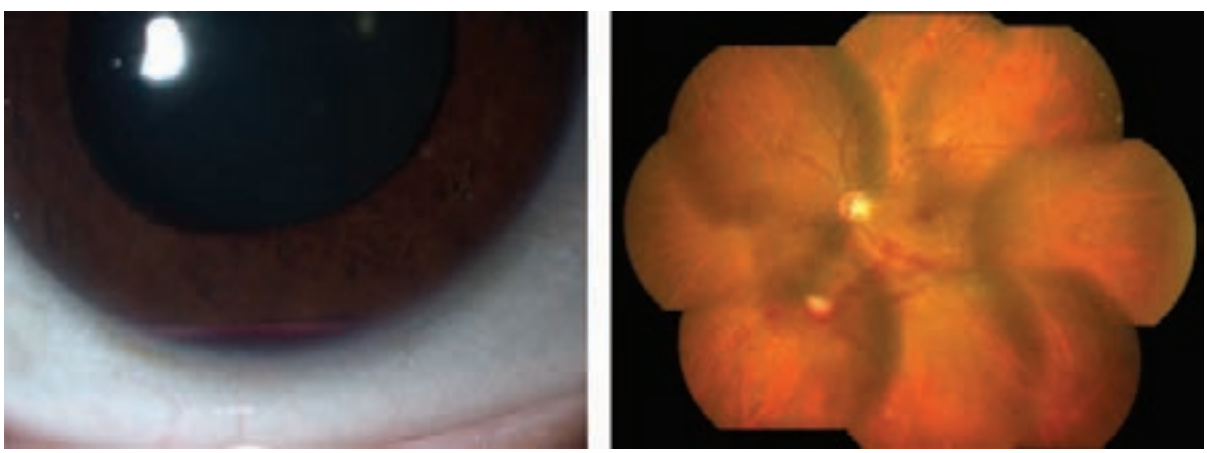
insight

Vol. XXX No. 2 JUNE 2012

Scientific Journal of
MEDICAL & VISION RESEARCH FOUNDATIONS
18, COLLEGE ROAD, CHENNAI - 600 006, INDIA



Editorial

Dear Readers

This issue has articles covering the practical aspects on the prevention of oculocardiac reflex, step wise evaluation of blepharoptosis and a brief overview on is this pupil normal? There is an interesting case report on the presence of proliferative retinopathy as the first sign of chronic myeloid leukemia. Interesting facts on the artifacts in macular optical coherence tomography and effectiveness of vision therapy in the treatment of the symptomatic convergence insufficiency are covered.

Enjoy reading.

Dr Shubhra Goel
Editor, Insight

Oculocardiac Reflex: Prevention of OCR Treatment

V. V. Jaichandran

Consultant Anaesthesiologist, Sankara Nethralaya, Chennai, India

HISTORY

The oculocardia reflex (OCR) is a physiological response of the heart to physical stimulation of the eye or the ocular adnexa, characterized by bradycardia or arrthymia, which sometimes leads to cardiac arrest. It was in 1908, both Bernard Aschner and Guiseppe Dagnini independently reported cardiac slowing after pressure on the eyeballs. Aschner demonstrated by animal investigations that the afferent impulse is carried via the ophthalmic division of the trigeminal nerve to the vagal centres, from where the impulses are relayed to heart via the vagus nerve.

The importance of OCR was brought to light when Sorenson and Gilmore in 1956 reported that traction on the medial rectus muscle caused cardiac arrest in a patient. This reflex is especially sensitive in neonates and children, as their vagal tone is higher. However, it is also reported to occur in adults. The overall incidence of OCR, both reflex bradycardia and dysrhythmias, was higher with general anaesthesia compared with regional anaesthesia.

Positive OCR is defined as dysrhythmia or bradycardia of 10% or more below relative baseline resulting from traction of an extraocular muscle. Vrabec et al. and Eustis et al. defined it as a 10% decrease, while Karhunen et al. defined it as a 20% decrease in the baseline heart. Mirakhur et al. have taken bradycardia of 70 or less heart beats per minute at any time during surgery and persisting for more than 15 seconds as study criteria and OCR as slowing of the heart rate by more than 20% or arrhythmia during traction irrespective of the heart rate.

MECHANISM

The reflex is mediated by nerve connections between the "trigeminal cranial nerve" and the "vagus nerve" of the "parasympathetic nervous system". The afferent tracts are derived mainly from the ophthalmic division of the trigeminal nerve. These afferents synapse with the "visceral motor nucleus" of the vagus nerve, located in the "reticular formation" of the brain stem. The efferent portion is carried by the vagus nerve from the "cardiovascular centre" of the "medulla" to the heart, of which increased stimulation leads to a decreased output of the "sinoatrial node" (Figure 1).

This reflex sets in due to stretching of the extraocular muscle or increase in the eye ball pressure. In clinical practice, this OCR is most often encountered during squint surgery in the paediatric age group. It can also occur in repair of detached retina, especially during tagging of extraocular muscles, encucleation of eye, intraorbital injection of local anaesthetics, following digital pressure to the eye, pinching of the conjunctiva with a forceps

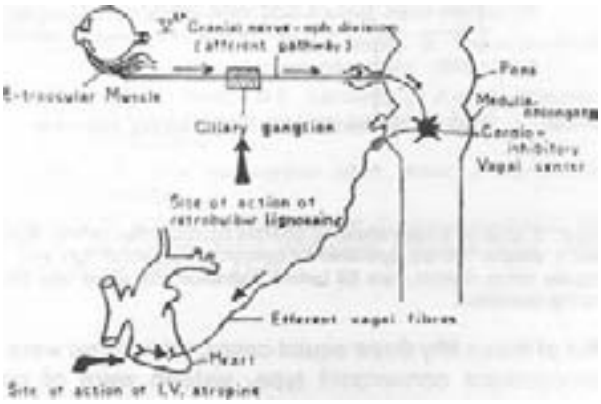


Figure 1. The afferent and effernt pathway of OCR. The site of action of drugs (i.e. atroprine and xyloaine) is indicated with arrows.

and postoperative pressure of the bandage. The reflex is also evoked by stimulation of eye lids (blepharocardiac reflex), face and oral cavity.

The various factors that can increase the risk of OCR reviewed by Scott Lang and van der Val are hypercarbia, hypoxaemia, hypoventilation, acidosis, light anaesthesia, young age due to higher resting vagal tone, pharmacological agents such as potent narcotics (sufentanil, alfentanil), β blockers and calcium channel blockers, and the nature of the provoking stimulus—namely, strength of stimulus—and duration.[1] Though retrobulbar anaesthetic infiltration is a method of prophylaxis to prevent OCR, yet still before the block has taken full effect, the reflex may be induced by stimulus of needle disturbance, retrobulbar bleeding or even the anaesthetic solution (2.5 ml) which probably has effects on intraorbital pressure within a short time. Ocular compression following regional block was found to be the most common triggering event in precipitating the OCR.

CARDIAC EFFECTS

OCR may manifest as bradycardia, bigeminy, ectopic beats, junctional (nodal) rhythm with disappearance, inversion or shift of the P wave on the ECG, AV block and cardiac arrest. Initial baseline baseline heart rate has no influence on the incidence of OCR: tachycardia is therefore not protective. Ohashi suggested that the depth and occurrence of bradycardia in the OCR were closely related to the strength of tension, and that the responses of extraocular muscles to stretch were quantitatively transmitted to the heart and then suppressed the heart rate. He also showed that there

were differences of depth and threshold of the reflex as well as the incidence among the medial rectus, the inferior oblique and the lateral rectus muscle. Stretch on the medial rectus and the inferior oblique evoked the reflex in all the patients tested, whereas, in the lateral rectus, the reflex could be induced only in about 50% of patients. The discrepancy in the heart rate sensitivity between surgical extraocular muscle tension and ocular compression might be due to different sensory receptors and brain stem processing for the trigeminally mediated oculocardiac reflex.

VAGAL ESCAPE

There is a tendency for the patient to adapt to the vagal tone with the traction of the extraocular muscle with the heart rate returning to the pretraction rate. The OCR normally fatigues with repetitive stimuli and thus lesser decrease in the heart rate is observed when the second muscle is stimulated compared to the first muscle traction. This was pointed out by Platen (1958) and Moonie (1964).

ASSOCIATION BETWEEN OCR AND POSTOPERATIVE NAUSEA AND VOMITING (PONV) IN STRABISMUS SURGERY

PONV is particularly common after strabismus surgery in children. In addition to the distress it causes both in child and parents, it is also the most frequent cause for unplanned admission after day-case surgery. Although many factors may influence the occurrence of PONV, its particular propensity for occurring after strabismus surgery has led to the theory that an oculo-emetic reflex is responsible. This has been described as a reflex resetting of the vomiting centre in the medulla following stimulation of the ophthalmic division of the trigeminal nerve during extraocular muscle manipulation. Allen et al. found that there was a significant association between a positive intraoperative OCR and PONV. Children with a positive OCR were 2.6 times more likely to vomit than those without the reflex.

Prevention of OCR

To avoid any untoward effect of OCR, it is recommended to monitor ECG, pulse oximetry, provide anticholinergic protection, encourage surgeon to be gentle and ensure an adequate depth of anaesthesia during the surgery. A quick traction on extraocular muscle provoked a reflex in 87% of instances, whereas progressive traction did so in only 51% of instances. Thus, to reduce the incidence of OCR occurring during strabismus surgery, minimal and gentle manipulation of the extraocular muscles must always be employed be the surgeons. The regional eye block (peribulbar or retrobulbar) can prophylactically block the afferent limb of the reflex, while intravenous injection of an "anti-muscarinic acetylcholine" (ACh) "antagonist", such as "atropine" or glycopyrrolate can prophylactically block or attenuate the efferent limb of the reflex. Topical lignocaine applied to eye muscles also significantly attenuates the OCR. Grover et al. have shown that local anaesthesia produces less bradycardia and ectopic arrhythmia and is better than general anaesthesia for surgeries in which traction of extraocular muscle is required.

