

Loss of sight from disuse of the eye (amblyopia ex anopsia) / by D.B. St. John Roosa.

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J. Tweedy

LOSS OF SIGHT FROM DIS-
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OPIA EX ANOPSIA).

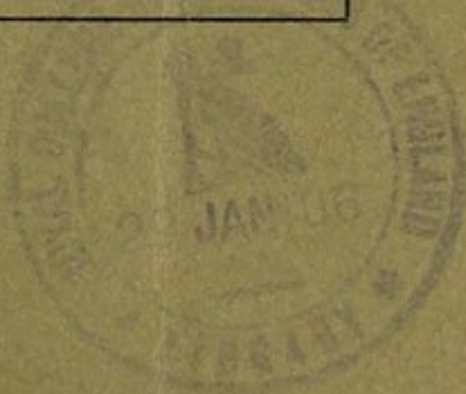
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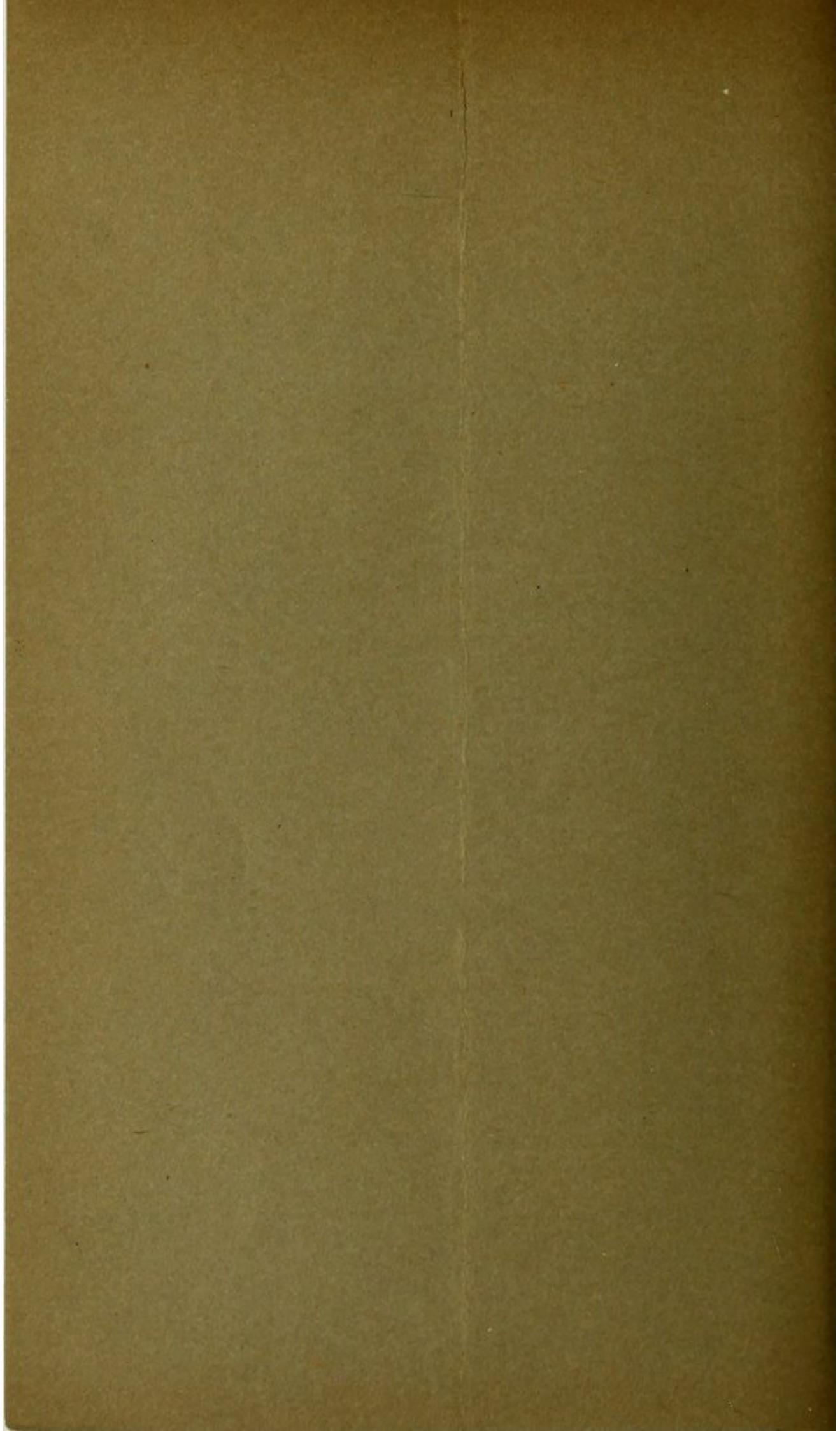
B. ST. JOHN ROOSA, M.D., LL.D.
NEW YORK

