinical treatment issue. Taken together with the developmental and neurophysiological data, they provide a basis for the study of the effects of asymmetrical visual pathology on binocular processes such as stereopsis. Additionally, an examination of a variety of visual tasks may allow for enlargement on

the speculations by Julesz concerning the levels of the visual system which are involved in any given perceptual task.

Single Vision Apart from specific topics like the brightness paradox and stereopsis, there has always been a great deal of interest in the question of how two visual images are interpreted as a single percept. These investigations have selectively taken issue with the classic theories of binocular perception and have generated data to explain "cyclopean vision".

One of the controversies has centered around the possibility that suppression may not be limited to amblyopic adjustment but may, in fact, be employed by a healthy visual system to achieve single vision. One of the findings which have led to these investigations concerned a positive correlation between the dominant eye which is preferred for sighting and the differential acuities of the two eyes (Crovitz, 1961). Fox and McIntyre (1967) have tested the assumption that suppression of one eye's input is contingent upon the complexity of the visual targets. In other words, they believe that suppression would occur only when the signal strength in the monocular channels exceeds some specified level. Their results supported this assumption and demonstrated that stimulation of one eye by a complex target initiates a contralateral inhibitory suppression process.

Blake et al. (1980) studied suppression in situations which induced binocular rivalry. Basically, they wished to determine whether the visual stimulus or the eye itself was being suppressed in these situations. Their psychophysical data indicate that rivalry involves competition between the eyes but that this competition does not

interfere with all binocular activity. In fact, they were able to demonstrate that despite the suppression imposed on it, the suppressed eye can still contribute to stereopsis. They suggest that rivalry and stereopsis are mediated by separate, parallel mechanisms, one essentially monocular and the other binocular. They further postulate that, in order to accomplish monocular suppression, it would be economical to locate the responsible mechanism at an early processing stage, prior to a point of inextricable combination. If this were the case, it would explain why "the suppressed eye seems to suffer a general reduction in sensitivity to all visual information while its dominating partner enjoys access to higher visual centers" (Blake et al., 1980, p. 230).

An equivalent amount of evidence exists to support the contention that the underlying process of binocular "single vision" is a summation of input from the two eyes. A great deal of data has been accumulated to identify the visual tasks which evoke a summation process. Harwerth, Smith and Levi (1980) found from their review of the literature that binocular contrast thresholds for sinusoidal gratings are reported to be generally lower than the monocular thresholds. Data obtained by Schmeisser and Dawson (1982), who used simple gratings to study visual evoked potentials, showed a small, though not statistically significant binocular versus monocular enhancement. However, there have been studies dealing with stereo-blind humans which showed that binocular summation was absent for threshold stimuli (Lema & Blake, 1977). Amblyopes, who do not have any appreciable stereoacuity, are also incapable of demonstrating binocular summation at both threshold and

suprathreshold levels. By using reaction time performance in a contrast detection task, Harwerth et al. (1980) were able to show binocular summation in normally sighted subjects for contrast levels near threshold. They obtained considerable variation in performance with contrast levels above threshold.

Blake and his colleagues have also examined the process of binocular summation and have acknowledged that, on a multitude of visual tasks that involve threshold measures, individuals benefit from using both eyes simultaneously rather than by relying on monocular input (Blake & Rush, 1980; Blake, Martens, & DiGianfilippo, 1980; Blake, Martens, Garrett, & Westendorf, 1980). Using measures of reaction time, these researchers have noted that the improvement of binocular performance is on the order of five per cent, compared to monocular performance. Thus, they find that, for normal observers, binocular reaction times are consistently faster than monocular ones and, furthermore, they state that the superiority of the two eyes over one exceed that predicted by probability summation (Lema & Blake, 1977). These authors also acknowledge that this pattern breaks down in amblyopes and stereoblind individuals either showing equivalence of monocular and binocular performance or, in some cases, poorer binocular performance. They conclude that binocular summation must reflect neural interaction between the eyes.

These experimental results naturally lead to speculation about the effects of other visual pathologies on "cyclopean vision." Determining whether summation capacity is completely eliminated in the presence of a unilateral impairment, for instance, would enhance the knowledge we have

about binocular vision. Similarly, the demonstration of a strong suppression mechanisms may also allow for this type of progress.

Visual Pathology

Few experimental studies have examined the effects of unilateral or asymmetrical visual pathology on the binocular processes. In 1966, Goldstein, Clahane and Sanfilippo alluded to the ample evidence which suggests that the peripheral visual fields significantly affect the binocular relationship. They also suggested, on the basis of past research, that peripheral stimuli could disrupt central fusion and supported their argument by demonstrating such disruption in patients with retinitis pigmentosa, which is usually characterized by extensive loss of peripheral vision. Another interesting study has demonstrated "binocular cooperation" after traumatic bi-temporal hemianopsia (Fisher, Jampolsky, & Flom, 1968). The presence of this visual problem with macular splitting and the loss of function of the decussating optic nerve fibers in the chiasm, causes each occipital cortical area to receive information from only the ipsilateral corresponding retinal area. This situation makes it impossible for a single spatial object to be perceived binocularly in a single visual direction. The results of the study showed that these patients could still achieve binocular single vision while being unable to appreciate stereopsis.

The existing problem in the clinical literature pertains to the almost exclusive concentration on symmetrical binocular impairment. While the present study examines asymmetrical visual pathology in its relation to binocular vision, much of the clinical literature is rele-

vant to its rationale.

Amblyopia By clinical definition, amblyopia is a condition in which there is a unilateral or bilateral decrease in visual acuity caused by abnormal binocular interaction which cannot be fully attributed to organic ocular abnormalities (Levi & Harwerth, 1980; Kaufman, 1974; Newell, 1978; Schapero, 1971). A considerable amount of evidence exists to demonstrate that, in addition to reducing visual acuity, amblyopia reduces contrast sensitivity, light flicker, and stereopsis (Manny & Levi, 1982a, b; Newell, 1978; Wesson & Loop, 1982). Recent studies have also employed the visual evoked response (VER) technique to study binocular vision which has been impaired by amblyopia (Lennerstrand, 1978; Wagner & Nilsson, 1978). It has been demonstrated in these experiments that the response of the dominant eye is the same under both monocular and binocular viewing conditions, which shows the lack of binocular interaction in stereoblind amblyopes.

Levi and Harwerth (1980) have reviewed numerous studies which have demonstrated that dark adaptation and scotopic as well as photopic luminosity in persons with strabismic amblyopia are similar in the amblyopic and non-amblyopic eye. They conclude, on the basis of this evidence, that the visual receptors function normally in the amblyopic eye, and Porac (1974) states that "...there is a great deal of evidence to support the view that the primary site of physiological involvement in amblyopia ex anopsia is at the central levels of the visual system" (p. 45).

Since amblyopia is predominantly a childhood disorder, much research has been devoted to measuring its development and determining critical

periods during which permanent impairment could be prevented (Schapero, 1971). The time course for the development of amblyopia has also been examined in infant monkeys that have experimentally induced strabismus (Boothe, 1980) or have had one eye chronically defocused by daily administration of a cycloplegic drug (Boothe, Kiorpes, & Hendrickson, 1982). The results demonstrate that acuity develops normally for approximately four weeks after the onset of esotropia, with a disruption of the normal pattern after that period. This supports the existence of critical time periods during which corrective surgery or training intervention could prevent permanent damage to the binocular mechanism. In the monkeys with induced anisometropia, decreased acuity remained stable for at least four months after the termination of treatment. However, no long-term results are available to determine whether the effects are permanent.

Recent studies have turned to more refined measures of visual functioning than the traditional Snellen techniques. For instance, Lennerstrand and Lundh (1980), among others, have discovered that contrast sensitivity is not only a good measure of visual capacity but is also capable of improvement even when no change is seen in standard visual acuity. In another study, dealing with adult amblyopes, Levi and Harwerth (1980) have demonstrated that amblyopic eyes show reduced contrast sensitivity over many spatial frequencies and stimulus durations. These authors also showed that the temporal integration time for high spatial frequencies is greatly increased in the amblyopic eye to almost twice the length of the non-amblyopic eye and, at all spatial frequencies, flicker detection sensitivity is reduced in the amblyopic

eye.

The interest in alternative assessment techniques for amblyopic visual functioning has led to some interesting findings. Freeman and Bradley (1980) used a two-alternative forced-choice method to show that functionally monocular individuals have significantly higher vernier acuities than those demonstrated by normally binocular individuals who are tested monocularly. The authors speculate that a form of neural recruitment may occur in amblyopes due to the disuse of one eye. Furthermore, Henson and Williams (1980) found that half of their strabismic subjects showed binocular depth thresholds which were significantly better than their monocular thresholds, when tested with the Howard-Dolman apparatus.

Colour vision has been studied in amblyopic subjects in order to assess the sensitivities of the cone mechanisms. Hansen (1979) used colour plates, the Farnsworth D-15 test, and static perimetry during chromatic adaptation to evaluate colour vision in both strabismic and anisometric amblyopes. The results indicated a generalized depression of sensitivity in amblyopia. However, normal colour vision was found when amblyopes were tested binocularly.

In relation to the present study, research concerning amblyopia provides a good basis for inference about the underlying mechanisms of adaptation to asymmetrically distorted vision. For instance, there has been much speculation concerning the role of the photoreceptors in this disorder. Bedell (1980) has concluded that "anomalous retinal receptor orientation apparently does not contribute to decreased amblyopic eye visual acuities. Within these amblyopic eyes, one must apparently look

to a more proximal site or sites within the visual system for the seat of the amblyopic visual loss" (p. 58). It may be the case that testing the levels of monocular and binocular visual functioning in the face of other major asymmetrical disorders will produce evidence concerning the locus of the underlying mechanisms.

Cataract A cataract is defined as any opacity in the crystalline lens (Newell, 1978; Vaughan & Asbury, 1977). The main symptom of cataract formation is a gradual decrease of vision that is not concurrent with pain or eye inflammation.

Research in this area has been devoted to medical problems such as the documentation of causes, the surgery involved in cataract extraction, and the post-surgical care of the patient. Perceptual studies have centered on patients' reactions to visual restoration after cataract surgery (von Senden, 1932; Tanner, 1971; Valvo, 1971). Only recently have investigators started to examine visual functioning before cataract removal. For example, a recent report examined patients' preoperative use of vision and engagement in visually guided activities in order to predict the postoperative visual performance and spontaneously expressed satisfaction with their surgery (Murphy & Donderi, 1980). In addition to the finding that preoperative activity levels were positively correlated with post-operative success ratings, these researchers found that adaptation to the differential image magnification after surgery was not uniform across their sample. In another examination of adaptation to spectacles by monocular aphakics, it was found that at first all individuals are extremely conscious of diplopia but some are successful in adaptating to it after extensive training

(Lubkin & Linksz, 1977). However, this has been the extent of attention devoted to asymmetrically impaired binocularity.

Another area of interest has developed around the need for a better indicator of visual function than the standard acuity measures. As in studies of amblyopia, researchers have turned to measures of contrast sensitivity with cataract patients (Hess & Woo, 1978). They have found that although cataracts decrease the contrast sensitivity function, in comparison to a healthy eye, just as they decrease standard acuity measures, the examination of contrast sensitivity provides a better index of remaining functional vision. This psychophysical technique has also been employed in the evaluation of vision in infants with congenital cataracts (Jacobson, Mohindra, & Held, 1981). By use of the "preferential looking technique" (Dobson & Teller, 1978), which is based on the discovery that infants prefer patterned to unpatterned visual stimuli, acuity can be assessed at a much younger age than would otherwise be possible. To date, few studies have been undertaken to examine the effects of a unilateral cataract or of asymmetrically dense cataract formation on binocular vision. Since this ocular disorder is one of the most important peripheral impediments to vision, it allows for a comparison with amblyopic suppression which many have concluded to be a centrally mediated process. It is also a process which does not necessarily involve damage to higher visual centers and thus makes it possible to examine the effects of optical transmission difficulties without the added complication of neural damage in the visual system.

Retinal Disease The potential disorders of the retina are numerous both in their effects on visual function and in their etiology.

Additionally, macular changes are a prevalent cause of visual impairment among the elderly (Blondin & Kenya, 1981; Delanee & Oates, 1982; Sperduto & Seigel, 1980). The retina has no pain nerve fibers, so the main symptom of a retinal abnormality is painless visual disturbance (Newell, 1978; Vaughn & Asbury, 1977). The classical research in both clinical and basic vision research has centered around the five main techniques that measure the function of the sensory retina. These include visual acuity, dark adaptation, colour vision, central and peripheral visual fields, and, more recently, electroretinography.

In the past decade, much work has been devoted to delineating the capacity of the peripheral retina to process different types of visual information (Phelps, Remijan, & Blondeau, 1981). By examining both clinical and experimentally induced cases of central visual loss, researchers have considered modifications of standard measures so that the acuity of the peripheral retina can be determined more accurately (Anstis, 1974; Millodot & Lamont, 1974). They have also tested peripheral vision under tachistoscopic conditions (Pailhous, Chesnais, & Leplat, 1975) as well as in stabilized-image situations (Gerrits, 1978). Functional vision has been evaluated by examining the potential of reading without foveal stimulation (Rayner & Bertera, 1979). These studies have provided much useful information regarding the capacity of the peripheral retina to discriminate form in psychophysical tests as well as in reading.

Once more, as with other visual disorders, there has been a recognition of the need for accurate assessment techniques directed not only at the quantity of remaining vision but also at its quality

(Skalka, 1980). Visual evoked response acuity and contrast sensitivity have become the measures of choice since they are sensitive to changes in retinal function which are not detected by Snellen techniques (Marmor, 1981; Sjostrand, 1979). Both visual evoked potentials and contrast thresholds have been shown to be higher in diabetic patients than in normals (Ghafour, Foulds, Allan, & McClure, 1982; Yamazaki, Adachi-Usami, & Chiba, 1982). Interestingly, even those diabetics without retinopathy show increased spatial thresholds at high frequencies. This finding has led Ghafour and his coworkers to suggest that the functional deficits associated with these frequencies may precede ophthalmoscopically visible retinopathy or decreased Snellen acuities.

Visual impairment caused by retinitis pigmentosa has also been evaluated using contrast sensitivity tests (Hyvarinen, Rovamo, Laurinen, & Paltomaa, 1981; Lindberg, Fishman, Anderson, & Vasquez, 1981). Patients with moderate and severe forms of this disorder show contrast sensitivity changes in their central vision even though their Snellen acuities are normal or near normal. Skalka (1980) states that the contrast sensitivity task is superior to other measures as an early indicator of macular dysfunction, and it has also been used successfully to monitor recovery of macular function after surgery for retinal detachment (Anderson & Sjostrand, 1981). Furthermore, the recent use of laser-generated sinusoidal gratings has allowed investigators to separate contrast sensitivity losses due to optical factors or refractive error from those caused by retinal diseases such as central serous retinopathy and branch vein occlusion (Kayazawa, Yamamoto, &

Itoi, 1982).

Colour discrimination has also been assessed in patients suffering from diabetic retinopathy (Kinnear, Aspinall, & Lakowski, 1972; Vassiliou, Simonetos, & Kastrantas, 1976; Zwas, Weiss, & McKinnon, 1980) and senile macular degeneration (Bowman, 1980). These investigators have concluded that, in some retinal diseases, alterations in colour vision may precede changes in visual acuity and they have emphasized the importance of reliable measures of colour vision in patients with retinal problems.

The topic of unilateral or asymmetrical retinal disorders has been recently addressed by Yanko (1980). The investigation focused on a case study of diabetic retinopathy and the author concluded that clinical observations of asymmetric retinal involvement might be valuable in ascertaining the possible causal associations and the pathogenesis of diabetic retinopathy. However, in most studies, when monocular and binocular performances were compared, the retinal pathology has been symmetrical. Therefore, the similarity of monocular and binocular results obtained by diabetics on the Farnsworth-Munsell 100 Hue Test (Lakowski, Aspinall, & Kinnear, 1972/73) leads one to question whether asymmetrical pathology would cause a significant change in binocular performance. Theoretically this type of observation might provide a clue concerning the perceptual process which deals with unequal retinal stimulation caused by a disease process rather than a difference in external stimulation to the two eyes.

The examination of asymmetrical retinal disease without the further complications of media opacities, optic nerve damage, or central

dysfunctions may demonstrate whether the retinal receptors are involved in the activation of summation, suppression or averaging. Furthermore, since the visual tasks to be examined allow different processing levels to become activated, the distinction of visual abilities may be contingent on the physiological state of the retina.

Optic Neuritis Newell (1978) states that "inasmuch as the optic nerve is composed of axons of the ganglion cell layer that form the nerve fiber layer of the retina, optic nerve disease may cause many of the same symptoms as retinal disease" (p. 321). It follows, then, that the main symptom is loss of vision with pain occurring only in retrobulbar neuritis which occurs far enough behind the optic disk so that no early changes are visible by means of the ophthalmoscope (Vaughan & Asbury, 1977). Since the central retina provides approximately ninety percent of the fibers of the optic nerve, central scotomas are the most common visual field defect. Optic neuritis is a general term which may include inflammation, degeneration, or demyelination of the optic nerve, and numerous causes of optic neuritis have been discovered (Newell, 1978; Thiel, 1963; Vaughan & Asbury, 1977).

Since optic neuritis is often associated with demyelinating and inflammatory diseases, systemic infections, as well as nutritional and metabolic disturbances, it is understandable that the majority of medical research has concentrated on the treatment of the underlying causes of the visual disorder. Optic neuritis, especially retrobulbar neuritis, characterized by acute unilateral loss of vision with a tendency toward recovery, is a frequent initial symptom of multiple sclerosis (Vaughan & Asbury, 1977). This fact has precipitated a number of

studies by researchers in visual perception who have attempted to find diagnostic procedures which would detect the early stages of multiple sclerosis.

Testing procedures which have been used for early detection of optic nerve disorders and monitoring subtle visual changes have included visual fields assessed with a tangent screen (Patterson & Heron, 1980) interferometric acuity tests (Campos, Enoch, Fitzgerald, & Benedetto, 1980) and Pulfrich's pendulum (Rushton, 1975). Additionally, Regan and his coworkers have found that, following acute retrobulbar neuritis, a patient displays an abnormal double flash threshold (Galvin, Regan, & Heron, 1976; Regan, 1980). They concluded that this measure is a more sensitive indication of visual damage in this type of disease than standard clinical tests such as the critical flicker fusion frequency. Multi-flash campimetry is an extension of the double-flash technique and has also been successfully used to identify poor temporal resolution in multiple sclerosis patients (Brussell et al., 1981/82).

Yet again, contrast sensitivity has been shown to be a more indicative reflection of visual loss in optic neuritis when compared to conventional clinical tests (Regan, Raymond, Ginsburg, & Murray, 1981; Regan, Silver, & Murray, 1977; Regan, Whitlock, Murray, & Beverley, 1980) and its use has helped to substantiate symptoms reported by patients which do not correlate with conventional test results (Woo & Long, 1979). The importance of using sensitive measures is clear in this diagnostic category where early detection of a disease entity is valuable. Confirmation of the contrast sensitivity findings has been provided by Zimmern, Campbell, and Wilkinson (1979). These studies

reaffirm the aforementioned belief that the quality of visual input, which can be substantiated by psychophysical tests, is as important as the limited quantitative measure that one obtains by traditional objective tests. Among other tests, the critical flicker fusion frequency has been assessed in patients with demyelinating disease and has shown gross abnormalities (Daley, Swank, & Ellison, 1979), as has colour vision which decreases with greater residual optic nerve damage (Griffin & Wray, 1978).

Given the accumulated knowledge concerning diseases of the optic nerve, it is reasonable to assume that binocular vision is affected by unilateral ocular involvement. This is especially the case when one considers the detrimental effects on binocular depth perception with asymmetrical optic nerve damage (Overbury & Bross, 1978). The use of suppression, summation, or averaging during binocular viewing may be contingent on the locus of impairment. If one assumes that optic nerve disorders damage the binocular mechanism, the process may differ from the one used when the visual information is degraded by ocular media opacity.

Present Study

This study investigated three major issues, the first two being theoretically oriented and the third addressing the applicability of perceptual theories in the clinical setting. The first theoretical question concerned the choice of adaptive process to deal with asymmetrical visual input. Secondly, an attempt was made to identify the locus of visual pathology which initiates the appropriate adaptive

process. The final issue dealt with the formulation of clinical recommendations concerning monocular and binocular visual functioning.

The primary intention was to determine the underlying process of binocular vision in an asymmetrically impaired system. Much evidence has accrued for both the fusion and suppression theories as well as other versions of these two basic formulations. Much of the fusion theory is based on studies of stereoscopic depth perception, but it is also supported by claims that the eyes have complementary shares in the production of binocular brightness if the contours of monocular patterns are congruent (Levelt, 1966) and by evidence which indicates that stereoblind individuals show some capacity for binocular summation although this is only demonstrable with high spatial frequencies (Lema & Blake, 1977).

Alternatively, Porac (1974) argues that fusion theories continue to survive because they have maintained that stereopsis depends upon the fusion of two monocular images and "...argue that if binocular combination is the result of a suppressive process of any great magnitude, there would be great losses in the amount of depth information available to the binocular system" (p. 17). Porac believes that degraded visual input forces the binocular perceptual system to choose the fastest physiological channel which has the best ability to quickly resolve the patterned properties of the incoming stimulation. Although Porac's support for the suppression theory is based largely on amblyopic perception, previous research had shown the existence of suppressive mechanisms in normal visual systems, particularly when the perceiver is confronted with complex stimuli (Fox & McIntyre, 1967) or

when non-corresponding contours are presented to the two eyes (Levelt, 1966). Here too, it would be instructive to demonstrate whether suppression is evident with differentially located visual pathology. This would provide a great deal of information concerning the flexibility of the visual system in an abnormal situation.

Secondly, this study compared monocular and binocular vision in patients with four major categories of visual impairment in order to determine if the system reacts identically to all perceptually detrimental conditions or if it maintains a degree of flexibility in its adaptive capacity. The first category involved media opacity which substantially degrades the quality of visual input purely in the optical transmission. This impairment often leaves intact the retinal, post-retinal, and central mechanisms involved in visual information processing. If an adaptive process is elicited by this type of disorder, it will demonstrate that neural involvement is not necessary to disrupt the normal functioning of the binocular system. The second category concerned the effects caused by retinal damage when there are no media opacities nor any post-retinal complications. Thirdly, the transmission inefficiency caused by optic nerve disease was assessed without the further interference of optical, retinal, or cortical involvement. Finally, amblyopia was reassessed in order to provide a comparison between visual impairment with demonstrable ocular abnormalities and those assumed to be centrally mediated.

The final issue addressed in this study involved the clinical prescription of lenses or occluders for the weaker eye in asymmetrical visual disorders. Since patients often complain about interference if

their weaker eye has a clear lens in front of it, practitioners resort to suggesting that either a frosted lens or an opaque occluder should be used to cover the weak eye. These two conditions ultimately produce either a monocular Ganzfeld or total visual deprivation for the affected eye. Many experimental studies, such as those reviewed by Harper and Bross (1978), have shown differential effects of these two types of monocular occlusion. For instance, with opaque occlusion over one eye one observes a depression-enhancement phenomenon, where the temporal acuity of the non-occluded eye is decreased after several hours but then increases with prolonged deprivation. It is also notable that lack of patterning achieved by a monocular Ganzfeld does not produce this effect. Further investigation dealing with the four categories of visual impairment outlined in this study may provide a basis for clinical applications of these experimental results.

Specifically, if the patients' complaints indicate a lack of fusion or suppression and accurately reflect serious diplopia, one must consider whether blocking the visual input with a frosted spectacle lens is adequate. In such circumstances, it may be necessary to induce total suppression of the weaker eye by the use of an opaque occluder. On the other hand, if the binocular system is capable of adapting, albeit slowly, to asymmetrical input, one might be interfering with an adequate natural process by using anything but a clear lens in front of the weaker eye. It was necessary, therefore, to examine monocular as well as binocular performance on the tests of interest.

In order to address the three issues that have been outlined, this study is divided into six related experiments. These assessed standard

visual acuity, contrast sensitivity, temporal acuity, colour vision, stereopsis, and reading performance. The visually impaired subjects belonged to one of the four diagnostic categories: cataract, retinal disorders, optic nerve disease, or amblyopia. These individuals' performances were compared to those of normally sighted persons. Thus, in each experiment, there are five categories of visual status, including the normally sighted group. Moreover, each individual performed each task monocularly and binocularly.

