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AMBLYOPIA EX ANOPSIA. OBSERVATIONS ON RETINAL INHIBITION, SCOTOMA, PROJECTION, LIGHT DIFFERENCE DISCRIMINATION AND VISUAL ACUITY*†

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INTRODUCTION

Amblyopia unassociated with organic findings, usually associated with anisometropia or strabismus or both, so-called "amblyopia ex anopsia," is seen clinically in varying degrees of severity. Considering that it may be preventable, a significant percentage of the population is affected. Of 10,000 Air Corps personnel, officers and enlisted men, examined for discharge by the author, 1% showed amblyopia ex anopsia. Of this 1%, only 20% showed obvious strabismus, as judged by inspection and cover test. Of 5,000 cases, for whom glasses were prescribed at the Drew Field Eye Clinic, 4% showed amblyopia ex anopsia.

Evaluation of the condition is important to organizations needing manpower. Classification of amblyopic subjects relative to selection for type of service in the armed forces, selection for jobs in industry, rating as insurance risks and evaluation for compensation subsequent to injury, is arbitrary because there is no accurate prognosis for return of vision to

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† This thesis has been condensed by the author.

the affected eye should the good eye be injured. As a result the selection is wasteful and in many instances unfair to the patient. Judging from the high incidence of amblyopia, early treatment is altogether inadequate, a fact due in part to a lack of consciousness of the extent of the problem by ophthalmologists, and in part to insufficient emphasis to the public through the public relations facilities of ophthalmological societies.[1, 25]

With our present knowledge of the subject we are at the stage of simply cataloguing clinical observations in an orderly fashion. Some of these observations are contradictory, requiring further study and clarification. Whether or not this type of amblyopia, without demonstrable fundus pathology to explain the poor vision, originates "ex anopsia" from disuse, is not certain. Clinical consensus, based on recovery of vision following forced use of the eye, corroborates this concept. More careful analysis reveals that many amblyopic patients show no improvement in vision after occlusion, and that most of the recoveries are only partial, even after prolonged periods of forced use of the eye, indicating that the resistance to recovery is extremely variable and that simple disuse is not the only factor involved. The familial incidence sometimes encountered and the central scotoma easily demonstrated in certain cases as compared to others necessitate a more critical evaluation. One group of patients may respond to educative training combined with forced use of the eye by techniques similar to those used for treating strephosymbolia. The possibility of setting up diagnostic criteria to differentiate groups that may recover warrants repeated analyses, reviews and correlations of observations on the condition. The next step in an attempt to explain clinical concepts in the point of view of increasingly more scientific units and theories may be the application of electrophysiologic methods.

The purpose of this paper is to record observations on retinal inhibition, scotoma, retinal projection, light difference

sense (discrimination) and visual acuity in amblyopic patients. This study is based on analysis of over 100 cases of amblyopia ex anopsia, and, in addition, 30 cases of organic macular lesions accumulated during the past 5 years.

For analysis of the findings the patients will be classified into 5 clinical grades, suggested by the classification of Chavasse[2]:

1. Amblyopia associated with inability to fixate but with the eye held in a relatively centric position.
2. Amblyopia associated with eccentric fixation of the eye.
3. Amblyopia associated with strabismus and with relatively good ability to fixate.
4. Amblyopia without apparent strabismus or with inconstant strabismus, and relatively good ability to fixate.
5. Amblyopia resulting from organic macular disease.

SCOTOMA, RETINAL INHIBITION AND RETINAL PROJECTION

The nature and significance of the central scotoma in amblyopia ex anopsia has been the subject of much speculation, but few attempts at qualitative or quantitative analysis have been reported. The outstanding work done in this country is that of Evans,[3] who found a definite central scotoma in 16 selected cases, which he interpreted as being large enough to explain the reduced visual acuity. Evans used a Lloyd stereocampimeter in this work. The presence of a normal angioscotoma showed the accuracy of his field studies and precluded, in his opinion, the possibility that the central scotomata demonstrated were inhibitory, incident to retinal rivalry.[4]

Travers[5] and Chavasse[2] stress the occurrence of inhibitory scotoma associated with retinal rivalry, and questioned the significance of Evans' findings. They feel that the scotoma of amblyopia ex anopsia is an exaggerated suppression scotoma or facultative scotoma, similar to that found in strabismus at the angle of squint when both eyes are being used. Apparently the consensus at present[6] is that in some cases of

amblyopia ex anopsia the scotoma is purely functional, depending on the inhibition incident to dominance of the fixing eye, and in other cases the scotoma has become obligatory, that is, persisting when the other eye is covered. In severe cases of amblyopia ex anopsia with eccentric fixation, a scotoma is readily found, and has many characteristics of an organic scotoma.[6] In less severe cases, a central scotoma may or may not be found, and visual acuity offers only partial indication as to the presence or absence of the scotoma.

To further elucidate the nature and significance of the scotoma in amblyopia ex anopsia a number of studies herein reported were made and compared with the findings on patients with a central scotoma due to organic macular lesion. These studies involved observations on amblyopic patients of many types, and instances of macular disease. Most of the patients were adults seen in the Army. The scotoma studies were made at different times with different types or modifications of apparatus and with different purposes in mind. Recently more complete studies on a limited number of patients with different clinical degrees of amblyopia and organic macular lesions were made with the over-all picture of the problem in mind. Earlier findings were interpolated with the more complete series and the aggregate was used in drawing conclusions as to the nature of the scotoma present in amblyopia ex anopsia.

Procedure and Apparatus

1. Tangent Screen: The screen was viewed at different distances with various sized white and colored test objects, and special markers on the screen to steady fixation.

2. Lloyd Stereocampimeter: A 0.4 mm. test object subtending an angle of 7 minutes and regular Lloyd stereocampimeter charts were used. Studies were made with both eyes fixing and with the amblyopic eye alone fixing.

3. Mirror-screen Test[5]: Two black tangent screens were placed at right angles to each other. A small oval mirror was

fastened on a gooseneck stand with a swivel joint allowing the mirror to be moved and set against the side of the nose so that the screen on the side could be observed. The subject fixated an object with the normal eye behind the mirror, and the field of the amblyopic eye was then plotted. The fixation object was moved to the desired point by adjusting the mirror. By this method retinal rivalry, inhibition and projection could be studied. In cases of strabismus with abnormal retinal projection, the eyes were aligned by moving the mirror until the cover test revealed no jump of either eye to fixate.

4. Prism-pinhole Technique of McCulloch[7]: A 2.0 mm. pinhole plus a 20 diopter prism, base down, was placed in front of the fixing eye. This projected the fixation point as seen through the pinhole to an area on the tangent screen 20 prism diopters above the actual fixation point. The eye to be tested could then fix on the displaced image and the field around this image could be plotted for this eye, the test object being out of view of the eye behind the pinhole. Fixation was excellent, but the factor of binocular vision and retinal rivalry was introduced in the determination of central scotoma by this method.

5. Amblyoscope: The Worth amblyoscope, the Holmes-Brewster stereoscope, and the synoptophore with various targets were used to study retinal inhibition, projection and rivalry.

6. After-image Test of Hering: This method of checking retinal projection was used in conjunction with other methods.

7. Ophthalmoscopic Beam: The projection of a narrow ophthalmoscopic beam of light shone on different parts of the retina was used to determine retinal inhibition and projection in cases of severe amblyopia with gross scotoma.

8. Snellen Letters: The size of the scotoma was judged by the Snellen letters that could be obscured in the scotoma.

9. Prism Displacement Test: The development of this test

by the author was the reason for the beginning of this investigation and is the basic test used throughout, and so the background for the test and its application will be described.

In the Army the problem arose of properly classifying patients with poor vision in one eye, the eye appearing normal otherwise. Small macular lesions were frequently seen; anisometropia was common; minimal strabismus was not unusual, many of the patients having a history of more pronounced squint in childhood; there were many cases of amblyopia ex anopsia who apparently had straight eyes; malingering at times was in epidemic proportions. The question of whether there existed a minimal strabismus, or had been strabismus at one time, or whether an indiscernible macular lesion was present, afforded a problem that attracted the author's interest. Use of the phi phenomenon was employed in this regard and has been reported elsewhere under the title of a simple test for binocular fixation.[8]

In 1940 Verhoeff[9] discussed the phi phenomenon in relation to strabismus. This phenomenon, also referred to as beta apparent movement, is literally the seen movement of displacement under discrete successive stimulation of the retina. If two light stimuli are presented briefly and in succession to different areas of the retina, in one or both eyes or alternate eyes, an apparent movement of the light is seen in the direction of the succession. The impression is that the original light moves across the retina to become the second light, rather than the impression of two separate stimuli. Presumably interpretation of this movement is dependent on overlapping of synaptic endings at all levels of the visual pathways, so that neural excitations are aroused successively in the cells between the stimulated areas similar to that which occurs when a single stimulus actually moves across the area.

Verhoeff demonstrated that, in strabismus, no apparent movement occurs when alternate eyes are stimulated within the area of the angle of squint because all the retinal localizing projection points in this area have the same directional

value, that is, of localizing stimuli straight ahead in conjunction with the macula of the nonsquinting eye. He showed that a squint could be differentiated from a phoria by the finding that retinal projection is normal in phoria, the phi phenomenon being present, and absent in squint. He suggested that all squints have false retinal projection of some degree, that is, sufficient to obscure the phi phenomenon. This observation suggested to the author that faulty retinal projection may be the first step in retinal inhibition or suppression. A stimulus may be seen but not localized and, having no directional value, is easily lined up with the directional value of the dominant fixing eye, and diplopia is avoided. It seemed that if a stimulus presented to an amblyopic eye which was apparently fixing straight ahead in conjunction with the dominant eye were to fall on an area involved in the angle of squint, it would not be seen because the involved retinal elements had been conditioned by the squint to project in the same direction as the macula of the dominant eye. If strabismus had not been present, or if an organic scotoma existed, no such areas adjacent to the macula in a conceivable angle of squint would be found, and diplopia would be elicited, particularly if the organic lesion were relatively recent, not allowing time for squint to take place. This idea was applied to the study of amblyopes. A prism moved quickly before one eye produces a second image in any area desired, and at any rate and duration desired. If an area adjacent to the macula were inhibited, an image thrown on it by means of a prism would not be seen. If this area were conditioned to project in the same direction as the macula of the dominant eye, the stimulus would not be seen. This was found to be true. The use of this test has been previously described.[8]

In using this so-called "prism displacement test" it became apparent that moving an image with a prism could be accomplished similarly to moving a test object with a pointer, with the added advantage of having the nontested eye sta-

bilize fixation, but with the disadvantage of having the phenomena of retinal rivalry and inhibition associated with binocular fixation. Originally the test was done in a lighted refraction room with the patient looking down the refraction lane at a muscle light, usually placed below the Snellen and astigmatic charts. Prisms of different strengths, and in different positions, were rapidly moved before one eye at a time at a rate precluding fusion. This determined whether, in any areas, the prism-induced image was not seen, or whether apparent movement of one image was seen. When the prism is flashed before the amblyopic eye, a second image may or may not be seen depending upon the degree of inhibition in this eye. This in turn depends on the extent of dominance of the fixating eye. The test proved valuable in determining ocular dominance, binocular fixation and organic defects of a size larger than one half degree, but inhibition factors were more difficult to measure quantitatively because of their variability depending on the state of attention of the subject.

To illustrate a case: If a 4-diopter prism is flashed before the amblyopic eye, the second light may or may not be seen by this eye in 1 or 2 positions of the prism. Now if the room is darkened so that only the light can be seen, and none of the surround, the attention value to the amblyopic eye is so increased that the chance of its being seen through the prism is much greater. Likewise, if the fixated object is a white pin on a black tangent screen, 1 or 2 meters away, the second image is almost always seen unless an organic scotoma exists or the inhibition is very marked. If the fixated object is altered in relation to the surround, as by putting a colored glass over it, the attention value may be changed so as to make it more readily visible. The surround then conditions the inhibition factors so that it is difficult to standardize inhibition into grades other than extreme degrees.

On the other hand, ocular dominance of a very minimal degree can be determined by flashing the prism over the good

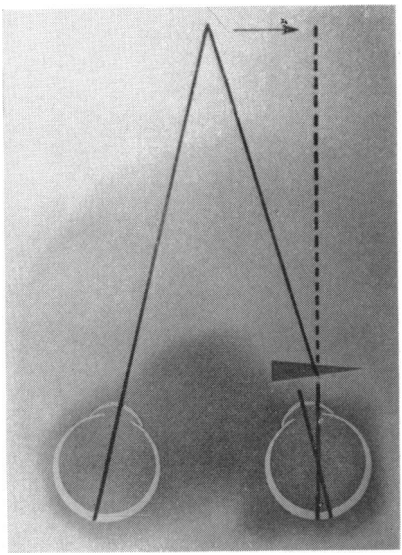


Fig. 1.—In normal binocular vision with fusion, 2 lights are seen when a 4 diopter prism is held briefly before 1 eye.

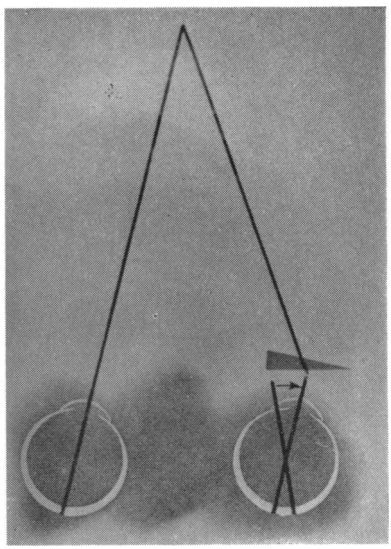


Fig. 2.—In normal binocular vision, if the prism is left in place, the eye under the prism moves toward the apex while the other eye remains stationary.

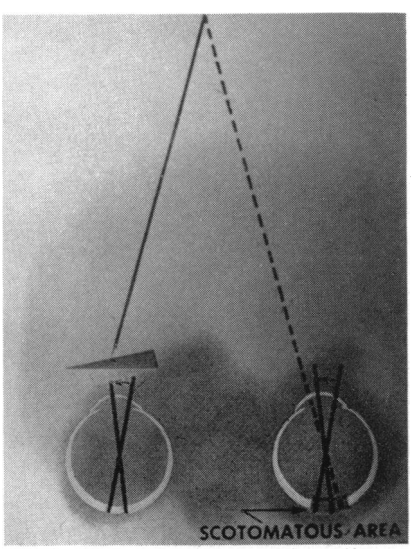


Fig. 3.—In amblyopia ex anopsia, with the prism before the sighting eye only 1 image is seen. This eye moves toward the apex of the prism and the amblyopic eye shows conjugate movement.

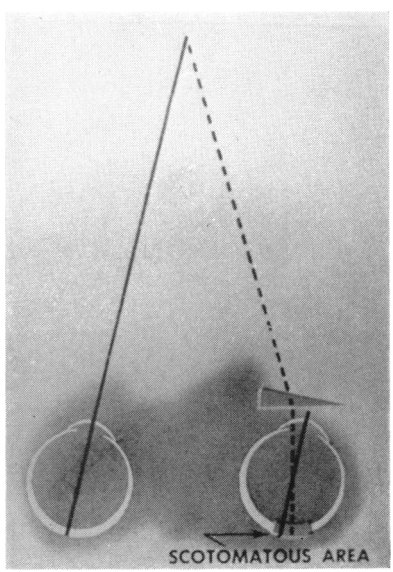


Fig. 4.—In amblyopia ex anopsia, when the prism is placed over the non-sighting eye there will be no diplopia. There will be no movement of the eyes.

eye. By this procedure, if this eye is dominant, the image will appear to move (monocular phi phenomenon) and the eye will move to fixate the new image produced by the prism, and conjugate movement of the eyes can be seen. If the eyes are equally dominant the image from the eye without the prism over it is more attractive to the brain than the newer prism-induced image, and fixation is maintained by this eye so that no movement of the eyes occurs[10] (Figs. 1, 2, 3, 4).

