Persistent Diplopia in Visually Mature Patients. Is it Intractable or something else? A Review and Case Series

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ABSTRACT

Diplopia is described as being intractable when there is inability to both fuse the two images and suppress the second image. Intractable diplopia persists despite achieving ocular alignment using either prisms, lenses, vision therapy, extraocular muscle surgery, or botulinum toxin injection. Treatment usually resorts to occluding or fogging the patient’s nondominant eye. Often times, however, adults having other causative mechanisms for supposedly persistent diplopia are able to achieve comfortable single vision with treatment that either establishes fusion or reactivates a preexisting sensory adaptation. This case series reviews these other causes of diplopia.

INTRODUCTION

Diplopia is the condition in which a single image is perceived simultaneously as being two rather than one.1 When on examination there is inability to fuse the two images and also suppress the second image, the diplopia is intractable.2,3 Intractable diplopia is constant and occurs in all positions of gaze despite achieving satisfactory ocular alignment with either prisms, lenses, vision therapy, extraocular muscle surgery, or botulinum toxin injection. Some patients may actively avoid fusion by performing involuntary ocular movements of their nonfixating eye, reporting one image jumping over the other when the strabismus is neutralized either with prisms or with haploscopic devices such as the synoptophore.4-6

Since fusion cannot be established, treatment for intractable diplopia is usually palliative. Occlusion or fogging of the nondominant eye with patches,3 frosted spectacle lenses,7 partially occlusive filters,8,9 monovision glasses or monovision contact lenses,10,11 occlusive contact lenses,3,7,12 corneal tattooing,13,14 opaque intraocular lenses,15,16 and botulinum toxin induced ptosis13 have been used to ameliorate intractable diplopia.

Although it has historically been reported to be a consequence of either amblyopia or strabismus treatment, the consensus is that intractable diplopia occurs infrequently.17-21 Cumulative findings from three studies involving adults manifesting constant strabismus without diplopia prior to extraocular muscle surgery reported intractable diplopia developing in only 12 of 899 patients (1.3%) postoperatively.22-24 Furthermore, strabismus that began in early childhood and either was untreated or treated and recurred is usually accompanied by sensory adaptations such as suppression
and/or anomalous retinal correspondence which prevent diplopia. However, in some instances these sensory adaptations may not be permanent and can be changed or lost over the years as illustrated in Case 1.

Case 1
A 17 year-old high school student was referred for constant and long-standing horizontal diplopia which he described as minimally bothersome. He desired to obtain a driver's license.

Ocular history was significant for bilateral optic neuritis and uveitis at 4 months of age. The patient’s systemic history was unremarkable. Extraocular muscle surgery in the left eye for probable infantile esotropia occurred at 3 years of age. Bilateral cataract surgery was performed at 8 years of age and YAG capsulotomy in the right eye at 14 years of age. Prism glasses were prescribed when he was 12 years old but was not helpful. He denied having had any patching or orthoptics/vision therapy.

On examination refractive correction and corrected visual acuities were right eye -1.25 – 2.50 x 14 (20/25) and left eye -1.50 -1.00 x 155 (20/25). The diplopia, described as being horizontal, was absent when either eye was covered. A constant left exotropia of 30 prism diopters (PD) was revealed with the cover test at distance and near. The exotropia was accompanied by bilateral dissociated vertical deviation. Latent nystagmus was present in the left eye. Versions showed bilateral superior oblique overaction with larger exotropia in downgaze.

The patient responded to sensory testing paradoxically as if he was esotropic. With the Worth 4-dot and red lens tests at both distance and near, the diplopia was uncrossed rather than crossed, suggesting anomalous retinal correspondence. The diplopia could neither be fused nor suppressed with any amount of prism. Synoptophore evaluation measured an objective angle of 30 PD base-in and a subjective angle of 22 PD - 24 PD base-out confirming anomalous retinal correspondence.

The diagnosis was consecutive exotropia with intractable diplopia. Bangerter filters and/or -2 D (diopter) and +3 D Fresnel lenses placed over the left spectacle lens to eradicate the diplopia were uncomfortable for the patient. This was attributed to the blur possibly increasing the dissociated vertical deviation. A 0.2 density Bangerter filter providing sector occlusion and restricted to the central portion of the left spectacle lens used exclusively for driving was more acceptable.

Strabismus beginning in adulthood causes diplopia that usually resolves and fusion reestablished when the eyes are accurately aligned prismatic or surgically. However, intractable diplopia has been reported following prolonged visual deprivation due to cataract or from uncorrected unilateral aphakia in adults with sensory strabismus who eventually received secondary intraocular lens implantation.

Intractable diplopia has also been reported following severe head injury, ocular trauma, central nervous system disorders, post viral syndromes, intracranial surgery, and cerebral vascular accident or stroke as illustrated in Case 2.

Case 2
A 56 year-old man was referred with a complaint of constant diplopia which began following a stroke three years ago. He described the diplopia as being both vertical and horizontal and more bothersome when reading. The diplopia was absent with either eye covered. He denied having strabismus and any other eye disorders during childhood. The referring practitioner prescribed prism glasses that did not alleviate the diplopia. There had not been any strabismus surgery performed.

On examination, refractive correction and visual acuities were right eye Pl – 0.50 x 75 (20/25) and left eye Pl – 0.50 x 110 (20/25). He manifested with the cover test a left hypertropia of 10 PD in primary gaze at distance and near.
An 8 degree right excyclotorsion was measured with the double Maddox rod. Versions showed limited elevation in the right eye and limited depression in the left eye. With the doll’s head (oculocephalic reflex) maneuver, versions in both eyes improved suggesting a supranuclear cause for the impaired ocular motility.

Sensory testing with the Worth 4-dot test showed vertical diplopia at both distance and near. Stereopsis with the contour stereotargets (circles) of the Randot stereotest (Stereo Optical Co, Inc, Chicago, ILL) was nil. With the synoptophore which compensated for the torsional as well as the vertical deviation, normal retinal correspondence and “fleeting sensory fusion” were demonstrated. Motor fusion was extremely poor. Moving the tubes of the synoptophore less than 1 PD in any direction caused diplopia.

