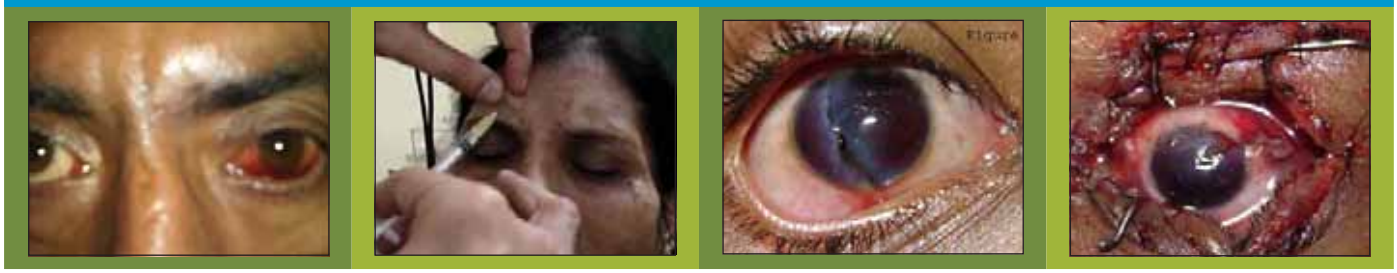
Contents



Editorial

My Dear Friends and Colleagues,

This Issue of DOS Times is different. Here is a Curtain Raiser for you.

The Community Approach in Ophthalmology

We all know that Our Country needs much more Ophthalmology Care than it gets. There are reasons of affordability, education , awareness and of approachability. Even today in Delhi and NCR,leaving aside other lesser developed areas of India-we routinely operate upon mature and hard cataracts. So much so that most phaco surgeons, think that operating on these types of cataracts is nothing to write home about.

So the need is there , and more ophthalmologists can get more work and every one can benefit.

The Modus Operandi

Why is it that some hospitals and institutes have adopted the community model very successfully and profitably, and others falter or not even try. Hospitals like Aravind Eye Hospital have proven that this is do-able and viable and desirable. Cleary, this model benefits the community and gives a wide area for clinical work to the Ophthalmologists. However, not many such successful models operate and many Ophthalmologists are looking for work in an Ocean full of it.

The Secret

The book by this name has recently been a bestseller, but here we are looking for a secret of a different kind. There are a lot of incentives and finances available from the government and international agencies for community Ophthalmology. For Cataract Surgeries,Eye Bank and Corneal Transplantations, School Eye Check Up programmes, Diabetic Retinopathy and Glaucoma Screening etc. One has to know how to go about and Start a community model and utilize these funds and benefits.

And this is what this issue of DOS times plans to tell you.
How to Start An Eye Hospital based on the community model.
How to Out reach to the general public in the towns and the villages.
How to finance the project and how to avail the funds available from the government and other charitable organizations.
How to make the project viable, so that at the end of the day ; you and your staff take home some money.
How to make a charitable trust or an NGO.
How to set up an eye bank. etc.

I am sure, and you'll agree that all of us need this knowledge. The young budding Ophthalmologist, so that he has better career options and the well established eye centres-so that they can provide quality eye care to the community and to give something back to the community.

I hope you enjoy this issue which we have painstakingly produced for you, requesting some of the pioneers in this field to personally write these crucial articles to benefit all of us.,

Yours Truly.

Thanking you,

Dr Amit Khosla
Secretary,
Delhi Ophthalmological Society

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Delhi
INDIA

Delhi Ophthalmological Society

15th to 17th April, 2011,
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INVITATION

Dear friends & colleagues,

Greetings from Delhi Ophthalmological Society. We wish to invite you to our 62nd Annual Conference from 15th to 17th April 2011 at Hotel Ashok, Delhi. Indian Ophthalmology is highly advanced and Ophthalmology in Delhi is vibrant and alive. Delhi has perhaps the maximum number of Ophthalmologists, the largest number of Ophthalmology training institutes and the largest number of Ophthalmology residents in the world. All subspecialities of Ophthalmology are highly developed and all surgeries-classic and recent advances are routinely performed.

The Delhi Ophthalmological Society is the Largest State Society in India with over 5000 members and The Annual Conference of our society is a 3 day celebration of Ophthalmology-live surgeries and wet labs, workshops, instruction courses and free papers showcasing original research work. Ophthalmologists of International repute participate in this conference. This year our theme is **"Trends in Ophthalmology"** and we will be abundantly pleased to welcome you to our conference and our city.

Delhi, the Capital city of India is a modern metropolis with all the world class facilities and a grand historical legacy. A brand new airport, comparable to the best in the world awaits your arrival. Wide roads and swanky radiotaxis provide excellent connectivity. The Delhi Metro Rail is fast, clean and punctual and there is a fast express link from the airport to the heart of the city. Delhi's air is clean and highly breathable. It is one of the greenest cities in the world. Your stay will be extremely comfortable. Our hotels are among the world's best and provide unmatched service. Indian cuisine is appreciated and duplicated the world over and Delhi Restaurants will provide you with a memorable culinary experience and the taste of India in Delhi.

The comfortable and Airconditioned Shopping Malls in and around Delhi always have a festive atmosphere and your shopping experience is bound to be incredible. In addition there are the traditional local markets like Karol Bagh and Chandni Chowk to provide you with the local flavour. Connaught Place in the heart of Delhi is a huge commercial centre built in a circular fashion. It was built by the British and is an icon for the city. There are number of theaters playing the classical and latest movies from Hollywood and Bollywood. The Golden Triangle Tour (Delhi-Agra-Jaipur-Delhi) offers you with an opportunity to witness the great Indian heritage.

I wish forward to welcome you to Delhi in the pleasant month of April.

Yours truly

Dr Amit Khosla
Secretary, Delhi Ophthalmological Society

Dr P.V. Chadha
President, Delhi Ophthalmological Society

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Diabetic Macular Edema

Dr. Lingam Gopal, *MS*

Dr. R. Kim, *MS*

Dr. Amit Khosla, *MD*

Dr. Pradeep Venkatesh, *MD*

Diabetic macular edema (DME), defined as a retinal thickening involving or approaching the center of the macula, represents the most common cause of vision loss in patients affected by diabetes mellitus. The pathogenesis and the course of DME require a complex approach with multidisciplinary intervention both at the systemic and local levels. In the last few years, many diagnostic tools have been proven useful in the detection and the monitoring of the features characterizing DME. In addition various therapeutic strategies (medical/ surgical) have been developed to treat DME. The aim of the present FOCUS on Diabetic Macular Edema is to thoroughly delineate the clinical and morpho-functional characteristics of DME and its current treatment perspectives.

(LG): Dr. Lingam Gopal*, MS, DNB, FRCS, Vitreo Retina Surgeon, Chairman, Sankara Nethralaya, Chennai, Tamil Nadu*

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(PV): Dr. Pradeep Venkatesh*, MD, Associate Professor, Vitreo Retina Services, Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Medical Sciences, Ansari Nagar, New Delhi*

(NV): Dr. Naginder Vashisht*, MD, Consultant, Sir Ganga Ram Hospital, Rajender Nagar, New Delhi*

NV: How do you diagnose diabetic macular edema?

LG: I diagnose diabetic macular edema first by clinical evaluation using slit-lamp biomicroscopy with +78 d lens. In case of suspicion as well as in confirmed cases, this is followed by OCT. I do not use vision as a criteria to diagnose.

RK: Diabetic macular edema is diagnosed clinically by slit- lamp biomicroscopy, using a 90 or 78 dioptre condensing lens.

AK: Diabetic macular edema is a clinical diagnosis. I usually do slit lamp biomicroscopy with +78 D/+90 D lens. Some of the clinicians make their diagnosis based on direct ophthalmoscope; however I recommend +90D lens aided slit lamp biomicroscopy as this is the best tool to evaluate retinal thickness clinically.

PV: Contact biomicroscopy is the Gold standard for clinical diagnosis of macular edema. As this needs topical anesthesia, special diagnostic lenses and is relatively more time consuming, I prefer non-contact fundus biomicroscopy for diagnosing diabetic macular edema. I use standard guidelines advocated in the ETDRS study to differentiate clinically significant from clinically non-significant macular edema.

My first clinical indicator to the presence of macular edema is the presence of foveal hard exudates. Careful fundus biomicroscopy for concurrent thickening would then decide that it is clinically significant. I think it is difficult to accurately diagnose edema in the absence of any foveal hard exudates by fundus biomicroscopy alone. Whenever the vision is subnormal and I am unable to clearly pick up CSME on clinical examination, I would be more interested in what the OCT reveals.

I do not perform stereophotography for the diagnosis of diabetic macular edema (except to fulfill some multicentric study protocols).

NV: What is your protocol for investigating DME?

LG: The first investigation is OCT. Fluorescein angiography is not routinely ordered. In selected cases, it is done to exclude gross diabetic macular ischemia. Systemic evaluation includes the lipid profile, renal function tests and hemoglobin estimation since anemia, hypercholesterolaemia and significant nephropathy are all associated with severe maculopathy. OCT is performed almost in every visit as long as the edema is persisting and a decision to treat or watch has to be made.

RK:
1. Optical coherence tomography (OCT) and fundus fluorescein angiography(FFA) only in cases with diffuse diabetic macular edema or recurrent DME.
2. A complete systemic evaluation which includes blood investigations and if the parameters are deranged, then a systemic evaluation by a physician.

AK: Our centres are equipped with OCT and FFA. However, I prefer to do OCT as an initial investigation, owing to its non-invasive nature. I do FFA in patients with diffuse diabetic macular edema (DDME) to look for pattern of leak: focal leak/ diffuse leak and plan my laser treatment. I also do FFA in patients with severe NPDR to look for subtle NVD/NVE. Patient's systemic evaluation is a must. This is dealt in the latter part of focus.

PV: I venture to perform fluorescein angiography and OCT only in patients wherein I suspect edema based on initial fundus biomicroscopy. Exceptions are in patients with subnormal visual acuity with no apparent CSME, in patients wherein media does not permit a reliable fundus biomicroscopic evaluation and in patients who have already received some form of treatment (laser/ injections) at presentation to me.

NV: What is your preference for the initial investigation FFA/ OCT? Why?

LG: As mentioned above OCT is the first investigation and the only one done in a majority of cases. OCT helps to precisely diagnose the type of edema- such as spongy type, cystoid spaces, foveolar detachment etc. It is an excellent tool to compare after any treatment to look for improvement and quantify the same. Presence of epi- macular traction can be identified and appropriately evaluated to see whether surgery would be treatment of choice.

RK: OCT is the first preference, as it is a non-invasive procedure.

AK: We prefer to do OCT as our initial investigation. OCT helps us to categorize the DME morphologically as: spongiform edema, DME with neurosensory detachment, DME with cystoid edema, tractional DME (Vitreo-macular traction syndrome/ epiretinal Membrane). OCT has become an essential part of management of DME in terms of quantification, morphological classification and to monitor the response of treatment. It helps us to selectively pick up cases which require FFA i.e. DME with spongiform edema, DME with neurosensory detachment, DME with cystoid edema and ischemic diabetic maculopathy. I also do FFA in patients with severe NPDR to look for subtle NVD/NVE.

PV: It is well known that FFA and OCT complement one another and so this question is superfluous. At baseline I would prefer to have both an angiogram as well as OCT data. FFA is important at baseline because it helps you detect concurrent macular ischemia as well as early new vessels (particularly in our country wherein it is not a routine practice to grade retinopathy based on seven field fundus photography). Most importantly it helps in detection of unsuspected deep/ diffuse leak in some patients wherein clinically only focal edema is presumed. In the presence of these your approach to managing and prognosticating the situation would be different. This opportunity is lost if you solely rely on OCT at baseline. Contrarily relying on FFA alone may result in not detecting the presence of a fine epimacular membrane or hyaloid traction. This situation also leads to poor decision making.

The greatest advantage of having baseline OCT is that it allows changes to be documented quantitatively and this lessens the need for subsequent angiograms.

NV: What are your recommendations for centers not equipped with OCT?

LG: It is definitely a less than ideal situation in the current scenario. One has to apply the norms we followed in the pre OCT era. Fluorescein angiography is a poor surrogate investigation. The amount of leakage does not correlate with the edema. In the absence of OCT, one may have to rely on clinical judgment with slit-lamp biomicroscopy coupled with whatever information can be garnered from FFA. Obviously improvement can only be judged from the vision.

RK: Clinical examination by slit-lamp biomicroscopy is the gold standard for the detection of DME.