Organic scotomata could be measured within the limits of 1 prism diopter by flashing the prism before the eye to move the prism-induced image into and out of the scotomatous area. Since the prism could be moved more rapidly than the eye would move, and good fixation could be maintained by the opposite eye, extreme accuracy could be obtained. This is exemplified in measuring field defects approaching the macula, as from glaucoma or hemianopsia.[11] The induced image can be moved into and out of the field defect so rapidly that the error due to unsteady fixation or inhibition attending retinal rivalry is greatly reduced.

The definitions for scotomata used by Traquair[12] are employed in this study; that is, pericentral scotoma is one in which the fixation area is equally surrounded by the defect; paracentral scotoma is one in which the defect approaches or overlaps the fixation area, but is mainly situated to one side. According to the relation to the fixation area, these scotomata are further classified as supracentral, infracentral, nasocentral and temporocentral. In relation to the blind spot or area ceca, scotomata are pericecal when surrounding it and juxtacecal or paracecal when adjoining it, the latter including supracecal, infracecal, nasocecal and temporocecal forms.

1. Amblyopia Associated with Inability to Fixate, but with the Eye Held in a Relatively Centric Position

This grade of amblyopia and the following grade showing eccentric fixation, recorded on Charts 1 and 2 respectively,

CHART 1.—GROUP 1.—INABILITY TO FIXATE: CENTRIC POSITION OF EYE

Case No.	*Age*	*Vision and Refraction*	*Strabismus*	*Fixation*	*Inhibition, Scotoma and Projection*
1	24	O.D. 5/200: −.50+1.00×75= 5/200 O.S. 20/20: emmetropia 20/20	Esotropia 5 to 10△D to exotropia 5 to 10△D Onset: infancy Prolonged occlusion	Centric, random, searching, 10 to 15△D	Tangent screen: cecocentral scotoma Prism test: 2 by 4° temporocecal scotoma Projection: indefinite inhibition area surrounding scotoma
2	13	O.D. 20/20: +.50+.50×95= 20/20 O.S. 8/200:+4.00+1.00×85= 8/200	Esotropia 0 to 5△D Onset: infancy Prolonged occlusion Family history of amblyopia	Centric, irregular excursions, 5 to 10△D	Tangent screen: 3 to 5° nasocentral scotoma Prism test: 2° nasocentral scotoma Ophthalmoscopic beam: inhibition between disc and macula, with false projection
3	28	O.D. 10/200:+2.00 cyl.×125= 10/200 O.S. 20/70: +2.75×85= 20/15	Esotropia 30△D Onset: infancy Accommodative element	Centric, nystagmoid, 10△D to either side	Tangent screen: 6° paracentral scotoma Prism test: 3° nasocentral scotoma Projection: normal between disc and paracentral scotoma, indefinite near scotoma
4	36	O.D. 15/200: emmetropia 15/200 O.S. anophthalmia	History of strabismus Strong family history of amblyopia	Centric, oscillating, 5 to 10△D	Tangent screen: 2 to 3° nasocentral scotoma Before forced use of eye by loss of good eye there was a large centrocecal scotoma
5	14	O.D. 20/15: +2.50−1.25×5= 20/15 O.S. 10/200:+4.00−2.00×30=10/200	Esotropia 25△D Onset: infancy, accommodative Family history of amblyopia	Centric, wandering, 8 to 10△D	Prism test: temporocecal scotoma 5 to 10△D Projection: indefinite between scotoma and macula
6	20	O.D. 15/200:+1.00 =15/200 O.S. 20/20: +.75 =20/20	Esotropia 30 to 40△D, accommodative element	Centric, wavering	Prism test: 10° temporocecal scotoma Projection: inhibition and false projection at macula

were the most severe forms of the condition encountered in the study. Chavasse[2] mentions an even more pronounced form, namely, amblyopia with inability to fixate, and light perception in one field only. The author has seen a number of patients who, on cursory examination, professed to have faulty light perception in the temporal field. After careful inquiry and testing with a bright pencil flashlight and a narrow ophthalmoscopic beam of light, these patients perceived light when the image was outside the area of retina in the squinting eye, between the fovea and the area stimulated in conjunction with the fovea of the fixing eye. This is designated as the area corresponding to the angle of squint. Patients were often surprised at how well they could see with a poor eye when forced to do so. If the angle of squint is large, and a complete inhibitory scotoma present throughout the angle of squint, and the eye is turned in so that the nose is an obstructing factor, it is easy to understand how light perception might appear to be faulty unless special precautions were taken to stimulate the retina outside the angle of squint.

CASE REPORTS (Group 1)

CASE 1.—Right eye convergent in early infancy. Left eye irregularly occluded during childhood. Right eye cosmetically straight without surgery at about 10 years of age, remaining so until patient was 22 years of age, at which time this eye started to turn out when patient was fatigued. Homatropine refraction O.D. $-.50+1.00\times75=5/200$, viewing unfamiliar Snellen chart, and 20/100 with E's presented singly in different positions. The eye was otherwise normal. O.S. emmetropia = 20/20. Under cover, for near, O.D. in 5 to 10 $\triangle$ D: and, for distance, when patient relaxed, out 15 $\triangle$ D. Fixation movements wandering, searching, randomlike, with excursions of 10 to 15 $\triangle$ D to either side of the midline.

Ribbonlike markers were placed radially from the center of a tangent screen, and the patient, using a long pointer, looking at the crossing of the ribbons, could map out her own scotoma. Apparently she was fixing with the eye turned in 3 to 4 degrees, as the blindspot was displaced inward this amount. The scotoma is shown on the

field chart. A small light held in the scotoma could be made to disappear, proving its density. A narrow ophthalmoscopic beam of light (½ disc diameter on the retina), shone into the eye, disappeared when the light was just temporal to the disc, and, as it was brought closer to the macula, became visible and was falsely projected to the nasal field.

The markers were then removed from the tangent screen, and the patient was asked to observe with both eyes the fixation spot, a white pin 3 mm. in diameter. Various sized prisms were flashed in front of the amblyopic eye to displace the image onto different areas of the retina. When the patient saw the second pin, she indicated its location on the screen. By this method, taking into consideration the fact that the eye was turned in approximately 10 △ D, a scotomatous area could be mapped out which included the entire papillomacular bundle. With practice this became considerably smaller, showing an area of absolute scotoma extending temporally from the normal blindspot for about 10 prism diopters. Between this area and the fixation point, the displaced image was always localized by the patient in the same place, regardless of the degree of displacement within this area. The patient was then asked to look at a grid and other complicated patterns, such as a person's face, and describe and outline the part of the image that was indistinct. From such observations it was apparent that there was an absolute scotoma extending from the disc halfway to the macula, and a relative scotoma involving the entire central area. In this latter area the projection was faulty.

CASE 3.—A 28-year-old male had had strabismus since birth. There had been no occlusion. Refraction: O.D.+2 cyl.×125= 10/200. O.S.+2.75 cyl.×85=20/15. Vision by 2-point discrimination was 20/100; single E's 20/100 to 20/50; near vision 14/42 (A.M.A. near vision test card equivalent to 20/60) when tested with single E's on a white background, but patient was unable to read even the largest print with this eye when letters were presented in a line. Foveolar reflex was normal. Attempted fixation with this eye caused gross nystagmoid movements of about 10 △ D to either side of the midline. Strabismus measured 30 △ D esotropia. It was apparent from the stereocampimeter and the mirror-screen tests that, with the good eye fixing, the image was falling on the blindspot of the amblyopic eye.

Flashing prisms over the amblyopic eye, the image could be moved off the blindspot, and the patient saw it and projected it normally. As the image approached to within 5° of the macula of

the amblyopic eye, the patient saw the image but could not tell whether it was to the right or left of the fixated image of the left eye. At times he saw 2 images with the amblyopic eye, one on either side of the fixation point as seen with the fixing nonamblyopic eye (triplopia). As additional prisms were added, the image disappeared at a point about 3° from the fixation point, that is 3° nasal to the macula. On occluding the good eye, the patient, after practice, could obscure the fixation point within the scotoma. His eye then appeared to be approximately straight ahead. With the pointer he could outline on the tangent screen a 3° central or paracentral scotoma. He could fixate with this scotomatous area for about 3 seconds. A small ophthalmoscopic light could be made to disappear in this area.

The interesting features of this case were the alignment of the blindspot in the amblyopic eye with the fovea of the good eye, the normal projection just temporal to the disc, the abnormal projection merging into absolute scotoma as the macular region was approached.

CASE 4.—A 36-year-old musician since losing his left eye about 3½ years ago has been forced to use the right eye which was known to be amblyopic. There was a history of strabismus in childhood. The patient's sister, brother, 2 nieces, mother's brother and cousin all have amblyopic eyes. The patient has been observed for 8 years. Vision with his right eye prior to loss of his good eye was "form vision only." Since loss of the good eye, his vision has improved for getting about, but he is still unable to read or distinguish features. A year ago vision was recorded as 6/200, and a centrocecal scotoma mapped out on the tangent screen. Recent scotoma studies showed nasocentral scotoma of 2°, apparently touching the fixation point. There was insignificant refractive error and the foveolar reflex was normal. Fixation movements were oscillatory, swinging about 4 to 8 △ D to either side.

Vision with Snellen letters was approximately 15/200, but as the patient continued to look at the chart he would occasionally get glimpses of a letter on the 20/120 line, and even one letter on the 20/60 line. He was unable to tell which line these letters were on. Then, using single Landolt circles and single E's he could tell the position of the 20/70 circle and the 20/50 E. Two-point discrimination, using black dots on a white card and bright pins on a black tangent screen, varied from 20/400 to 20/50. If the patient knew that only 2 points were to be distinguished as 2, he could study the dots and after a moment see a flash of separation between them and

interpret them as 2 when they were 1.5 mm. apart at 2 meters distance, that is, 20/50 vision. When confronted with a number of points, separated by varying distances, there was considerable confusion and he could only recognize separation of 12 mm. or 20/400. When only 2 points were presented, but he did not know there were only 2, he would interpret them as 2, 3 or 4 until separations were reached corresponding to 20/400 visual acuity. It was easier for him to interpret dots and pins separated vertically than horizontally.

This patient showed a definite lessening of the scotoma over a period of 3½ years of forced use of the eye due to loss of the non-amblyopic eye. A definite nasocentral scotoma persisted so that reading vision was impossible.

Analysis

The characteristic feature of Group 1 illustrated on Chart 1 is the poor fixation, fixation movements oscillating to either side of the midline, the eye being held in a relatively centric position as compared with Group 2 in which the eye is held in a relatively eccentric position. All amblyopes show grossly apparent searching fixation movements, but in Group 1 the movements are extreme, 5 to 10 △ D to either side of the central position. One would expect to find a large central scotoma. Instead, a large cecocentral or centrocecal scotoma is easily demonstrable by all methods used, and within this area are relative degrees of inhibition and indefinite projection.

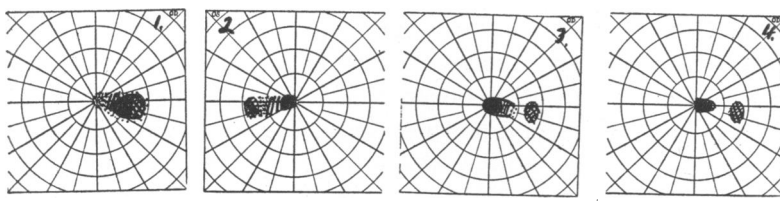
An absolute scotoma, even to light, was demonstrated in 4 cases (Chart A). This corresponded to a point on the retina just temporal to the disc in Case 1 and just nasal to the macula in Cases 2, 3 and 4. Surrounding the scotoma, within the cecocentral area, the light or test object can be seen if sufficiently bright, as for instance, a white test object on a black tangent screen displaced with prisms. As the scotomatous area is approached, the sense of projection becomes indefinite. The patient will locate the test object each time it is seen in approximately the same place in spite of successively increased strengths of prisms being used. The posi-

tion may vary depending on whether the patient considers the object to be coming from the right or from the left. A small light, as a pencil flashlight or narrow ophthalmoscopic beam of light, can be made to disappear in the scotomatous area. The inhibition and the poor projection account for the faulty fixation movements. Failure to use an eccentric area for fixation, as is done in Group 2, is related to the poor visual acuity and the large size of the inhibition area, but primarily to the lack of fixed false projection. Even in these cases, with practice, the patient can fix with the scotomatous area or to one side of it.

The visual acuity in all cases in Group 1 was less than 20/200. There was insignificant refractive error in 4 of the 6 cases. All of the 6 cases had had strabismus. In Cases 2 and 5 this was associated with high refractive error, and the strabismus had a marked accommodative element. In Cases 3 and 6 the strabismus was of an amount to allow the normal blindspot to be stimulated in conjunction with the macula of the other eye. The strabismus in Cases 1, 2 and 4 was minimal, Case 2 having been corrected by surgery, and Cases 1 and 4, originally esotropic, showing tendency to exotropia in adult life. Because there was an accommodative element in the strabismus, or no fixed angle of strabismus, fixed false retinal projection had not developed, perhaps accounting for the absence of obligatory eccentric fixation. Apparent inability to use the eye properly was noted in infancy in all cases in Group 1. Since refractive and strabismic factors were relatively unimportant, one is forced to suspect that the amblyopia was on a congenital basis in this group, a supposition correlated by a strong family history in Cases 2, 4 and 5. Three of the patients had prolonged occlusion of the good eye, starting at an early age, without effect. Case 4, an adult, had 3½ years of forced use of the eye after loss of the non-amblyopic eye without developing vision sufficient for reading. The amblyopia definitely became less, suggesting that active training methods might possibly further improve

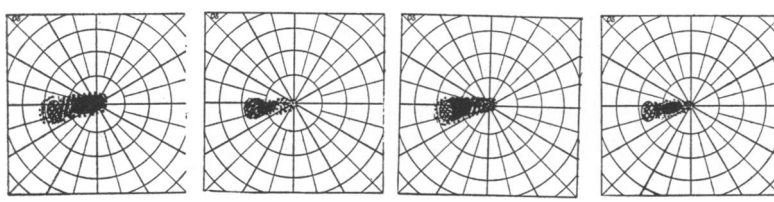
CHART A

GROUP 1

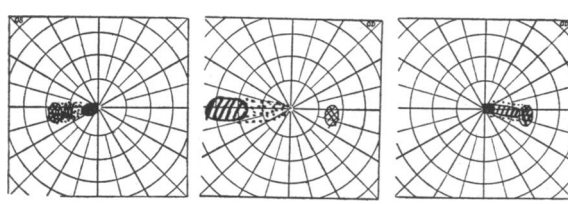


Case 1 Case 2 Case 3 Case 4

GROUP 2



Case 7 Case 8 Case 9 Case 10



Case 11 Case 12 Case 13

Scotoma

Inhibition

Indefinite Projection

Blindspot

vision. One is inclined to believe that in this type of amblyopia there may be a congenital defect of a more serious nature than in the cases that respond to occlusion. A final practical note is that the strabismus in Group 1 can be corrected or overcorrected easily by surgery, in contrast to the group of obligatory eccentric fixators with fixed false projection described in Group 2.

II. Amblyopia Associated with Eccentric Fixation of the Eye

The majority of so-called "poor fixators" belong to Group 2. When the nonamblyopic eye was covered, the amblyopic eye assumed an eccentric position, from which position fixation movements were gross, searching excursions of less extent than in Group 1. Twenty-six cases are reported on Chart 2. In a number of patients observed in the first part of the investigation, no attempt was made to estimate the degree of eccentric fixation, but some of these are included as there was a definite notation that fixation was eccentric.