It was noted that the present glasses had ground-in prism with 3 PD base-down in the right lens and 8 PD base-up in the left lens, which corrected for left hypotropia, not left hypertropia. Using glasses without ground-in prism, treatment included 8 PD base-up Fresnel prism over the upper portion of the right spectacle lens. Since diplopia was more bothersome when reading and achieving fusion at both distance near seemed improbable, a 0.6 density Bangerter filter over the lower portion of the right spectacle lens was included.

One month later diplopia persisted. In this clinician’s opinion, the absence of motor fusion and impaired ocular motility in each eye made prism therapy and any other therapy likely ineffective. Treatment consisted of prescribing a 0.3 density Bangerter filter that eliminated the diplopia over the entire right spectacle lens.

Comment

Both patients, Case 1 with childhood onset strabismus and Case 2 with adult onset strabismus, met the criteria for intractable diplopia, showing absence of both fusion and suppression. Case 2 demonstrated “fleeting sensory fusion” with the synoptophore. Due to the lack of demonstrable motor fusion, this most likely indicated superimposition of the synoptophore targets rather than actual sensory fusion of the targets.

Since intractable diplopia can have a substantial impact on the patient’s quality of life and occurs infrequently, it should be a diagnosis of exclusion and made only after other causes of diplopia have been ruled out. Many patients I have examined over the years had other types of diplopia that could be treated without constantly occluding or fogging one eye. They were able to achieve comfortable single vision by either changing their optical prescription, altering their fixation pattern or head posture, using prisms, or undergoing vision therapy or extraocular muscle surgery.

The purpose of this report is to review other types of diplopia that should be considered in adults and visually mature patients with persistent diplopia. Representative case reports are included.

Monocular Diplopia

Monocular diplopia is when two images of the object of regard are seen by one eye alone. It persists when one eye is covered. It may be constant or intermittent, unilateral or bilateral, vertical or horizontal, and occur with or without strabismus. The patient frequently describes perceiving a halo, ghost image, image overlapping or stacking rather than two distinct separate images. Approximately 6% to 12% of patients with diplopic symptoms have monocular diplopia.

Causes of monocular diplopia are numerous being either extraocular or optical, or organic. Extraocular or optical are the most common and can be associated with uncorrected or inadequately corrected refractive error (especially astigmatism), reflections from spectacle lens surfaces, incorrect placement of a bifocal segment, tear film abnormalities, lid abnormalities, corneal irregularity (i.e.,
scarring from refractive surgery or keratoconus which frequently can be corrected with rigid contact lenses), iris abnormalities, cataract, or a dislocated intraocular lens. 34,35,36,40 Incipient cataract with zones of increased optical density in the anterior and posterior subcapsular or nuclear layers of the lens is frequently causative. 34,35 Monocular diplopia attributed to optical causes usually resolves when placing a pinhole before the affected eye. 34

Organic causes include neurogenic disease, migraine, and retinal abnormalities such as epiretinal membrane, choroidal neovascular membrane, and macular edema. Monocular diplopia that is organic does not resolve with a pinhole. 34

When occurring with strabismus where diplopia is expected to be binocular, monocular diplopia may not be considered as the source of the patient’s symptoms.

**Case 3**

A 62 year-old woman was referred for long-standing diplopia. She described the diplopia as being vertical in direction. She reported her eyes being misaligned since childhood. Treatment included glasses and vision therapy for many years, the latter given both as a child and as an adult. She denied having had prism glasses, patching, or surgical treatment.

On examination, refractive correction and visual acuities for the right eye were +5.75 - 0.50 x 70 (20/30) and the left eye +6.50 - 3.50 x 107 (20/50). Cover test revealed orthophoria at distance and intermittent left exotropia of 10 PD at near. With the vertical Maddox rod, a left hyper deviation of 0.5 PD was measured at both distance and near. Versions were full in all positions of gaze. Stereopsis with the contour stereotargets of the Randot stereotest was 200 arc seconds. Fusional vergence amplitudes at near were 16/10 and 10/8 for divergence and convergence, respectively.

Further testing indicated that when the right eye was covered, diplopia persisted and when the left eye was covered, diplopia was absent, confirming monocular diplopia. When placing a pinhole aperture before her left eye, diplopia resolved. Biomicroscopy and dilated ophthalmoscopy revealed no apparent causes for the monocular diplopia. It was noted that her spectacle lenses were scratched, a large diagonal scratch occurring on the left lens. A manifest refraction was right eye + 6.25 - 0.50 x 73 and left eye + 7.00 - 3.00 x 100 and gave 20/30 visual acuity in each eye. Placing the refraction in a trial frame eliminated the monocular diplopia in her left eye.

The diagnosis was intermittent exotropia with optically-induced monocular diplopia. She was prescribed the updated refraction with a +2.50 add and no longer had diplopic symptoms.

**Comment**

Case 3 illustrates the importance of ruling out monocular diplopia with patients manifesting strabismus having diplopic symptoms. The patient had intermittent exotropia and had undergone vision therapy both as a child and as an adult. Checking the patient’s refraction and quality of the spectacle lenses should not be overlooked or discounted in patients having monocular diplopia. 37,38

**Spontaneous and Rapid Alternate Fixation**

Adults having strabismus since childhood may notice a shift or jump in apparent position of the fixation target if they rapidly change fixation from one eye to the other eye. 25,41 They frequently describe this phenomenon as diplopia. Rapid alternate fixation is more common with patients unable to fuse having small-angle deviations and is particularly bothersome when driving, playing sports, and doing close work.

Observing the patient’s fixation pattern and relating it to findings from sensory tests is essential for the correct diagnosis. With the Worth 4-dot test, for example, patients may report diplopia, seeing five lights. The clinician should inquire whether the five lights
are present simultaneously or whether they alternate between two and three lights very quickly. When maintaining fixation with each eye in turn, “diplopia” is not present. Treatment of rapid alternate fixation encourages the patient to develop and fixate exclusively with his/her dominant eye.

**Case 4**

A 44 year-old woman was referred for diplopia which reportedly began in childhood. She described the diplopia as being both horizontal and vertical. Ocular history included amblyopia treatment as a child that involved full-time alternate day patching, patching the right eye one day and patching the left eye the next day. This was done for 3 years. There had been no other treatment.

On examination visual acuity refractive error and visual acuities were +0.50 -0.25 x 115 and 20/20 in each eye. The patient manifested a constant, alternating esotropia of 2 PD at distance and 14 PD at near. No vertical deviation was detected. Versions were full in all positions of gaze.