In a control double-blind study, Kundra demonstrated that combining peribulbar block with general anaesthesia in children undergoing intraocular surgery completely abolished OCR, reduced the requirement of anaesthetics and produced early recovery from anaesthesia with satisfactory postoperative analgesia.

Treatment

If bradycardia does occur, the removal of the inciting stimulus is immediately indicated, and is essential for successful termination of this reflex. The surgeon operating on the eye should be asked to cease their activity and release the applied pressure or traction on the eyeball. This often results in the restoration of normal sinus rhythm of the heart. If not, the use of atropine or glycopyrrolate will likely successfully treat the patient and permit continuation of the surgical procedure.

CONCLUSION

Prevention of OCR requires awareness of the anatomical structures involved, cooperation between the surgeon and anaesthesiologists and knowledge of the factors known to favour or evoke these reflexes.

REFERENCES

1. Lang S, Lanigan D, van der Wal M. Trigeminocardiac reflexes: maxillary and mandibular variants of the oculocardiac reflex. Can J Anaesth 1991;38:757–60.
2. Paton J, Boscan P, Pickering A, et al. The yin and yang of cardiac autonomic control: vago-sympathetic interactions revisited. Brain Res Brain Res Rev 2005;49:555–65.
3. Kim H, Kim S, Kim C, et al. Prediction of the oculocardiac reflex from pre-operative linear and nonlinear heart rate dynamics in children. Anaesthesia 2000;55:847–52.
4. Smith R. Death and the oculocardiac reflex. Can J Anaesth 1994;41:760.
5. Ashner B. Concerning a hitherto not yet described reflex from the eye on circulation and respiration. Wien Klin Woschanschr 1908;21:1529–30.
6. Apt L, Isenberg S, Gaffney WL. The oculocardiac reflex in strabismus surgery. Am J Ophthalmol 1973;76:533–35.
7. Dauchot P, Gravenstein JS. Effects of atropine on the electrocardiogram in different age groups. Clin Pharmacol Ther 1971; 12:274–80.
8. Kirsch RE, Samet P, Kugel V, et al. Electrocardiographic changes during ocular surgery and their prevention by retrobulbar injection. Arch Ophthalmol 1957;58: 348–56.
9. Bosomworth PP, Ziegler CH, Jacoby S. The oculocardiac reflex in eye muscle surgery. Anaesthesiol 1958;19:7–8.
10. Mendelblatt FI, Kirsch RE, Lemberg L. A study comparing methods of preventing the oculocardiac reflex. Am J Ophthalmol 1962;53:506–8.
11. Berler DK. The oculocardiac reflex. Am J Ophthalmol 1963;56: 954–9.

Bilateral Visual Impairment Due to Proliferative Retinopathy as a First Sign of Chronic Myeloid Leukemia in an Adult

Mamta Agarwal[1], Aditya Verma[2] and Jyotirmay Biswas[3]

[1]Consultant, Dept. of Uvea & cornea services, MRF, Sankara Nethralaya, Chennai

[2]Ass. Consultant, Dept. of Vitreoretinal Services, MRF, Sankara Nethralaya, Chennai

[3]Director, Dept. of Uvea, MRF, Sankara Nethralaya, Chennai

INTRODUCTION

Leukemic retinopathy was first described by Liebreich in 1860s. It is more common in acute than chronic leukemia. It presents either as nonproliferative retinopathy or hypertensive retinopathy and is seen in 50% of patients with leukemia.[1] However, proliferative retinopathy with neovascularization is rarely seen and is found only in patients with chronic disease. We report an unusual case of bilateral visual impairment due to proliferative retinopathy as a first sign of chronic myeloid leukemia in an adult.

CASE REPORT

A 36-year-old male presented with complaints of pain and blurred vision in the right eye of 15-day duration and in the left eye of 4-day duration. It was associated with headache. There was no history of any systemic illness, although he reported a weight loss of 6 kg in the last 6 months.

Visual acuity was counting fingers at 1 m in the right eye and 6/24 in the left eye. Slit-lamp examination of both eyes revealed anterior uveitis, rubeosis iridis and hyphema (Figure 1). Indentation gonioscopy showed closed angles in the right eye and peripheral anterior synechiae with angle neovascularization in the left eye. Intraocular pressure by applanation tonometry was 20 mmHg in the right eye and 18 mmHg in the left eye. Indirect ophthalmoscopy in the right eye showed vitreous hemorrhage and neovascular frond in the inferior periphery (Figure 2, arrow). Left eye examination revealed tortuous retinal vessels, intraretinal hemorrhages, cotton wool spots, vascular sheathing and dispersed vitreous hemorrhage (Figure 3). Neovascularization of the retina was also seen along the superior and inferior temporal arcades.

The patient was referred to an internist for a systemic evaluation. Physical examination revealed severe hepatosplenomegaly. Routine blood investigations showed decreased RBC count (3.0 million/mm^3), low packed cell volume (28%), leukocytosis (316,000/mm^3) and thrombocytosis (572,000/mm^3). Differential leukocyte count revealed neutrophils 38%, lymphocytes 7%, eosinophils 7%, basophils 5%, monocytes 3%, myelocytes 24% and

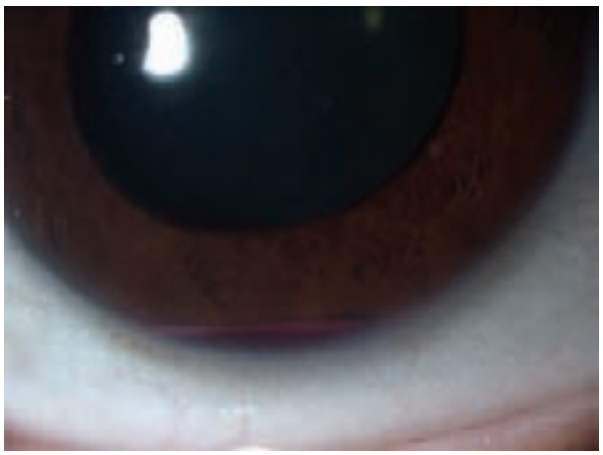


Figure 1. Slit-lamp examination of the right eye showing 360° rubeosis iridis and hyphema.

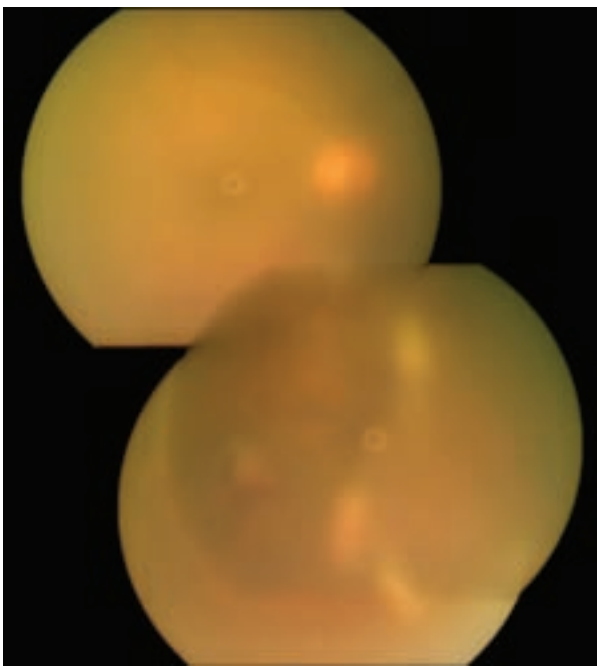


Figure 2. Fundus montage of the right eye showing dispersed vitreous hemorrhage with neovascular frond (arrow).

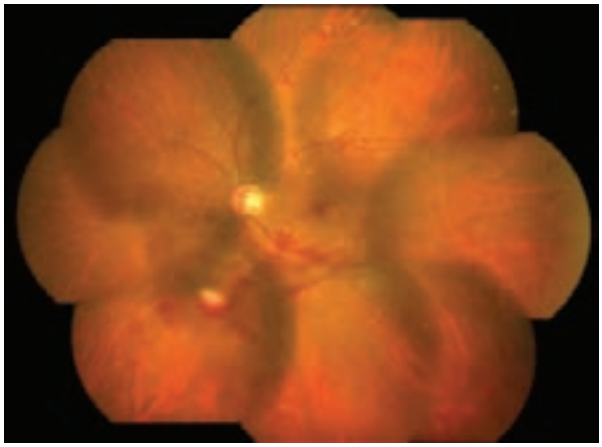


Figure 3. Fundus photoghraph of the left eye showing neovascularization of the retina, superficial hemorrhages, hard exudates over the posterior pole, venous loops along superotemporal arcade and minimal vitreous hemorrhage.

metamyelocytes 12%. Peripheral blood smear showed hyperleucocytosis with myeloid left shift, eosinophilia, basophilia, thrombocytosis, 16% metamyelocytes and 3% myeloblasts. Bone marrow aspiration cytology revealed hypercellular bone marrow with features consistent of chronic myeloid leukemia in the chronic phase. Cytogenetic analysis was positive for the Philadelphia chromosome in all metaphase cells. Patient was then started on imatinib mesylate at a dose of 400 mg orally per day by the oncologist.

He underwent bilateral panretinal photocoagulation and was treated with topical steroids, cycloplegics and antiglaucoma medications. Two months later, the patient was reviewed in our clinic. Visual acuity in both eyes was light perception. Clinical examination revealed advanced neovascular glaucoma in the right eye and vitreous hemorrhage in both eyes. He underwent parsplana vitrectomy with endolaser photocoagulation in the left eye. At the last follow-up after 4 months, visual acuity was light perception in the right eye and 6/60 in the left eye.

DISCUSSION

Chronic myeloid leukemia is a clonal myeloproliferative disorder of the hematopoetic stem cell origin characterized by diffuse replacement of the bone marrow by neoplastic cells. Ocular involvement may be either due to leukemic infiltration of ocular tissues or as a result of secondary complications of the disease like anemia, leukocytosis and thrombocytopenia which can cause hemorrhages and ischemia.

