A paralysis of the patheticus, superadded to a paralysis of the oculo-motorius, may be recognized by the absence of the characteristic rotation around the sagittal axis, which would otherwise occur on looking down (Wernicke).

If several muscles of one eye which are supplied by different nerves are paralyzed, or if there exist paralysis of the muscles of both eyes, we speak of an ophthalmoplegia (Hirschberg, Mauthner), and we distinguish an external ophthalmoplegia if only the extrinsic, and an internal ophthalmoplegia if only the intrinsic, muscles of the eye are paralyzed (sphincter, dilator, ciliary muscle). The so-called ophthalmoplegia progressiva (von Graefe) will be described in the eleventh chapter of this part under the name of poliencephalitis superior (Wernicke). Quite lately attention has been drawn to a so-called recurrent paralysis of the third nerve, of which Mauthner has analyzed fourteen instances. This disease is characterized by the fact that only one, and always the same, oculo-motor becomes affected, and that the paralysis is always complete—that is, takes in all the branches. Females, especially those of a nervous or hysterical temperament, seem more predisposed to the affection than males. The duration of the individual attacks varies from one, three, four, to even six months. They may recur after an interval of from four weeks to a year. Other nervous symptoms-migraine, vertigo-may or may not accompany them. The attacks may recur during the whole life of the patient, and even in the intervals traces of paralysis may remain (Möbius, Remak). Whether there are instances in which the disturbance is only functional, or whether in all cases there exists a distinct organic basis, we are with our present material unable to decide definitely, and we are equally in the dark with reference to the seat of the affection, as to whether it is of peripheral or of central origin. That there are instances where the former is true is proved by a case published by Richter (cf. lit.), where a new growth in the nerve itself was found.

In a suspected paralysis of the ocular muscles we endeavor to make out in our examination any defects in the mobility of the eyeball. For this purpose the patient is asked to follow with his eyes the finger of the examiner in different directions without moving his head. In this way every asymmetry in the movements of the two eyes can be noted. If the mobility in the direction of the action of the affected muscle is defective ("primary

deviation"), nystagmus-like twitching is sometimes observed on attempts at extreme rotation in that direction. But it may happen that the paresis of a muscle is not recognized if its innervation is particularly strong; then we have in the corresponding muscle of the other eye so abnormal an innervation that in the latter the effect is excessive, and we get a so-called "secondary deviation" of the sound eye. This can easily be demonstrated if the presumably healthy eye is first covered with the hand and the patient endeavors to fix with the paretic eve a point which it can not reach at all or only with the utmost exertion. If, then, the fixing eye is covered, we observe whether the healthy eye be in a proper position or not, and shall find that the latter has been moved too far in the desired direction. If this method does not give any satisfactory results, we have to examine into the nature of the double images. One eye of the patient having been covered with a colored glass, he is asked to follow with his eyes (of course, again without moving his head) the flame of a candle which is moved to and fro. If there exists paralysis or paresis in one eye, the patient complains of seeing, on the side toward which the affected muscle moves the eye, two flames, which become the farther apart the more the affected muscle is exerted. But if now, for instance, the patient looking toward the left complains of diplopia, this may be due to paralysis of the left external or the right internal rectus, as both of these muscles move the eyeball to the left. To determine which of these two is not performing its function properly, we must ascertain from the patient whether the double images are homonymous or crossed—that is, whether the colored picture be on the same or on the opposite side to the eye covered with the colored glass (homonymous and crossed diplopia respectively). In the former case the abducens (rect. ext.) is the nerve affected; in the latter the oculo-motorius (rect. intern.). For a minute study of the double images the reader is referred to the plates and the work of Landolt, of Paris, which has been translated into German by Magnus (Landolt-Magnus, Breslau, Kern, 1887; also Landolt, Les champs de fixation monoculaires, le champ de fixation binoculaire, etc., Arch. d'Ophthalm., 1893, No. 5).

The associated lateral movements of the eye to the right and to the left may be interfered with in the following ways:

I. There may exist a so-called conjugate deviation of the eyes—that is, a permanent fixation of both eyeballs to one side—

which can only be overcome, and then but temporarily, by the strongest effort. We shall refer to this symptom again in our account of hemiplegia.

2. Motion of both eyes toward one side may be permanently lost. In this case we have a paralysis of the abducens of the one and paralysis of the internal rectus of the other side, and the eyes are turned not toward the affected but toward the opposite side. In such cases the lesion is situated in the lateral portion of the pons, near the abductor nucleus. If the centres of both sides which lie close together are paralyzed, the eyes which are fixed in the middle can be moved neither to the right nor to the left, but only upward and downward, the upper eyelid moving normally (Wernicke).