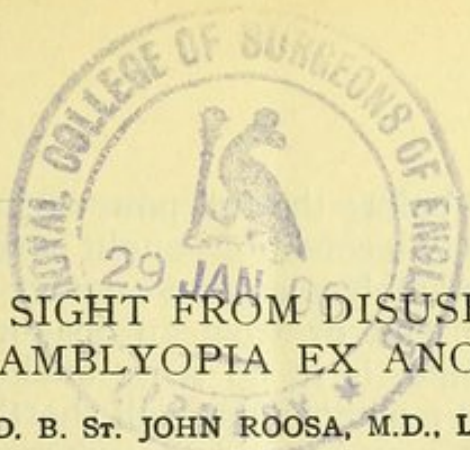
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LOSS OF SIGHT FROM DISUSE OF THE
EYE (AMBLYOPIA EX ANOPSIA).*

By D. B. St. JOHN ROOSA, M.D., LL.D.,

NEW YORK.

FAILURE of sight from disuse of the eye is a subject upon which all authorities in Ophthalmology are not agreed, either as to what class of cases come under this head, nor as to all the facts bearing upon the subject. By some writers, of whom the present is one, the term, *amblyopia ex anopsia*, is limited to those cases where use of the eye has been given up, because to use it involves double vision, the maculæ luteæ being no longer in exactly corresponding positions, as is the case in any form of strabismus. By others, cases where the retina is sound, but no image can be formed upon it, and thus it is diseased, are classified under the head of *amblyopia ex anopsia*. Where an opacity of the cornea, lens or vitreous prevents the entrance of light, there can be no image. The loss of vision here is no proper sense from disuse, but is from shutting out of the illumination. If the obscuration of the media is removed, as in the operation for cataract, the vision becomes at once, with correction of the refractive error, exceedingly good, in some instances perfect. This is amblyopia from obscuration of the ocular media.

In all cases where true *amblyopia ex anopsia* has been fully recovered from, it has been a matter of time. Months or—as in the case I am now reporting

* Read at the Annual Meeting of the Medical Society of the State of New York, January 31, 1905.

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—years elapse before the full power of the retina is restored. The two conditions ought never to be confounded. The amblyopia from cataract, either from opacity of the lens, or from an opaque membrane remaining behind, is in no sense analogous to the cases where, in a manner that will shortly be spoken of, the patient may be said to suppress the visual image by a mental action.

The subject of my paper is that form of *amblyopia ex anopsia*, which occurs very temporarily in using one eye for an ophthalmoscopic or microscopic examination, while the other eye in disuse—the eye not occupied with the instrument—sees nothing, while the fellow eye is examining the details in the retina, choroid or optic nerve, or those of a pathological or anatomical specimen with a monocular microscope. This is a condition completely analogous to even more, perhaps, exactly like the continued loss of vision in an eye turned inward or outward from the proper line of vision, where the visual power is suppressed. Another form of the same kind of suppression of the image, or rather the suppression of its perception—the image is always formed on the retina, as surely as it is on the sensitive plate of the photographic camera, if an object be placed in front of it—is that which may occur in our daily walks abroad, to any of us, when we are so abstracted by our thoughts that we may look into a friend's face without seeing him. It is still denied on some sides that this condition ever results from strabismus, but that the impairment of vision is congenital, and itself produces the squint. On other sides, it is admitted that it may occur, in part, at least, as a result of the strabismus, but, if so, that the function of the retina never again becomes perfectly normal. In spite of all that has been done in the actual exhibition of cases where the amblyopia has been removed by use and prac-

tice, and of the cases where amblyopia has actually occurred after the squint, it has been said, not so long ago, by no less an authority than Priestly Smith, "there is little evidence to show that an eye