Schachat et al. in their study described leukemic infiltration of ocular structures in 3% of patients. Secondary complications like intraretinal hemorrhages, cottonwool spots, central retinal vein occlusion and white centered retinal hemorrhages were seen in 39% of patients.[1] In another case series by Reddy et al., 10% of patients presented with eye signs at the initial presentation who were later diagnosed to have asymptomatic leukemias.[2] Visual loss as a first sign of adult type of chronic myeloid leukemia in a child has also been recently reported by Rudolph et al.[3]

Our patient also presented with bilateral visual loss due to proliferative retinopathy as a first clinical symptom of chronic myeloid leukemia without any systemic complaints. Proliferative retinopathy has been reported rarely in chronic leukemias.[4,5] Retinal neovascularization secondary to vascular occlusion and capillary drop out occurs due to leukocytosis and thrombocytosis. Intravascular sludging by leukemic cell infiltration also plays a role.[5] Raised intraocular pressure occurs as a result of leukemic infiltration of the trabecular meshwork.

To the best of our knowledge, medline search did not show any report of chronic myeloid leukemia presenting with bilateral vitreous hemorrhage, hyphema and neovascular glaucoma as was seen in our patient. A diagnosis of chronic myeloid leukemia was made in this patient based on clinical suspicion, ophthalmic findings and laboratory investigations. Although the role of an ophthalmologist is secondary in the management of leukemias, early recognition of ophthalmic signs as a presenting feature of a more severe and life-threatening disease makes their role quite important.

REFERENCES

1. Schachat AP, Markowitz JA, Guyer DR. Ophthalmologic manifestations of leukemia. Arch Ophthalmol 1989;107:697–700.
2. Reddy SC, Jackson N, Menon BS. Ocular involvement in leukemia—a study of 288 cases. Ophthalmologica 2003;217:441–5.
3. Rudolph G, Haritoglou C, Schmid I, et al. Visual loss as a first sign of adult-type chronic myeloid leukemia in a child. Am J Ophthalmol 2005;140:750–1.
4. Huynh TH. Johnson MW, Hackel RE. Bilateral proliferative retinopathy in chronic myelogenous leukemia. Retina 2007;27:124–5.
5. Frank RN, Ryan Jr SJ. Peripheral retinal neovascularization with chronic myelogenous leukemia. Arch Ophthalmol 1972;87:585–9.

Step-wise Evaluation of Blepharoptosis

Md Shahid Alam[1] and Shubhra Goel[2]

[1]Senior Fellow, Dept. of Orbit, Oculoplasty, Reconstructive & Aesthetic, Sankara Nethralaya, Chennai
[2]Consultant, Dept. of Orbit, Oculoplasty, Reconstructive & Aesthetic Services, Sankara Nethralaya, Chennai

INTRODUCTION

Ptosis refers to drooping of the eye lid and is sometimes more specifically described as blepharoptosis. It is not only a cosmetic concern but can also lead to obscuration of visual field and sometimes prefrontal headaches secondary to the overuse of the frontalis muscle. Managing a case of ptosis requires a detailed clinical history and examination. This article is an attempt to summarize the important parameters while evaluating a patient with blepharotosis.

HISTORY TAKING

A proper and thorough history taking constitutes the first step in the evaluation. The following points are important while eliciting history:

1. Onset and duration and progress (increasing, decreasing or constant)
2. Diurnal variation
3. Family history
4. Associated history
    a. Diplopia
    b. Weakness in any other body part (limb weakness or difficulty in deglutition)
    c. Trauma including birth trauma in case of congenital ptosis
    d. Previous surgery (ptosis surgery, any other eyelid, orbital or cataract surgery)
    e. Long-term use of steroid drops
    f. Allergies (blepharochalsis)
    g. Chronic use of contact lens

EXAMINATION

In words of Crowell Beard "The preoperative examintation is without doubt the most important step in the treatment of the ptosis patient. If the examination has not provided sufficient information for adequate classification of the ptosis the surgeon should not hesitate to request re examination or to postpone consideration of surgery until a satisfactory examination is possible."

Important parameters are

1. Is it a true ptosis?
2. Age of presentation?
3. Laterality?
4. Important general signs:
    a. Head posture: The patient with ptosis often adopts abnormal head positions, commonly a chin elevation in order to clear the visual axis . An abnormal head tilt may be seen in cases of superior rectus muscle weakness A persistent chin up position to clear the pupillary axis in a child can cause torticolis and is also one of the indications for ptosis surgery.
    b. Facial asymmetry/dysmorphism: Congenital ptosis can be part of the craniofacial syndromes like blepharophimosis, Turner's, Noonan's and others.
    c. Periocular skin: Conditions like blepharochalasis can have the wrinkled tissue paper or classically what is called as the cigarette paper skin. Skin scars within the eyelid or surrounding periorbital and adnexal tissue may suggest a past surgery or trauma that could have caused the ptosis.
5. Opthalmic examination
    a. Visual acuity and refraction: The visual acuity must be documented for every patient. Young children with ptosis must undergo cycloplegic refraction. Ptosis where the visual axis is occluded can lead to amblyopia in children and must be urgently addressed.
    b. Hirschberg/cover test: The presence of squint necessitates the management of squint before ptosis management. Hypotropia can be mistaken for ptosis. Intermittent phoria or tropia can also be an indicator of underlying myasthenia gravis.
    c. Pupillary evaluation: An ipsilateral miotic pupil is seen in Horner's syndrome in contrast to a widely dilated, nonreactive pupil in cranial nerve III palsy. A sluggish and ill-sustained pupillary reaction may be indirect evidence of dense amblyopia.
    d. Bell's phenomenon and corneal sensitivity
    A weak or absent Bell's phenomenon would be a relative contraindication for corrective lid surgery. Poor corneal sensation would also carry a risk of exposure complications following surgery.
    e. Dye eye evaluation
    A detailed dry eye workup, including Schirmer tear secretion or measurement of the tear meniscus height, may be valuable if the history or the examination suggests baseline dry eyes. If the tear

secretion is below normal, the ocular surface will have to be closely monitored post-operatively. In older patients with marked reduction in tears, an undercorrection of the ptosis may well be indicated.

f. Ocular motility
Elevation limitation suggests superior rectus weakness which is commonly seen in congenital ptosis. A generalized limitation in the actions of all extraocular muscles is seen in CPEO. Unusual lid movements associated with ocular motility may suggest aberrant nerve regeneration. In III nerve palsies, abduction is preserved, which helps confirm the diagnosis. Myasthenia gravis should always be considered as it can present with varied ocular motility limitation.

g. Fundus evaluation: Ptosis in progressive external ophthalmoplegia and Kearns–Sayre syndrome has been found to be associated with pigmentary retinopathy.

6. Measurements in a case of ptosis

A detailed examination of the ptotic eyelid would encompass the following signs:

- Marginal reflex distance (MRD)
- Palpebral fissure height and symmetry
- Levator function
- Marginal crease distance
- Downgaze ptosis
- Hering's effect
- Brow position and dermatochalasis
- Frontalis muscle overaction
- Lagophthalmos
- Associated lid signs

a. Palpebral fissure height (PFH): Palpebral fissure height is the vertical distance between the upper and lower lid in the pupillary plane. The normal human corneal diameter is 11 mm and the superior 1–2 mm of the cornea is normally covered by lid, therefore the normal PFH is approximately 9 mm. Normal PFH in primary gaze is 9 mm on average (males: 7–10 mm; females: 8–12 mm) . In downgaze, a PFH of 1 mm or less indicates visually significant complete obstruction of the pupil in reading position.

b. Marginal reflex distance-1 (MRD-1): MRD1 is the distance in millimeters between the central upper lid margin and the corneal light reflex in primary gaze. MRD2 measures the distance between the central lower lid margin and the corneal light reflex. A normal MRD is 4–4.5 mm. Measuring the MRD not only helps quantify the severity of ptosis, but also helps guide the appropriate management plan for each case.

The amount of ptosis is the difference between the MRD1 of two eyes in unilateral cases. The difference between the measured and the normal values gives an indication of the amount of ptosis in bilateral ptosis. An MRD2 that measures the lower lid to rest below the inferior limbus indicates some degree of lower lid retraction which should be considered in the preoperative planning. A retracted lower lid should be horizontally tightened or elevated to a more anatomic position at or just above the inferior limbus when planning surgical elevation of the upper lid in order to avoid postoperative lagophthalmos and exposure.

Quantitatively, on the basis of MRD, ptosis can be classified as

- Mild ptosis 2 mm or less
- Moderate ptosis 3 mm
- Severe ptosis 4 mm or more

c. Margin crease distance (MCD): This measures the distance between the upper lid margin and the eyelid crease. The eyelid crease may help in the diagnosis of some types of ptosis and is an aesthetically crucial landmark for placement of the incision in ptosis surgery. The normal MCD in males is 6–8 mm and 8–12 mm in females. An abnormally elevated or asymmetric MCD should alert the physician to the presence of underlying levator disinsertion and involutional ptosis. In cases where more than one lid crease is present, the most prominent crease should be measured.

d. Levator function: It is assessed by the speed and amount of eyelid excursion by Berke's method. Normal levator function is 15 mm or greater. A difference between the measured and the normal levator function guides the surgical plan, as a decreased levator function would predispose a risk of postoperative lagophthalmos, and therefore a more conservative elevation would be more appropriate. The assessment of levator function in pediatric population is difficult. The presence of lid fold and increase or decrease in its size on the movement of the eyelid gives a clue about levator action. The test allows the assessment of levator function during first year of life. The upper eyelid of child is everted as the child looks down. If the levator action is good, lid reverts back on its own.