Sensory testing with the Worth 4-dot test revealed uncrossed diplopia at both distance and near. Stereopsis was nil with the contour stereotargets of the Randot stereotest. The synoptophore indicated normal retinal correspondence without evidence of fusion.

Observation of the patient’s fixation pattern indicated that she rapidly alternated fixation between the eyes. Repeating the Worth 4-dot test showed that she did not perceive five lights simultaneously. Maintaining fixation with either eye resulted in suppression of the contralateral eye, seeing either two or three lights depending on which eye was fixating at the time.

The diagnosis was esotropia with rapid alternating fixation. In an attempt to disrupt her habitual fixation pattern, a 0.3 density Bangerter filter was placed over the right spectacle lens. This was the least dense filter that eliminated the rapid alternate fixation. On follow up, the patient reported less “diplopia” but found wearing the filter annoying. Switching the filter to the left spectacle lens was more acceptable.

The patient returned to her referring practitioner with the treatment plan of gradually tapering the density of the filter and possibly its discontinuation while maintaining fixation exclusively with her right eye.

**Case 5**

A 45 year-old police officer was referred for diplopia since childhood. The referring practitioner diagnosed intractable diplopia and treated him with an occlusive contact lens. Ocular history included four strabismus surgeries, the first at 4 years of age and the last when he was 25 years old. He had undergone vision therapy from 4 years old to 12 years old. The latter was given by an orthoptist and reportedly included fusion and antisuppression procedures. The patient denied any patching or prism therapy.

Present examination revealed refractive correction and corrected visual acuities for right eye Pl – 1.00 x 73 (20/25) and for left eye -0.75 – 0.75 x 75 (20/20). The patient manifested a constant right exotropia of 12 PD at distance and 18 PD at near. Despite the slight difference in visual acuities, he could readily maintain fixation with his right eye. No vertical or torsional deviation was detected. Versions indicated full ocular motility in all positions of gaze.

When covering either eye, diplopia was absent. With the red lens test, uncrossed diplopia was perceived at distance and crossed diplopia at near. The Worth 4-dot test showed uncrossed diplopia at distance and near. Fusion could not be demonstrated when neutralizing the strabismus with prisms. The Randot stereotest, however, revealed 70 arc seconds and 250 arc seconds with contour stereotargets and random dot stereotargets, respectively. Synoptophore testing indicated normal retinal correspondence, sensory fusion with stereopsis, and a limited range of motor fusion.
Due to the presence of reasonably good stereopsis, it was concluded that the patient did not have constant exotropia with intractable diplopia but rather intermittent exotropia with fusion capability, albeit tenuous. Home-based vision therapy procedures using stereoscopic vectograms 20 minutes per day were prescribed to enhance fusional vergence amplitudes and vergence facility.

On follow-up, he reported compliance with the home-based exercises but no relief of diplopia. Sensorimotor testing showed findings consistent with the previous visit. Careful questioning with the Worth 4-dot test, however, indicated that he perceived not five lights simultaneously but rather two and then three lights seen in rapid succession. Spontaneous alternate fixation between eyes was apparent. Having the patient consciously maintain fixation with either eye in turn resulted in suppression of the contralateral eye. These findings were confirmed with the red lens and Bagolini striated lens tests.

The diagnosis was recurrent intermittent exotropia with rapid spontaneous alternate fixation. Treatment was altered to encourage fixation exclusively with his dominant left eye. Placing a 0.4 density Bangerter filter on the spectacle lens over his right eye was the minimal density filter that inhibited the rapid alternate fixation and resolved the “diplopic” symptoms. The goal was to eventually taper and discontinue the filter with the possibility of improvement in fixation.

Comment

The extensive treatment during childhood for both patients may have caused the problematic fixation pattern in adulthood. Case 4 had full time alternating patching for 3 years and Case 5 had vision therapy for 8 years and also four extraocular muscle surgeries. Additional vision therapy and/or surgery to enhance the fusion capability for Case 5 were declined by the patient.

Both patients were given Bangerter filters (Reyser Optik AG, St. Gallen, Switzerland) which are used to treat amblyopia in children, and in adults having intractable diplopia (Case 1 and Case 2), and other types of diplopia that may not be immediately amenable to prisms, vision therapy, or extraocular muscle surgery. Bangerter filters or foils are thin plastic and translucent partially occlusive filters that degrade central vision and eliminate diplopia while allowing a full visual field and favorable cosmesis. Similar to a Fresnel prism, they are usually placed on the inner surface of the spectacle lens usually before the patient’s nondominant eye. The filters create a functional central scotoma in the eye with the filter under binocular viewing conditions. They vary in strength from the most dense (<0.1), which produces marked reduction in visual acuity (20/300), to the barely occlusive (0.8), in which nearly normal visual acuity (20/25) is possible. Since degradation in visual acuity is patient dependent and not always equal to manufacturer estimations, the weakest density filter that resolves the diplopic symptoms is prescribed. With less dense filters, peripheral fusion and stereopsis can be maintained in some cases.

The long term treatment effect of Bangerter filters for diplopia is unknown. The preferred method is to initially prescribe the weakest density filter that eliminates the diplopia and over time taper the filter’s density either partially or entirely if possible.

Fixation Switch Diplopia

As illustrated in Cases 4 and 5, when alternating strabismus occurs in patients with a history of childhood onset strabismus, suppression usually changes from one eye to the other eye with a switch in fixation. Patients having nonalternating strabismus and strong fixation preference may not be able to transfer suppression to their preferred or fixating eye. They instead experience diplopia when fixating with their nonpreferred or deviating eye. For example, a patient having left esotoropia with
suppression may experience diplopia when forced to fixate with the left eye. Referred to as fixation switch diplopia, the diplopia can be either intermittent or constant.\textsuperscript{44-50} Its diagnosis is confirmed when diplopia is present with only the right eye or only the left eye fixating under binocular viewing conditions.