AK: There are many centers in India which are still not equipped with OCT. I would recommend the following (a) Correct estimation of best corrected visual acuity, (b) Through examination with +90D lens. Occasionally, I use Goldmann three mirror lens to look for macular elevation in cases with subtle macular edema and (c) Initial fundus color photography and FFA. For follow-up similar protocol is to be followed and compared with previous visits to monitor the response to treatment.

PV: I am fortunate to be working at a centre wherein both facilities are easily available. Lack of baseline data from these modalities has therapeutic implications. I hence suggest that they team up with colleagues (who have OCT) and have the satisfaction of following current guidelines. This may not apply to patients with minimal focal edema however (these patients, a rarity in our country, could be managed by FFA and follow up fundus photography alone).

NV: What systemic investigations do you order for patients with DME?

LG: As mentioned above one would like to rule out hyper cholesterolemia, anemia and nephropathy.

RK: Blood investigations which include Fasting blood sugar, glycosylated haemoglobin (HbA1c), Lipid profile, Blood Pressure.

AK: Diabetes Mellitus is a multi-systemic disease. We work in conjunction with endocrinologist, nephrologist, cardiologist and neurologist to look for/ screen the other complications secondary to diabetes. We order following systemic investigations: Blood sugar profile (Fasting blood sugar, post prandial bold sugar, glycosylated hemoglobin), blood lipid profile, renal function tests (Blood urea and serum creatinine), hemoglobin estimation and urine for microalbuminuria.

PV: Before ordering investigations I would be eager to know the type of diabetes, any recent attempt at insulin usage (implying poor control) in a previously controlled type 2 diabetic, clarify if proglitazones have been prescribed (check with endocrinologist), any preceding stress (infection/ non-ocular surgery) and recent ocular surgery (usually cataract surgery). I would obtain the HbA1C and blood pressure control records over the last 4-8 weeks. I request for a lipid profile only in patients with

extensive hard exudates. In patients with higher grades of retinopathy I am interested in knowing their renal status as well (creatinine/ microalbuminuria). Anemia may have a bearing only if the hemoglobin values are significantly low.

NV: What are your indications for macular lasers (Focal/ grid)?

LG: In most cases except those with gross edema with whole posterior pole swollen, I do grid or focal laser as the first treatment of choice. If there is no response in 3-4 months, then I consider injection of anti VEGF drugs. In selected cases with gross edema, I sometimes inject anti VEGF drugs first and follow it up with laser once the edema is partially reduced. I believe that a good response with laser always gives a more stable result long term than a good result with anti VEGF drugs.

RK: Focal DME and diffuse DME without severe hard exudates or serous detachments are the indications.

AK: Most of the patients with DME require macular lasers except ischemic maculopathy and tractional DME. I prefer to do FFA, OCT and systemic evaluation before choosing the type of laser treatment. Adequate systemic control is especially renal status is essential prior to laser therapy. Usually I prefer to do macular lasers in the primary sitting, however at times it is preceded by anti-VEGF drugs/ steroids depending on the central macular thickness pattern and morphology of DME on OCT.

PV: I venture to undertake focal laser in the presence of focal leaky microaneurysms (away from FAZ) on FFA and absence of cystic edema on OCT. I use a print out of an early and late frame (on ordinary A4 sheet) as a guide during focal laser.

It is unusual to have diffuse leak on FA without spongy/ cystic retinal changes on OCT. In this situation I prefer some form of local injection followed by laser (focal/ grid), once the edema subsides.

The above procedures are performed only after paying attention to factors discussed in Q.5. It is important to remember that unlike PDR, treatment of macular edema in diabetics is not an emergency (implying that one should allow for control of systemically deranged parameters before deciding on laser).

NV: What are your indications for anti-VEGF agents/PST/ IVTA?

LG: This answer can be taken in continuation of the above. In general I always prefer anti VEGF as the first drug of choice in view of lack of risk of glaucoma and cataract. If however there is no response with previous injections, I switch to steroid injection. In most cases I prefer intra vitreal injection and not posterior subtenon's.

RK: Anti-VEGF agents are used in cases of DME with co-existing glaucoma and in cases recalcitrant to lasers and IVTA

PST steroid is given in cases of DME, who have developed or worsened after PRP or after cataract surgery and in whom there is a component of post –surgical cystoid macular edema.

IVTA is used as a secondary therapy in cases, where focal/ grid laser is not effective. Also primarily in cases laser is not possible due serous detachments or in presence of of hard exudates threatening fovea.

AK: As discussed in the question above I make a choice between the anti-VEGF agents and steroids depending on the central macular thickness pattern and morphology of DME on OCT. I usually prefer to inject anti-VEGF agents for spongiform edema and DME with neurosensory detachment. Anti-VEGF agents are also preferred over steroids in view of lack of cataract and glaucoma following their use. In patients with central macular thickness > 500μ and in patients not responding to anti-VEGF agents I prefer IVTA. However, regular monitoring for intraocular pressure is to be done.

PV: I prefer these agents in two situations

1. Presence of diffuse leakage on FFA and concurrent spongy/ cystic edema on OCT (recheck contribution of systemic factors before decision making)
2. Lack of response to focal laser therapy (such patients usually also have leaky aneurysms close to FAZ)

Despite DRCR results, I still use posterior subtenon injections in patients with moderately increased CMT. You will see useful results not so infrequently even with this approach.

NV: What is your preference IVTA/IV anti-VEGF agents?

LG: See above

RK: IVTA is preferred as the first choice, as it has both anti VEGF and also anti-inflammatory effect.

AK: Discussed in Ans. 6 and Ans. 7. However, I switch to other mode of therapy if there is no response to previous therapy. Some studies in world literature report the worsening of diabetic macular edema (ischemic component) following use of anti-VEGF agents and recommend the use of IVTA in these cases.

PV: As a general rule I prefer IVTA (usually 2mg in 0.05ml) in patients with cystic edema on OCT/FA and anti-VEGF in patients with non-cystic edema. Other than the nature of edema on OCT one needs to weigh in other history such as IOP elevation with earlier steroid therapy, history of cardiac ailment/ stroke (more prevalent in the diabetic population) and presence of concurrent neovascularization. I consider switching from IVTA to IVA and vice versa if the initial therapeutic agent does not achieve satisfactory reduction in CMT.

NV: In what situations you plan combined treatment (laser+ anti-VEGF agents/PST/IVTA)?

LG: See above

RK: We combine laser with IVTA/grid as a second procedure in all cases where primary laser could not be performed.

AK: Discussed in Ans. 6 and Ans. 7. I would like to remind that laser still remains the gold standard for the treatment of DME except in ischemic maculopathy and tractional maculopathy. The use of anti-VEGF agents/ steroids helps to decrease the edema temporarily, for laser to work effectively.

PV: I use a combination of PST and laser (PRP) in patients with CSME and neovascularization. For patients with DME alone, I treat with these agents sequentially based on the response to initial therapy. The most appropriate sequence (not considering focal edema) to me would be intravitreal injection-laser-intravitreal injection.

NV: Which anti-VEGF agent you choose for patients with DME & why?

LG: I give them the options and let them choose based on the cost factor.

RK: Avastin is the choice, as it is affordable for most of our patients.

AK: I prefer avastin and lucentis over macugen. However, amongst the first two agents I let the patient choose based on their affordability.

PV: The options (Bevacizumab/ Ranibizumab/ Pegaptanib) available are discussed with the patient/ family and I allow them to decide. To ensure better decision making by the family, they must also be made aware that repeated injections would be necessary in most situations. Patients who have medical insurance/ reimbursement facilty usually opt for Ranibizumab. Herein again I think initial pan-VEGF injection followed if necessary by selective VEGF injection may be safer in the long run compared to continuous pan-VEGF inhibition.

NV: What are your views regarding cocktail regimen (Anti-VEGF agents+IVTA)?

LG: I do not have much experience with combining both in one injection session.

RK: Cocktail regimen can be used in some cases of DME recalcitrant to laser+ IVTA/VEGF combo.

AK: This is one of the latest forms of treatment in vogue for DME world over. It offers the dual advantage in terms of immediate/ early reduction in DME (anti-VEGF effect) and sustained effect (due to triamcinolone acetonide). The dose of steroid given is 2mg. I have tried cocktail regimen in a few cases of recalcitrant edema with good results. The dose of 1mg IVTA also gives satisfactory results.

PV: I have never treated any patient with a cocktail regimen. I am not convinced of its necessity.

NV: How do you follow-up patient of DME?

LG: 3-4 monthly unless the condition is very stable- in which case 6 monthly or longer.

RK: In cases where focal/grid laser has been done, the patients are followed up after 4 months.

In case of IVTA, we review the patient after a week to check for the IOP.

In cases where anti-VEGF or IVTA was injected, the patients are followed up every month, until the edema resolves.

AK: I follow up my patients of DME as: (i) with focal/ grid laser: at 3 months, (ii) with anti-VEGF agents: Day 1 to look for anterior chamber reaction and intraocular pressure (IOP) and at 6 weeks and (iii) with IVTA: Day 1 to look for anterior chamber reaction and intraocular pressure (IOP) and every week to monitor IOP. I repeat all the investigations i.e. systemic investigations and ocular investigations (Color fundus photography, OCT and FFA if required) at 3months for first group and for second and third group at 6 weeks. Then I compare them with previous visit to assess the response to treatment and to decide whether any additional treatment is required.

PV: If I have undertaken only conventional laser, I request follow up OCT at 6 weeks and 3 months. At these visits I also take photographs of field 2 and compare with baseline images. If I have performed subthreshold micropulse diode laser I would wait for 4-6 months before considering re-treatment/ alternate treatment.

In cases of inadequate response or worsening edema (particularly in both eyes) one must again assess the possible contribution of systemic factors (as discussed earlier).

With longer intervals of follow up for DME, one must not forget to look for evolution of features of proliferation.

When intravitreal injections have been used, I see them on day 1, day 7 and day 28. Day 1 and day 7 are essentially to pick up unexpected inflammation/ infection early (patient is advised to visit the eye casualty immediately if he develops features of pain, blurred vision). I request a follow up OCT at week 4. Patients given intravitreal steroids also need long term monitoring for elevation of IOP.

NV: What is recalcitrant DME? What are the causes do you attribute for recalcitrant DME?

LG: Recalcitrant DME is one that does not respond at all to any of the modalities of treatment such as photocoagulation, anti VEGF or steroids. The reasons could be sick RPE, systemic associated problems that could not be corrected such as nephropathy, pre retinal traction.

RK: Recalcitrant DME is that DME persisting even after maximum laser therapy. The causes are poor systemic control (poor glycaemic control, uncontrolled hypertension, uncontrolled nephropathy, severe hyperlipidaemia) and chronic DME leading to cystoid macular degeneration.

AK: Recalcitrant DME was the term which was coined for the DME unresponsive to laser treatment. However in present era, we call DME recalcitrant if it is unresponsive to all modalities of treatment i.e. laser, anti-VEGF agents and steroids. The reasons for recalcitrant DME could be: uncontrolled systemic condition, tractional DME and ischemic maculopthy.

PV: Recalcitrant DME may be defined as lack of anticipated response to appropriate intervention (e.g. of focal laser

in focal edema). Earlier, failure of response to two laser attempts was bracketed as recalcitrant edema. With the advent of other therapeutic options the definition is now become blurred.

Causes for recalcitrant DME include

a) Inappropriate laser technique (related to choice/ placement/ power)

b) Aggressive PRP (with very short duration between sessions) in the presence of even mild CSME

c) Failure to detect concurrent taut hyaloid/ epiretinal membrane/ vitreomacular traction before initial intervention

d) Evolution of Epiretinal membrane/ traction following intravitreal injection (of IVTA and anti-VEGF respectively)

e) Worsening systemic condition (poor diabetes/ blood pressure control; onset of microalbuminuria)

f) Complicated intraocular surgery (usually phacoemulsification)

g) Worsening grade of retinopathy

h) Long standing DME

i) Rapid lowering of blood sugar/ control of diabetes using Insulin

j) Use of Proglitazones

k) Idiopathic

NV: How do you management recalcitrant DME?

LG: Pre retinal traction can easily be corrected with surgery. Patient can be encouraged to have better control of the systemic status if possible. We do understand that there are a set of patients with severe nephropathy and anemia (bordering on the need for renal transplant) who are best watched if all avenues have been exhausted. In select cases one may try surgery with ILM peel even if the OCT does not show visible significant traction.

RK: If the systemic parameters are deranged, those are addressed first.

A cocktail regimen can be repeated. Cases with cystoid macular degeneration, are only observed.