CLASSIFICATION OF LEVATOR FUNCTION

Levator function (mm)	Classification
10–15	Excellent (normal)
>8	Good
5–7	Fair
<4	Poor

e. Downgaze ptosis: It is important to note the relative position of the eyelids in downgaze or reading position. In severe involutional ptosis, the eyelids not uncommonly block most of the pupils in downgaze.

f. Hering's effect: In cases of unilateral ptosis or asymmetric ptosis, the presence or absence of Hering's effect masking a bilateral ptosis should be determined. This is done by elevating the more ptotic upper lid manually or with 2.5% phenylephrine topically. A prominent Hering's effect would indicate the need to perform bilateral ptosis correction bilaterally in order to avoid contralateral ptosis postoperatively. The phenylephrine test is also helpful in predicting the result of conjunctival mullerectomy.

g. Associated lid signs

- Lid lag: The ptotic lid remains at a higher level than normal when looking down, due to failure of a fibrotic or maldeveloped levator muscle to relax in downgaze
- Fatigability: Progressive increase in degree of ptosis of upper lid on constant focusing or inability to look upwards without the eyelids gradually falling down.

Ask the patient to look up without blinking at your finger for 30 s to observe whether the elevated lid position can be sustained without dropping.

- Cogan's lid twitch: Overshoot of upper lid on rapid saccade from down to primary gaze. Ask the patient to look at your finger for several seconds in downgaze and then ask him to fixate at an object in primary gaze. Look for the lid overshooting to a higher position before settling into the normal MRD.
- Orbicularis oculi tone: Ask the patient to close the eyes forcefully and try to open them against resistance. In individuals with normal orbicularis tone, the examiner should not be able to open the lids. Ask the patient to close eyes gently. In eyes with weak orbicularis tone, the lids may close very slowly, or the eyes may be seen through the open lids (lagophthalmos).
- See-saw ptosis: Manually lift the ptotic lid and look for droop of the contralateral eyelid. This is due to Hering's law of equal innervation, in which increased innervation to lift the ptotic lid also concurrently elevates the opposite lid leading to its retraction.
- Marcus Gunn jaw-winking: This is a synkinetic movement that occurs due to misdirection of the mandibular branch of the trigeminal nerve to the ipsilateral levator muscle. Clinically, the upper lid elevates on wide opening or sideways movement of the mouth. Ask the patient to open the mouth wide, say the syllable "aa," or move the jaw side to side. In children, biscuits or chocolates can be given to simulate the chewing mechanism.

7. Ancillary work up
   1. Family photos
      This may help demonstrate the onset and progression of ptosis and also to document similar findings in family members.
   2. Visual fields
      Goldman ptosis visual fields are performed to document the visual fields with the lids in ptotic position, and again with the lids taped up to simulate the corrected position. The difference in the improvement of the central and peripheral visual fields is calculated. Insurance companies may often require this documentation.
   3. Preoperative photo documentation
      Current photographs taken in primary, up and downgazes document the ptosis severity and are helpful for comparison following surgery. Side-view photos also document any associated brow ptosis or temporal skin hooding over the lid margin.

CONCLUSION

Blepharoptosis may impair normal visual development in children and become visually significant to limit normal activities in adults. The etiologies of the various ptosis types and how they manifest differently are important to understand prior to undertaking surgery. Careful assessment, patient selection, and thorough preoperative evaluation are necessary to choose the most appropriate surgical plan.

Artifacts in Macular Optical Coherence Tomography

K. P. Mohana[1,2] and Dr. Muna Bhende[1]

[1]*Sri Bhagvan Mahavir Vitreoretinal Services, Sankara Nethralaya*

[2]*Elite School of Optometry*

INTRODUCTION

Optical coherence tomography (OCT) is a novel non-invasive instrument that gives a cross-sectional image of the retina in a micron-level resolution using low coherence Interferometry technique. Apart from macular evaluation, it also has its application in optic nerve head and anterior segment evaluation. The advantage of obtaining a highly resolved cross section of the retina is that it enables us to visualize the microstructure of the retina in detail and to monitor subtle changes in the retina over a period or after an intervention objectively.

In addition to having a qualitative use, OCT also provides a quantitative measure of the thickness of the retina using automated segmentation algorithms (Figure 1). With the advent of spectral domain OCT (see below), quantitative data have become more reproducible and reliable due to an increased scan density and a greater retinal coverage, apart from an improved visualization of retinal layers. Although this helps to monitor the disease progression and treatment response in conditions such as macular edema, choroidal neovascular membrane (CNVM), sub-retinal fluid, there are certain potential image artifacts which can mislead us to wrong diagnosis. Knowledge about the possible artifacts in an OCT image will aid in better interpretation of the disease condition. In this article, we have compiled those artifacts in OCT that will affect the interpretation.

TIME DOMAIN (TD) OCT VS SPECTRAL DOMAIN (SD) OCT

The OCT technologies that are in extensive clinical use are TD technology and SD technology. TD OCT is named so because the images are acquired as a function of time. It requires mechanical movement of the reference mirror to measure the echo time delays. In SD OCT, also known as Fourier domain OCT, the echo time delays of light are measured by using a stationary reference mirror and taking the Fourier transform of the interference spectrum. This results in a scanning speed ranging from 18,000 to 50,000 A-Scans/s in different SD OCT machines as compared to only 400 A-Scans/s in TD OCT. Also, SD OCT gives a better axial resolution of 5 μm compared to 10 μm in TD OCT. The first commercially introduced OCT was Stratus TD OCT, and there are different SD OCT machines including Cirrus HD-OCT, Topcon 3D OCT, Spectralis HRA, RT Vue etc.

TD OCT has been reported to give higher rates of significant errors than any other SD OCT machines.[1] Giani et al. have compared the artifacts in five SD OCT instruments and Stratus and have reported highest error frequency in Optopol Copernicus SD OCT instrument compared to other instruments, probably due to complex analysis of retinal segmentation.[2] Sull et al. have documented an increased incidence of artifacts in SD OCT volume scans than radial scans used in Stratus and few other SD OCT machines like Topcon 3D OCT, but also mentioned that an artifact in a single line scan will produce a larger impact on the retinal thickness map since it is based on the interpolated data from only 6 radial scans compared to 128 scans creating volumetric data.[3]

TYPES OF ARTIFACTS

Artifacts in OCT can be software related, operator related or patient related. Most of the artifacts in SD OCT are related to software errors. This can be corrected only by modifying the segmentation lines and placing it over the respective retinal boundary using the tools provided by the inbuilt software.

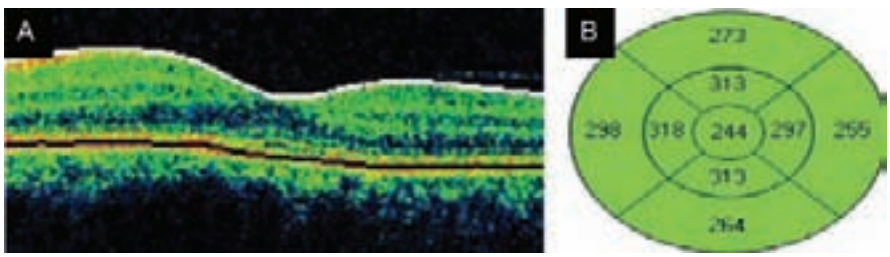


Figure 1. (A) Segmentation algorithm in Cirrus OCT instrument measuring retinal thickness by placing white and black lines along internal limiting membrane (ILM) and retinal pigment epithelium (RPE), respectively. (B) Color-coded ETDRS (Early Treatment Diabetic Retinopathy Study) thickness map showing average thickness values derived by measuring the distance between the segmentation lines, within each sector in 1-, 3- and 6-mm rings of the map.

Software-related artifacts

Misidentification of retinal boundary

OCT software requires a clear delineation of the inner and outer retinal interface to accurately identify them.[4] In conditions where the integrity of the interfaces is affected, misidentification of the boundaries occurs. This is the most commonly observed artifact among all OCT artifacts.[1–10]

Inner retinal boundary

This is the most frequently reported artifact[1] in which the inner segmentation line does not overlay the actual position of ILM. This is due to various reasons like poor quality of image due to media haze[4] or certain high reflective membranes simulating ILM like posterior vitreous detachment, vitreomacular traction, Epiretinal membrane[1] in which cases there is a possibility of overestimation of retinal thickness[2] (Figure 2A). Macular holes are also more prone for such artifact.[1,5] In eyes with macular edema not associated with CNVM, the inner and outer segmentation lines are clearly delineated,[4] unless there is degraded signal strength.

Outer retinal boundary

All instruments use ILM as the inner retinal border whereas the outer retinal boundary varies with different OCT machines. Stratus measures retinal thickness till inner segment/outer segment (IS/OS) junction, Topcon 3D OCT till OS tips of photoreceptors, Cirrus HD-OCT till RPE. Misidentification of outer retinal boundary occurs in CNVM (Figure 2B), sub-retinal fluid associated with macular edema and diseases that disturb the outer retinal integrity.[1,4]

In most cases, OCT scan artifacts affect only the retinal thickness map, but not the cross-sectional image itself. Hence, the clinician can rely on the morphologic information to assist in diagnostic and therapeutic decisions.[4] But, in multicenter clinical trials, central subfield thickness (CST) is an important parameter to monitor the patient; hence, it is better to either make the measurements manually or modify the segmentation lines if specified in the protocol.

Inverted artifact

This artifact is seen only in SD OCT images due to a mathematical function called Fourier transformation involved in extracting the image from the raw data.[7] This artifact is mostly associated with out-of-register scans in which the truncated portion of the image is seen inverted and overlapping with the real erect image (Figure 3A). Segmentation errors that are often seen in such artifacts are misidentified inner retina and incomplete outer retinal segmentation line.