Based on past research concerning amblyopic suppression and on clinical reports from patients with asymmetrical visual disorders, it was hypothesized that the visually impaired subjects in each diagnostic category would exhibit significantly impaired performance under the binocular viewing conditions as opposed to the monocular conditions using the stronger eye. It was also expected that experimentally induced suppression by complete occlusion of the weaker eye would aid subjects with asymmetrical impairment in their task performance, and that the extent of impairment would vary in the four diagnostic categories depending on the visual task.

Experiment 1

The first experiment determined the extent of visual loss present in each subject through the use of Snellen tests for both distance and near acuity. The measure is considered important in the present context since it is the one most frequently used by practitioners, notwithstanding the degree of criticism it has received in the scientific literature. Additionally, these measures will continue to be used in order to evaluate appreciation of fine detail (Leibowitz, Post, & Ginsburg, 1980) and must be studied in terms of fusion and suppression capabilities.

Method

Subjects Twenty-five subjects participated in this study, with five individuals in each of five diagnostic groups. These consisted of normally sighted individuals, amblyopes, those with unilateral or asymmetrical cataracts, maculopathy, or optic neuritis.¹ The mean age across groups was 39, with a range of 18 to 80. The normal subjects and one amblyope were students at Concordia University; all of the other participants were private patients of ophthalmologists at the Royal Victoria and the Montreal General Hospital. With the exception of the normal group, all participants had asymmetrical visual disorders with neither eye having a Snellen acuity lower than 20/200 (6/60). In every test, the participants wore standard corrective lenses if they had significant refractive error. None of the participants used low vision aids in any of the experiments.

Apparatus A standard projected Snellen chart was used to assess static distance acuities. Near-distance acuities were assessed using

standard reading cards which are also calibrated according to Snellen notation.

Procedure The subjects were asked to read the chart monocularly, with a black patch covering either the weaker or the stronger eye for the first two conditions. They also read the chart monocularly, with a frosted lens in front of the weaker eye and a fourth reading with binocular vision. These four conditions were randomly ordered across subjects, with the exception of the weaker eye not being tested first. The test was terminated when the subject identified fewer than 75 percent of the optotypes on any line of the chart. The subject's score was recorded in terms of the smallest visual angle which was discriminated on at least 75 percent of the trials.

In order to assess the subject's near acuity, both word and number charts were employed in order to minimize the probability of memorization. Once more, the subject's score reflected the smallest resolvable visual angle with a 75 percent accuracy rating. The two monocular conditions and the binocular condition remained identical to the distance acuity testing procedures. In both the near and distance tests, the subjects were given no feedback concerning their responses.

Results

The measure of distance acuity recorded in this experiment was the smallest visual angle in minutes resolved by the observer on 75% of the trials. This angle was determined by the reciprocal of the Snellen fraction. The means for each group in the four viewing conditions are shown in Table 1. The data indicate that, for distance acuity, the four

TABLE 1

Group Means of Distance Acuity

	Strong Eye Black Patch	Strong Eye Frosted Lens	Weak Eye	Binocular
Amblyopia	1.5	1.1	6.5	1.1
Cataract	1.7	1.5	7.2	1.5
Optic Neuritis	1.3	1.2	2.3	1.1
Macular Degeneration	2.4	3.5	10.0	3.5
Normals	.9	.9	1.0	.8

Group Means of Near Acuity

	Strong Eye Black Patch	Strong Eye Frosted Lens	Weak Eye	Binocular
Amblyopia	.4	.4	2.9	.4
Cataract	.4	1.1	2.3	.4
Optic Neuritis	.4	.5	.4	.4
Macular Degeneration	.7	.9	3.5	.7
Normals	.4	.4	.4	.4

viewing conditions produced scores in the optic neuritis group which were not significantly different. A repeated measures analysis of variance showed that there were significant differences in the amblyopes, $F(3,12) = 11.22$, $p < .01$, those patients with cataracts, $F(3,12) = 10.51$, $p < .01$, macular degeneration, $F(3,12) = 15.03$, $p < .01$, and in the normals, $F(3,12) = 3.78$, $p < .05$. Tukey's post hoc analysis revealed that the normal observers showed better acuity binocularly than with their non-dominant eye only but all other pair-wise comparisons showed no difference between viewing conditions. In the other three groups, the acuity of the weaker eye was always significantly lower than that measured in any other viewing conditions. All other pair-wise comparisons showed no significant differences. These findings suggest that for a high contrast distance task the input from the weaker eye is suppressed resulting in acuities equal to that of the stronger eye whether a frosted lens covers the weaker eye or binocular viewing is possible.

The results of the near acuity assessment show no difference being due to the viewing conditions in the cataract, optic neuritis, and normal groups. Individuals with macular degeneration and amblyopia exhibited near acuities which were significantly different across the four viewing conditions, $F(3,12) = 4.87$, $p < .05$; $F(3,12) = 3.80$, $p < .05$. In both groups the near acuity of the weaker eye was significantly lower than the other three acuity measures but there were no other differences among conditions. The means are shown in Table 1. Once more, the implication that suppression of the weaker eye takes place in binocular viewing for some patient populations is supported by

the data.

Discussion

The significant differences found between the weaker eye's acuities and those resulting from the other viewing conditions are not surprising. The outcome which requires further examination involves the lack of acuity difference among the other three conditions. The results demonstrate that distance acuity does not change appreciably when tested binocularly, with a frosted lens over the weaker eye, or with an opaque occluder over the weaker eye. One can conclude that the input from a weaker eye is suppressed regardless of the locus of pathology. It is crucial to recall that Snellen optotypes are not comprehensive measuring instruments of functional visual acuity (Hess & Woo, 1979; Lennerstrand & Lundh, 1980; Marmor, 1981; Regan et al., 1977) and, therefore, these results should not be generalized beyond the specific tests used in this experiment.

Experiment 2

As pointed out in the literature review, a current technique that has gained a degree of clinical acceptance is that of contrast sensitivity evaluation (Derefeldt, Lennerstrand, & Lundh, 1979; Dobson & Davison, 1980; Virsu & Rovano, 1979). It has been established as a measure that is discriminative in all the diagnostic categories which were examined herein and that, furthermore, it may be a good measure of binocular interaction.

Method

Subjects The same 25 subjects participated in this experiment.

Apparatus A PDP11/10 computer interfaced with Wavetek function generators allowed sine wave grating patterns to be produced on a large screen cathode ray tube (Hewlett Packard 1310A equipped with a P15 phosphor). The luminance of the display was measured with a Spectra Spot Meter.

Procedure The subjects sat at a distance of one meter from the CRT screen and viewed a square field that subtended a visual angle of 4 degrees. This distance was maintained by the use of a chin rest. The room was in darkness other than the stimulus display with mean luminance of 1.5 cd/m^2 . The sine wave grating pattern was observed against a dark background. Testing was done with a natural pupil and all observers wore any corrective lenses that they normally used for distance.

A psychophysical technique was utilized which required the observer to press a key to indicate that the square field no longer appeared homogeneous. Combinations of six spatial frequencies, ranging from 1 to

13.45 cpd in 0.75 octave unit steps, and four temporal frequencies of 0, 2, 8, and 32 cycles per second were presented. For a given flicker rate, the six spatial frequencies were tested in random order. For each spatial frequency, seven trials were presented, the first two of which were considered practice. Interspersed randomly among these were three additional trials during which no grating was presented. This allowed for an assessment of false alarm rates. A given block of trials ended when a grating was either detected or missed seven times. Each trial was preceded by a warning tone and began with the presentation of a subthreshold grating whose contrast increased in steps of 0.05 log units. An increase in contrast occurred every 250 milliseconds or after two complete flicker cycles whichever was longer.

Before a testing session began, a simple reaction time was assessed for a 2.8 cycle per degree (cpd) grating that was approximately 0.1 log unit above threshold. During the testing trials the computer used this reaction time in specifying the contrast of the grating that was displayed at an estimated time of detection rather than at the time at which the response key was pressed. Once more, no feedback was provided to the subjects.

The test was repeated three times: monocularly by each eye and binocularly. In both monocular conditions the non-viewing eye was covered by a black patch. The frosted lens was not used in this test. The three viewing conditions were randomized, except that the weaker eye was not tested first.

Results

An examination of the contrast sensitivity data reveals interesting differences between the groups. The normally sighted observers show no differences at any of the spatial frequencies between their dominant and non-dominant eye nor between the monocular and binocular results. These results are shown in Figure 1.

Figure 2, contains the results of the amblyopic group, where a repeated measures analysis of variance showed a statistically significant difference at 8.00 cpd, $F(2,8) = 5.47$, $p < .05$. Tukey's post hoc analysis showed that this difference existed between the amblyopic eye and the other two conditions. There is no difference between the non-amblyopic eye's contrast sensitivity and that of the two eyes together at any spatial frequency. One should note, however, that the amblyopic eye shows lowered sensitivity at low and middle frequencies.

The group with optic neuritis showed no significant differences among the three viewing conditions. In Figure 3, one notes a tendency for binocular viewing to exhibit better contrast sensitivity when compared to either eye alone, with the notable exception of the lowest spatial frequency. Here one sees no difference between the stronger eye, and the combination of the two eyes. The curve of the weaker eye's sensitivity is interesting in the lower spatial frequencies, where a decrease is observed compared to the other viewing conditions.

Observers with asymmetrical or unilateral cataracts also show a tendency for better binocular contrast sensitivity compared to the stronger eye although these differences do not attain statistical significance. The one exception exists at the lower spatial frequencies.

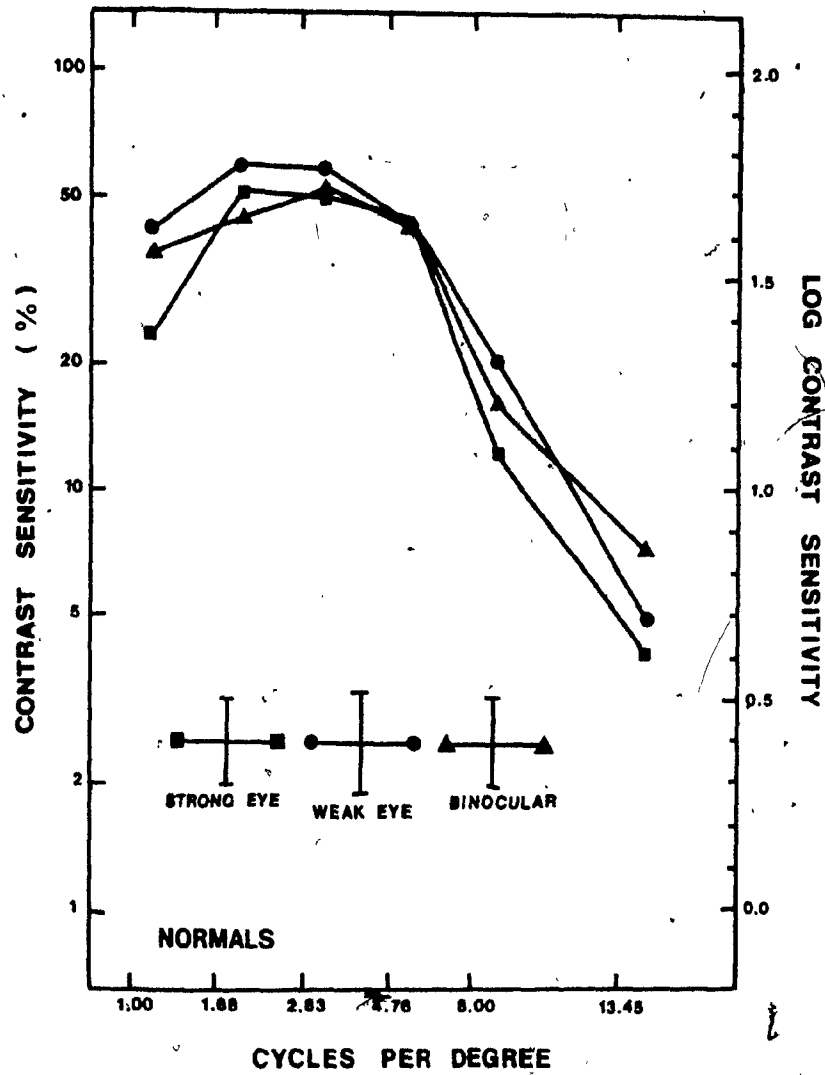


Figure 1. Contrast sensitivity as a function of spatial frequency in normals, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

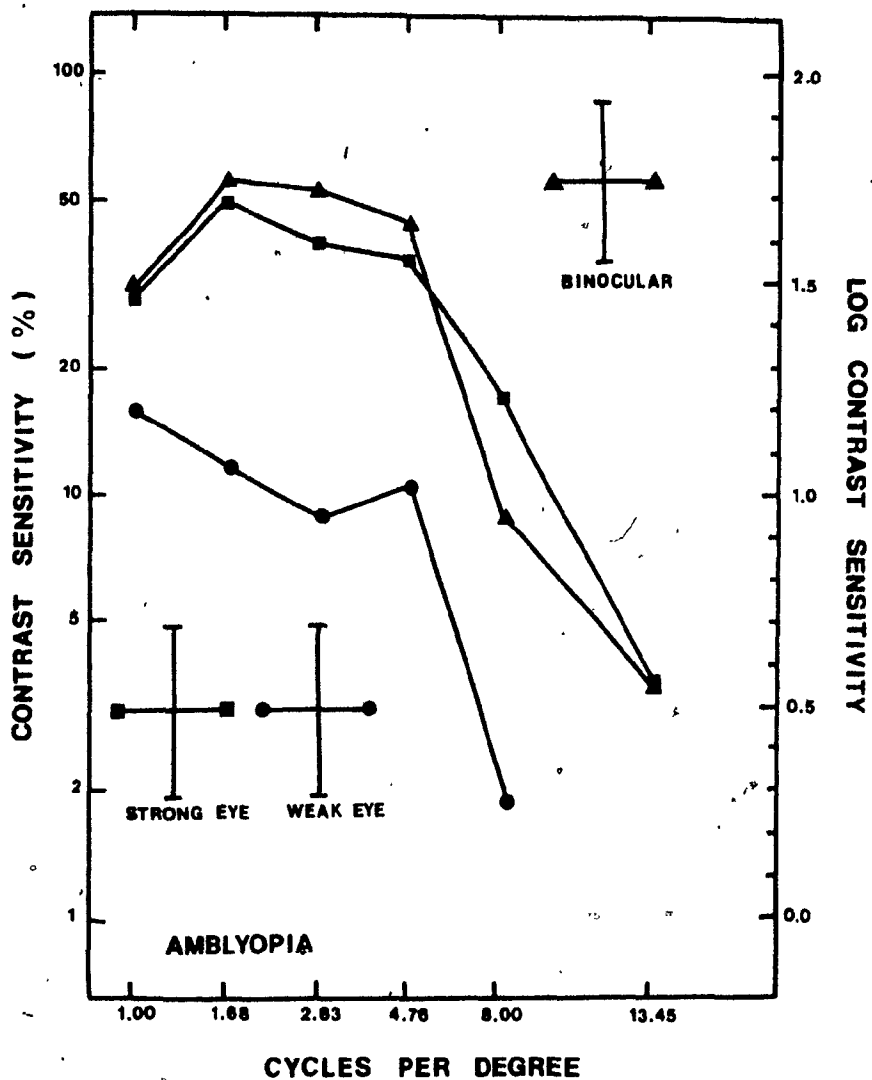


Figure 2.

Contrast sensitivity as a function of spatial frequency in amblyopia, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

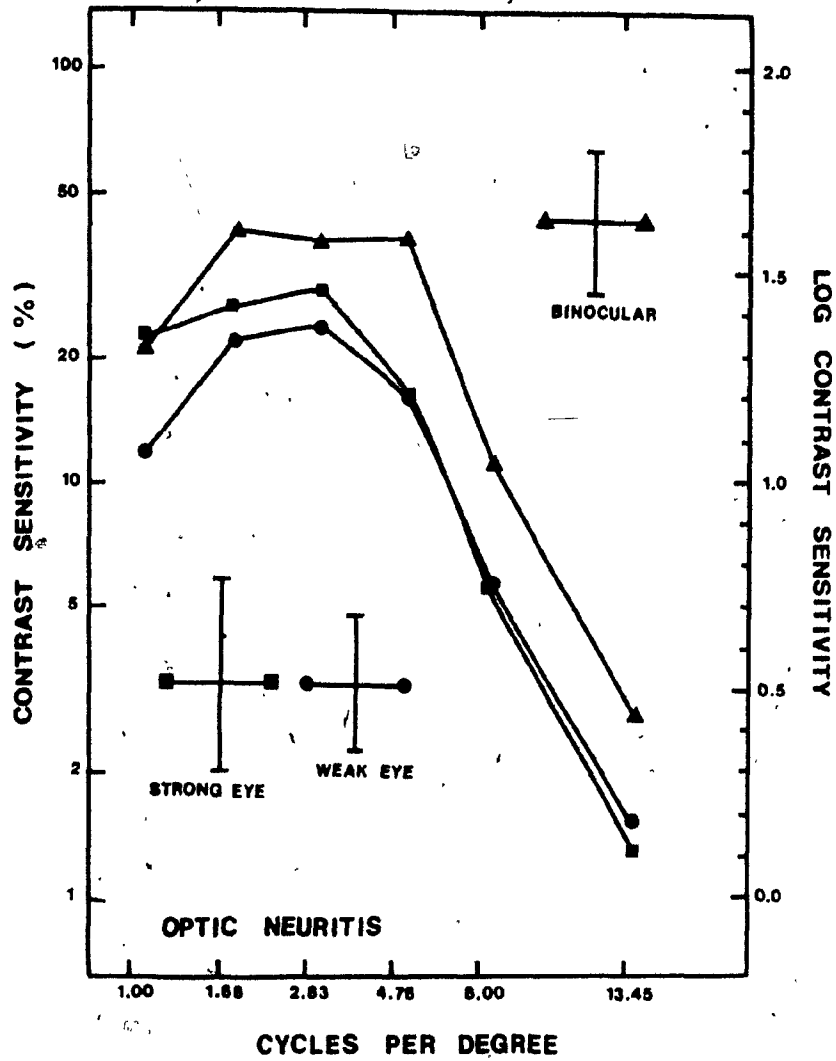


Figure 3. Contrast sensitivity as a function of spatial frequency in optic neuritis, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

The contrast sensitivity of the weaker eye is significantly reduced at all spatial frequencies, as may be seen in Figure 4.

Finally, in the macular degeneration group, there are no statistically significant differences among the viewing conditions across all spatial frequencies. There is a consistent trend, however, for the binocular contrast sensitivity to be lower compared to the stronger eye. This tendency shown in Figure 5 is especially striking in the lower spatial frequencies where the results in the binocular condition are approximately equal to those produced by the weak eye.

Discussion

The results of the amblyopic group support the findings of Levi and Harwerth (1980) who demonstrated that amblyopic eyes show reduced contrast sensitivity over many spatial frequencies. Additionally, the demonstrated decrement of sensitivity in low spatial frequencies for individuals with cataracts, macular degeneration, and optic neuritis substantiates a great number of previous results which led to the acceptance of this test in assessing visual acuity. The optic neuritis group did not show as dramatic a loss in the lower frequencies as did the other groups. However, it must be stressed that these individuals showed the least degree of asymmetry, with the lowest Snellen acuity in a weak eye being 20/40 (6/12). One may speculate that the loss in the low spatial frequencies is exacerbated when there is a greater degree of impairment in the weaker eye.

Considering the small samples and the lack of statistically significant differences between the results of the strong eye and binocular viewing, only tentative conclusions may be drawn regarding the apparent

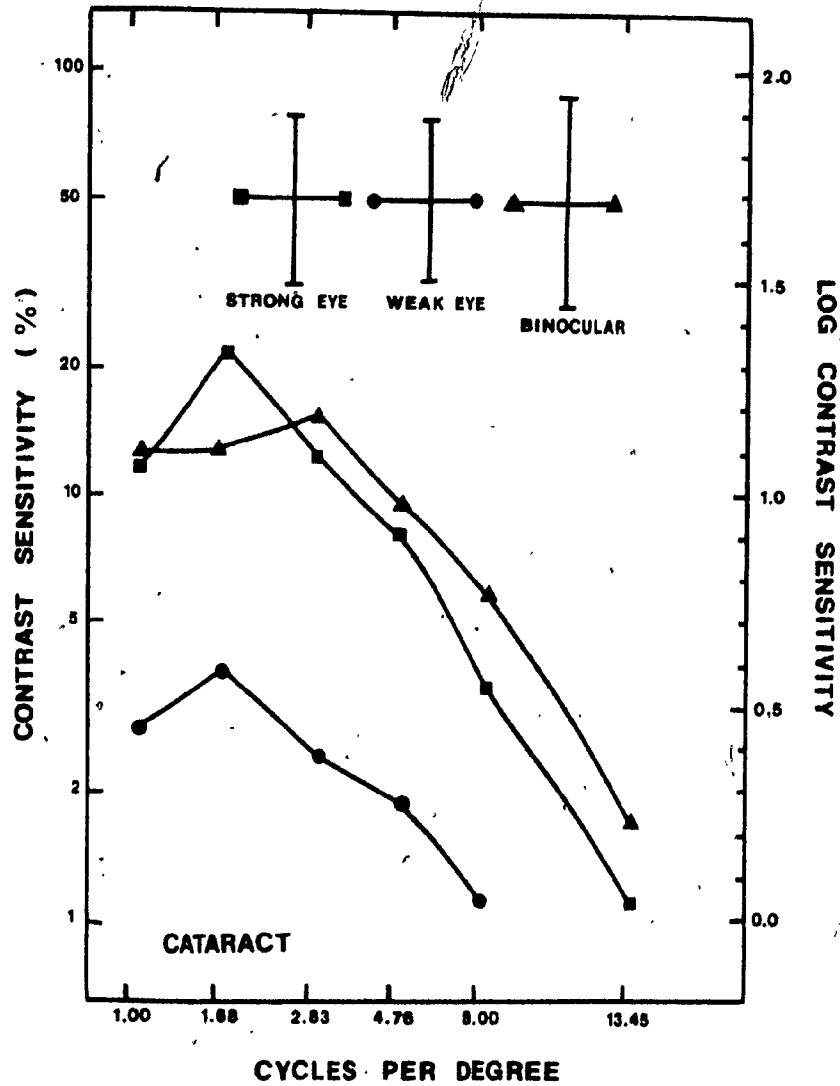


Figure 4. Contrast sensitivity as a function of spatial frequency with cataract, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

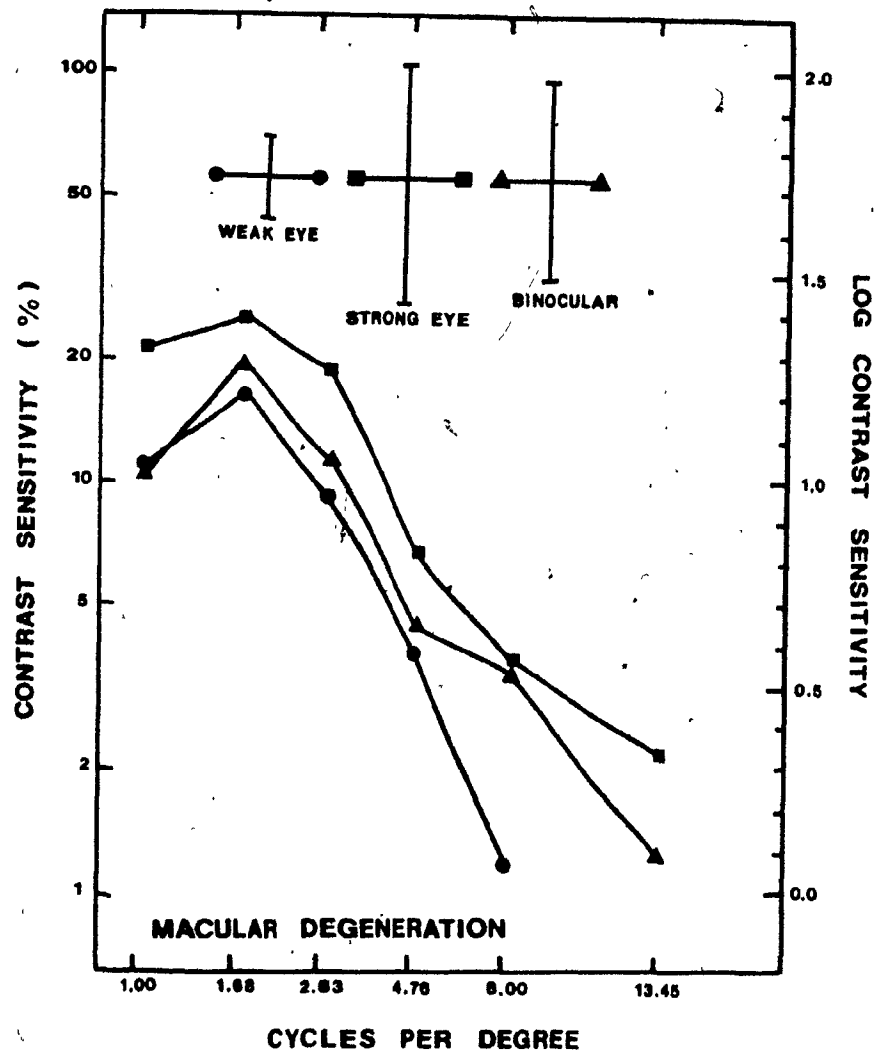


Figure 5. Contrast sensitivity as a function of spatial frequency in macular degeneration, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

use of summation or suppression in all but the macular degeneration groups. It seems that averaging is not occurring in any of the pathological groups with the possible exception of macular degeneration, but this trend must be interpreted cautiously.

