Chapter 2

Diplopia Developed in Later Life, An Ophthalmologic Approach

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Additional information is available at the end of the chapter

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Abstract

Patients suffering from double vision first are commonly suspected to have acquired a neurological disorder. Over many years, we have observed numerous elderly patients complaining of double pictures for distant objects but lacking any other neurologic symptom. For this condition, no other causality was found than aging; therefore, the name "Age-related Distance Esotropia" has internationally been accepted. These days we know that the onset of comitant strabismus may occur, not only in childhood but even in the late years of life. Physicians are generally unaware of this fact; thus, these patients fail to find timely help for their double vision. Other geriatric eye disorders, such as cataract and maculopathy, often lead to a loss of binocularity without causing diplopia, the only signs being blurred vision or the habitual closing of one eye. The treatment of this so-called "masked diplopia" by prismatic correction of the squint will restore clear binocular vision and improve the reading ability. To avoid expensive neurological examinations for ocular-caused symptoms, simple diagnostic methods have been described in this article to help distinguish between ocular and neurologic disorders.

Keywords: diplopia, esotropia, prism correction, masked diplopia, geriatric eye diseases, squint

1. Introduction

For 40 years so far, the author has studied certain problems of vision, such as eye deviations with or without diplopia, not only in young but preferably in elderly patients. Although these symptoms are not infrequent, they are badly neglected. A typical case example may explain the matter concerned:

Case 1: A 70-year-old woman was suffering from double vision but could not remember the time of onset. Her family doctor sent her to a neurologist, supposing some kind of palsy, but the neurologic state was normal. After this she consulted an ophthalmologist who prescribed new glasses, which did not correct the double pictures. Several other ophthalmologists stated correspondingly, her troubles were due to her age and there was no help. Finally, she was seen at the orthoptic department of the First University Eye Clinic of Vienna; the opinion of our colleagues proved to be correct, our diagnosis was "Convergent squint of presbyopic age" [1], which was the first denomination we allocated to this condition in 1976 and, of course, treatment was possible and not difficult.

In the following years I paid more attention to other age-related diseases of the eyes, such as cataract, glaucoma and maculopathy, in order to find out their influence on binocularity and reading ability. Out of 15,000 index cards from my private office for general ophthalmology, 901 were selected. All the patients older than 40 and were suffering from severe binocular problems; 526 patients of this cohort had double pictures as a consequence of an acquired squint position but not every eye deviation was connected with the emergence of subjective diplopia; because of visual field defects, the patient is not aware of the second picture. We have called this condition "masked diplopia" [2].

In the following sections the reader mainly finds pathologic alterations of the eyes and their symptoms to be the cause of double vision. In addition to these ophthalmologic findings, we have recognized many interactions with nervous functions and even systemic diseases of the body; the eyes are not an isolated functional system. Unfortunately, many physicians treat the symptom of diplopia with mental reservation because of their lack of knowledge. Therefore, this chapter should help to survey this difficult matter and enable colleagues to discern important details to differentiate between eye-related and/or neurologic disorders. The targeted groups of readers may be: ophthalmologists without training in the subspecialty of orthoptics, neurologists, general practitioners and orthoptists.

2. Diplopia and ophthalmopathology

2.1. Double vision: monocular or binocular or both?

By covering each eye alternately, it is easy to decide which eye perhaps sees two objects in the proximity, or a shadow-like double contour or even three objects (triplopia). These symptoms may be caused by opacities in the optic system of the eyes, such as cataract, corneal scars and so on. But most commonly, we found the cause to be a high degree of astigmatism, uncorrected or with faulty correction.

2.2. The role of refraction anomalies

Even elderly people do not like to wear glasses. As we know, uncorrected ametropia may lead to a latent misalignment of the eyes, for example, myopia to exophoria and hyperopia to esophoria. Thus, the development of a convergent squint in childhood is caused by an overactive accommodation to overcome the hyperopia. Although the accommodative power

is highly diminished with age, the first step to evaluating the degree of an eye deviation is, independent of the patient's age, the exact correction of their refractive error.

2.3. Extraordinary symptoms equivalent to diplopia

2.3.1. Peripheral double pictures

Usually double vision arises from a squint deviation of the eyes and is checked with a torch and a red glass before one eye to separate the pictures. Some patients report double pictures, but when checked with the torch, only one light is seen. Yet, the patients' complaint is credible. An accurate description could typically be "When watching TV I see only one speaker, but the screen seems to be double, shifted to one side". Such statements may occur in cases of age-related maculopathy or atrophy of the optic nerve, caused by an extended central scotoma. But both peripheral retinas are still in function and indicate the malposition of the eyes, provided that the fixed object exceeds the size of the scotoma.