AK: The recalcitrant edema reduces/ resolves partially if co-morbid conditions like hyperlipidemias, hypertension and diabetic nephropathy are kept under proper check. This is further supplemented if adequate blood sugar control is achieved. However, tractional form of DME (VMT/ ERM) is amenable to surgical treatment i.e. pars plana vitrectomy with membrane peeling. Correction of anemia could help in otherwise unresponsive ischemic form of DME. If patients with chronic kidney disease who are advised to undergo renal transplantation, macular edema resolves subsequent to transplantation procedure. The pioglitazone group of drugs should be avoided in patients with recalcitrant DME.

PV: Consider each of the above factors and address them whenever possible. One could switch from one option (e.g. consider IVTA if anti-VEGF does not show results) to another.

NV: What is the role of surgery in patient with diabetic maculopathy?

LG: Some of this has been covered in the previous question. In the presence of visible traction- surgery is the treatment of choice with or without intra vitreal injection of anti VEGF drugs or steroid same time. In the absence of significant traction, the role of surgery is questionable but can be tried in selected cases where all other forms of treatments have been tried and the RPE does not look that sick. Hard exudates under the fovea can be sucked out through a Retinotomy but the RPE in these cases is usually unhealthy and the visual results are not good. However although one may not perform surgery for these exudates, if incidentally surgery is being done for other reasons, there is probably merit in removing the sub retinal exudates same time.

RK: It is reserved for cases with VMT or taut posterior hyaloid causing traction.

AK: Surgical treatment of DME picked up with the advent of OCT technology. Pre-retinal traction secondary to VMT or ERM is perfect indication for surgery. However, I do not anticipate drastic improvement in vision of patients with documented tractional DME for more than 6 months. Recalcitrant DME with collection of hard exudates at fovea could respond better to parsplana vitrectomy with ILM peeling as it helps in faster resolution of exudates. Some surgeons also advocate removal of hard exudates/ long standing cholesterol crystals if deposited in the macular region (crystal aspiration).

PV: Surgery is traditionally recommended in patients with documented foveomacular traction/ taut hyaloid/ epimacular membrane. It is said to be useful for patients with recalcitrant edema even in the absence of the above OCT features.

Patients in whom I have performed surgery for taut hyaloid and vitreomacular traction have shown long term resolution of macular edema and marginal improvement in vision. I do not perform surgery in the presence of only a fine epimacular membrane and when OCT fails to show any vitreomacular anomalies.

NV: What are your surgical results?

LG: In the cases with pre retinal traction, the results are satisfactory. It is obviously not comparable to idiopathic ERMs. Recurrence of DME is not uncommon despite initial resolution after surgery even without recurrence of membranes. In eyes with sub retinal exudates, the visual results have not been satisfactory despite the anatomical result being OK.

RK: Surgical results are good in cases of Vitreomacular traction. The results are poor in chronic cases and in cases associated with macular ischaemia.

AK: The resolution of DME is achieved once the tractional component has been removed. However, the edema may reoccur if patient's systemic parameters are not under control. In addition, ILM peeling also helps in faster resolution of edema.

PV: The best result I have seen is a visual improvement from 4/60 to 6/36 after removal of taut posterior hyaloid. This patient has maintained resolution of macular edema after surgery with no supplemental intervention.

NV: How do you approach patient with cataract and DME?

LG: If the retina is well ablated and the eye is relatively stable (without florid disease or NVI), I will combine phacoemulsification with IOL implanatation along with the VR surgery same time. If however, the condition is not that stable, I would avoid cataract surgery same time if it is permissible (i.e the cataract is not interfering with satisfactory completion of VR surgery). In bad cases where in silicone oil etc is used, I tend to combine cataract surgery with IOL implantation along with the silicone oil removal at which stage the retinal condition is likely to be more stable and the retinal ablation would have been well done.

RK: If the view is good Focal /grid laser is given first and cataract surgery is planned after a month.

In cases with dense cataract, the cataract surgery is done first followed by laser after 1 month or IVTA.

AK: I prefer to treat the edema if the media is clear and poses no danger for laser treatment and postpone the cataract surgery for 2-3 months. However, if the cataract is significant I go ahead with phacoemulsification and explain the patient of worsening of DME post cataract surgery. I deal with DME 6 weeks following cataract surgery. In addition if DME is severe I prefer to combine my cataract surgery with intravitreal anti-VEGF agents/ steroids.

PV: I document the nature of DME on FFA and OCT. I find the FFA useful even in situations wherein OCT is unable to capture images with adequate signal intensity. In cases with focal edema, I would follow the standard recommendation of performing focal laser before cataract surgery (if media allows adequate visualization). In patients with diffuse/ recalcitrant DME I would prefer to treat with intravitreal agents, note any change in vision and OCT values before recommending cataract surgery. In those with significant cataract related haze, I recommend phacoemulsification followed by repeat FFA/ OCT to decide on the choice of treatment. I discourage use of any posterior subtenon/ intravitreal injections at the end of cataract surgery.

Phacoemulsification must be performed only when systemic control in adequate and by a surgeon with greater expertise (one who can consistently place the IOL in the bag and also have a low rate of capsular opacification, even over longer periods of follow up).

NV: How you manage patient with worsened DME post cataract surgery?

LG: I would like to see whether there is an added component of Pseudophakic CME. I do understand that it may be difficult to identify these two components apart in a given clinical situation. One can try sub tenon's or intra vitreal steroid which can have benefit under both circumstances.

RK: In cases with severe DME, IVTA or anti -VEGF are used. In mild to moderate DME, PST or the newer topical NSAID Nepafenac eye drops are used.

AK: The worsening of DME following cataract surgery is a well established fact. I usually prefer to wait for 4-6 weeks following cataract surgery. At the end of 6 weeks I do OCT evaluation of DME and plan my treatment on the basis of central macular thickness and morphological profile of DME on OCT.

PV: The usual recommendation was to just follow up these eyes for 6 months before considering any intervention (as surgery induced edema is known to resolve naturally over a period of time). I would however treat such eyes earlier if vision drop is significant and OCT shows severe CMT worsening.

NV: How do you diagnose and manage ischemic maculopathy?

LG: Diagnosis of ischemic maculopathy can be suspected clinically from biomicroscopy but the definitive diagnosis is by fluorescein angiography. One should also differentiate between gross ischemia from mild ischemia, because some amount of enlargement of FAZ is very common and should not deter us from trying the treatment options. In cases with gross ischemia, it is unlikely that any treatment will benefit.

RK: Ischaemic maculopathy is diagnosed by fundus fluorescein angiography(FFA) and the criteria are irregular and enlarged foveal avasular zone.

No treatment is required unless CSME is associated.

AK: Ischemic maculopathy is a diagnosis confirmed on FFA. However, I suspect ischemic maculopathy clinically if :(a) the vision of patient is not corroborating with the fundus findings, (b) featureless macula on slit-lamp biomicroscopy and (c) recalcitrant DME after tractional and exudative forms of DME have been excluded. I usually recommend OCT in patients with ischemic maculopathy to look for subtle element of mixed form of maculopathy which is amenable to treatment. Ischemic maculopathy itself is usually unresponsive to any form of treatment.

PV: I confirm a clinical suspicion of macular ischemia using FFA. If there are associated cystic changes I consider a trial of IVTA.

NV: How you approach patient with PDR and DME?

LG: In an unlasered eye, I will first differentiate between the severity of PDR Vs severity of DME. If there are subtle NV and significant DME, I will treat the macula first and start the PRP after 2 months. If however the PDR is also significant, I will treat the macula and along with it start the PRP. The Temporal sector is avoided and the same is filled up after 3-4 months. If there is severe DME necessitating anti VEGF drugs, I will inject the same and continue with PRP. The injected drug could be beneficial to both. Macular grid can be done once the macular edema responds to Anti

VEGF drugs.

Going by the recent publications injecting anti VEGF routinely along with laser seems to be a good option in DME.

RK: In a patients with early PDR and DME, focal laser is done first and PRP is done 1 month later in multiple sittings.

In cases of severe DME, PRP is combined with IVTA.

AK: I prefer to treat DME and PDR with high risk criteria of DRS in the same setting. I space out the pan retinal photocoagulation (PRP) over 3-4 sittings, each session at an interval of 4 days-1 week. In the first sitting, I do macular lasers (focal/grid) and combine them with PRP in the nasal/infero-nasal quadrant. This is followed by PRP in the superior and inferior quadrants. The temporal PRP done in the last sitting. For patients with DME with PDR (non HRC) I do macular lasers initially and start PRP after 2 weeks. However, if the DME is severe and the new vessels are flat, I combine laser with anti-VEGF agents. I inject anti-VEGF agents followed by PRP over 2-3 sessions. I do macular laser within one week of injection (usually with the second sitting of PRP).

PV: In patients with early PDR and macular edema, I do not perform PRP. I undertake focal laser to leaky microaneurysms (in the absence of cystic changes) and scatter laser confined only to the sector carrying the new vessel. In those with cystic edema I give a posterior subtenon injection followed a week later by scatter laser to a defined region. I avoid using intravitreal drugs in patients with early PDR.

In those with PDR and HRC I prefer to first treat with an intravitreal anti-VEGF agent. About a week later, I combine macular laser with first session of PRP. I then complete PRP with two more sessions spaced over 10-14 days.

With PASCAL laser it may be possible to treat these eyes in one session. I however am yet to gain experience with this laser system and so would be ill suited to comment on the same.

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Botulinum Toxin for Oculofacial Aesthetic Rejuvenation

Poonam Jain *MS*

Injection of Botulinum toxin for aesthetic purposes is one of the most common cosmetic procedures performed today. It has become gold standard in non surgical facial rejuvenation for eradicating dynamic wrinkles of the face. As dynamic wrinkles predominantly occur in the periocular region, Botox is a useful adjunct in the armamentarium of the ophthalmologist. It is safe and easy to perform if one adheres to the basic principles of therapy and has a thorough understanding of facial and orbital anatomy and muscular interactions.

Introduction

Botulinum is a neurotoxin derived from the gram- negative anaerobic bacterium Clostridium botulinum. The neurotoxin interferes with acetylcholine release from peripheral cholinergic nerve terminals, causing temporary paralysis of the injected muscles. There are seven distinct serotypes of botulinum toxin that have been identified but only serotype A is commercially available for cosmetic and clinical use. Serotypes B and F have been investigated for use in cervical dystonia and blepharospasm.

Botulinum toxin was first used in 1973 and 1979 for the treatment of strabismus and in 1985 for blepharospasm and dystonia. In 1989, it gained US FDA approval and ophthalmologists began using it to treat blepharospasm, strabismus and hemifacial spasm. Aesthetic indications were added after patients noticed that treatment in the brow area produced a relaxed, unworried appearance.

Preparation of injection

The formulation is available in lyophilized form and must be reconstituted with physiological preservative free saline prior to use. It is available as 50 or 100 U vial.

The toxin quickly loses effectiveness if it gets too hot or cold during transport and storage. It is therefore essential to maintain an unbroken cold chain for the vials from the point of manufacture, during transport and during storage in the freezer of a refrigerator until they are used. After reconstitution, it can be stored up to 6 weeks at 4°C in the refrigerator.

The appropriate diluent volume is selected based on the concentration desired. Although volumes of 1cc to 10cc have been used with varying results to dilute100cc of Botox, pack insert recommendation and consensus of experts is 2.5cc dilution making it 4 units of Botox per 0.1 ml. The vial if intact, contains a vacuum that will automatically withdraw the saline from the syringe when the diluent is injected into the vial.

1 ml tuberculin syringe or insulin syringe with a 30 G needle is used for injecting the precise amount.

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Change the 30 gauge needle after every 4 - 5 pricks. A sharp needle is less painful.

Use ice pack to chill injection sites, to reduce pain of injection. Topical anaesthetic cream can also be applied 20 to 30 minutes before the procedure in sensitive patients.

The patient may be seated or lying while injecting depending on the choice of the surgeon but it is best to plan and mark the injection sites while they are seated so as to observe the patient's natural facial expressions as the fat pads and skin folds fall naturally.

Cosmetic Use of Botox in upper face

The muscles of the human face can be divided into the more superficial mimetic muscles (muscles of facial expression) and deeper muscles of mastication (chewing muscles). It is the mimetic muscles that are targeted during treatment for facial wrinkles. Botox injection works by relaxing the muscles of facial expression, thereby removing the dynamic wrinkles, creating a smoothened, rejuvenated, more youthful face. The effect becomes visible 1-2 days after the injection and generally lasts for 4-6 months. Retreatment can be offered after the initial effect has worn off.