If one assumes that averaging is occurring in asymmetrical maculopathy, the reason for this strategy requires some speculation. In all the other groups examined in this study the retinal receptors were intact. Although visual information is substantially degraded by optical aberrations, or slowly transmitted by pathological optic nerves, or not processed at more central levels of the system it still activates the retinal receptors. This is not the case in macular degeneration, where large groups of retinal receptors no longer function normally. It may well be that the "patchy" firing from the retina may activate an averaging process, whereas the other conditions may evoke summation or suppression.

Experiment 3

Temporal resolution has proven to be a very useful measure of visual function. In this experiment, a new psychophysical technique called multi-flash campimetry, developed by Brussell, White, Bross, Mustillo, and Borenstein (1981/82), was used to map visual fields based on temporal resolving power. Additionally, flickering sine wave gratings were used to assess sensitivity to the temporal component of the contrast sensitivity function described in Experiment 2, since it has been previously determined that flickering a grating leads to changes in contrast sensitivity (Levi & Harwerth, 1977).

Method

Subjects The same 25 subjects participated in this experiment.

Apparatus Both the multi-flash and contrast sensitivity tasks were implemented on a PDP11/10 computer interfaced with a large screen cathode ray tube (Hewlett Packard 1310A equipped with a P15 phosphor). The luminance of both displays was measured with a Spectra Spot Meter.

Procedure Multi-flash campimetry is an extension of the double-flash discrimination test. By presenting many, rather than two, flashes to a given retinal location and by increasing the amount of time separating the flashes, one can more rapidly assess temporal resolution. This technique tests 120 retinal locations per eye by using the display shown in Figure 6. Each of the six concentric circles consists of 20 points separated by 18 polar degrees. At a viewing distance of 57 cm, maintained by the use of a chin rest, each point subtends a visual angle of about 5 min. The radius of the innermost circle subtended 0.625 deg

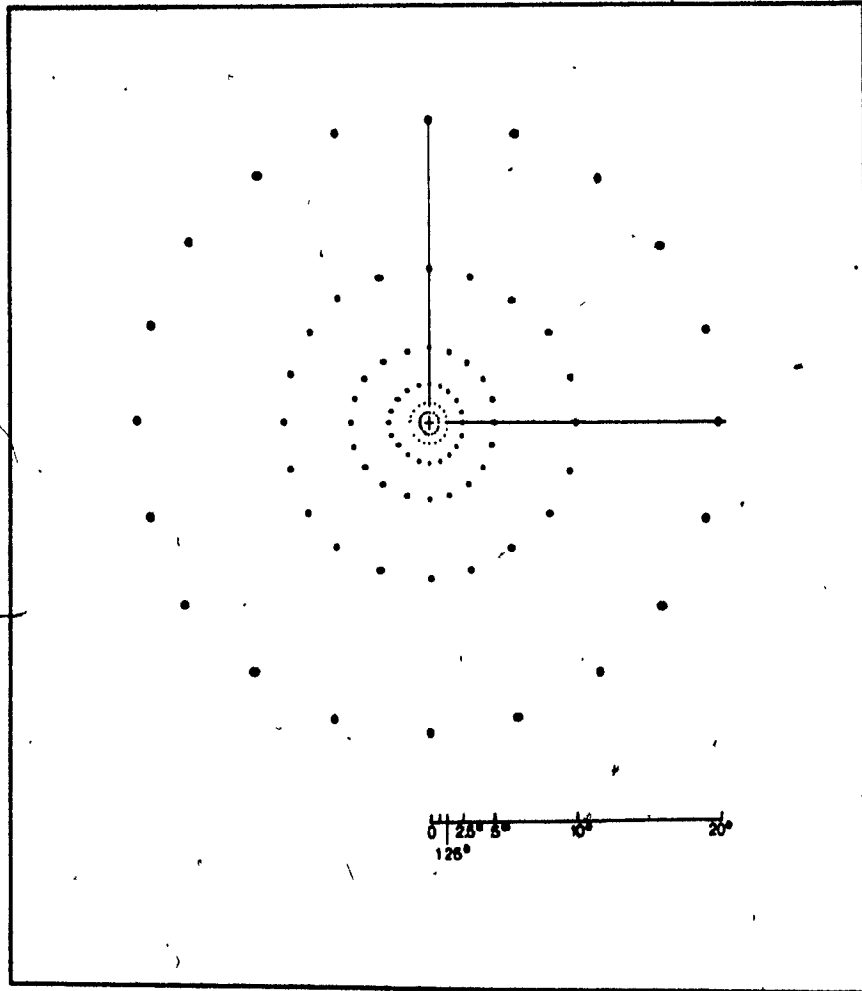


Figure 6. A representation of the stimulus display in multi-flash campimetry. The perpendicular lines indicate that only one quadrant was presented at any given time.

of visual angle. The radius of any other circle was double that of the previous one, such that the radius of the outermost circle subtended a visual angle of 20 deg. Only one quadrant of the display was visible at a time and both the order of the quadrants and the tested points within a quadrant was random.

The luminance of a lit area that contained all of the points placed adjacent to one another was 1.5 cd/m^2 . In an otherwise dark room, each observer first scanned an image that consisted of the superposition of all four quadrants of the display. This established an initial state of light adaptation in the tested eye. There was also a practice trial which utilized a simplified display of only 12 points. The observer was required to fixate a small cross-shaped target in one corner of the screen and to press a key as soon as one of the 12 points appeared to flicker. The task was identical in the test trials but there were 36 points on the screen at one time. The computer flickered each testing point at a frequency of 5 Hz. Within each 200 msec cycle the amount of time during which the point was turned off was increased in steps of 2.8 msec until the observer pressed the response key.

At the end of each session the computer printed the critical off periods for each quadrant and for both eyes, and noted any points that were statistically deviant. Specifically, an off period was considered deviant if it was more than seven standard errors longer than the mean for all points within its circle or more than 21 standard errors longer than the mean for all points within an eye. The statistically deviant points were then immediately repeated in order to ascertain whether they

reflected lapses in attention, momentary loss of fixation, or whether they corresponded to retinal regions whose temporal resolving power was genuinely impaired. This test was only done monocularly to assess each eye's temporal resolving power.

The spatio-temporal sensitivities for each observer were determined by the procedure described in Experiment 2. The only difference was in the spatial and temporal frequency combinations. In this experiment, spatial frequencies of 1.00, 2.83, and 8.00 cpd were paired with each of seven temporal frequencies ranging from 1 to 32 Hz (plus 0 Hz) in one octave unit steps.

Results

The multi-flash campimetry provided an assessment of temporal resolution in 120 retinal points per eye, where the dependent measure is the critical off period required for an observer to indicate that a point appeared to flicker. The data are mapped in such a way that light gray areas of the map reflect good temporal resolution and increasingly dark areas show a decrement in temporal resolution acuity. These maps substantiate the degree of asymmetry experienced by the participants in this study. No response to any given point was noted if the off-period was 200 msec and this would constitute the darkest area on the maps. Figure 7 contains one map from each of the groups having some form of visual impairment and allows comparison of the four pathological conditions examined in this study. Individual maps for each observer are shown in Appendix A.

Amblyopic eyes and those with cataracts generally produce a dark

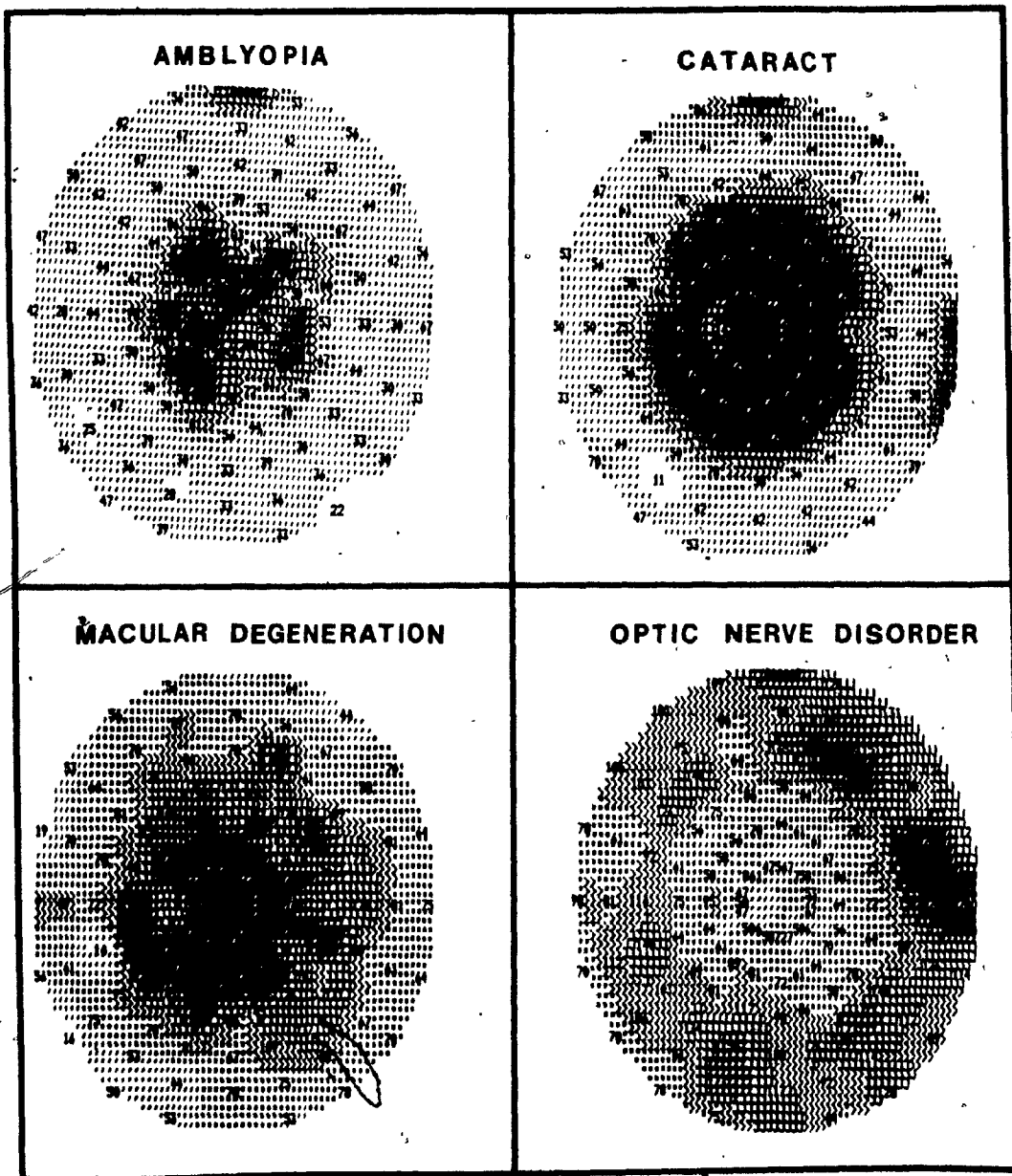


Figure 7. Representative temporal resolution fields in eyes with the pathological conditions examined in this study.

central area indicating a loss of temporal resolution in the foveal and parafoveal area. Macular degeneration and optic neuritis may show a more scattered pattern of loss. Specifically, early retinopathy may show small 'islands' in the macular area which eventually grow to form a central scotoma. In patients with optic neuritis, on the other hand, a scotoma appears only in acute stages of the disorder. However, some individuals retain 'islands' of temporal resolution loss even in remission stages.

The data obtained in the spatio-temporal task allowed for the assessment of binocular resolution. As in Experiment 2, only one spatial frequency is presented since it best reflects the observers' temporal sensitivity at different frequencies. The 2.83 cpd grating was chosen since it produced the least number of "no response" trials. For the normally sighted observers, there were no significant differences between their monocular scores obtained with either eye and their binocular performance at any of the temporal frequencies, as shown in Figure 8. It is notable that binocular sensitivity is higher when the temporal component is introduced. The observers with amblyopia and cataracts showed no difference between their binocular scores and those produced by the stronger eye. However, the weaker eye always showed a lower performance. Figures 9 and 10 contain the results for the amblyopic and cataract groups, respectively.

In the optic neuritis group several points are to be noted. First, there is no significant difference among the three conditions when the grating was not flickered. As soon as the temporal component is intro-

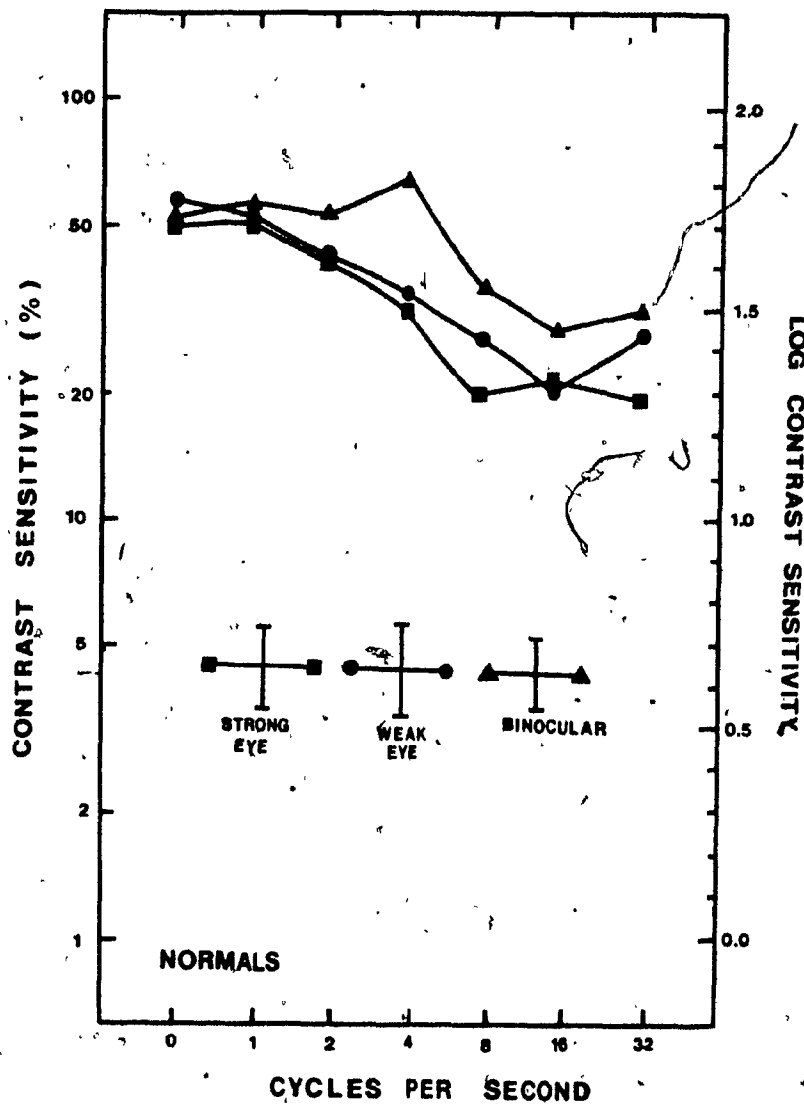


Figure 8. Contrast sensitivity as a function of temporal frequency in normals, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

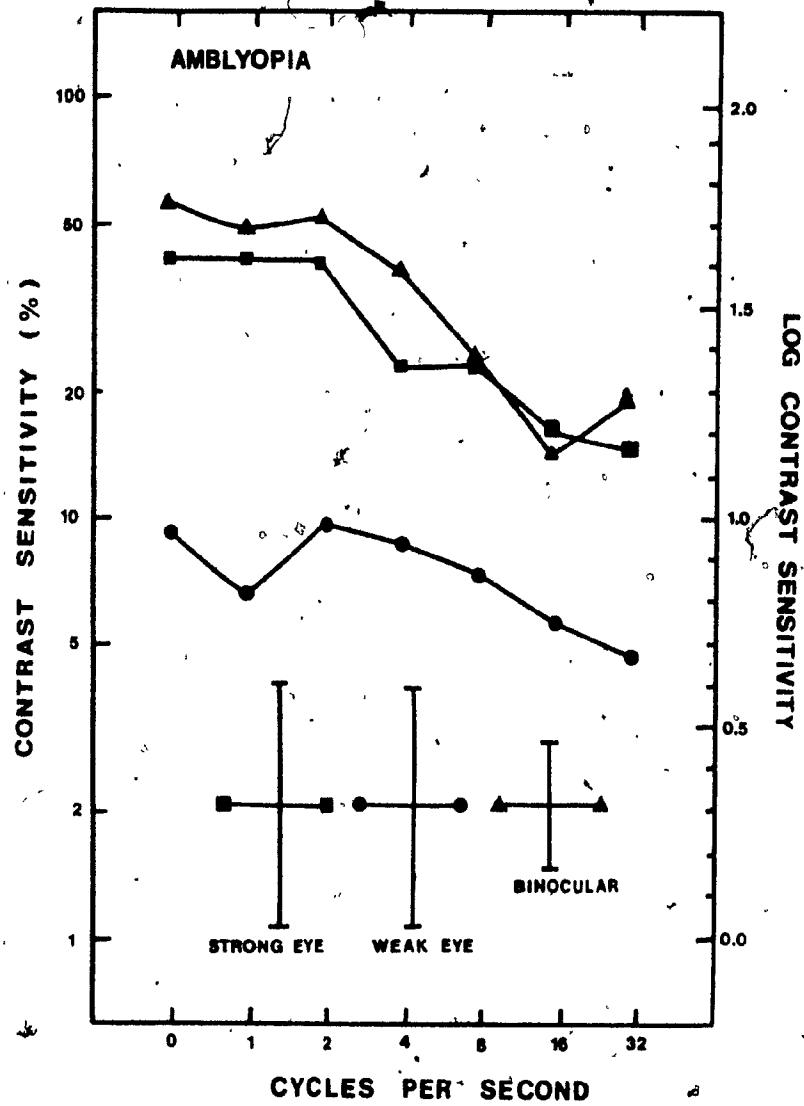


Figure 9. Contrast sensitivity as a function of temporal frequency in amblyopia, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

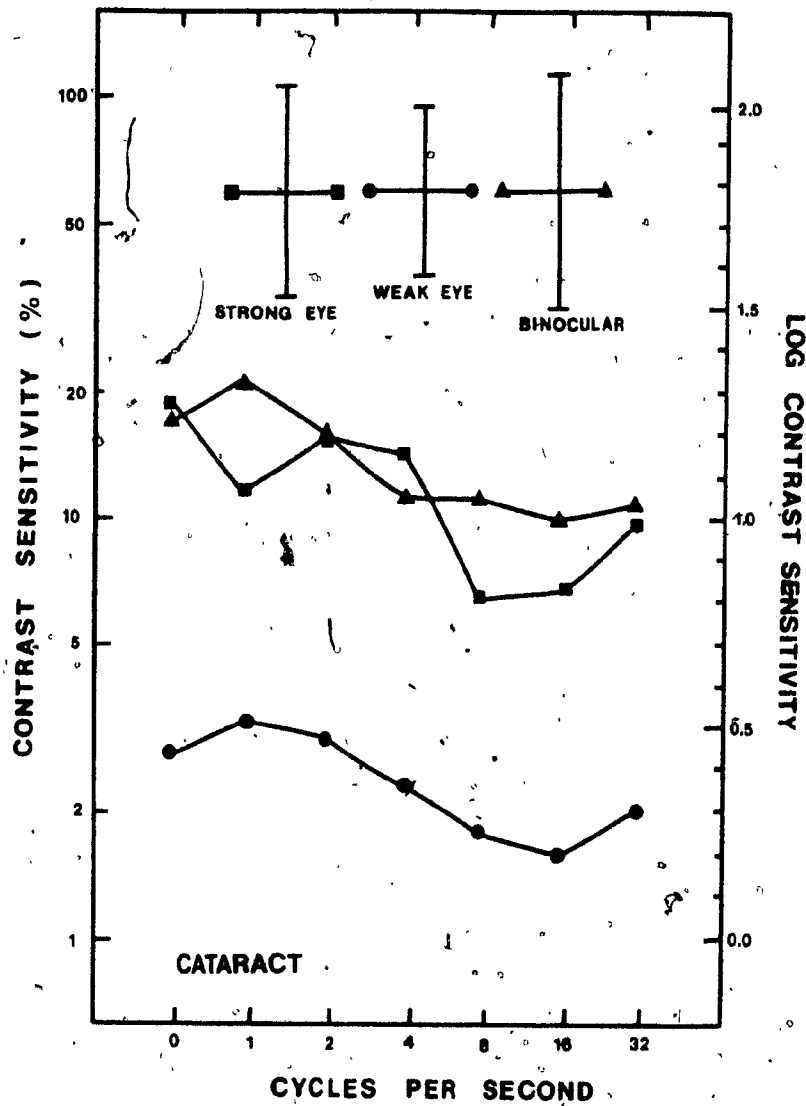


Figure 10. Contrast sensitivity as a function of temporal frequency with cataracts, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

duced, however, the weaker eye's sensitivity differs significantly at least from the binocular condition, as shown in Figure 11. A repeated measures analysis of variance shows that a statistically significant difference occurs when the grating is flickered at 8 Hz $F(2,8) = 7.22$, $p < .05$. Additionally, binocular sensitivity was greater at all temporal frequencies even though this difference was not statistically significant.

Figure 12 shows the results of the macular degeneration group. Although there are no statistically significant differences among any of the viewing conditions at any of the temporal frequencies, the trends in the data are interesting. As in the previous experiment, this is the only group that shows consistently higher scores when viewing the test stimulus with the strong eye alone. Also, the weaker eye shows higher sensitivity at 4, 8, and 16 cycles per degree, which did not occur in any other group. The other temporal frequencies produce scores that parallel the performance of the other groups with visual pathology.

Discussion

Previous research has indicated that amblyopes show reduced sensitivity to temporally modulated stimuli. The results of this experiment support these earlier findings of Manny and Levi (1982). Additionally, the expected equality of performance between the non-amblyopic eye and binocular viewing was obtained. Studies of temporal resolution in persons with cataracts and macular degeneration are less common and the differences obtained here between these groups requires further investigations.

