Diplopia-Visual Confusion

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Abstract

Diplopia –double vision. It is defined as perception of two images of a single object. In fact diplopia is derived from Greek words diplous and ops, meaning "double" and "eye" respectively. Diplopia may binocular or monocular when it persists in affected eye despite covering the other eye.

Introduction

Monocular diplopia usually do not need to undergo neurological evaluation since a careful eye evaluation itself reveals the cause **.**A large chalazion or other eyelid tumor may produce enough distortion of cornea to cause monocular diplopia[1] which can be evaluated on simple torch light examination. A careful refraction including retinoscopy may reveal irregular corneal or lenticular astigmatism[2].Corneal topography can further reveal an early keratoconus or abnormal corneal contour.Large iridectomy resulting in polycoria can lead to formation of second image. Tilting of lens with or without subluxation can cause double vision especially when the edge if subluxated lens lie within visual axis. Formation of prominent cleft within the crystalline lens or vacuole in posterior sub capsular area[3]can also lead to diplopia.Viterous opacities sometimes present with double vision. Slit lamp bio microscopy can reveal central serous choroidopathy or macular edema due to any cause which causes distortion of amsler grid and can be confirmed on optical coherence tomography .Pituitary tumor that produces large scotoma bisecting fixation[4] which can be demonstrated on visual field charting.
Binocular diplopia indicates breakdown of fissional capacity of the binocular system. Binocular diplopia is usually associated with ocular deviations which can be same in all gaze positions (comitant) or vary with different gaze positions (incomitant).Comitant deviations do not have diplopia. Various causes of ocular deviation include extraocular muscle weakness like congenital myopathies, mitochondrial myopathies, muscular dystrophy. Extra ocular muscle restriction like muscle entrapment, thyroid associated ophthalmopathy, extraocular muscle injury or hematoma due to ocular surgery can lead to double vision. Orbital disorder like trauma, tumor, thyroid associated ophthalmopathy, infection causes diplopia. Myasthenia gravis, botulinum injection cause neuromuscular junction dysfunction resulting in diplopia. Palsies of 3rd, 4th or 6th cranial nerve can result in ocular deviation and diplopia .These can be due to ischemia, trauma, hemorrhage, aneurysm, tumor, vascular malformation, multiple sclerosis. Supranuclear pathway injury[5] secondary to hydrocephalus, neurosyphilis, Wernicke's encephalopathy, neurodegenerative disease, trauma, hemorrhage, and tumor is one of the causes of diplopia

1. History- taking

Careful description of the diplopic images can help differentiate monocular or

Refractive diplopia from a problem of the ocular motor system, the former often being described as a 'ghosting' rather than a true diplopia.

- Is it horizontal, vertical, oblique or torsional diplopia?
- Is it worse (or better) with changes in gaze or head position (comitancy)?
- Is it worse for distance or near viewing?
- Is there any variability or fatigability?
- Is the onset acute or gradual?
- Are there any associations, particularly a history of strabismus or amblyopia, loss of vision, ptosis, pain or systemic health problems (e.g., diabetes or hypertension)?

3. Monocular Tests

Monocular diplopia should always be excluded before attempting to further classify the diplopia. Monocular occlusion is used to rule out monocular diplopia .After occlusion diplopia may be present in each eye (e.g. cortical diplopia) or in affected eye only. Visual acuity with and without pin-hole is required to rule out
Refractive monocular diplopia which normally disappear through a pinhole.
Amsler chart may be used to demonstrate metamorphopsia which indicate associated macula pathology.

4. Cover test

The cover tests to be done, at least initially, using an accommodative target for fixation. Binocular diplopia is normally due to misalignment of the visual axes, a movement of the deviating eye to take up fixation can be seen on occlusion of the fixating eye. Long-standing deviations usually have intermittent suppression, thus cover test can reveal a misalignment of the visual axes even when there is no subjective report of diplopia. Fixation may alternate in recent onset deviations, observation of the movement as the eyes swap fixation can be sufficient to determine the direction of the deviation without the use of a cover .The direction of eye movement and subjective descriptions of the images can be used to determine whether the diplopia is horizontal (crossed or uncrossed), vertical or torsional.
Where there is an incomitancy, the angle of deviation normally differs depending on the fixating eye, with the secondary deviation being larger than the primary.