CASE REPORTS (Group 2)

CASE 7.—A 31-year-old studio worker had been under the author's observation for 10 years. The left eye had turned in since 5 months of age. Refraction: O.D. emmetropic: 20/15. O.S. +2.00 Sph. = 5/200. Foveolar reflexes were normal. When the patient fixated with the right eye, the left eye turned in 25 to 30 △ D as judged by the corneal reflex. On attempting to fix with the left eye, he turned this eye in 40 to 45 △ D. From this point, oscillations of 6 to 10 △ were noted. On the tangent screen the patient, using a pointer, could map out a large cecocentral scotoma. Fixating with the good eye behind a mirror, the mirror turned so that the amblyopic eye was in the primary position, the normal blindspot was plotted, and a scotoma extending from the blindspot to the fixation point. By varying the size, intensity and attention value of the fixated object presented to the nonamblyopic eye varying degrees of inhibition were made manifest in the amblyopic eye (see Groups 3 and 4). An absolute scotoma was mapped out in an area 10 by 4° between the fixation point and the blindspot, touching the fixation point. A relative scotoma surrounded this. Similar inhibition was

demonstrated by flashing a prism over the amblyopic eye to make the image of a central white pin appear on different parts of the retina of the amblyopic eye, fixation being held steady by the good eye looking in the mirror. There was an area around the absolute scotoma in which the patient saw the prism-induced image, but was unable to localize the moving image. Outside this area projection was normal in sign. A narrow ophthalmoscopic beam was described as dim when directed to a point just nasal to the fovea, was projected to the nasal field when the spot was placed on the temporal border of the disc, straight ahead when on the nasal border of the disc and to the temporal field when placed slightly more nasally. The interpretation of the after-image test was a complete symmetrical cross. This paradoxical finding is explained by the fact that the patient fixed the eye eccentrically when viewing the luminous line with the amblyopic eye. This eccentric position was considered by him to be straight ahead.

The patient volunteered this information: with the left eye turned out so as to stimulate an area on the retina just temporal to the macula, the image appeared clearer, but was projected 15 to 20° to the right (nasal field); he preferred to keep the eye turned in where the image was projected straight ahead rather than to have a clearer image projected to one side. This indicates that where a conflict exists between acuity and projection, acuity is not always the deciding factor in determining the position of the eye. This eye has been operated on 3 times during the past 10 years and the strabismus is only slightly improved over the original position. This is a case of marked obligatory eccentric fixation with a large centrocecal scotoma and marked fixed abnormal retinal projection.

CASE 8.—A 36-year-old musician gave a history of left convergent strabismus since infancy. Refraction: O.D.$+.50-.37\times50=$ 20/20. O.S.$+.75$ sph.$=10/200$. Fundi were normal. Fixing with the right eye, the left eye turned in 25 to 30 $\triangle$ D. Attempting to fixate with the left eye, this eye turned in 15 to 25 $\triangle$ D and there were irregular jerking fixation movements. In this position the patient felt that his eye was straight ahead, and with this eye viewing a tangent screen, he could map out a scotoma which obviously included his normal blindspot and a 3 to 4° extension toward the macula. Having the patient fixate a 2 mm. white pin on the tangent screen 1 meter away with his good eye, a prism was flashed in front of his amblyopic eye to displace the image in this eye to different areas of the retina, and the patient stated when and where he saw the second image. The eye was turned in so that he was aligning an

area on the retina just temporal to the disc of the amblyopic eye with the fovea of the right eye, as judged with the stereocampimeter and the mirror-screen test. That is, the eye was turned in 20 to 25 △ D. Flashing a 2 to 5 △ prism base in over the left eye, the patient failed to see the prism-induced image as it was on the blind-spot. Flashing a 12 △ prism base in, the patient readily saw the prism-induced image and projected it correctly to the left (temporal field). Flashing a 2 to 3 △ prism base out over the left eye, the patient saw no image; but, increasing to a 4 △ prism he saw the image and projected it correctly to the left. As prism was gradually added, he continued to project the image to the left until a 16 △ D was used, when he suddenly projected the image to the right 5 to 10°, even though the image was still nasal to the macula (eye turned in 20 to 25 △ D). Continuing to increase the prism gradually no scotomatous area was found as the macula was approached, while the projection of the image continued to be to the right. Now reducing the base out prism, the patient continued to project to the right until the prism had been reduced to 7 △ D base out. Then suddenly he started projecting the image to the left again. In other words, no real scotomatous area could be found, except possibly 2 to 3° area adjoining the temporal side of the disc. The area between the disc and the macula showed indefinite projection; to the left, if the increment was from the nasal side, and to the right if from the temporal side, even though the same area of the retina was involved. As the macula was reached there was fixed false projection. This phenomenon of the projection being influenced by the site of the original stimulus is common. If the patient was asked to look with his eyes turned out slightly so as to throw the image of a letter subtending an angle of 50′ placed on the tangent screen onto the temporal retina or onto the macula, he failed to note any dimness of the letter. However, when asked to observe a line of letters, the patient stated that the letters to the right were clearer than to the left. Since the eye was turned in 10 △ D when fixating, this observation suggested that the macular region saw better than the retinal region nasal to it, used for fixation. This finding, and failure to find a scotoma at the macula by prism test, suggested that vision in the macular region was not depressed more than in the adjacent area. Likewise, judging from the poor vision, it was not developed more than the adjacent area. This patient then showed a temporocecal scotoma, fixed false projection at the macula, and indefinite projection between the scotoma and the macula. The macular area had no scotoma, but approximately the same visual acuity as the area peripheral to it.

CASE 9.—A 28-year-old accountant had had convergent strabismus of the left eye since childhood. Refraction: O.D.+.75×80= 20/15. O.S.+.50 sph.=15/200. Fixing with the right eye, the left eye turned in approximately 25 △ D, and on attempting to fixate with the left eye, this eye turned in slightly more on casual gaze; but when the patient was told to look carefully, he turned the eye out 5 to 10 △ D. He could hold the eye relatively still in this position. On a tangent screen he could project his own scotoma which included the entire cecocentral area. A similar type of scotoma was found on the stereocampimeter, but with the prism test, the absolute scotoma was found to extend only 7° toward the macula from the edge of the disc. In the area of relative inhibition, between this and the macula, projection was indefinite. This patient, in contrast to Cases 7 and 8, preferred to fix at the site of clearest vision, namely, just temporal to the macula, and the obligatory sense of eccentric fixation was less well established. The macular area must have been depressed more than the immediately adjacent temporal retina yet only a relative scotoma was demonstrable.

Analysis

Cases 8, 9, 11, 15 and 22 showed relatively insignificant errors of refraction. Visual acuity varied from light projection in one case to 20/200, in most instances comparable to what would be expected from the part of the retina stimulated as a result of the eccentric fixation. All cases in Group 2 showed esotropia except Cases 12 and 30, which showed exotropia. The exotropia in Case 14 was the result of surgery. Amblyopia can develop with divergent strabismus but it seems to be a rare coincidence, probably because the eyes in a divergent position have both maculas frequently stimulated so that typically divergent squinters become alternators, using the right eye for the right field and the left eye for the left field, whereas in convergent strabismus the nose helps to obscure objects from the macula.

Scotoma observations were similar to those in Group 1 (Chart A). Inhibition was found on the area of retina involved in the angle of squint extending to the macula, with most dense inhibition, or absolute scotoma, at the angle in

some cases, and touching one side of the macula in others. The scotoma was readily found by all tests. The patients could project the inhibition area onto a tangent screen and outline it with a pointer. They could lose light from a pencil flashlight in the area of absolute scotoma. In some cases, shining an ophthalmoscopic beam into the eye, one could determine the part of the retina that was most inhibited and the existence of false projection as the image fell closer to the macula.

The distinguishing feature of Group 2 is the eccentric position assumed for fixation. This position depends on 3 factors: the size of the inhibition area, its position and the abnormal subjective sense of the straight-ahead position of the eye. The patient may turn the eye farther in or out than the position assumed when the nonamblyopic eye is fixing. In some of the cases, there is an obvious attempt to place the image on a less inhibited area of the retina, in the interest of better visual acuity. In others, the abnormal retinal projection is the more determining factor. The patient will turn his eye to a position in which he considers it straight ahead, even if visual acuity is not as good in this position as in some other position closer to the macula, outside of the angle of squint, in which position he considers the eye to be turned.

The cases with eccentric fixation resulting from fixed abnormal retinal projection are designated by Chavasse[2] as those having obligatory eccentric fixation. There is a strong tendency to force the eye into this position even after repeated surgery (Case 7). This tendency may be anticipated by the involuntary turning of the eye when prisms are placed in front of the eye, displacing the image closer to the macula or on the opposite side of the macula. Eyes with this tendency will continue to turn as prism is added in order to keep the fixed anomalous straight-ahead position in line with the macula of the other eye. The same tendency for obligatory movement of the eye under prisms is seen in some centric fixators, with or without amblyopia. Evidently a strong

peripheral fusion sense is present. This group may represent an advanced form of the type of amblyopia described in Group 3.

The prognosis for Group 2 is equally as poor as for Group 1. An adult (not on the chart) was seen in whom eccentric fixation persisted 3 years after loss of the nonamblyopic eye. The patient said a fixated object was seen straight ahead when his eye was turned in; but that when his eye was relatively straight the object was seen double, the clearer image projected considerably to one side, and the duller image projected straight ahead.

III. Amblyopia Associated with Strabismus and Abnormal Retinal Projection, but with Relatively Good Ability to Fixate

CASE REPORTS (Group 3)

CASE 34.—A 45-year-old school teacher showed intermittent convergent strabismus and amblyopia of the right eye. Refraction: O.D. $+4.50 = 20/200$. O.S. $+4.50 - .25 \times 5 = 20/15$. Two-point discrimination: 20/100 to 20/50. Fundi were normal. There was an accommodative strabismus of the right eye of 4 to 15 $\triangle$ D. Fixation movements were searching and 3 to 4 $\triangle$ D in extent. Without glasses her right eye was in 8 to 10 $\triangle$ as judged by the cover test.

Examined by prism test, using the tangent screen, at 1 meter, fixing a 3 mm. white pin, flashing prisms over the right eye, the patient saw 2 images with 2 $\triangle$ D base up, down and in, but not base out, until a 10 diopter prism was used. This demonstrated an inhibited area of retina nasal to the macula. With glasses on, the patient showed 0 to 2 $\triangle$ D esotropia. Repeating the prism test, she responded as follows: 2 $\triangle$ base out, no image seen; base in, up and down, second image seen; with 3 to 14 $\triangle$, diplopia in all fields, and projection correct in direction. She saw 2 images now in the same area that was inhibited when the eye was forced to accommodate by not wearing glasses. With mirror-screen test, the patient showed a 14 $\triangle$ D scotoma nasal to and touching the fixation point. The size of this scotoma varied depending on the size and interest value of the fixation object presented to the left eye. The smaller the fixation object the smaller the scotoma found in the right eye. Stereocampimeter revealed the same findings. On the tangent screen or