3. The upward and downward motion of the eyes may be lost and only the lateral motion be possible. This form of the associated ocular palsy, in which also both upper lids may be paralyzed, is caused by a lesion of the centres situated in the central gray matter of the third ventricle and the aqueduct of Sylvius—that is, in the region of the oculo-motor nucleus. If this be accompanied by a hemiplegia, we are justified in diagnosticating a lesion of the pyramidal tract at the level of the upper corpus quadrigeminum, the posterior commissure, and the adjoining portion of the optic thalamus (Wernicke).

The treatment of the ocular paralyses is very problematical, and rarely produces unquestionable results. Usually a trial is made with iodide of potassium, a course which may be justified if there is a history of syphilis; but this drug is frequently of no avail whatever. Electricity is used either by applying one electrode over the closed lid of the diseased eye and the other over the base of the neck, so as to pass the current through the whole course of the eye muscles, or by allowing the current to pass transversely through the head from one mastoid process to the other. Medium-sized electrodes should be used and a weak current be applied about four times a week, each session occupying from one to two minutes. Now and again after prolonged galvanization we are really fortunate enough to perceive an improvement in the paralysis, or even to see it disappear. That much of this is to be attributed to the treatment seems doubtful, if we remember that it is utterly impossible to stimulate the ocular muscles with the current; for the same reason an electrical examination in ocular palsies is impossible (cf. Hirt, Lehrbuch der

Electrodiagnostik und Electrotherapie, Stuttgart, Enke, 1893, p. 751).

Passing over the different spasms of the eye muscles which occur in some brain diseases, we shall pay attention here only to one form with which the neurologist ought to make himself familiar, viz., nystagmus. This consists in a to-and-fro motion of the eyeballs in a certain plane, usually horizontal (nystagmus oscillatorius), which continues on voluntary movements of the eyes, but which is itself not under the control of the will. These movements are usually present in both eyes, and vary quite markedly in frequency and extent, according as the patient is made to fix a point or to change the direction in which he is looking. The condition is supposed to be due to weakness of sight of both eyes, dating from early childhood-that is, to impairment in the functions of the retina at a time when these have an important regulating influence in the establishment of the normal fixation of the eyes (von Graefe). However, there are undoubtedly cases which do not belong to this class, for it is a well-known fact that nystagmus may be an occupation disease, as it is often observed in miners who have to use their eyes in the dark (Schroeter, Mooren, Nieden, Foerster, Snell [British Med. Jour., July 11, 1891]; Priestley Smith [ibid., Oct. 15, 1891], and others); and, secondly, it appears in the course of certain nervous diseases—perhaps in connection with repeatedly occurring cerebral anæmia (Knoll, Ueber die nach Verschluss der Hirnarterien auftretenden Augenbewegungen-Sitzungsber. d. Akademie d. Wissenschaften in Wien, Abtheilung III, 1886). In both these classes of cases sight is often not diminished at all, and some other than the one given above must be the underlying cause; and, as a matter of fact, this nystagmus of the miners is simply due to overstrain of the eyes in an insufficient light, while the nystagmus occurring in the course of nervous diseases, more especially of multiple sclerosis, but also of tabes and epilepsy, is to be regarded as a symptom and attributed to the same influences as the main disease. That nystagmus, finally, may also be a symptom of hysteria, and may persist during the whole course of the disease, is shown by a case published by myself (cf. Deutsche med. Wochenschr., No. 30, 1887, lit.). C. S. Freund has observed nystagmus in a case of Basedow's disease (Deutsche med. Wochenschr., 1891, No. 3).

## LITERATURE.

## I. Isolated Oculo-motor paralysis.

Richter. Typisch-recidivirende Oculomotoriuslähmung mit Sectionsbefund. Arch. f. Psych. u. Nervenhk., 1887, xviii, 1.

Suckling. Brain, 1887, xxxviii, p. 241 (attacks of migraine followed by transitory oculo-motor palsy).

Senator. Ueber periodische Oculomotoriuslähmung. Zeitschr. f. klin. Med., 1887, xiii, No. 3 u. 4.

Joachim. Fall von periodischer Oculomotoriuslähmung. Jahrb. f. Kinderhk., 1888, xxviii, 1.