4. Anomalous head postures

Observe the patient's head, chin and face as many forms of diplopia are relieved by compensatory head postures .AHP occurs as patient consciously wanting to avoid a gaze position that relies upon contraction of paretic muscle, an AHP could also put the eyes into a position that will bring the visual axis of one eye into a suppression scotoma. It separates the images to minimize the effect of confusion. It moves the eyes out of the direction of a mechanical limitation of eye movement. It also improves visual acuity in patients with ptosis or nystagmus.With horizontal muscle weakness patient tends to turn his face toward weakened horizontal rectus muscle to relieve diplopia.Underaction of the superior and inferior rectus muscle, which primarily move the the eyes in the vertical plane is compensated by a chin flexion or extension, which reflexly evokes a vertical change in gaze. Torsional diplopia is

almost always caused by underaction of superior or inferior oblique muscle at least in primary gaze and is lessened by angular tilt of the head towards the shoulder

5. Maddox double rod test[6]

Maddox rods at the same orientation in front of each eye (normally vertically orientated to produce a horizontal streak) can be used to assess the angle of torsion.
If the streaks seen by each eye are not parallel, then the rods can be rotated until the streaks become parallel and horizontal, thus giving a measure of the rotation required and hence the torsion. This test is maximally dissociating and can produce erroneous results (possibly as a result of small angles of head tilt).

6. Bagolini lenses

It can be used in the same way, but this method tends to underestimate the angle of torsion, due to fusion.

7.Forced duction test[8]

The forced duction test can be used to differentiate whether limitation of extra ocular movement is related to paresis of agonist or tethering of antagonist extraocular muscle.

8. Diplopia Chart

A vertical bar of light is viewed through red and green goggles at a fixed distance from the eye. The bar light is moved into each direction of gaze, and the patient describes the image separation and appearance. The image separation can be measured. By convention, the red filter is always placed before the right eye. It should be remembered that the most distal image belongs to the under-acting eye. The position of the image is the reverse of the position of the eye. So it is important to mark the patient's right and left on the chart.

9. Field of binocular single vision (BSV)

The field of BSV is a test used to describe the areas of BSV, and hence diplopia. It can be done using a kinetic perimeter, or approximated from ocular motility. The patient sits at the perimeter, with the chin central to fixation. The target is moved outwards until the patient recognizes diplopia, and the point is marked. The target is then moved further until one image disappears, this point is again marked. The inner ring describes the area of BSV; the outer ring describes the limits of the binocular field of fixation.

10. Hess chart

The patient is seated squarely facing the screen being plotted, with head centered on the central fixation spot. The center position is plotted first, and then the 15° fixation points, and finally as many of the 30° points as can be plotted without moving the head. On interpretation of Hess plot the smaller field belongs to the eye with the defect. Neurogenic parses shows the muscle sequelae to a greater or lesser extent

(dependent on the duration of the condition and the eye used for fixation). The largest under action is normally in the direction of action of the paretic muscle and the largest over-action is normally the contralateral synergist. Mechanical defects show a compressed field. There is usually no obvious over-action of the direct antagonist, nor under-action of the contralateral antagonist, so the effects of the defect are limited to the direction of action of the mechanical restriction. The most obvious feature of a mechanical defect is normally the marked by over action of the contralateral synergist.

11.Treatment

The management of diplopia is individualized according to its specific cause; each etiology has its own natural history and associated morbidity[9]. A careful refraction and fitting of a contact lens will eliminate approximately 60% of all cases of monocular diplopia[10].The goal is to try to restore binocular vision if possible. This can be done by various means, such as

11.1 Paste-on Fresnel or ground-in prism

In clinical practice, prismatic power is usually split equally between the two eyes If vertical prism is to be ground into one spectacle lens only, a base-up orientation is preferable because distortion is greater towards the base and will also result in reflections from overhead lights if placed base down[11]. Fresnel prisms have the advantage of conforming and adhering to the spectacle lens surface through surface tension[11].
Other advantages of Fresnel prisms include modest cost, negligible thickness and weight, and wide range of power (up to 30Δ).

11.2 Optometric vision therapy

Vision therapy exercises are directed at improving ocular motility**.** One can change the neurophysiological vergence-control mechanism through various visual stimulations[12].Optometric vision therapy can help patients reduce the amount of prism required.

11.3 Botox chemo denervation.

It helps in preventing secondary contracture. It can be injected in to the antagonist in recent onset palsy. Fusion in these cases results from relaxation of antagonist muscle and neural recovery from agonist.
Often, a combination of treatments is used. If such measures are unsuccessful, patients may be advised to occlude the poorer Seeing Eye, preferably with translucent tape on their glasses. As is the case with most ischemic ocular motor palsies, the translucent monocular occlusion is only required temporarily, until the diplopia spontaneously resolves.

11.4 Strabismus surgery for diplopia

Any recent onset strabismus is allowed to stabilize for a minimum of 6 months up to a year. Typical recession or resection is rarely indicated. Transposition procedures can be done to treat diplopia in primary gaze.

12. References

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