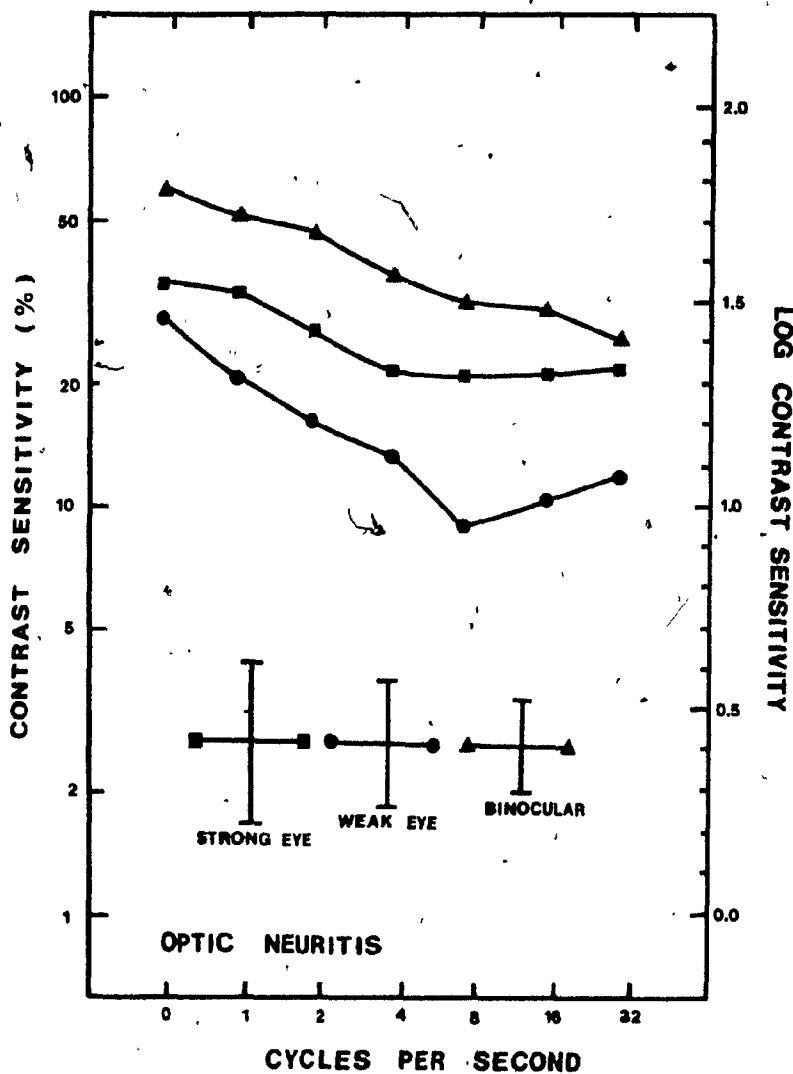


Figure 11. Contrast sensitivity as a function of temporal frequency in optic neuritis, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

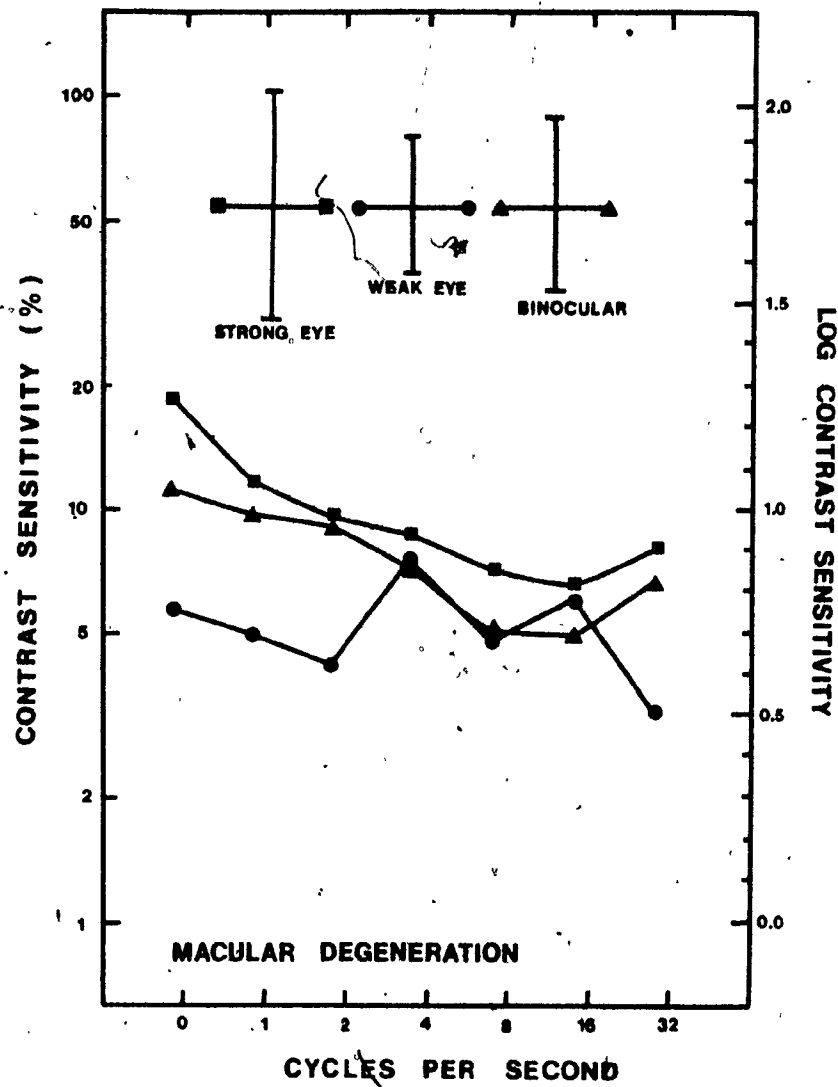


Figure 12. Contrast sensitivity as a function of temporal frequency in macular degeneration, showing monocular and binocular sensitivity. Average standard errors are shown for each of the three conditions.

As was the case in the previous experiment the group with cataracts showed little difference between the sensitivity of their strong eye alone and their binocular sensitivity. If anything, there was a tendency for these individuals to show slightly better binocular performance. It seems that when the visual input is degraded by media opacity a healthy binocular mechanism might add the two messages. If, on the other hand, the photoreceptors degrade the information to the binocular mechanism, one might observe an averaging phenomenon which seems to occur in the case of macular degeneration.

Finally, there is no shortage of evidence that optic nerve disorders affect temporal resolution (Daley et al., 1979; Galvin et al., 1976; Regan, 1980). In this study, all participants with optic neuritis were in remission and their Snellen acuities were not lower than 20/40 (6/12). Yet, a flickering grating was more difficult for them to detect with their weaker eye. It is reasonable to assume that if damage to their optic nerves became more extensive the sensitivity curve produced by their weaker eye would be even lower.

Experiment 4

This experiment tested for stereopsis in all four pathological conditions to assess the effect of the unequal input to the two eyes on this task. Stereoacuity is reflected by the smallest amount of horizontal retinal image disparity that gives rise to a sensation of relative depth and is expressed in seconds of visual angle (Romano et al., 1975). This assessment was made with the Titmus Stereotest and the Howard-Dolman apparatus.

Method

Subjects The same 25 subjects participated in this experiment.

Apparatus Stereoacuity was assessed by using the standard Titmus Stereotest, (Titmus Optical Co., Inc.) using Polaroid Vectographs and glasses. Additionally, the Howard-Dolman depth perception apparatus was used since none of the participants in this study had any problems dealing with eye-hand coordination.

Procedure While wearing the polarized viewers which are necessary for the Titmus Stereo test, the subject was asked to indicate which of several objects on a Vectograph looked closer. The best stereoacuity score possible on this test was 40 seconds of arc. The standard scoring procedure entails continuation of the test from the largest angle to the smallest until the subject no longer responds or until two successive errors occur. In either case, the score is the angle of stereopsis associated with the last test stimulus which the subject discriminates correctly. No meaningful judgements can be made monocularly with this test and normal Titmus stereoacuity is 50 seconds of arc or better

(Schmeisser & Dawson, 1982).

The Howard-Dolman apparatus is constructed in such a way that the observer can align two suspended rods in the device by pulling either of two strings attached to the rods. The viewing distance was three meters and the room was dark except for the illuminated panel at the rear of the apparatus. The observer, wearing corrective lenses for distance if necessary, aligned the two rods so that they were perceived to be equidistant from any chosen reference point, such as the front of the apparatus. At the outset of each trial the rods were separated by some randomly chosen distance. Five trials were performed binocularly, five with the stronger eye covered by a black patch, five with the weaker eye covered by the opaque occluder, and five with the weaker eye covered with a frosted lens. These viewing conditions were randomly ordered except that the weaker eye was never tested first. The measure recorded at the end of each trial was the displacement of the rods in centimeters.

Results

There were great differences among groups on the stereopsis measures, as can be seen in Table 2 which shows the range of Titmus stereocutities and in Table 3 which shows the average displacements of the rods in the Howard-Dolman apparatus. Only the normals showed excellent stereopsis of 40 or 50 seconds of arc with the Titmus Stereotest and their binocular performance on the Howard Dohlman apparatus was significantly better than in any of the monocular conditions, $F(3,12) =$

TABLE 2

Titmus Stereotest Scores
in Seconds of Arc

Amblyopia	Cataracts	Optic Neuritis	Macular Degeneration	Normal
100	800	400	800	40
200	400	50	800	40
60	800	100	800	40
800	200	40	140	50
800	400	60	800	40

TABLE 3

Mean Displacements of the
Howard-Dolman Test Rods

	Strong Eye Black Patch	Strong Eye Frosted Lens	Weak Eye	Binocular
Amblyopia	7.1	4.7	8.4	5.2
Cataract	3.8	4.6	5.1	5.7
Optic Neuritis	5.3	4.6	4.7	1.8
Macular Degeneration	5.1	5.4	9.3	3.6
Normal	3.1	3.3	2.8	.6

4.61, $p < .05$. Individuals with optic neuritis varied on the Titmus Stereotest from 40 to 400 seconds of arc but, like the normals, their Howard Dolman performance was significantly better binocularly, $F(3,12) = 3.78$, $p < .05$. Tukey's post hoc analyses were performed to determine which performances were significantly different.

Individuals with cataracts and amblyopia showed virtually no stereopsis on either test. The depth judgements were not significantly different across all four viewing conditions. The group that had macular degeneration also showed no stereopsis on the Titmus test. When viewing the Howard-Dolman apparatus with the weaker eye, four out of five people could not see the bars and therefore made no adjustments. The statistical analysis showed this to be a significantly worse performance than in both of the other monocular conditions as well as in binocular viewing. There were no other statistically significant differences in this group.

Discussion

The results of this experiment indicate that stereopsis can only be achieved when the degree of asymmetrical impairment is not extensive. Since the group with optic neuritis maintained better vision in their weak eye than did the other visually impaired subjects, binocular depth perception was still possible. Their binocular judgements on the Howard-Dolman apparatus are much less accurate, however, when compared to normally sighted subjects.

In all the other groups, the observers admitted that they were guessing when trying to position the rods in the apparatus. The varia-

bility of their scores attested to this fact. Henson and Williams (1980) have reported that half of their strabismic subjects had significantly better binocular thresholds when tested with the Howard-Dolman apparatus. In this study, both strabismic and anisometropic amblyopes were studied and none of the five subjects had monocular scores which were better than their binocular ones. The methodology of the two studies differed as well, and this difference may account for the discrepancy in the results since the individuals tested by Henson and Williams were given a forced choice on every trial and their viewing distance was double that used in this experiment.

Experiment 5

Colour vision has often been shown to be vulnerable to early onsets of visual pathology and was, therefore, considered to be a potentially informative index for this study. Pinckers (1982) has stressed the utility of colour vision tests for the differential diagnosis of retinal and optic nerve disorders and Mainster and Dieckert (1980) reported that colour brightness comparison is a useful technique for detecting monocular or asymmetrical deficits in optic nerve or macular function. Their test consisted of presenting a brightly coloured object to each eye and determining whether the object was perceived to be of equal brightness. This is also a measure which allows one to evaluate the quality of the visual stimulus which has been shown to be of equal value to quantitative measures.

Method

Subjects The same 25 subjects participated in this experiment.

Apparatus The American Optical H-R-R Pseudoisochromatic Plates (AO H-R-R) as well as the Farnsworth-Munsell 100-hue test were used to ascertain each participant's degree of colour vision. Both tests required the use of an Easel Lamp (Macbeth Corporation).

Procedure Participants who normally wore corrective lenses for near work were asked to wear them for this task. The plates of the AO H-R-R test were presented to each eye alone and to both eyes at the same time. The observer was told that three symbols would be used in the test: a circle, an X, and a triangle, and on each plate the symbols were named and pointed out by the observer. The book of plates rested on the rack of the Easel Lamp and the viewing distance was not con-

trolled during testing due to the wide range of participants' acuities.

The Farnsworth-Munsell 100-Hue test (FM-100) which measures colour discrimination and colour confusion was also administered under two monocular conditions using the black patch as well as under the binocular condition. The order of the viewing conditions was random, except that the weaker eye was not tested first. The frosted lens was not used in this experiment. The 100-Hue test consists of 85 coloured disks which the observer must arrange in the correct sequence of shades. The test is divided into four boxes with permanent reference colours at the ends of each box to indicate the beginning and end of the sequence. The score is based on the observer's deviations from the correct order where a high error score reflects poor colour discrimination.

Results

The H-R-R plates allow one to assess the type as well as the extent of colour defect. The results showed all the normals to have excellent colour vision. Additionally, all but one amblyope, who had a mild red-green defect, made no errors or omissions on this test.

The subjects who had cataracts could not see any figures on the plates with the weaker eye but the dominant eye had normal colour vision and binocular performance was not impaired. In the optic neuritis group, two observers had normal colour vision and three showed a mild red-green impairment in the eye which had suffered an acute attack in the past. This weakness was not seen in the binocular condition.

The most severe colour defects were found in the group with macular degeneration, although one observer with 20/70 (6/21) and 20/200

(6/60) Snellen acuities showed no color defect. The other four observers, however, ranged from a mild red-green defect in the weaker eye to strong defects which affected binocular performance as well. Only one participant had extremely low colour discrimination which approached monochromatism.

The results of the FM-100 test showed that normals and individuals with cataracts have equal colour discrimination whether they view the coloured caps monocularly or binocularly. Of course, the eyes with the more advanced cataracts could not see the caps at all, but only one participant in this study did not make an attempt to do the test with his weaker eye. The mean error scores, obtained on the FM-100 by each group are shown in Table 4. Individual results are given in Appendix B.

Statistically significant differences were found in the amblyopic group, $F(2,8) = 5.49$, $p < .05$, in the optic neuritis group, $F(2,8) = 7.53$, $p < .05$, and in the macular degeneration group, $F(2,8) = 11.32$, $p < .01$. In all cases, however, Tukey's post hoc analysis showed that these differences were caused by the weaker eye. There were no statistically significant differences between binocular colour discrimination and monocular discrimination when using only the stronger eye. It is interesting to note that individuals with optic neuritis and macular degeneration showed lower performance binocularly than with their stronger eye. Again, the small samples and large variability in the data prevent one from concluding that an averaging process has been utilized by the visual system.

TABLE 4
Mean Error Scores for the
Farnsworth-Munsell 100-Hue Test

	Strong Eye	Weak Eye	Binocular
Amblyopia	72	116	65
Cataract	129	415	132
Optic Neuritis	77	214	146
Macular Degeneration	352	631	419
Normal	54	48	53

Discussion

Although binocular performance is not impaired when tested with the AO H-R-R plates, it has a tendency to be lowered in those individuals with optic neuritis and macular degeneration. Amblyopes and persons with cataracts are able to suppress the input from the weaker eye but retinal and optic nerve disorders seem to disturb suppression. Since the FM-100 is used to assess a person's ability to discriminate colour as well as to identify colour defectiveness, error scores are obtained on this test even when the H-R-R plates indicate normal colour vision. It is easier, therefore, to compare monocular and binocular performance by examining either the error scores or the patterns which are drawn on the basis of those scores.

The observers with asymmetrical pathology did not produce any typical bipolar patterns on the FM-100 which would indicate a specific colour defect but, rather showed high error scores which reflect poor colour discrimination. The lower binocular performance in the optic neuritis group is interesting since this is the only occasion where an averaging of their monocular inputs may be occurring. Those observers with macular degeneration show similar trends to those found in the spatio-temporal experiments.

Experiment 6

Finally, reading performance was assessed under all four viewing conditions since this is a functional measure which is subjectively important to visually impaired individuals and the task for which a frosted lens is often recommended. It was, therefore, considered worthwhile to supplement the numerous psychophysical findings with those measures which may be more frequently used in clinical settings.

Method

Subjects The same 25 subjects participated in this experiment.

Apparatus A reading text, extracted from Time Magazine, was prepared using large print equivalent to 14-point print size which is expressed as 2M print in Snellen notation.

Procedure The subjects wore standard correction for near vision, if necessary, and were allowed to hold the text at any distance. The text was divided into four parts and the order of the viewing condition was randomized. Each participant was asked to read the text aloud at a comfortable speed. The reading was timed and the number of errors were recorded. In determining the final score, the formula used to assess typing skills was used in order to specify speed-error relationship. This consisted of counting the number of words read in 60 seconds and by subtracting from this the number of errors multiplied by five. The formula is based on the assumption that an average word in a text contains five letters. By this formula, a high score reflects a good performance.

Results

The reading scores obtained in this experiment were derived from the reading speed with a penalty for errors. The reading scores are shown in Table 5. Except the normal and the optic neuritis group, the weaker eye produced a low performance and some subjects could not read at all with their weaker eye. The results in all other viewing conditions were not significantly different; there was no binocular advantage or disadvantage.

Discussion

While it is difficult to generalize from the results obtained in this experiment to clinical procedures, it should be noted that the within-groups differences were extensive. For example, two participants in the macular degeneration group, with equivalent acuities, behaved very differently in the reading test. One person could read binocularly with only a strong bifocal correction but the other could only read monocularly with a powerful magnifier, while closing his weaker eye. Additionally, the older participants with asymmetrical pathology of any kind were more likely to complain about their binocular reading, despite the objective evidence. This may well reflect a change in the degree of adaptability which may be independent of the locus and extent of the pathology.

TABLE 5

Mean Reading Scores

	Strong Eye Black Patch	Strong Eye Frosted Lens	Weak Eye	Binocular
Amblyopia	96	76	41	95
Cataract	90	88	0	106
Optic Neuritis	94	95	84	90
Macular Degeneration	59	73	17	68
Normal	89	65	93	82

General Discussion

Three major issues were addressed in this study. The first dealt with the identification of binocular processes that are activated by asymmetrical visual pathology. The second question was concerned with the possible task-dependence of these strategies. Finally, the third issue related to the prescription of lenses or occluders for individuals with asymmetrical cataracts, optic neuritis, or macular degeneration.

Of these, the third issue is the most straightforward: there was never any difference between task performances when the weak eye was covered by a frosted lens and when it was covered by a black occluder. Therefore, on the basis of these data, no statement can be made regarding the prescription of one or the other occluder and the patient's preference may be the best guideline.

The first two issues are more complex since there are both group and task differences which must be addressed. Primarily, it must be emphasized that Snellen optotypes, which are used to assess distance and near acuity, are limited in their utility. This point has been stressed by other workers who have studied visual impairment caused by amblyopia (Lennerstrand & Lundh, 1980), cataracts (Hess & Woo, 1978), retinal disorders (Marmor, 1981), and optic neuritis (Regan et al., 1977). In this study, the acuities of the stronger eye and those obtained from binocular viewing were equivalent in all four diagnostic groups. The normally sighted individuals had significantly higher binocular acuities, in agreement with previous research results (Blake et al., 1980). All individuals with asymmetrical pathology, regardless of its locus, were able to suppress the input of the weaker eye during binocular

viewing. This indicates that high-contrast, single visual targets are recognized equally well binocularly or with the stronger eye alone, and implies suppression.

This is not the case when one examines the results from the contrast sensitivity test. Here, differences emerge which are dependent on the locus of pathology. Normally sighted individuals and amblyopes produce virtually equal results binocularly and monocularly, using the stronger eye. Individuals with cataracts and optic neuritis show evidence for summation of the monocular inputs since their binocular sensitivity is higher regardless of spatial frequency. However, those with macular degeneration demonstrate a decrement of sensitivity in the binocular viewing condition. Averaging of the two inputs seems to be occurring only when the retinal receptors are impaired. Perhaps an explanation of these results lies in the extension of the "misuse-disuse" notion which was discussed by Herman et al. (1980). A cataract or other media opacity can be responsible for "disuse" of the binocular mechanism by decreasing the signal strength of the stimulus below some critical level which may be required for efficient binocular processing. On the other hand, macular degeneration may create a potential "misuse" of the binocular system. The limited reactivity of the photoreceptors in the presence of this disorder may provide a very segmented signal which evokes an averaging mechanism. It must be stressed that even though these are compelling possibilities, one should exercise caution in their generalization due to the small number of individuals examined in this study.

Similar results were obtained when examining temporal resolution.

Normally sighted individuals demonstrated higher binocular sensitivity whenever the grating was flickered, which is in agreement with the data of Blake and his coworkers. Amblyopes again showed suppression of the weaker eye and their binocular sensitivity was essentially equal to or slightly better than that of the dominant eye. In the groups with asymmetrical cataracts, binocular and stronger-eye performance was virtually equal over all temporal frequencies. Individuals with optic neuritis again showed evidence of summation since their binocular performance was consistently higher than their monocular sensitivity. Finally, averaging seemed to be occurring in those with macular pathology. These results are strikingly similar to those obtained with steady gratings at various spatial frequencies. It seems that the decrement in sensitivity during binocular viewing is independent of the temporal component when macular degeneration is the cause of visual impairment.

One additional occasion where an averaging process occurs in other than the macular degeneration group is in the colour discrimination test. In their performances on the FM-100, only one out of five amblyopes did worse binocularly than with their dominant eye and normally sighted individuals showed no differences across viewing conditions. However, three out of five observers with cataracts, four out of five with optic neuritis, and all five of the macular degeneration group showed poorer colour discrimination binocularly than with the stronger eye. Thus, optical aberrations, as well as neural damage at either the retinal or the optic nerve level may elicit an averaging strategy when colour discrimination is required and the individual might be well

advised to close the weaker eye when making these judgements.

The difference in the colour experiment regarding possible averaging of the two inputs is difficult to explain. A possibility suggests itself if one examines the tests used in this experiment in light of foveal and peripheral vision. Every test in this study other than colour discrimination could be performed without excellent foveal capacities, including the text reading, since relatively large print size was used. Colour discrimination, however, requires good foveal vision, and when this is interrupted by cataracts or when the foveal information is not transmitted optimally due to an optic nerve disorder, the averaging alternative is observed. Averaging of colour discrimination performance in the cataract group is not as dramatic as it is in macular degeneration or optic neuritis and may be due to statistical variability. One can speculate that the process in the other two groups may be affected by the opponent cells which are found at retinal and post-retinal levels (Cornsweet, 1970). The notable exception occurs in amblyopes who use suppression of the weaker eye routinely. This may indicate that early asymmetrical impairment may allow a by-pass of the averaging mechanism and a constant reliance on suppression.

The most surprising outcome occurred in the reading experiment. Based on clinical reports, it was expected that averaging of the monocular inputs would take place in all but the amblyopic and normally sighted groups. The data did not support this hypothesis since reading performance was equivalent in all conditions across groups, the only obvious exception being the performance of the weaker eye alone. One

is led to the conclusion that in reading, which is a high-contrast, high spatial frequency task, the weaker eye is suppressed. These results parallel those of the first experiment which examined Snellen acuities for distance and near vision.

In summary, the three binocular strategies of summation, suppression and averaging seem to depend mainly on the locus of asymmetrical visual pathology and secondly on the task. In spatial and temporal resolution, averaging occurs only in those visual systems which are impaired by macular degeneration. In colour vision, all groups except the amblyopes and normals showed at least some evidence of averaging. All other tasks seem to evoke either binocular summation or suppression of the weaker eye. In practical terms, this indicates that binocular mechanism in these tasks are unimpaired by asymmetrical pathology.

Cogan (1982) examined binocularity but used a dichoptic viewing condition. He stated that if the contours of two images, each presented to one eye, are grossly different, the eye which has a contour-rich target is predominant and the other eye with the contour-poor target is perceptually suppressed. Porac (1974) also proposed that where visual input is of poor quality there is a reliance on the physiologically superior eye, and Levelt (1966) discussed this phenomenon in view of the contour mechanism and the law of complementary shares. If it can be assumed that asymmetry of the two eyes provides this sort of contour-rich/contour-poor situation, then all of the groups in this study should have showed suppression of the weaker eye. Yet, the group with macular degeneration produced data which supported the averaging alternative in spatial and temporal sensitivity tasks. Additionally, there was some

evidence of summation in the case of asymmetrical cataract and optic neuritis. The difference in methodology used by Cogan (1982) and that of the present study may explain this discrepancy or it may be that visual pathology, other than amblyopia, selectively impairs suppression.

The results of this study also support the claims of many investigators regarding the utility of contrast sensitivity and temporal resolution tests in comprehensive assessment of visual function. These tests, along with the FM-100 test of colour discrimination, were able to differentiate between groups in terms of their use of adaptive binocular strategies. More traditional tests such as Snellen acuities and reading performance did not yield the same amount of discrimination. Longitudinal studies of all four diagnostic categories examined in this study are necessary if the effects of asymmetrical pathology are to be understood. The adaptation of the visual system over time may provide further explanation of binocular processing.