2.3.2. Masked diplopia

Despite an acquired squint deviation of the eyes the patient is not aware of a second picture because the visual field of one or both eyes has become defective and causes a lack of perception in the central and peripheral parts. Even while wearing corrective glasses, the subjective symptoms are described as blurred vision, seeing of "clouds" and, most noticeable, the irresistible impulse to close one eye. These complaints by a patient cause suspicion of the unknown presence of binocular difficulties. After correction of the squint angle by prisms, the binocularity will be restored again and the patient will have clear vision from one moment to the other, an impressive effect for the patient and the treating physician. The symptoms mentioned are most common in cases of age-related maculopathy.

2.3.3. Vague statements of patients with binocular defects

Without mentioning double vision or squint the patients let the observer assume some kind of anomaly of binocular vision, saying sentences like these: "One eye feels to be stuck", "I am not able to estimate distances" and "The picture seem to tremble when I look with both eyes".

2.4. Physiological aging processes and binocularity

The aging of the eye muscles, starting from the age of 30, is connected with an increase of their ring bands, which leads to a disruption of the myofibrils, and hence the weakening of muscle power (Mühlendyk) [3]. Sachsenweger [4] describes a thickening of the elastic fibers and an augmentation of the collagen tissues in the eye muscles. The result is an increased rigidity, combined with a reduction of their exact and fine functions. A step-wise decline of the ocular ductions between 20 and 80–95 years of age was measured: Supraduction was diminished by 35%, abduction by 21% and adduction and infraduction by 25% [5]. A reduction of the fusion amplitude and a weakening of the accommodative convergence were found to be signs of senile involution. Additionally, the impairment of neurological functions with age was noted:

The reduction of visual field sensitivity, slower but sometimes hyperkinetic motor reactions, changes in alertness and attention as well as early fatigue [6].

2.5. Age-related distance esotropia

This *designation*, created by Mittelman in 2006 [7], will probably be internationally accepted to define a certain disorder of binocularity, acquired in the late years of life. Previously, this condition (see page 1) carried different terms: "Strabismus convergens des Presbyopenalters" (Esotropia of presbyopic age) in Austria; "Konvergenzschielen im Senium" in Germany [8] and "Divergence insufficiency esotropia" in the USA. Although the clinical symptoms are similar to those of Bielschofskys' distance esotropia of highly myopic patients [9], there is a difference to be pointed out: High myopia causes anatomical alterations of the connective tissues in the orbit; age per se only exceptionally produces severe degenerative orbital changes like, for example, the "sagging eye syndrome" [10].

2.5.1. The patients

A total of 125 relevant patients were registered in my office, 74 female and 51 male, aged between 45 and 93 years with a maximum between 71 and 85, the peak being at 71–75. The distribution of the refraction errors of 250 eyes was emmetropia 33 (mostly pseudophakic eyes), 114 hyperopia and 103 slight-to-medium myopia. Many patients showed organic lesions corresponding to their age: Pseudophakia (26), incipient cataract (26), age-related macular degenerations (24), glaucoma (7), lesion of the optic nerve (4), synchisis scintillans (2), heavy eye syndrome (7) and enophthalmus after loss of weight (1). Of course frequent internal diseases must be considered to influence the patients' general condition as well as their eyes.

2.5.2. Anamnesis

For an example, see patient 1.

Some typical statements given by the patients may be mentioned:

a. Once two distant cars were observed coming down the street, and when it passed nearby it proved to be only one car.

b. The patient sees the actor on stage in a theatre as two actors.

c. When driving a car the median line of the street seems to divide at a certain distance.

Almost all patients have no difficulties and good reading ability in near distances. But they cannot date the onset of their symptoms, which will set in gradually and therefore cannot be realized immediately. As a consequence, an ophthalmologist is consulted with a delay of months or even years.

2.5.3. Pathophysiology

Both visual axes of a test person without a squint are in a parallel position when looking at far distances, but the visual lines intersect at a near object to match the degree of convergence

necessary for binocular fixation. In our aged patients, the visual axes are crossed at some distance nearer than about 20 m, according to the degree of convergent squint, the result being uncrossed double vision of distant objects. Fixing a near object the patient needs less convergence power to gain binocular perception; in relaxing the convergence the patient gains a normal eye position for near distances. Other patients cannot correct their eye deviation in this way; they maintain a convergent position even in near spaces; on the contrary, a convergence insufficiency may lead to a latent divergence at reading distance, that is, exophoria.

2.5.4. Clinical state

The first point is, of course, the complete inspection of the eyes and measuring the refractive error.

Visual acuity (VA): Almost all patients had 0.5 or better in both eyes. Only 11 had one eye worse between 0.4 and 0.01, and three of them did not complain about double pictures but habitually would close one eye. The corrected VA for reading was sufficient in all cases, in some only monocular.

