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How to deal with diplopia

Conduite à tenir devant une diplopie

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ABSTRACT

Diplopia is a frequent neuro-ophthalmologic symptom with diverse etiologies. This article describes elementary diagnostic tests and frequent causes of diplopia. Monocular diplopia persists when the other eye is closed and usually disappears when the patient looks through a pinhole. It is usually caused by errors in the optical media of the eye and has to be differentiated from spectacle-induced side effect and non-organic disorders. A sign of non-organic etiology is absence of change in image position when the head is tilted. Binocular diplopia disappears regardless of which eye is closed. Binocular diplopia occurs when the images of both eyes cannot be fused. The most frequent direct cause of diplopia is acquired strabismus. Knowledge of several specific types of strabismus enables efficient patient management. Congenital and decompensating strabismus like accommodative esotropia, pathophoria, strabismus surso- and deorsaudentorius, retraction syndrome, Brown’s syndrome and esotropia in high myopia only need ophthalmologic treatment. Orbital injury, orbital tumor, ocular myositis, Graves orbitopathy and vascular disease usually require multidisciplinary management. Neurogenic paresis, superior oblique myokymia, ocular neuromyotonia, myasthenia, chronic progressive external ophthalmoplegia (CPEO), inter-nuclear ophthalmoplegia (INO) and skew deviation require specific neurologic examination. Treatment of diplopia includes treatment of the fundamental disorder, monocular occlusion, prisms and strabismus surgery.

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RÉSUMÉ

La diplopie est un symptôme neuro-ophthalmologique fréquent de causes variées. Cet article décrit les étapes du diagnostic devant une diplopie et les étiologies les plus fréquentes. Il existe deux types de diplopies : monoculaire et binoculaire. La diplopie monocular persiste lorsque l’œil controlatéral est fermé et disparaît généralement quand le patient regarde à travers un trou sténopéique. Elle est presque toujours la conséquence de phénomènes optiques dans les milieux transparents de l’œil et doit être différenciée de la diplopie anorganique (et aussi de la palinopsie et de la persévération visuelle). Le diagnostic spécifique et le traitement sont effectués par l’ophtalmologue. Les symptômes typiques de la diplopie anorganique, qui peut être monoculaire ou binoculaire, sont à la fois l’absence de changement de position des images doubles debout ou en position latérale à 90° et la...
Diplopia is a frequent symptom in neurology and ophthalmology. Diplopia can be caused by optical phenomena, ocular disorders, oculomotor disorders, disturbance in binocular fusion and more central perceptual disturbances. Diplopia means to see an image simultaneously in two directions. When we hold our thumbs at different distances in front of the eyes and look at one of them, the other appears twice. Normally, this physiologic diplopia is not disturbing. We can also simulate pathologic diplopia: by pressing the finger on the upper lid of one eye, the inferior rectus muscle is a little bit relaxed, the visual axis moves upward. Thus, the image appears below that of the other eye.

Some basics on the physiology of binocular vision facilitate understanding.Physiologically, the centres of the right and left retina and all other retinal points correspond with each other in the visual cortex. If identical stimuli fall on both retinas, a single image is perceived as the result of fusion that can be seen as the conjunction of the two cortical representations of the corresponding retinal points. Manifest squint goes along with global disparity of the retinal images of both eyes. The same stimulus that falls on the retinal centre of the fixating eye falls on a disparate point in the squinting eye. So it appears in two directions (diplopia). The stimulus that falls on the retinal centre of the squinting eye simultaneously appears in the same direction as the fixated stimulus (confusion). Tropia means manifest strabismus (esotropia, exotropia, etc.), while the term phoria refers to latent strabismus that means one eye deviates not before binocular vision is being artificially dissociated in the cover test. More on diplopia and strabismus can be found in textbooks (Cron, 1973; Spielmann, 1997; Rosenbaum and Santiago, 1999; von Noorden and Campos, 2002; Kaufmann and Steffen, 2012).

Subjective measurement of the squint angle is possible by asking the patient to look at a small white light, and then placing a red glass in front of one eye, and asking him to tell where the red light is located in the image of the other eye (Fig. 1). In esotropia, the image of the right eye appears to the right (uncrossed). Exotropia causes crossed diplopia. Correspondingly, the image of the hypertropic eye appears downwards. Incyclotropia causes clockwise tilt of the image of the right compared to the left eye, excyclotropia causes the opposite tilt. The horizontal/vertical squint angle can be estimated by the ratio of the horizontal/vertical distance between the double images and the test distance: 1 cm/1 m roughly corresponds to 0.5°, 1 cm/0.2 m corresponds to 2.5°, 10 cm/1 m correspond to 5°, and so on.

