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RETROBULBAR NEURITIS AND FACIAL PALSY OCCURRING IN THE SAME PATIENT, WITH CASES.[1]

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As is well known an interstitial optic neuritis, most severe in the optical canal and at first chiefly located in the papillo-macular tract, from which it may extend, however, through the whole diameter of the nerve, usually manifests itself by the following symptoms: Obscuration of vision, beginning in the center of the visual field and rapidly progressing to complete or nearly complete blindness; either negative ophthalmoscopic appearances, or at most blurring of the margins of the disc, hyperemia of its surface and alterations in the caliber of the retinal vessels, the arteries being diminished in size and the veins distended and pulsating; and marked pain on movement of the eyeball, or when the globe is pressed backward into the orbit. Such an affection may arise because of the presence in the blood of an infecting agent existing in association with some disease, for example, rheumatism, gout, syphilis, influenza, scarlet fever, etc., or because this infecting agent comes directly from a diseased focus in the mucous membrane of the nose, the ethmoidal cells or the sphenoidal sinus, or arises as the

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direct result of an inflammatory process in the orbit, *e.g.*, cellulitis, or in the optical canal, *e.g.*, periostitis, gummatous deposits, etc. The same disease at times appears to be the result of certain toxic agents, such as alcohol or lead, or may be provoked by menstrual disturbances, especially sudden suppression of the menses, by "catching cold," or by overwork and prolonged eye-strain. That a similar ocular disease is at times part of the symptomatology of an insular sclerosis, or of an acute or subacute myelitis, and is then of most serious prognostic import, need not be more than mentioned in this society.

Retrobulbar neuritis may be acute, subacute or chronic, and the acute cases are at times exceedingly rapid in onset and development, forming the fulminant class. With the subacute and chronic cases the present paper does not deal. The affection now under consideration, as described in the opening paragraph, may be (1) bilateral from the onset, (2) monolateral, which is perhaps the most frequent manifestation, or (3) bilateral after an interval, that is to say there may be a shorter or longer period of time between the onset of the affection in the first and in the second eye, and it is with this third class of cases that I will deal and call attention, if not to a new group, to an uncommon variety of this group, to wit: one in which the retro-ocular inflammation is preceded by an attack of peripheral facial paralysis, either upon the same or upon the opposite side.

In illustration the following cases are reported:

Case I.—Miss J. S., aged 30, a seamstress, born in Russia, presented herself for treatment on May 9, 1898, upon the kind recommendation of Dr. Barcus.

History.—The patient's early clinical history is unimportant, so far as it could be ascertained; of the health of her parents nothing is known; her sister, whom I examined, was sound. Between two and three years before her visit, after exposure to a draft, she developed facial palsy of the right side which lasted between two and three weeks, so I am informed by Dr. Barcus, her attending physician, and disappeared under the influence of blistering, alteratives and electricity. After her recovery the patient pursued her ordinary vocation as seamstress with comfort, her eyes, which were decidedly astigmatic, having been provided with proper glasses. Four days before she

consulted me, after a period of moderate right-sided headache, the sight of the right eye began to fail rapidly. She denied menstrual difficulties, either at this time or at any previous time in her history, and gave no very clear account of exposure to cold or draft, although she was accustomed to sit near an open window while sewing. Knee-jerks and station normal; no tremor; there was no anesthesia and the intellection was good.

Examination.—The girl was rather pallid, but gave no evidence of organic disease.

The vision of the right eye equaled seeing the movements of the hand in the upper field. The disc was a vertical oval, of pallid hue; both sets of vessels were slightly diminished and there was a strong venous pulse and faint retinal veiling. There was distinct pain above the eye and on movement. The center of the visual field was occupied by a large scotoma.

The vision of the left eye, after the correction of a mixed astigmatism, was 6-6, the accommodation normal, and the ophthalmoscope showed a rather pallid disc, and vessels smaller than normal. There were no traces of the former facial palsy except twitching of the right orbicularis and of the muscles at the angle of the mouth on the right side.

The usual treatment for retrobulbar neuritis, for practically all of the classical signs of this condition were present, was instituted, and after a vigorous course of iodide of potassium, and bichloride of mercury, with counter-irritation to the temple, and later full doses of strychnine, the vision gradually improved, and by the 25th of July had risen to 6-12 in the right eye, or fully one-half of normal.

At this time confusion of vision began upon the left side, preceded as before by pain in the brow and tenderness in the orbit. The disc appeared paler upon the temporal side than at previous visits, the veins being about normal, but the arteries small. The vision sank to 6-12, the accommodation failed, but a positive scotoma could not be demonstrated. Under ascending doses of iodide of potassium the vision rapidly improved, and from that date until the present time there has been no relapse, and now normal vision is restored to each eye and the patient has returned to her occupation.

CASE II.—Miss Anna K., aged 20, consulted me on September 29, 1896, for relief from keratitis.

History.—The patient's parents are healthy Germans, and the girl herself gives no history of severe illness or constitutional complaint other than much discomfort during menstruation. In 1893 she had an attack of sclero-keratitis of the right eye and soon afterwards facial palsy of the right side, which lasted for six weeks and disappeared under the influence of al-

teratives and electricity. A second attack of sclero-keratitis occurred in 1895 and was unassociated with facial palsy. The present attack did not differ from the others.

Examination.—The girl is well formed, of good coloring, and with the exception of dysmenorrhea and ocular disturbances, in fair condition. The vision of the right eye was 6-60, and the eye contained the ordinary lesions of patchy opacity of the cornea following severe scleritis. In the left eye the vision was 6-5, the accommodation normal, the media clear, the optic disc was of good color and the retinal circulation normal. The eye rapidly improved under treatment.