Motility of the eyes: The ductions into all directions of gaze are free without restrictions, the near point of convergence being commonly between 15 and 25 cm. Everything seems to be in order unless you check the position of the eyes for *different* fixation *distances*. A fixation light installed *at a distance of 5–6 m* reveals diplopia, and by cover test (CT) or using a red-glass before one eye you can diagnose uncrossed double vision—that means esotropia. A Maddox-Cross at the same distance could indicate the degree of eye deviation; using the cover test you can measure the angle of squint by a prism bar, both in primary position. To differentiate, whether the deviation is due to an eye muscle palsy or not, you have to check the squint angle in different directions of gaze by turning the head of the patient while fixing the light. An increase of the eye deviation into a certain direction means incomitant squint and mostly indicates a neurogenic palsy. Our cases of age-related distance esotropia showed no indication of a palsy even if there was a very slight bilateral abduction deficit, not more than 2–3°. The range of squint angle while fixing into the distance varied between 2 and 20°, the average being 5°.

The binocular state at a *reading distance*, measured with the Maddox-Wing test, varied; many cases had an angle around zero, others had exo- or esophoria, ranging between −4 and +15°, on an average + 1°. The average difference between the angle that was far/near was 4.5° (**Table 1**).

Far distance: Uncrossed double pictures, comitant in all directions of gaze	Near distance: Eso - ortho - exophoria
Onset gradually and unnoticed	No reading problems
Peak age: 70–75 years	Independent of refraction error

Table 1. Age-related distance esotropia: main symptoms.

2.5.5. Prognosis without an operation

36 patients were observed over 2–19 (average 7.2) years. The squint angle did not change in 23 cases, increased in 9 (+4°) and decreased (−3.5°) in 4 patients.

2.5.6. Treatment

2.5.6.1. Prismatic glasses

The fitting of prismatic glasses is not quite easy and must be done by a highly qualified and experienced ophthalmologist or optician. Here only some principles are discussed. One main factor in assessing the strength of the prism is the patients' fusion power, that is, the ability to overcome the eye deviation, either totally or partly. A total interruption of the fusion ability while measuring the angle of deviation is to be avoided, as done, for example, with Maddox-Rods or by covering with an additional prism bar. The examination should take place under visual conditions as natural as possible, and only slight dissociation of fusion should be used, such as that possible with the Bagolini striated glasses or a light-coloured red glass. Thus, the habitually compensated part of squint will remain and only the manifest part, which causes diplopia, would be corrected; the patient probably will tolerate well the prismatic glasses.

Another problem is the prismatic difference far/near. The glasses for distant vision should correct the diplopia, and for this purpose ground-in prismatic glasses are recommended; press-on prisms are blurring, tend to promote suppression and will not be well tolerated. But these distant-glasses are not suitable for reading. Depending on the near position of the eyes, the patient may need no prism at all or a different prismatic strength. The possibilities are two separate spectacles or only one frame with progressive glasses and ground-in prismatic correction, suited for distance viewing. As the progressive glasses have the same prismatic power in both distances, an exophoria for near fixation will be produced, but most patients are able to correct this deviation by forced convergence. If the difference far/near does not exceed eight prism diopters, progressive glasses are well tolerated and sufficient for daily life; reading of books for a longer time will require separate reading glasses.

2.5.6.2. Motility exercises

Motility exercises at home, especially to the lateral side, in my experience, can prevent or slow down the progression of muscular degeneration.

2.5.6.3. Eye muscle operations

This topic is frequently discussed; operations are always of higher interest than conservative therapy; every operator is convinced of his method as the best. In general, aged patients will refuse operations and prefer prismatic glasses, but there is a technical limit for prismatic glasses. If the eye deviation exceeds 25 prism diopters (Δ), the only possibility is an operation. Difficulties after surgery arise from the age-related loss of elasticity in the eye muscles and the reduced fusion strength; both are needed to accomplish the fine adaptation of the eye position which takes more time with increasing age.

In my last active years I operated 14 relevant cases, on average 71 years old and squint angle on average as 25Δ. I performed recession-resection of the non-dominant eye in 10 cases and bilateral resection of the external rectus in 4 cases in order to avoid a weakening of internal rectus muscles. The results after 1–16 (average 6.7) years were the following: 11 cases had orthotropia and 3 cases showed a small residual angle without diplopia. The results are much more stable than in childhood—a short-term repeated increase of the squint angle, very common in children, never occurs in elderly patients.

2.5.7. Discussion concerning the pathogenesis

As usual, there are multiple determinant factors to be considered.

- Divergence insufficiency or palsy:

  Neurologists point out correspondingly that divergence palsy exists only within the scope of neurological diseases. Indeed, I checked 17 of my age-related convergent patients at the synoptophore and all patients showed a fusion range into divergence of 4–5 degrees.

- Abducens nerve palsy—bilateral or unilateral:

  The onset of palsy is always a shocking event which arises suddenly. One patient, I remember, was wearing prismatic correction for distance esotropia; some months later, a unilateral abducens palsy had occurred additionally; the neurologic findings in MRI were vascular encephalopathy with multiple lesions, especially in the pons region. Curran [11] was of the opinion that simulated divergence palsy could be a phase in the evolution of real abducens palsy.

