

Pacific University

CommonKnowledge

College of Optometry

Theses, Dissertations and Capstone Projects

5-1989

Psychological disturbances of vision

Susan L.Y. Chiu
Pacific University

Recommended Citation

Chiu, Susan L.Y., "Psychological disturbances of vision" (1989). *College of Optometry*. 153.
<https://commons.pacificu.edu/opt/153>

This Thesis is brought to you for free and open access by the Theses, Dissertations and Capstone Projects at CommonKnowledge. It has been accepted for inclusion in College of Optometry by an authorized administrator of CommonKnowledge. For more information, please contact CommonKnowledge@pacificu.edu.

Psychological disturbances of vision

Abstract

Patients can present themselves in an optometrist's office and report diplopia, tunnel vision, blindness, decreased acuity and other visual and ocular anomalies. It is the optometrist's job to separate the true visual conditions with an underlying organic or visual cause from a psychological cause. Ocular malingering, hysterical amblyopia, Streff syndrome, psychological blindness, and psychosomatic ophthalmology will be presented. This literature review will serve as a guide to rule out visual anomalies of an organic cause or psychological cause and offer management and treatment options.

Degree Type

Thesis

Degree Name

Master of Science in Vision Science

Committee Chair

Nira R. Levine

Subject Categories

Optometry

Copyright and terms of use

If you have downloaded this document directly from the web or from CommonKnowledge, see the "Rights" section on the previous page for the terms of use.

If you have received this document through an interlibrary loan/document delivery service, the following terms of use apply:

Copyright in this work is held by the author(s). You may download or print any portion of this document for personal use only, or for any use that is allowed by fair use (Title 17, §107 U.S.C.). Except for personal or fair use, you or your borrowing library may not reproduce, remix, republish, post, transmit, or distribute this document, or any portion thereof, without the permission of the copyright owner. [Note: If this document is licensed under a Creative Commons license (see "Rights" on the previous page) which allows broader usage rights, your use is governed by the terms of that license.]

Inquiries regarding further use of these materials should be addressed to: CommonKnowledge Rights, Pacific University Library, 2043 College Way, Forest Grove, OR 97116, (503) 352-7209. Email inquiries may be directed to: copyright@pacificu.edu

PSYCHOLOGICAL DISTURBANCES OF VISION

By

SUSAN L.Y. CHIU

**A thesis submitted to the faculty of the
College of Optometry
Pacific University
Forest Grove, Oregon
for the degree of
Doctor of Optometry
May, 1989**

Adviser

Nira R. Levine, Ed.D.

Susan L.Y. Chiu Susan L.Y. Chiu

Nira R. Levine, Ed.D. Nira R. Levine

BIOGRAPHY

I am a 1980 honor graduate of McKinley High School. A three year scholarship from the Chinese Women's Club of Honolulu was awarded in 1980 to attend the University of Hawaii. At the University of Hawaii, I was active in Phi Eta Sigma Freshman Honor Society and the Pre-optometry society and served as secretary in 1984-1985.

After entering Pacific University in 1985, I've been active in the Student Optometric Association, Phi Theta Upsilon Service Fraternity, Amigos Optometric Service Organization and served as President in 1987-1988. With Amigos, I have served as an optometric intern in Mulege, Mexico, providing optometric care to its people. In 1988 I was nominated to Who's Who in American Colleges and Universities. Most recently I was nominated to the Outstanding Young Women of America for 1989 and a recipient of the Hawaii Optometric Association Scholarship this Spring 1989. I will be looking forward to receiving my Doctor of Optometry degree on May 21, 1989.

ACKNOWLEDGEMENTS

My sincere thanks to my adviser Dr. Nira Levine, for her concern, support and ever helpful reminders. This thesis would not have been possible if it were not for her suggestions and inspiration.

ABSTRACT

Patients can present themselves in an optometrist's office and report diplopia, tunnel vision, blindness, decreased acuity and other visual and ocular anomalies. It is the optometrist's job to separate the true visual conditions with an underlying organic or visual cause from a psychological cause. Ocular malingering, hysterical amblyopia, Streff syndrome, psychological blindness, and psychosomatic ophthalmology will be presented. This literature review will serve as a guide to rule out visual anomalies of an organic cause or psychological cause and offer management and treatment options.

PSYCHOLOGICAL DISTURBANCES OF VISION

I. INTRODUCTION

The purpose of this thesis is a synopsis of the more common psychologically based visual and ocular anomalies that can present in an optometrist's office. Little has been presented at Pacific University on specific entities such as ocular malingering, hysterical amblyopia, the Streff syndrome, psychosomatic optometry and ophthalmology, and their methods of testing, diagnosis, treatment, and management. This paper may serve as a simple guide for optometrists and interns in familiarizing them with these specific visual and ocular conditions so they can better differentially diagnose an optometric and ophthalmological condition, be it functionally, psychologically, or organically based.

