when the ignorance of the pathology is so great, the description may be made too narrow.

One must always remember that a recurrent oculomotor palsy may be the first sign of a general organic disease of the nervous system, like tabes or multiple sclerosis, but in the case we have reported no sign of such disease could be found.

MYASTHENIA GRAVIS, WITH PARALYSIS CONFINED TO THE OCULAR MUSCLES.[1]

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and
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Myasthenia gravis may present symptoms of implication of the ocular muscles in association with other signs of the disease, but a limitation of the weakness to the ocular muscles is exceedingly uncommon, and cases of this character are not always easy to diagnosticate. W. Sterling[2] has recently reported a case in which the paresis was observed only in a drooping of the upper lids, the ptosis varying in intensity from time to time. The myasthenic reaction was obtained in the biceps, deltoid, and sternocleidomastoid muscles. This was the only case of this character Sterling knew of, but one of us (Spiller), in association with Dr. William Campbell Posey, has observed a similar case, in which bilateral ptosis of varying intensity was the only sign of the disease, except that one other ocular muscle was slightly affected. Dr. Posey will probably report this case. Sterling refers to the cases of Camuset, Karplus, Kunn, and Wilbrand and Sänger, in which the weakness was only in the ocular muscles, although in the first two cases other symptoms of the disease developed later. The myasthenic reaction to the electric current does not seem to have been obtained in any of these four cases.

A case reported by Oppenheim is as follows:

A man, aged forty-seven years, not syphilitic and not alcoholic, in February, 1904, noticed suddenly diplopia. He had some headache during a few days. He was strong, and only the ocular muscles were affected. Both internal recti muscles were paralyzed, and within a few days ptosis, especially on the right side, developed. The intensity of the ptosis varied greatly; at times it was only slightly indicated,

[1] Read before the Philadelphia Neurological Society, January 24, 1905.

[2] Monatsschrift f. Psychiatrie und Neurologie, vol. xvi. p. 183. (Ergänzungsheft.)

at other times the pupils were almost covered by the upper lids. The levator palpebræ superioris, therefore, appeared to be easily exhausted. If the man were made to look upward, the upper lids began to fall within a few seconds. The action of other muscles (presumably ocular) caused exhaustion of the levator. Occasionally the ptosis was more pronounced on the left side. He had neither diplopia nor ptosis in the morning during an hour after awakening. Myasthenic reaction was observed in the left deltoid. This muscle was not fully exhausted by the electric current, but the contractions became weaker with every irritation. There were no other symptoms of involvement of the nervous system at this time. The right large toe was unusually large. Later bulbar symptoms developed, and the disease terminated fatally.

Oppenheim remarks that these ocular forms of myasthenia gravis have not received sufficient attention, and that probably they are more common than is supposed. The diagnosis of cerebral syphilis or tabes is likely to be made in such cases. Tabetic ptosis may be a hypotonia of the levator palpebræ superioris, and may disappear for a time, and variation in intensity of the ocular palsy is recognized in tabes. The signs of tabes at first may be in the ocular muscles and may be confined to these for a long time. The internal muscles of the eyes are not affected in myasthenia, but these muscles may also escape in tabes. The myasthenic electric reaction is a positive means of diagnosis.

Oppenheim[1] points out that congenital defects have been observed in myasthenia, such as the enlargement of the toe he observed in his case.

The case of myasthenia gravis we report is remarkable, in that the weakness was observed only in the ocular muscles. The rapid exhaustion of the levator palpebræ superioris, first of one side and then of the other, especially when both eyes were uncovered at the same time; the recovery after rest, the variation in the paralysis of other external ocular muscles of the oculomotor distribution, the integrity of the inner muscles of the eyeballs, a response of the sternocleidomastoid muscle to the faradic current, suggesting the myasthenic reaction, and the absence of all other signs of implication of the nervous system, make the case probably one of myasthenia gravis of the ocular type.