Objective measurement of the squint angle is performed by covering one eye of the patient who is constantly looking at the white light (cover test). When the fixating eye is covered, the squinting eye has to move from its deviated position to take up fixation (Fig. 2). A movement of 1 mm corresponds to 5°. That ratio also allows a relatively precise measurement of the motility range in each direction by using a liner and watching the movement of the corneal limbus (limbus test).

1. Diagnostic procedure

An important decision is whether the diplopic patient should primarily or exclusively be treated by the ophthalmologist, or whether neurologic and neuroradiologic diagnostics is urgent. At first, physiologic and non-organic diplopia should be sorted out.
Fig. 1 – Red glass test on the Maddox scale (patient’s view). With the red glass in front of the left eye, the red double image of the white fixation light appears uncrossed and downward, corresponding to 4.5° esodeviation and 1.0° left over right deviation.

2. Physiologic diplopia

At times, patients or parents are worried about physiologic diplopia which they or their child had not realized or told before. When the other history, ocular motility and stereopsis are normal, patients can be informed about the innocence of that phenomenon. Myopic patients occasionally report diplopia in peripheral directions. The reason may be that their spectacles refract light beams to the periphery. Therefore, objects visible outside the glasses may appear twice through the margin of the glasses.

3. Non-organic diplopia

Non-organic (psychogenic or pretended) diplopia is relatively rare. Every true double image tilts when the patient tilts his head. If the double image is horizontal in upright head position, then it will be nearly vertical in a 90° ear down position. Constant spatial orientation of the images in spite of that postural change of the head is suspect of non-organic origin. Random-dot-stereopsis is incompatible with binocular diplopia at the same time. The statement that random-dot-stereograms appear double is typical of non-organic disorder.

4. Monocular diplopia

Monocular and binocular diplopia can be differentiated by alternately covering the patient’s right and left eye. Binocular diplopia will disappear regardless of which eye is closed, monocular diplopia only when the involved eye is closed, whereas bilateral monocular diplopia will not disappear. Monocular diplopia may be caused by astigmatism, corneal scars or pathologies of the cristallin lens such as cataract that can also cause triplopia and polyopia (Table 1). The closely spaced double contour typically disappears when a pinhole of 1 to 2 mm diameter is placed in front of the affected eye(s) (what would not be expected in palinopsia and visual perseveration). These patients need only ophthalmologic treatment.

5. Binocular diplopia

Binocular diplopia occurs when fusion of the images from both eyes is disturbed in the visual cortex or when suppression of important parts of the displaced image from one eye is impossible (in infantile strabismus, suppression is the rule). Fusion may be disturbed by different image sizes (aniseikonia), shape or location or may be pure central problems. Aniseikonia may result from refractive or cataract surgery. Metamorphopsia due to retinal disease may also affect

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<tr>
<th>Etiology</th>
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<td>Regular astigmatism</td>
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<td>Corneal scar</td>
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Fig. 2 – Alternating cover test. Top: esotropia of the left eye. Middle: occlusion of the squinting eye does not cause a movement. Bottom: occlusion of the fixating eye causes a gaze saccade from the squinting to the straight position. At the same time, the right eye moves into a squinting position behind the occluder.
binocular fusion and be reported as diplopia. Lines of a graph paper appear undulated or distorted to the patient.

The by far most frequent cause of binocular diplopia is manifest strabismus. The further procedure depends on the specific type of strabismus and its causes. However, diplopia and its pattern in different directions of gaze alone do not allow for reliable diagnosis of the fundamental disorder. The patient’s history, additional symptoms and findings have to be considered.