Successful use of Botox depends on a thorough understanding of the actions and anatomy of the muscles controlling facial expression. (Figure 1). One must understand not only the primary function of each muscle but also the interplay and interactions with the antagonistic muscles. (Table 1)

The three most common areas treated in the upper face are the glabella, forehead and lateral canthus for the wrinkles in the respective regions.

Glabellar Frown Lines

Glabellar lines or "frown lines" are vertical creases seen adjacent or in between the medial aspects of the eyebrows. These are often the first wrinkles of concern to the middle aged patient.

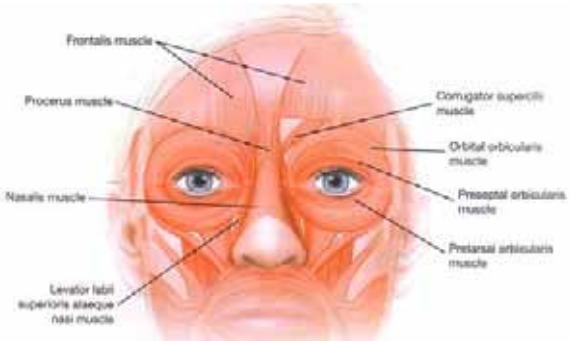

Figure 1: *Mimetic muscles are superficial muscles and are responsible for controlling facial expressions*

Table 1: Actions of Facial Muscles		
Muscle	**Action**	**Expression Lines**
Frontalis	Brow elevator	Horizontal
Corrugators	Pull eyebrows medially	Frown lines
Procerus & Depressor supercilii	Brow depressors	Frown lines & Horizontal nasal lines
Orbicularis oculi	Brow depressors- Especially laterally	Crows Feet

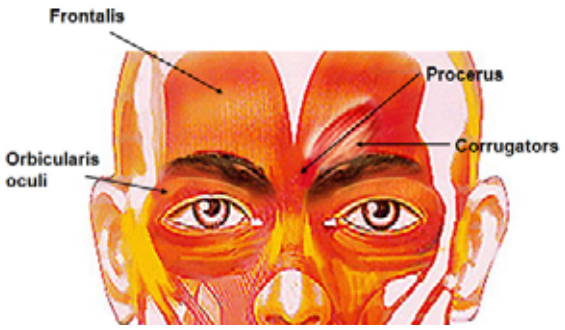

Figure 2: *Target muscles for botox treatment*

The muscles of facial expression that contribute to frown lines include the corrugator supercilli and the medial orbital portion of the orbicularis oculi muscles and the procerus and depressor supercilii muscles. (Figure 3)

These muscles are termed the "glabellar complex."

Target Muscles -

Corrugator supercilii and orbicularis muscles move the eyebrow medially

Procerus and depressor supercilii pull the eyebrows inferiorly

Injection sites: 1 in the procerus and 2 in each corrugator (Figure4).

These are the general guidelines. The injection sites have to be customized after noting which muscles are being recruited.

Range of doses: 12.5 – 30 units (2.5 to 5 U per puncture)

Technique

- Place injections in a V- like pattern but do not pass midpupil to prevent brow flattening
- Inject deeper, at the intramuscular level, but avoid hitting the periosteum to avoid causing pain.
- The injections can be perpendicular or tangential into the muscles. Pinching the muscles makes it more comfortable for the patient and prevents diffusion into the orbit. (Figure5)
- Stay at least 1 cm above the orbital rim to reduce the risk of brow ptosis

Horizontal Forehead Lines

Target Muscles

Frontalis muscle which is the sole brow elevator. Contraction of the frontalis muscle is responsible for the elevation of the brows and also induces horizontal wrinkles across the forehead, the so called 'worry lines'.

Injection sites: 4-10 sites , 2.0 – 2.5 U per site (Figure 7)

Range of doses: 10- 25 U (women), 15 – 30 U (men)

Technique

- It is important to recognize that the lower two centimeters of the frontalis is largely responsible for elevation of the eyebrows

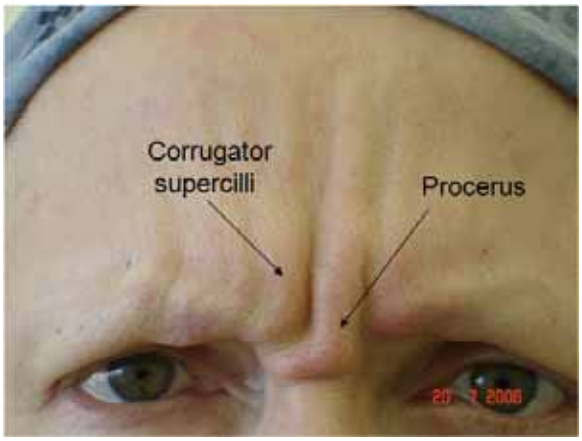

Figure 3: *In treating the glabellar frown lines, we are focusing on the corrugators and the procerus*

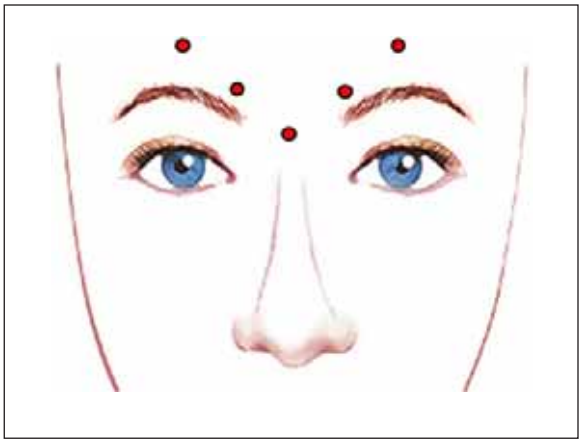

Figure 4: *Frown Lines - Injection Sites*

Figure 5: *You may pinch the corrugator and procerus over the orbital rim to protect against BOTOX® diffusion into orbital rim*

Figure 6: *Glabellar Frown Lines & Forehead Lines*

- therefore, stay at least 2 cm above the orbital rim to avoid causing brow ptosis (Figure 8)
- inject in between wrinkles within the interpupillary area in a gridlike pattern
- the depth of the injection is subcutaneous
- avoid central raphe
- Don't over-treat: leave some facial expression
- Look for pre-existing brow Ptosis (which the patient may be elevating their frontalis to compensate for)
- Look for any brow asymmetry and tailor the doses and sites accordingly.

Crow's Feet

Target Muscle - Lateral Orbicularis oculi

Injection sites: 2- 4 each side, 2.5 – 4.0 U per site (Figure 10)

Range of doses: 12 – 15 U each side

Technique

- Injection sites should be 1 to 1.5 cm (one fingerbreadth) from the orbital rim
- Level of the injection is immediately subdermal, whereby a wheal is observed with each puncture. (Figure 11)
- Inject in between the wrinkle lines.

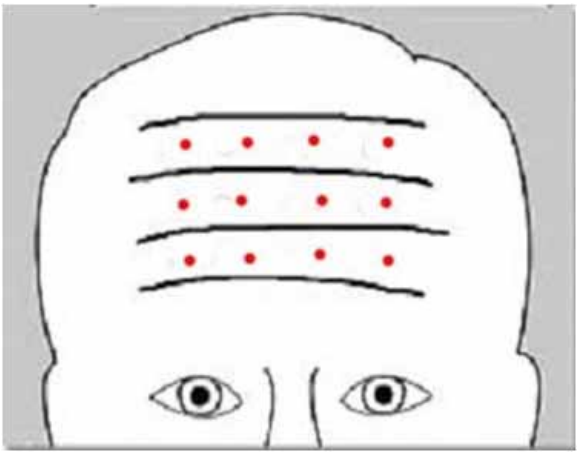

Figure 7: *Approximate injection sites for horizontal forehead lines*

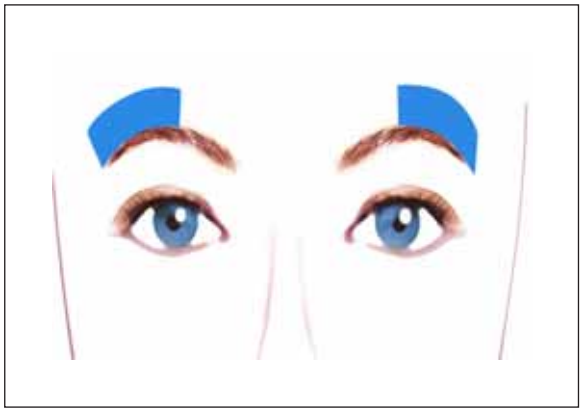

Figure 8: *To minimise brow ptosis risk, avoid injecting upto 1cm above the brow, lateral to the mid-pupillary line (the shaded area)*

- Ask the patient to smile and distribute the injection sites accordingly. A second row of injections can be added if the wrinkles extend fairly laterally.
- If the patient's crows feet extend under the eye, one may continue injections around to the mid- pupillary line in small doses, but do not be tempted to move down into the cheek area due to the risk of injecting the zygomaticus muscle and causing lip ptosis.

Other Oculofacial Uses

With more experience and high patient acceptance, use of Botox continues to expand to many other cosmetic and functional indications, such as

- hyperlacrimation
- eyelid retraction
- spastic entropion
- migraine/ stress headache
- improving symmetry in facial palsy
- brow arching and browlift, called chemical browlift

Adverse Reactions

The most common untoward reactions are related to a wider than expected local paralysis. Ptosis of the upper eyelid can occur as

Figure 9: *Horizontal forehead lines*

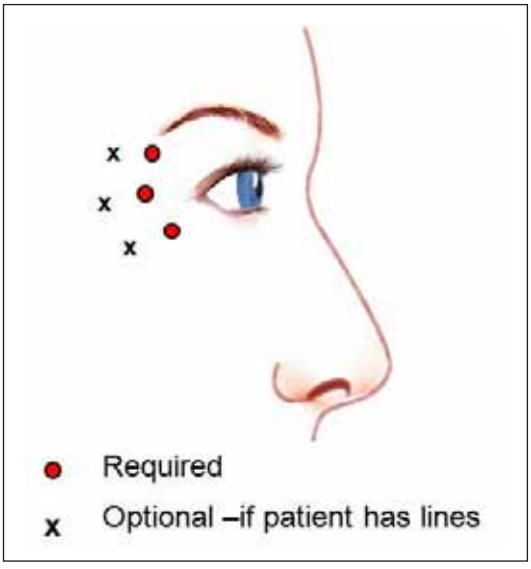

Figure 10: *Crow's Feet - Injection Technique the injection sites follow the curve of the orbicularis oculi*

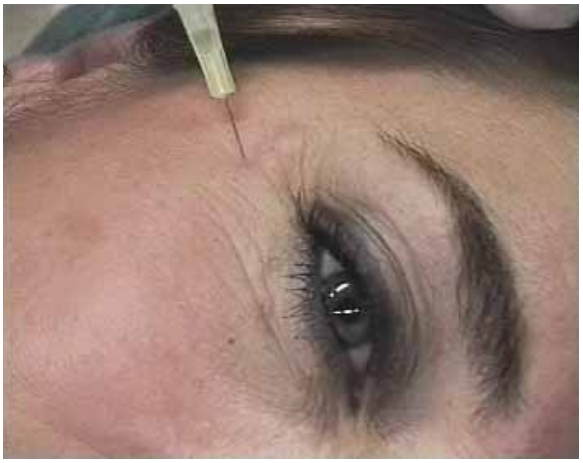

Figure 11: *Inject superficially into the orbicularis muscle in between wrinkles*

Figure 12: *Crow's feet*

discussed above. Diplopia may occur if the botox penetrates the orbital septum. It is usually transient and resolves spontaneously. General reactions such as headache, flu-like symptoms, pain and nausea have infrequently been reported.

There are no systemic risks from injecting directly into a blood vessel by accident.

Contraindications

- Pregnancy
- Lactation
- Allergy to human albumin

Relative Contraindications

- Disorders of neuromuscular junction: Myasthenia gravis, Eaton Lambert syndrome
- Disorders of muscle weakness: multiple sclerosis, amyotrophic lateral sclerosis,
- Concomitant use of aminoglycoside antibiotics, quinine, penicillamine.

Conclusion

Treatment of facial wrinkles through the use of Botox has revolutionized the treatment of facial aging. It is highly effective in treating a wide variety of hyperfunctional facial lines. It is easy to use, well tolerated by patients and extremely safe. Performed as an OPD procedure .Effect is rapid and does not require time away from work or social activity. Knowledge of muscular anatomy and physiology, awareness of potential complications and appropriate injection techniques are keys to the successful use of Botox, the treatment being rewarding for both the patient and the treating doctor.