CHART 1 ON PAGE 536

CHART 2.—GROUP 2.—INABILITY TO FIXATE: ECCENTRIC POSITION OF EYE

Case No.	Age	Vision		Strabismus	Fixation	Studies of Inhibition, Scotoma and Projection
7	31	O.D. 20/15; Emmetropic O.S. 5/200; +2.00	20/15 5/200	Esotropia 25 to 30△D. Anisometropia Onset: infancy	Obligatory. Eccentric 40 to 45△D. Exotropia	Tangent Screen: Paracentrocecal scotoma Prism Test: 10×4° nasocentral scotoma Projection: False in region of relative scotoma
8	36	O.D. 20/20; +.50−.37×50 O.S. 10/200; +.75	20/20 10/200	Esotropia 28 to 30△D. Onset: infancy	Eccentric. 15 D△. Esotropia	Tangent Screen: 3 to 4° temporocecal scotoma Prism Test: 2° temporocecal scotoma Projection: Indefinite between scotomatous area and macula; fixed false at macula
9	28	O.D. 20/15; +.75−.75×170 O.S. 15/200; +.50	20/15 15/200	Esotropia 25△D. Onset: childhood	Eccentric. 30 Esotropia or 5 to 10△D. Exotropia	Tangent Screen: Paracentrocecal scotoma Stereocampimeter: Paracentrocecal scotoma Prism Test: Temporocecal scotoma 14△D. Projection: Indefinite
10	12	O.D. 20/25; +2.50+.62×80 O.S. 5/200; +2.25	20/25 5/200	Esotropia 30 to 40△D. Onset: 8 weeks of age	Eccentric. 35△D. Esotropia	Prism Test: Temporocecal scotoma Projection: False between scotomatous area and macula Ophthalmoscopic Beam: Inhibition at angle of squint; projection false between area of angle and macula Amblyoscope: Inhibition between macula and disc
11	27	O.D. 20/20; +.25 O.S. 20/100; +.50 cyl.×90	20/20 20/100	Esotropia 10 to 20△D. Macula slightly granular Onset: infancy	Obligatory. Eccentric 10+△D. Esotropia	Pinhole Prism Test: ½×1° infracentral scotoma, 3/1,500 white test object Prism Test: 5° nasocentral scotoma. Strabismus increased as prism added Mirror-screen: Scotoma to 5/1,000 white and red test object; relative scotoma around this. Retinal rivalry: O.D. predominates Projection: False or normal, depending on attention. After-image test: normal Ophthalmoscopic beam: Inhibition temporal to disc; projects nasally at macula
12	25	O.D. 20/200; −1.75+1.50×105 O.S. 20/200; −2.50+.75×25	20/200 20/20	Exotropia 20△D. Vertical element Onset: childhood	Obligatory. Eccentric 10 to 20△D. Esotropia	Mirror-screen: Angle of false projection equals angle of squint After practice, could align maculas. Large inhibitory scotoma, temporal retinal area Ophthalmoscopic Beam: Dull on temporal side and at macula Bright on nasal side
13	20	O.D. 5/200; +2.50+.75×75 O.S. 20/20; +2.25−.62×90	5/200 20/15	Esotropia 10 to 25△D. Accommodative element	Eccentric	Prism Test: Scotoma throughout angle of squint Postoperative Prism Test and Mirror-screen: Fingerlike cecocentral scotoma; 2° absolute nasocentral, 10° relative cecocentral; indefinite projection Amblyoscope: 5° nasocentral scotoma. Projection: Normal
14	20	O.D. 20/15; +2.00 O.S. L. P. +2.00	20/15 L. P.	Esotropia Postoperative at 16 years of age, exotropia 40△D.	Eccentric. 30△D. Exotropia	Light projection in extreme nasal and temporal fields Marked inhibition in central field
15	33	O.D. 20/40; −.75 O.S. Hand motion straight ahead 20/300 (Temporally) −.50+.50×90	20/20 20/300	Esotropia 20 to 30△D. Onset: infancy	Obligatory. Eccentric. 20△D.	Prism Test: Nasocentral scotoma Ophthalmoscopic Beam: Inhibition temporal to disc Projects to right at macula
16	20	O.D. 20/200; +4.25+1.50×15 O.S. 20/40; +5.50−1.50×80	20/200 20/40	Esotropia 20 to 25△D.	Eccentric. 5△D. Exotropia	Amblyoscope: Inhibition throughout angle of squint, except at macula; projecting as exotropia
17	20	O.D. 20/200; +1.00 O.S. 20/20; +2.00−.75×90	20/200 20/20	Esotropia 35 to 40△D.	Eccentric. 25△D. Esotropia	Amblyoscope: Inhibition throughout angle of squint Prism Test: Temporocecal scotoma 8°, projected as exotropia between scotoma and macula
18	20	O.D. 20/30; +3.50−1.00×180 O.S. 10/200; +4.50−.50×180	20/25 10/200	Esotropia 40 to 50△D.	Obligatory. Eccentric	Amblyoscope: Inhibition at angle and at macula projection normal and abnormal Prism Test: Inhibition at angle; abnormal projection between this and macula
19	20	O.D. 20/200; +5.50−.50×180 O.S. 20/40; +5.25−.50×180	20/200 20/15	Esotropia 45 to 55△D. Accommodative element	Eccentric	Small scotoma at angle. Normal and abnormal projection between scotoma and macula. Small nasocentral scotoma at macula
20	20	O.D. 10/200; +3.00−5.00×180 O.S. 20/50; +.50−.25×180	10/200 20/50	Esotropia 30△D. Anisometropia	Eccentric	Tangent Screen: No scotoma found Amblyoscope: Inhibition at angle of squint Projection: False at macula
21	19	O.D. 20/15; +1.00−.50×180 O.S. 20/200; +2.00−.50×15	20/15 20/200	Esotropia 40 to 50△D. Accommodative element	Eccentric. Postoperative: Fixation improved	Amblyoscope: Marked inhibition throughout angle Small area adjoining macula with false projection Postoperative: Esotropia 3 to 5°
22	22	O.D. 20/20; +.50−.25×180 O.S. 4/200; +1.00	20/20 4/200	Esotropia 35△D.	Obligatory. Eccentric 45 D. Esotropia	Vision inhibited in nasal field
23	34	O.D. 20/20 O.S. 5/200	20/20 5/200	Esotropia 50△D.	Obligatory. Eccentric 20 to 30△D. Esotropia	Marked inhibition Postoperative: Fixes in old area, causing opposite eye to turn out
24	20	O.D. 20/30; +4.00−.25×90 O.S. 5/200; +6.00−3.00×180	20/20 5/200	Esotropia 50 to 60△D, Anisometropia	Eccentric	Amblyoscope: Inhibition throughout angle of squint
25	29	O.D. 20/200; +4.50−1.00×180 O.S. 20/50; +4.50−1.00×105	20/200 20/40	Esotropia 0 to 25△D. Accommodative element	Eccentric. 5 to 10△D. Exotropia	Prism Test: Moderate inhibition; nasocentral scotoma Projection: Normal when seen
26	30	O.D. 20/400; +7.25 O.S. 20/40; +3.00+.50×130	20/400 20/30	Esotropia 20△D. Accommodative element Anisometropia	Eccentric	Inhibition throughout angle of squint
27	24	O.D. 20/30; +4.00−.50×165 O.S. 5/200; +6.00−1.00×180	20/15 5/200	Esotropia 20 to 40△D. Accommodative element Anisometropia	Eccentric	Amblyoscope: Scotoma throughout angle of squint Prism Test: Scotoma throughout angle of squint. Fixing muscle light Projection: False at macula
28	22	O.D. 5/200; +5.50−3.25×18 O.S. 20/20; +.75−1.00×140	5/200 20/20	Esotropia 40 to 50△D. Accommodative element Anisometropia Onset: infancy	Eccentric	Scotoma throughout angle of squint
29	20	O.D. 20/400; +4.25−1.00×5 O.S. 20/20; +2.25−.25×80	20/400 20/20	Esotropia 20 to 30△D. Accommodative element Anisometropia Onset: infancy	Obligatory. Eccentric 10△D. Esotropia	Scotoma throughout angle of squint Projection: False at macula Postoperative: Eccentric fixation unchanged
30	25	O.D. 7/200; +3.50 O.S. 20/20; +.75	7/200 20/20	Exotropia 20△D. Anisometropia Onset: infancy	Eccentric	Amblyoscope: Inhibition throughout angle of squint Prism Test: Scotoma throughout angle, but occasionally sees image, and then projects normally
31	29	O.D. 20/30; +4.25−.62×17 O.S. 4/200; +4.00	20/15 4/200	Esotropia 20 to 40△D. Accommodative element Onset: 4 years of age	Obligatory. Eccentric	Prism Test: Inhibition throughout angle of squint Fixing muscle light. Fixed false projection at macula Strabismus increases as correcting prisms added
32	20	O.D. 20/40; +2.00−.75×180 O.S. L. Pr; +5.00−1.25×120	20/25 L. Pr.	Esotropia 50△D. Anisometropia		Marked cecocentral scotoma Projection: Normal in direction outside scotoma

CHART 3.—GROUP 3.—ABILITY TO FIXATE: FALSE PROJECTION AND STRABISMUS

(Fixation Central, Slightly Wavering)

Case No.	*Age*	*Vision*	*Strabismus*	*Studies of Inhibition, Scotoma and Projection*
33	20	O.D. 20/200; +4.00×95 20/200 O.S. 20/30; +2.00+3.25×70 20/25	Esotropia 35△D. Onset: 3 years of age	Prism Test: 9° scotoma at angle of squint Projection: False between scotoma and macula Slight nasocentral scotoma
34	45	O.D. 20/200; +4.50 20/200 O.S. 20/20; +4.50−.25×5 20/15	Esotropia 10 to 25△D. Accommodative element Onset: childhood	Prism Test: Scotoma at angle of squint Projection: False between scotoma and macula With glasses on: No inhibition found Mirror-screen: Centrocecal scotoma, variable, 3 to 4° Letters: 20′ scotoma Tangent Screen: 2/1,000 test object dim to right of fixation point 3 or 4° scotoma between macula and disc Stereocampimeter: Same as tangent screen
35	18	O.D. 20/70; +3.50+.50×90 20/50 O.S. 20/20; +3.50+.50×90 20/20	Esotropia 10△D. Accommodative element Onset: childhood	Stereocampimeter: No scotoma to 7″ test object; false projection Tangent Screen: No scotoma, 1/1,000 test object Projection: False at macula Amblyoscope: Fixed false projection at macula Prism Test: ½° inhibition at angle of squint Snellen Chart: 20/40 letter to right, dim; 8 to 10′ nasocentral scotoma Mirror-screen: Minimal inhibition at angle of squint to 3/1,000 test object No inhibition at macula. No retinal rivalry
36	8	O.D. 20/25; +4.00−1.00×10 20/25 O.S. 20/50; +4.00−1.25×170 20/50	Esotropia 12 to 40△D. Accommodative element	Prism Test: Inhibition at angle of squint Abnormal projection between scotoma and macula After-image Test: Normal projection Strabismus increases as correcting prisms added Amblyoscope: Inhibition at angle of squint Projection: Normal and false between angle and macula No effect on strabismus from 5 mm. recession of internal rectus muscle
37	19	O.D. 20/100; +4.00 20/70 O.S. 20/20; +2.25+.75×7 20/20	Esotropia 50 to 60△D. Anisometropia Onset: age 3 Family history	Prism Test: 15° scotoma at angle of squint. Fixing muscle light Projection: False between scotoma and macula Postoperative Prism Test: Inhibition at macula and normal projection Amblyoscope: Small nasocentral scotoma and no inhibition at angle Mirror-screen: Peripheral inhibition and nasocentral scotoma. Projection normal except at macula
38	23	O.D. 20/400; +5.00−4.00×15 20/400 O.S. 20/20; +1.00−.25×170 20/20	Esotropia 10△D. Anisometropia Onset: infancy Family history	Prism Test: Scotoma at angle and nasocentral scotoma False projection between Strabismus increases as correcting prisms added
39	23	O.D. 20/20; +1.50−.50×75 20/20 O.S. 20/50; +1.75−.50×90 20/40	Esotropia 45△D. Onset: childhood	Scotoma at angle of squint by prism test and amblyoscope Projection false between scotoma and macula After-image; normal projection. Pinhole prism test: no central scotoma; false projection between blindspot and macula Mirror-screen: Scotoma at angle, 25° peripherally; indefinite projection between blindspot and macula Postoperative Prism Test: Peripheral scotoma and 2° nasocentral scotoma Projection: Normal if stimulus from temporal side; abnormal if from nasal side
40	27	O.D. 20/20; +3.25 20/20 O.S. 20/200; +2.75+1.50×105 20/200	Esotropia 40△D. Onset: 1 year	Prism Test and Pinhole Prism: Scotoma at angle Postoperative Prism Test and Pinhole Prism: No scotoma at macula Amblyoscope: Aligns macula O.D. with blindspot O.S. Mirror-screen: No scotoma; indefinite projection between disc and macula
41	23	O.D. 20/20; +2.25+.25×90 20/15 O.S. 20/40; +1.75+.50×90 20/40	Esotropia 40 to 45△D. Onset: age 4	Prism Test: Vague inhibition at angle Amblyoscope: Inhibition at angle; false projection between angle and macula Mirror-screen: Normal and false projection; retinal rivalry Postoperative: 7½° esotropia. Prism test: 5° scotoma at angle False projection between angle and macula Amblyoscope: Normal projection; no fusion Mirror-screen: No scotoma; normal projection Pinhole Prism: No scotoma. After-image: Normal projection Strabismus neutralized by prisms, nasocentral scotoma by prism test
42	21	O.D. 20/200; +5.00 20/100 O.S. 20/20; Emmetropic 20/20	Exotropia 30△D. Anisometropia Onset: infancy	Prism Test and Red Lens: Inhibition at angle; indefinite projection between angle and macula Image clearer at macula than at inhibited area
43	23	O.D. 20/15; Emmetropic 20/15 O.S. 20/200; +.50−.25×180 20/200	Esotropia 25 to 30△D.	Amblyoscope: 4° scotoma at angle of squint and false projection between scotoma and macula. No scotoma at macula
44	20	O.D. 20/20; +1.75 20/20 O.S. 20/70; +2.50+2.50×80 20/70	Esotropia 40 to 50△D. Anisometropia	Prism Test: 10° inhibition at angle of squint. Fixing muscle light Projection: False between inhibition area and macula
45	20	O.D. 20/20; +2.50+.75×30 20/20 O.S. 20/70; +1.75+.75×180 20/70	Esotropia 30 to 50△D. Accommodative element	Prism Test: 10° scotoma at angle; false projection between scotoma and macula. Fixing muscle light Amblyoscope: Same as with prism test Postoperative: Sensory examination shows no change in scotoma
46	23	O.D. 20/300; +1.00−.25×180 20/200 O.S. 20/25; +.50−.25×150 20/20	Esotropia 30 to 50△D.	Prism Test and Amblyoscope: Scotoma at angle Projection: False between scotoma and macula No scotoma at macula
47	20	O.D. 20/20; Emmetropic 20/20 O.S. 20/50; Emmetropic 20/50	Exotropia 28△D.	Prism Test: 2½° scotoma at angle; false projection between scotoma and macula Amblyoscope: 5° scotoma at angle
48	20	O.D. 20/400; −2.25−.50×90 20/15 O.S. 20/400; −2.25−1.25×90 20/50	Exotropia 30 to 35△D. Onset: infancy	Prism Test: 10° inhibition at angle; indefinite projection between scotoma and macula Postoperative: Small central scotoma O.S. by prism test and amblyoscope Fixing O.D., normal projection Fixing O.S., abnormal projection
49	20	O.D. 20/30; +1.50−.25×115 20/15 O.S. 20/50; +1.75−1.25×130 20/50	Esotropia 11 to 24△D. Accommodative element	Prism Test: Scotoma at angle and fixed false projection between scotoma and macula Strabismus increased as correcting prism added
50	19	O.D. 20/70; +1.50+.75×60 20/50 O.S. 20/25; +1.25−1.25×5 20/20	Esotropia 25△D.	Prism Test: Scotoma throughout angle
51	19	O.D. 20/60; +3.50 20/60 O.S. 20/20; +2.50−1.00×145 20/20	Esotropia 38△D.	Prism Test and Amblyoscope: 10° scotoma at angle of squint. Fixing muscle light Projection: Abnormal between scotoma and macula
52	25	O.D. 20/20; +.75−.25×165 20/20 O.S. 20/40; +.75 20/40	Esotropia 38△D.	Prism Test and Amblyoscope: Scotoma at angle Projection: False between scotoma and macula
53	20	O.D. 20/30; +1.50−.25×115 20/15 O.S. 20/50; +1.75−1.25×180 20/50	Esotropia 11 to 24△D. Accommodative element	Prism Test and Amblyoscope: Scotoma at angle of squint Projection: False between scotoma and macula