II. OCULAR MALINGERING

Malingering is the "conscious pretense of false physical, physiologic, or psychologic condition."³⁵ Ocular malingering is one of the most common types. Ocular malingerers may feign decreased vision (most common), visual loss, ie., blindness in one or both eyes, and/or decreased peripheral

vision. People will malingering to obtain financial benefits, such as lawsuit awards and tax benefits, sympathy, or avoidance for military assignment. Negative malingerers, such as pilots, may report their visual conditions to be better than it actually is in order to maintain their pilot's license or to qualify for higher positions that require finer vision.²⁴

The positive malingerer feigns a visual condition to be worse than it actually is; may appear hesitant or evasive; the negative malingerer feigns a condition to be better than it actually is and may appear self-assertive or over confident.²⁴ If malingering is suspected, a careful case history must be taken and tests should be administered in the presence of witnesses. A good clue is when the objective and subjective findings consistently do not agree and there is a lack of correlation between distance and near visual acuity. ^{33, 35,39}

Here are some important clues to ocular malingering:

1. Vagueness about the time of onset and etiology of his/her claimed condition.^{26,35}
2. Wearing of tinted lenses indoors, particularly if he/she feigns blindness.^{26,39}
3. Being discomforted by questions and being unwilling to talk of his /her condition.³⁹
4. Symptoms which are not substantiated by pathologic findings.^{31,35}

5. Being wary of examination, often alternately blinking the eyes before reading the visual acuity charts and reading all letters of all lines on the charts with equal hesitancy (same with 20/200 as with 20/40); the malingerer will frequently read all the letters of one line, but claim inability to see any on the line directly below.^{26,46}
6. Partial loss of vision in one or both eyes is frequently claimed; this happens to be the most difficult to prove and is that which most frequently simulated conversion hysteria; diplopia is next in frequency.^{26,33}
7. Partial loss of vision in one or both eyes is frequently claimed; this happens to be the most difficult to prove and is that which most frequently simulates conversion hysteria; diplopia is next in frequency. ^{33,35,46}

Total blindness, where only light and form perception or a worse condition is claimed, is seldom used as a feigned illness because it requires a commitment to a prolonged period of very difficult simulation.^{25,33,46} The person with true blindness may walk upright or with a slight backward tilt, where as the malingerer will often lean slightly forward and may purposely bump into objects. The ocular hysteric, one who is affected by stress, anxiety, etc., and manifests a

visual disturbance, will carefully and purposely walk around any object in his path. If a truly blind person is asked to look at his hand or touch fingertips, he will do so with no difficulty. Whereas a malingerer may hesitate or fail to do so. Reflex tearing and flinching upon sudden movement toward the face is intact in malingerers, but a skilled one may be able to inhibit it. Strong illumination with a slit lamp will produce tearing and or blinking in the most skilled malingerer. A blind eye will not normally demonstrate a direct pupillary response to light, nor will it elicit a consensual response. A pupillary light response indicates a seeing eye or the lesion causing blindness is in the visual system above the point of exit of the pupillary fibers from the optic tract. A very determined malingerer may instill a mydriatic to suppress the pupillary light response. ^{1,26,35,46}

In a true unilateral blindness, the pupillary light reflex on the blind side will be missing, but the convergence and accommodative reflex is intact. Therefore, if a malingerer instills a mydriatic to inhibit the pupillary light reflex, he will also eliminate the consensual reflex as well as the accommodative and convergent reflex. ^{1,26,35,46}

Quam ³⁵ notes that the negative malingerer is usually talkative and overconfident. He will try to memorize the 20/20 line with the better eye before the poorer eye is tested. He may also use miotics to improve visual acuity. Therefore, always test the weaker eye if it is known. ^{33,35,46}

The differential diagnosis can include psychoneurosis, conversion hysteria, multiple sclerosis, pituitary tumor, ocular myasthenia, syphilis of the central nervous system, vascular pathology, disturbances of metabolism, bizarre drug reactions, malignancy, and some acute diseases of early onset.^{1,11,35,46}

Some simple subjective tests for malingering can be performed with the usual optometric equipment:

1. Bar-reading with a ruler used as a septum; if the patient is able to read the lines completely,, binocular vision exists (disproves diplopia, amblyopia, visual field defect).^{26,32}
2. A +12 diopter sphere can be placed before the good eye and a +6 diopter sphere before the tested eye; the patient is handed a near visual acuity chart at the focal length of the +12. While he is reading the card aloud the examiner withdraws the card past the focal range of the +12; if the patient continues to read he is doing so with the tested eye (disproves monocular amblyopia).^{26,35}
3. A variation of the above is to instill a local anesthetic in each eye and then 2% cyclogel in the good eye and distilled water in the eye to be tested. After 30 minutes record the visual acuity (the local anesthetic is needed so the patient will be unable to differentiate the difference in "stings" of the drops

in the two eyes).^{35,46}

4. With the patient at the phoropter place two cylinders of 2 diopters each and of the same sign at right angles to each other before the good eye; put the necessary corrective lens before each eye. Have the patient read rapidly and , as he is doing so, slowly rotate one of the cylinders to blur the vision. If the patient continues reading he is doing so with the tested eye (disproves monocular amblyopia).^{32,35}
5. Probably the easiest, quickest, most revealing, and least suspected test is that used for stereopsis at near; if stereoacuity is 40 seconds of arc or better the visual acuity is 20/40 or better in each eye; the vectograph slide in the AO projector can also be used to good advantage. The examiner must be wary of alternate winking and "brushing the hair out of the eyes; (momentary occlusion) by an astute malingerer.^{33,35}
6. An excellent test for a patient feigning complete blindness in one eye is to seat him before a tangent screen at a distance of one-third meter and to place a "diaphragm" between his eyes and extending to a point to that he is unable to see the center white dot with either eye but is not able to see a white spot placed in the blind spot of the opposite eye. The diaphragm

is then removed and if the patient sees three white dots he sees with both eyes.^{26,33}

7. A simpler variation of the above is to hand the patient the diploscope and ask him what letters he sees; any of the binocular responses (DOG, DOOG, DG, OGDO) all indicate vision in both eyes.³⁵
8. Another test actually relies on the malingerers need to determine the testing situation and react accordingly. A 15 prism diopter prism is held base up over the normal eye so as to bisect the pupil; the eye in which sight loss is feigned is uncovered as the patient is told to look at the test card and asked if he sees vertical diplopia. Malingerers will discover the situation by quickly closing the "blind" eye and will affirm diplopia. As he is asked to alternately read the double charts one line at a time, first upper and then lower, the prism is surreptitiously raised slightly to cover the entire pupil. If the loss of vision is real, one chart immediately disappears. If the patient continues to read both charts, he is alternately reading with each eye and one can continue to test the real visual acuity of the "blind" eye. However, this test takes considerable practice to perfect the timing.^{33,35,39}

The above subjective tests can be easily "passed" by a skilled malingerer. The following objective tests which does not give an actual acuity, can still give concrete evidence of better vision than claimed by the patient:

1. Careful and close observation of the direct and consensual pupillary reactions; complete blindness in one eye with a normal fundus and normal vision of the other eye may occur only in two conditions: retrobulbar lesion of the optic nerve and functional disturbance. Pupillary light reaction is normal in hysterical blindness as is the optokinetic nystagmus. In retrobulbar lesions illumination of the blind eye does not cause pupillary constriction in either eye whereas illumination of the sighted eye causes constriction in both.^{26,31,38}
2. An excellent test for a patient claiming complete blindness in one or both eyes is called the "swinging mirror test." A relatively large mirror, such as is used to teach contact lens insertion, is rapidly rotated and moved in a wide arc repeatedly in front of the blind eye (normal being occluded) or eyes. Any motion of the observed eye or eyes indicates vision in that eye.^{31,38}

3. As a test for malingering vs. hysteria, the patient is told to look at an extremity. A truly blind patient and the hysterically blind patient will do so with ease, but the malingerer will invariably look elsewhere. Except in the rare case of total hemianopsia, a patient who is unable to turn his eye toward his own hand is consciously malingering.^{38,39}
4. Using a 4 diopter prism base up before one eye while the patient is reading out loud will cause slowed reading or confusion, or both, in the malingerer.³²
5. A variation of the above test is to insert an 8 diopter base out in front of one eye while the patient is reading out loud. Close observations will reveal a fusion movement in the bifoveal malingerer.³²
6. If the patient complains of diplopia he can be asked to estimate the distance between the two images; this will cause an alternating movement of the eyes if real diplopia is present. If no alternating is seen, there is usually no diplopia.^{26,35}
7. By rapidly rotating the head of the patient to 20 degrees to each side one can observe fixation patterns which proves visual acuity of greater than finger counting at two

meters.^{32,35}

8. An excellent way to plot actual visual fields without the patient being aware of it is to ask the patient to look for an object in the periphery where there allegedly is no vision. If the eye turns toward the object by the shortest route, the eye must have seen it previously. The test can be continued in all 12 different meridians and at different distances between the periphery and center of the field. The patient is unaware that visual fields are actually being tested as he is permitted to move his eyes to bring the object into the center of his field. ^{31,38}
9. Lastly, the examiner can simultaneously place bifoveal targets in the major amblyoscope preset to the patient's objective angle (assuming a normal examination so far and ask him to immediately report what he sees. If he reports all check points present he must be bifoveally fusing. Bifoveal fusion is inconsistent with visual acuity much below 20/50 in one or both eyes.^{26,33,35}