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Not Just Cosmetic
Botulinum Toxin for Strabismus

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Botulinum toxin (marketed as Botox®) is the buzzword for cosmetic surgeons, but it was first used and approved to treat strabismus. Botulinum toxin has become recognized and accepted as both an adjunct and alternative to strabismus surgery in many types of strabismus.[2] First described by Scott the toxin temporarily paralyses the extra ocular muscle resulting in a changed ocular alignment that resolves over time (usually a two to three month time interval).[3] During this period of altered eye position, the visual axes may adopt an ocular alignment that permits binocular single vision.

How It Works?

Botulinum toxin is an exotoxin of a bacterium clostridium botulinum. Botulinum toxin selectively blocks the release of acetylcholine from the cholinergic synapses found within a muscle, therefore blocking the nerve impulses and preventing contraction of the muscle cells.

Binding

The heavy chain portion of the active ingredient in BOTOX binds to the cell membrane of the motor nerve via an unidentified high affinity "acceptor" molecule. This high-affinity binding action allows for efficient uptake of BOTOX by the motor nerve and facilitates selective, targeted treatment at the injection site.

Internalizing

After binding, the BOTOX protein molecules pass through the cell membrane of the motor nerve and into its cytoplasm via a process called endocytosis. It is here that the enzymatic component (light chain) of the BOTOX protein molecule is activated.

Blocking

Inside the motor nerve, the light chain of the BOTOX protein molecule cleaves apart a protein (called SNAP25) that enables vesicles which store the neurotransmitter acetylcholine to attach to the cell membrane. Cleaving SNAP25 prevents these vesicles from fusing with the membrane and prevents the release of acetylcholine into the neuromuscular junction (the space between the motor nerve and the muscle). Thus, nerve impulses that control muscle contractions are blocked decreasing muscle activity.

Paralysis (which is temporary) follows within days after injection of the toxin into the extraocular muscle, and the toxin becomes fully effective within three to seven days of the injection. The duration of paralysis is dependent on the individual, but generally lasts for three months.

In cases of paralytic strabismus it prevents contracture of ipsilateral antagonist and improve ocular motility thus reduces diplopia, increase binocular field and correct abnormal head position.

Uses

Botulinum toxin is being used both as diagnostic tool and therapeutic purposes.

Diagnostic

- Investigation of postoperative diplopia
- To detect whether fusion is present preoperatively

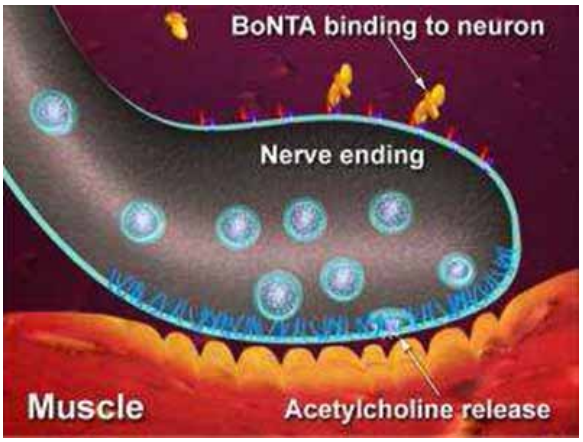

Figure 1: *Binding*

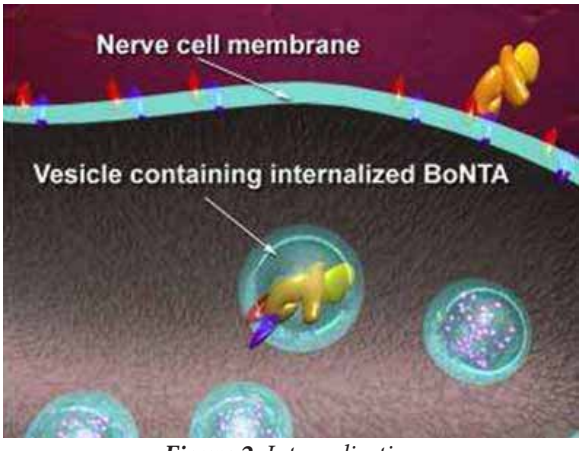

Figure 2: *Internalisation*

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Figure 3(a): *Pre injection,*
3(b),(c),(d): *Post injection (case no. 1)*

- To differentially diagnose between a part and complete sixth nerve palsy
- To aid in the prediction of surgical results for incomitant deviations

Therapeutic

- To restore fusion in those patients with decompensating deviations
- Paralytic strabismus
- To aid surgical overcorrections and undercorrections
- Augmentation of strabismus surgery
- Post RD surgery strabismus
- Nystagmus

Some examples

Case No.1 (Figure 3a,3b,3c,&3d)

62yr female C/o sudden onset double vision with in turning of left eye for past one week. She was suffering from hypertension & diabetes mellitus for past 3yrs, which were controlled on medications. On examination, CT for distance and near- Left Esotropia and PBCT for distance-14pd, for near 16pd.limitation of movement in left eye on levoversion. She was diagnosed with left sixth nerve palsy and investigated. All investigations were normal including MRI. To relieve her diplopia inj. botox was injected in left medial rectus and after 2 weeks she was orthophoric and had full ocular movements.

Case No. 2 (Figure 4a,4b,)

25 year old female with intermittent outward deviation of left eye had been operated for squint twice and now didn't want surgery. She measured 35 pd for distance, 25 pd for near left exotropia. Injection botox was injected in lateral rectus. She remained ortho for distance and near for 3 months and injection was repeated.

Case No. 3 (Figure 5a,5b)

A 56 yr old man came with double vision of sudden onset for 1 week. He was known case of hyperthyroidism. On cover test he had small angle right esotropia of 15 pd. There was mild limitation in abduction in the right eye. Investigations were done and he was given option of prisms. He was injected Botox in right eye medial rectus. He was orthophoric after a week.

Injection technique: Any of the techniques can be used:

- Under EMG guidance directly in to the muscle
- Subtenon's over the muscle

Figure 4(a): *Pre injection*
4(b): *Post injection (case no. 2)*

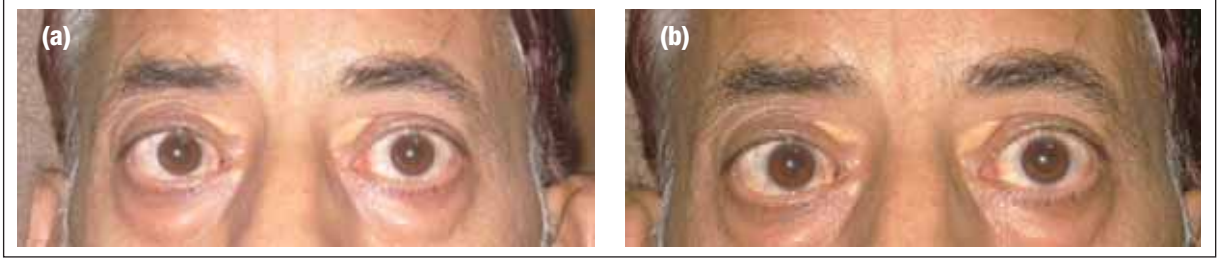

Figure 5(a): *Pre injection,* ***5(b):*** *Post injection (case no. 3)*

Figure 6: *Subconjunctival Haemorrhage*

- Opening up the conjunctiva (done in Operation Room)

Advantages

- Can be used as an OPD procedure.
- It leaves no scar

Disadvantages

- More than one injection may be needed as dose effect relationship is still unknown
- Alignment may not be stable

Adverse Outcomes

Most common complications are transient ptosis, induced vertical deviation and subconjunctival hemorrhage. The overall complication rate range from 8.4% to 55.54%.[4,5,6]

Subconjunctival hemorrhage after injection Botox in Medial rectus (Figure 6).

Conclusions

Botulinum toxin has been shown to achieve levels of binocular vision comparable to surgery in cases of acute onset esotropia, sixth nerve palsy and infantile esotropia. We have found it useful in cases of small angle strabismus, residual strabismus and particularly sixth nerve palsy, where it helps in resolution of diplopia and increases field of vision thus making quality of life better for the patient. It can be considered as an independent treatment option.

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Refractive Lens Exchange

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The idea of removing the clear crystalline lens in highly myopic and hypermetropic patients for refractive purposes is not new. Refractive lens exchange (RLE) was first described by Boerhaave in 1708. RLE is also commonly known as Clear Lens Extraction. Austrian surgeon Fukala and his French counterpart Vacher are credited with the first reported series of lens removal in highly myopic and hypermetropic patients. A retrospective study of 10 years apparently showed unacceptable long-term complications. This led to the abandonment of the technique until 1980. A few surgeons continued in small numbers to treat high myopia in this way throughout the 1980s and the early 1990s. A long-term study of a group of highly myopic patients reported in 1999 by Colin et al.[1] apparently showed 8% rate of retinal detachment (RD), approximately double that expected in a similar population group not having lens exchange surgery. But recent studies have shown encouraging results. A meta analysis of papers on RLE and cataract in high myopia done by Richard Packard[2] between 1996 and 2004, which included 2036 eyes and mean follow up of 43.5 months showed that the overall incidence of retinal detachment was only 1.85%. In recent past, RLE is again being accepted as viable alternative to other refractive procedures. RLE is appealing because this procedure is not technically difficult for most of the ophthalmologists, and good visual outcome can be achieved with very few complications.

Indications

- Myopia when other refractive procedures are contraindicated (if the cornea is too thin, too flat or too steep etc.).
- Very high myopia , beyond correction by other refractive procedures (usually myopia >8 D; low cost alternative to Phakic IOL)
- Hypermetropia when other refractive procedures are contraindicated (due to insufficient AC depth).
- Very high hypermetropia when other refractive procedures would give an inadequate result (Hypermetropia >5D; low cost alternative to Phakic IOL)).
- High hypermetropic patients of both pre-presbyopic and presbyopic age groups are probably the best candidates because of the smaller risk of postoperative retinal detachment and the fewer modalities available to treat patients with high hypemetropia.
- High myopia or hypermetropia along with minimal lens opacity that is presently visually insignificant but may eventually progress and cause visual loss.
- Poor patients, in spite of being good candidate for other refractive procedures (an important indication in developing countries like India).

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Contraindications

- Very young patients (pre-presbyopic age group patients are considered a relative contraindication).
- Axial length of >29mm.
- Presence of chorioretinal degeneration, retinal tear, nanophthalmos etc[3].

Pre-Operative Work-Up

- Visual acuity and Best corrected visual acuity.
- Proper slit lamp examination.
- Intraocular pressure.
- Direct and Indirect Ophthalmoscopy, to look for degenerative changes in retina.
- Keratometry.
- A-Scan.
- Informed consent to be taken.

Intra Ocular Lens (IOL) Power Calculation

High expectation for excellent uncorrected visual acuity makes accurate IOL power calculation more critical than it is in a routine cataract surgery. IOL power calculation formulae are less accurate for high myopia and hypermetropia. Also, in high myopic patients, posterior staphyloma can make axial length measurement inaccurate. IOL power calculation is preferably done by Immersion A-Scan. Immersion vector A-Scan is even more accurate. If available, IOL Master is superior and yields an accuracy of significantly less than ± 0.25 D. SRK-T formula is considered to be most accurate in moderate and high myopic patient, whereas Hofffer-Q and Holladay are most accurate for moderate and high hypermetropia.

Many surgeons believe that an IOL should always be implanted after RLE, even with little or no optical power in high myopic eyes. Plano IOLs should be implanted in these patients, which acts as barrier to anterior prolapse of the vitreous in case Nd:YAG laser posterior capsulotomy is required. IOLs with posterior square edge design also prevent the development of PCO.

Which IOL to be Implanted

Multifocal IOLs have the advantage of providing both near and far vision without the dependence on spectacles. This is especially important among myopes of pre-presbyopic age group who often remain unsatisfied in spite of gaining good distance vision, because they lose their accommodation. Side effects of multifocal technology including unwanted photic phenomena and deterioration in contrast sensitivity are being further defined and evaluated to better assess the effects of these intraocular lenses on functional vision and patient satisfaction.

Accommodative IOLs are also a good choice. The first FDA-approved accommodating lens is available now. It maintains binocular function and also avoid the unwanted mesopic and scotopic visual disturbances that are experienced with multifocal lens technologies.