- Decompensated esophoria:

  Two of my patients showed the transition from distance orthophoria to convergent squint because of forced accommodative convergence to overcome near exophoria, a spasm of convergence which cannot be relaxed in a short time. One of the patients was a dentist aged 55; 10 years later after retiring from his profession, the distant deviation regressed to orthophoria. A similar mechanism can be supposed for hyperopic patients refusing to wear glasses. We do not observe such binocular reactions exclusively in the elderly but also in younger patients.

- Connective tissue degeneration in elderly patients [10]:

  A degeneration of the connective band between the lateral and superior rectus muscle causes the lateral muscle to slip downwards, followed by esotropia and hypotropia of the bulb. This clinical entity was called "sagging eye syndrome" and resembles the "heavy eye syndrome" of high myopia, the first additionally being connected to ptosis and loss of orbital fat.

- Age-related myopathy of the eye muscles:

  In my opinion, the high-grade loss of elasticity is an essential factor explaining the disparity between the positions far/near. Performing surgery on patients in their 80s, I could notice the extreme density of their external rectus muscle, described [12] as senile fibrosis.

2.6. Cataract, aphakia, pseudophakia and binocularity

Everyone has to expect the acquisition of cataract, if the life is long enough; commonly, the cataract will develop bilaterally but not to the same degree. These days, we can operate every stage of cataract, be it binocular or monocular, by intraocular lens implantation. Yet, problems occur and disappointed patients will complain of diplopia.

2.6.1. Cataract per se causes diplopia

Cortical opacities of the lens, such as a central spindle, generally cause monocular double contours or even triplopia. Cloudiness of the lens nucleus causes myopia and, if decentered, may additionally have a prismatic effect, evoking binocular diplopia.

2.6.2. Diplopia after long-standing unilateral cataract or aphakia

The most difficult cases were those we saw 20–30 years ago, after the new technique of intraocular lens implantation (IOL) had entered into practice everywhere. A lot of patients with long-standing unilateral cataract or aphakia hoped to gain binocularity again, but instead of binocular depth perception they acquired intractable diplopia. Medical science had to learn some new facts [13]: Interruption of fusion over some years caused by a unilateral visual defect, effectively approaching blindness, represents visual deprivation in adults, which results in a total loss of fusion ability, reversible only in few exceptional cases. Unlike the visual deprivation of newborn children, which almost always causes irreversible amblyopia, the visual acuity of adults is hardly impaired. Through own binocular infrared oculography (IROG) studies [14] we were able to find out that the excursions of the interfixation movements (normally less than 30′ = arc minutes) of the deprived eyes were enlarged by up to 4°30′; that is an absolute obstacle for fusion.

Finally, a recommendation for the daily work of ophthalmologists: A patient having unilateral cataract and VA less than 0.3 should not hesitate longer than 1 year to be operated to avoid the postoperative risk of double vision.

2.6.3. Unexpected diplopia after cataract operations with IOL implant

Even patients who have never had binocular troubles may acquire double vision after cataract surgery. Studying such cases we found that most of them had a pre-existing compensated imbalance of their eye muscles, for example, a slight horizontal or vertical deviation.

During the period of progressive lens opacity not only will the VA be slowly reduced but so will the fusion power and thereby misalignments of the eyes are manifest, and even a distance esophoria may develop.

2.6.4. Prismatic glasses before and after cataract surgery

A group of 25 patients wearing prismatic glasses because of asthenopic complaints or diplopia returned to my office after receiving cataract surgery and wanted the prescription of new glasses. The results are shown in **Table 2**. An improvement (by up to 12Δ) was remarkable,

6 patients	Prisms unchanged
11 patients	Improvement 2–12Δ included: 8 distance esotropia, 2 exotropia and 1 hypertropia
8 patients	Deterioration 1–4Δ included: 6 hypertropia, 2 esotropia

Table 2. Change of prisms after cataract surgery.

especially in cases having convergent deviations based on high astigmatic anisometropia which could be eliminated by the operation. Vertical deviations are severe obstacles for fusion and mostly remain unchanged after cataract surgery.

2.7. Glaucoma and binocularity

In early stages, glaucoma does not impair binocular functions. Difficulties arise with the slowly progressive defects of the visual field, mainly in the nasal areas, causing loss of fusion power and stereo vision. Subjective complaints of glaucoma patients are rare; the event of seeing double pictures only occurs exceptionally because there is no perception in damaged areas of the visual field. Yet, there are some consequences to be mentioned.