CHART 4.—GROUP 4.—CENTRIC FIXATION (SLIGHTLY WAVERING): NORMAL PROJECTION

Case No.	*Age*	*Vision*	*Strabismus*	*Studies of Inhibition, Scotoma and Projection*
54	15	O.D. Emmetropic 20/15 O.S. Emmetropic 20/50	Inconstant esotropia 0 to 17△D.	Snellen Letters: 11′ central or nasocentral inhibition Tangent Screen: No inhibition Stereocampimeter: No inhibition
55	35	O.D. 20/100; +2.00 20/100 O.S. 20/20; −1.25 cyl.×130 20/15	No strabismus Onset: infancy Positive family history	Stereocampimeter: 30′ scotoma Prism Test: 1 to 4° nasocentral inhibition, depending on surround Mirror-screen: ½° facultative scotoma; retinal rivalry elicited; dominance reversible
56	50	O.D. 20/25; +1.00+.62×100 20/15 O.S. 20/100; +3.25−.50×45 20/50	No strabismus Anisometropia Positive family history	Prism Test: 30′ central scotoma; Letters: 10′ central inhibition Stereocampimeter: 1° scotoma. Tangent screen: No scotoma Mirror-screen: ½ to 3° facultative scotoma; retinal rivalry elicited; dominance reversible
57	13	O.D. 20/20; +1.00 20/20 O.S. 20/60; +2.25 20/50	No strabismus Anisometropia	Stereocampimeter: 7′ scotoma, with binocular fixation; no scotoma with fixation O.S. Tangent Screen: No scotoma to a 2/1,000 test object Prism Test: 1 to 2△D. inhibition, nasocentral
58	27	O.D. 20/20; +3.50 20/15 O.S. 20/100; +4.00 20/100	Esotropia 7 to 10△D.	Prism Test: 2△D. nasocentral inhibition Mirror-screen: Retinal rivalry elicited; dominance reversible Snellen Letters: 11′ central and nasocentral inhibition Tangent Screen: No scotoma to 2/1,000 test object fixing center of circle of pins 1° in diameter
59	23	O.D. 20/20; +.25−.25×170 20/20 O.S. 20/200; +4.50−2.00×20 20/200	Anisometropia No strabismus Convergence normal Onset: infancy	Prism test: 3 to 5△D. central and paracentral inhibition Mirror-screen: Retinal rivalry Amblyoscope: Central and nasocentral inhibition Tangent Screen: 10′ to 30′ nasocentral scotoma
60	23	O.D. 20/70; +4.00−1.75×172 20/30 O.S. 2/200; +6.25−2.50×180 20/200	Anisometropia Intermittent accommodative esotropia Onset: infancy	Prism Test: 4△D. nasocentral inhibition Pinhole Prism Test: Retinal rivalry elicited; dominance reversible; facultative scotoma 1 to 2° Amblyoscope: 4 to 5° nasocentral facultative scotoma Mirror-screen: ½ to 5° facultative scotoma Tangent Screen: ½ to 1° central scotoma
61	43	O.D. 20/100; +3.50 20/100 O.S. 20/20; +1.25−1.00×80 20/20	Anisometropia No strabismus	Prism Test: 2 to 5△D. lateral inhibition Projection: Normal in direction; inaccurate in amount
62	31	O.D. 20/200; +4.25 cyl.×90 20/100 O.S. 20/40; +1.50 cyl.×90 20/25	Anisometropia No strabismus Onset: infancy	Prism Test; 1 to 2△D. lateral inhibition at macula Mirror-screen: Retinal rivalry elicited, favors O.S.; indefinite pericentral scotoma
63	27	O.D. 20/15; +.50+.25×25 20/15 O.S. 20/300; +2.00 cyl.×125 20/300	Esotropia; accommodative 0 to 25△D. Anisometropia Onset: infancy	Prism Test: 2△D. nasocentral inhibition Mirror-screen: Retinal rivalry elicited; dominance reversible
64	24	O.D. 20/400; −2.25−1.25×180 20/100 O.S. 20/25; −1.75 cyl.×125 20/20	Anisometropia No strabismus Onset: infancy	Prism Test: 3△D. nasocentral inhibition
65	11	O.D. 20/30; +.50−.25×15 20/20 O.S. 20/100; +2.75−1.00×160 20/40	Anisometropia No strabismus Convergence good Onset: infancy	Prism Test: 1△D. nasocentral inhibition Prism Pinhole Test: 5′ scotoma, facultative; retinal rivalry elicited; dominance easily reversible Tangent Screen: No scotoma
66	23	O.D. 20/15; Emmetropic 20/15 O.S. 20/70; +4.00 cyl.×75 20/70	Anisometropia No strabismus Onset: infancy	Prism Test: Prism over O.S., no inhibition. Prism over O.D., conjugate movement Pinhole Prism Test: 10 to 20′ facultative scotoma; retinal rivalry elicited, dominance reversible
67	26	O.D. 20/20; +1.50 20/20 O.S. 20/70; +2.75−.50×45 20/50	Anisometropia No strabismus Onset: infancy	Prism Test: No inhibition with prism over O.S.; over O.D., conjugate movement Depth perception present
68	30	O.D. 20/400; +5.50−1.50×180 20/100 O.S. 20/20; +.25−.25×90 20/20	Anisometropia No strabismus Onset: infancy	Prism Test: 2△D. nasocentral inhibition Pinhole Prism Test: Facultative 18′ scotoma; retinal rivalry elicited; dominance reversible
69	20	O.D. 10/200; +5.50 20/200 O.S. 20/15; +3.50 20/15	Anisometropia Esotropia 0 to 20△D. Accommodative	Prism Test: Nasocentral inhibition Amblyoscope: Central inhibition
70	21	O.D. 20/20; +.50−.50×15 20/15 O.S. 20/60; +3.00−2.00×165 20/40	Anisometropia No strabismus Onset: infancy	Prism Test: Prism over O.S., no inhibition. Prism over O.D., conjugate movement Fusion and stereopsis present
71	20	O.D. +4.75−2.25×15 20/40 O.S. +4.75−2.75×180 20/20	No strabismus Hyperopic astigmatism Onset: infancy	Prism Test: Prism over O.S., no inhibition. Prism over O.D., conjugate movement Fusion and depth perception. Most minimal case
72	22	O.D. 20/20 O.S. 20/200	Esotropia 4 to 10△D.	Prism Test: Nasocentral inhibition
73	21	O.D. 20/60; +1.50−2.00×145 20/50 O.S. 20/20; +2.75 20/20	Esotropia 0 to 28△D. Onset: Age 5	Amblyoscope: No inhibition at angle of squint. No inhibition at macula
74	18	O.D. 20/50; +1.00−.50×180 20/50 O.S. 20/25; +.50−.37×160 20/20	Esotropia 0 to 15△D.	Prism Test: 10△D. inhibition; normal projection between angle and macula Amblyoscope: Inhibition at angle; very slight inhibition at macula
75	29	O.D. 20/20; −.25−.25×155 20/15 O.S. 20/40; −.25−.50×10 20/40	No strabismus Convergence good	Prism Test: 2△D. inhibition at macula
76	20	O.D. 20/100; −2.75 20/40 O.S. 20/40; −1.00 cyl.×110 20/25	Anisometropia No strabismus Onset: infancy	Prism Test: Minimal to normal response
77	20	O.D. 20/40; +3.50−1.50×165 20/25 O.S. 20/60; +4.50−1.25×12 20/50	Anisometropia No strabismus Convergence good Onset: infancy	Prism Test: Normal response suggesting no inhibition, yet poor vision

campimeter with the nonamblyopic eye closed, a scotoma of 2 to 4° was found between the fixation point and the blindspot, the scotoma appearing to shift to the left as the test object approached from the right, and shifting to the right as it approached from the left. Looking at a line of letters on the Snellen chart at 20 feet, with the amblyopic eye, one entire letter on the 20/80 line would disappear, suggesting a 20′ central or nasocentral scotoma. The findings in this case show the variability of retinal inhibition as an adaptation process.

CASE 35.—An 18-year-old girl had had her right eye turned in since childhood. When seen at the age of 7, the eye was convergent 30 △ D. Fundi were normal. At age 12, strabismus measured only 6 △ D. At age 18, strabismus measured 10 △ D by cover test. Refraction: O.D.+3.50+.50×90=20/50. O.S.+3.50+.50×90=20/20. Fixation movements with the amblyopic eye were horizontal 3 △ D fluctuations. The fixated object seemed to drift to the left and when the patient moved her eye the object would go back to the original position. Looking at 3 or 4 letters in a row, the letter on the right was less distinct than the letter on the left. The normal blindspot could be mapped out readily on the tangent screen, but a central scotoma was not found with 1/1,000 test object, and the fixation point did not disappear. Careful stereocampimeter studies revealed no central scotoma. There was abnormal projection with the blindspot displaced nasally 5° when the patient was allowed to align the 2 charts so that they appeared fused, or as 1. With the mirror-screen test a minimal scotoma, approximately ½° was found at the angle of squint, but not at the macula or between the macula and the angle of squint. There was false projection in the area between the angle of squint and the macula.

With the mirror-screen test an attempt was made to stimulate both maculas at the same time by using in front of the amblyopic eye as a fixation object a triangular piece of adhesive tape 3 mm. in size marked with lines, and in front of the amblyopic eye various sized round white test objects. Retinal rivalry or replacement as found in normal eyes could not be elicited. Flashing prisms in front of the amblyopic eye with the normal eye fixing a 2 mm. test object on a black tangent screen at 1 meter, indicated a 1 △ D scotoma at the angle of squint, and false projection between this and the macula.

This patient obviously had amblyopia ex anopsia, yet it was impossible to find a definite central scotoma. She stated that when

looking at 20/40 letters in a line, at 20 feet, the letter on the right became dimmer than the one on the left of the fixation point, and that the letter she tried to read became dim, moved and came back, confusing the letters on the line. This suggested either a 10′ scotoma which should be readily measured by a 1/1,000 test object subtending an angle of 3.5′, or that the apparent movement of the letter resulted from a conflict between normal and abnormal projection near the macula. To lessen this possible conflict, single isolated E's were presented to the patient, and she could read 20/20 easily. This ability to distinguish single E's in contrast to letters in a line is further described in the section on visual acuity. This case indicates the importance of false projection as a basis for poor fixation and poor vision in amblyopia ex anopsia.

Analysis

In this group, visual acuity was usually 20/200 or better, only 1 of the 21 patients having less than 20/200 vision. Four of the patients had negligible refractive error. There were 3 instances of divergent strabismus. The degree of esotropia was quite variable. Although this group is classified as having the ability to fixate, fixation is not normal, excursions being apparent to even casual observation.

The inhibition tests showed inhibition or scotoma primarily at the angle of squint of varying degrees and extent, and false projection between this area and the macula. A true scotoma was difficult to demonstrate at the macula. The failure to elicit retinal rivalry is noteworthy. One might imagine that the macular area of the squinting eye is disregarded in binocular vision as a result of lack of attention rather than being actively inhibited as occurs in retinal rivalry. This is in agreement with the type of macular vision that alternating squinters have, namely, that when both maculas are presented with dissimilar images, both images can be seen simultaneously, projected into different places in space as if there were no binocular relationship.[13, 14]

Some of the patients showed strong fixed false projection with peripheral fusion sufficiently strong to force the strabismic eye to continue to turn in the direction of the original

squint, in order to keep prism-displaced images on the area of the retina originally at the angle of squint. This phenomenon is similar to that seen in cases of obligatory eccentric fixation described in Group 2.

Cases in this group show: (1) how extremely variable projection can be, depending upon the circumstances under which it is measured; (2) that the projection may be an adaptation to the squint; (3) that as alternating strabismus is approached, amblyopia is less, and false projection factors increased. Since true alternators do not have amblyopia ex anopsia and have markedly abnormal projection, one would expect a negative correlation between abnormal projection and amblyopia. Theoretically, in severe amblyopia fixed false projection is not necessary for adaptation and, conversely, if false projection is well established, amblyopia is unnecessary for avoidance of diplopia.

However, in this study, the presence of both indefinite projection and inhibition in the same area have been demonstrated repeatedly by use of the prism displacement test. It is as if there were a merging of these factors. These phenomena are frequently seen in an area adjoining one of absolute scotoma. In severe amblyopia one frequently finds an absolute scotoma adjoining the disc with an area of relative inhibition and indefinite projection between the scotoma and the macula, and at the macula failure of differentiation, explaining the lowered visual acuity and, in addition, fixed false projection so strong as to compel eccentric fixation (see Group 2). These findings indicate that the retinal sensory system has the capacity to develop inhibition, relative or complete, false projection, indefinite or definite, any or all of these phenomena being present in a particular case. In this group with strabismus and amblyopia, the prism displacement test reveals some degree of false projection in every case. As measured by other methods, the projection was normal in some instances and false in others.

In these patients the central scotoma, per se, is not suffi-

cient to explain the visual acuity. Since isolated letters can be seen better than similar-sized letters in a group, factors other than 2-point resolving power are concerned in the amblyopia. These factors would appear to be attention, false projection and poor fixation, causing letters seen in a group to become confused and less readily distinguishable than when seen singly (see section on visual acuity).

Relative to prognosis for ultimate return of vision in the amblyopic eyes in Group 3, a hopeful indication is that a degree of improvement could be attained comparable to the improvement seen when reading single isolated letters. In reading single letters, in contrast to letters in a group, factors of false projection and faulty fixation are minimized. Theoretically these functions could be trained more easily than 2-point resolving power could be improved, particularly if a central scotoma existed.

IV. Amblyopia without Apparent Strabismus or with Inconstant Strabismus, with Normal Retinal Projection and Relatively Good Ability to Fixate

CASE REPORT (Group 4)

CASE 56.—A 50-year-old woman had had "weak" eyes all her life but no history of strabismus. The mother and sister of the patient had a similar condition. The foveal region appeared normal in both eyes. Refraction: O.D.$+1.00+.62\times100=20/15$. O.S.$+3.25-.50\times45=20/50$. There was apparent orthophoria when looking at letters, but on closing the right eye, and attempting to read with the left eye, searching movements of 2 $\triangle$ D were apparent. On the stereocampimeter a 1° scotoma was found with a .4 mm. Evans test object. On the tangent screen the blindspot was readily found and no scotoma demonstrable using a 1/2,000 test object. In the mirror-screen test with the right eye behind the mirror the patient fixated between 2 pins separated by 2°. These were then projected onto the tangent screen seen by the amblyopic eye, and a 2/1,000 test object passed between the pins to determine the presence of a scotoma. Under these conditions a scotoma of ½° to 3° could be found, depending on the size of the test object. With the smallest test object, 1/1,000, a 3° scotoma was found; with 8/1,000

CHART 5.—GROUP 5.—ORGANIC LESIONS

Case No.	Age	Vision	Etiology	Fixation	Studies of Inhibition, Scotoma and Projection
78	38	O.D. 20/20; +.50−.50×60 20/20 O.S. 20/60; +.75−.75×150 20/40	Reddish plaque in macula	Can fix on scotomatous area	Prism Test: Prism over O.S., diplopia; Prism over O.D., conjugate movement Minimal ocular dominance Snellen Letters: 15′ scotoma
79	36	O.D. 20/20; +.50 20/20 O.S. 20/60; +.50−.50×180 20/40	Granular macula; eclipse burn at age 16 years	Centric	Prism Test: Normal response; no ocular dominance Snellen Letters: 10′ scotoma Tangent Screen: 20′ scotoma
80	11	O.D. 20/100; −4.50 20/25 O.S. 20/200; −4.50 20/50	Hole in macula; eclipse burn	Searching movements 1 to 2△D.	Tangent Screen: 1 by 1/2° scotoma Stereocampimeter: 1° scotoma
81	52	O.D. 20/50; +1.25−.37×170 20/40 O.S. 20/20; +1.00−.37×170 20/20	Central angiospastic retinopathy	Centric	Prism Test: Normal response Stereocampimeter: 7′ scotoma Tangent Screen: 10′ central scotoma Snellen Letters: 10′ to 20′ scotoma
82	20	O.D. 20/50; +.50−.25×30 20/50 O.S. 20/20; +.75 20/20	Eclipse burn; veiled macular markings, pigmentary change	Centric	Prism Test: Normal response
83	20	O.D. 20/200; Unimproved 20/200 O.S. 20/20 20/20	Gun explosion; age 8 Granular macula; no strabismus	Centric, slightly wavering	Prism Test: 1 by 3½° scotoma
84	27	O.D. 20/20; Emmetropic 20/20 O.S. 20/70; +1.50−.62×90 20/60	Concussion blow at age 12 years; granular macula No strabismus	Centric, slightly wavering	Prism Pinhole Test: Dominance easily reversible Mirror-screen Test: Retinal rivalry favors O.D.; 2° scotoma; inhibitory as well as absolute Snellen Letters: 10′ scotoma Tangent Screen: 10′ scotoma Prism Test: 1 to 1½° nasocentral scotoma; dominance indefinite
85	26	O.D. 20/20; Emmetropic 20/20 O.S. 20/200; −.25+.75×100 20/200	Concussion blow at age 24 years; no strabismus Break in retina and choroid at macula	Centric, slightly wavering	Prism Test and Tangent Screen Test and Pinhole Prism Test: 2° temporocentral scotoma
86	20	O.D. 20/60; −.75−.50×175 20/20 O.S. 20/50; −1.00−.50×180 20/20	Eclipse burn, age 10	Centric	Prism Test: Normal response. Small scotoma on grid Tangent Screen: Scotoma Snellen Letters: 10′ scotoma
87	33	O.D. 10/200; +1.00+1.25×70 20/100 O.S. 20/20; +.25+.50×95 20/20	Injury O.D. age 3; strabismus 4 to 5 prism diopters Macular reflex poor Dilated pupil	Centric, slightly wavering	Prism Test: 1° scotoma. Ocular dominance marked Tangent Screen: Indefinite scotoma Amblyoscope: Inhibition at macula
88	20	O.D. 20/50; +.50−.75×180 20/40 O.S. 20/20; +.50−.37×165 20/15	Hole in macula	Centric	Prism Test: Normal response Snellen Letters: 10′ scotoma

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CHART 7.—LIGHT DIFFERENCE DISCRIMINATION