Incomplete segmentation error

Here, the automated segmentation lines are placed correctly along the inner or outer retina, but are found missing in between before reaching the lateral edges of scan (Figure 3B). This occurred when the segmentation line approached outer retinal pathology or shadows from vessel or a partially degraded scan. Cirrus OCT also showed such artifact, but only in those B-scans that had degraded image quality. This artifact was frequently observed in Spectralis HRA + OCT and it commonly involved in the outer segmentation line.[5]

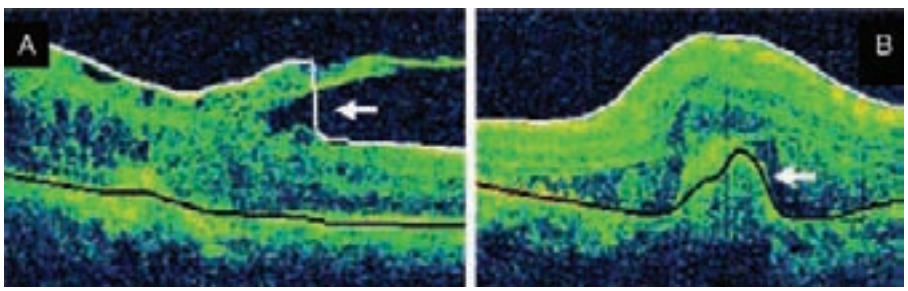


Figure 2. Misidentification of inner (A) and outer (B) segmentation lines (arrows) in vitreomacular traction and CNVM, respectively.

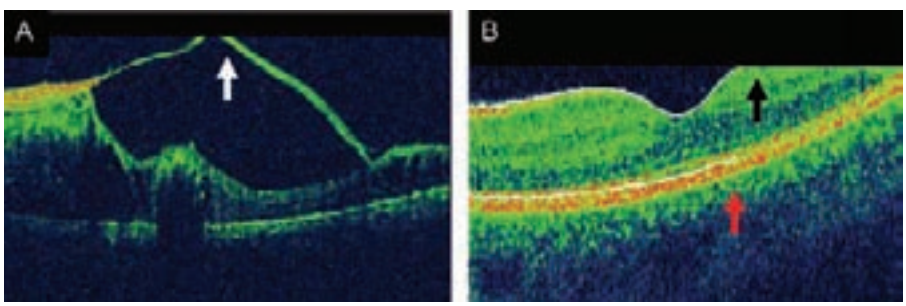


Figure 3. (A) Inverted artifact shows fibrovascular proliferative membrane (arrow) overlapping on the upright image which appears to cause traction on the right-hand side of the image. (B) Incomplete segmentation error (red arrow), out-of-register artifact (black arrow).

Complete segmentation error

Even when identifiable inner and outer retinal borders were present, there was no segmentation lines present along the inner and outer retina resulting in a complete segmentation failure. This was reported in some cases scanned in Spectralis.[5]

Operator-related artifacts

Apart from segmentation errors which are software related, there are certain avoidable artifacts which are operator related. The following three artifacts are due to poor scan acquisition and can be eliminated by careful attention during the acquisition process and by repeat scanning.

Out-of-register artifact

This artifact occurs when the inner or outer retinal layers are cropped when the image is shifted up or down while acquiring the scan (Figure 3B). This causes segmentation errors in the truncated portions of the image.[5] This artifact can be avoided by aligning the scan properly at the center of the image window than at the extreme top or bottom.

Off-center scans

It occurs when the scan is not centered on the foveal center. If the CST of the ETDRS map is more than 0.25 mm away from the true center based on the topographic and OCT B scan data, it is termed an off-centered scan[1] (Figure 4A). In cases where there is no foveal depression due to conditions such as macular pucker, cystoid macular edema and CNVM, centration errors are common, which always give incorrect CST.

To avoid off-center scans, the patient has to fixate well on the given target. Although this artifact is reported as a patient-induced error,[1] it is the operator's responsibility to monitor the fixation through the infrared camera and provide a larger target for a stable fixation or move it to an appropriate location to position the scan lines at the foveal center during the scanning.[4] In Spectralis, RTVue-100 and Topcon 3D-OCT, slightly off-center scans can be manually re-centered after image acquisition by moving the ETDRS map to the actual foveal center, but significantly decentered scans would result in an incomplete scan and erroneous retinal thickness measurements.[1,5]

Cut edge artifact

Cut edge artifact is the least reported artifact seen in Stratus[3] and Spectralis OCT[5] machines where the lateral edges of the scan, usually the left edge, are truncated (Figure 4B). This occurs without producing much effect on the surface map. In most of the scans obtained in Stratus, the inner and outer segmentation lines continue till the edge of the scan as a close approximation to the true interfaces[4] and it occurs only when the scanning starts and hence can be avoided by ignoring the first scan.[8]

Degraded scan images

Degraded scan images may be due to poor image acquisition by the operator (e.g. failure to adjust the OCT focus)[5,8] or due to some pathology in the media affecting the signal strength like cataract, vitreous hemorrhage/opacities. The lower signal strength is associated with significant segmentation errors affecting the retinal thickness measurements[9] (Figure 5).

Patient-related artifacts

Motion artifacts

Motion artifacts are common in TD OCT and it manifests as undulations in the neurosensory retina and RPE that may mimic retinal folds/drusens/PED.[7] With an improved scan acquisition speed in SD OCT machines, the frequency of this artifact has reduced considerably.[5] Spectralis has an eye-tracking system that maps certain landmarks on the retina and registers the OCT image with the fundus image to track the eye movements and to reduce the artifacts related to it. But when volume scans are obtained, motion artifacts (e.g. vertical microsaccades) are still a problem, which creates unrepresentative abnormal features on retinal thickness maps and affects the retinal thickness values[7] (Figure 6). Baskin et al. have reported such artifact as double fovea artifact.[12]

A new artifact was reported in Cirrus OCT related to eye movement that caused motion waves in the ILM (Figure 7) and RPE layer maps without affecting retinal thickness measurements, but may be misinterpreted as either a true pathologic feature or a significant algorithm error.[5]

EFFECT OF RETINAL DISEASE

Diseased eyes have more frequent artifacts than normal eyes.[2,4,5] In a study done in TD OCT, none of the normal eyes ($n = 11$) had segmentation errors.[4] Certain

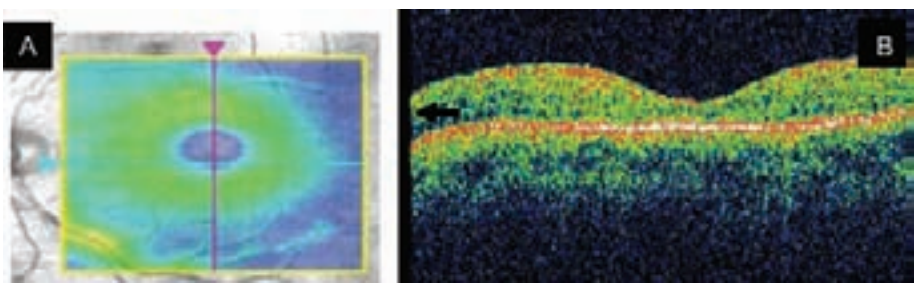


Figure 4. (A) The off-centered scan showing the blue circle in the thickness map corresponding to the fovea deviated away from the scan center (intersection point of purple and cyan lines). (B) The cut edge artifact in Stratus OCT image (arrow).

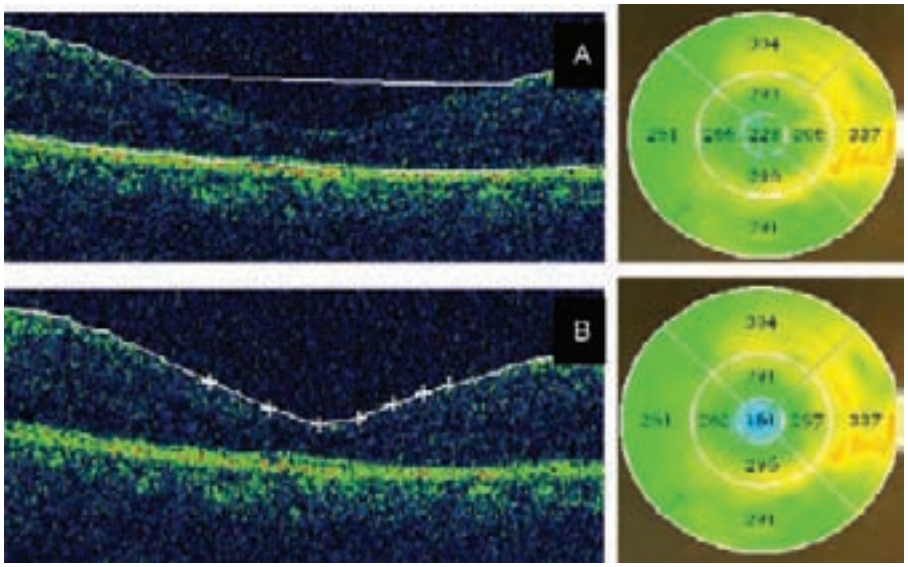


Figure 5. Degraded scan image. (A) Misidentification of inner retina due to media haze in a case of Uveitis, which resulted in abnormal thickening in ETDRS map. (B) After manual correction, ETDRS chart shows recalculated retinal thickness values.

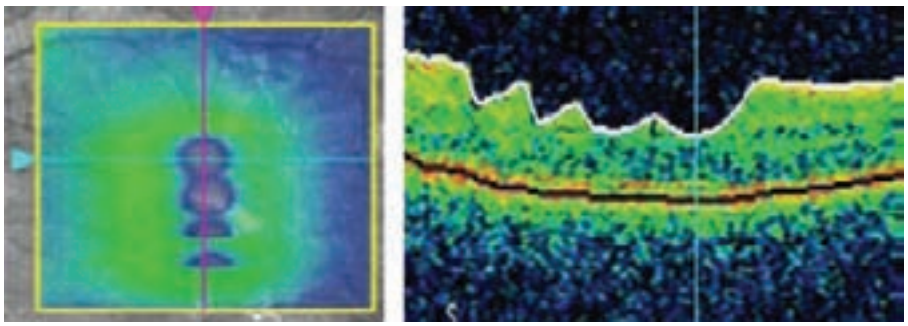


Figure 6. (A) Retinal thickness map appears to have a multiple foveal region due to vertical saccades. (B) Vertical section along purple line in thickness map shows multiple foveal dip.

