ORBITAL OPTIC NEURITIS, INCLUDING ALCOHOL AND TOBACCO AMAUROSIS

BY

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ORBITAL OPTIC NEURITIS, INCLUDING ALCOHOL AND TOBACCO AMAUROSIS.[1]

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THE singular cases in which people notice a haze, a blurr, even a perfectly dark patch, in the centre of their field of vision, known under the name of central amblyopia or central scotoma, as well as those cases in which persons lose their sight in one or a few days without exhibiting symptoms of any ocular or general disease to account for this appalling occurrence, were, more than twenty years ago, accounted for by A. v. Graefe[2] and Theo. Leber[3] by the supposition of an inflammation in the orbital part of the optic nerve, **retrobulbar neuritis.** By anatomical investigations during the last ten years, this hypothesis has been proved to be a reality, as we shall see further on.

Retrobulbar neuritis may be idiopathic or the result of different kinds of intoxication, alcoholism, nicotinism, lead-poisoning, diabetes, syphilis, etc. It has an acute and a chronic form.

A.—Acute retrobulbar neuritis.

SYMPTOMS.

1. *Headache*, in various degrees, sometimes quite severe.

2. *Orbital pain*, increased by movements of the eye and pressure upon it.

[1] Read before the New York Academy of Medicine to introduce discussion of the subject, Dec. 18, 1890.

[2] On Neuro-Retinitis and Certain Cases of Fulminant Blindness. His *Archives*, xiv., 2, pp. 114–149 (see particularly pages 146–149), 1866.

[3] Amblyopia with Central Scotoma. *v. Graefe's Arch.*, xv., 3, p. 65, etc., 1869; and "Gräfe-Sämisch's Handbuch," vol. v., p. 809, 1876.

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3. *Impairment of sight*, in all degrees. It may begin without any warning and advance rapidly, leading, in exceptional cases, to total blindness of both eyes in a day or two.

4. *Central scotoma*, for color and form. It may be of little intensity or so marked that no object is recognized in its area—partial and complete (or absolute) scotoma. The peripheric boundaries of the visual field remain normal.

5. *General diminution of color-perception.*

6. *Moderate retinal congestion and serous effusion* of the optic disc and adjacent retina. This condition may be followed by *ischæmia*, or the latter may be noticed from the beginning. The congestive swelling is to be considered as the distal boundary of the inflammatory focus, whereas the ischæmia is the result of the compression of the retinal vessels by the inflamed parts in the orbit.

7. The *termination* reveals three ophthalmoscopic pictures:

(*a*). Normal condition;

(*b*). Partial atrophy—always in the temporal half—of the optic disc;

(*c*). General atrophy of the optic nerves.

ETIOLOGY.

1. *Exposure and overwork.*

2. *Acute infectious diseases, e. g.*, measles, diphtheria, rheumatism, etc.

3. *Alcohol, nicotine, opium, lead poisoning*, etc.

4. *Suppression of menses.*

5. *No assignable cause.*

PROGNOSIS. Doubtful. We find complete and permanent recovery and partial recovery of sight, as well as permanent blindness, sometimes with preservation of one or several islets in the visual field, like oases in the desert. Their explanation later on.

TREATMENT. Rest, diaphoresis, salicylate of soda, mercury, iodide of potassium.

Let me detail one **illustrative case** from my own practice.

May 5, 1883, I was called to see Mr. Nathan Steinam, of New York City, forty years old, a successful merchant, healthy, without a constitutional taint, with no bad habits. The previous day

he had complained of nausea and severe headache. In the evening his sight was dim ; the next morning he was totally blind in both eyes. I found S = o, both pupils large, immovable, eyeballs of normal tension, their movements undisturbed. The media clear, the retinal arteries small, the veins enlarged and tortuous, the retina in the vicinity of the disc turbid, *i. e.*, a slight degree of congestive neuro-retinitis. The patient was a well man excepting his binocular blindness, and that he passed only a few ounces of urine during the day. The two next days the same condition. The urine drawn with a catheter still scant, but free from albumen, casts, and sugar. On the fourth day he passed more urine. Eyes the same, arteries more filled, but no pulsation on pressure ; veins dark, like thrombosed. Dr. Wiener, his family physician, Dr. Wm. H. Draper as consultant, and myself thought that the patient suffered from a uræmic attack. On the seventh day he had a spell of somnolence, but recovered soon and felt well. The excretions of urine became normal, the retinal arteries filled gradually to about two thirds of the natural calibre and pulsated on pressure, the veins lost their turgescence, the retina became transparent, and in about six weeks the optic discs were white, distinctly atrophic. Seven weeks after the onset of blindness he had, at various hours of the day, transient glimpses of light, during which he recognized the window and a lamp, both of which he correctly localized. When I came to see him I never could verify these observations, and was inclined to consider them as subjective sensations of light ; yet the patient, a man of well-balanced mind, positively asserted that he had seen light, though it disappeared quickly. He was treated with salicylate of soda, calomel *refracta dosi*, strychnia hypodermically, large doses of iodide of potassium, electricity. Dr. A. L. Loomis, in consultation, was of the opinion that some cerebro-spinal lesion would sooner or later manifest itself.