Although it can be idiopathic, fixation switch diplopia is more frequently associated with a reduction in visual acuity in the preferred or fixating eye.\textsuperscript{49} This can occur naturally when a myopic refractive shift occurs either exclusively or more so in the preferred eye relative to the nonpreferred or deviating eye. If the distance visual acuity is reduced to a level below that of the nonpreferred eye, fixation may be switched to the nonpreferred eye, causing diplopia. Prescribing the optimal refractive correction usually restores the previous fixation preference and suppression. Fixation switch diplopia can also occur when a cataract or retinal disorders such as macular edema, age related macular degeneration, and retinal detachment reduces visual acuity in the preferred eye causing fixation to be switched to the nonpreferred eye having better visual acuity.\textsuperscript{4,49}

Fixation switch diplopia can be iatrogenically induced. Refractive corrections determined subjectively and without cycloplegia for adults having strabismic amblyopia can be causative.\textsuperscript{45} The possibly better visual acuity obtained in the amblyopic eye can lead to a fixation switch to that eye, resulting in diplopia. Adults with mild strabismic amblyopia without fusion capability should not have the visual acuity in their deviating eye improved to a level equal or better than that of their fixating eye to avoid fixation switch diplopia.

The use of monovision to treat presbyopia using contact lenses, corneal refractive surgery, or cataract surgery with intraocular lenses has become more common. Since monovision forces fixation with one eye at distance and the other eye at near, it may induce fixation switch diplopia in visually mature patients with a history of nonalternating childhood strabismus.\textsuperscript{45-47,49} Restoration of the original fixation pattern by reversing the monovision and prescribing the optimal refractive correction for each eye with glasses, contact lenses, or further refractive surgery usually resolves the diplopia.

Fixation switch diplopia is not uncommon. In a report on 152 adults with history of childhood strabismus having diplopic symptoms, 20 (13%) cases were attributed to fixation switch diplopia.\textsuperscript{40}

**Case 6**

A 34-year-old physician’s assistant was referred for long-standing and occasional diplopia. She had undergone two surgeries for childhood onset esotropia, the first at 5 years of age and the second two months before the current examination. The recent surgery did not alleviate the horizontal diplopia which was reported to be variable and more problematic when doing suturing. The patient also reported poor depth perception and blurred vision. She denied having had any occlusion, prisms, or vision therapy. She wore contact lenses for myopic anisometropia (right eye -7.00 D and left eye -5.25 D).

On examination corrected visual acuities were 20/20 in each eye. The patient manifested an esotropia that was barely noticeable at casual glance. Cover testing revealed an incomitant deviation. When fixating with her right eye at both distance and near, esotropia of 25 PD was measured whereas when fixating with her left eye, esotropia of 12 PD was measured. Version testing revealed a mild adduction deficiency in the right eye causing exotropia in extreme left gaze.

Sensory testing indicated that the diplopia was dependent upon which eye was fixating. With right eye fixation, diplopia occurred whereas with left eye fixation, there was suppression. This was confirmed with the Worth 4-dot test which showed with right eye fixation, five lights (uncrossed diplopia) and with left eye fixation, two or three lights (suppression) at distance and near. Sensory testing also
revealed that the strabismus was intermittent as indicated with the Randot stereo test (100 arc seconds for contour stereotargets and 250 arc seconds for random dot stereotargets). Cover test during stereotesting revealed absence of strabismus.

The diagnosis was incomitant strabismus with fixation switch diplopia. Since the visual problems were more troublesome for near and the patient had fusion capability, the minimum prism (8 PD base-out) and minimum added plus lenses (+1.50 D) that provided continuous fusion when suturing were prescribed over her contact lenses.

Comment
Treatment for fixation switch diplopia involves restoring the patient’s original fixation pattern which allowed suppression to occur. Case 6 was atypical for fixation switch diplopia since there was absence of visual acuity reduction and presence of fusion. The latter was revealed with stereotesting and reinforced with treatment. Additional extraocular muscle surgery was declined by the patient.

Changes in Ocular Alignment
Suppression associated with childhood onset strabismus tends to be regional as well as facultative. Hemiretinal suppression zones are specific to the direction of the strabismus developing mostly in the nasal hemiretina with esotropia and mostly in the temporal hemiretina with exotropia. Since suppression is not developed immediately or at all in visually mature patients, diplopia can result when there is a sudden change in the angle of strabismus. This can occur either spontaneously or following strabismus surgery, trauma, illness, paresis or mechanical restriction of an extraocular muscle(s), or changes in the patient’s refractive management or refractive needs.

Diplopia is likely when strabismus changes direction. If the eyes were esotropic since early childhood and become exotropic, diplopia is probable since the extra-foveal image is no longer on the nasal hemiretina but on the non-suppressed temporal hemiretina. Similarly if the eyes were exotropic since early childhood and become esotropic, diplopia is probable since the extra-foveal image is no longer on the temporal hemiretina but on the non-suppressed nasal hemiretina. Diplopia may also occur when the angle of strabismus increases or decreases yet remains in the same direction. Reverting back to the previous angle of deviation either with prisms or extraocular muscle surgery usually eliminates the diplopia.

When there is anomalous retinal correspondence and the magnitude of the strabismus changes, diplopia can be paradoxical. For example, in a patient with moderately-sized esotropia and anomalous retinal correspondence preoperatively and a small angle esotropia postoperatively, any diplopia will be perceived as being crossed rather than uncrossed.

Rather than actual hemiretinal suppression zones, a “trigger mechanism” operating on a hemiretinal basis has been proposed. This “trigger mechanism” determines whether suppression or diplopia occurs and is activated by the image of the fixation target crossing the midline of the retina from the nasal side to the temporal side or vice versa.

Changes in ocular alignment are a common cause of diplopia, occurring in 95 of 152 adults (63%) having strabismus dating back to childhood.

Case 7
A 39 year-old woman was referred for recent onset diplopia. The diplopia, which was horizontal in direction, developed three months earlier. She denied having recent trauma, illness, ocular surgery, or taking any medication. The patient’s ocular history included childhood onset left esotropia which was treated with glasses and surgery, the latter occurring at 7 years of age. Occlusion therapy was given for amblyopia. Records from the
referring practitioner indicated esotropia of 30 PD at distance and near prior to the onset of diplopia. All current ocular, systemic, and neurological health evaluations were normal.

On examination, corrected visual acuities with small hyperopic refractive correction were 20/25 and 20/50 for the right and left eyes, respectively. A constant left esotropia of 50 PD was measured at distance and near. Version testing revealed full ocular motility in all positions of gaze. Sensory testing with the Worth 4-dot and Bagolini striated lens tests at distance and near showed uncrossed diplopia which did not resolve with neutralizing prisms. Synoptophore evaluation showed an objective angle of 38 PD base-out and a subjective angle of 18 PD base-out, suggesting unharmonious anomalous retinal correspondence. With 15 PD base-out before her left eye, the diplopia was abated.