Bernhardt. Recidivirende Oculomotoriuslähmung. Berliner klin. Wochenschr., 1889, No. 47.

Manz. Die recidivirende Oculomotoriuslähmung. Berliner klin. Wochenschr., 1889, xxvi, No. 34.

Vissering. Ueber einen Fall von recidivirender Oculomotoriuslähmung. Münchener med. Wochenschr., 1889, xxxvi, No. 41.

Manz. Doppelseitige Oculomotoriuslähmung bei cerebraler Kinderlähmung. Wiener klin. Wochenschr., 1892, v, No. 42.

Goldschmid. Ein Fall von traumatischer totaler Oculomotoriuslähmung. Wiener med. Wochenschr., 1893, xliii, No. 7.

Dalichow. Aetiologie und ätiologische Diagnostik der Oculomotoriusparalyse (from Senator's clinic). Zeitschr. f. klin. Med., 1893, xxii.

## 2. Palsies of the Eye Muscles in General.

Mauthner. Die nicht nucleäre Augenmuskellähmung. Wiesbaden, Bergmann, 1886.

Mauthner. Die nucleäre Augenmuskellähmung. Wiesbaden, Bergmann, 1886. Möbius. Ueber die Localisation der Ophthalmoplegia exterior. Centralbl. f. Nervenhk., 1886, ix, No. 17.

Westphal. Arch. f. Psych. u. Nervenhk., 1887, xviii, 3, p. 846.

Landolt-Magnus. Uebersichtliche Zusammenstellung der Augenbewegungen im physiologischen und pathologischen Zustande. Breslau, Kern, 1887.

Remak, E. Doppelseitige Trochlearisparese. Neurol. Centralbl., 1888.

Mauthner Differentialdiagnostik der Lähmung der Erhebungsmuskeln.

Mauthner. Differentialdiagnostik der Lähmung der Erhebungsmuskeln des Auges. Wiener med. Wochenschr., 1888, No. 24.

Landolt. Une forme particulière de Paralyse des muscles oculaires. Clermont (Oise), 1889.

Böttiger. Arch. f. Psych., 1889, xxi, 2, p. 517.

Bernhardt. Zur Lehre von der nucleären Augenmuskellähmung und ihrer Complicationen. Berliner klin. Wochenschr., 1890, No. 43.

Thomsen (Bonn). Zur pathologischen Anatomie der progressiven Ophthalmoplegie. Festschrift. Hamburg, 1891.

Barth. Beitrag zur chronischen progressiven Ophthalmoplegie. Jahrb. d. Hamburger Staats Krankeninstitutes, 1892, ii, p. 100.

Schlesinger. Augenmuskellähmung nach Herpes zoster. Wiener med. Presse, 1892, xxxiii, No. 43.

Stower. Ein Fall von doppelseitiger Augenmuskellähmung. Münchener med. Wochenschr., 1892, xxxiv, No. 48.

Schlesinger. Zur Diagnose der chronischen nucleären Ophthalmoplegie. Inaug-Dissert., Tübingen, 1893.

Cheneys, Frederic. Boston Med. and Surg. Journ., June 24, 1893, cxxviii. Bach. Centralbl. f. Nervenhk. u. Psych., N. F., 1893, iii, p. 57.

Roth. Doppelbilder bei Augenmuskellähmung. Berlin, Hirschwald, 1893.

Barabaschew. Wiener klin, Wochenschr., 1893, vi, No. 17.

Braunstein. Petersburger med. Wochenschr., 1893.

Dalichow. Die Aetiologie und die ätiologische Diagnostik der Oculomotoriusparalyse mit Berücksichtigung der pathologischen Anatomie. Inaug.-Dissert., Tübingen, 1893.

Hotz. Arch. f. Augenhk., 1893, xxvi, 3, 4. Jackson, Hughlings. Lancet, July 3, 1893, ii.

## 3. Paralysis of the Abducens.

Purtscher. Traumatische Abducenslähmung. Arch. f. Augenhk., 1888, xviii, 4. Bennett and Savill. Brain, July, 1889, xlv u. xlvi (nuclear paralysis of the abducens).

Blocq et Guinon. Sur un cas de paralysie conjugée de la sixième paire. Arch. de méd. expérim. et d'anat. path., 1891, i.

Bloch. Statistisch-casuistischer Beitrag zur Lehre von den Abducenslähmungen. Inaug.-Dissert., Berlin, 1891 (438 cases of paralysis of the abducens).