Thus far, almost eight years, no general disease whatsoever has appeared. The patient is physically and mentally as well as any person of his age. He goes regularly to his business, and is an intelligent and cheerful man, resigned to his loss of sight. But what has appeared is an islet of useful vision nasally and downward from the point of fixation in the visual field of the right eye, in which he counts fingers at a distance of 15′. Yet he takes in only three fingers at a glance and has slightly to move the eye to see the others. This represents an angle of a little

over 1°. His optic discs are perfectly white, the blood-vessels about two thirds the normal calibre. The arteries pulsate on pressure. The picture resembles that of atrophy after total embolism of the central retinal artery. On examination, a few days ago, the patient's general and ocular conditions were found unchanged.

B.—Chronic retrobulbar neuritis.

SYMPTOMS.

1. *Diminution of sight*, binocular, gradual, yet in some cases with acute aggravations.

2. *Day blindness*, patients see better in the dusk, the disease first described by Arlt under the name of nyctalopia. The patients complain of a fog and are dazzled; at night they feel more comfortable, but in reality see no better.

3. *Diminished color-perception*, contrast shadows not distinguished.

4. *Central scotoma*, at first for colors, then for forms (absolute). Ring scotoma, by restitution of the central part.

5. *Diminished range of accommodation.*

6. *Boundaries of visual field normal.*

7. According to the stage of the disease we find *ophthalmoscopically:*

(*a*) Fairly normal condition or slight congestion.

(*b*) Sector-like atrophy in the lower outer part of the optic disc.

(*c*) General atrophy of the od.

OCCURRENCE.

Almost exclusively in males, and in the great majority of cases from abuse of *alcohol or tobacco*, or more frequently *of both.* Opium, stramonium, lead, carbon sulphide, malaria, syphilis, diabetes, rheumatism and gout are mentioned as causes. Of late cases have been reported as sequels of epidemic influenza.[1]

PROGNOSIS good in the first stage, when no atrophy of od.

[1] Bergmeister, *Wien. klin. Wochenschr.*, 1890, No. 11. Landsberg, *Centralbl. f. Augenh.*, May, 1890, p. 141. Remak, *ibidem*, July, p. 201. C. M. Hansen, *West. Med. Reporter*, Oct., 1890, p. 231. Eperon, *Progrès Méd.*, Dec., 1890, p. 471.

is present. Rarely ending in complete blindness. The triangular atrophy of the od. may persist, but sight become normal again. Incomplete recovery is the rule.

TREATMENT.—Insist on total abstinence from alcohol and tobacco. Order strychnia, iodide of potassium, turkish baths, pure and invigorating air—sea, mountain, forest. Strychnia hypodermically, gr. $\frac{1}{30}$ or more, acts as an excitant and a tonic, with transient improvement of vision.

Diagnosis of cases of retrobulbar neuritis.

I beg to make a few remarks about the *examination of such patients.* The majority complain of impairment of sight. With test types at a distance we find both eyes affected, S from $\frac{20}{30}$ down to counting figures at several feet, excentrically.

Near by they require stronger glasses than their age and amblyopia would warrant.

Their field of vision has normal boundaries.

With the O S we notice one of the three stages: no abnormity, atrophy on temporal side, or general atrophy of the optic disc.

Examining for color-perception, we find that the patients of the last category are color-blind, those of the second recognize pigments well enough, but fail in contrast shadows; all of them show a color scotoma. The best mode to find this out is to hold a small piece of colored paper, not more than 3 *mm* long, with a pair of forceps in the visual line while you look at the patient's eye and let him look at yours. He will either be unable to see the color, especially green or red, or will confound it with another. If you want to measure the extent of the scotoma the handiest instrument is the perimeter of Schweigger. We shall, of course, not omit to examine the patient's general condition as to the well-known symptoms of alcoholism, and the other diseases mentioned before.

Pathological anatomy.