2.7.1. Divergent squint for reading distance and diplopia

Bilateral total defects of the nasal visual field as far as the fixation point are equivalent to the total loss of fusion and of convergence. The consequences are a divergent eye position for near distances, inability of reading, lack of stereo vision and crossed diplopia or unilateral suppression, although the VA of both eyes may be 1.0. Imagine the difficulties such a patient has with reading: The left eye is able to perceive the beginning of a line but the following words have vanished and can only be seen by the right eye, which cannot join them to the word seen by the left eye because of the squint position. The prismatic correction of the squint angle could enable both eyes to read the continuous chain of words alternatingly.

2.7.2. Diplopia evoked by an almost blind eye

Case 2: A woman aged 75, whose glaucoma I had treated years ago, called me in despair because the newly consulted ophthalmologist did not believe her complaints: Her left blind eye caused her to perceive double pictures. Indeed, she had a total atrophy of the optic nerve with only peripheral remnants of the visual field and the left bulb had drifted into a divergent position. The trial to correct the squint angle by a press-on prism 15 Δ base attached to the left glass was successful, and the patient was content.

2.7.3. Masked diplopia and glaucoma

Case 3: A chemist, aged 76, treated for glaucoma elsewhere, called me, saying "My vision is very bad, can you help me?" His eye tension was well adapted, the visual field showed intermediate defects and his corrected VA was RE: 0.8–1.0 and LE: 0.7–0.8. While testing his vision we noticed that the binocular vision was worse than the monocular, and the test objects seemed to be blurred; a state like this is a clue to binocular problems. In fact there was a

manifest distant deviation of +6° and a latent near angle of +2°. Prisms of 6 Δ added to the distance glasses immediately produced "clear" binocular distant vision.

2.8. Age-related maculopathy and binocularity

Although modern treatments are able to prevent wet maculopathies with neovascularization from developing into tumor-like fibrotic scars, enough problems remain to be discussed such as the influence of binocularity on reading ability and hence the quality of life; such considerations seem to be neglected in ophthalmology.

2.8.1. Correction of the refraction error even for eyes with impaired vision

Subjective vision testing of visually disabled old patients is fraught with difficulties: The patient cannot decide which glass is the better one, wants magnification and prefers plus glasses too much; the duochrome test is not predicative due to the color vision defect of a damaged retina. And last, the refractometer measuring is not exact for eyes which cannot fix centrally. To get exact data of the refractive error we have to perform retinoscopy in mydriasis and some days later do a subjective test once more.

In our experience the refraction error changes with aging: Myopia increases just as astigmatism against the rule.

Case 4: An intelligent woman, aged 79, maculopathy LE > RE, could read only using a magnifying lens; the left eye had no reading ability at all, no squint position. The patient complained that her ophthalmologist had not given her a new glass for the left eye, but this eye supported the right eye and she needed it. The left glass was strengthened with +2,25sph +1,0x180°; with the new glasses the patient could read 0.4 text without magnification.

There exist some more examples to teach us that the refractive correction of an almost blind eye is highly beneficial for the master eye provided that the eyes are in a parallel position.

2.8.2. Correction of divergent eye positions for reading distance

In spite of fitting glasses, the next complaint of a patient may be: "I can only read the newspaper when closing the worse eye". This is a typical statement indicating the presence of a divergent squint when looking at near objects. These patients never complain of diplopia but show the symptoms of masked diplopia. For cases with one highly impaired eye we use Fresnel prisms, added to the glass of the worse eye; this procedure will be well tolerated and the prism can be changed easily as soon as the squint angle varies. With restored binocularity, the patient has a sudden experience of regained clear vision and no longer feels compelled to close the worse eye. Unfortunately, maculopathy is progressive and the initial success may be lost quickly.

2.8.3. Squint position and maculopathy

The onset of a squint position depends on the size and the distance to the foveola of the central scotoma and whether it is sieve like or dense. No squint will develop if both eyes are deteriorating to the same degree.

Binocularity supports the reading ability most effectively if the damage is bilateral but with a large difference in vision, for example, 0.5 and 0.05. The peripheral fusion of the damaged eye is conductive to the macular orientation of the better eye and causes an increase of reading speed, provided an existing squint position is corrected by prisms.

2.8.4. Central retinal defects and binocular problems

The use of optical coherence tomography (OCT) allows seeing the pathology of the retina, swellings, cysts, distortions of the retinal layers and many more. The visual acuity of such eyes often may be almost normal but the optical pictures of both eyes don't fit one to the other; the central fusion will be disrupted, the reading speed will be slower binocularly than monocularly and the patient will close one eye. If the affected eye is the non-dominant one, central suppression will develop. The confusing quality of the vision of a dominant eye can be reduced temporarily by hazy foils (**Figure 1**).

2.9. Strabismus since childhood

Although patients in general have been accustomed to their visual defects since their youth, situations evoking diplopia may occur even in later life.

2.9.1. Spontaneous change of the squint angle of amblyopic eyes

Amblyopic eyes have fewer zones of suppression than alternating eyes. With aging, strabismic eyes have the tendency to drift into divergence, causing double pictures. The treatment consists of bringing back the deviating eye into the original position, either by prisms or by an operation.