In summary, the results obtained in this study indicate that the visual system may react differently when required to resolve fine detail in the environment than it does when other functions are assessed. Specifically, the strategies used during binocular viewing are not mutually exclusive given the existence of visual pathology or the locus of the impairment. Thus, individuals may use one or another strategy depending on the visual task at hand but might show a preference depending on the cause of their visual asymmetry. These findings suggest that the visual system maintains an appreciable degree of functional plasticity even when its physiological development is complete.

References

- Anderson, C., & Sjostrand, J. Contrast sensitivity and central vision in reattached macula. Acta Ophthalmologica, 1981, 59, 161-169.
- Anstis, S.M. A chart demonstrating variation in acuity with retinal position. Vision Research, 1974, 59, 161-169.
- Bedell, H.E. Central and peripheral retinal photoreceptor orientation in amblyopic eyes as assessed by the psychophysical Stiles-Crawford function. Investigative Ophthalmology and Visual Science, 1980, 19(1), 49-59.
- Blake, R., & DiGianfilippo, A. Spatial vision in cats with selective neural deficits. Journal of Neurophysiology, 1980, 43(5), 1197-1205.
- Blake, R., & Rush, C. Temporal properties of binocular mechanisms in the human visual system. Experimental Brain Research, 1980, 38, 333-340.
- Blake, R., Breitmeyer, B., & Green, W. Contrast sensitivity and binocular brightness: dioptic and dichoptic luminance conditions. Perception and Psychophysics, 1980, 27(1), 180-181.
- Blake, R., Martens, W., Garrett, A., & Westendorf, D. Estimating probability summation for binocular reaction time data. Perception and Psychophysics, 1980, 27(4), 375-378.
- Blake, R., Westendorf, D.H., & Overton, R. What is suppressed during binocular rivalry? Perception, 1980, 9, 223-231.
- Blondin, M., & Kenya, P.R. A view of the literature concerning the epidemiology of senile macular degeneration. American Journal of Optometry and Physiological Optics, 1981, 58(8), 643-647.

- Boothe, R.G. The time course for the development of strabismic amblyopia in infant monkeys (*Macaca nemestrina*). Investigative Ophthalmology and Visual Science, 1980, 19(7), 841-845.
- Boothe, R.G., Kiorpes, L., & Hendrickson, A. Anisometric amblyopia in *Macaca nemestrina* monkeys produced by atropinization of one eye during development. Investigative Ophthalmology and Visual Science, 1982, 22, 228-233.
- Bowman, K.J. The clinical assessment of colour discrimination in senile macular degeneration. Acta Ophthalmologica, 1980, 58(3), 337-346.
- Boynton, R.M. Some temporal factors in vision. In Rosenblith, W.A. (Ed.) Sensory Communication. Cambridge: MIT Press, 1961.
- Braun, R.G. Binocular enhancement of colour discrimination in a deutan. American Journal of Ophthalmology, 1976, 81(2), 219-222.
- Braunstein, M.L. Depth perception through motion. New York: Academic Press, 1976.
- Brussell, E.M., White, C.W., Bross, M., Mustillo, P., Borenstein, M. Multiflash campimetry in multiple sclerosis. Current Eye Research, 1981/82, 1(11), 671-677.
- Burian, H.M., & Boeder, P. Some newer aspects of binocular vision. In Sorsby, A. (Ed.) Modern trends in ophthalmology. London: Butterworth & Co. (Publishers) Ltd., 1955.
- Campos, E.C., Enoch, J.M., Fitzgerald, C.R., & Benedetto, M.D. A simple psychophysical technique provides early diagnosis in optic neuritis. Documenta Ophthalmologica, 1980, 49, 325-335.
- Cogan, A.I. Monocular sensitivity during binocular viewing. Vision

- Research, 1982, 22, 1-16.
- Cornsweet, T.N. Visual perception. New York: Academic Press, 1970.
- Crovitz, H.F. Differential acuity of the two eyes and the problem of ocular dominances. Science, 1961, 134, 614.
- Curtis, D.W., & Rule, S.J. Fechner's paradox reflects a nonmonotone relation between binocular brightness and luminance. Perception and Psychophysics, 1980, 27(3), 263-266.
- Daley, M.L., Swank, R.L., & Ellison, C.M. Flicker fusion thresholds in multiple sclerosis: a functional measure of neurological damage. Archives of Neurology, 1979, 36, 292-295.
- Delaney, W.V., & Oates, R.P. Senile macular degeneration: a preliminary study. Annals of Ophthalmology, 1982, 14(1), 21-24.
- Derefeldt, G., Lennerstrand, G., & Lundh, B. Age variation in normal human contrast sensitivity. Acta Ophthalmologica, 1979, 57, 679-690.
- Dobson, J.S., & Davison, P.A. A new rapid test of contrast sensitivity function utilizing spatial bandwidth equilization. Investigative Ophthalmology and Visual Science, 1980, 19(2), 213-217.
- Dobson, V., & Teller, D.Y. Visual acuity in human infants: a review and comparison of behavioral and electrophysiological studies. Vision Research, 1978, 18, 1469-1483.
- Fisher, N.F., Jampolsky, A., & Flom, M.C. Traumatic bitemporal hemianopsia; Part II. Binocular cooperation. American Journal of Ophthalmology, 1968, 65(4), 574-577.
- Fox, R., & McIntyre, C. Suppression during binocular fusion of

- complex targets. Psychonomic Science, 1967, 8(4), 143-144.
- Freeman, R.D., & Bradley, A. Monocularly deprived humans: non-deprived eye has supernormal vernier acuity. Journal of Neurophysiology, 1980, 43(6), 1645-1653.
- Galvin, R.J., Regan, D., & Heron, J.R. Impaired temporal resolution of vision after acute retrobulbar neuritis. Brain, 1976, 99, 255-268.
- Gerrits, H.J.M. Differences in peripheral and foveal effects observed in stabilized vision. Experimental Brain Research, 1978, 32, 225-243.
- Ghafour, I.M., Foulds, W.S., Allan, D., & McClure, E. Contrast sensitivity in diabetic subjects with and without retinopathy. British Journal of Ophthalmology, 1982, 66, 492-495.
- Goldstein, J.H., Clahane, A.C., & Sanfilippo, S. The role of the periphery in binocular vision. American Journal of Ophthalmology, 1966, 62(4), 702-706.
- Griffin, J.F., & Wray, S.H. Acquired colour vision defects in retrobulbar neuritis. American Journal of Ophthalmology, 1978, 86(2), 193-201.
- Hagedoorn, A., Haan, D., Tiesinga, G., Vonhoff, D.J., Buys, J.J., Hooft, R.H., & van Rhijn, S. Binocular vision (The flip-flop phenomenon). American Journal of Ophthalmology, 1972, 74(5), 907-914.
- Hansen, E. The colour receptors in amblyopia investigated by specific quantitative perimetry. Acta Ophthalmologica, 1979, 57, 612-622.
- Harper, D.W., & Bross, M., The effect of unimodal sensory deprivation

- on sensory processes: a decade of research from the University of Manitoba. Canadian Psychological Review, 1978, 19(2), 128-144.
- Harwerth, R.S., Smith, E.L. III, & Levi, D.M. Suprathreshold binocular interactions for grating patterns. Perception and Psychophysics, 1980, 27(1), 43-50.
- Helmholtz von, H. Physiological optics, 1867. Translated 1925.
 Edited by Southall, J.P.C. Optical Society of America.
- Henson, D.B., & Williams, D.E. Depth perception in strabismus. British Journal of Ophthalmology, 1980, 64(5), 349-353.
- Hering, E. The theory of binocular vision 1868. Translated by Bridgeman, B., & Stark, L. New York: Plenum Press, 1977.
- Herman, J.H., Roffwarg, H.P., Rosenmann, C.J., & Tauber, E.S. Binocular depth perception following REM deprivation or awake state visual deprivation. Psychophysiology, 1980, 17(3), 236-242.
- Hess, R., & Woo, G. Vision through cataracts. Investigative Science, 1978, 17(5), 428-435.
- Hubel, H.D., & Wiesel, T.N. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. Journal of Physiology 1962, 160, 106-154.
- Hurvich, L.M., & Jameson, D. The perception of brightness and darkness. Boston: Allyn & Bacon, Inc., 1967.
- Hyvarinen, L., Rovamo, J., Laurinen, P., & Peltomaa, A. Contrast sensitivity function in evaluation of visual impairment due to retinitis pigmentosa. Acta Ophthalmologica, 1981, 59, 763-773.
- Jacobson, S.G., Mohindra, I., & Held, R. Development of visual acuity in infants with congenital cataracts. British Journal of

- Ophthalmology, 1981, 65, 727-735.
- Julesz, B. Binocular depth perception without familiarity cues. Science, 1964, 145, 356-362.
- Julesz, B. Foundations of cyclopean perception. Chicago: The University of Chicago Press, 1971.
- Kaufman, L. Sight and mind: an introduction to visual perception. New York: Oxford University Press, 1974.
- Kayazawa, F., Yamamoto, T., & Itoi, M. Contrast sensitivity measurement in retinal diseases by laser generated sinusoidal grating. Acta Ophthalmologica, 1982, 60, 511-524.
- Kinnear, P.R., Aspinall, P.A., & Lakowski, R. The diabetic eye and colour vision. Transactions of the Ophthalmological Society, United Kingdom, 1972, 92, 69-78.
- Lakowski, R., Aspinall, P.A., & Kinnear, P.R. Association between colour vision losses and diabetes mellitus. Ophthalmic Research, 1972/73, 4, 145-159.
- Leibowitz, H., Post, R., & Ginsburg, A. The role of fine detail in visually controlled behavior. Investigative Ophthalmology and Visual Science, 1980, 19(7), 846-848.
- Lema, S.A., & Blake, R. Binocular summation in normal and stereoblind humans. Vision Research, 1977, 17, 691-695.
- Lennerstrand, G. Binocular interaction studied with visual evoked responses (VER) in humans with normal or impaired binocular vision. Acta Ophthalmologica, 1978, 56, 628-637.
- Lennerstrand, G., & Lundh, B.L. Improvement of contrast sensitivity from treatment for amblyopia. Acta Ophthalmologica, 1980, 58, 292-

294.

- Levelt, W.J.M. Binocular brightness averaging and contour information. British Journal of Psychology, 1965, 56(1), 1-13.
- Levelt, W.J.M. Some demonstrations of the complementary functioning of the eyes. Perception and Psychophysics, 1966, 1, 39-40.
- Levi, D.M., & Harwerth, R.S. Spatial-temporal interactions in an isometric and strabismic amblyopia. Investigative Ophthalmology and Visual Science, 1977, 16(1), 90-95.
- Levi, D.M., & Harwerth, R.S. Contrast sensitivity in amblyopia due to stimulus deprivation. British Journal of Ophthalmology, 1980, 64(1), 15-20.
- Lindberg, C.R., Fishman, G.A., Anderson, R.J., & Vasquez, V. Contrast sensitivity in retinitis pigmentosa. British Journal of Ophthalmology, 1981, 65, 855-858.
- Lubkin, V., & Linksz, A. A ten-year study of binocular fusion with spectacles in monocular aphakia. American Journal of Ophthalmology, 1977, 84(5), 700-707.
- Mainster, M.A., & Dieckert, J.P. A simple haploscopic method for quantitating colour brightness comparison. American Journal of Ophthalmology, 1980, 89(1), 58-61.
- Manny, R.E., & Levi, D.M. Psychophysical investigations of the temporal modulation sensitivity function in amblyopia: uniform field flicker. Investigative Ophthalmology and Visual Science, 1982, 22, 515-524.
- Manny, R.E., & Levi, D.M. Psychophysical investigations of the temporal modulation sensitivity function in amblyopia:

- spatiotemporal interactions. Investigative Ophthalmology and Visual Science, 1982, 22, 525-534.
- Marmour, M.F. Contrast sensitivity and retinal disease. Annals of Ophthalmology, 1981, 13(9), 1069-1071.
- McDougall, W. The principle underlying Fechner's 'paradoxical experiment' and the predominance of contours in the struggle of the two visual fields. British Journal of Psychology, 1904-05, 1, 114-115.
- Millodot, M., & Lamont, A. Peripheral visual acuity in the vertical plane. Vision Research, 1974, 14, 1497-1498.
- Murphy, S.B., & Donderi, D.C. Predicting the success of cataract surgery. Journal of Behavioral Medicine, 1980, 3(1), 1-14.
- Natsoulas, T. On homogeneous retinal stimulation and the perception of depth. Psychological Bulletin, 1963, 60, 385-390.
- Newell, F.W. Ophthalmology: principles and concepts (4th Edition) St. Louis: The C.V. Mosby Company, 1978.
- Overbury, O., & Bross, M. Improvement of visual acuity in partially and fully sighted subjects as a function of practice, feedback, and instructional techniques. Perceptual and Motor Skills, 1978, 46, 815-822.
- Pailhous, J., Chesnais, M., & Leplat, J. Detection et localisation de lacunes en presentation tachistoscopique: role des vision foveale et periphegrique. Annee Psychologique, 1975, 75, 445-456.
- Patterson, V.H., & Heron, J.R. Visual field abnormalities in multiple sclerosis. Journal of Neurology, Neurosurgery, and Psychiatry, 1980, 43, 205-208.

- Perry, N.W. Jr., & Childers, D.G. Monocular contribution to binocular vision in normals and amblyopes. In Arden, G.B. (Ed.) The visual system: neurophysiology, biophysics, and their clinical application. New York: Plenum Press, 1972.
- Phelps, C.D., Remijan, P.W., & Blondeau, P. Acuity perimetry. Documenta Ophthalmologica, 1981, 26, 111-117.
- Pinckers, A. Colour vision as a diagnostic aid. Documenta Ophthalmologica, 1982, 52, 393-396.
- Porac, C. Ocular dominance and suppressive processes in binocular vision. Unpublished Doctoral Dissertation, 1974.
- Porac, C., & Coren, S. Suppressive processes in binocular vision: ocular dominance and amblyopia. American Journal of Optometry and Physiological Optics, 1975, 52, 651-657.
- Porac, C., & Coren, S. The dominant eye. Psychological Bulletin, 1976, 83, 880-897.
- Rayner, K., & Bertera, J.H. Reading without a fovea. Science, 1979, 206, 468-469.
- Regan, D. Detection and quantification of neuro-ophthalmological abnormalities using psychophysical measures of visual delay and temporal resolution. In Sokol, S. (Ed.) Electrophysiology and psychophysics: their use in ophthalmic diagnosis. Boston: Little, Brown & Company, 1980.
- Regan, D., Raymond, J., Ginsburg, A.P., & Murray, T.J. Contrast sensitivity, visual acuity and the discrimination of Snellen letters in multiple sclerosis, Brain, 1981, 104, 333-350.
- Regan, D., Silver, R., & Murray, T.J. Visual acuity and contrast

- sensitivity in multiple sclerosis - hidden visual loss: an auxiliary diagnostic test. Brain, 1977, 100, 563-579.
- Regan, D., Whitlock, J.A., Murray, T.J., & Beverley, K.I. Orientation-specific losses of contrast sensitivity in multiple sclerosis. Investigative Ophthalmology and Visual Science, 1980, 19(3), 324-328.
- Romano, P.E., Romano, J.A., & Puklin, J.E. Stereoacuity development in children with normal binocular single vision. American Journal of Ophthalmology, 1975, 79(6), 966-971.
- Rushton, D. Use of the Pulfrich pendulum for detecting abnormal delay in the visual pathway in multiple sclerosis. Brain, 1975, 98, 283-296.
- Schapiro, M. Amblyopia, New York: Chilton Book Company, 1971.
- Schmeisser, E.T., & Dawson, W.W. Dichoptic interaction of harmonically related spatial and temporal frequencies. Documenta Ophthalmologica, 1982, 53, 37-50.
- Senden, von M. Raum-und Gestaltauffassung bei operierten Blindgeborenen vor und nach der Operation. Leipzig: Varth, 1932.
Cited in Hebb, D.O. The organization of behavior. New York: Wiley, 1949.
- Sherrington, C.S. On binocular flicker and the correlation of activity of "corresponding" retinal points. British Journal of Psychology, 1904-05, 1, 26-60.
- Sjostrand, J. Contrast sensitivity in macular disease using a small-field and a large-field TV-system. Acta Ophthalmologica, 1979, 57, 832-846.

- Skalka, H.W. Comparison of Snellen acuity, VER acuity, and Arden grating scores in macular and optic nerve diseases. British Journal of Ophthalmology, 1980, 64(1), 24-29.
- Sokol, S. Visually evoked potentials: theory, techniques and clinical applications. Survey of Ophthalmology, 1976, 21(1), 18-44.
- Sperduto, R.D., & Seigel, D. Senile lens and macular changes in a population-based sample. American Journal of Ophthalmology, 1980, 90, 86-91.
- Sperling, G. Binocular vision: a physical and a neural theory. American Journal of Psychology, 1970, 83, 461-534.
- Staller, J.D., Lappin, J.S., & Fox, R. Stimulus uncertainty does not impair stereopsis. Perception and Psychophysics, 1980, 27(4), 361-367.
- Tanner, W.P. Adaptation of vision following cataract removal. New Outlook, 1971 (November), 281-286.
- Thiel, R. Atlas of diseases of the eye. Vol. 2, New York: Elsevier Publishing Co., 1963.
- Vaegan, & Halliday, B.L. A forced-choice test improves clinical contrast sensitivity testing. British Journal of Ophthalmology, 1982, 66, 477-491.
- Valvo, A. Sight restoration after long-term blindness: the problems and behavioral patterns of visual rehabilitation. New York: American Foundation for the Blind, 1971.
- Vassiliou, G., Simonetos, G., & Kastrantas, A. Colour vision deficiencies in young diabetics. Modern Problems in Ophthalmology, 1976, 17, 299-301.

- Vaughan, D., & Asbury, T. General ophthalmology (8th Edition) Los Altos: Lange Medical Publications, 1977.
- Virsu, V., & Rovamo, J. Visual resolution, contrast sensitivity, and the cortical magnification factor. Experimental Brain Research, 1979, 37, 475-494.
- Wanger, P., & Nilsson, B.Y. Visual evoked responses to pattern-reversal stimulation in patients with amblyopia and/or defective binocular functions. Acta Ophthalmologica, 1978, 56, 617-627.
- Weekers, R. Critical frequency of fusion: clinical applications. In Sorsby, A. (Ed.) Modern trends in ophthalmology. London: Butterworth & Co. (Publishers) Ltd., 1955.
- Wesson, M.D., & Loop, M.S. Temporal contrast sensitivity in amblyopia. Investigative Ophthalmology and Visual Science, 1982, 22, 98-102.
- Wod, G.C., & Long, W.F. Use of contrast sensitivity function to measure residual vision following a demyelinating disease. Australian Journal of Optometry, 1979, 62(7), 293-295.
- Yamazaki, H., Adachi-Usami, E., & Chiba, J. Contrast thresholds of diabetic patients determined by VECP and psychophysical measurements. Acta Ophthalmologica, 1982, 60, 386-392.
- Yanko, L. Diabetic retinopathy in visual deprivation: a case report. Acta Ophthalmologica, 1980, 58, 283-287.
- Zimmern, R.L., Campbell, F.W., & Wilkinson, I.M.S. Subtle disturbances of vision after optic neuritis elicited by studying contrast sensitivity. Journal of Neurology, Neurosurgery, and Psychiatry, 1979, 42, 407-417.

Zwas, F., Weiss, H., & McKinnon, P. Spectral sensitivity measurements
in early diabetic retinopathy. Ophthalmic Research, 1980, 12(2),
87-96.

Reference Note

As was mentioned previously, the four categories of visual impairment which were examined in the present study are quite broad and include a wide variety of diseases. It seems appropriate therefore, to specify which particular visual disorders would allow one to best address the present problems.

Opacity problems, for instance, may occur on the cornea, in the lens, in the vitreous, and/or in the retina. Corneal problems may involve scars following injuries to the surface of the eye, corneal vascularization or edema, and pigmentation of the cornea which is sometimes due to blood staining following injury or surgery. Vitreous opacities often consist of cell aggregates, coagulated exudate and fibrin, and strands of degenerated vitreous body. They may sometimes be due to exogenous material such as parasites or foreign bodies. Vitreous opacities give rise to symptoms of floaters which move about in the field of vision. Similarly, hemorrhages, exudates, cotton-wool patches, edema, microaneurysms, and tissue proliferation in the sensory retina cause loss of transparency and, thus, an opacity problem (Newell, 1978; Vaughn & Asbury, 1977).

Since one would ideally attempt to avoid opacities that change location in the eye or do not cause a significant decrease in visual acuity, lens opacity or cataract was examined in this study. Since cataract formation is often asymmetrical, this furthers its potential in this type of examination. Specifically, senile or traumatic cataracts were studied in order to avoid the confounding effects of amblyopia developing in a congenital case. To further avoid any systemic or other

ocular abnormalities, cataracts due to either toxic and diabetic onset were not considered.

Disturbance of retinal function may likewise be caused by many diseases. These include congenital and developmental abnormalities, vascular disorders, inflammations, degeneration, detachment, and tumors. Many of these disorders are bilateral and symmetrical, which excluded them from this study, and some generate additional involvement of the other visual structures. Two conditions which are either unilateral or asymmetrical in a considerable number of cases are central retinal vein occlusion and macular degeneration. The former is a typically unilateral condition which causes a slow, painless loss of vision. In this disorder, the retinal veins are dilated and tortuous and retinal hemorrhage is possible (Vaughn & Asbury, 1977). Central retinal or macular degeneration is also commonly unilateral, or at least asymmetrical, and is associated with decreased blood supply from the choriocapillaris. This disorder may be divided into two major categories. The first is the "dry", atrophic degeneration where there is a gradual visual loss with a stabilized maintenance of residual peripheral vision. The second type is the hemorrhagic variety which is often associated with problems of retinal edema and microaneurysms in the retina.

Diseases of the optic nerve are particularly difficult to study if one is to avoid concurrent systemic or generalized central nervous system involvement. Multiple causative factors are again apparent, with optic nerve damage being possibly due to developmental anomalies, optic neuritis, papilledema, atrophy, or optic nerve tumors (Newell, 1978).

Optic neuritis, which was examined in the present study may be caused by demyelination, pressure on the optic nerves, inflammation, or any number of other factors. These disorders may display hyperemia of the disk, distention of the large retinal veins, retinal edema, and flame-shaped hemorrhages in the nerve fiber layer of the retina. Often, however, one sees a clear retina and, although it is usually concomitant with generalized demyelinating disease, one can often show no central visual disorders.

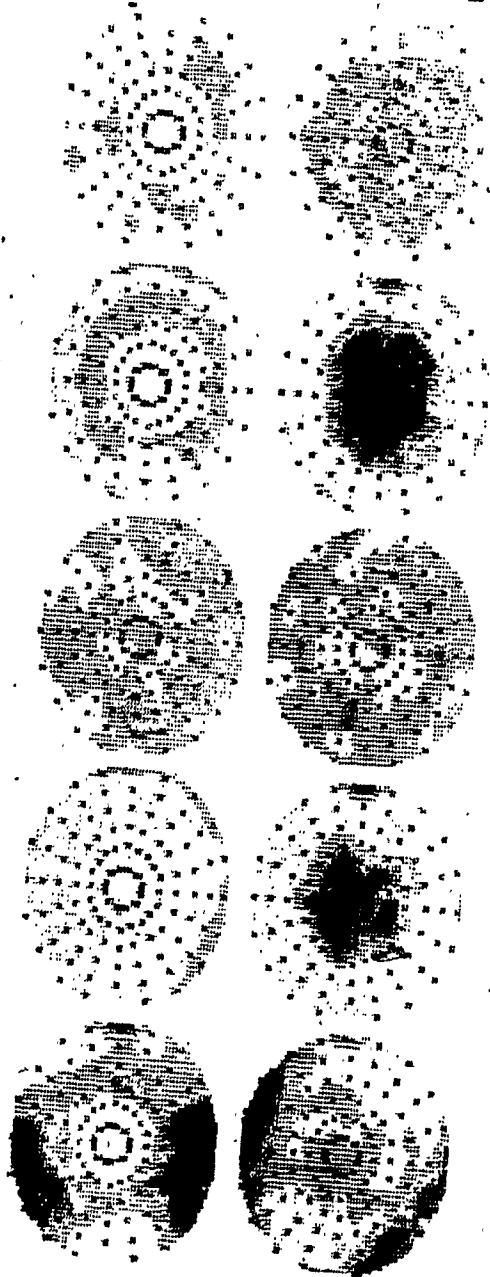
Finally, in the amblyopic category, the examination will be restricted to amblyopia caused by anisometropic conditions or by strabismus. Anisometropia is a condition which exists when there is a significant difference in the refractive error of the eyes.