6. Referral to ophthalmologist

Table 2 shows types of strabismus which can be completely managed by an ophthalmologist. Accommodative exotropia occurs in hyperopic patients whose binocular fusion does not resist the convergence impulse coupled with accommodation which is required to compensate for hypermetropia. The squint angle is variable, often increases at near, and the patients have difficulties to focus at near. Harmless pure esotro- or exophoria and microstrabismus (Lang and Arruga, 1968) with a phoric component can compensate. These patients often report visual problems with car driving, reading, near work and when they are tired. Normosensoric squint without any obvious neurologic or oculomotor abnormality can suddenly occur in young children. Surgical correction should be pursued within few weeks to months to restore normal stereopsis. Some colleagues recommend neurologic examination including MRI, because the disorder can be exceptionally caused by intracranial tumor. Quarterly follow-up exams should be performed for 2 years. Patients with infantile strabismus in their later life can be affected by diplopia due to spontaneous loss of suppression. Then, neither suppression nor fusion are possible even after prism correction of the squint angle (“horror fusionis”). Typically, in those types of strabismus the squint angle does not depend on gaze direction and the ocular motility is not limited in any direction.

The term congenital or idiopathic superior oblique palsy (strabismus surosaadductorius) is used for hypertropia which increases in contralateral gaze and ipsilateral head-tilt (Fig. 3).

Table 2 – Binocular diplopia to be managed by the ophthalmologist alone.

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<thead>
<tr>
<th>Etiology of squint/diplopia</th>
<th>Underlying disorder</th>
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<tr>
<td>Uncorrected hypermetropia</td>
<td>Accommodative esotropia</td>
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<td>Sensori-motor decompensation</td>
<td>Decompensated esophoria</td>
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<td>Strabismus surosaadductorius</td>
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<td>Strabismus deorsoaductorius</td>
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<td>Normosensoric esotropia</td>
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<td>Loss of suppression</td>
<td>Infantile esotropia</td>
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<td>Congenital cranial dysinnervation disorder (CCDD)</td>
<td>Retraction syndrome (Stilling, Türk, Duane)</td>
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<td>Brown’s syndrome (also acquired)</td>
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<td></td>
<td>Elevator palsy</td>
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<tr>
<td>Pure mechanical</td>
<td>Esotropia (hypotropia) in high myopia</td>
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In ipsilateral gaze as well as contralateral head-tilt, the squint angle decreases and fusion is usually possible. Earlier photographs may show the compensatory head-tilt. Superior oblique hypoplasia and possibly absence of the trochlear nerve (Yang et al., 2012) can be the underlying cause and visible on MRI but may vary largely (Uchiyama et al., 2010). MRI is not necessary for clinical purposes, when the motility pattern is typical (Figs. 3–5). The term strabismus deorsoad-ductorius describes the less frequent motility pattern of hypotropia that increases in contralateral gaze. Congenital inferior oblique palsy may be the cause. MRI is not necessary. In contrast to Brown syndrome, monocular motility is not restricted.

The retraction syndrome (Fig. 6) is a congenital dysinnervation disorder (CCDD) that, like diverse types of congenital fibrosis of extraocular muscles (CFEOM), is based on a genetic defect in the brainstem or the development of cranial nerves, respectively (Engle, 2007a,b; Lorenz, 2012). Due to hypo-/aplasia of the 6th nerve fibres of the 3rd nerve originally destined for the medial rectus muscle innervate the lateral rectus muscle. Abduction of the eye is reduced, and patients typically turn their head to the parietal side to get normal binocular vision. In contrast to 6th nerve palsy, adduction causes retraction of the eye with narrowing of the lid fissure (Fig. 6). Up- or downshoot of the eye is caused by simultaneous contraction of the co-innervated horizontal muscles. In contrast to pure 6th nerve palsy, the squint angle is rather small in spite of significantly limited abduction. If the dysinnervation predominates, adduction is markedly limited and the patient is exotropic and turns the head to the opposite side to achieve binocular vision. Aplasia of the 6th nerve can be shown by MRI (Parsa et al., 1998), which is not necessary for clinical decisions.
Trochlear nerve palsy RE

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Congenital SO palsy RE

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Fig. 4 – Acquired right 4th nerve palsy compared to congenital superior oblique palsy. Large excyclotropia and incomitant vertical deviation, increasing in downgaze in 4th nerve palsy. This is in contrast to small cyclodeviation and large vertical deviation, also in upgaze and adduction in congenital superior oblique palsy.

Fig. 5 – Congenital superior oblique palsy left eye. Spontaneously, the head is tilted to the contralateral (i.e., right) shoulder. Left: with increased right tilt, there is no manifest strabismus. Middle: with straight head, the boy can still compensate his hyperdeviation. Right: with left head-tilt, the strabismus becomes manifest. Such large hypertropia is not typical of acute acquired 4th nerve palsy.