How does an optometrist decide to handle the malingering patient? If the patient is a child who wants eyeglasses because his friends have

them, or wishes to gain sympathy, or avoid accepting the responsibility for his poor schoolwork, the best way to handle the child is to merely reassure him that things will return to normal shortly and that there is no need for him to be worried. It is often best not to confront the child with his malingering, although in specific instances, the optometrist may find that it may be best to confront the child.^{26,33,35}

In counseling the parents of the child, it is important to emphasize the importance of trying to understand the reasons for their child's malingering. The parents may or may not wish to confront the child. The best course may be to talk with the child and solve the underlying problem.^{26,33,35,39}

The adult malingerer who does so to gain financial or emotional benefits should be confronted and if a hysterical basis is suspected; he should seek psychological counseling.^{26,35,46}

It is important to realize that ocular disease can coexist with malingering, so therefore the presence of malingering should not cause the practitioner to overlook the presence of an organic dysfunction and lastly, diagnosis of malingering can never be by exclusion, it must always be supported by positive findings.^{26,31,35,46}

III. OCULAR HYSTERIA (HYSTERICAL AMBLYOPIA)

We discussed that ocular malingerers tend to be younger, usually in

their second or third decade, and are under some particular pressure. which may be associated with employment, frequently had some minor ocular insult or the insult may be self inflicted. Ocular hysteria usually manifests visual field defects and decreased visual acuity. Ocular hysterics are usually under some emotional stress and can reach wider age groups, which can include persons in their third and fourth decades. Typically, hysterics tend to be children, adolescents, and young adults and they tend to be female. Stress in school aged children can include parental pressure to improve school work, sibling rivalry, child abuse, death of a loved one; the list can include any number of emotional situations.^{10,37,40}

The ocular hysteric's main objective findings are constricted or tubular fields. The linear size of the field does not change with the test distance.^{13,23, 30,40,44}

In Eames'¹³ study on 193 unselected school children 9% exhibited classical hysterical visual field defects. Schaegel and Quilala's³⁷ study on 800 unselected patients, 5.25% were found to have hysterical typed field defects. Yasuna⁴⁷ feels that hysteria should be considered in all cases of amblyopia of unknown origin.

An interesting feature of a patient with this field loss is that the patient doesn't appear very concerned.^{13,40} This contrasts with a patient with an organic disease such as retinitis pigmentosa who is terribly concerned. The ocular malingerer will exaggerate his loss and bumps into

objects. ^{13,26,33}

Also associated with hysterical field loss is an associated amblyopia which is usually bilateral. The decreased acuity can often be reduced by prescribing low powered plus lenses and the use of suggestion on the part of the doctor. ^{8,10,28,40}

The hysterical tubular fields are typically bilateral.^{23,40} The fields can range from 5 to 15 degrees when this field is found, change the test distance and retest with an isopter size of the same visual angle as the original stimulus. In hysteria the usual findings will show that the field does not change, making the field appear tube-shaped instead of the expected normal conical shape. Harrington ²³ feels that this finding is almost pathognomonic of ocular hysteria and cannot be indicative of an organically caused field defect.

Another type of hysterical field is the spiral or fatigue field. The extent of the field diminishes as each new meridian is tested. This type of defect is also found to have psychogenic etiologies.²³

Central scotomas have been reported in hysteria.^{3,30,36} These are usually bilateral and show an inconsistency between the denseness of the scotoma and the measured visual acuity. This type of defect should again be checked at different testing distances to evaluate it for a functional etiology in cases where other neurological testing indicates no organic abnormality. ^{3, 23,30}

Ring scotomas have also been found in hysteria. ^{26,34,44} These scotomas

may be parital or complete rings and can be differentially diagnosed from ring fields with organic causes ie. retinitis pigmentosa, certain toxic amblyopias, etc., by again using different test distances. ^{8,30,34,44}

Hemianopsias are another field defect that have been found in hysteria. ^{19,34,35,39} It is often found in these cases that the functional nature of the hemianopsia can be shown by demonstrating it under binocular conditions, with monocular testing disclosing the defect only for the eye on the side of the binocular defect. ^{13,23,26}

Organic lesions rarely give perfect symmetric bilateral field defects. Hysterical field defects also give sharp borders even when targets of varying sizes are employed. ^{13,23,26}