Appendix A

Multi-flash maps - Amblyopia.....	98
Multi-flash maps - Cataract.....	99
Multi-flash maps - Optic Neuritis.....	100
Multi-flash maps - Macular Degeneration.....	101
Multi-flash maps - Normals.....	102

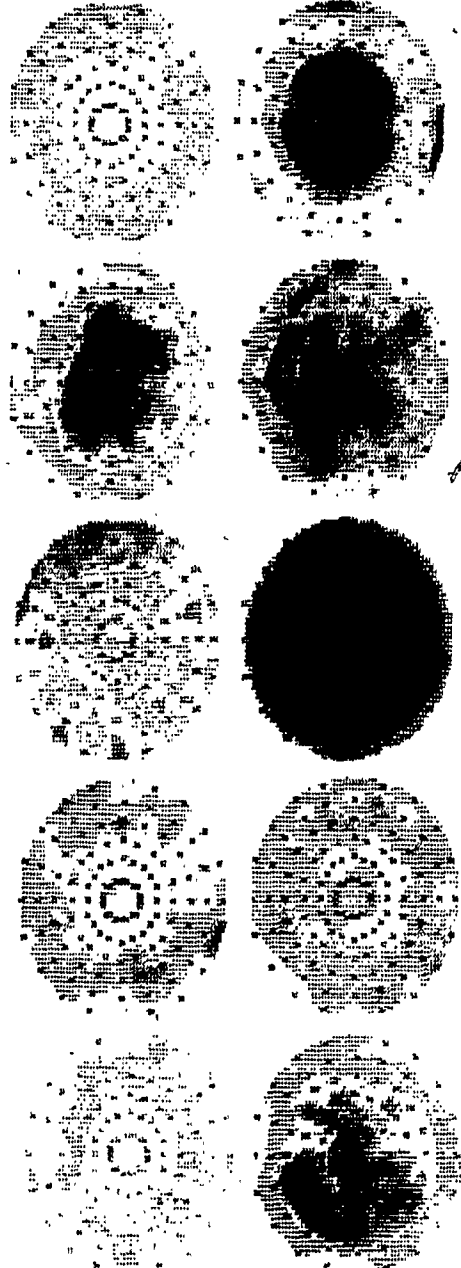
AMBLYOPIA

STRONG EYE WEAK EYE



CATARACT

STRONG EYE WEAK EYE



OPTIC NERVE DISORDER

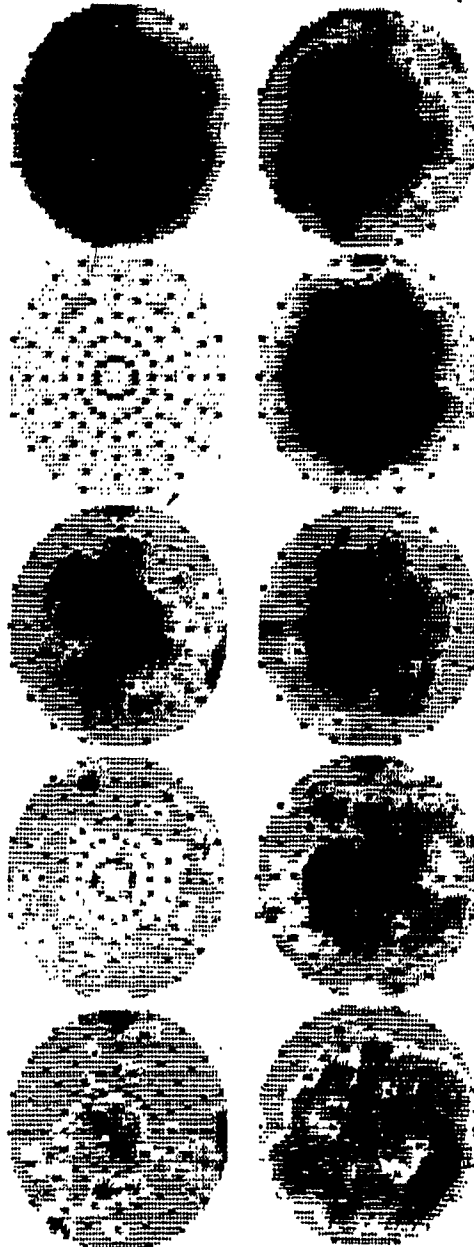
STRONG EYE

WEAK EYE



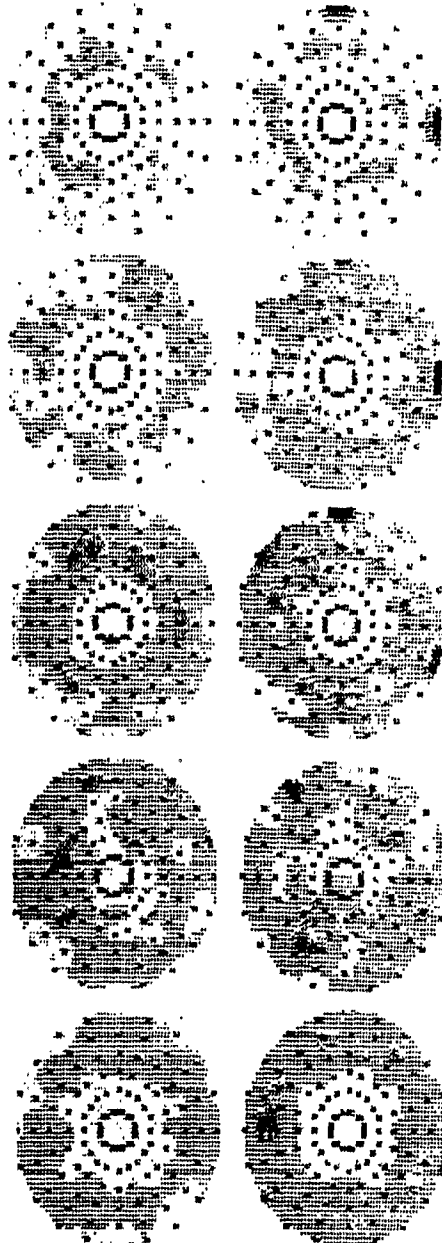
MACULAR DEGENERATION

STRONG EYE WEAK EYE



NORMAL

STRONG EYE WEAK EYE

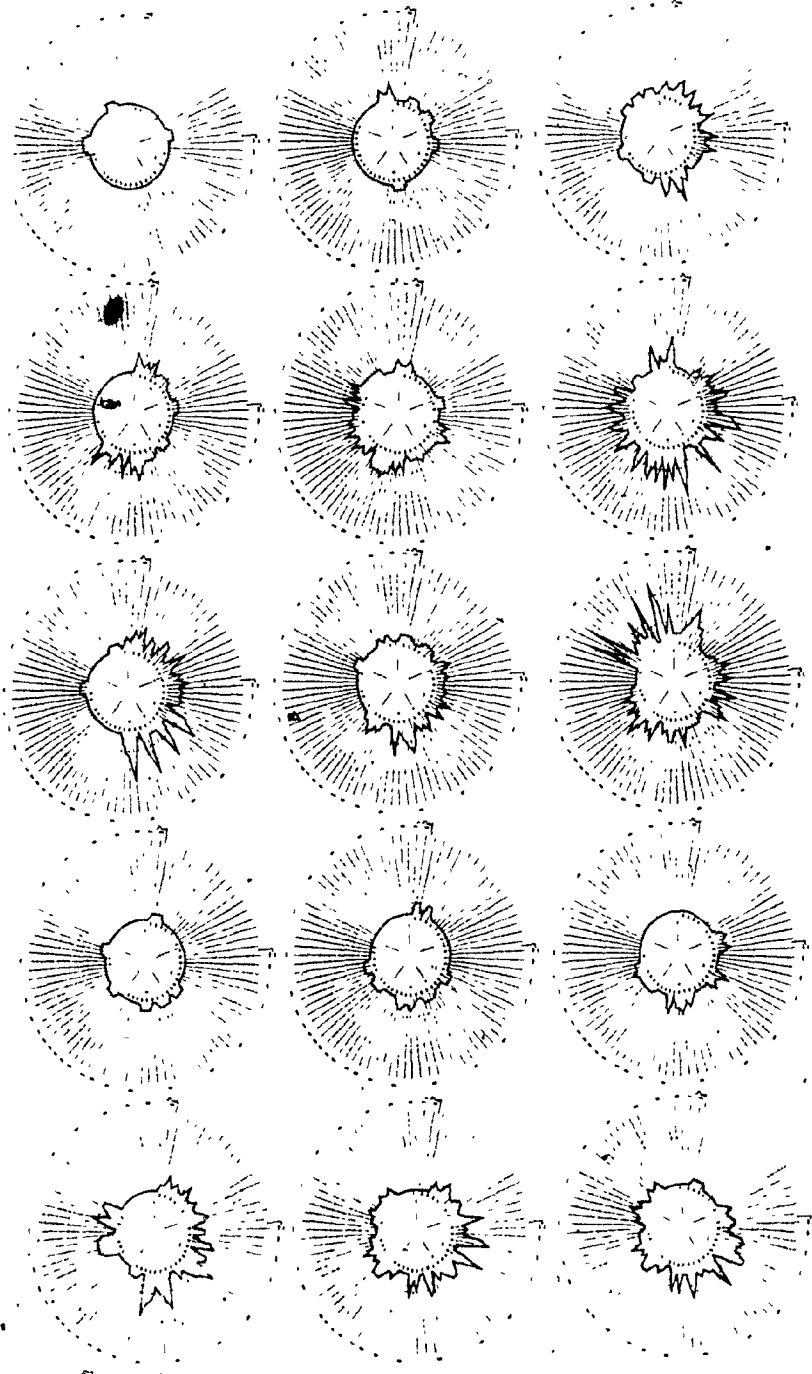


Appendix B

Farnsworth-Munsell charts - Amblyopia.....	105
Farnsworth-Munsell charts - Cataract.....	106
Farnsworth-Munsell charts - Optic Neuritis.....	107
Farnsworth-Munsell charts - Macular Degeneration.....	108
Farnsworth-Munsell charts - Normals.....	109

AMBLYOPIA

BINOCULAR STRONG EYE WEAK EYE



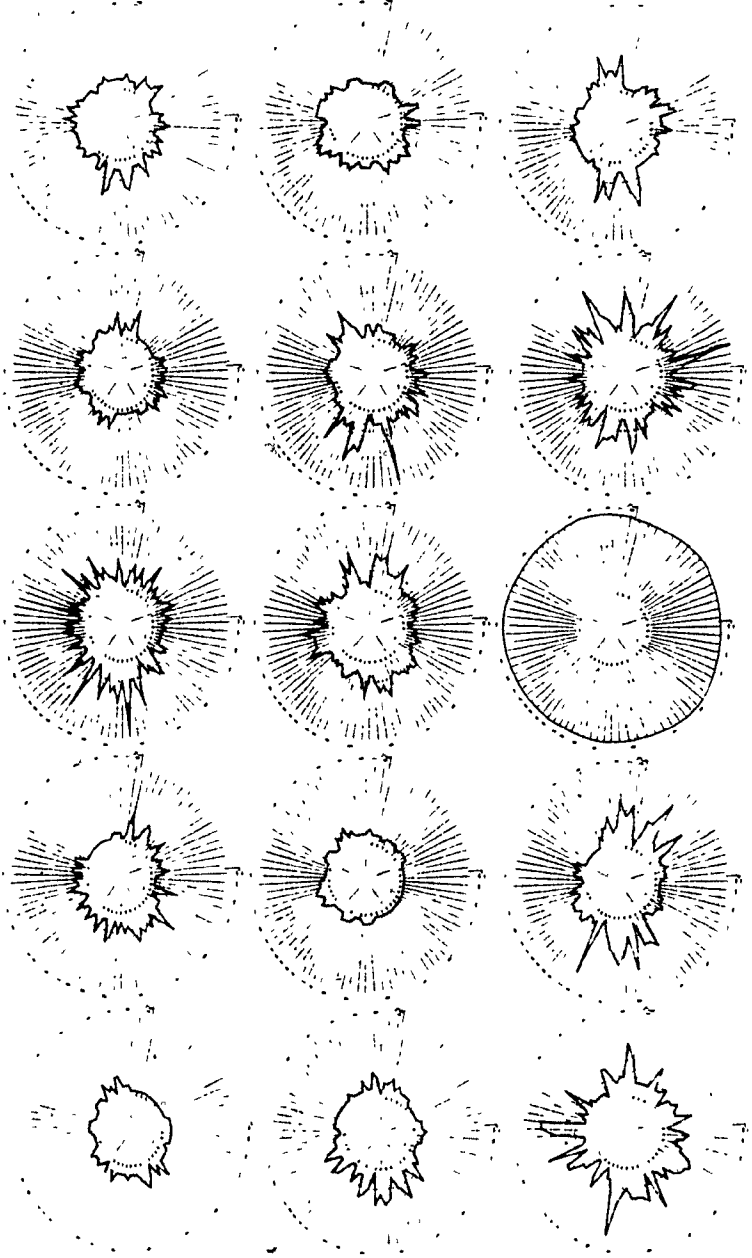


CATARACT

BINOCULAR

STRONG EYE

WEAK EYE

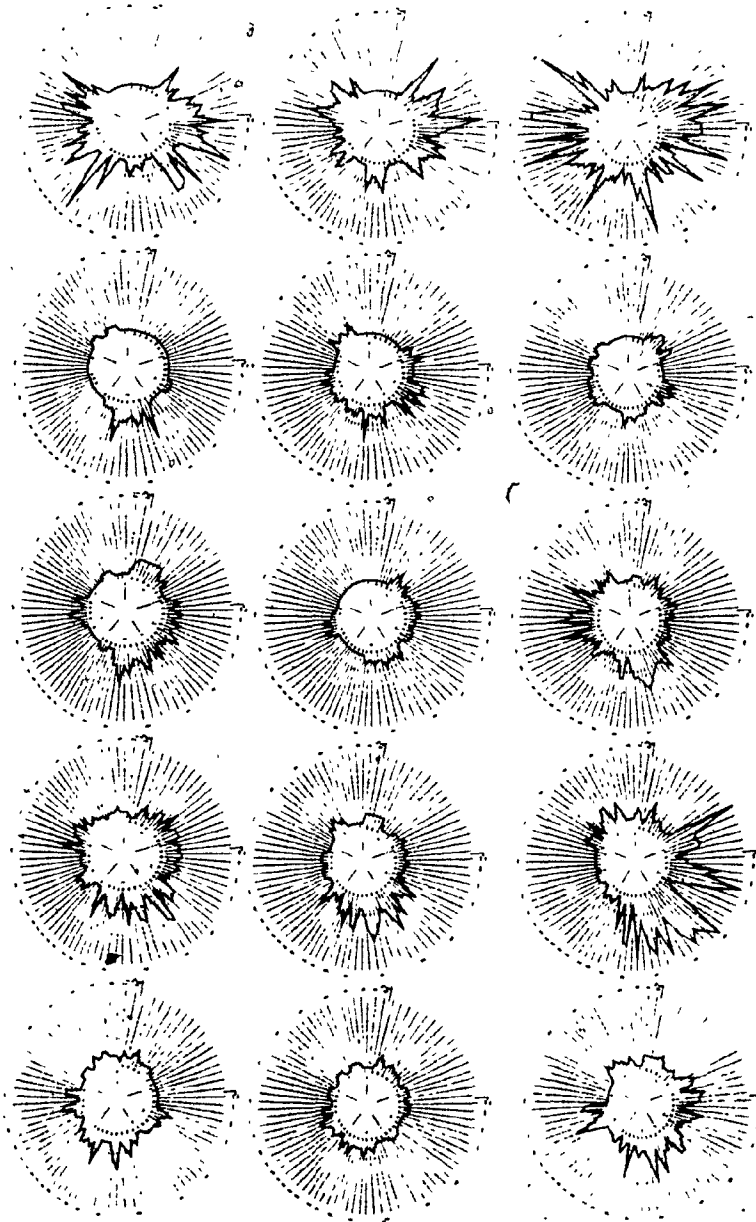


OPTIC NERVE DISORDER

BINOCULAR

STRONG EYE

WEAK EYE

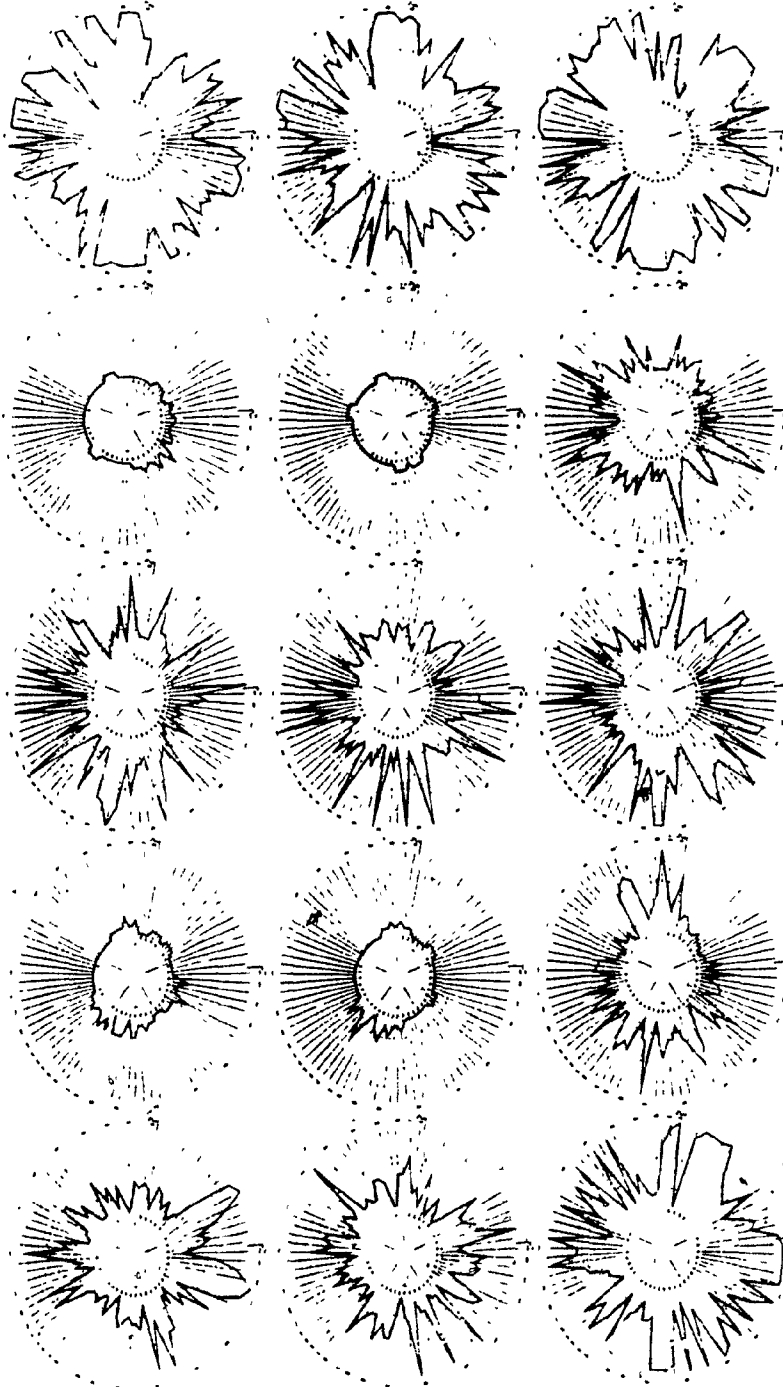


MACULAR DEGENERATION

BINOCULAR

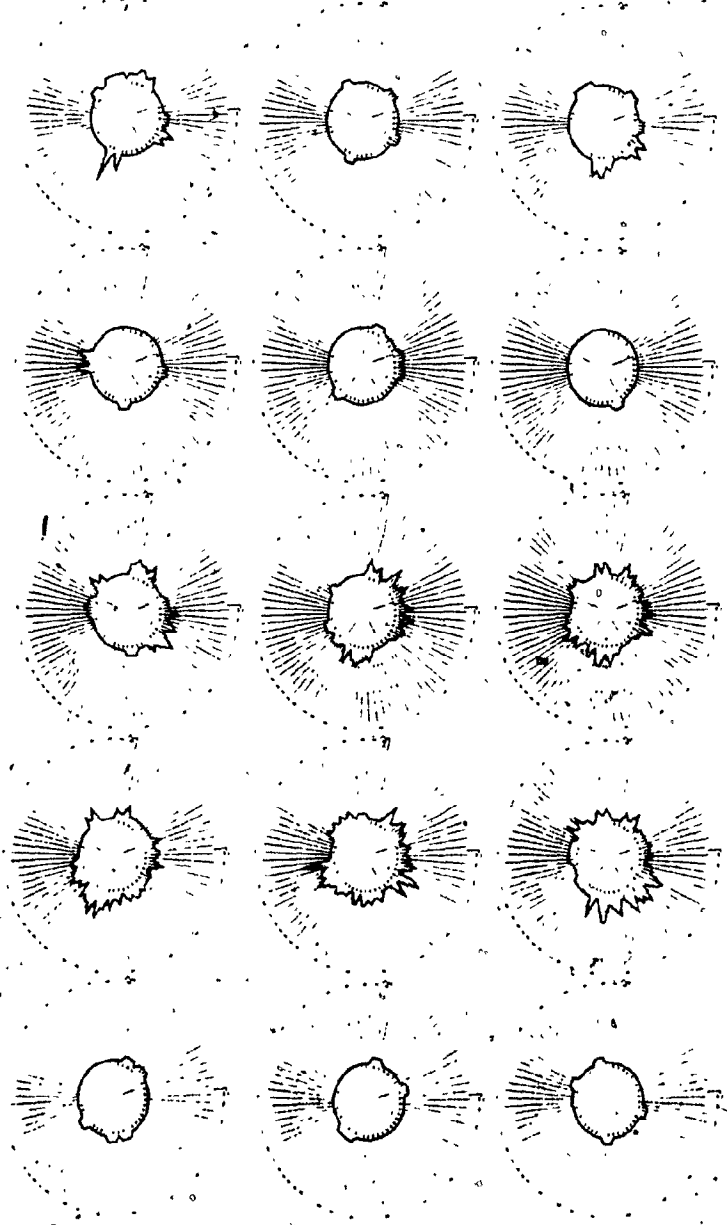
STRONG EYE

WEAK EYE



NORMAL

BINOCULAR STRONG EYE WEAK EYE



Appendix C

Source Tables for Analysis of Variance - Experiment 1.....	111
Source Tables for Analysis of Variance - Experiment 2.....	112
Source Tables for Analysis of Variance - Experiment 3.....	113
Source Tables for Analysis of Variance - Experiment 4.....	115
Source Tables for Analysis of Variance - Experiment 5.....	119
Source Tables for Analysis of Variance - Experiment 6.....	120

Distance Acuity - Amblyopia

SOURCE	SS	df	MS	F
Condition	106.01	3	35.34	11.22*
Subjects	8.80	4		
Error	37.8	12	3.15	

$p < .01$

Distance Acuity - Cataract

SOURCE	SS	df	MS	F
Condition	120.53	3	40.17	10.51*
Subjects	18.54	4		
Error	45.88	12	3.82	

$p < .01$

Distance Acuity - Macular Degeneration

SOURCE	SS	df	MS	F
Condition	181.53	3	60.51	15.03*
Subjects	70.55	4		
Error	48.32	12	4.03	

$p < .01$

Distance Acuity - Normals

SOURCE	SS	df	MS	F
Condition	.16	3	.05	3.77
Subjects	.11	4		
Error	.17	12	.01	

$p < .05$

Near Acuity - Amblyopia

SOURCE	SS	df	MS	F
Condition	23.19	3	7.73	3.79
Subjects	8.58	4		
Error	24.44	12	2.04	

$p < .01$

Near Acuity - Macular Degeneration

SOURCE	SS	df	MS	F
Condition	27.47	3	9.16	4.88
Subjects	4.87	4		
Error	22.57	12	1.88	