Case No.	Corrected Vision	Ratio: Recognizable Intensity Difference						Fixation Movements	Approximation of Absolute Scotoma
		Fixation Central Ambl./Normal		Peripheral/Central Fixation					
		Spot—12°	Spot—1°	Spot—12°		Spot—1°			
				Temp.	Nasal	Temp.	Nasal		
Amblyopia Ex Anopsia									
57	O.D. 20/20 O.S. 20/50	1.8	1.3	1.2 1.0	2.1 0.9	2.3 2.2	3.5 2.5	Searching excursions 3△D., amblyopic eye Large spot: 5△D., both eyes Small spot: 5△D., amblyopic eye	Stereocampimeter: 7′ paracentral scotoma
34	O.S. 20/15 O.D. 20/200	1.4	1.5	1.2 0.9	1.0 1.1			Searching excursions 4 to 8△D., amblyopic eye Large spot: 5 to 15△D., amblyopic eye Large spot: 5 to 10△D., good eye	Letters: 20′ paracentral scotoma
2	O.D. 20/15 O.S. 20/400	0.8	2.8	1.7 1.6	1.4 1.5	2.9 1.0	3.1 1.1	Irregular, 5 to 10△D., amblyopic eye Large spot: 10 to 15△D. with left; 5 with right Small spot: 10 to 15△D. with left; 5 with right	Tangent Screen: 1½ to 2° nasocentral scotoma
9	O.D. 20/15 O.S. 20/200	1.4	2.1				1.5	Eccentric; 10△D.; amblyopic eye	Tangent Screen: 10° cecocentral scotoma Prism Test: 5° cecocentral scotoma
3	O.S. 20/15 O.D. 20/200	1.5	1.5					Nystagmoid: 15△D., amblyopic eye; 1 or 2△D., good eye Large spot: gross, searching with right; 5△D. with left Small spot: gross, searching with right; 1 to 2 with left	Tangent Screen: 3° nasocentral scotoma
7	O.D. 20/15 O.S. 5/200	1.7	1.6					Eccentric fixation, 30△D. esotropia. Irregular, 4 to 15△D. movements, left eye Right eye: large spot 5△D.; small, 1 to 2△D.	Tangent Screen: 10 by 4° centrocecal scotoma
4	O.S. Anoph. (related to control) O.D. 12/200	0.7	1.0	1.1	1.3	2.7	2.0	Large spot: irregular, 3 to 4△D. jerks Small spot: more gross excursions than with large spot	Tangent Screen: 2° centrocecal scotoma, touching fixation point
1	O.S. 20/20 O.D. 5/200	1.1	5.0			2.9 1.4	2.6 1.3	Searching, random, 15△D. excursions Large spot: 15 with amblyopic, 5△D. with left eye Small spot: 15 with amblyopic, 2 or 3 with left eye	Tangent Screen: 2 to 4° cecocentral scotoma
8	O.D. 20/20 O.S. 15/200	1.4	1.9			2.7 1.0	2.4 1.4	Eccentric fixation Irregular jerking movements	Tangent Screen: 3° cecocentral scotoma touching blind-spot
56	O.D. 20/15 O.S. 20/50	1.1						Large spot: 5△D. excursions both eyes	Snellen Chart: 22′ scotoma
Organic Lesions of the Macula									
78	O.D. 20/20 O.S. 20/40	1.0	1.2 (45′) (30′) (20′) not seen 1.6 2.4	1.2 1.0	1.5 1.0			Slightly irregular Left eye: large spot; 5△D.; small, 1 or 2△D.	Letters: 15′ scotoma Judged by disappearance of spot: 20′ scotoma Light difference discrimation decreased with smaller spots
79	O.D. 20/20 O.S. 20/60	1.2	1.0					Large spot: 5 to 8 with right; 5 to 15△D. with left Small spot: 5△D. with left; only 2 to 3 with right	Letters: 10′ scotoma
80	O.D. 20/25 O.S. 20/50	0.9	1.0	1.1 1.1				Large spot: 5 to 10△D. movement with both eyes Small spot: 5△D. with both eyes	Tangent Screen: ½° scotoma Stereocampimeter: ½° scotoma
81	O.S. 20/15 O.D. 20/40	1.2	1.3	1.5 1.5 1.4	1.5 1.5 1.4	2.0 2.0 1.6	1.8 1.8 1.4	Large spot: 5 to 10△D. movements of both eyes Small spot: 2△D. movements with both eyes	Letters: ½° scotoma
Normal Controls: Ratio of Right to Left Eyes									
	Normal vision both eyes	1.07 1.09 1.00 1.15 1.07	1.00 1.04 1.17 1.04					Large spot: fixation movements the same with either eye approximately 5△D. Small spot: fixation movements definitely less than with large spot, 1 to 3△D.	

CHART 8.—VISUAL ACUITY

Case No.	Strabismus	Refraction	Snellen	Circles Ferree-Rand	Two Point Discrimination			Movable Black Squares	Single E's	Letters C's	Line of E's	Scotoma
					Lights	Black Dots on White	Bright Pins on Black					
9	Esotropia 20 D.	O.D.: Emmetropia O.S.: +1.00×110	20/20 20/1,300		20/20 20/1,200	20/20 20/1,200	.. ..	.. ..				Prism Test: 10° temporocecal
38	Esotropia 10 D.	O.D.: +5.00−4.00×15 O.S.: +1.00−.25×170	20/400 20/20		20/600	20/600	..	..				Prism Test: 1° nasocentral
62	None	O.D.: +4.25 cyl.×90 O.S.: +1.50 cyl.×90	20/100 20/25		20/100	20/100	..	..				Prism Test: ½° paracentral
58	Esotropia 7 to 10 D.	O.D.: +3.50 O.S.: +4.00	20/15 20/100		20/50	20/50	..	20/30				Letters: 11′ nasocentral Prism Test: 1° nasocentral, facultative
63	Esotropia 0 to 25 D.	O.D.: +.50+.25×25 O.S.: +2.00 cyl.×125	20/15 20/300		20/200	20/200	..	..				Prism Test: 1° nasocentral, facultative
64	None	O.D.: −2.25−1.25×180 O.S.: −1.75 cyl.×125	20/100 20/20		20/70	20/70	..	..				Prism Test: 1½° nasocentral facultative
59	None	O.D.: +.25−.25×170 O.S.: +4.50−2.00×20	20/20 20/200		20/200	20/100	20/100	..				Prism Test: 2° centronasal Tangent Screen: 7′ nasocentral
94	None	O.D.: Emmetropia O.S.: +4.00 cyl.×75	20/15 20/70		20/70	20/70	..	..				Prism Test: ½° paracentral, facultative
60	Intermittent	O.D.: +4.00−1.75×172 O.S.: +6.25−2.50×180	20/30 20/200		20/100	20/200	..	..				Prism Test: 1½° central or nasocentral facultative
92	None	O.D.: +2.50+2.50×95 O.S.: +2.50+2.00×80	20/50 20/20		20/50 20/20	20/50 20/20	20/50 20/20	20/30 20/20				
93	None	O.D.: +2.00−.25×90 O.S.: +2.00−.25×170	20/20 20/50		20/20–50 20/100–20	20/15 20/50	20/20 20/75	20/15 20/30				Prism Test: ½° central, facultative
55	None	O.D.: +2.50 O.S.: −1.25	20/100 20/20	20/175<..	20/70–200<..	20/150<..	..<..	20/30<..				Prism Test: ½° facultative Tangent Screen: 7′ central
34	Esotropia	O.D.: +4.50 O.S.: +4.00−.25×5	20/200 20/15	20/125	..<..	20/100<..	20/50<..	20/50<..				Prism Test: 3° nasocentral facultative Letters: 20′ central
2	By history	O.D.: O.S.: +4.00+1.00×95	20/20 20/600		20/20 20/400	20/20 20/400	20/20 20/300	20/20 20/400	Looks to left to see E<..	..<..	..<..	Prism Test: 1½° nasal or nasocentral
56	None	O.D.: +1.00+1.75×102 O.S.: +3.25−.50×45	20/15 20/50	20/25 20/85	20/20–50 20/50–200	20/15 20/70	20/15 20/50	20/15 20/30	20/15 20/40	.. 20/50	.. ..	Prism Test: 30′ central scotoma Letters: 10′ central; stereocampimeter: 1° central
4	By history	O.D.: Emmetropia O.S.: Anophthalmic	15/200		20/400–50	20/400–50	20/400–50	20/40	20/50–40	20/70	20/200	Tangent Screen: 2 to 3° nasocentral
3	Esotropia 30 D.	O.D.: +1.25×125 O.S.: +.25+2.75×84	15/200 20/15		20/200<..	20/100<..	20/200<..	..<..	20/60<..	..<..	20/200<..	Tangent Screen: 1½° central scotoma
54	Esotropia inconstant	O.D.: Emmetropia O.S.: Emmetropia	20/15 20/50		..	..	..	..	..	..	20/15 20/25	Letters: 15′ central scotoma
1	By history	O.D.: −.50+.75×180 O.S.: Emmetropia	5/200 20/20		..	..	..	..	..	..	20/100	Prism Test: 2 by 4° cecocentral Tangent Screen: 10° cecocentral
35	Esotropia 10 D.	O.D.: +3.50+.50×90 O.S.: +3.50+.50×9	20/50 20/20		..	..	..	..	..	..	20/20	8 to 10′ nasocentral
8	Esotropia 2 D.	O.D.: +.50−.37×50 O.S.: +.75	20/20 20/200		..<..	..<..	..<..	..<..	..<..	..<..	20/20 20/100	Tangent Screen: 3° cecocentral
89	Esotropia 20 D.	O.D.: −2.25+3.25×115 O.S.: −2.25+3.25×75	20/70 20/40		..<..	..<..	..<..	..<..	20/30<20/30	..<..	20/70<..	
90	Esotropia 20 D.	O.D.: +1.50−.50×15 O.S.: +1.00	20/30 20/70	20/70	..<..	..<20/40	..<..	..<20/20	20/25<20/25	..<20/25	..<20/70	
91	Esotropia 5 to 20 D.	O.D.: +3.50+.75×70 O.S.: +3.00+.75×90	20/70 20/30		..<..	..<..	..<..	..<..	20/25<20/25	20/40<..	20/70<..	
79	Organic etiology	O.D.: +.50 O.S.: +.50−.50×180	20/20 20/50–40		..	..	..	..	20/40	..	..	Letters: 10′; Tangent Screen: 20′ scotoma Eclipse burn; fixation normal; no ocular dominance
78	Organic etiology	O.D.: +.50−.50×60 O.S.: +.75−.75×150	20/20 20/40		..	..	..	..	..<20/40	..<..	..<..	Hole in macula; fixation normal; minimal ocular dominance; letters: 15′; stereocampimeter: ½° scotoma
					extra letters help patient focus							
87	Organic etiology	O.D.: +1.00+1.25×70 O.S.: +.25+.50×95	20/100 20/20		..<..	..<..	20/100<..	..<..	..<..	..<..	..<..	Prism Test: ½° central scotoma; tangent screen less; contusion injury; macula mottled

a ½° scotoma was found. With the right eye occluded, the patient fixated between 2 pins placed at similar distances on a tangent screen and no scotoma was discernible.

The scotoma seemed to be an inhibition scotoma induced by the image from the sound eye. In a stereoscope a black card with a white fixating pin was placed in front of the right eye and a red card in front of the left eye. The white pin and black background stood out. The right eye was then blurred with a plus 4 lens, equalizing visual acuity in the 2 eyes, at the testing distance. The white pin on the red background then stood out, showing reversal of ocular dominance. This was more apparent if a gold-colored ring were placed around the pin projected on the red card.

This patient had normal projection, facultative scotoma only, and reversal of retinal rivalry and ocular dominance if the attention value of the image in the amblyopic eye was sufficiently enhanced.

Careful visual acuity studies were repeated a number of times on this patient, with the final results as follows: Snellen letters 20/50 with recognition of 1 or 2 letters on the 20/40 line. Only 4 letters were seen when 5 letters were present on this line. The middle letters were most difficult to see, tending to run together. Number chart, E's in a line and Landolt circles all gave 20/50 visual acuity. Presenting single E's on a white background, the patient interpreted the position of the 20/50 E readily and guessed at the 20/40. Using single circles, the same results were obtained. The single E was seen a little better than the circle. Two-point discrimination was checked, using 2 black dots on a white card and bright pins on a black tangent screen (see section on visual acuity). Acuity varied from 20/70 to 20/50. When the points were in a vertical line, discrimination was easier than when horizontal.

This patient was tested on a number of different days and each time fixation movements were steadier as continued effort was made. The original estimation was 2 to 4 △ D fixation excursions, and the final appraisal was recorded as nearly normal.

Analysis

Most of the patients in Group 4 showed anisometropia of varying degrees. Only 2 patients in a series of 24 had no significant refractive error. As in Group 3, fixation movements were searching and 1 to 3° in extent. The severity of the amblyopia was comparable to that seen in Group 3. Only 1 case had less than 20/200 vision. The easily demon-

strable retinal rivalry is in keeping with normal retinal projection and no strabismus. Ocular dominance and retinal rivalry could be changed from the normal eye to the amblyopic eye in many instances by increasing the interest value of the image presented to the amblyopic eye over that presented to the normal eye. The facility with which this could be done might be used as a measurement of the degree of amblyopia.

Absolute scotoma is definitely recorded in only 3 cases (Cases 55, 59 and 60), the largest scotoma being 1 degree. In the other patients only a facultative scotoma dependent upon binocular vision was recorded. It is possible that a minimal absolute scotoma within the facultative scotoma, which of itself does not explain the visual acuity, interferes with development of the fixation reflex. Fixation in this group was better than in Group 3, but, even so, fixation movements were grossly apparent. It is interesting that this group with normal projection did not show as much improvement in acuity when viewing single letters compared to a group of letters, as did the group with false projection (Group 3).

One might surmise that inhibition attending normal retinal rivalry is the mechanism allowing inattention to the blurred image in the amblyopic eye and consequent failure to develop the fixation reflex. That this modus operandi is not a complete explanation is apparent from the fact that many patients with rather marked degrees of anisometropia show no amblyopia, even though the condition has always been present. The high incidence of a family history of squint or amblyopia in these cases suggests additional etiologic factors.

One might assume that this group would have the best prognosis, having the fewest obstacles to normal vision to overcome. Paradoxically, consideration of the findings would lead one to offer a poor prognosis. Fixation is relatively good, there is fusion of the image of the amblyopic eye with that of the good eye, binocular habits are normal. There are, then, fewer bad habits to correct and the question arises of what to do to improve vision. The failure of vision to develop even if

the eye is used normally suggests an organic defect. Evans[4] is of the opinion that none of this group improves completely; that these patients simply learn to use an isopter closer to the central area, but never at the central area, where a small absolute scotoma exists.

V. Amblyopia Resulting from Organic Disease

Thirty patients with small lesions of the macula were studied. Data on scotoma and inhibition were sufficient on only 12 of these to be of value for comparison with cases of amblyopia ex anopsia.

CASE REPORT (Group 5)

CASE 81.—A 52-year-old housewife had normal eyes until 3 years ago when a central scotoma developed suddenly, large enough to blur the width of 1 column in the newspaper. This cleared gradually, but left reduced vision which has remained unchanged for a year. Refraction: O.D.$+1.25-.37\times170=20/40$. O.S.$+1.00-.37\times170=20/20$. The fundus of the right eye showed loss of the foveal reflex and mottling of the macula. On the tangent screen and stereocampimeter a 20′ central scotoma was readily found. As judged by the loss of 1 letter on the 20/40 line, a 10′ scotoma was estimated. Fixation movements were normal. Prism test: the patient experienced diplopia when a 1 diopter prism was flashed over either eye in any position. There was no tendency for movement of the eyes. This was a normal response.

Analysis

Eyes with organic lesions of the macula show interesting differences from amblyopic eyes. The patients with organic lesions could more readily hold fixation either to fix with the scotomatous area or with an area eccentric to the scotoma. The scotoma was readily found by all methods and was much larger in proportion to the reduction in visual acuity than that found in amblyopic patients. Nevertheless the visual acuity in some of the organic patients seemed less than one would predict from the size of the scotoma as judged from peripheral acuity ratings published in the literature.[3, 15, 16, 17]

A few of the patients showed what appeared to be an inhibition area surrounding the absolute scotoma, suggesting that inhibition factors similar to those in amblyopia may develop subsequent to organic lesions (Cases 84 and 85). However, in most cases inhibition outside the absolute scotoma was minimal. Retinal projection was normal.