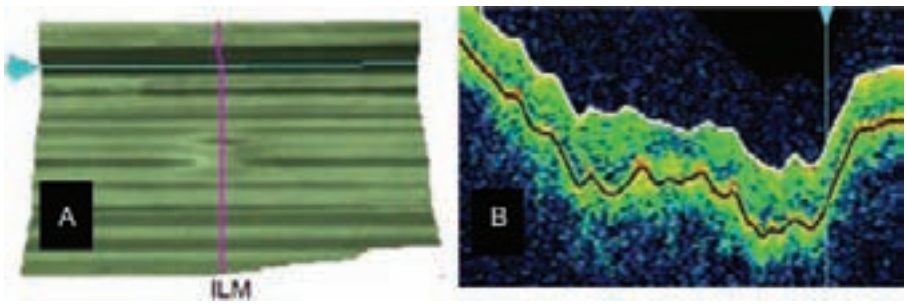


Figure 7. Motion artifacts. (A) Waves seen in the ILM map in Cirrus HD OCT device. (B) A vertical section along the purple line in ILM map shows irregularity in neurosensory retina due to eye movements. RPE undulations are seen due to both drusens and eye movements.

artifacts are associated more frequently with a particular disease condition. Macular diseases like CNVM and SRF that affect the outer retinal boundary are associated with an increased incidence of outer retinal segmentation errors,[1,2,4–6] whereas diseases like ERM, VMT and macular holes affecting the inner retina are significantly associated with an increased percentage of inner retinal segmentation errors.[1,2,4]

Neovascular age-related macular degeneration (nARMD) had the highest percentage of segmentation errors (90% of 50 eyes).[10] Uveitis and diabetic retinopathy was also reported to have inner segmentation errors,[5] but significantly lower error scores than CNVM.[9] Hard exudates present in macular edema would result in misindentification of outer retina, but in a study done by Sadda et al., it did not contribute significantly to retinal thickness measurement errors.[9] Retinal occlusive diseases had a higher prevalence of segmentation errors.[1,6]

Degraded scan images were caused by non-retinal diseases like cataract and corneal scarring.[4] Off-center scans

were more frequently associated with nARMD,[4] diabetic macular edema[6] and uveitis.[5] Out-of-register artifacts and inverted artifacts are observed in conditions which span a larger axial distance such as in high myopia, bullous retinal detachment and structures in vitreous like asteroid hyalosis and largely detached hyaloid face.[7,11]

EFFECT ON FOVEAL THICKNESS

The central 1-mm area of the ETDRS map contributing to foveal thickness is essential as a treatment criterion and is related to visual acuity. Han et al. showed that 32.7% of 98 and 37.5% of 88 scans taken in Cirrus and Spectralis, respectively, are having at least 1 artifact in the central 1-mm area affecting the CST. They also graded the severity of segmentation errors within central 1 mm based on the ratio of segmentation error to overall retinal thickness; mild error as that affecting less than one-third of actual retinal thickness at the point of error; moderate error as one-third to two-thirds and severe error as more than two-thirds of retinal thickness.[5] An error of 11 μm or more in the central subfield after the manual correction was regarded as a clinically significant inaccurate foveal thickness.[1]

Regardless of the retinal disease, misidentification of retinal boundary and off-center artifacts always produced an incorrect thickness map, either an abnormal thickening or a thinning which led to erroneous interpretation of the disease response to treatment.[1,4] Other artifacts like incomplete and complete segmentation failure rarely affected the central 1-mm area but altered the peripheral subfields.[5] With increasing centre foveal thickness, the proportion of decentration artifact reduced whereas segmentation errors increased.[9]

CONCLUSIONS

OCT image artifacts occur frequently and certain artifacts are associated with a particular disease. Few artifacts are never observed in the center 1-mm area of scans but when present it will affect the CST measurements greatly. Some are avoidable with a careful attention to the scanning procedure. Those which are unavoidable should be recognized and interpreted with caution. Thus, an insight into the scan artifacts, diseases that produce them and ways to avoid them will increase the diagnostic value of this imaging technique.

REFERENCES

1. Ho J, Sull AC, Vuong LN, Assessment of artifacts and reproducibility across spectral- and time-domain optical coherence tomography devices. Ophthalmology 2009;116:1960–70.
2. Giani A, Cigada M, Esmaili DD, et al. Artifacts in automatic retinal segmentation using different optical coherence tomography instruments. Retina 2010;30:607–16.
3. Sull AC, Vuong LN, Price LL, et al. Comparison of spectral/Fourier domain optical coherence tomography instruments for assessment of normal macular thickness. Retina 2010;30:235–45.
4. Ray R, Stinnett SS, Jaffe GJ. Evaluation of image artifact produced by optical coherence tomography of retinal pathology. Am J Ophthalmol 2005;139:18–29.
5. Han IC, Jaffe GJ. Evaluation of artifacts associated with macular spectral-domain optical coherence tomography. Ophthalmology 2010;117:1177–89.
6. Domalpally A, Danis RP, Zhang B, et al. Quality issues in interpretation of optical coherence tomograms in macular diseases. Retina 2009;29:775–81.
7. Sadda SR, Keane P. Pitfalls to consider when using spectral domain OCT. *Retina Today*, March 2009, 38–41.
8. Hee MR. Artifacts in optical coherence tomography topographic maps. Am J Ophthalmol 2005;139(1):154–5.
9. Sadda SR, Wu Z, Walsh AC, et al. Errors in retinal thickness measurements obtained by optical coherence tomography. Ophthalmology 2006;113:285–93.
10. Patel PJ, Chen FK, da Cruz L, et al. Segmentation error in Stratus optical coherence tomography for neovascular age-related macular degeneration. Invest Ophthalmol Vis Sci 2009;50:399–404.
11. Ho J, Castro DPE, Castro LC. Clinical assessment of mirror artifacts in spectral-domain optical coherence tomography. Invest Ophthalmol Vis Sci 2010;51:3714–720.
12. Baskin DE, Gault JA, Vander JF, et al. Double fovea artifact. Ophthalmology 2011;118 (2):429–9.

Effectiveness of Vision Therapy in the Treatment of Symptomatic Convergence Insufficiency: Results from the Pilot Study

M. Revathy[1,2], H. Jameel Rizwana[1,2] and R. Krishna Kumar[1]

[1]*Elite School of Optometry, 8 G.S.T Road, St. Thomas Mount, Chennai 600 016*

[2]*Sankara Nethralaya, 18, College Road, Nungambakkam, Chennai 600 006*

ABSTRACT

The purpose of this study was to determine the effectiveness of vision therapy (VT) in the treatment of symptomatic convergence insufficiency (CI). Symptomatic CI subjects were given 10 sessions of 60 minutes of in-office VT (IVT) followed by 30 minutes of home vision therapy (HVT) per day for 3 months. Near exophoria, near point of convergence (NPC) break and near positive fusional vergence (PFV) break were the primary outcome measures which were reviewed after IVT and at the end of three consecutive months of HVT. Out of 29 subjects recruited (age 23.9 $\pm$ 7 years [mean $\pm$ SD]; range 9–39 years), 10 have completed the 3-month follow-up visit, 8 were in the HVT phase, 6 were in the IVT phase and 5 were lost to follow-up. There was a significant improvement in the primary outcome measures at the end of 3 months. The near exophoria improved to 4.0Δ from 7.0Δ, NPC from 11.0 to 5.0 cm and PFV from 16.0Δ to 45Δ after VT. The success rate was 80% after IVT and 90% at the end of 3-month follow-up visit. IVT combined with HVT is very effective in treating CI.

1. INTRODUCTION

Convergence insufficiency (CI) is a common binocular vision disorder that is often associated with a variety of symptoms, including eyestrain, headache, blurred vision, diplopia, sleepiness, difficulty in concentrating, movement of print while reading and loss of comprehension.[1,2] Clinically, CI is defined as "a syndrome characterized by greater exophoria at near, receded near point of convergence (NPC), reduced positive fusional convergence, and a low AC/A ratio".[1,3,4]

To the best of our knowledge, six studies have been published in India on CI and its treatment since 1961.[5–10] Most of them were retrospective studies reporting the frequency of CI in the clinical setting.[5–8] Efficacy of treatment has been reported only with synoptophore exercises and home-based pencil push-up exercises.[7,10] These studies lack in clear description of diagnostic criteria, sequence of treatment protocol, definition of endpoints and duration of home exercise given. Recent multi-centered, randomized, clinical trial (Convergence Insufficiency treatment trial—CITT)[11] has shown that pencil push-up exercises are no more effective than IVT and prism glasses were equivalent to placebo treatment in the treatment of CI. Hence we proposed to determine the effectiveness of VT in treating symptomatic CI.

2. METHODS

The study was a prospective interventional study, conducted between August 2010 and May 2011, at the Binocular Vision and Vision Therapy Clinic of Medical Research Foundation, Sankara Nethralaya. The study adhered to the declaration of the Helsinki and was approved by the Institutional Review Board and Ethics Committee of Vision Research Foundation. Symptomatic subjects were recruited from Binocular Vision Clinic. Comprehensive binocular vision assessment was done, which included best-corrected visual acuity (distance and near), cycloplegic refraction and a sensorimotor examination that included near stereoacuity with Randot stereo test, sensory fusion for distance and near using Worth four dot test, prism base cover test (distance and near), NPC with Astron International Convergence ruler (ACR rule), NPC using red and green filter, positive and negative fusional vergence at near using prism bar, vergence facility using 12 BO/3 BI prism flippers, monocular accommodative amplitude by push-up method, accommodative response by monocular estimation method, monocular and binocular accommodative facility with $\pm$2.00D flippers. Near exophoria, NPC with ACR rule and near PFV were the primary outcome measures of the study. All the diagnostic tests, IVTs and follow-up visits were reviewed by a single examiner (RM) to avoid inter-examiner variability. In order to minimize examiner's bias during the follow-up visits, the objective assessment was done first and then the baseline values were reviewed.