It is also found that a hysteric's visual field is very open to suggestion. ⁴⁰ This factor can cause the fields to be inconsistent when testing is repeated but this can also be an important aid in the diagnosis. An example of this would be to find another blind spot opposite the physiologic one after mild suggestion by the doctor. It is also interesting to note that although a hysteric's visual fields can be found to be extremely constricted, there is almost no subjective complaint. ^{23,26,39,40}

The most common diagnostic techniques are low powered plus lenses to see if the vision and/or field loss can be modified. In more difficult diagnoses, special electrodiagnostic techniques such as visual evoked potentials can be utilized to eliminate an organic cause for the decreased vision and field loss. ^{3,8,16} Hysteria is felt to be best tested by

psychoanalysis by a professional counselor to unveil the underlying emotional stress. The prognosis in almost all cases is good if the full scope of treatment methods is utilized. Symptoms sometimes spontaneously disappear although this is not the rule. ^{10,34,40}

IV. THE STREFF SYNDROME

The Streff or non-malingering syndrome includes bilateral amblyopia and tubular fields without an organic etiology as described by Streff.⁴¹ The reduction in visual acuities is usually equal in both eyes. Distance acuities may only be slightly less than 20/20, but near acuities will often be more reduced than the far acuities, therefore it is important to take near acuities at the appropriate distance because the patient may tend to bring the near acuity task extremely close.^{4,9}

Streff syndrome patients also exhibit decreased accommodative facility, reduced stereopsis, aberrant color vision, constricted visual fields, and a subjective response to low-plus lenses is positive, usually +0.25, +0.50, or +0.75 D.^{9,22} A low plus prescription will usually provide immediate improvement in near acuities and stereopsis, but no effect on distance acuities, visual fields or color vision.^{9,18,19} These areas will return to normal at a much slower rate as the patient's accommodative system regains flexibility with the plus lens wear and vision therapy consisting of training of ocular motilities, accommodation, fusion and

binocularity.^{15,37,43}

The etiology of the Streff syndrome is not clear but may be conjectured to be an accommodative stress response to close work. This near point stress may cause overlying psychological problems like visual field constriction. Whereas, the opposite is true of hysterical amblyopia. Optometric treatment appears to be effective and predicatable for this functional-behavioral type of amblyopia.^{19,41,42}

V. CASES IN PSYCHOSOMATIC OPTOMETRY

Optometrists with expertise in low vision and psychosomatic optometry review several cases where patients have been told by ophthalmologists that they are "going blind", have been visually crippled secondary to the emotional and psychological stress. With low vision devices, and more importantly, with counseling, patients have a renewed life.

It was Feinbloom⁵ who, prior to 1932, first proposed the correct approach to low vision as being one of visual rehabilitation. He indicated that the rehabilitation must proceed on both an intellectual and motor level. To carry this work out clinically, a great deal of time must be spent with the patient through a series of visits. The work cannot be completed by only writing the prescription. The visits to the office and

the work done in learning new habits will make the patients feel that their improvement is a logical consequence of the treatment. No attempt is made to push the work. It is not too important that too much be accomplished in any one session. The patient is won over by facing facts as part of the functional treatment. Ellerbrock¹⁵ suggests more good can be done by listening sympathetically than by dispensing information. Each successive visit reveals more and more underlying disturbing elements that have accumulated through the years of so-called "blindness." The knowledge that optometrists have gained from psychosomatic optometry clearly show the pitfalls of trying to prescribe optical aids in low vision cases without proper treatment of the psychological factor. It is not surprising, therefore, that so many failures are on record of patients who were prescribed for optically, but not psychologically. ¹⁵

Psychosomatic optometry suggest that in cases of low vision, the existing visual behavior found in the examination is , in part, the manifestation of psychological disturbances in the patient. It is this approach to the psychological treatment of the cases reported below that will likely yield a higher therapeutic average.⁴⁵

Ellerbrock¹⁵ reports a case where Mrs. S., age 68, was first seen March, 1946. She had secured ophthalmological opinion that ultimate blindness would result because of her incipient senile cataract. The patient accepting the concept of ultimate blindness from high authority,

in effect gradually became blind psychologically, and manifested all the emotional and physical symptoms of such newly blind - namely, dependence upon others, disuse of even small visual cues, dependent more on other sense cues, became more introspective and introverted and even contemplated suicide. The patient's uncorrected visual acuity on the first visit was finger count at two feet and this was improved with telescopic spectacles to 20/200. One pair of telescopic spectacles and one pair of regular glasses for near work were designed for her. She came back for a progress check two months after receiving her lenses, and her findings now were: Uncorrected visual acuity 20/200, with telescope 20/90. A month later her visual acuity with her telescope was 20/70. Mathematically it is obvious that a 2X telescope can increase visual acuity only a proportionate amount. The difference between her first visit where she registered finger count and her last when she recorded 20/70 could never be brought about by any ordinary telescopic spectacles. It is significant too that she now even does without the telescopic spectacles in daily use -- using only the regular spectacles for her work at the near point, having been taught to utilize to the fullest the visual acuity she had. Certainly a one or two-visit trip to the office would never have undone the damage done her mentally. Only psychosomatic optometry could have elicited her fears first and replaced them with a program of work. In place of the original hopelessness, a program of

hopefulness and optimism was substituted, and a release obtained for her psychic trauma.^{15,45}