CHART 6.—PERIPHERAL VISUAL ACUITY

Eccentricity (Central Scotoma) Degrees		*Ludvigh*[15]	*Sloan*[16]	*Evans*[3]	*Weymouth*[17]	*Wertheim*[17]
Diameter	*Radius*					
0	0	20/11	20/15	20/15		20/20
½	¼	20/12			20/23	
	⅓			20/22		
1	½	20/13	20/27		20/24	
2	1	20/17	20/30	20/40	20/30	20/23
3	1½				20/45	
4	2	20/27	20/38			
	2½			20/95		
	3		20/50			
10	5	20/45	20/70	20/150		20/40
14+		20/100+				
20	10	20/77	20/150			20/70
	12		20/200			
25+		20/200				

These cases of organic lesions further indicate that the scotoma alone in amblyopia ex anopsia is too small to account for the visual acuity. Eleven cases of holes in the macula were seen who had 20/20 vision. Central scotoma was readily demonstrable in these cases. The exact size of the scotoma was not recorded and these cases are not included on the chart. Nevertheless they further emphasize the difference between the effect of the scotoma on vision in acquired organic lesions and that in cases of amblyopia ex

anopsia. Chavasse[2] has published cases of congenital holes in the macula with vision and fixation movements simulating those found in amblyopic patients. One differentiation of the congenital from the acquired form of organic lesions with scotoma at the macula is the variation in type of fixation movements. In amblyopic patients without demonstrable scotoma one might assume a lack of differentiation of the macula with failure of development of the fixation reflex to explain the reduced visual acuity.

LIGHT DIFFERENCE SENSITIVITY

Burian and Wald[18] have reported that the amblyopic eye, in the condition known as amblyopia ex anopsia, can see minimal stimuli of light and color when in the dark-adapted state, to the same degree as the good eye. They concluded that visual acuity and minimal perception of light are separable functions. Ludvigh[19, 20] tested the light discriminatory sense in the light-adapted eye, using high levels of illumination and found in some instances sensitivity of the amblyopic eye essentially the same as in the sound eye, while in other instances the light difference sensitivity in the amblyopic eye was anywhere from 5 to 20 times as poor as that of the sound eye. These results suggested to him that (1) if the patients pass through a transitional stage from normal to extremely subnormal light difference sensitivity, they must do it quite quickly, since no one was observed in a transitional stage in which the amblyopic eye was only 2 or 3 times as poor as the second eye; (2) the patients in the first group responded better to occlusion than the second group, and for this reason the test might be used as a prognostic device; (3) the test demonstrated that light difference sensitivity and visual acuity are distinct and separate functions.

In an attempt to classify amblyopes into the groups suggested by Ludvigh, light difference discrimination was tested on a series of patients, including cases of amblyopia ex anopsia, cases with organic scotomata and normal controls.

Procedure and Apparatus

Two 100-watt Eastman Kodaslide projectors were focused on an opalescent screen from behind and, in the final experiments, slides with apertures of different sizes were used in order that 1 projector should illuminate a large background circle of light and the other furnish a small, brighter spot upon this background. To the observer, seated 19 inches in front of the opalescent screen, was visible the large background circle of light subtending an angle of 40° at the nodal point of the eye, and in the center of this circle a smaller, brighter spot of light. The measurement of this smaller spot in one series of experiments was 12°, and in a second series 1°. A variable transformer (General Radio 200-B Variac) was attached to the second projector to change the illumination of the spot.

To prevent too great a spectral shift into the red as illumination was diminished, and to permit greater accuracy in calculating relative intensities from voltage formulas, a neutral density filter, transmitting only 25% of the light, was placed in front of the spot. In a few instances a filter transmitting only 10% of the light was used in order that the voltage be at a higher level where conversion formulas are more accurate. The results with the 10% filter checked satisfactorily results with the 25% filter, and results with the 25% filter are recorded in Chart 7. The patient's head was supported on a chinrest to keep the eye at the proper distance from the screen. The spot was flashed on and off the center of the screen at irregular intervals, with exposures of approximately ¼ second, controlled by interposing a card in the beam of light from the second projector. The patient was asked to observe the spot on the center of the screen and indicate its presence by tapping on the desk each time it became visible. The illumination of the background, as measured by a Luckiesh-Taylor brilliance meter, was 13 ± 2 foot lamberts, which is equivalent to 850 photons of retinal illumination, when the pupillary diameter is 5 mm. It is

noted from Hecht's work[21] that the light difference detectable for normal observers is fairly constant after the intensity reaches 260 photons. Since the intensities were above this level, it was not necessary to know the exact diameter of the pupil, although this was recorded in order that no gross abnormality go unobserved. The results interrupting the beam of light with a card were compared with those obtained by use of a camera shutter calibrated to give a $^1/_{25}$ second exposure, and were found to be the same by either method in a series of normals. The click of the shutter introduced a distracting auditory factor, so use of the card seemed preferable. The variable transformer, controlling the intensity of the spot, was turned so that the spot became fainter and fainter until it was barely perceptible, while flashed on and off. The procedure was repeated a number of times until consistent results were obtained. At each end point, the illumination was measured by a Weston photronic cell connected to a low resistance micro-ammeter and the intensities of the background and spotlights computed directly and the increment of light necessary for recognition of a difference recorded. This method of calculation decreased the error from possible change of line voltage and variance in the burning of bulbs. It introduced an error due to inability to read the micro-ammeter scale within 2 to 5%.

On consideration of the results calculated by the direct method, it was noted that all the amblyopic eyes were within the 5% difference appreciation which would not be significant considering the range of error of the instrument reading, and still the voltmeter readings indicated a distinct and consistent difference between the normal and amblyopic eye in several instances. Since the problem was primarily one of relating the relative increase in light of the spot to that of the background for the amblyopic eye as compared with the normal eye, the expedient of utilizing the empirical voltage luminosity formula as determined by the Bureau of Standards for this type of bulb[22] was adopted. When I and $I + \triangle I$

are 2 light intensities which can just barely be perceived as different, then the ratio $\triangle I/I$ is considered the measure of intensity discrimination.[23] Taking the voltage readings obtained with the incremental spot hooked to the variable transformer, we calculated the relative luminosity of $\triangle I_1$ to $\triangle I_2$, the increments required for recognition by the amblyopic eye and the normal eye respectively. The calculation is as follows:

$$\frac{\text{lumens}}{\text{LUMENS}} = \left(\frac{\text{volts}}{\text{VOLTS}}\right)^{3.38}$$

This is interpreted to read: "The luminous output at voltage V_1 is to the luminous output at voltage V_2 as $V_1^{3.38}$ is to $V_2^{3.38}$." For example, Case 79: the voltage required for the normal eye to perceive an increment $\triangle I_2$ was 68, and for the amblyopic eye to perceive the increment $\triangle I_1$ 71, the resulting ratio

$$\frac{\triangle I_1}{\triangle I_2} = \left(\frac{71^{3.38}}{68^{3.38}}\right) = 1.04$$

The normal eye was tested first, and then the amblyopic eye, using first the 12° and then the 1° spots.

Results

It is seen that the amblyopic eye required slightly more light than the normal eye to see the 12° spots. Six of the 10 amblyopes showed a difference between amblyopic and nonamblyopic eyes. When the periphery of the retina was stimulated, by having the patient look 20° to the side, at the edge of the large field, and judging when the 12° spot appeared, again there was slight but questionably significant difference between the normal and the amblyopic eye, the normal eye showing more of a gradient between central and peripheral light difference appreciation than the amblyopic eye.

Using a 1° spot there was a greater difference between the amblyopic and the nonamblyopic eye. Eight of the 10 amblyopic eyes required significantly more illumination to see

the 1° spot than did the nonamblyopic eye. Using the periphery of the retina, 20° nasal or temporal, the gradient of central to peripheral light discrimination was significant in the normal eye, the peripheral retina requiring definitely more light to appreciate the spot than did the central area. The gradient was insignificant in the amblyopic eye, the readings for the central and peripheral retina being approximately the same.

The fixation movements recorded on the chart were estimated by the author observing the patient's eyes and are accurate only to the extent that an observer experienced with the cover test can estimate movement of the eye. The fixation movements increased in amplitude as the end point of the light discriminatory observations were reached, the maximum movements being recorded. With the large 12° spot, the fixation excursions were relatively gross even with the normal eye. With the 1° spot, the fixation movements were definitely smaller in the normal eye, but still grossly searching in the amblyopic eye.

From this observation of eye movements and the finding of no such definite gradient between central and peripheral retina in the amblyopic eye as is found in the nonamblyopic eye, one might assume that the amblyope when supposed to fix centrally, actually uses retina outside the macular region. This would explain the relative lack of gradient between central and peripheral vision.

Eyes with small organic macular lesions resembled normal eyes in discrimination of both 12° and 1° spots but showed slightly less gradient from center to periphery. Fixation was good, as in the normal eyes, the spot being larger than the scotoma. In Case 78 the spot was made progressively smaller, to a size covered by the scotoma, with resultant findings of proportionately increased light difference necessary for discrimination as the projected spot approached the size of the projected scotoma.

In early experiments done with apparatus which was

not as well controlled as that used in the reported experiments, similar findings were noted, plus the fact that the difference between the normal and the amblyopic eye was increased by using a smaller background and smaller spots. The difference in fixation movements is exaggerated under such conditions, the normal eye fixating well and the amblyopic eye poorly. The amblyopic eyes requiring the greatest increase in intensity of spot over background were those with poorest fixation and a large scotoma.

Since estimation of fixation movements and measurements of scotoma are comparatively simple, these tests at present for classification of amblyopic patients would seem to have greater clinical applicability than estimation of light difference sense. To study accurately contrast interpretation or light difference sense as a quantitative photochemical response in the area of the retina concerned in amblyopia, fixation movements must be accurately controlled. In addition, the illuminations of background and spot and the sizes of background and spot must be carried through more extreme variations. The extreme differences in light difference sensitivity between normal and amblyopic eyes encountered by Ludvigh were not found in the series of patients studied with the above-described apparatus using spots of 12° and 1°.

VISUAL ACUITY

Visual impressions originating in the retina can be classified into light sense, light projection, motion sense, color sense and form sense. All of these may be affected to some degree in severe amblyopia, but the form sense is primarily affected. The visual acuity of a number of amblyopic patients was tested by several methods.

Procedure and Apparatus

The visual acuity was determined with Snellen charts illuminated with approximately 25 foot-candles and viewed at a distance of 20 feet. The normal eye was occluded and

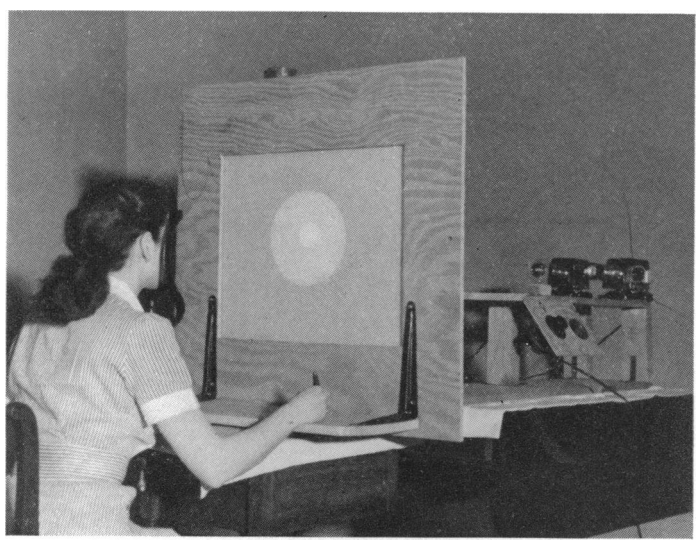

Fig. 5.

the correction, previously determined by atropine refraction, worn before the amblyopic eye. The patient's fixation movements and description of what he saw were recorded. The visual acuity of the normal eye was reduced to that of the amblyopic eye by blurring with lenses, and the difference in appearance of the letters to the two eyes was described.

Two-point discrimination was then measured, using as points a number of different-sized objects, the most satisfactory being two black dots on a white card viewed at different distances. Two sizes of dots were employed. The first was the smallest size that could be seen as a dot at 4 or 5 meters. Two such dots, whose separation was variable, were viewed at 5 meters distance. The minimum separation at which the two dots could be distinguished as two, was recorded for this distance and for other shorter distances, such as 4, 3, and 2 meters. The visual acuity was calculated by determining the visual angle subtended at the eye by the two dots. The second size of dot used corresponded in diameter to the limb of the smallest visible Snellen letter. Two such dots separated by a distance less than the diameter of one, were viewed at a distance of 5 meters and the card approached toward the observer, until the two dots were distinguished. The visual acuity was calculated as above.

In addition, two black 8 mm. squares placed on white cards, fashioned in the form of a slide rule so that the squares could be separated or brought together, were similarly used. The higher acuity values obtained with this test are explained by the fact that break in continuity rather than two-point discrimination was being tested.

A special apparatus for determining two-point discrimination of light was made. In a metal disc were drilled ½ mm. round apertures, in pairs, separation of which was calculated to correspond with Snellen chart acuity ratings, when viewed at a distance of 2 meters. This disc was mounted on a flashlight with parallel ray reflector, single lamp and 3 dry cells. To prevent diffusion of light from the holes, a diffusing plastic

sheet was cemented behind the light port. The observer was shown one point of light, then two points at separations corresponding to acuity ratings as recorded in column 2 of Table 1. The observer stated when he could distinguish two points of light.

TABLE 1.

Instrument Reading	*Equal to Snellen*	*Edge-to-edge*		*Center-to-center*
		mm.	*Inches*	*Inches*
1	. . .	.00	.00	.00 (one hole only)
2	20/20	.59	.0232	.0429
3	20/30	.83	.0327	.0524
5	20/50	1.50	.0591	.0787
10	20/100	3.00	.1181	.1378
20	20/200	6.00	.2362	.2559
40	20/400	12.00	.4724	.4921

A similar system was devised using ordinary straight steel pins, with heads filed to a uniform diameter of 1.2 mm., placed on a tangent screen and viewed at distances of 2 and 4 meters. In most cases the pinhead subtended an angle on the retina smaller than that subtended by the identifying points on a recognizable Snellen letter. Under cover from the patient, a second pin was then placed, varying the distances from the first pin until the minimum distance at which the two pins were recognized as two was determined. Third, fourth, fifth and sixth pins were placed in line with the second pin, but at distances slightly less than that separating the first two pins. The pattern would then be recognized as a line with one pin separated from it at one end. The effect of the line on the recognition of the original separation was then determined. The procedure was repeated with the pins lined in various directions. The pins served as points of light but with less interfering radiation than the instrument described above, for the pins were viewed in a room under ordinary

conditions of illumination. The distance separating the pins was measured as the distance between the edges of the pins.

Comment

Chart 8 shows a rather marked variation in visual acuity, depending upon the method by which it is measured. Letters could be identified with slightly greater ease by the literate person because of ability to use additional learned clues, whereas the reverse was true in children just learning their letters. A child's visual acuity, as determined by use of the E test[24] is better than when tested with Snellen letters, whereas an adult's visual acuity, as determined by the Ferree-Rand or Landolt circle chart,[24] is slightly less than when determined with letters. If a patient knows that he is simply to recognize a break between two points, or the direction in which a letter is pointing, as in the E test, discrimination is easier than if he has no previous conception of what is to be identified but is simply told to identify points within a pattern.