2.9.2. Postoperative muscular insufficiency simulating palsy

Case 4: A middle-aged woman once noticed double pictures when looking to the right side; a neurologist diagnosed a palsy of the right abducens nerve. This diagnosis would have been correct in the presence of a convergent eye position while looking to the right, but, in fact, the eyes were divergent. The patient told us that a recession of the internal rectus of the left eye had been performed many years ago. With aging, the power of a weakened muscle will decrease even more; thus, an incomitant squint is produced without being caused by the palsy of a nerve.

2.9.3. Intractable diplopia after squint surgery

Squint surgery in childhood previous to the age of 3–4 years is a precondition for the establishment of certain imperfect cooperation between both eyes, a sort of anomalous fusion. Such

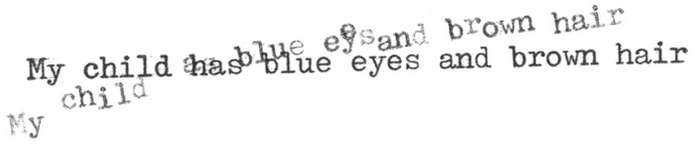

Figure 1. This is to imagine how a patient, having macular pathology, sees.

patients commonly do not develop diplopia in later life. But this danger is high for patients squinting since early childhood and operated with a delay of 10–20 years. In my office I have seen a lot of patients having had diplopia all their lives. When asked if they were suffering from their immanent double pictures the patients stated in agreement that these unreal shadows were only a problem when they paid attention to them.

2.10. Heavy eye syndrome

This name is derived from the anatomical position of an eye, more myopic than the fellow eye, and hence larger and weightier, which causes a slight, unnoticeable displacement downwards. Our fusion ability to overcome vertical deviations is only 2°, and deviations more than this result in diplopia.

Case 5: A lawyer, aged 60, consulted me in despair. Since a few months, reading had become extremely difficult; the letters appeared double in the vertical direction, and he was hardly able to practice his profession. Some ophthalmologists he had consulted could not help. His glasses were RE −4.5 s + 1.0x15°, LE −6.0 s +0.5x90° and +2.5 s addition for near distances. VA was 1.0 for both eyes. The vertical deviation for near fixation in primary position was +VD 4° (=8Δ); for measuring we used the Lees-Screen, producing full dissociation of fusion.

Prescription of glasses: For his myopic correction ground-in prisms were added—RE 2.5 Δ base down, LE 2.5 Δ base up. With these glasses the patient was highly content and could work again without difficulties. As explained in Section 5.6.1, we gave prisms with just the strength necessary to overcome the deviation.

Patients having such problems are often middle aged and always myopic. In my experience, myopia may increase gradually from youth until the age of 60–65 and afterwards decrease again, due to the influence of presbyopia. These changes in refraction are not always developing symmetrically in both eyes, with the consequence of anisometropia and heavy eye syndrome.

3. Diplopia and general diseases without age limit

3.1. Parkinson's disease

When dealing with this disease, neurologic symptoms will, of course, have priority. Patients consulting an ophthalmologist will usually not declare themselves spontaneously to suffer from "Parkinson". An experienced medical practitioner, however, can easily recognize those patients, observing their stiff movements and the blank, expressionless stare. The diagnose makes us consider a number of ocular symptoms connected with this disease (**Table 3**).

The complaints of 17 patients at my office were diplopia at all distances (4), only at reading distance (12) and unilateral blindness due to glaucoma (1).

The most important problem is the convergence insufficiency with a consequent divergent eye position for near distances, causing reading inability. The first step to helping these patients is to measure the most comfortable reading distance between the eyes and the book, which

Convergence insufficiency
Loss of stable fixation
Dry eyes due to a decreased blink rate
Hypometric saccades
Decreased excursions of up-gaze

Table 3. Ophthalmologic symptoms of Parkinson's disease.

should be seen binocularly. Many patients use a reading stand because of their hands' tremors. For this specific distance we determine not only the strength of the reading glasses but also the prisms necessary to compensate the divergent position. Press-on prisms are not advisable to be put on the patients' own reading glasses for trial; they would be rejected for blurring.

Additionally, a squint position for distance may be present. Two of our patients had a convergent and three a slightly vertical deviation. Those patients needed separate prismatic glasses for each distance.

Another problem is the instability of fixation, resembling a tremor of the eyes. Using IROG we found an anomalously high number of square-wave jerks in both eyes when fixing an object [2]. This fact, combined with inexact movements, may evoke an impression of double vision, perceptible only by the patient and only for a split second.

Finally, a prescription for wetting eye drops must not be forgotten.

3.2. Endocrine ophthalmopathy: Graefe's disease

This disease commonly affects middle-aged patients; the onset may be acute with swelling of the eyes and exophthalmos or insidious with asthenopic complaints and slowly developing diplopia. The first sign may be a restriction of elevation and convergence, unilateral or bilaterally asymmetric, caused by the swelling of the eye muscles. The treatment of the acute stage requires the cooperation of several medical disciplines.