Von Graefe in 1866, and Leber in 1869, found the pallor in the temporal half of the od. and pronounced it to be the result of retrobulbar neuritis. Leber felt confident

that the anatomical proof would soon be furnished. This was done in 1880 by Samelsohn[1] of Cologne, and Nettleship and W. Edmunds[2] of London. These investigators ascertained that in a limited area of the optic nerve there were augmentation of nuclei, hypertrophy of connective tissue, and waste of nerve fibres, *i.e.*, *interstitial, sclerosing inflammation*, in the same way as alcohol produces cirrhosis of the liver.

Following up the process from the eye toward the brain they discovered that the degenerated segment near the eye had the shape of a triangle, with its base on the lower outer margin of the nerve, its apex at the central vessels. Toward the brain it gradually passed from the periphery of the nerve to the centre, which it reached in the optic canal. A case of Vossius,[3] and one of Sachs,[4] confirmed these investigations. The authors followed the atrophic segment into the chiasm and the optic tracts.

The white sector in the od. seen with the O S is, of course, the direct observation, *intra vitam*, of the atrophic segment. I have repeatedly seen it, and pass a number of sketches around. By these investigations the course of the papillo-macular fibres through the optic nerve is traced, and we know now what is the lesion in central amblyopia from alcoholism and kindred affections. Some years ago, after my attention had been directed to this triangular optic atrophy in alcoholism, etc., I was astonished to find it clear and well marked in one eye of a healthy and absolutely temperate girl, of eighteen years of age. On further examination I noticed that there was, in that eye, **a coloboma of the macula lutea.** Since that time I have found the same in other cases of macular coloboma. This affection being either a congenital defect, or the result of choroidal hemorrhage or circumscribed exudation, gives, according to Gudden's law of ascending atrophy, a neat confirmation of the position of the macular fibres in the optic-nerve entrance. In a recent paper, by G. Lindsay Johnson,[5]

[1] *Graefe's Arch.*, 1882, No. 1, pp. 1–109.
[2] Trans. Ophth. Soc. Un. Kingdom, 1881, p. 124, etc.
[3] *Graefe's Arch.*, xxviii., 3, p. 301.
[4] THESE ARCHIVES, 1889, p. 133.
[5] THESE ARCHIVES, 1890, pp. 1–29.

of London, on extra-papillary colobomata, which is illustrated by excellently executed colored drawings, there are several cases of coloboma of the macula lutea which show this triangular atrophy of the od.

An excellent paper by Dr. W. Uthoff, of Berlin, appeared, four years ago, in *Graefe's Archiv*,[1] on the Influence of Chronic Alcoholism on the Human Visual Organ. Uthoff utilizes a clinical material of 30,000 patients (Schöler's Klinik), and has examined, in different asylums of Berlin, 1,000 inebriates functionally and ophthalmoscopically. He found the triangular white atrophy of the od. in 14%. Of six fatal cases he received the eyes and examined them with the microscope most thoroughly. I pass his plates round, exhibit some with the magic-lantern, and am happy to show you under the microscope some of his specimens which he had the kindness to present me with. The one shows the central interstitial neuritis in the period of nuclear infiltration, the other in the stage of connective-tissue hypertrophy. The process is characteristic by the limitation of its area of inflammation, circumscribed and triangular near the disk, of which we gain a view in the living with the ophthalmoscope, then oblong and round as it ascends toward the optic foramen. In one of the lantern specimens you see the changes pass like a band from the temporal border of the nerve straight across to the nasal border. This shows how the atrophy of the nerve may become total.

It is remarkable that in all the specimens a certain number of healthy nerve fibres are seen preserved in the atrophic parts. This explains on the one hand why alcoholists rarely become totally blind, and on the other why in the visual field of persons blind from retrobulbar neuritis islets of useful sight may permanently be preserved, oases in the desert, as I called them in the case reported before.

Retrobulbar optic neuritis presents an example of peripheric neuritis of great scientific and practical importance, the nature of which, correctly foretold by exact clinical analysis, has of late been verified in the most gratifying manner by pathologists. The process may start at different points of

[1] Year 1886, part iv., p. 95, and year 1887, part i., p. 257.

the nerve. Samelsohn found in his case the origin in the optic canal; in two of Uthoff's cases the changes in the nerve reached the optic foramen and entered the cranial cavity; in the four others they were present only in the distal end of the orbital portion of the nerve, at its entrance into the eye, and from 6–12 *mm* back. In all of these cases the temporal pallor of the od. was seen with the ophthalmoscope. In the fatal cases in which the ophthalmoscope showed no changes, none were found *post mortem.*

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