The examiner taking vision on an amblyopic patient is impressed by a number of phenomena:

1. The increased oscillatory fixation movements of the amblyopic eye which accompany the patient's efforts to see. These movements apparently increase, with an increase in the effort to see, and are proportionately greater as visual acuity is decreased.

2. The relative improvement of vision as the effort to see is continued. The first impression of the patient's vision may indicate 20/400, and this may improve to as much as 20/70 in a matter of minutes after a series of tests have been made on the patient. It is as if the patient learns to concentrate more as he becomes aware of the result of his efforts. Fixation movements seemed to be better controlled with the practice obtained during the testing.

3. The patient's interpretation of what he sees. He states that he can see the letter, that it is black, not blurred, but

that the identifying points of the letter cannot be determined, or that the letter on a line is lost in relation to other letters. For example, he may be able to say that in a certain row he sees a letter, as "D," but is unable to state what position that letter occupies. Other amblyopes may note a "fading" of the fixated letter and others describe a "crowding together" of the letters on a line as if the macular area and a portion of the retina to one side of it project in almost the same direction. An amblyope gave the reason for his being unable to hold the eye steady as the fact that the fixated letter moved over toward the letter beside it and seemed to merge with it. Then he felt compelled to move his eye to follow it. When letters are pointed out on a Snellen chart, the amblyope frequently reads the letter next on the nasal side if the amblyopia is associated with convergent strabismus (Cases 60 and 90).[2]

If the good eye is blurred to a visual acuity equal to that of the amblyopic eye the contrast to the patient is obvious, i.e., to the good eye the minimal visible letter is blurred and hazy, whereas to the amblyopic eye it is black, easily seen, but uninterpretable. The amblyopic eye can see a point stimulus, such as a black dot, or a point of light, many times smaller than the minimal size letter than can be interpreted, whereas the good eye, blurred to the same visual acuity as the amblyopic eye, is unable to see a similar stimulus because of the diffuseness of the image. It is well known that a dot or spot of light depends upon contrast for visibility, that is, its perception varies with its brilliance and not its size, whereas the interpretation of letters depends on ability to differentiate two points. If the letters are large enough the amblyope can interpret them correctly. There is no defect then in the synthesis of stimuli into pattern providing the original units of two-point discrimination are resolved. Apparently synthesis of stimuli at a lower level, two-point discrimination, is an important factor at fault. Ability to judge alignment and contour is probably also involved, but theoretically to a lesser

degree since the recognition of contour and alignment may involve percipient elements outside the macular area.

4. Difference in visual acuity determined by letters in a group and single letters. The amblyopic eye was tested at frequent intervals with both Snellen chart and with single E's and C's on separate cards. In observing the visual acuity of amblyopic children who are learning to read, an interesting phenomenon was observed (Cases 89, 90 and 91). After patching of the good eye, the amblyopic eye showed improvement when tested with the letter E to an extent nearly equal to that of the uninvolved eye. However, when tested on the Snellen chart, the vision was only 20/70 to 20/100 with the amblyopic eye, while it was 20/20 with the good eye, proof that the letters could be read. At first, this seemed to suggest a form of strephosymbolia, which is commonly seen in children learning to read, but involving only the amblyopic eye. That this is not so was demonstrated by the fact that the child's visual acuity appeared similarly decreased in the amblyopic eye if the E's were placed in lines, as on the regular illiterate E chart. That is, single letters or direction of identifying points on an E could be identified by the amblyopic eye, if viewed one letter at a time, but when placed in conjunction with other letters in a line, confusion affected the interpretation. The fixated letter apparently comes and goes, is lost in the inhibited area, and then reappears to get mixed up with other letters on the line. To further elucidate this observation a number of adults with amblyopia were examined in a similar way as well as two adults with organic scotoma.

It is seen from Chart 8 that the patients with strabismus and relatively poor fixation showed improved visual acuity when looking at a single letter as compared with letters in a line. Patients with no apparent strabismus showed less change. The scanning movements of patients with strabismus and their faulty projection interferes with interpretation of letters in a line more than with interpretation of single letters. Patients without strabismus had normal projection, ambly-

opia was sometimes less severe, and fixation better, so that their vision reading single letters as compared with vision reading letters in a line showed less change than in the strabismus cases with faulty projection. There was the same lack of improvement in organic cases for the same reason. In fact, two of the organic cases stated that when looking at a pattern of letters, fixation was steadied and interpretation easier (Cases 78 and 79). Fixation movements in these two patients appeared to be nearly normal. They could readily project their scotomatous area onto a point and hold it there. When asked to move their eyes to simulate the fixation movements of amblyopic eyes, they were unable to do so.

One would expect that measurement of visual acuity by two-point discrimination would be better than by reading of letters in a line in cases with strabismus and false projection. The discrimination of two points as such would be unaffected by false projection and poor fixation. Chart 8 shows a general tendency confirming this. If, however, the patient is unaware of how many points are to be discriminated, poor fixation and faulty projection lead to confusion and the visual acuity rating is reduced because the patient is not certain whether two or more points are present as the eye moves and the points become obscured in the scotoma and are projected falsely in the inhibition area. In general, two-point discrimination was not as good as interpretation of the position of a single letter, as E or C, in cases with faulty projection where the identifying points could be lost in the scotomatous area and would then reappear as fixation changed, without the general pattern of the letter being lost to the patient. Whereas in interpretation of two points, the existence of clues of continuity or pattern is absent. Comparing the findings in the amblyopic eye with the eye with an organic lesion suggests that the amblyopic eye has not learned the fixation reflex, which the normal eye, once having attained, has difficulty in losing, even if a scotoma develops larger than that demonstrated in the cases of amblyopia reported here.

Understanding of the defect in amblyopia ex anopsia necessarily demands further investigation of the fixation reflex and the effect of eye movements on visual acuity in amblyopic patients.

SUMMARY

Verhoeff observed that nonparalytic strabismus patients failed to note apparent displacement of a fixated light by the cover test (absence of binocular phi phenomenon) because the percipient elements of the retina involved in the angle of squint have the same directional value as the macula of the fixing eye. This observation suggested to the author the following possibility: that with one eye fixing, if prisms of varying strengths were moved before the strabismic eye displacing the image varying amounts within the angle of squint, the image would not be seen, or, if seen, it would be falsely projected. Furthermore, it suggested that moving prisms before an eye might be used to study projection, inhibition and scotoma in strabismus and amblyopia. Amblyopia based on organic macular lesions might be differentiated from amblyopia ex anopsia if retinal inhibition and projection were more altered in amblyopia ex anopsia.

In general, these suppositions were verified. The prism-displacement test proved valuable in the study of retinal inhibition and projection in strabismus and amblyopia. The advantages of the prism-displacement method are:

1. Fixation is better controlled than with other binocular methods because of the rapidity with which a prism can be flashed before the eye being tested while the other eye is fixing.

2. Retinal rivalry and suppression attending binocular methods are reduced to a minimum because the stimulus is momentary.

3. Maximum and minimum degrees of inhibition can be elicited by varying the duration and intensity of the stimulus and the attention value of the contrasting surround. For

example, inhibition at the angle of squint is minimal when the fixation object is a white pin and the background a black tangent screen, or when the object is a small light in a dark room. The scotoma found is smaller than that found by any other method where fixation is controlled. Contrariwise, when the object is a light fixated at the end of a refraction lane beneath additional charts, inhibition factors may be increased and the scotoma may appear to be 10 times as great as judged by the amount of prism required for the patient to see a second image with the amblyopic or strabismic eye.

4. Field defects approaching the macula may be judged with an accuracy of 15 minutes (¼ degree).

5. No special equipment is needed for the test.

6. The eyes are examined under ordinary conditions of use.

7. The examiner can readily visualize what part of the retina is being stimulated.

The disadvantages of the test are:

1. Areas 20° in the periphery cannot be examined because of the distortion caused by strong prisms.

2. Prisms must be flashed before the eye at a rate fast enough to preclude fusion and slow enough to avoid monocular diplopia. A little experience overcomes this difficulty.

Scotoma, retinal projection and inhibition studies were made on patients who had amblyopia ex anopsia of various grades. The prism-displacement test was used as well as the usual methods for obtaining data on these conditions.

Severe amblyopes, Group 1, with gross inability to fixate, but with the eye held in the primary position, show an area of relative inhibition between the disc and the macula, with a smaller absolute scotoma, either adjoining the nasal side of the macula or on the temporal side of the disc. In the area of relative inhibition outside the absolute scotoma an image may be seen, but it is projected indefinitely. There is apparently a merging of factors of abnormal projection and inhibition in this area to account for the poor fixation and poor vision in this group of cases. The absence of fixed false pro-

jection, the variable inconstant strabismus, the strongly positive family history and the early onset in infancy, all suggest that Group 1 is an advanced grade of Group 4 (amblyopia without strabismus) and that a congenital defect as well as simple disuse of the eye may be a factor in etiology.

Group 2, amblyopes with eccentric fixation, show the same type of centrocecal or cecocentral scotoma, but the false retinal projection in the area adjoining the absolute scotoma is firmly fixed as to directional value. In some cases the fixed false projection accounts for the eccentric position of the eye more than the position and density of the scotoma, the patient preferring to fix in the eccentric position even though the vision is better in another position. These patients, like Group 1, represent a severe form of amblyopia but strabismus factors are more definite. The fixed false projection makes it difficult to obtain good results by surgery, the eye tending to go back to the original position. This can be judged by the tendency of the eye to turn under prisms so as to keep the image aligned on the same area of the retina.

Group 3 comprises cases of amblyopia with strabismus with relatively good ability to fixate. All amblyopes show increased amplitude of fixation movements as compared with the normal eye or with cases of organic macular disease. Group 3 shows degrees of inhibition at the angle of squint varying from absolute scotoma to mild facultative inhibition. Infrequently there is a small scotoma touching the side of the macula involved in the angle of squint. Between the inhibition area at the angle and the macula, degrees of faulty projection are found varying from fixed false to indefinite projection. No case with strabismus was seen in this group that showed normal projection to the prism-displacement test, a finding in keeping with Verhoeff's contention that all nonparalytic, constantly strabismic patients show faulty retinal projection as judged by absence of binocular phi phenomenon in the area of the angle of squint.

Group 4 comprises amblyopia cases without strabismus or

with inconstant strabismus showing normal retinal projection outside the central inhibited area. Anisometropia is found in most cases. A facultative type of central or paracentral scotoma is usually present and rarely a very small absolute scotoma. The scotoma is too small to account for the visual acuity. The relatively poor fixation is an important contributing factor. Retinal rivalry can be elicited and might afford a mechanism whereby the nondominant eye could be easily inhibited, preventing development of the fixation reflex. However, all patients with anisometropia do not develop amblyopia, and other factors, such as congenital defects, must be considered in the etiology.

In order to further elucidate the role of scotoma in explaining visual acuity of patients with amblyopia exanopsia 30 cases of acquired organic macular disease (Group 5) were studied. In these cases the scotoma was large as compared to that found in amblyopia. Notwithstanding the larger size of scotoma, the visual acuity was much less reduced. Fixation was steadier and projection was normal. In the cases with good fixation and normal projection, the size of the scotoma could be estimated as the size of the letter on the Snellen chart that could just be lost in the scotoma. In fact, scotomata were found by this method that had been missed with the stereocampimeter and the tangent screen. A suggestion of a scotoma in amblyopia can be indicated in the same way, particularly in the cases with relatively good fixation and normal projection. If fixation and projection are abnormal, one is not certain whether the "missed" letter is in a scotoma or is being projected falsely.

The scotoma of amblyopia ex anopsia, when near the macula, is usually eccentrically placed toward the angle of squint. An absolute central scotoma was demonstrated infrequently. The eccentric position of the scotoma suggests that the amblyopia is acquired. Absolute central scotoma in strabismic cases suggests an organic defect.

To further analyze the nature of the defect in amblyopia

ex anopsia, contrast discrimination, or determination of light difference sense, was attempted in a series of amblyopic cases. The idea for this work was suggested by Ludvigh.[19, 20] The results show a correlation of lowered light discrimination with increased fixation movements and with the size of the scotoma. The problem is to control fixation sufficiently to be certain which area of the retina is being stimulated. The light difference sense of an area of the retina peripheral to the inhibition area in the amblyopic eye appears to be the same as in the normal eye. The method herein described cannot be used to classify grades of amblyopia any more satisfactorily than studies of scotoma, fixation and projection.

Visual acuity in amblyopic cases was appraised by two-point discrimination and by Snellen letters, singly or in groups. Upon casual observation the visual acuity varied greatly in any one case, depending on the method used, but after carefully repeated examinations a final estimation could be made. The visual acuity could not be explained on the basis of the size of the scotoma, nor entirely on the basis of two-point resolving power. The area of inconstant inhibitions surrounding the scotoma, inattention, poor fixation and faulty projection, were also factors. This concept was brought out by the fact that certain amblyopes, particularly those with faulty projection and poor fixation, can identify single isolated letters much smaller than those they can identify when seen in a group. Patients with normal projection and relatively good fixation, as those with moderate amblyopia without strabismus, or with organic scotoma, showed less discrepancy between identification of single letters and letters in a group. The importance of the contributory factors emphasizes the need for special measures to train fixation and correct faulty projection in the treatment of amblyopia ex anopsia. One might devise a method whereby a series of brief successive visual stimuli might be associated with auditory or kinesthetic stimuli to improve the fixation reflex. The test could be done under conditions of dark adaptation,

using red stimuli, based on the finding of Burian and Wald[18] that fixation is relatively good even in severe amblyopes in the dark adapted eye. There is a possibility that fixation might be slightly eccentric under these conditions as the scotoma is usually nasocentral, touching the macula, a fact to be considered in the training.

This study was undertaken in an attempt to ascertain criteria helpful in prognosticating return of vision in the adult with amblyopia upon loss of the nonamblyopic eye. Two cases, representative of Groups 1 and 2, have been observed for 3 years following loss of the nonamblyopic eye. In one case, representing Group 1, vision was light projection only before loss of the good eye. Two years later a centrocecal scotoma was found. The patient now has a residual 2 to 3° nasocentral scotoma with retinal projection faulty in the centrocecal area. This precludes reading vision, but the patient can identify single leters of a size corresponding to 20/40 vision. Vision is reduced to less than 20/200 when letters of the same size are seen in a group. As yet the patient has had no formal training that might improve the fixation reflex. In the second case, representative of Group 2, also showing a nasocentral scotoma and fixed false projection, the patient continues to fix eccentrically 3 years after loss of the good eye. Consideration of these 2 cases, illustrative of the most severe forms of amblyopia, indicates that an absolute scotoma adjoining the macula gives a poor prognosis. Since one of these cases has shown improvement, particularly when viewing single letters, the prognosis might be better if the absolute scotoma were adjoining the disc with only relative inhibition at the macula instead of scotoma at the macula. Scotomata in both positions were encountered in different cases in these two groups.

In Group 3, with strabismus and false projection, but ability to fixate, one might expect improvement of vision to the degree found when viewing isolated letters, faulty projection and fixation movements then being minimized. Here

again absolute scotoma near the macula might offer a poorer prognosis than scotoma at the angle of squint.

In the group without strabismus (Group 4) one might expect the prognosis to be particularly favorable since false projection is not present as an obstacle to normal vision. Contrariwise, there is less room for improvement in this group and more evidence that an organic defect is present. The presence of an absolute scotoma would definitely offer a less favorable prognosis than the presence of a facultative scotoma.

Relative to prognosis, one cannot make more definite statements than the above at this time. The problem, however, is not unsolvable. Careful follow-up on amblyopic patients who have lost the good eye is one way in which the problem can be solved. The armed forces afford a unique opportunity for conducting this research provided studies similar to those suggested in this paper are carried out over a period of years.

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