The treatment of late stages is the task of the ophthalmologists. Owing to the inflammation, the muscle fibers are replaced by fibrotic tissues and lose their elasticity; consequently, the excursion of the bulb to the opposite side of the affected muscle is restricted, a condition proved by *the forced duction test.* First, the elevating muscles are afflicted, rectus inferior and oblique inferior, and then a neurogenic palsy of superior rectus and oblique is simulated. The treatment of choice is an operation, which is possible once the deviation has stabilized.

3.3. Myasthenia gravis

This disease is based on a defective neuromuscular junction, a lack of acetylcholine and it is interesting for ophthalmologists because the first symptoms often are a uni- or bilateral ptosis and diplopia. In the course of a day the symptoms will deteriorate. The double pictures cannot be related to a certain eye muscle, like in a neurogenic palsy, and they are highly changeable;

therefore, they cannot be corrected by prisms. Blood tests and stress tests will confirm the diagnosis. In general, neurologists will take charge of the treatment.

Yet, we recommend the colleagues of ophthalmology to consider the possible presence of myasthenia in cases of unsettled asthenopic complaints or unstable heterophoria.

4. Diagnostics

This chapter is aimed at ophthalmologists without orthoptic equipment and at colleagues of other disciplines, such as neurology and geriatrics. With simple methods and a minimum of instruments, it is possible to find out whether double vision is due to an ophthalmologic or a neurologic disorder.

What we need:

1. A non-blinding penlight for the inspection of the eyes, for fixation in a near distance and to check the motility of the eyes.
2. A Maddox-Cross, mounted at a 5-m distance, serving as a fixation light for measuring the angle of squint in various directions of gaze.
3. Two red glasses, one bright and the other dark, for different degrees of fusion dissociation, while measuring the eye deviation.
4. Bagolini glasses for examinations under near physiological conditions.
5. A Maddox-Wing test for measuring deviations in near distances.
6. Occluder.

Partially, diagnostic instructions have already been given on the previous pages. A few most important items are to be further explained: The cover test, the motility of the eyes and their muscles and the Bagolini glasses.

4.1. Cover tests

Cover tests (CTs) are the most important diagnostic tools for the comitant and the incomitant squint. To estimate the existence of muscle palsy, the alternating CT is useful, especially for patients who are not able to speak (**Tables 4** and **5**).

4.2. The movements of the eyes and their muscles

To check the eye motility, a torch is moved in the main directions of gaze, monocular and, what is more important, binocular, while the head is fixed. For the main action of each muscle, see **Figure 2**. In the presence of double pictures the examiner has to ask in which direction the lights have the largest space between them; a red glass before the right eye enables to differentiate the eye which each light belongs to; the light in the most outward position indicates the palsied muscle.

The patient fixes an object	a. in the distance b. in the proximity
Alternate covering of both eyes, every second, without interruption	
No movement = Orthophoria,	
but exceptionally strabismus + amblyopia + eccentric fixation	
(corneal reflexes to be noticed!)	
Movement from nasal side = Esotropia or esophoria	
Movement from temporal side = Exotropia or exophoria	
-tropia = manifest squint	
-phoria = latent squint, decompensation by fusion interruption	
both can be differentiated by the unilateral cover-uncover test	

Table 4. The alternate cover test.

Both eyes must be checked for distance and near fixation, and an interval to regain the original position is necessary.
Observation of the **free eye**
1. While **covering** the fellow eye:
a. No fixation movement = Heterophoria
b. Fixation movement = Heterotropia
2. While **uncovering** the fellow eye:
a. Fixation maintained = alternating squint
b. Fixation lost = unilateral squint

Table 5. The unilateral cover-uncover test.

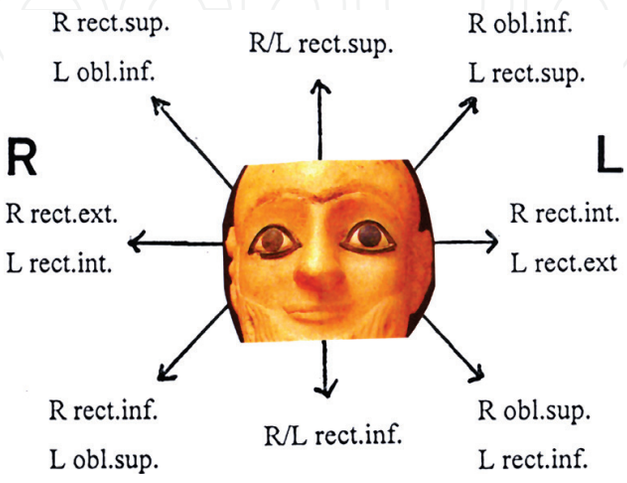

Figure 2. The action of the eye muscles. The face is a photo of an antique statue.