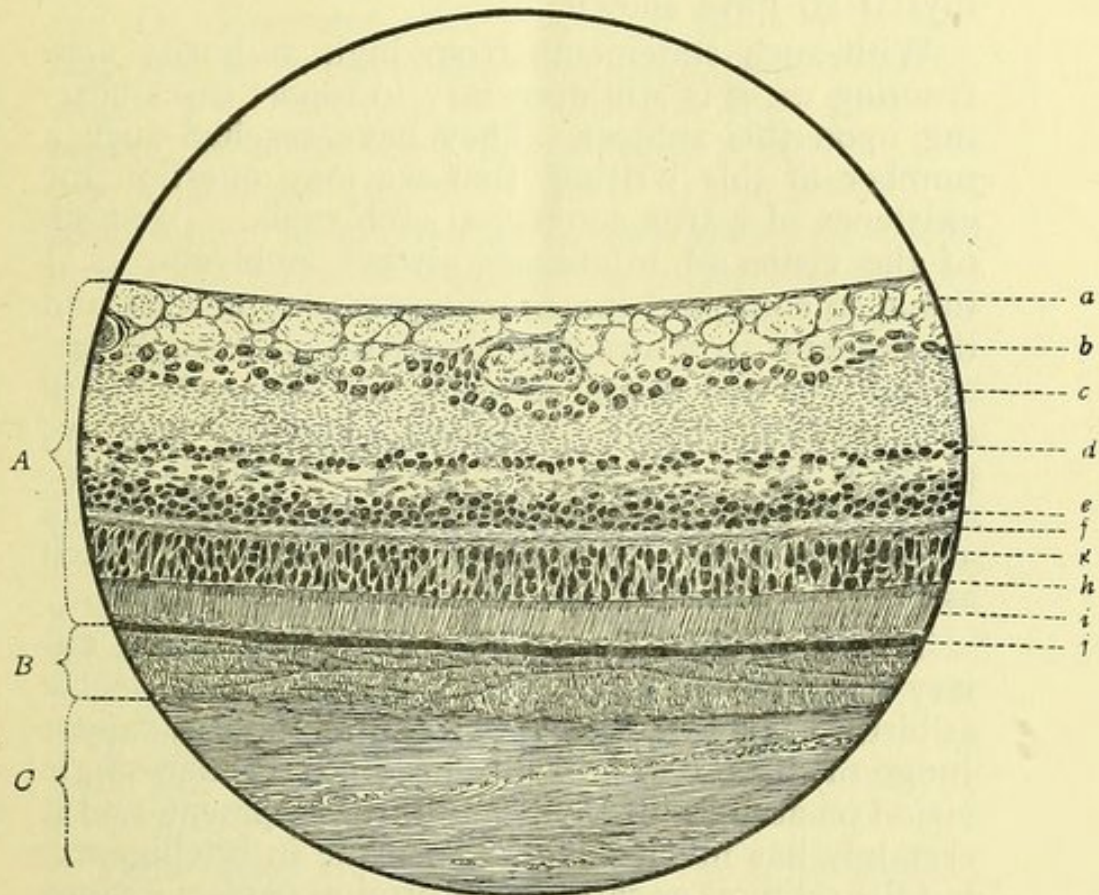


FIG. Retina of a Newborn Child. All the histological elements are fully developed. From a photomicrograph, by Dr. E. L. Oatman, Obj. $\frac{1}{2}$, Oc. 1. A, Retina; B, choroid; C, sclera. Principal layers: a, nerve fiber; b, ganglion cell; c, internal reticular; d, internal granular; e, external reticular; f, Henle's fibers; g, external granular; h, external limiting; i, rods and cones; j, pigment.

which has once acquired good vision, can lose it by squinting." ¹ It is in the very nature of things impossible to produce many such cases, but even one, and I, myself, have reported long since one, is enough to show that many more exist.