Monovision is another alternative to provide both near and far vision, in which the dominant eye is made emmetropic and the other eye is made myopic of -1.00 to -1.50 D. A contact lens trial can be given to the patients prior to surgery to evaluate the acceptance of monovision. Impairment of stereoacuity is of concern in monovision.

Surgical Technique of RLE

Under topical anaesthesia, a clear corneal incision or a sclero-corneal tunnel is made, depending upon surgeon's preference. If peribulbar injection has to be given, care should be taken to avoid perforating the larger and softer myopic eyes. Two side-port incisions are made to facilitate proper bimanual cortical cleaning. With the help of 2.8 mm keratome, anterior chamber is entered. Ophthalmic viscosurgical device (OVD) is injected into the anterior chamber and a continuous curvilinear capsulorhexis of 5.0–6.0 mm size is made. In high axial myopia, the tendency of excessive deep anterior chamber can be minimised by avoiding OVD-overfill and by lowering the bottle height. Copious hydrodissection is done in order to lift the nucleus out of bag. It is then rotated and prolapsed completely out of bag. Phaco power is kept at minimum. Mostly, much of phaco power is not required for these soft nucleus and the nucleus is aspirated by phacotip. Cortical matter is properly aspirated. Thorough posterior capsular polishing is done to prevent posterior capsular opacity in future. Foldable IOL is implanted in the bag. Optic of IOL should be 6.00 to 6.50 mm to facilitate future retinal examination. Large optic also allow wide YAG laser capsulotomy without exceeding the IOL edge, thus avoiding anterior movement of vitreous ,which is an important factor for retinal detachment in high myopic patients. OVD is removed thoroughly. Stromal hydration is done at the side-port entries. Sterile pad and bandage is applied at the end of the procedure. Pad is removed after 6 hours and topical antibiotic and steroid drops are started.

Post-Operative Follow-Up

Following parameters should be monitored at every visit:

- Any complaints, for example diminution of vision, pain in eyes, floaters or flashes of light.
- Uncorrected and Best corrected visual acuity.
- Refraction.
- Intraocular pressure.
- Slit-lamp examination.
- Fundus examination by Direct and Indirect Ophthalmoscopy.

Advantages of RLE

- Cheapest refractive surgery. It can be made even cheaper by implanting monofocal IOLs and providing monovision to the patient.
- Technically easy to perform by most of the cataract surgeons.
- No extra instrumentation required.
- Visual prognosis is excellent. Most of the myopic patients have improvement of Snellen's acuity by 1 or 2 lines post-operatively.
- Stable refraction. Regression doesn't occur like some corneal refractive procedures.
- No chance of cataract development in future. For this reason, RLE is a definite indication in patients with high refractive error along with minimal cataractous change.
- ?Better optical quality of vision, compared to corneal refractive surgeries.
- Can be performed even in eyes with anterior chamber depth lesser than that required for Phakic IOL.

Disadvantages of RLE

Complications include the following:

- Posterior capsular opacification. Nd:YAG laser capsulotomy further increases the risk of detachment of retina.
- Retinal detachment. Recent studies have encouraging results in terms of RD. A meta analysis of papers on refractive lens exchange and cataract in high myopia between 1996 and 2004 included 2036 eyes[2]. The mean follow up was 43.5 months and overall incidence of retinal detachment was only 1.85%. This is only slightly different from that expected in the highly myopic population as reported by Burton[3] at 1.5%. The recent reports in 2003 of Ravalico et al[4]. and Guell et al., with 4 year follow up, also showed only 1 detachment for 422 eyes.
- Macular oedema.
- All of these complications are particularly prevalent in cases of extreme refractive error; macular oedema is more common in patients with hypermetropia.
- The remaining complications are the same as for any cataract surgery:
- Endophthalmitis.
- Corneal oedema from endothelial disruption or vitreous touch.
- Corneal melting with ocular surface disease.
- Wound distortion, leading to astigmatism and iris prolapse.
- Wound leak, shallow or flat anterior chamber.
- Hypotony.
- Iridodialysis.
- Glaucoma.
- Uveitis.
- Intraocular lens dislocation.
- Hemorrhage (anterior segment or vitreous).

- Capsular rupture.
- Zonular dialysis.
- Malignant glaucoma.
- Retained lens material.
- Suprachoroidal haemorrhage.
- Choroidal effusion (particularly in patients with hypermetropia).
- Implantation of wrong power of IOL.

Outcome and Prognosis

Visual outcome is usually excellent. A meta analysis of papers on refractive lens exchange on 2036 eyes, done by Richard Packard[2] showed RLE with posterior chamber IOL implantation to be safe, predictable, and effective. RLE was shown to achieve excellent visual acuity and refractive outcome with few complications. Some reports with prophylactic 360° therapy of peripheral retina showed a statistically lower rate of retinal detachment in those eyes, than if they had not been subjected to prophylactic laser treatment.

RLE vs Other Refractive Surgeries

Broadly, refractive procedures for myopia and hyperopia are classified into "Corneal procedures" and "Lenticular procedures". Corneal procedures include PRK, LASEK, LASIK, Thermokeratoplasty etc., whereas lenticular procedure includes Phakic IOL (PIOL) and RLE.

Corneal refractive procedures have a propensity to create optical aberrations. Conventional myopic excimer laser surgery reverses its shape from prolate to oblate, increasing the spherical aberration. Moreover, corneal refractive procedures have an upper limit of correction of myopia or hyperopia, whereas lenticular procedures have much wider range than the former.

Out of the two lenticular surgeries, Phakic IOL needs a minimum central anterior chamber (AC) depth from 2.8 to 3.2 mm, depending upon the location of IOL implantation (Ant. Chamber angle supported IOL / Ant. Chamber Iris fixated IOL / Post. Chamber sulcus fixated IOL). Thus, a patient with a crowded anterior segment from a thickened crystalline lens would not be a candidate for a PIOL, and could benefit from a reduced risk of angle closure glaucoma following RLE. These hypermetropic patients have a lower risk of retinal detachment as well. Several complications like corneal decompensation, pupillary block glaucoma, persistent flare etc. have been reported with PIOL. Anterior lenticular opacity has also been seen, necessitating removal of Phakic IOL and crystalline lens.

Normal corneal contour is retained after Refractive lens exchange, enhancing the quality of vision. Several studies have shown that, for quality of vision, an un-operated cornea is optically superior to an operated cornea. Any surgery on the cornea creates abnormal contours, which, in turn, create optical aberrations. The more the correction, the greater is the amount of induced aberration and the concurrent decrease in quality of vision, especially in low-contrast situations (e.g., driving at night). Clearly, RLE is an optically superior choice in many situations.

RLE is probably the most stable refractive procedure available. It also yields good contrast sensitivity. It has been found to be useful even in high ametropic children, who have anterior chamber depth insufficient for a Phakic IOL. Anterior surface ablation procedures have a significantly higher risk of regression or progression.

Conclusion

Refractive lens exchange has the advantage of greatly expanding the range of refractive surgery beyond the currently available methods, as well as providing cheaper and most stable alternative to corneal refractive procedures. Modern surgical techniques, IOLs and choosing the appropriate group to whom this procedure should be offered, has led it to become an accepted, safe and common refractive surgery.

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Pseudophakic Monovision: Pros and Cons

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Presbyopia is a condition in which the lens of the eye loses its ability to focus, making it difficult to see objects up close. Presbyopia cannot be cured. Instead, prescription glasses, contact lens, reading glasses, progressive addition lenses or bifocals, conductive keratoplasty, lasik can help to correct the effects of presbyopia.Cataract in old patients provides unique opportunity to ophthalmologist to correct dependency on glasses with cataract extraction. Presbiopic lens exchange (PRELEX) aim to correct the loss of accommodation by removing the crystalline lens by phacoemulsification and implantation of a multifocal intraocular lens (IOL) in the capsular bag[1]. This technique was first described in 1997 by C. Claoue. Multifocal lenses provide spectacle free life, but problem of nocturnal halos[2], photic phenomena[3], decrease contrast sensitivity[2,4,5,6,7] and cost are limiting factors. Monovision is a method of presbyopic correction whereby one eye is corrected for distance and the other for near, by means of contact lens, refractive surgery or intraocular surgery[8] (Figure 1). Monovision in pseudophakic patients was first described in 1984 by Boener and Trasher[9]. Greenbaum[10] reported a high level of acceptance of monovision in 140 patients who had bilateral cataract or refractive lens exchange (RLE). Similarly, Handa[11] et al found 80% satisfaction among pseudophakic monovision patients. Pseudophakic monovision is a cost effective alternative to multifocal IOL in reducing dependence on glasses in presbyopic population (Figure 2).

Types of Monovision

- Conventional monovision
- Cross monovision.

Figure 1: *Concept of Monovision*

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When dominant eye is optimized for distance vision, it's called as conventional monovision and when nondominant eye optimized for distance, it's called cross monovision.

Factors affecting success of monovision

Success of monovision depends on various factor and complex interplay between them.

There are 4 major factors

- Ocular dominance
- Degree of anisometropia
- Stereopsis
- Patient's motivation

Other factors include age of the patient, preoperative trial of monovision and patients counseling.

The mechanism that enables monovision to succeed is interocular blur suppression (i.e. the ability to suppress the blur image from 1 eye) and it is assumed that it is easier to suppress blur in nondominant eye[12]. Few old studies suggested that correction of dominant eye for distance increase the chance of success rate of monovision[13,14] but others believe that selection of eyes for correction may not be a determinant of the success of monovision[15]. However, no studies have conclusively quantified

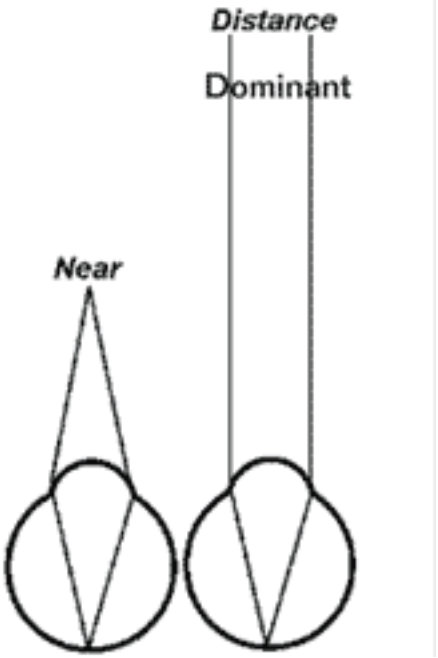

Figure 2: *Pseudophakic Monovision*

which eye to correct for distance in a monovision patient.[7] Several theories have been proposed for selection of the eye for correction;

- Correcting the dominant eye to maximize performance of visual tasks requiring spatial perception;
- Correcting the left eye for increased driving safety;
- Correcting the less myopic eye to decrease the peripheral blur during distance vision; and
- Correcting the dominant eye for the most commonly used viewing distance to maximize blur suppression.

In the absence of rigorous clinical studies the dominant eye for distance has become the convention. Ideal monovision required alternating dominance and interocular blur suppression[16] for dependable distance vision. A study in which comparison of convention & cross monovision was done in presbyopic patients, it was found that satisfaction level was same in both the groups and patients satisfaction with monovision showed no relationship to gender, age at the time of initial surgery, preoperative trial of monovision, laterality of treatment, type of monovision predictability outcomes.

Strong sighting preference reduces the success of monovision[10]. Ideally the patients of monovision should be able to see clearly at all distances. The binocular clear vision range should be equal to the sum of monocular clear range without interference of blurred image in one eye. Therefore strong dominance may render strong stress in visual systems preventing altering dominance & intraocular blur suppression. Sipplel & co authers[17] indicated that strong sighting dominance is difficult to preserve in successful monovision and weak sighting dominance (altering dominance) seems to be an important factor in successful monovision. However inputs from the dominant eye produce a greater response to a given stimulus than input from the nondominant eye. Therefore ocular dominance may play an important role in the temporal fluctuation in interocular blur suppression. For successful monovision, interocular blur suppression should be flexibly changed in each eye in all distances. Therefore, it is best that the ocular dominance in patients with monovision be as low as possible. Binocular rivalry is not primarily viewed as a tool for measuring ocular dominance but rather for studying the neural correlates of visual perception[11].