Vics⁴⁵ reports a case where Mrs. G illustrates most dramatically how visual acuity may really exists but low vision patients permit it (or unfortunately have it suggested to them) to drop far below its own potential. Fifteen years ago she witnessed a fire which took the lives of her closest friends. Her uncorrected visual acuity was O.D. hand movement only. O.S., 20/1500, improved to 20/150 with telescopic spectacles on her first visit to the office. She was taken under treatment which proved slow and sometimes tedious. As is usual in low vision work, she did all her seeing with one telescope and reading addition. In this instance it was before the left eye. The right eye was occluded. Vics' ⁴⁵ experience with more than 1,000 cases indicates that very few low vision patients have binocular vision and beyond examining nothing was ever done with the right eye during the entire course of treatment. The patient had always thought her right eye to be impossible of correction and aid. On the day she received her device she was told that the right eye had recorded visual acuity of 20/180 and this fact was shown to her. The amazement that her right eye now had a measurable visual acuity was beyond her control and she broke down emotionally. Since no treatment was instituted for the right eye either at near or at distance, a conclusion to be assumed is that the lowered acuity for the

many years previous had been on a psychogenic basis.⁴⁵

Ellerbrock¹⁵ reported that if J.D.'s parents had accepted the suggestion of an ophthalmologist, the boy would have been put into a Braille school. Either the physician had not heard of telescopic spectacles or refused to accept the possibility that they might help. The patient's visual acuity 20/400 was due to a primary optic atrophy that may have had its origin in an automobile accident when he was seven months of age. This acuity was increased (on the final visit) to 20/140 and he was enabled to read the finest Jaeger type. The nystagmoid movements of his eyes which were apparent at the first visit had disappeared at discharge. It would seem that the psychosomatic effect occasioned by his being told he was to be put into a Braille school was resolved when he was shown that there was no necessity for such a move. During the course of treatments it was demonstrated that he could have sufficient vision to carry on normal work with with telescopic spectacle. It was a simple matter to work with this patient, since he was in the midst of his high school career. He went on to finish high school with a great deal of ease and confidence. The work he now does is not the same were he allowed to continue as patient with low vision.¹⁵

VI. PSYCHOSOMATIC OPHTHALMOLOGY

There are many forms of simulated eye disease, among them acute or chronic ocular anxiety neurosis, traumatic neuroses, hypochondriasis, as well as delusion and obsessions related to ocular manifestations of the major psychoses.²⁰ Any ocular disease may be complicated by any psychoneuroses. Some of the more common ocular diseases or defects that are associated, either complicated or caused, by the psychoneuroses include: (a) the refractive errors and muscle anomalies (squint); and (b) primary ocular diseases, such as glaucomas, cataract, iritis, keratitis, etc.; (c) neurologic ocular diseases, such as nystagmus, field changes, etc.; (d) medical ocular diseases, such as retinal hemorrhage, etc.; and (e) surgical ocular conditions.^{7,12,36} The ocular anxiety neurosis is the most commonly seen of these conditions. It is manifested by an anxiety pattern of behavior, with ocular symptoms despite the fact that no ocular pathology or other pathology is present. The ocular symptoms consist of: (1) severe headaches that do not respond to aspirin; (2) visual loss, varying from a complete "blackout" to spots of varying sizes before the eyes; (3) ocular pains; (4) photophobia, burning, epiphora; (5) twitching of the lids; (6) dizziness, perspective distortion; and (7) easy fatigue.^{2,7,20} Byrnes⁶ reports that a 38-year old woman who complained of spots before left eye and failing vision in the past six weeks. She consulted an

eye care practitioner who said he was unable to tell her the cause of the "spots" or scotoma. He studied her for several hours, spent long periods in the dark room raising questions about the possibility of iris melanoma, and the patient became very upset. In fact she was in 'a state of acute anxiety, which was obvious. Further examination of subjective scotoma showed it to be larger at two meters than at four meters. After reassurance, all findings were normal. There was no defect of her field of vision. The fundus was normal and so was the tension. The diagnosis was ocular anxiety state, with no ocular disease, and the treatment consisted of reassurance, with follow-up when necessary.⁶