Brown syndrome (Fig. 7) is a restrictive disorder caused by thickening of the superior oblique tendon proximal to the trochlea or scars that prevent free movement of the tendon in distal direction. The hypothesis that congenital Brown syndrome may be a kind of CCDD resulting from dysinnervation has been highlighted by Neugebauer and Fricke, 2010. The lacking trochlear nerve fibres could be substituted by 3rd nerve fibres innervating the superior oblique muscle. Corresponding MRI findings have been published recently (Kaeser et al., 2012). Brown syndrome is characterized by limited elevation of the adducted eye while in abduction elevation is significantly better or normal. In upgaze, the eye deviates in an exotropic position. MRI and specific neurologic diagnostics are not necessary. Further diagnostics (ENT, diagnostics for rheumatic disorder) for acquired Brown syndrome may be indicated if there are signs of inflammatory origin.

Congenital double elevator palsy with and without ptosis is due to an innervation defect of the superior rectus and inferior

Fig. 6 – Top: acquired left 6th nerve palsy (top) compared to left retraction syndrome (bottom). The common feature is more or less limited abduction (right). Bottom: retraction in adduction with narrowing of the palpebral fissure (here also upshoot) of the affected eye is typical of this harmless congenital cranial dysinnervation disorder.
oblique muscles and eventually the levator muscle. Elevation of the eye is equally reduced in addition and abduction.

Abnormal axial length of the eye causes high myopia. The posterior segment of the elongated eye tends to get outside the muscle cone temporal upwards, the lateral rectus muscle shifts downward and the eye becomes eso- and hypotropic (Herzau and Ionnikas, 1996). If the mechanism is symmetric in both eyes only esotropia results. The disorder may be mistaken for 6th nerve palsy because abduction is reduced. It may also be confused with Graves disease or orbital tumor because of pseudo-exophthalmus and elevation deficit. An MRI is not necessary to diagnose high myopia but can visualize the pathological etiology of the acquired strabismus (Krizizok et al., 1997).

7. Disorders requiring multidisciplinary management

The orbital and vascular disorders listed in Table 3 always require imaging. Diplopia is a late symptom when the eye is displaced by a slowly growing tumor. Carotis sinus cavernous fistula and sinus thrombosis are often accompanied by diplopia. However, their leading symptoms are vascular dilatation and exophthalmus. Reduced and painful motility combined with reduced intraocular pressure are signs of orbital ischemia. Neovascularisation of the iris and retina are signs of chronic ischemia as well as collateral circuits (duplex-scan).

7.1. Ocular myositis

Ocular myositis causes painful motility deficit and diplopia in and against the pulling direction of the muscle. Typical findings are dilatation of the conjunctival vessels over the muscle, swelling of the muscle including the tendinous insertion (MRI) and rapid improvement on systemic corticosteroids. Blood should be taken for serologic diagnostics prior to treatment.

7.2. Graves disease

Graves disease may occur together with autoimmune thyreopathy. Besides involvement of the orbital fat, lacrimal gland and connective tissues, every single extraocular muscle can be affected, often in an asymmetric pattern. The inferior and medial rectus muscles are preferentially affected. They become swollen, their relaxation is reduced. Therefore, motility in the opposite direction is reduced. Involvement of the medial rectus muscle causes an abduction deficit with horizontal diplopia that may be mistaken for 6th nerve palsy. The elevation deficit caused by inferior rectus involvement can be differentiated from superior rectus or vertical gaze palsy by tonometry (Wulle et al., 1987). An increase in intraocular pressure is typical of restrictive disorders when the patient is asked to look to the limited gaze direction, it is never present in paresis. Perimetry is mandatory to rule out optic nerve compression. Measurement of the circumpapillary retinal nerve fibre layer by spectral domain optic coherence tomography (SD-OCT), imaging of the extraocular muscles by CCT/MRI and eventually pattern VECF help make the diagnosis and document morphological and functional deficits. Further diagnostics including endocrinology and general internal examination will be asked for by the ophthalmologist when the ocular manifestation precedes other signs of thyreopathy (Bartalena et al., 2008; Eckstein and Esser, 2010).