Studies of visual performance demonstrated slightly reduced binocular visual acuity and no effect on binocular peripheral visual acuity or binocular field size. There is reduced depth perception, which improves following adaptation.[13,18,19,20]

In monovision, because of induced anisometropia detailed depth perception (stereopsis) may be reduced[21]. Study done by Wright et al[11] showed that near stereopsis decreased slightly in monovision patients as compared to control group but the difference was not statistically significant. Recent study done by Yaron et al showed that mean near stereopsis in monovision patients was 175.6 sec of arc; however stereopsis for distance was not assessed in this study[22]. In our study mean near stereopsis was 173.33(±65.31) second of arc. In a study done at our centre23 it was found that the mean distance stereopsis on FD2 was 51(±11.21)) sec of arc, which was similar to that shown in previous studies done with monofocal contact lenses[13,18]. Reduced Stereopsis has been considered to be the major disadvantage associated with monovision. Jain & Coauthors[8] found that patients with unsuccessful monovision had a reduction in stereopsis than compared with patients with successful monovision.

Monovision with intraocular lens implantation may be advantageous in presbyopic patients having cataract surgery. However this option should be pursued only after careful preoperative screening especially after examination of the quality of ocular dominance.

Age is an important factor which affects success of monovision. Studies showed that chances of monovision success are less in young because young patients are more demanding and have excellent distance best corrected visual acuity and often good uncorrected visual acuity[24], but on the other hand young patients are highly motivated with early visual adaptation which improves success rate. In old age poor and delayed visual adaptation decreases chance of successful monovision.

Preoperative trial of monovision by putting +1 to +2 D lens in front of eye to be corrected for near vision is also a good preoperative exercise and few studies claim increased success of monovision by this method[7,22,23].

When counseling patients preoperatively, it is important to consider occupation, sports, hobbies, and the need to maintain uncorrected near vision. To make choice, the patients must receive an explanation of Presbyopia and accommodation. This is time consuming and demands effective communication. It is also important to communicate that monovision is a compromise that does not restore normal physiology but compensation for normal accommodation. When patients understand the trade off, they are more likely to adapt to and to be happy with monovision correction[25].

Conclusion

Pseudophakic monovision is a good alternative of multifocal IOLs.[2,3,4,5] The cost of monovision is also less than compared to multifocal IOLs, which makes it an effective alternative of multifocal IOLs in developing countries. The rate of secondary interventions are less in monovision then multifocal IOLs[22]. In conclusion we recommend conventional monovision with moderate anisometropia to improve success of monovision and patients satisfaction level.

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Congratulation

Congratulation **Dr. N. K. Agarwal** joined the DTE GHS, Nirman Bhawan, New Delhi as Deputy Director General (Ophthalmology) as incharge of National Program for Control of Blindness (NPCB).

Newer Trends in Evaluation of Ocular Injuries

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Despite the strong anatomical barrier and vigilant physiological protection provided by nature to the eye, the incidence of ocular injuries remains high. The fate of traumatized eye depends upon the treatment adopted. Timely reporting of cases and early surgical management reduces the visual loss.

"Trauma is one of The Captains of the Men of Death"
-Duke Elder

"Promptness in decision of action in emergency is the best test of the powers and resources of any man, especially medical men".

Need for Evaluation

Annually more than 2 million cases of eye trauma are reported, out of which more than 40000 cases end up with severe visual impairment accounting for socioeconomic burden.

Most of the cases are less than 40 yrs of age and 90% blindness due to trauma is preventable.[1]

How to Classify Ocular Trauma?

Various authors from different countries classified ocular trauma but the one given by Duke Elder is most acceptable hence quoted.

Etiological Classification

Duke Elder classified ocular injury from etiological (Nature of Injury) point of view into the following groups[2]:

Mechanical

- Concussions & contusions caused by blunt trauma
- Penetrating & perforating injuries due to sharp Objects.
- Effect of retained intraocular foreign bodies.

Non mechanical

- Electrical injury
- Ultrasonic injuries,
- Stress injury
  - Barometric stress,
  - Vibrational stress
  - Acceleration stress
- Thermal injury
  - Hypothermia,
  - Hyperthermia
- Radiational injury
- Chemical injuries

- Intrauterine Injury,
- Non occupational domestic injury,
- Injuries in agriculture
- War injuries
- Iatrogenic injuries

He further classified injuries into:

- Birth Injury,

Figure 1: *External photograph showing full thickness corneal tear from 12 O'clock to 5 O'clock (zone 1 injury)*

Figure 2: *Intraoperative external photograph showing limbal tear from 11 o'clock to 2 o'clock hours with iris tissue prolapse and hyphema (zone 1 injury)*

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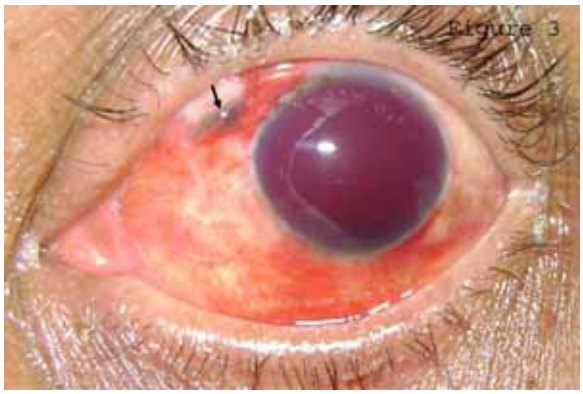


Figure 3: *External photograph showing scleral tear 4 mm away from limbus (zone 2 injury) with iris tissue prolapsed (black arrow) with eight ball haemorrage (toatal hyphema) in anterior chamber*

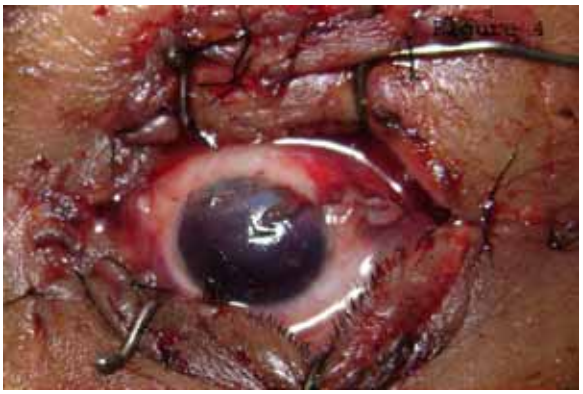


Figure 4: *Corneoscleral tear extending 10 mm (Zone 3 injury) into the sclera*

- Injuries in sports & travel
- Industrial accidents
- Self inflicted injuries

The importance of the initial evaluation of eye trauma can not be overemphasized because the result of this examination determines the subsequent diagnostic & therapeutic decisions; a comprehensive workup is mandatory.

Anatomical Classification

Stenberg & Pieramici et al. in 1997 (Ocular trauma classification group) Classified ocular injuries as[3]:-

Open Globe Injury

Classification

Type

- Rupture
- Penetrating
- Intraocular foreign body
- Perforating
- Mixed

Grade

Visual acuity

- > 20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception
- No light perception

Pupil

- Positive: relative afferent pupillary defect present in affected eye
- Negative: relative afferent Pupillary defect absent in affected eye

Zone

- Isolated to cornea including the corneoscleral limbus (Figure 1&2)
- Corneoscleral limbus to a point 5 mm posterior into the sclera (Figure 3)
- Posterior to the anterior 5 mm to sclera (Figure 4)

Closed Globe Injury

Classification

Type

- Contusion
- Lamellar Laceration
- Superficial foreign body
- Mixed

Grade

Visual acuity

- >20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception
- No light perception

Pupil

- Positive: relative afferent pupillary defect present in affected eye
- Negative: relative afferent Pupillary defect absent in affected eye

Zone

- External (limited to bulbar conjunctiva, sclera, cornea).
- Anterior segment (involving structures in anterior segment internal to the cornea and including the posterior lens capsule also includes pars plicata but not parsplana)
- Posterior segment (all internal structures posterior to the posterior lens capsule)

Trauma Index (Shukla et al)

Magnitude of trauma depends on structural or functional loss, which has to be weighed against the time factor. Based on this convention, a formula for overall assessment of ocular injury has been workout by Shukla et al[4]. Visual loss up to 6/60 is graded in steps of 10 and beyond those steps of 5.

- T. I. = 1/2 (S/2+F) T/100
- **S** – Structural loss **F** – Functional loss **T** – Time factor

Grades of Structural loss are:

Mild (25%): Slight corneal and lenticular opacities, scar/notching of lids, slight congestion, swelling, and hemorrhages.

Moderate (50%): Dense corneal/lenticular opacities, ptosis or lagophthalmos, squint, marked subconjunctival haemorrhage and hyphaema. acute inflammations.

Marked (100%): Large scleral/corneoscleral tears, total anterior staphyloma, pthisis bulbi, and multiple fractures leading to gross displacement of globe.

Functional loss - Graded from 0 to 100% depending on the visual acuity (6/6 to >NLP).

Vision	Loss	Vision	Loss
6/6	0%	5/60	65%
6/9	10%	4/60	70%
6/12	20%	3/60	75%
6/18	30%	2/60	80%
6/24	40%	1/60	85%
6/36	50%	C.F.	90%
6/60	60%	H.M.	95%

No Perception of light = 100%

(Based on the presumption that pre trauma vision was 6/6.)

To simplify, the Ocular Injuries were further classified in three grades depending on the Trauma Index as - Mild, Moderate and Severe.

Classification

S. No.	T.I. Group	Grade
1.	0-10	Mild
2.	11-50	Moderate
3.	Above 50	Severe

Figure 5: *External photograph showing Hyphema and closed globe injury*

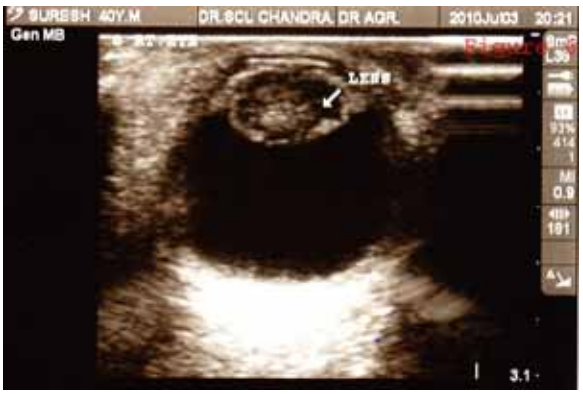


Figure 6: *B-Scan of a closed globe injury showing disorganized cataractus lens (white arrow) and almost flat anterior chamber*

Both the eyes are considered separately.

Trauma index up to 10 indicates that injuries are mild and recover in short time however over 50 indicates grave damage.

The Birmingham Eye Trauma Terminology (BETT)

Lack of Unambiguous Common Language a major limiting factor in effective sharing eye injury information. Varying responses are given to simple questions such as

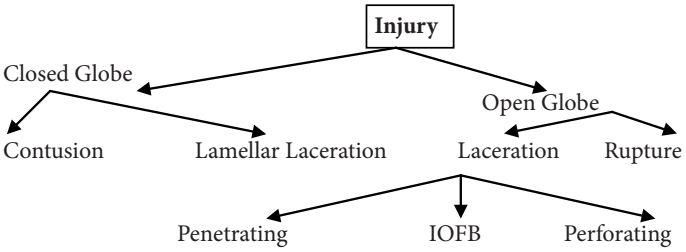
- What is the distinction between laceration and rupture, penetration and perforation?
- Is full thickness scleral wound without obvious choroidal & retinal involvement, an open globe injury?
- If a foreign body has traversed the eye & lodged in the orbit, is it perforating? Double perforating? Double penetrating?

*Kuhn F et al has introduced "**The Birmingham Eye Trauma Terminology (BETT)**" which is uniformly acceptable terminology as below*[5]:

This comprehensive, standardized system of eye trauma terms, should be utilized when reporting to USEIR

Endorsed by the: American Academy of Ophthalmology, International Society of Ocular Trauma, Retina Society, United States Eye Injury Registry, Vitreous Society, World Eye Injury Registry,

Mandated by: Grafe's Archives, Klinische Monatsblatter, Ophthalmology



BETT

Glossary of Terms

Term	Definition and explanation
Eyewall	- Sclera and cornea
Closed globe injury	- No full-thickness wound of eyewall (figure 5,6 & 7) 55
Open globe injury	- Full-thickness wound of the eyewall.
Contusion	- There is no (full-thickness) wound - Due to direct energy delivery by the object (e.g., choroidal rupture) or to the changes in the shape of the globe (e.g., angle recession)
Lamellar laceration	- Partial thickness wound of the eyewall
Rupture	- Full-thickness wound of the eyewall, caused by a blunt object. - Inside – out mechanism (Figure 8&9)
Laceration	- Full-thickness wound of the eyewall, caused by a sharp object. - By an outside – in mechanism.
Penetrating injury	- Entrance wound. - If more than one wound is present, each must have been caused by a different agent. - Retained foreign object/s (figure 10) Technically a penetrating injury, but grouped separately because of different clinical implications.
Perforating injury	- Entrance and exit wounds. - Both wounds caused by the same agent.