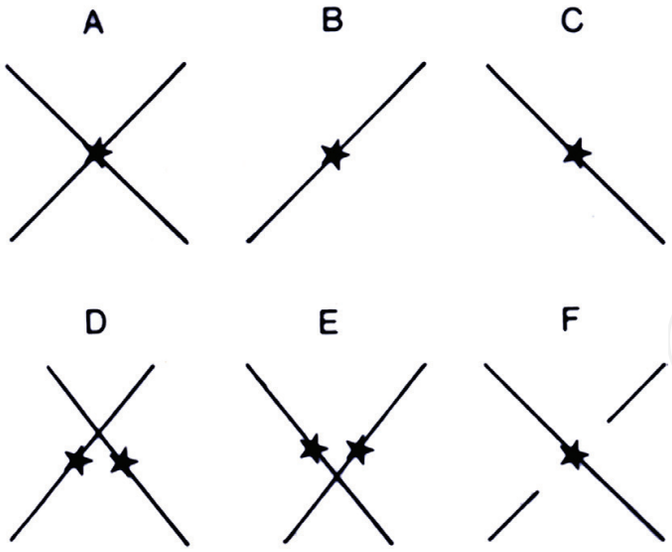

Figure 3. Some results using Bagolini glasses: (A) orthophoria (or microtropia), (B) suppression of RE, (C) suppression of LE, (D) uncrossed diplopia (esotropia), (E) crossed diplopia (exotropia), and (F) central scotoma without squint.

4.3. The Bagolini glasses

The Bagolini glasses, always used binocularly, give information about the eye position, fusion ability or suppression. They should only be used in addition to the best correcting glasses of the refraction error. The possible results are shown in **Figure 3**.

5. Conclusion: differential diagnosis of double vision in the elderly

Ocular diplopia:

- anamnesis most important
- onset slowly, unnoticed
- concomitant motility pattern
- loss of binocularity without diplopia = "masked diplopia"
- intractable diplopia in the adulthood of infantile squinters or after the operation of a long-standing cataract

Neurogenic diplopia

- onset sudden, flash-like
- incomitant motility pattern
- intractable diplopia after severe craniocerebral trauma

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Other declarations

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References

[1] Stangler-Zuschrott E. Strabismus convergens des Presbyopenalters (convergent strabismus in the prebyopic age). Klinische Monatsblätter Augenheilkuinde. 1976;**168**:775-783

[2] Stangler-Zuschrott E. Geriatric Orthoptics and Non-Paretic Diplopia in Adults. Nova Science Publishers; 2016. 126 p. ISBN 978-1-63485-244-9

[3] Mühlendyk H, Syed A. Histological and ultrastructural studies on the ringbands in human extraocular muscles. Albrecht von Graefes Archiv für Klinische und Experimentelle Ophthalmologie. 1978;**108**:177-191

[4] Sachsenweger R. Altern und Auge. Leipzig: VEB Georg Thieme; 1971

[5] Shechtman D, Shallo-Hoffman J, et al. Maximum Angle of Ocular Duction During Visual Fixation as a Function of age. Strabismus. 2005;**13**:21-26

[6] Piper HF. Formen des motorischen Abbaus im Senium. Bericht 63.Zus. Dtsch. Ophthalm. Ges. Berlin 1960. pp. 308-313. Bergmann Verlag 1961

[7] Mittelman D. Age-related distance esotropia. Journal of AAPOS. 2006;**10**:212-213

[8] Dannheim-de Decker E, de Decker W. Strabismus convergens im Senium. Zeitschrift für praktische Augenheilkunde & augenärztliche Fortbildung. Heidelberg: Kaden Verlag; 1994;**16**:263-266

[9] Bielschowsky A. Das Einwärtsschielen der Myopen. Jena: Zusammenkunft DOG; 1922. p. 245

[10] Chaudhuri Z, Demer JL. Sagging eye syndrome: Connective tissue involution as a cause of horizontal and vertical strabismus in older patients. JAMA Ophthalmology. 2013;**131**:619-625

[11] Curran R. True and simulated divergence palsy as a precursor of benign sixth nerve palsy. Binocular Vision Quarterly. 1989;**4**:125-130

[12] Stangler-Zuschrott E. Schieloperationen an presbyopen Patienten. Klinische Monatsblätter Augenheilkuinde. 1982;**181**:397-399

[13] Pratt-Johnson JA. Fusion ability lost and regained in visual adults. Albrecht von Graefe's Archive for Clinical and Experimental Ophthalmology. 1988;**226**:111-112

[14] Stangler-Zuschrott E. Horror fusionis. Eine Störung sensomotorischer Funktionen (Central fusion disruption, a disorder of sensomotoric functions), Spektrum Augenheilk. 2005;**19**: 280-289