8. Specific neurologic disorders

The disorders listed in Table 4 require specific neurologic diagnostics. Incomitant strabismus is a typical but non-specific sign of peripheral cranial nerve palsy. Taking into account an eventually accompanying cyclodeviation, the paretic muscle can be identified in two steps:

- the gaze direction in which the distance between the double images is maximal represents the pulling direction of the paretic muscle;
- the position of the double images in that gaze direction represents the functional deficit of the paretic muscle.
Table 4 – Neurologic disorders associated with binocular diplopia.

<table>
<thead>
<tr>
<th>Etiology of squint/diplopia</th>
<th>Underlying disorder</th>
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<tr>
<td>Peripheral neurogenic palsy</td>
<td>Abducens nerve palsy</td>
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<td>Troclear nerve palsy</td>
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<td>Oculomotor nerve palsy</td>
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<tr>
<td>Neurogenic hyperactivity</td>
<td>Superior oblique myokymia</td>
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<td>Ocular neuromyotonia</td>
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<td></td>
<td>Cyclic spasm</td>
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<tr>
<td>Systemic disease</td>
<td>Myasthenia, Lambert-Eaton myasthenic syndrome, chronic progressive external ophthalmoplegia</td>
</tr>
<tr>
<td>Acquired supranuclear disorder</td>
<td>Internuclear ophthalmoplegia</td>
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<td></td>
<td>Skew deviation</td>
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<tr>
<td>Neurogenic/psychogenic</td>
<td>Near spasm</td>
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Examples:

- vertical and tilted diplopia is maximal in right and down gaze. This means that a depressor is paretic, either a superior oblique or an inferior rectus muscle;
- the inferiorly located image belongs to the left eye (reduced depression of the left eye) and appears clockwise tilted compared to the image of the right eye (i.e., excyclodeviation). This means that an incylorotating depressor of the left eye is paretic. This is the superior oblique muscle (4th nerve palsy).

8.1. Abducens nerve palsy

Abducens nerve palsy (Fig. 6) causes a deficit in abduction with an increasing uncrossed diplopia in ipsilateral gaze. A small (<20°) esotropia in spite of completely lacking abduction as well as (abnormal) adduction (caused by vergence substitution) of the paretic eye in attempted ipsilateral gaze point to an additional ipsilateral gaze palsy. Mild bilateral 6th nerve palsy causes diplopia only at far not at near.

8.2. Acquired trochlear nerve

Acquired trochlear nerve palsy causes tilt and vertical diplopia increasing in contralateral and down gaze. The maximal excyclotropia is similar in extent as the maximal hypertropia which often is unapparent (use cover test!). Large hypertropia combined with small or lacking cycloptropia is typical of congenital strabismus sursoadductorius (Fig. 4). In both cases, hypertropia increases when the patient tilts the head to the affected side (Fig. 5). In acute palsy, this sign is less obvious and should be measured by cover testing or subjective diplopia localization. In the course of time, the head-tilt phenomenon increases due to gain modulation of the otoith reflex (Robinson, 1985). Vertical deviation and head-tilt phenomenon are often very small in bilateral symmetric 4th nerve palsy (Gräf et al., 2005). Excyclotropia is always present and increases in down gaze where typically esotropia (uncrossed diplopia, V-pattern) will be found due to loss of the abducing effect of the superior oblique muscles.

8.3. Oculomotor nerve paralysis

Oculomotor nerve paralysis is unequivocal because of nearly absent motility, mydriasis, accommodation palsy and ptosis. Diplopia may help identify partial 3rd nerve palsy. Diplopia may be crossed (exotropia) or change in vertical direction with vertical gaze movement, because the paretic eye is less mobile in up and down gaze. Third nerve palsy without involvement of the pupil is mostly caused by ischemia. Pupillary involvement is typical of traumatic or compressive lesion that requires emergency imaging (MRI). Tolosa Hunt syndrome (see Landau, this issue) and in elderly patients arteritis should be considered as a differential diagnosis (Rosen et al., 1998; Leigh and Zee, 1999; Thömke, 2008).

8.4. Superior oblique myokymia

Superior oblique myokymia is a rare disorder but should be kept in mind when patients report intermittent pain behind the eye together with oscillating vertical diplopia. The discreet typically unilateral cyclovertical eye movement caused by rhythmic spontaneous activity of the 4th nerve may be invisible without magnifying optics. Occasionally, neurovascular compression of the nerve can be found on MRI. The symptoms often improve with 200 to 400 mg/day oral carbamacepine (Dieterich, 2008; Strupp et al., 2011).