In pure neuroses there is no ocular disease, only simulated ocular disease. The differential diagnosis of a central scotoma, or any scotoma, includes ocular, as well as brain and neurologic disease, such as multiple sclerosis, and requires a thorough examination and careful consideration. Often consultations are required, and it is important to reassure the patient during these periods rather than to allow the psychoneurosis to become acute.^{2,20,36}

Dunbar¹² reports a case of a young, married woman who had dendritic keratitis illustrates a state of acute anxiety. She had been doing well, with the slow improvement noted in these cases. One day after having been seen by an associate who treated her in the absence of her usual doctor, she developed an acute anxiety state. This occurred because of

some unplanned remark that she would have a permanent scar on her cornea. In the next twelve hours she became so upset because her beauty would be smirched by the scar, she imagined a pearly white cornea, which is not a mark of beauty in any way, that she feared she would not be attractive any more. She envisioned her husband leaving her, and her world ruined. In the wee hours of the morning the doctor was awakened by her husband, also in a state of acute anxiety. His wife had taken a handful of sleeping tablets. He spent the night correcting his colleague's mistake in handling an ocular anxiety neurosis. As the case turned out, her corneal scar was nearly imperceptible and her vision some months later was 20/30 in that eye.¹²

Gordon²¹ reports how glaucoma may be related to an anxiety state: J.B. was a male, aged 68. He worked as night watchman in a bank for 27 years without missing a day of employment because of illness. One night a fire occurred in the basement. In opening the doors to let firemen in, smoke and flames struck him in the face. He became ill, vomited, and was taken home. The next day he went to work as usual, but his eyes smarted and were slightly red. He noted occasional spells of blurred vision and thought he saw smoke in the room at certain times. These symptoms persisted ten days, when he consulted his family physician. He was treated for ten more days for nervous shock, and then was sent to an eye care practitioner who diagnosed acute glaucoma in his left eye, which

was operated upon. The patient suffered agonizing pain in the left eye for the next six weeks. Operation was performed, but absolute glaucoma developed in this eye. The right eye was under a tension of 40 mm of Hg and had a complete loss of field superiorly. This patient was badly scared by the fire. He said he shook all over when he discovered it. Possibly his vomiting was the result of an acute anxiety state. From the history it is apparent that the glaucoma developed immediately after the nervous shock of the fire. This patient lost an eye entirely, and may well lose all useful vision in the second eye as a result of glaucoma.²¹

While many pages could be written about this case to substantiate claims for liability, it is Birge's ⁷ opinion that this patient lost one eye as a direct result of events arising out of the fire. His remaining sight has been seriously endangered permanently, and he is therefore entitled to compensation and necessary medical and hospital care for the rest of his life, for the disease contracted in the performance of duty.⁷

The relation between definite organic and visual eye disorders either caused or complicated by ocular psychoneuroses is a tremendous subject to which conscientious doctors can make an important contribution. It is up to them to pool their experience and knowledge for the benefit of mankind.

REFERENCES

1. Agatston, H, Ocular malingering, Arch Opth,31:223, 1944.
2. Bahn, CA, The Psychoneurotic Factor in Ophthalmic Practice, Am J Ophthalmol,26(3): 369-378, 1943.
3. Behrman J: The Visual Evoked Response in Hysterical Amblyopia, Brit J Opth 53(12): 839-845, Dec 1969.
4. Brown, J, Functionally Reduced Visual Acuity, A Case Report, Optometric Monthly, Vol 75, No. 12, p.489, 1984.
5. Feinbloom, W, Introduction to Principles and Procedure of Subnormal Vision Correction." Foundation of Visual Rehabilitation, 1935.
6. Byrnes and Shier. Amer J Opthal, 32, (268),1949.
7. Birge, HL, Psychosomatic Ophthalmology, Sight-saving Review, Vol. 18 No. 5, 1949.
- 8 Bowers BT, Vision Testing with Objective Response. Ann Opthal 4:689, 1972.
9. Catalano, RA, Functional Visual Loss in Children, Am J Opthal, Vol 93, No. 3, p.385, 1986.
10. De Schweinitz GE, Neurosis and Psychoses; Ocular Manifestations of Hysteria, 1906: 614-96.
11. Duke-Elder, S. System of Opthal, Vol. 5, 487,1970.
12. Dunbar, Emotions and Bodily Changes, NY, 1935.
13. Eames TH, A Study of Tubular and Spiral Central Fields in Hysteria, Am J Opth, Vol 30, No.5, pp. 610-611, May 1947.