Notes by Dr. Buckman, January 8 *and* 13, 1905. C. F. L., aged thirty-three years, a groceryman, muscular, consulted me April 26, 1904, on account of dizziness and blurring of sight, which had lasted about one week. His vision was, and now is, about normal. He complained that everything looked crooked with both eyes, but perfectly normal with either alone. The slight error of refraction

[1] Deutsch. med. Wochenschrift, July 14, 1905, p. 1053; Berliner klin. Wochenschrift, February 6, 1905, p. 164.

was corrected under atropia, and he was ordered for the right eye +.25 ⊂ + .25 c. ax. 90°; for the left eye, + .50 ⊂ + .25 c. ax. 90°, combined with prism 2° base up and ½° base out. This correction lasted but a short time, and the muscular deviation gradually increased. He could use either eye alone, but could not use both eyes together, and has not been able to do so since that time.

On November 14th there was almost complete paralysis of the inferior rectus muscle of the right eye. The condition improved under large doses of iodide and daily applications of electricity. In a short time from the last date ptosis of the left eye developed, with occasional ptosis of the right. He took mercury to ptyalism and is now taking large doses of strychnine.

Sometimes one muscle is paralyzed and sometimes another. The sixth nerve has never been affected. He has not had optic neuritis, nausea, vomiting, headache, nor vertigo during the past six months. He has not had syphilis, and is not hysterical. His mother and several of his maternal relatives are afflicted with some defect in the ocular muscles.

The headache from which he suffered was purely nasal in origin. He has some hypertrophic rhinitis, and the few headaches he has had since he has been under treatment have been almost immediately relieved by a reduction of the pressure from the enlarged and engorged middle turbinates. The pain has always been unilateral, and it has been entirely absent since his catarrh has been treated regularly.

When he lies flat on his back both lids remain wide open without effort, and all the muscles appear well balanced.

Notes by Dr. Risley, January 12, 1905. When first seen by us, October 28, 1904, the following conditions were present: The vision in the right eye, with his correcting glass, was 6/x; in the left eye, 6/viii. The ophthalmoscope showed the fundus oculi cherry-red and fluffy, with large central excavations in the disks, but no signs of atrophy or swelling. There was present diplopia caused by a partial paralysis of the internal rectus, and a complete paralysis of the inferior rectus on the right side. At his next visit, November 25, 1904, the double images were closer together, and a curious ptosis was present, usually affecting the left eye, but at times both eyes. *He stated that on arising in the morning the eyes were wide open, and the double images very close together, and at times he had no perceptible diplopia, but as he resumed the duties of the day all the symptoms recurred.* The left eye at this visit was slightly turned upward, apparently by the effort to overcome the ptosis. There were crossed diplopia and left hyperphoria.

Examination. Eyes left=exophoria disappears, hyperphoria increases. Eyes left and up=hyperphoria increases, slight exophoria. Eyes right and up=exophoria increases, hyperphoria disappears. Eyes right=exophoria increases and hyperphoria increases

Eyes right and down=hyperphoria and exophoria increase. Eyes down=exophoria and hyperphoria diminish. Eyes down and left=exophoria and hyperphoria diminish.

Four years ago a friend of the patient, an optician, told him he had muscle trouble when testing his eyes.

Notes by Dr. Spiller, January 10, 1905. The patient had severe headache during ten years until about six or seven months ago. Vision was not affected when he had the headache and has always been good.

When he takes off his glasses the upper lids begin almost immediately to droop and continue to do so until the eyeballs are covered, first one then the other. He wears a cover over one eye, and changes this from one glass to the other frequently. When he looks downward both upper lids fall. The external rectus on each side is normal. He cannot look upward or downward very well with the right eye, but can perform these movements with the left eye. He cannot keep both eyes open at the same time more than a minute, but if he covers one eye the other remains open. The iritic response to light and accommodation is prompt in each eye. Sensations for touch and pain are normal in all parts of the body. The muscles of the face, of the tongue, and of mastication are normal. The biceps reflex is not unusually prompt on either side. The grasp of each hand is powerful. Voluntary power in the lower limbs is normal. The patellar reflex is normal on each side. Ankle clonus is not obtained. Gait and station are normal with eyes open or closed. The muscles of the facial distribution respond promptly to the faradic current.