$p < .01$

Spatial Frequency (8.00 cpd) - Amblyopia

SOURCE	SS	df	MS	F
Condition	2.42	2	1.21	5.47
Subjects	.82	4		
Error	1.77			

$p < .01$

Spatial Frequency (1.00 cpd) - Cataract

SOURCE	SS	df	MS	F
Condition	1.35	2	.67	6.20
Subjects	3.20	4		
Error	.87	8	.11	

$p < .01$

Spatial Frequency (1.68 cpd) - Cataract

SOURCE	SS	df	MS	F
Condition	1.51	2	.75	10.53
Subjects	3.80	4		
Error	.57	8	.07	

$p < .05$

Spatial Frequency (2.83 cpd) - Cataract

SOURCE	SS	df	MS	F
Condition	1.90	2	.95	8.56
Subjects	2.81	4		
Error	.89	8	.11	

$p < .01$

Spatial Frequency (4.76 cpd) - Cataract

SOURCE	SS	df	MS	F
Condition	1.50	2	.75	5.61
Subjects	2.66	4		
Error	1.07	8	.13	

$p < .01$

Spatial Frequency (8.00 cpd) - Cataract

SOURCE	SS	df	MS	F
Condition	1.44	2	.72	7.60
Subjects	1.50	4		
Error	.76	8	.09	

$p < .01$

Spatial Frequency (4.76) - Optic Neuritis

SOURCE	SS	df	MS	F
Condition	.52	2	.26	5.67
Subjects	1.94	4		
Error	.36	8	.04	

$p < .01$

Temporal Frequency (0Hz) - Cataract

SOURCE	SS	df	MS	F
Condition	2.12	2	1.06	10.59
Subjects	3.16	4		
Error	.80	8	.10	

$p < .05$

Temporal Frequency (1Hz) - Cataract

SOURCE	SS	df	MS	F
Condition	2.09	2	1.04	7.97
Subjects	3.13	14		
Error	1.05	8	.13	

$p < .01$

Temporal Frequency (2Hz) - Cataract

SOURCE	SS	df	MS	F
Condition	1.78	2	.89	7.91
Subjects	2.57	4		
Error	.90	8	.11	

$p < .01$

Temporal Frequency (4Hz) - Cataract

SOURCE	SS	df	MS	F
Condition	1.86	2	.93	11.87
Subjects	2.35	4		
Error	.62	8	.08	

$p < .05$

Temporal Frequency (8Hz) - Cataract

SOURCE	SS	df	MS	F
Condition	1.84	2	.92	10.75
Subjects	2.66	4		
Error	.69	8	.08	

$p < .05$

Temporal Frequency (16Hz) - Cataract

SOURCE	SS	df	MS	F
Condition	1.76	2	.88	10.37
Subjects	2.48	4		
Error	.68	8	.08	

$p < .05$

Temporal Frequency (32Hz) - Cataract

SOURCE	SS	df	MS	F
Condition	1.69	2	.84	10.29
Subjects	3.07	4		
Error	.66	8	.08	

$p < .05$

Temporal Frequency (1Hz) - Optic Neuritis

SOURCE	SS	df	MS	F
Condition	.43	2	.21	5.52
Subjects	.72	4		
Error	.31	8	.04	

$p < .01$

Temporal Frequency (4Hz) - Optic Neuritis

SOURCE	SS	df	MS	F
Condition	.48	2	.24	11.91
Subjects	.94	4		
Error	.16	8	.02	

$p < .05$

Temporal Frequency (8Hz) - Optic Neuritis

SOURCE	SS	df	MS	F
Condition	.83	2	.41	7.22
Subjects	.55	4		
Error	.46	8	.06	

$p < .01$

Temporal Frequency (32Hz) - Optic Neuritis

SOURCE	SS	df	MS	F
Condition	.33	2	.16	4.76
Subjects	1.32	4		
Error	.27	8	.03	

$p < .01$

Depth Perception - Optic Neuritis

SOURCE	SS	df	MS	F
Condition	37.40	3	12.47	3.78
Subjects	22.02	4		
Error	39.54	12	3.29	

$p < .05$

Depth Perception - Macular Degeneration

SOURCE	SS	df	MS	F
Condition	91.19	3	30.39	6.47
Subjects	33.08	4		
Error	56.34	12	4.69	

$p < .05$

Depth Perception - Normal

SOURCE	SS	df	MS	F
Condition	22.50	3	7.50	4.61
Subjects	8.43	4		
Error	19.53	12	1.63	

$p < .01$

Farnsworth-Munsell-100 - Amblyopia

SOURCE	SS	df	MS	F
Condition	7454.53	2	3727.27	5.49
Subjects	24484	4		
Error	5430.80	8	678.85	

$p < .05$

Farnsworth-Munsell-100 - Optic Neuritis

SOURCE	SS	df	MS	F
Condition	26477.2	2	13238.6	7.53
Subjects	84516.27	4		
Error	14070.13	8	1758.77	

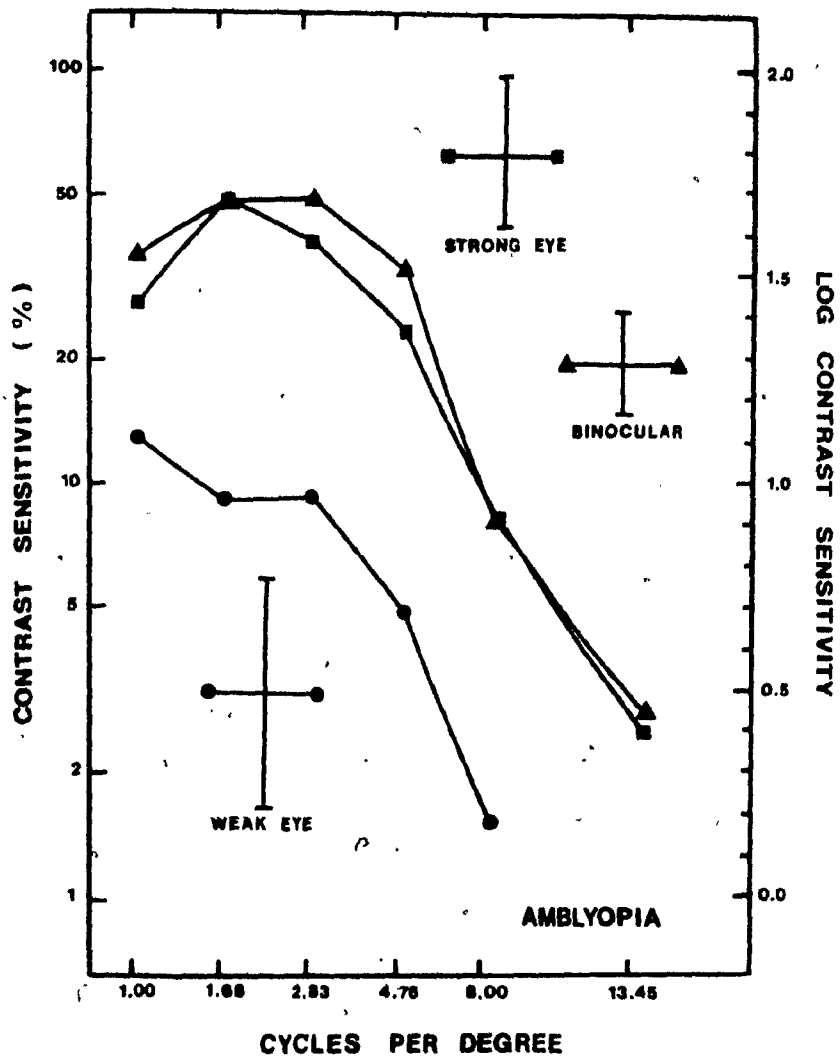
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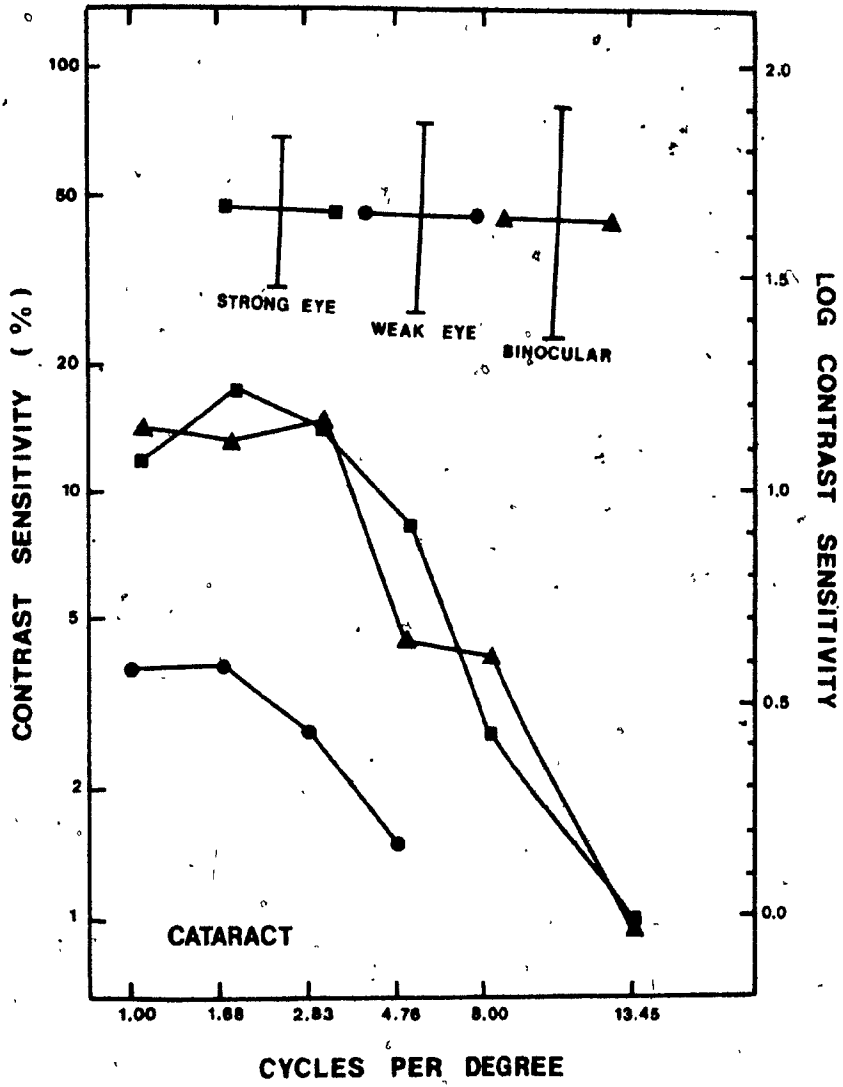
Farnsworth-Munsell-100 - Macular Degeneration

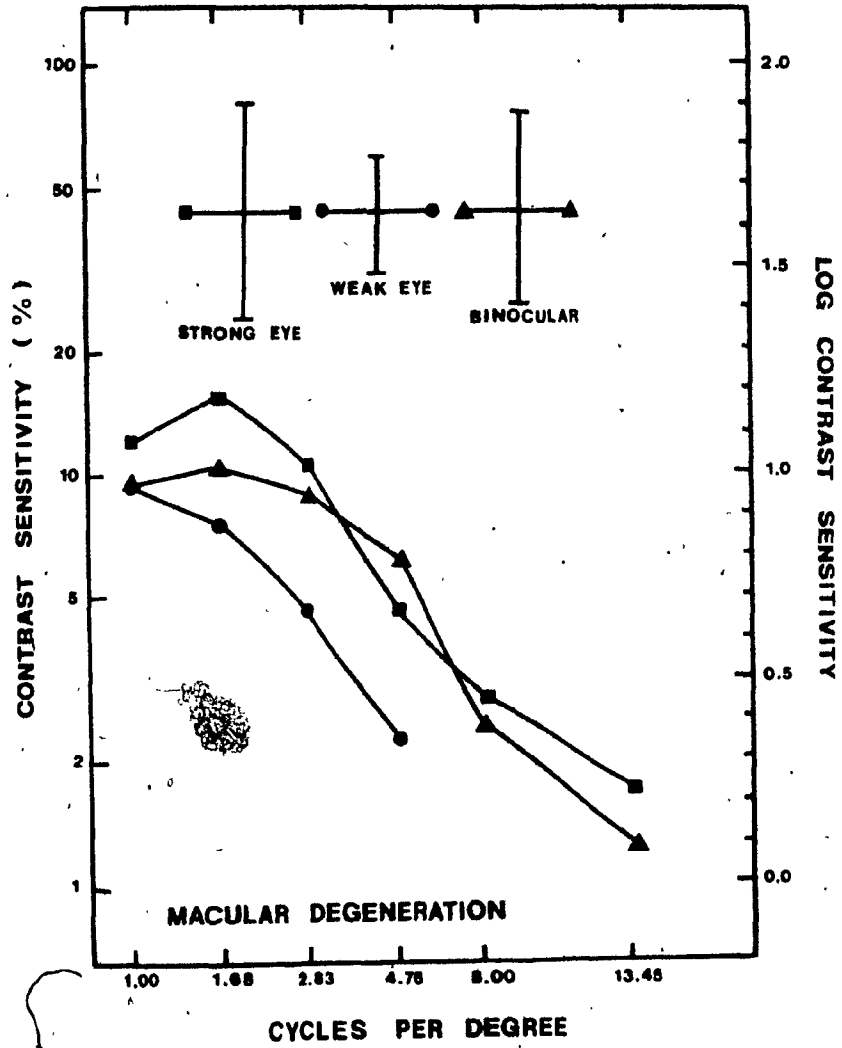
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Condition	211930.53	2	105965.27	11.32
Subjects	1599585.73	4		
Error	24861.47	8	9357.68	

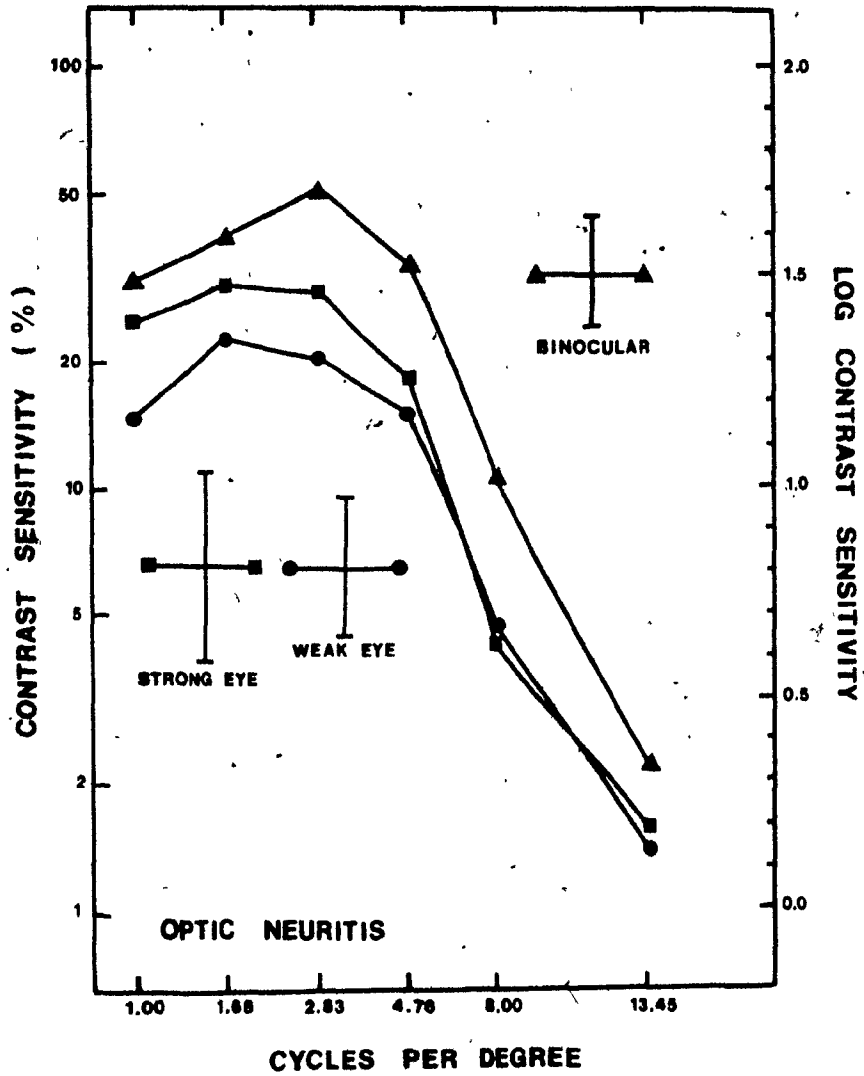
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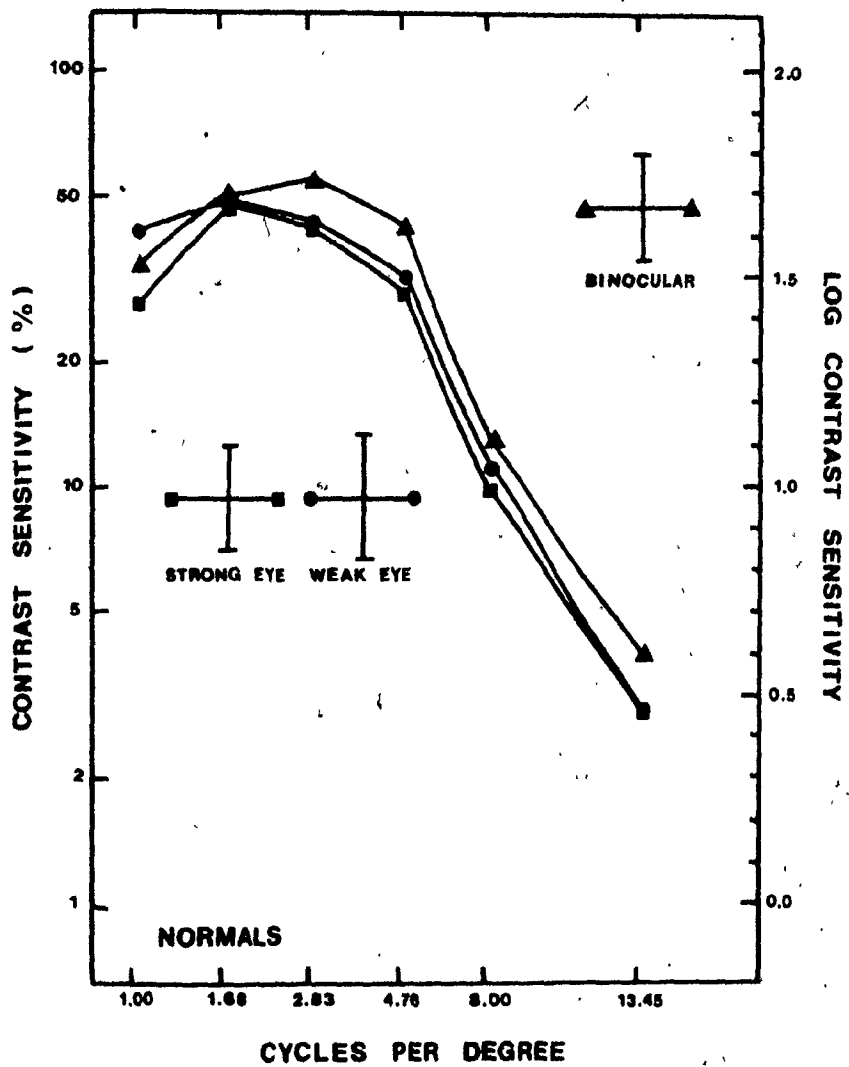
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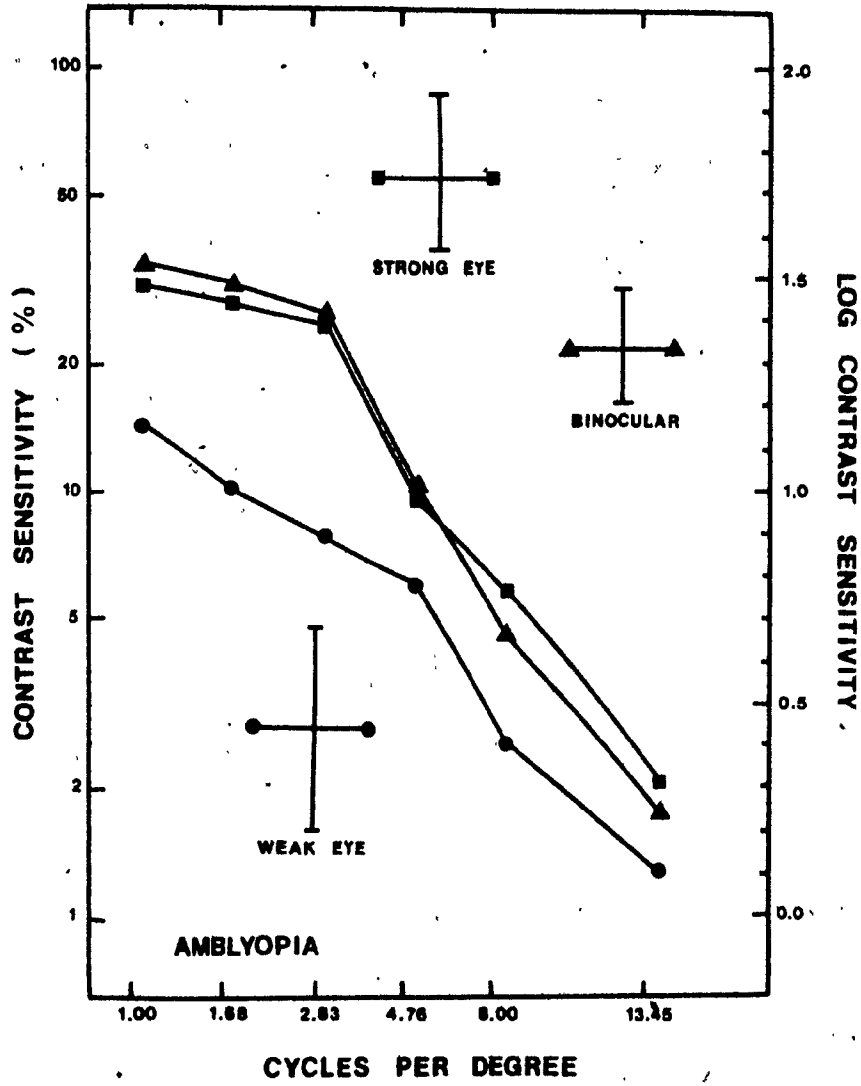