Treatment included prescribing the minimum prism power that provided continuous suppression (Fresnel prism 15 PD base-out over the left spectacle lens) and subsequent strabismus surgery.

Comment
When prior records indicate a change in the angle of strabismus for patients with recent onset diplopia, prisms can frequently provide single vision. For Case 7, 15 PD base-out prism optically simulated her prior angle of strabismus and placed the extrafoveal image within a preexisting suppression zone on her nasal hemiretina. Extraocular muscle surgery or botulinum toxin injection would be expected to produce similar results.

Regarding prisms, they can be used diagnostically to help identify patients at risk for diplopia by simulating changes in ocular alignment to be achieved surgically. This is more of a concern when strabismus surgery is to be performed on adults having neither diplopia nor fusion capability.

While wearing his/her refractive correction, the patient fixates an isolated Snellen optotype at distance. Loose prisms, a prism bar, or rotary prisms of increasing power are placed before the deviating eye. The patient is instructed to report if and when diplopia occurs and disappears as prism power is changed. When diplopia is reported, prism is decreased to determine at what point it can be eliminated and if findings are reproducible. During testing, the patient maintains fixation with his/her preferred eye so that rapid alternate fixation, which can mimic diplopia, is not induced (Case 4 and Case 5). If diplopia is elicited at or near the target angle to be achieved with surgery, Fresnel prisms can be prescribed to determine whether the diplopia subsides or persists over time.

Regarding usefulness of this procedure, reports indicate that although it is very sensitive, it has a relatively low positive-predictive value. Perceiving diplopia with prisms revealed only a small risk for temporary diplopia and minimal risk for developing intractable diplopia postoperatively. On the other hand, absence of diplopia with prisms was excellent assurance that diplopia would not occur postoperatively, indicating a high negative-predictive value.

The low positive-predictive value has been attributed to the different mechanisms involved when comparing the effect of prisms and extraocular muscle surgery. The use of prisms to simulate postoperative alignment does not account for changes in proprioceptive afferent input that occur following extraocular muscle surgery. Botulinum toxin injection reportedly allows better simulation of surgical alignment and has been shown to reduce the proportion of false-positive diplopia tests.

Diplopia occurring with horizontal strabismus that is vertically incomitant can appear to be intractable. When the magnitude of esotropia or exotropia changes by 10 PD-15 PD or more in up versus downgaze, it is referred to as an alphabet pattern strabismus (i.e., A, V, X, or Y pattern). For patients with alphabet patterns,
sensorimotor testing should not be restricted to primary gaze. Diplopia that appears to be intractable in one position of gaze does not rule out either suppression or fusion in other positions of gaze.

**Case 8**

A 42 year-old man was referred because of long-standing diplopia which became more troublesome with his new glasses. The diplopia was described as being horizontal and worse when reading and less when depressing his chin. The patient recalled having strabismus as a child. Prism glasses and contact lenses had been prescribed but did not relieve the diplopia. There had not been any strabismus surgery. He denied head trauma or any health concerns. The referring practitioner recently prescribed progressive addition lenses for incipient presbyopia.

On examination, refractive correction and visual acuities were right eye +1.25 D (20/20) and left eye +1.00 D (20/20) with a +1.50 D add. The patient manifested a constant left esotropia of 18 PD and 20 PD at distance and near, respectively. The amount of esotropia was similar in right and left gaze. In upgaze, there was absence of strabismus whereas in downgaze, esotropia measured 25 PD. Version testing showed full ocular motility in all positions of gaze.

Sensory findings concurred with the patient’s ocular alignment status. With the Worth 4-dot test, uncrossed diplopia occurred in primary and downgaze whereas in upgaze, the patient reported 4 lights (fusion). Stereopsis with the contour stereotargets of the Randot stereotest was nil in primary and downgaze whereas 100 arc seconds was measured in upgaze. Synoptophore testing showed normal retinal correspondence, sensory fusion with stereopsis, and fusional vergence amplitudes.

The diagnosis was diplopia due to long-standing V-pattern esotropia. Because the esotropia and diplopia increased in downgaze and there was demonstrable fusion, progressive addition lenses were discontinued. Diplopia resolved and fusion was maintained using multifocal contact lenses combined with prism glasses (9 PD base-out), the prism amount being the minimum that permitted fusion.

**Comment**

Progressive addition lenses may be contraindicated in presbyopes with alphabet pattern strabismus and fusion potential. Since having a wide transition zone between the distance and near segment, progressive addition lenses force patients to view further into downgaze when reading than with conventional flat top “line” bifocal lenses. Switching to high- set flat top bifocals or multifocal contact lenses may be helpful. This justifies the patient in Case 8 with V-pattern esotropia reporting the diplopia being more troublesome with his new glasses.

**Torsion**

Torsion or cyclotorsion is a wheel-like movement of the eye around its antero-posterior or fixation axis. Extorsion or excyclotorsion is wheel-like movement of the eye outward away from the nose whereas intorsion or incyclotorsion is wheel-like movement of the eye inward toward the nose.

Torsion occurs mostly with cyclovertical strabismus and can affect one or both eyes. When misalignment between the eyes involves the cyclovertical extraocular muscles, torsional diplopia may occur. Since patients with cyclovertical strabismus usually have coexisting vertical and horizontal deviations, the diplopic images will be described as being not only tilted, oblique, or slanted, but also vertical and horizontal, the amount being incomitant.

The most common cause of torsional diplopia is superior oblique palsy, accounting for approximately 67% of all cases. Other causes include palsies or restrictions involving the other cyclovertical extraocular muscles, thyroid myopathy, and skew deviation.
Optical conditions such as uncorrected astigmatism with an oblique axis or improper placement of the axis of an astigmatic correction can also cause torsion. Torsional diplopia can occur secondary to certain retinal disorders and macular translocation surgery.

Assessment of torsion is advisable for any patient whose diplopia cannot be resolved with offset of the deviation with prisms, particularly when the deviation is vertical. Torsion is measured in degrees subjectively using the double Maddox rod test which is considered the gold standard, the Bagolini striated lenses, the synoptohore, or the Hess-Lancaster test. It can also be measured objectively using either binocular indirect ophthalmoscopy or fundus photography to determine whether the fundus is anatomically rotated from its normal position.