The inclusion criteria for CI were (i) exophoria at near should be at least 4Δ greater than at far; (ii) the NPC break point should be $>$6 cm; (iii) near positive fusional vergence (PFV) should be at least $\leq$20Δ base out blur or break; (iv) best corrected visual acuity (BCVA) $\geq$20/25 in both eyes; (v) stereoacuity of at least 500 arc seconds; (vi) monocular amplitude of accommodation $>$15–0.25 $\times$ age (Hofstetter's minimum expected); (vii) informed consent and willingness

to participate in the study. The exclusion criteria were (i) CI previously treated with vision therapy/orthoptics within 1 year; (ii) strabismus; (iii) amblyopia; (iv) any ocular surgery; (v) manifest or latent nystagmus; (vi) systemic diseases. The vision therapy regimen comprised 60 minutes of in-office VT per day for 10 sessions. IVT sessions comprise computerized orthoptic training (VTS software®, HTS Inc., USA) and traditional VT equipments like tranagalyphs, aperture rule and synoptophore. Home VT exercises were prescribed for the next 3 months. The subjects were called at the end of every month for reviewing the outcome measures for 3 months. All subjects underwent a specific therapy regimen of IVT[11,12] for 60 minutes per day for 10 sessions. IVT was divided into three phases to train the gross convergence, vergence amplitudes/facility and accommodative amplitude/facility. After 10 sessions of IVT, home vision therapy (HVT) was given for the consecutive 3 months. Home log books were given to ensure compliance. Outcome measures were assessed at the end of every month.

Table List of home vision techniques

S. no.	Procedure	Purpose	Duration (min)
1	Brock string	Gross convergence	5
2	Transparent eccentric circles	NFV amplitude	5
3	Opaque eccentric circles	PFV amplitude	5
4	Transparent Life Saver card	NFV amplitude and facility	5
5	Opaque Life Saver card	PFV amplitude and facility	5
6	Hart Chart	Accommodative facility	5

3. RESULTS

Out of 68 (9.7%) CI subjects identified, 29 participated in the study (8 males and 21 females; age 23.7 ± 7 years [mean $\pm$ SD]; range 9–39). The baseline mean spherical equivalent refractive error of the right eye was $+0.73 \pm 1.45$D and for the left eye was $+0.05 \pm 1.15$ D. The mean duration in days for completing IVT from baseline was 21 ± 9 days. The overall duration of all follow-up visits were 120 ± 8 days [mean $\pm$ SD]. Out of 29 recruited, 10 (34.5%) subjects had completed the overall 3 follow-up visits; 8 (34.5%) are in IVT phase; 6 (20.7%) are in HVT phase; 5 (17.2%) subjects were lost to follow-up during the IVT phase.

Table 1 shows the distance and near phoria measures for all the follow-up visits. The median values were given as the data were not normally distributed. The Friedman test was used to compare the overall differences in the exophoria for all the follow-up visits ($p<0.05$). Post hoc comparisons (Wilcoxon signed-rank test) were done to find the significant difference between baseline and all follow-up visits with a conservative p value of 0.01. Wilcoxon signed-rank test was used as a post hoc test. Significant differences were obtained between baseline vs. post IVT follow-up and baseline vs. 3-month follow-up visit (baseline vs. post IVT, $p=0.006$; baseline vs. 1st

Table 1 Phoria measure of all the follow-up visits

Characteristics ($n=10$)	Baseline	Post IVT	1st month	2nd month	3rd month	p
Mean age (years) $\pm$ SD			24.2 ± 7.7			
Distance phoria, prism dioptre (Δ), median (IQR)	0.0 (0.0–3.0)	0.0 (0.0–4.5)	0.5 (0.0–3.0)	0.0 (0.0–3.0)	0.0 (0.0–4.5)	0.790
Near phoria(Δ), median (IQR)	7.0 (2.0–8.5)	4.0 (3.5–8.5)	4.0 (2.0–7.5)	3.0 (2.0–9.0)	4.0 (1.7–7.5)	0.001*

*$p<0.01$ by Friedman's test.

Table 2 NPC for all the follow-up visits

Characteristics ($n=10$)	Baseline	Post IVT	1st month	2nd month	3rd month	p
NPC break (cm), median (IQR)	11.0 (7.8–15.0)	5.0 (4.0–6.8)	5.3 (3.5–7.0)	6.0 (4.9–6.6)	5.0 (5.0–6.5)	0.000*
NPC recovery(cm), median (IQR)	14.0 (10.0–20.5)	6.3 (5.5–9.5)	7.3 (5.5–9.3)	8.5 (6.8–10.3)	7.5 (6.0–8.0)	0.000*

Table 3 PFV break at all follow-up visits

Characteristics ($n=10$)	Baseline	Post IVT	1st month	2nd month	3rd month	p
Near PFV break (Δ), median (IQR)	16.0 (13.5–18.5)	37.0 (35.0–46.2)	45.0 (32.5–46.2)	45.0 (35.0–50)	45.0 (33.7–46.2)	0.00*
Near PFV recovery (Δ), median (IQR)	13.0 (9.0–16.5)	30.0 (27.0–41.3)	10.0 (23.6–41.3)	40.0 (27.5–45.0)	37.5 (23.8–41.3)	0.00*

*$p<0.01$ by Friedman's test.

month, p=0.01; baseline vs. 2nd month, p=0.01, baseline vs. 3rd month, p = 0.007).

Table 2 lists the NPC break measures for all the follow-up visits. The median NPC break measure changed to 5.0 cm (5.0–6.5) from 11.0 cm (7.8–15.0) [median (IQR)] after the IVT which was statistically significant (Wilcoxon signed-rank test, $p<0.05$). There was a significant difference in the NPC among all follow-up visits ($p<0.05$, Friedman test) and post hoc comparisons (Wilcoxon signed-rank test) revealed significant difference between baseline and every follow-up visits (p = 0.005).

Table 3 shows the near PFV break measures for all the follow-up visits. The PFV break increased from 16Δ (13.5–18.5) to 45.0Δ (33.7–46.2) [median (IQR)] at the end of IVT (Wilcoxon signed-rank test, $p<0.05$ for all the follow-up visits). Table 4

4. CURE RATES AT THE END OF IVT AND AFTER 3-MONTHS OF HVT

Subjects who achieved NPC <6 cm at final follow-up or change of 4 cm NPC from baseline to final follow-up visit and PFV >20Δ or change of 10Δ from baseline measures were considered "Success". Subjects who did not achieve both the criteria were considered as "failures". Cure rates were calculated for 18 subjects who completed IVT. Out of 18 subjects, 13 subjects (72.2%) were "successful" and 5 subjects were (27.8%) "Failures". Cure rates were calculated after 3 months of HVT. Out of 10 who completed HVT, 9 (90%) were successful and 1 (10%) failed. The cure rates were significantly predicted by near PFV break ($p<0.05$) by the regression model. Age, gender and NPC break were not predicting the success rates.

5. DISCUSSION

There was a significant improvement in the primary outcome measures (near exophoria, near PFV and NPC) and the differences were significant both statistically and clinically at the end of 3-month follow-up visit. The median near exophoria improved significantly from 7.0Δ to 4.0Δ. The magnitude of phoria was well within the normal range as set by our protocol at all follow-up visits. The phoria difference between the distance and near was not more than 4Δ after IVT and had maintained the same throughout the HVT phase. The phoria did not revert back or increase during the follow-up visits. NPC break improved significantly after VT. The normative data for NPC break are ≤6 cm.[13] In CITT,[11] the NPC break improved from 13.3 cm to 4.0 cm. In our study, 60% of subjects achieved a normal NPC of less than 6 cm at the end of 3-month follow-up and remaining 40% achieved less than 10 cm in the 2- and 3-month follow-up visit. In CITT, the PFV break improved from 11.0Δ to 30.5Δ and another study by Cooper and Feldman,[14] improved the PFV to 53Δ using computerized VT. The near PFV break in our study significantly improved to 45Δ from 16Δ. 90% of the subjects had PFV improved to more than 20Δ. Only 10% of the subject had reduced PFV to less than 20Δ especially in the 3-month follow-up.

In CITT,[11,12] the success rate reported was 53.3% among the IVT group. In our study, 80% were successful after IVT and there was 90% success rate at the end of 3-month follow-up. The 10% failure rate could be due to the severity of CI and non-compliance with HVT though adequate steps were followed to ensure maximum compliance. The compliance of HVT could not be tracked even after providing home log books and reminders through weekly phone calls. The success rate in our study is higher when compared to CITT. This could be due to the difference in the IVT administrations sessions in CITT when compared with our study. CITT study had 60 minutes per day of IVT for 12 weeks compared to 60 minutes per day of IVT completed within a month in our study. To avoid the decrease in divergence (NFV) amplitudes due to convergence training, we gave simultaneous NFV training during each session. We compared the pre- and post-NFV amplitudes from the baseline to the end of the follow-up visits and no significant differences were found.

There are certain limitations to our study. No masked examination was done during the follow-up visits though the follow-up assessments were done first before reviewing the baseline measures. The duration of days taken to complete the IVT ranged from 12 to 31 days. In spite of clear instructions given during the recruitment phase, subjects failed to follow the IVT regularly. This resulted in difference in continuity of IVT follow-ups, i.e. 4 of 10 underwent IVT continuously with a few days time gap in between where as outstation subjects completed the IVT on consecutive days without any gap. Out of 29 subjects, 10 subjects completed the overall follow-up visit, 8 subjects are in the HVT phase, 6 subjects are in the phase of doing IVT and 5 did not turn up for IVT. This loss to follow-up may be due to practical problems of visiting the hospital for 10 sessions on consecutive days.

We conclude that VT was effective in treating symptomatic CI. There was a significant improvement in the primary outcome measures (near exophoria, NPC break and near PFV break) following VT. The improvement achieved was stable during the 3 months of HVT. Thus multi-method protocol of VT of shorter period of time 10 sessions were effective in improving the clinical signs of CI. HVT is recommended to maintain the stability of improved parameters.