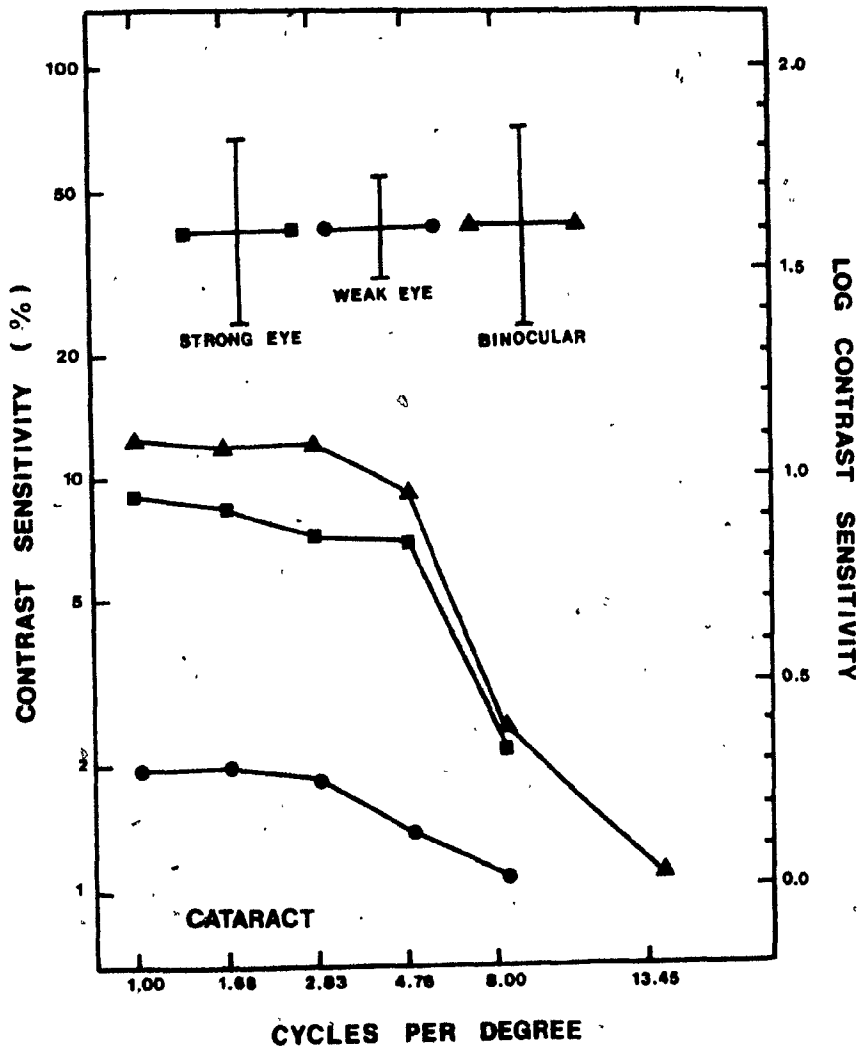


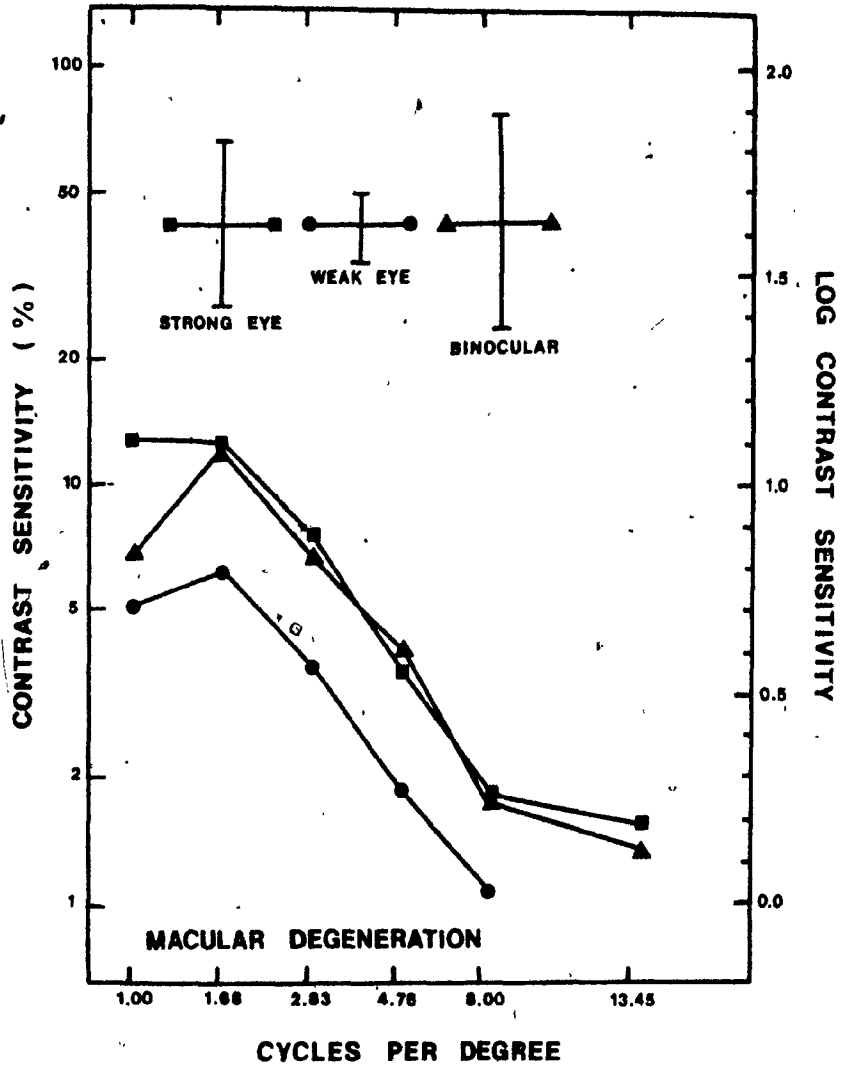


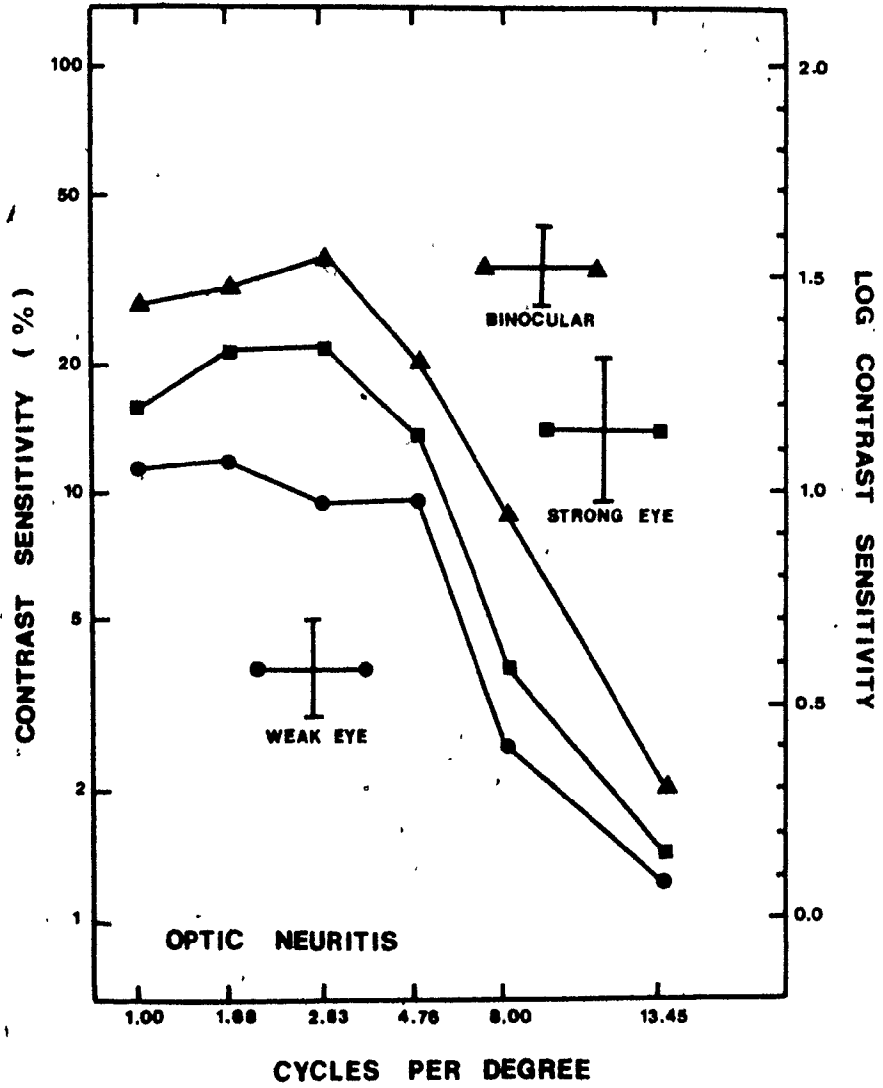




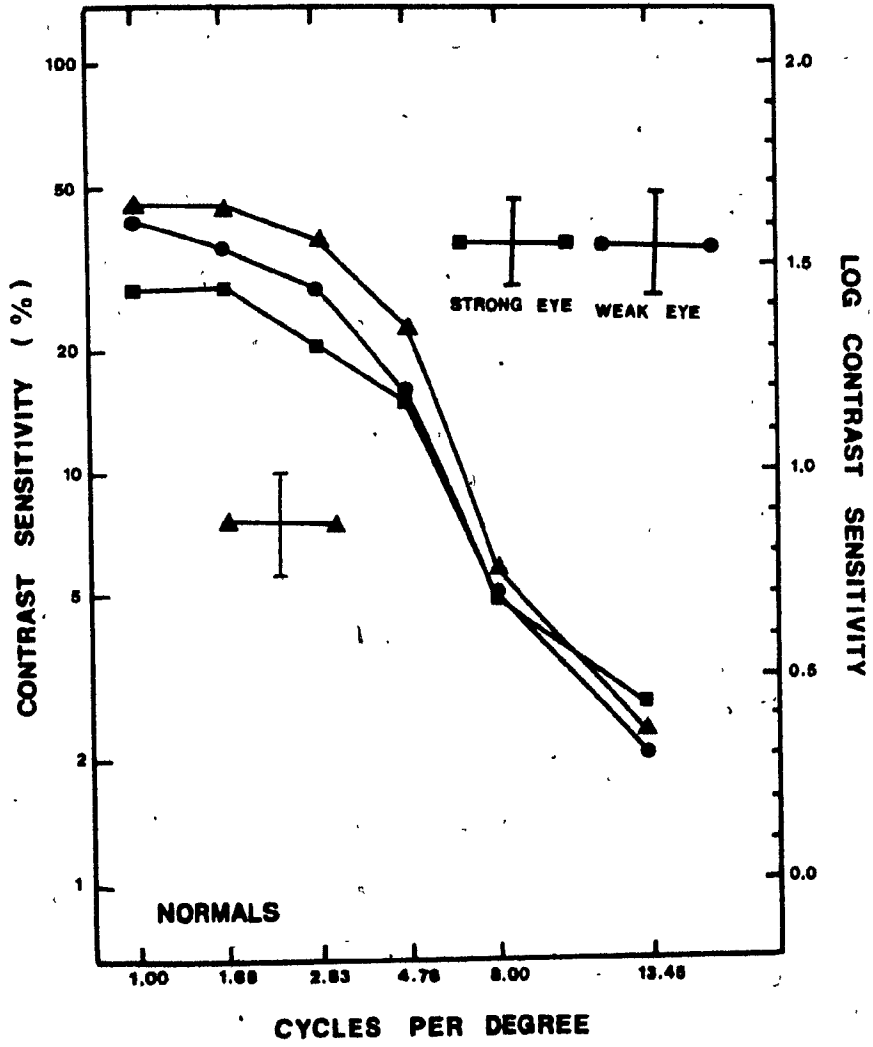


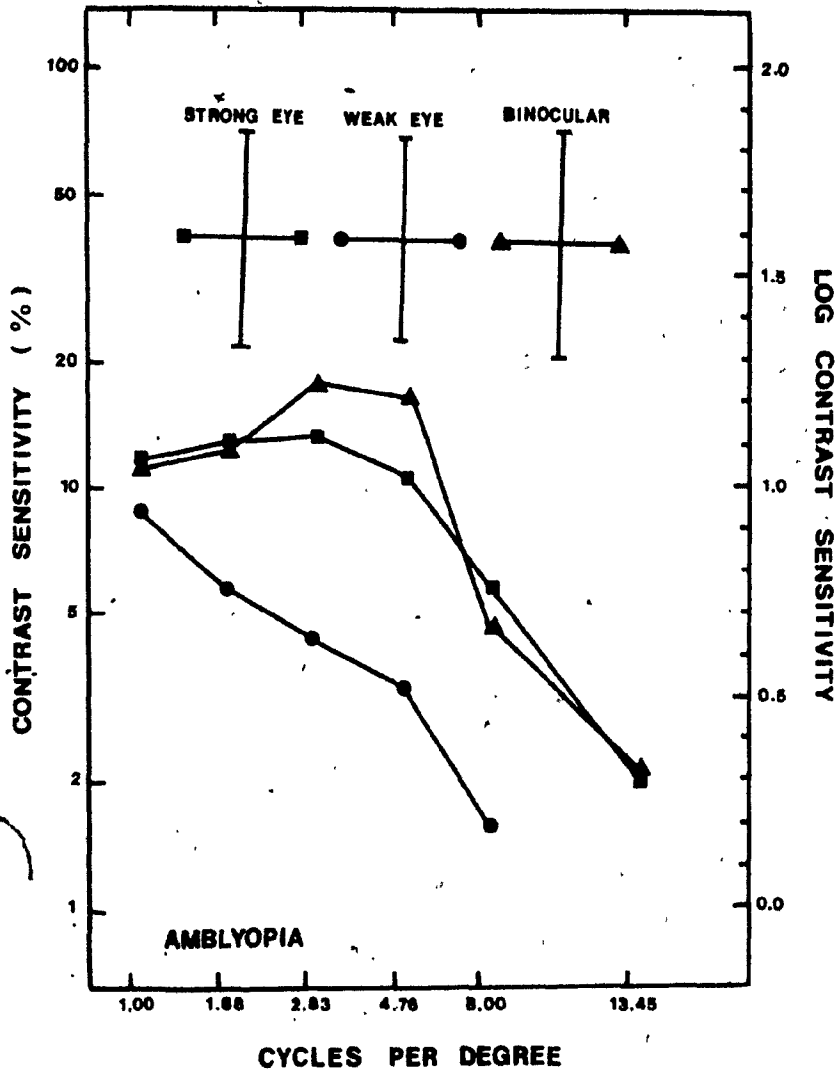


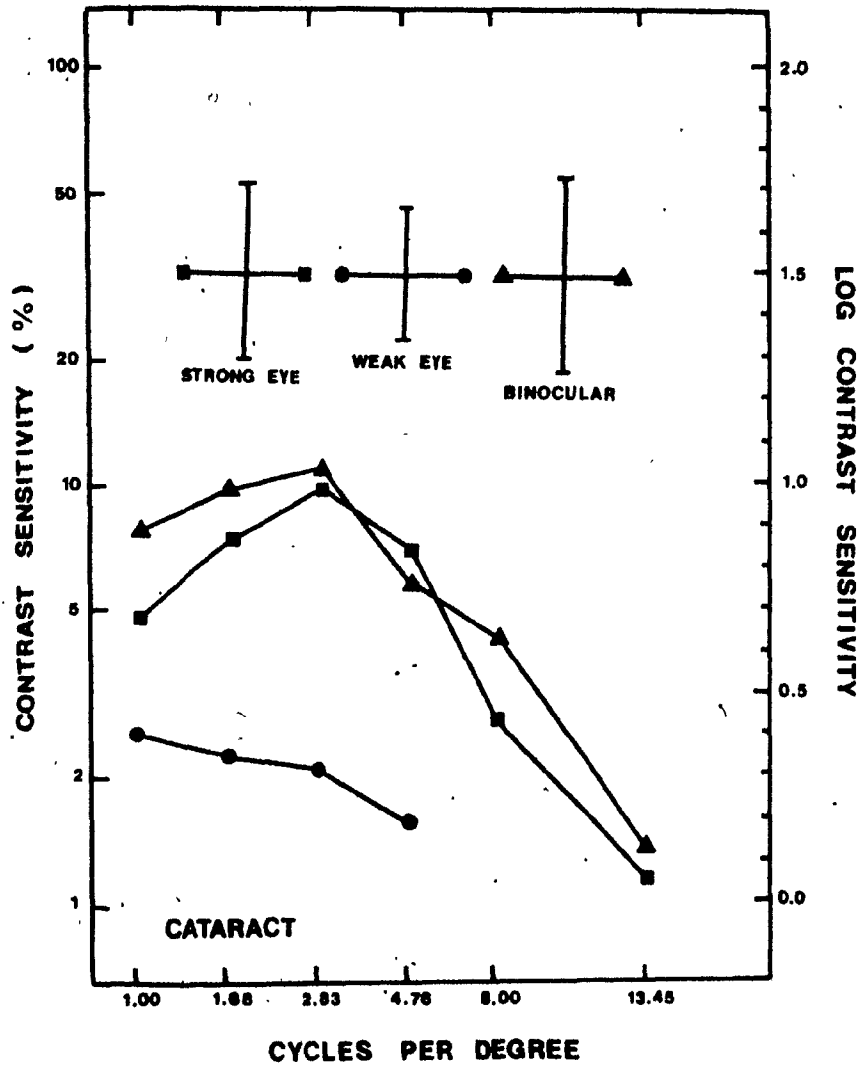


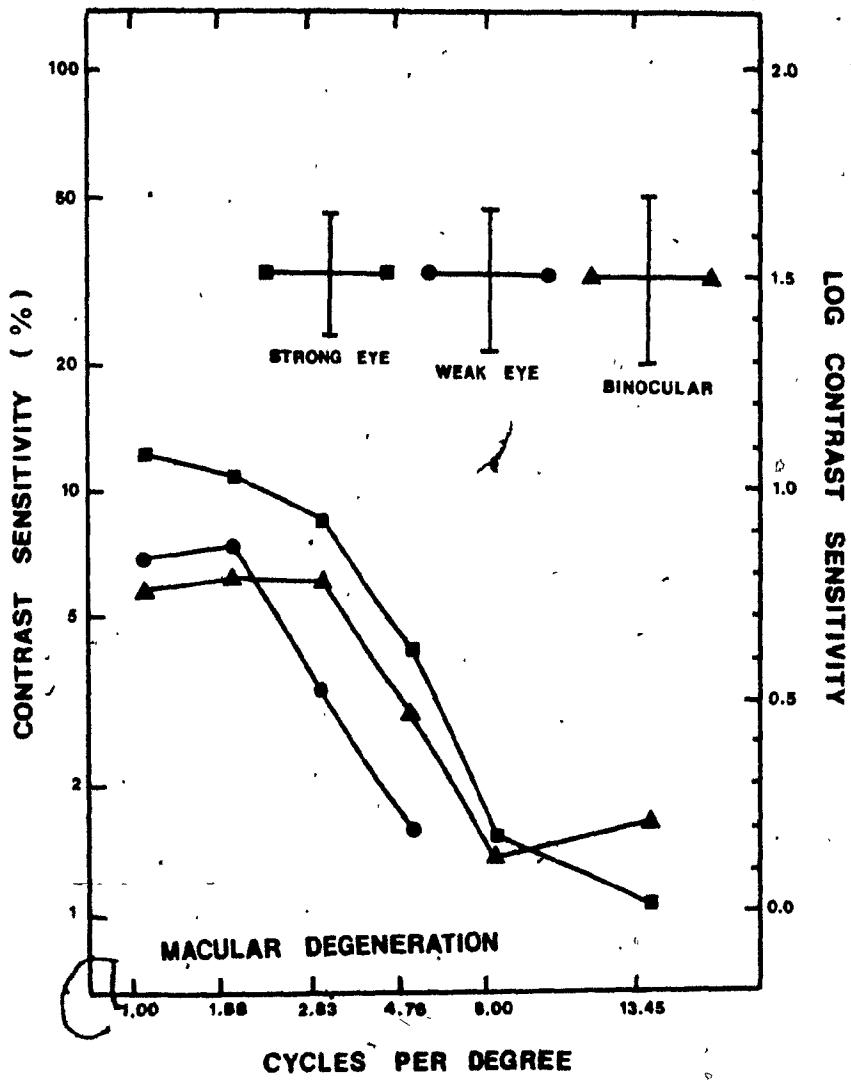


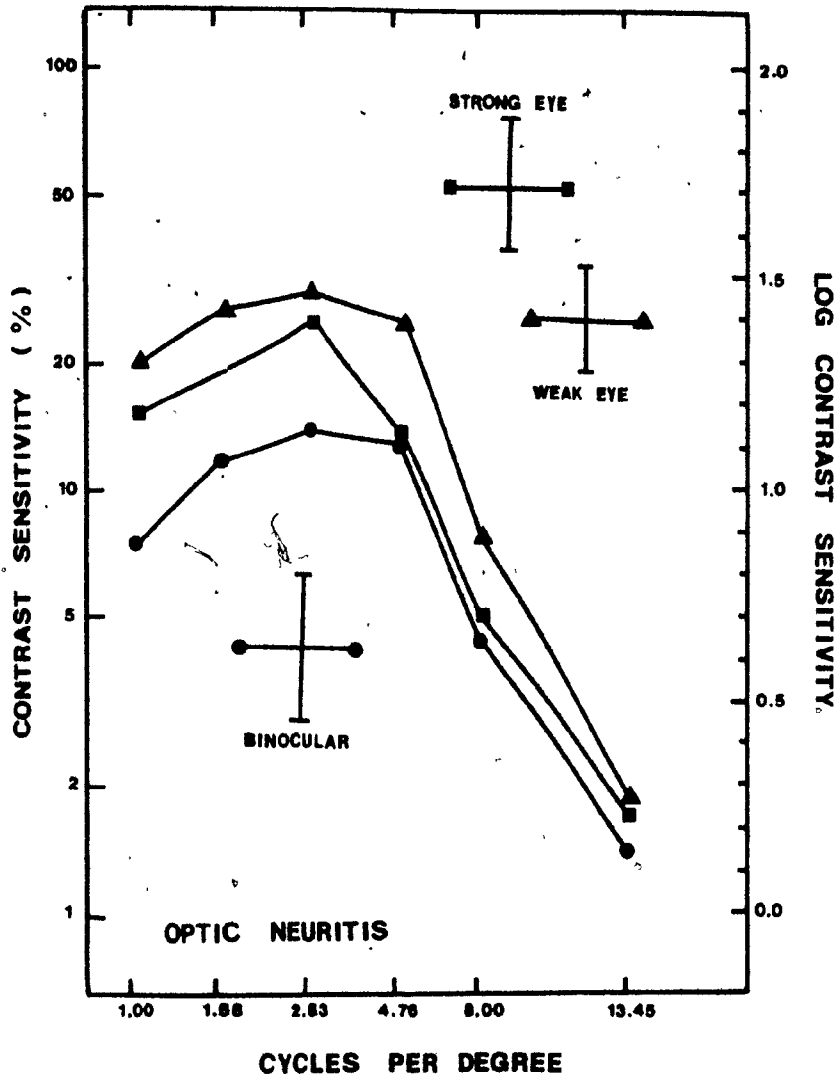
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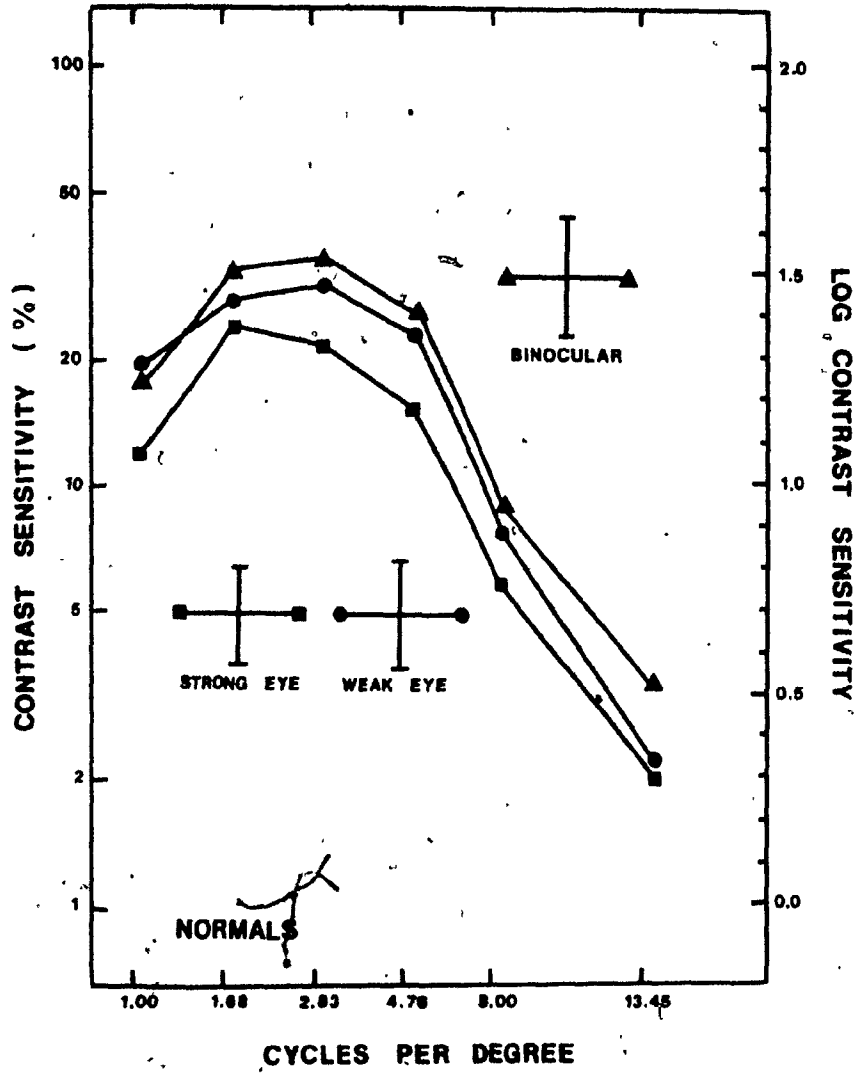












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