January 24, 1905. When he rises in the morning he cannot open his eyes so well as later in the day; formerly this was not the case. He can open his eyes better in the evening; during the entire day he uses his eyes in reading orders in his store (he is a groceryman) or in writing, therefore the disturbance is greater during the day, and is less in the evening, because he lies down.

When he takes off his glasses the upper lid of one eye, depending on which eye he had been using last, begins to fall, and gradually the upper lid falls until there is complete ptosis; while this is occurring, the upper lid of the other eye gradually droops, until ptosis may be complete or nearly complete on this side. The falling of the upper lid cannot be due to an attempt to overcome the diplopia, because the second lid droops after the first pupil is covered by the lid, and the falling of the lids occurs in the same manner when occasionally he has no diplopia while looking directly forward. He usually has diplopia in looking directly forward, but occasionally he has no diplopia for a few minutes, and then one eye "shoots off," as he expresses it, and diplopia develops. *When the upper lids have fallen, if he closes his eyes for two or three minutes he can open them again,* but with some ptosis of one lid; to-day it is in the left.

FIG. 1.

The ptosis of the right eye is not so great as that of the left. The photograph was taken one or two minutes after the glasses were removed. At times he is able to open both eyes fully.

FIG. 2.

The eyeballs are almost completely covered. The photograph was taken after the glasses had been removed three or four minutes.

He cannot keep both eyes open at the same time more than one or two minutes. If he covers the left eye with his hand, the right eye stays open better, but after two or three minutes the right upper lid falls. He now can keep the left eye partially open if he covers the right eye. The weakness of the other ocular muscles seems now to be in the elevators and depressors of the right eyeball. The ocular palsy, he says, has seldom been the same at any two examinations. The deltoids react well to a rapidly interrupted faradic current, but there seems to be some exhaustion by this current in the left sternocleidomastoid muscle.

MYASTHENIA GRAVIS:

WITH SPECIAL REFERENCE TO THE OCULAR SYMPTOMS, AND A REPORT OF A CASE INVOLVING THE EYES ONLY.[1]

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MYASTHENIA gravis is rare, especially in children. Of the earlier investigators, six are pre-eminent, viz., Wilks, Erb, Oppenheim, Hoppe, Goldflam, and Jolly. The disease was first described by Wilks[2] in 1877. He recorded a case of apparent bulbar paralysis, in which death resulted in a few weeks from failure of respiration. No lesion of the medulla was found at the autopsy. In 1878 Erb[3] published three cases, in two of which the principal symptoms were bilateral ptosis, while in the third, the intrinsic muscles of the eye were also involved. Weakness of the neck muscles, paresis of the muscles of mastication and weakness of the tongue and extremities were other symptoms observed. No muscular atrophy was present. Nothing more was added to the subject until nearly ten years later, when Oppenheim,[4] in 1887, published "A Case of Chronic Progressive Bulbar Paralysis without Anatomical Findings." Shortly afterward Eisenlohr[5] reported a case in an eighteen-year-old girl, in which the prominent symptoms were ptosis and paresis of other ocular muscles. In this case, as in Oppenheim's, no lesion was found. In 1891, Goldflam[6] added four new cases to those previously reported and collected by him. In 1892 Hoppe[7] contributed an exhaustive account of the subject, signalizing a new epoch in the history of the disease. Goldflam showed that the characteristic

[1] Read before the Chicago Medical Society, November 2, 1904.
[2] Guy's Hospital Reports, 1877, vol. xxii.
[3] Ueber Einen Neuen, wahrscheinlich bulbären Symptomenkomplex, Arch. f. Psych., Bd. ix.
[4] Virchow's Archiv, Bd. cviii.
[5] Neurol. Centralbl., 1887, Bd. vi.
[6] Ibid., 1891, Bd. x.
[7] Berl. klin. Woch., 1892, Bd. xxix.