8.5. Ocular neuromyotonia

Ocular neuromyotonia is related to myokymia. It mostly concerns a rectus muscle. The muscle fails to relax after intended contraction because its innervation persists. The typical history is surgery and radiation for intracranial tumor months or years ago. Diplopia occurs after the patient has been looking in the pulling direction of the affected muscle. Then, eye movement to the opposite side is painful and reduced, and by contraction of both the affected muscle and its antagonist, the eyeball is retracted. After a couple of seconds the pathologic contraction resolves and motility improves. Treatment by oral carbamacepine (200–400 mg/day) is effective (Strupp et al., 2011).

8.6. Myasthenia

Myasthenia can involve first or exclusively extraocular muscles and the levator muscle. Non-striated intraocular muscles are not involved because of different receptors. Motility deficit, squint angle and diplopia increase when the patient is asked to look permanently in the concerning direction. After a short break, looking in the opposite gaze direction, squint angle, diplopia and ptosis decrease. The positive response to cholinesterase inhibiting drugs supports the assumption of myasthenia, but the drug test is not very sensitive. Oral administration of increasing doses for several days may be necessary. Additional diagnostic tests (EMG, autoimmune antibodies, thorax CT, in some cases single fibre EMG, which is the most sensitive test) are necessary, also with regard to Lambert-Eaton syndrome.
8.7. **Chronic progressive external ophthalmoplegia (CPEO)**

Chronic progressive external ophthalmoplegia progresses slowly and involves all extraocular muscles symmetrically. Diplopia occurs relatively late and starts at near. Bilateral ptosis is often the initial symptom. Retinal involvement has to be considered warranting fundus examination, OCT, fundus auto fluorescence testing, and electrophysiology (Fraser et al., 2010). A 24-hour ECG must be performed to detect arrhythmia (Kearns-Sayre syndrome). Laboratory, molecular biologic and genetic diagnostics concern the fundamental mitochondrial disorder (Deschauer and Zierz, 2008).

8.8. **Internuclear ophthalmoplegia (INO)**

Internuclear ophthalmoplegia (INO) manifests as dissociated paresis of the medial rectus muscle due to lesions of the internerve between the contralateral 6th nerve and the ipsilateral 3rd nerve nucleus. The functional interruption causes an adduction deficit with crossed diplopia that increases in contralateral gaze (where dissociated nystagmus with oscillopsia occurs), while adduction by convergence is better. INO can be bilateral.

8.9. **Skew deviation**

Skew deviation is part of the ocular tilt reaction (OTR), together with head-tilt to the side of the hypotropic eye and cycloverision. The patients report vertical diplopia and a tilt of the images of both eyes. The head-tilt, vertical deviation, cycloverision and a line that the patient perceives vertical, point to the same direction. OTR can be caused by medullar (ipsiversive OTR) or mesencephalic (contraversive OTR) lesions (Brandt and Dieterich, 1998).

8.10. **Near spasm**

The near spasm is characterized by variable esotropia, diplopia, blurred vision at distance and bilateral miosis. Refractometry by an ophthalmologist is essential. The disorder is believed to be psychogenic in most cases. Neurologic examination is necessary to exclude an organic origin.

9. **Treatment of diplopia**

Monocular diplopia caused by regular astigmatism is treated by spectacle correction or contact lens, irregular corneal astigmatism by contact lenses only. Cataract surgery will eliminate monocular diplopia due to pathologies of the crystallin lens. Binocular diplopia due to the first two groups of disorders listed in Table 2 may resolve by fully correcting the underlying refractive error. Large and inconstant strabismus as well as cyclotropia require eye muscle surgery. Small squint angles may be treated by prismatic lenses. In all other cases, treatment of the fundamental disorder alone or together with eye muscle surgery is indicated. When there is the potential of spontaneous recovery, surgery should be postponed for up to 1 year. For the interim time, press-on prisms on the glasses or occlusion foils can be worn. Usually the paretic eye will be occluded. However, in young children alternating occlusion may be indicated to avoid amblyopia. Problems can also arise when the dominant eye is paretic.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

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