The fovea is normally 0.3 disc diameters below and temporal to the center of the optic disc. While viewing the retina with indirect ophthalmoscopy when there is no torsion, an imagined horizontal line through the fovea will pass through the upper half of the optic disc. When there is torsion, the imagined line will appear either above (excyclotorsion) or below (incyclotorsion) the upper half of the optic disc in the involved eye. This technique requires much skill and experience to observe.

An amount of 6 degrees or more can degrade fusional vergence amplitudes and reduce stereopsis. Excyclotorsion exceeding 10-15 degrees frequently accompanies bilateral superior oblique palsy and can disrupt fusion entirely. Since prisms only have horizontal and vertical effect, torsional diplopia cannot be neutralized and thus can mimic intractable diplopia. It can be offset with the synoptophore along with any coexisting vertical/ horizontal deviations, allowing assessment for fusion. When fusion is demonstrated with the torsion neutralized, the diplopia is not intractable. Similar results can likely be achieved with accurate extraocular muscle surgery that specifically addresses the torsion along with other aspects of the strabismus.

Case 9
A 33 year-old man reported long-standing diplopia which he described as being torsional, vertical, and horizontal. Ocular history included three surgeries for strabismus caused by a car accident when he was 21 years-old. The last surgery was six years ago. Prism therapy had not been successful and it was suspected by the referring practitioner that he had intractable diplopia. The patient denied having strabismus or any other binocular vision disorder as a child.

On examination, corrected refractive error and visual acuities were for the right eye -0.75 – 0.50 x 22 (20/25) and for the left eye -0.25 D sphere (20/20). A compensatory head posture was not present. The patient manifested at distance a constant right exotropia of 14 PD with 2 PD right hypotropia and at near right exotropia of 20 PD. In upgaze, there was 10 PD right exotropia and in downgaze, 10 PD right hypotropia. Versions showed moderate superior oblique underaction and inferior oblique overaction in the left eye. With the double Maddox rod, 26 degrees excyclotorsion (18 degrees right eye and 8 degrees left eye) was measured in primary gaze. The bilateral excyclotorsion was confirmed with fundus photography and binocular indirect ophthalmoscopy. Compensating for the strabismus with prisms did not eliminate the diplopia.

Sensory testing with the Worth 4-dot test revealed diplopia at distance and near. Stereopsis was nil with the contour stereotargets of the Randot stereotest. With the synoptophore which also offset the torsional deviation, normal retinal correspondence, sensory fusion with stereopsis, and fusional vergence amplitudes were demonstrated.

The diagnosis was residual cyclovertical strabismus with large excyclotorsion. Since fusion capability was present, additional extraocular muscle surgery was recommended. However, the patient decided against surgery at this time and used a 0.2 density Bangerter filter that resolved the diplopia over his right spectacle lens.
Comment

Diplopia accompanied by large amounts of torsion can mimic intractable diplopia. That fusion could be demonstrated with the synoptophore implies a favorable prognosis once torsion is reduced or eliminated and ocular alignment is achieved with extraocular muscle surgery.63

Accommodative Disorders

Patients with strabismus may have poor control of accommodation and not accommodate as well or as steadily as patients without strabismus. Accommodative insufficiency and accommodative spasm have been reported with both exotropia and esotropia.25,64-70

Exotropia occurring with accommodative insufficiency can be either primary and the accommodative insufficiency secondary or vice versa.64 When exotropia is primary, the increased vergence demand stimulates the accommodative system to produce additional accommodative convergence to compensate for the exotropia and maintain fusion. The accommodative function can become chronically fatigued, leading to accommodative insufficiency. When accommodative insufficiency is primary, the poor focus of the target and a low accommodative vergence level can cause the vergence function to become fatigued, leading to exotropia.

A series of adolescent and young adults having exotropia and severely reduced accommodation was reported by Rutstein and Daum.64 Symptoms included long-standing diplopia and blurred vision that had not responded to either to vision therapy, prisms, or extraocular muscle surgery. Orthophoria or small exophoria was present at distance and intermittent exotropia ranging from 5 PD to 20 PD was present at near. Clinical testing indicated that the patients had markedly reduced amplitudes of accommodation and difficulty sustaining accommodation. Accommodative response determined with dynamic retinoscopy (monocular estimate method) showed a large and varying accommodative lag relative to the accommodative demand indicating inability to sustain accommodation for any period of time. This involuntary fluctuation of accommodation as determined with dynamic retinoscopy appeared to be related to the presence or absence of the exotropia. With accommodation sustained, there was ocular alignment as revealed by cover test and single, clear vision whereas with accommodation released, there was exotropia, diplopia, and blurred vision. Rutstein and Daum concluded that the defective accommodation was a contributing cause for the exotropia and persistent diplopia. Treatment with base-in prism and bifocal glasses resolved the chronic visual symptoms for most patients.

Some investigators have attributed the poor accommodation as being secondary to the exotropia, it being due to under accommodation resulting from loss of convergence.70-72 Horwood and Riddell, for example, reported that during decompensation of distance intermittent exotropia, accommodation normally driven by disparity cues becomes reduced as the drive from convergence is extinguished.70-72 Regardless of the exact mechanism, the resulting diplopia can be persistent if the accommodative disorder is not addressed.

Case 10

A 27 year-old man was referred for long-standing horizontal diplopia and blurred vision which were more bothersome at near. He wore glasses for a small refractive error. He denied a history of strabismus, head trauma, systemic illness, or taking any medications. Prism glasses did not alleviate the diplopia.

Examination revealed refractive error and visual acuities for the right eye +0.75 – 0.75 x 110 (20/24) and the left eye +0.50 – 0.50 x 91 (20/25) Cover testing showed poorly controlled intermittent and alternating exotropia of 6 PD at distance and 14 PD at near. Versions and pupillary testing were normal. Near point of convergence was 50 cm. With the Worth 4-dot
test, fusion was present at distance and crossed diplopia at near. Stereopsis with the contour stereotargets of the Randot stereotest was 140 arc seconds.