14. Eggers, H.: Estimation of Uncorrected Visual Acuity in Malingerers. Arch. Ophthalmol, 33: 23-27, 1945.
15. Ellerbrock VJ, "Report on Survey of Optical Aids for Subnormal Vision.", Committee on Sensory Aids, National Research Council, 1946.
16. Feinsod M, Hoyt WF, Wilson Spire JP: Visually Evoked Response; Use in Neurologic Evaluation of Post-Traumatic Subjective Visual Complaints., Arch Ophthalmol, 94:237-240, 1976.
17. Friesen H, Follow-up study of Hysterical Amblyopia. Am J ophth 62(6): 1106-115, Dec 1966.
18. Gilman GD: Hysterical Amblyopia or Stress Syndrome? Can Optom 5(9): 6, 23, Sept 1979.
19. Gilman, GD, Optometric or Psychological Problem?, J of the AM Optom Assoc, Vol 52, No.7, 1981, pp.609-610.
20. Glaser JS: Neuro-Ophthal, Hagerstown, MD: Harper & Row, 1978, pp11-12.
21. Gordon, The Neurotic Personality, London, 1927.
22. Grala, PE, When the Patient Can't See 20/20, Review of Optometry V.121, No.8, p.42, 1984.
23. Harrington, DO, The Visual Fields, ed 3, St. Louis, LV Mosby, 1971 p. 368.
24. Hesterberg, RC, A Review of Ocular Malingering and Hysteria for the Flight Surgeon, V.67, No. 8 p554, 1983.
25. Kleckner, JF, Malingering in Relation to Visual Acuity, Am J Ophth, 35:47, 1952.

26. Kramer, KK, et al, Ocular Malingering and Hysteria: Diagnosis and Management , Survey of Ophthal, Vol. 24 No. 7 1979, pp. 89-90.
27. Krill AE, Hereditary Retinal and Choroidal Diseases. Vol 1: Evaluation. Hagerstown, MD: Harper & Row, 1972, pp 258-260, 280.
28. Krill AE, Newell FW: The Diagnosis of Ocular Conversion Reaction Involving Visual Function. Arch Ophth 79(3): 254-261, Mar 1968.
29. Levey NS, Stereoscopic Perception and Snellen Visual Acuity. Am J Ophthal, 78:722, 1974.
30. Lincoff HA: Bilateral Central Scotoma of Hysterical Origin. Arch Ophth 62(2): 273-279, Aug 1959.
31. Lincoff HA, Ennis J.: Differential Diagnosis of Hysteria and Malingering. Am J Ophthal, 42:415, 1956.
32. Lytton, H: Neutralizing cylinder Glasses as a Test for Malingering, Br J Ophthal, 26: 512, 1942.
33. Miller, WB, A review of Practica Tests for Ocular Malingering and Hysteria, Survey Ophth, 17: 241, 1973.
34. Parinaud M, The Ocular Manifestations of Hysteria, Philadelphia: Lippincott, 1900:727-69.
35. Quam, K, Ocular Malingering, Am Orthoptic J, Vol. 24, 1974, pp.73-76.
36. Schlaegel TF: Psychosomatic Ophthalmology, Baltimore, Williams and Wilkins, pp. 377-378.
37. Schlaegel TF, Quilala FV, Hysterical Amblyopia, Arch Ophth, 54 (6); 875-884, Dec. 1955.

38. Singhal NC: Hysterical Blindness Versus Malingering. Indian J Ophthal 20:173, 1977.
39. Spaeth, EB, The differentiation of the Ocular Manifestations of Hysteria and of Ocular Malingering, Arch. Ophthalmol, 4(6): 911-938, 1930.
40. Spaulding, DH, Visual Fields and Hysteria, J of the AM Optometric Assoc., Vol. 51, No. 11, 1980 pp. 855-858.
41. Streff JW: Preliminary Observations on a Non-Malingering Syndrome, Optom Wkly 53(12); 536-537, Mar 1962.
42. Streff JW, Regimens of Prescribing, Optometric Extension Program Seminar Series, San Diego, June 10-11, 1978.
43. Thompson, HS, Function Visual Loss, Am J Ophthal, V.100, No.1, p.209, 1985
44. Traquair HM, An Introduction to Clinical Perimetry, ed 6, St. Louis, CV Mosby, 1949, p.283.
45. Vics, II Comments on Psychosomatic Optometry with report of Subnormal Vision Cases, Am J of Optometry and Arch of Am Acad of Optometry, Vol 26, No.2, Feb 1949.
46. Wetzel, JO, Malingering Tests, Am J Ophthal, 26: 577-586, 1943.
47. Yasuna, ER, Hysterical Amblyopia in Children and Young Adults, Arch Ophth 45(1):70-76m Jan 1951.