REFERENCES

1. Cooper J, Duckman R. Convergence insufficiency: incidence, diagnosis, and treatment. J Am Optom Assoc 1978;49:673–80.
2. Adler P. Efficacy of treatment for convergence insufficiency using vision therapy. Ophthal Physiol Opt 2002;22:565–71.
3. Borsting E, Rouse M, De Land NP. Prospective comparison of convergence insufficiency and normal binocular children on CIRS symptom surveys. Optom Vis Sci 1999;76:221–8.
4. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders*, Chapter 8. Philadelphia: Lippincott Williams and Wilkins, 2002, p. 226.
5. Dhir BK. Convergence insufficiency. Indian J Ophthalmol 1961;9:33–35.
6. Desai R, Desai S, Desai N, et al. Visual status of industrial workers. Indian J Ophthalmol 1990;38:64–65.
7. Deshpande SB, Ghosh RK. Study of primary convergence insufficiency. Indian J Ophthalmol 1991;39:112–4.
8. Archana S, Kailwoo K, Vijayawali. Convergence insufficiency in patients visiting eye OPD with headache. JK Sci 2008;39:135–36.
9. Patwardhan S, Pradeep S, Rohit S et al. Preferred clinical practice for convergence insufficiency in India—a survey. Indian J Ophthalmol 2008;56:303–6.
10. Reddy RH, Vidyavati DA. Accommodation synoptophore in the treatment of convergence insufficiency. Indian J Ophthalmol 1983;31:367–8.

11. CITT Group. Randomized clinical trial of treatments for symptomatic convergence insufficiency in children. Arch Ophthalmol 2008;126:1336–49.
12. Scheiman M, Mitchell GL, Cotter S, et al. A randomized clinical trial of treatments for convergence insufficiency in children. Arch Ophthalmol 2005;123:14–24.
13. Hayes GJ, Cohen B, Rouse M, et al. Normative Values for the near-point of convergence of elementary school children. Optom Vis Sci 1998;75:506–12.
14. Cooper J, Feldman J. Reduction of symptoms in binocular anomalies using computerized home therapy – HTS. Optometry 2009;80:481–6.

Is this Pupil Normal?

Ashwin Mohan[1] and Rashmin Gandhi[2]

[1]Resident, MRF, Sankara Nethralaya, Chennai

[2]Senior Consultant, Neurophthalmology, and Director E learning, MRF, Sankara Nethralya, Chennai

WHAT IS A NORMAL PUPIL?

Anatomically

- One single round opening of about 3–4 mm diameter in the center of the iris that has a pupillary ruff at its circumference. The size can vary depending on the illumination.
- The pupil aperture generally appears grayish black.

Physiologically

- One that dilates in dark illumination and constricts in bright light uniformly
- Both eyes should have the pupil of the same diameter irrespective of whether both eyes are illuminated uniformly or one eye is illuminated more than the other.

Now that we know the characteristics of a normal pupil, we can say that if any of the above findings in the pupil are inconsistent, the pupil has some abnormality. We will now analyze each of the above characteristics mainly focussing on the physiological aspects and try arriving at a diagnosis.

The pupillary diameter is controlled by a number of factors and hence understanding of these factors can help us gain insight into any underlying pathology. The pupillary reflexes can be used as an excellent screening test to rule out or rule in defects in the visual system. The pupillary reflex, i.e. constriction and dilatation, is mediated by parasympathetic and sympathetic outflow, respectively. The pupil reacts to changes in light intensity and to the object in focus, i.e. the near reflex. Thus pathology in the afferent visual pathway, the integration nuclei in the CNS or the efferent autonomic system can all independently result in an abnormal pupillary appearance.

Visual stimuli from the rods and cones, via the ganglion cells, reaches the pretectal nuclei, after decussating at the optic chiasma. From here, via the internuncial fibers, reaches both the Edinger Westphal nuclei equally traversing the posterior commissure, explaining why both the eyes react to the light perceived by one eye alone. Although supranuclear afferent input from the cerebral cortex, brain stem and even spinal cord may have a modulatory effect (both excitatory and inhibitory) on the pupil response to a light stimulus, it is not established that these pathways influence the presence or magnitude of the RAPD.[1] Efferent parasympathetic fibers from Edinger Westphal nuclei travel via the 3rd cranial nerve to the ciliary ganglion. Short ciliary nerves from this ganglion innervate the sphincter of the iris to cause a constriction, thus limiting the light entering the eye.

Thus we can categorize the pathology into two broad categories:

1. An afferent pathway defect
2. An efferent pathway defect

The pupils are best examined in a room with moderate lighting, and the subject focussing at a distant object, thus eliminating any accommodative reflex of the pupils. Any anatomical factors that can impede the smooth movement of the pupil should be ruled out before we proceed for the testing of the pupillary function. Posterior synechiae can cause a mechanical obstruction as can an IOL haptic post surgically. These will cause an unequal reaction of the pupil.

First, in diffuse illumination, once the above basic characteristics of each pupil are ascertained, we must compare the two pupils to check for any inequality. Assuming that the pupils are round and equal in size in both eyes, we can examine for the afferent defects. Testing the near response if the light reaction is normal does not add any further information, because the near response is always normal with a normal light reaction.[2]

AFFERENT PATHWAY DEFECTS

- Both eyes constrict to a lesser extent when the pathological eye is stimulated with light.
- In a purely afferent defect, both the pupils are of the same size, inequality in size, almost always indicates an efferent pathology.
- In general, the association between RAPD and visual acuity is poor. There is a closer correlation between RAPD and visual field loss.[3]

The direct and the consensual reflexes are tested, first by shining a torchlight into each eye in a dimly lit room and observing the pupillary reaction in that eye with respect to constriction and speed. The consensual reflex is tested by the swinging flashlight test where light is shone alternatively into each eye. If the pupil dilates when light is shone into that eye, then a Marcus Gunn pupil or relative afferent pupillary defect is diagnosed. This should be graded with neutral density filters in log mar units to document the extent of RAPD.

RAPD depends on

1. The total amount of light that enters and stimulates the rods and cones
2. The area of functional retina that can be stimulated

3. The integrity of the retrobulbar optic pathway

Advanced *Glaucomatous damage* can decrease the functional nerve fiber layer and thus carry less light stimulation resulting in an RAPD.

Optic nerve diseases like retrobulbar optic neuritis will result in an impedance to the transmission of the impulse.

If an RAPD is present, in an eye with a cataract, it must alert us to the possibility of a retrolental pathology.

EFFERENT PATHWAY DEFECTS

An efferent pathway defect is characterized by the presence of an Anisocoria. An anisocoria can produce an RAPD because the smaller pupil will limit the amount of light that enters that eye. We must remember that constriction is the result of parasympathetic outflow that is a response to bright light and dilatation is due to sympathetic outflow, a reaction to dim light. Hence if the sympathetic outflow is affected to one eye, that eye will dilate poorly in response to dim light, compared to the other eye that has normal sympathetic innervation. Similarly, a parasympathetic defect will result in a loss of miosis in response to bright light in that eye. We must also remember that an action can occur due to the inhibition of the antagonistic action, i.e. dilatation can occur in dim light even in the absence of sympathetic stimulation due to the inhibition of parasympathetic tone. Thus, the anisocoria should be evaluated in scotopic (dim light) and photopic (bright light) conditions to ascertain when the anisocoria seems to be more apparent.

If it is found to be more in dim light, then it is suggestive of a Horner's syndrome or any other pathology in the sympathetic innervation of that eye. Other ocular manifestations of decreased sympathetic innervation like ptosis and anhydrosis also should be noted. Extra-ocular movements to note any oculomotor palsy should also be done.

Horner's syndrome can be confirmed with administration of either 2–10% cocaine HCl which aggravates the anisocoria, or apraclonidine which produces a reversal of anisocoria due to its weak alpha1 agonistic action on the supersensitive denervated iris muscle.[4]

The location of this lesion can be determined with the hydroxyamphetamine test, which acts by releasing norepinephrine from the terminal nerve endings. Thus in a post-ganglionic lesion, classically no dilatation will be produced because the nerve is dead, which is not the case with central or pre-ganglionic Horner's syndrome. As ophthalmologists we be aware of life-threatening causes of Horner's syndrome like Pancoast's tumor and multiple sclerosis and take necessary action.

If it is found that the anisocoria is greater in bright light, then reduced action of the iris sphincter in the eye with larger pupil is the cause. This can be either due to abnormalities of the iris sphincter itself due to ischemia or surgical trauma, or due to defective parasympathetic flow to, or receptor blockage in the iris sphincter. Segmental denervation of the sphincter or Adie's Tonic pupil is an important cause especially in young women. Characteristic vermi-form movements of the pupil are seen due to segmental contractions. Re-innervation then succeeds in due course or time to ultimately make this pupil the smaller of the two.

Third nerve compressive lesions can also present in the same way and is called Hutchinson's pupils. Segmental dilator muscle spasm due to sympathetic hyperactivity can produce the transient "tadpole pupil" which is often seen in migraine.

If a pupils constricts better to near accommodation than to light stimulation, it is known as Argyll–Robertson pupil or light near dissociation. This can occur due to damage to the Edinger Westphal nucleus or aberrant re-innervation or regeneration of the third nerve. Tertiary Syphillis is a well-known infectious cause of this disease. Dorsal Midbrain syndrome or Perinaud's syndrome will also result in a light near dissociation along with a vertical gaze palsy and contraction nystagmus.

REFERENCES

1. Kawasaki A, Miller NR, Kardon R. Pupillographic investigation of the relative afferent pupillary defect associated with a midbrain lesion. Ophthalmology 2010;117:175–9.
2. Wilhelm H. Neuro-ophthalmology of pupillary function—practical guidelines. J Neurol 1998;245:573–83.
3. Bremner FD Pupil assessment in optic nerve disorders. Eye (Lond) 2004;18:1175–81.