The possible source of the exotropia, diplopia, and blurred vision became evident when evaluating accommodation. His amplitude of accommodation was less than 1 D in each eye. Dynamic retinoscopy showed a variable and large underaccommodative response (accommodative lag ~ 2.25 D) in each eye.

The diagnosis was intermittent exotropia with severe accommodative insufficiency. The minimum prism (6 PD base – in) and plus lens add (+1.75 D) that provided single and clear vision combined with the refractive correction was prescribed.

Two months later, he reported absence of diplopia and clear vision. Exotropia could not be elicited with the new glasses. Home based vision therapy utilizing vergence and accommodation procedures were given. The goal was to gradually taper and possibly eliminate the prism and bifocals.

Seven months following his initial visit, the patient reported absence of diplopia and clear vision. Exotropia could not be elicited with the new glasses. Home based vision therapy utilizing vergence and accommodation procedures were given. The goal was to gradually taper and possibly eliminate the prism and bifocals.

Comment

Clinical notes from the referring practitioner made no mention of any accommodative disorder. It cannot be assumed that patients with intermittent exotropia or any type of strabismus accommodate as well or as steadily as non-strabismic patients.70

Spasm of the near reflex or convergence spasm is a rare syndrome that can either mimic or occur simultaneously with esotropia.73-76 It usually consists of intermittent and variable episodes of sustained maximal convergence, accommodative spasm, and pupillary miosis.77,78 Ocular symptoms include diplopia, blurred vision, ocular pain, photophobia, or eye strain. Nonspecific symptoms such as headache, nausea, and dizziness may also occur. Dynamic retinoscopy shows a large and unstable over accommodative response or lead of accommodation.75,77 Limited abduction, unilateral or bilateral, can accompany the episodes.79 Mostly functional in etiology and self-limiting, organic causes have also been reported.80

Spasm of the near reflex is more frequent in children, adolescents, and young adults and is a differential diagnosis for other acquired esotropias, both comitant and incomitant.74,76,78,79 Treatment usually includes inhibiting the accommodative and convergence spasm using cycloplegic agents combined with bifocal glasses. For persistent cases, botulinum toxin has had some success.81,82

When occurring in older patients, convergence spasm and pupillary miosis can occur without accommodative spasm. The accompanying diplopia can be persistent and diagnosed as being intractable.

Case 1183

A 65 year-old man was referred for long-standing horizontal diplopia. When he was 19 years old, he suffered an injury to his right cornea that required pressure patching. He reported that the patch was applied for 3 months. Following its removal, his right eye turned inward and he had experienced diplopia ever since. He had been examined annually the past 10 years in the primary care clinic at
our institution and esotropia with diplopia had always been documented. Since prism glasses had not helped, he was diagnosed with intractable diplopia and placed on medical disability. Ocular history also included myopic astigmatism, bilateral vitreous detachment, and bilateral cataracts. There had not been any orthoptics/vision therapy or extraocular muscle surgery.

Examination revealed refractive error and visual acuities for the right eye -8.00 – 1.75 x 75 (20/40) and left eye -6.00 – 1.50 x 95 (20/40). The patient manifested a fluctuating and difficult to measure right esotropia of approximately 25 PD at both distance and near. The variability of the strabismus appeared to be due to a superimposed convergence spasm. With versions, a marked abduction deficit occurred bilaterally accompanied by pupillary miosis. With duction eye movements, abduction in each eye became full and the pupils were not miotic.

Despite reporting diplopia, the patient suppressed the right eye with the Worth 4-dot test at distance and near. Stereopsis with the contour stereotargets of the Randot stereotest was nil. With the synoptophore, normal retinal correspondence, sensory fusion with stereopsis, and fusional vergence amplitudes were demonstrated.

One month later, the patient also reported experiencing occasional superimposition of two dissimilar images, suggesting possible visual confusion. Corrected visual acuities were 20/30 in each eye. A variable right esotropia of 25 PD with an apparent superimposed convergence spasm continued to be measured. Sensory testing was consistent with the previous visit.

Because of the variable esotropia, pupillary miosis, and limited abduction which improved with duction eye movements, the diagnosis was diplopia due to esotropia and convergence spasm. The patient was referred for strabismus surgery which included a 7.5 mm recession of the right medial rectus. Postoperative visits revealed visual acuity of 20/30 in each eye, absence of diplopia, orthophoria at distance and near, normal pupillary size, full ocular motility, and 100 arc seconds with the contour stereotargets of the Randot stereotest.

Comment
It was speculated that the convergence spasm increased the magnitude of the esotropia and thus the separation between the diplopic images, allowing the patient to occasionally ignore rather than suppress the second image as revealed with the Worth 4-dot test. Eliminating the esotropia eliminated the need for the convergence spasm.

Retinal Disorders/Surgery
Diplopia can occur secondary to retinal disorders such as epiretinal membrane, retinal wrinkling, vitreomacular traction, macular hole, macular edema, subretinal neovascularization, macula-off retinal detachment, and surgery that involves the macula. Its cause is attributed to mechanical distortion and displacement of the macula from its normal centration relative to the peripheral retina leading to rivalry between central and peripheral fusional mechanisms. The ocular alignment necessary to superimpose corresponding points in both foveae is different from the alignment that superimposes corresponding points in the peripheral retina of each eye. The patient’s peripheral fusion tries to help align the anatomically misaligned central macular points preventing central fusion, resulting in diplopia. The diplopia is frequently intermittent and tends to improve in dim illumination. Cover testing usually reveals a small-angle comitant vertical strabismus. Cyclotorsion may also occur with absence of any cyclovertical muscle dysfunction. Often visual acuity is not greatly affected. Aniseikonia and metamorphopsia frequently contribute to the diplopia and make it more difficult to treat.
other eye. The image size disparity, referred to as anatomic or retinally induced aniseikonia, results from forces causing either stretching or compression of the retinal photoreceptors, the former producing micropsia (perceiving a smaller image) and the latter macropsia (perceiving a larger image). As this disruption of the photoreceptors is typically not uniform across the retina, the amount of aniseikonia is heterogenous and field dependent. It can be larger or smaller in one meridian than the other. Methods used to treat optically induced aniseikonia due to anisometropia such as iseikonic lenses, contact lenses, or contact lens/spectacle lens combinations (Galilean telescopes) may be less helpful for aniseikonia that is retinally induced. Anectodotal case reports have reported some success.