Priestly Smith then goes on to say, "when this is the case, we should expect to find the faculty recoverable by use." This is exactly just what does occur, as the cases of Agnew,² W. B. Johnson,³ and myself all have shown.

With such statements from high authority confronting us, it is still necessary to report cases bearing upon this subject. They have reached such a number at this writing, that we may question the existence of a true congenital amblyopia. To speak of the vision of infants as always amblyopic is, I think, to confound the functions of the muscles and the cerebral structure with the anatomical conditions of the rods and cones. The former are certainly in a congenitally feeble condition, but the sensitive plate on which the visual image is formed is as accurate as in childhood. We learn to accommodate and to think, but we are born with retinae on which as exact an image may be formed, as ever in life.

The accommodative power, the action of the ciliary and internal recto, with the intellect are feeble at birth. The infant has poor fixation power, cannot judge of distances, probably has no binocular single vision on account of this want of development, and it certainly has much advance to make in intelligence, but the retina is as capable of forming perfect images of objects as ever in life. My colleague, Dr. Edward L. Oatman, has many specimens of the infantile retina which prove that the layer of rods and cones is perfect at birth. I cannot for these reasonings accept Priestly Smith's conclusion, that "all eyes are amblyoptic at birth—highly amblyoptic. Those which later reach the standard of normal vision, do so by a process which occupies probably several years." This author (*loc. cit.*, p. 37) has confounded, I think, the power of using the ocular muscles and

the brain with the perceptive structure of the retina. If I am right, his deductions fall to the ground.

The case* reported by myself to this society in 1886—a case carefully and critically observed by two of my colleagues, the late Dr. Edward T. Ely and Dr. Emerson, was that of a child with normal vision, who developed convergent strabismus, which was followed by amblyopia from disuse—proves the existence of *amblyopia ex anopsia*. This case is the one quoted by Javal⁴ in his treatise on strabismus. If no other had been reported similar to it, it is of itself sufficient to demonstrate that the loss of vision in cases of strabismus is, at least, not always dependent upon a congenital condition. That it ever is, I may add in parenthesis, is, at least, a debatable question. The cases reported by Agnew and Johnson prove that the amblyopia from disuse of the eye, occurring in strabismus, may be perfectly recovered from, and I have other cases in my notebook to prove this. The cases reported by Javal, myself and others show also that even where no accident has occurred to deprive the fellow eye of sight, the amblyopic eye may regain its full power of vision by exercise. Indeed, this may be said to be now generally, but not always, conceded.

These cases are entirely against the contention of Priestly Smith, the retinae of infants are deficient in visual power. If this be assumed, the amblyopia is merely owing to the fact that the eye, out of the proper range of vision, is not exercised in sight, and does not fully develop. I am not aware of any anatomical or pathological demonstration that converts this theory into a fact. As said above, and as the photograph I exhibit shows, the retinae of infants, like the cochleae, are well developed at birth. It is the ocular muscles, not the perceptive apparatus,

and perhaps, also, the cerebral ocular centers that go on to full development after birth.

It is because the case I am about to relate supports the proposition that the amblyopia in the deviating eye is functional, and not organic, acquired and not congenital, and also that it may be recovered from perfectly, that I report it. It certainly *proves* that this is true in some cases. Incidentally, it also furnishes evidence that the retinal perceptive layer does not cease to develop, if not used, but requires only to be called into activity.

Case illustrating recovery of sight in amblyopia

*Transactions of Medical Society of the State of New York, 1886. Results of the operation for convergent squint. *ex anopsia. Convergent strabismus with amblyopia from early childhood. Operation. Over effect. Second operation, cure of strabismus. Loss of fellow eye from accident. Recovery of sight in amblyopic eye.*

John A. R., aged 46, consulted me on the 17th of May, 1900, with the following history: He stated that his right eye, which then turned inwards, was operated on convergent strabismus, when he was about eight years of age, and that divergent strabismus resulted, which was corrected by the writer of this paper, at one of his clinics, in Burlington, Vt., in 1883.