About six weeks after her first visit, and without any special history of exposure to cold or draft, and with no ear disease, she appeared with right facial palsy, inability to close the eye or wrinkle the brow, etc., which never, however, became complete. There was gradual restoration of function, although the right side of the face continued to be rather stiff, with a good deal of twitching at the angle of the mouth.

The patient was seen from time to time, but did not require special treatment until June 16, 1898, when she reappeared with the history that four days previous to her visit, after sleeping in a draft and when the night had become suddenly chilly, she awakened with a blur before the *left* eye, which rapidly developed until at the date of her visit she had only faint light perception in the peripheral field, the entire center of which was occupied by an absolute scotoma. The ophthalmoscope revealed moderate congestion of the disc. She was at once admitted to the Polyclinic Hospital and placed upon a treatment which consisted at various times of mercurial inunctions, pilocarpine sweats, iodide of sodium and counter-irritation. In two weeks vision was 3-60, excentric. The optic disc had lost its congestion, but there was decided pallor in the lower and outer quadrant and the ophthalmoscope revealed a large scotoma breaking through below. From that time until the present the condition has remained much the same, although the vision of the left eye has decidedly improved, and at the last examination, 10, 24, 98, was 6-50 when the head was rotated slightly to the left.

On August 9 I sent the patient to Dr. John H. W. Rhein, in order that he might examine the case electrically. He reports as follows: "I find quantitative changes only, in slight degree, in the muscles of the affected side of the face, reaction of degeneration cannot be obtained, and the nerve transmits the currents well, though not so well as on the unaffected side." A careful application of galvanism to the face improved its appearance; similar application to the eye was not followed by improvement. There were no signs of central nervous

disease; knee-jerks normal; station good; no patches of anesthesia; no tremor, nor change in voice, and no defect of intellection.

In résumé we find the first patient had, first, right facial palsy; two years later right retrobulbar neuritis, followed two and one-half months later by a mild left retrobulbar neuritis; ultimate complete recovery. The second patient had, first, right sclero-keratitis, probably depending on menstrual disturbance (whether there was retrobulbar neuritis at this time cannot be ascertained, but probably not); soon after right facial palsy; two years later again sclero-keratitis, without facial palsy; one year later sclero-keratitis, and six weeks later right facial palsy; finally, two years later, *left* retrobulbar neuritis, terminating in partial atrophy of the optic nerve. Omitting the attacks of sclero-keratitis, which probably have a different etiology, the patient had two attacks of right facial palsy, followed two years later by a severe left retrobulbar neuritis.

Naturally, there is no reason why a similar cause operating should not at one time attack the optic nerve as it passes through the optical canal and at another the facial nerve in the Fallopian canal, or, to quote the words of Mr. Marcus Gunn,[2] "Cases of retro-ocular inflammation of the optic nerve may be compared with those numerous instances of inflammation of the portio dura in its bony canal, many of which are attributed to cold or rheumatic inflammation of the sheath." It would seem, however, that not only may these affections be compared, but also that they may occur in the same subject, in a certain sense one affection replacing the other, or, perhaps, more accurately, the same cause operative in the same susceptible subject may produce either neuritis in the Fallopian canal, or neuritis in the optical canal. In other words, the subject of common facial paralysis due to neuritis again exposed to the original cause of the palsy may develop, not a recurrence of the Fallopian neuritis, but a neuritis in the optical canal.

With reference to the pathological lesions found in cases of retrobulbar neuritis, the investigations of Elschnig and others indicate that there is an interstitial neuritis with secondary degeneration of the nerve fibers. It is probable that a perineuritis, or a peri-osteitis in the optical canal would produce the

[2] Trans. Ophth. Soc. of the U. K., Vol. XVII, 1897, p. 118.

same symptoms, and as Gifford suggests,[3] it is not likely that a pachymeningitis spreading into the optical canals would be followed by the ophthalmoscopic appearances and clinical signs already described as characteristic of this disease.

That a peripheral facial paralysis may be caused by a lesion of the nerve in the Fallopian canal was demonstrated by May's case of leucocythemia with facial paralysis, caused by an infiltration of lymphoid cells and destruction of the nerve fibers in the Fallopian canal. Adolf Meyer[4] found in a case of facial palsy of ten days' duration, occurring in a paretic dement, hemorrhagic infiltration of the periosteum of the internal auditory canal and changes of reaction to a peripheral lesion in the facial nucleus of the same side. Dejerine and Theohari[5] report a case of peripheral facial paralysis, of the so-called rheumatic type, in which they found considerable lesions of parenchymatous neuritis in all of the terminal branches of the facial, but especially in the inferior facial. The degeneration was less intense in the intra-petrous portion of the nerve.

In the more chronic types of retrobulbar neuritis, producing the clinical symptoms ordinarily known as central amblyopia, we are accustomed to describe the histological changes as an interstitial sclerosing inflammation, which Samelsohn compared with the same pathological process visible in interstitial hepatitis, but it has been well stated that the descriptions of the optic nerves in the recorded cases agree quite as well with an inflammation of the connective tissues of the nerve causing secondary atrophy of the nerve fibers, as with an atrophy of the latter, associated with secondary interstitial changes following in its track.

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[3] An American Textbook of Diseases of the Eye, Ear, Nose and Throat, p. 441.

[4] Journal of Experimental Medicine, Vol. II, No. 6, 1897.

[5] Comptes rendus de la Société de Biologie, 1897, p. 1033.