Treatment with prisms alone for the small-comitant vertical strabismus should be attempted initially. Although rarely successful, if compensating for the deviation provides stable fusion, prisms can be prescribed.

Case 12

A 66 year-old woman was referred for diplopia. Its onset was 3 months ago and coincident with retinal detachment surgery in her right eye. The diplopia was vertical in direction, more troublesome at distance, and absent when either eye was occluded. She also reported the image in her right eye being approximately 20% smaller than the image in her left eye. She presently used “over the counter” reading glasses. The patient denied having strabismus or any other binocular vision disorder as a child.

Habitual visual acuities were 20/40 in each eye. With refraction right eye -0.75 - 0.25 x 35 and left eye +1.50 - 0.75 x 100, visual acuities improved to 20/30 and 20/20 for the right and left eyes, respectively. Amsler grid testing revealed slight metamorphopsia along the horizontal meridian in the right eye.

The patient manifested a constant right hypertropia of 8 PD at distance and 4 PD at near. The hypertropia was similar in amount for right and left gaze and with forced right head tilt and forced left head tilt. Versions revealed full ocular motility in all positions of gaze. With the double Maddox rod test, 5 degrees right exyclotorsion was measured.

The Worth 4-dot test indicated vertical diplopia at distance and fusion at near. Stereopsis was nil with the contour stereotargets of the Randot stereotest.

Aniseikonia measured with the New Aniseikonia Test (Handaya Co., Tokyo, Japan) showed 10% image disparity between the eyes, the right eye requiring the magnification. Despite the aniseikonia, metamorphopsia, and torsion, prism 7 PD base-down right eye combined with the refractive correction produced stable fusion and was prescribed.

More frequently, diplopia induced by retinal disorders or surgery can be intractable and requires occlusion or fogging of the nondominant eye. Bangerter filters alone or combined with Fresnel prisms have been used with intractable diplopia due to maculopathy.

Case 13

A 78 year-old diabetic man complained of long-standing vertical diplopia and image size disparity. The image seen by his right eye was described as being much smaller than that of his left eye. Ocular history included bilateral pseudophakia, bilateral pars plana vitrectomy, bilateral macular edema worse in the left eye, and bilateral epiretinal membrane peel. The visual symptoms commenced with the retinal disorders. Prism glasses had been unsuccessful. The patient was presently using “over the counter” reading glasses.

On examination, unaided visual acuities were 20/30 and 20/25 for the right and left eyes, respectively. With the Amsler grid, metamorphopsia without scotoma was present in both eyes, the right eye more than the left eye. Cover test showed right exotropia of 6 PD with right hypotropia of 2 PD at distance and 8 PD right exotropia with 2 PD right hypotropia.
at near. The vertical deviation was confirmed subjectively with the Maddox rod. Versions were full in all positions of gaze. With the double Maddox rod, 5 degrees right excyclotorsion was measured. Sensory testing with the Worth 4-dot test showed diplopia at distance and near. Stereopsis with the contour stereotargets of the Randot stereotest was nil.

With the New Aniseikonia Test, 3.5% aniseikonia was present, the right eye requiring the magnification. Placing a 3.5% size lens before his right eye provided more visual comfort.

The diagnosis was retinally induced aniseikonia and small-angle strabismus. Since the patient used “over the counter” reading glasses and was nearly emmetropic, prescribing iseikonic lenses to minimize aniseikonia was not feasible. Applying 2 PD base-up Fresnel prism before the right eye did not eliminate the diplopia. Adding a low density Bangerter filter (0.6) with the prism provided single vision.86 The patient was bothered by the blur with the filter and after two months used only the prism which did not provide single vision.

The patient had been examined 4 years earlier, prior to any cataract and retinal surgery. His refractive error and best visual acuities at that time were right eye -1.00 - 0.75 x 107 (20/40) and left eye -2.25 - 0.75 x 105 (20/50). He reported vertical diplopia and the two images being unequal in size. Orthophoria and 7 PD exophoria were present at distance and near, respectively. A 4 degree right excyclotorsion was measured with the double Maddox rod. Findings with the Worth 4- dot test and stereotesting were identical to the later visit. Interestingly, the aniseikonia measured 11% in the vertical meridian and 9% in the horizontal meridian, the left eye rather than the right eye requiring the magnification.

Comment
Both patients had diplopia and aniseikonia secondary to retinal disease or surgery. Case 12 surprisingly did well with vertical prism despite the large aniseikonia whereas Case 13 with less aniseikonia could not achieve fusion with either size lenses or prisms and was diagnosed with intractable diplopia.

CONCLUSION
Although intractable diplopia can develop in visually mature patients having either childhood or adult onset strabismus, it occurs rarely and therefore should be considered a diagnosis of exclusion. Being diagnosed with intractable diplopia can cause significant stress and has substantial quality of life implications. In this case series, one patient (Case 11) had been placed on medical disability for many years, another patient (Case 5) used an occlusive contact lens while serving as a police officer, and another patient (Case 6) could not perform suturing procedures as a physician’s assistant. All three patients had long-standing diplopia that had been resistant to earlier treatment and demonstrated fusion capability on examination.

Gruzensky and Palmer’s paper in 1988 categorized complicated cases of diplopia into two types, absolute and resistant.3 Absolute diplopia is synonymous with intractable diplopia as presently defined and implies constant diplopia in all positions of gaze and fixation distances that cannot be eliminated with prisms or other types of treatment. Occlusion or fogging of the nondominant eye is usually required. Resistant diplopia includes patients who sometimes may seem to have intractable diplopia but are found on examination to be able to obtain single vision with treatment that either establishes fusion or reactivates a preexisting suppression and/or anomalous retinal correspondence. The other types of diplopia reviewed in the present manuscript fit the resistant diplopia category described by Gruzensky and Palmer.3

Summarizing, the diverse causes of diplopia in this case series emphasize the fact that when examining an adult with persistent diplopia that is possibly intractable, these mechanisms for
diplopia should be considered first. Determining the exact cause of diplopia requires a careful case history, thorough examination, and appropriate use of specialized testing. Many of these patients can be spared of either occluding or fogging their nondominant eye and should have access to other forms of treatment.

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Acknowledgement

The author thanks Dr. John Amos, Dr. Richard London, and Dr. Lanning Kline for critiquing the manuscript.

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