On the 30th of December, 1899, he was accidentally struck in the left eye by a piece of wood. The vision in the injured eye, previous to the accident, was $\frac{2}{30}$. This was the eye which the patient used, and, in fact, was his only dependence. The vision of the right eye has always been very defective. In the right eye the vision is $\frac{2}{160}$, with a cylindrical glass of + 4 D. It is $\frac{2}{200}$ without any glass. He has 5 diopters of astigmatism on that side. The cornea of the injured eye is opaque in the lower portion, the

pupil is irregular, and filled with broken-up lens matter. There is a small opening in the upper part of the lens. The right eye, which, in early youth, was affected with convergent strabismus, and, as has been stated, after an operation, with divergent strabismus, was now somewhat turned downward, and had eccentric fixation.

I operated upon the left eye for the purpose of removing the lens matter and breaking up the anterior synechia. An iridectomy was performed upon the temporal side, and the lens matter removed. An excellent opening resulted.

I pass from this portion of the case merely by saying that the corneal astigmatism was so much and so irregular that no better vision could be obtained than he had before the operation, $\frac{2}{8}$. I now turned my attention to the amblyopic eye, as being the hope of the patient, if the theory were correct, that it was amblyopic from disuse simply. I found no lesion of the fundus oculi, and it was pronounced to be normal by my associate, Dr. Emerson, and my colleague, Dr. A. E. Davis, who saw the case in consultation, and by myself. After thoroughly testing the patient, he was ordered the following glasses:

R. + 3.50c 130°
L. + 11

A + 4 was added to the cylindrical, on the right, for reading. He could not then read ordinary type. The patient was advised to exercise his eyes in reading, beginning with children's primers, and the hope was held out to him that his vision would ultimately be improved. One year and a half afterward his distant vision was not in the least improved, *but he read No 1 Jaeger with his glass.* He was urged to persevere, and he appeared, on October 26,

1904, with his vision in the formerly amblyopic eye, equal to $\frac{2}{30}$, with an appropriate glass, and his corneal astigmatism reduced to 4 D instead of 5 D, as in the beginning. The fixation in the right eye was now central and steady. Vision in the left eye remained as before, $\frac{2}{30}$, but he prefers and wears the glass for the correction and improvement of the sight, over the eye, although double vision can be brought out, and probably exists, under certain conditions, but evidently it does not annoy the patient, who is perfectly content with his condition.

Remarks.—The interest of this case is twofold: first, that a man more than forty years old, after having had an amblyopic eye ever since his early youth, could develop his eyesight to very nearly normal visual power. Second, that the vision for fine type was improved some months before that for infinity,—letters at twenty feet, was improved at all.

The case also shows, in a most interesting way, not only the possibility of relief in such cases, where the good eye is so much injured as to cause a great impairment of sight, but, also, it demonstrates, if any demonstration were needed, the practicability and importance of the treatment first thoroughly carried on by Javal, and widely taught by him, of exercise of an amblyopic eye, for the purpose of removing what turns out to be functional amblyopia.

There certainly are cases where the amblyopia precedes the strabismus, as in membranous cataract, but these are not, as I have already indicated, cases of amblyopia from disuse, but from obscuration, and have no place in this category, nor in this discussion. The cases of a very high degree of astigmatism in one eye, while the other has no defect, are, however, strictly analogous. In some of them it is impossible,

with the most exact correction of the error of refraction, to bring the vision up to that of the normal fellow eye. In such cases, we may conclude, I think, not that the retina is not developed, but that it is functionally amblyopic from suppression of the image. Probably all of these cases where the ophthalmoscope shows no lesion, would require normal vision, were the fellow eye lost, as in the case just detailed.

This whole subject of strabismus and its cure, with restoration of singular binocular vision, and of normal visual power, is now undergoing a thorough investigation at the hands of some of the best observers in Ophthalmology. Many points hitherto obscured are already cleared up. It is, as a contribution to this gradual elucidation of an important part of our science, that the preceding case is presented.

REFERENCES.

1. Transactions of the Ophthalmological Society of the United Kingdom, Vol. XVIII, p. 36.
2. Verbal communications.
3. Transactions American Ophthalmological Society, 1903.
4. Javal, Manuel du Strabisme, p. 239.

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