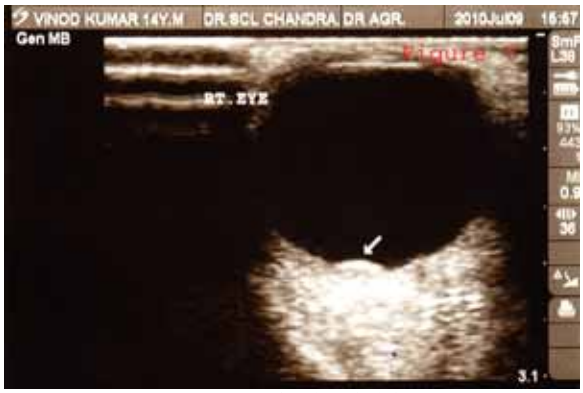


Figure 7: *B-scan image of a closed globe injury patient showing Berlin's edema*

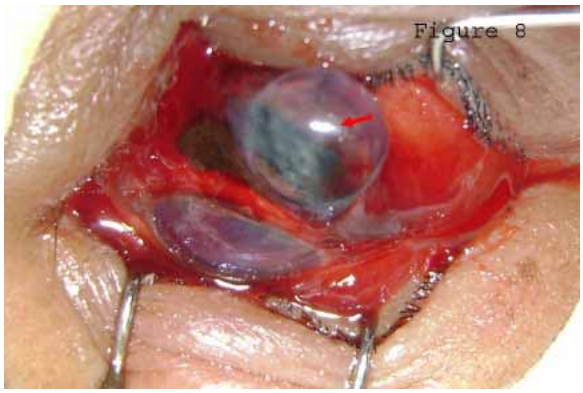


Figure 8: *Intraoperative photograph showing crystalline lens (red arrow) found underneath conjunctiva on exploration of a patient with globe rupture*

The Ocular Trauma Score (Version 11.1) Computational Method for Deriving the OTS Score[6]:

Initial Visual Factor	Raw points
A. Initial Visual acuity category	NLP = 60 LP to HM = 70 1/200 to 19/200 = 80 20/200 to 20/50 = 90 > 20/40 = 100
B. Globe rupture	-23
C. Endophthalmitis	-17
D. Perforating injury	-14
E. Retinal detachment	-11
F. Afferent pupillary defect (Marcus Gunn) pupil)	-10
Raw score sum = sum of raw points	

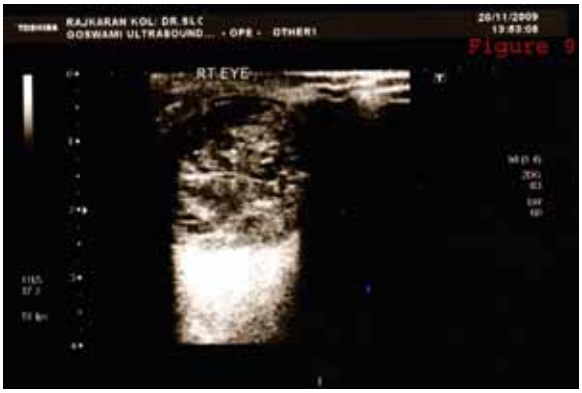


Figure 9: *B-Scan image of a patient of occult globe rupture showing disorganized globe*

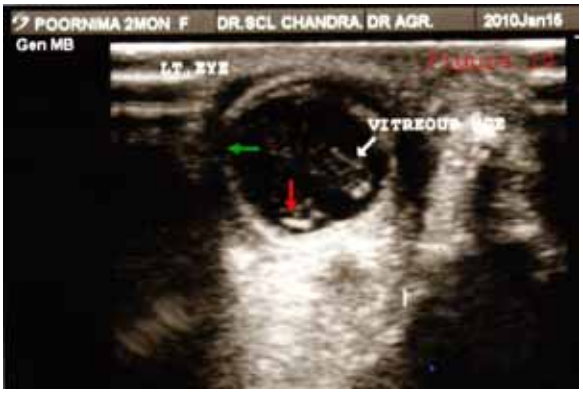


Figure 10: *B-Scan image of a penetrating intraocular injury showing vitreous hemorrhage (white arrow) and an intraocular foreign body (red arrow) and globe laceration (green arrow)*

Estimated Probability of Follow up Visual Acuity Category by the OTS Score						
Raw Score Sum	**OTS Score**	**NLP**	**LP/HM**	**1/200-19/200**	**20/200 -20/50**	**>20/40**
0-44	1	73%	17%	7%	2%	1%
45-65	2	28%	26%	18%	13%	15%
66-80	3	2%	11%	15%	28%	44%
81-91	4	1%	2%	2%	21%	74%
92-100	5	0%	1%	2%	5%	92%

Time factor plays an important role in restoration of structural and functional integrity in cases of penetrating ocular trauma. BETT and OTS are helpful in classifying, documentation and predicting the final visual outcome in case of trauma.

Goals of Initial Evaluation

Clinical Evaluation

The goals of initial evaluation can be organized on four levels.[1,7,8]

Complete evaluation of the eye and ocular adnexa.

Recognition of emergent conditions.

- Life threatening injuries.
    - Respiratory distress.
    - Cardiovascular compromise.
    - Massive bleeding and shock.
    - Major trauma to any organ system

Recognition of complete extent of ocular involvement.

Identification of compounding factors

Other associated non-life threatening injuries.

- Bleeding.
- CNS Trauma.
- Other injuries.

Concurrent Medical conditions.

- Diabetes Mellitus.
- Atheroscterotic cardiovascular disease.
- Sickle cell hemoglobinopathy.
- Bleeding disorders
- Infectious diseases (a) Hepatitis. (b) AIDS

Foreign bodies

Need for further testing

- Radiological
- Ultrasonographic
- Electro Physiologic
- Hematological / Serologic

Development of initial therapeutic plan

In conclusion newer terminology and ocular trauma classification are helpful not only in management of ocular injuries but also excellent tools in proving uniformity in ocular trauma research.

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Use of Interferons in Ocular Diseases

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Interferons were discovered as natural antiviral substances produced during viral infection and were initially characterized for their ability to 'interfere' with viral replication, reduce cell proliferation, and alter immunity. They are involved in defence against viral infections, tumor growth, and tolerance induction as suspected inducers of autoimmune disease. At the same time they have been used successfully as treatment for autoimmune diseases such as multiple sclerosis. Thus interferons appear as double agents, being involved in inducing as well as in treating autoimmunity.

Types

Based on amino acid composition and biological properties, interferons have been classified as type 1 and type 2. Type 1 includes at least 13 different isotypes of interferon a and a single interferon b, both of which bind to a common receptor, IFNAR. Type 2 includes a single member g which binds to a different receptor IFNGR.

Role of interferons in immunity

Interferons are synthesized and secreted by monocytes, macrophages, T lymphocytes, neurons, and glial cells. Early viral infection leads to stimulation of plasmacytoid dendritic cells, the main producers of IFN 1 and subsequently IFN g production. This finally leads to

- Induction and maintenance of T-helper type 1 cells, CD8 cytotoxic T cells, and natural killer cells that fight the viral intrusion.
- Anti-proliferative and proapoptotic effect on T cells.
- Development of tolerance promoting regulatory T cells.
- Positive as well as an inhibitory effect on B-cell development and survival.

Role of interferons in autoimmunity has been implicated in

- SLE
- Diabetes mellitus
- Uveitis
- Multiple sclerosis
- Sarcoidosis including sarcoidosis associated uveitis

Interferon as a therapeutic substance

IFN-a has been approved in the treatment of Hepatitis B and C infections. IFN-b1a at 44µgms three times a week has been used successfully in reducing the number and severity of multiple sclerosis attacks. Recently pegylated interferons have been introduced which have a covalently attached polyethylene glycol chain and thus have a longer plasma half life, which improves efficacy, allows less frequent dosing, and reduces immunogenicity.

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Local use in ocular disease

Viral keratitis and conjunctivitis

Wilhelmus reported in a 2003 Cochrane review that interferon monotherapy had a slight beneficial effect on dendritic epithelial keratitis, but interferon was not better than other antiviral agents. A compelling observation was the significantly improved treatment success produced by a combination of a topical interferon with a topical nucleoside antiviral agent such as trifluridine or acyclovir. Interferon-a eye drops have an effect on reducing the frequency of infection with viral conjunctivitis.

Ocular surface neoplasias

Interferons have been shown to be effective in management of small primary and recurrent corneal and conjunctival neoplasias. In a dose of 1 to 1.5 million IU per ml 4-5 times a day topically, IFN a2b has been observed to reduce recurrence rates after primary excision. Similarly intralesional injection of interferon-a2b in conjunctival mucosa-associated lymphoid tissue lymphoma in a dose of 1.5 million IU 3 times weekly achieved complete clinical and histopathological remission in significant number of cases with very few recurrences. The only side effect was a transient flu-like syndrome in all patients.

Mooren's Ulcer

Topical IFN alpha2a as a single therapeutic agent has been reported as an effective alternative in the treatment of patients with Mooren's ulcer. It offers the benefits of topical therapy and may avoid surgical intervention in unresponsive cases.

Intravitreal use in ocular disease

Isolated case reports of the intravitreal use of a single injection of 100 000U interferon-a in 0.1 ml total volume in patients with advanced neovascular AMD showed a small subjective and objective improvement in visual acuity. This was however associated with a reversible reduction in the light response on electroretinography after 1 month.

Systemic use in Ocular Disease

Multiple Sclerosis-associated Ocular Disease

Interferon-b has been shown to have beneficial effects in patients with multiple sclerosis or optic neuritis. The approach to treating patients with optic neuritis has been modified by the results of the Controlled High-Risk Subjects Avonex Multiple Sclerosis Prevention Study (CHAMPS). Patients with initial episode of demylination and subclinical lesions of MS on MRI brain when treated with weekly intramuscular injection of 30 µgms of IFN b1a showed a 50% reduction in progression to clinically definite MS.

Also treatment of uveitis associated with multiple sclerosis that was refractory to corticosteroid treatment with type 1 IFNs appeared to have beneficial effects on visual acuity, intraocular inflammation activity, and the presence of CME in a significant number of study subjects. Interferon-b1a and interferon- b1b block interferon-g induced disintegration of endothelial junctions and therefore protect endothelial barriers. The reduction of vascular leakage and hence reduction of edema of the posterior pole seems to be an important effect of interferon in the treatment of patients with uveitis associated with multiple sclerosis.

Behcet disease

The uveitis in a patient with Behcet disease is accompanied by severe inflammation with occlusive vasculitis that can lead to visual impairment. Herpes simplex virus type 1 is thought to play a role in the pathogenesis of Behcet disease. Therefore, IFN a was introduced in 1986 for its antiviral activity. IFN a was administered at a dose of 6 million units daily, with dose and frequency adjusted depending on the clinical response. Subjects showed significant improvements in both visual acuity and posterior uveitis score in 92% of patients.

Cystoid macular edema in uveitis

In patients with inactive uveitis and CME not responsive to systemic corticosteroids and acetazolamide, IFN a2a has been shown to be efficacious in a dose of 3 to 6 million IU daily for 2 to 4 weeks with complete resolution of CME.

Side effects

Most common side effects of type I interferons are injection site reactions and flulike symptoms fever, headache, myalgia, arthralgia, sweating, and fatigue). These acute effects can be alleviated or eliminated by concomitant use of paracetamol. Usually, severity of these symptoms will decrease in the course of therapy or after adjustment of dosage.

Depression and suicidal intentions can occur during therapy with interferon independent of preexisting psychiatric disease. An asymptomatic increase in liver enzymes and a decrease in leukocyte count are seen in as many as 10% of patients. Most of the side effects are mild and reversible.

Interferon may also produce ophthalmologic side effects, particularly retinal lesions such as cotton wool spots, hemorrhage, microaneurysms, and neurovisual impairment. These are common but rarely symptomatic. There are isolated case reports of severe ophthalmologic complications such as acute exophthalmos, subconjunctival hemorrhage, papilledema, retinal artery occlusion, and retinal vein thrombosis. Risk of ocular side effects seems to be higher in patients treated with pegylated interferons.

Conclusion

Interferons are interesting cytokines that form a network of complex interactions with other cytokines and connect innate and adaptive immunity. They seem to be involved in induction of autoimmune disorders as well as in their treatment. Interferons have well documented role as immunologic therapy in the treatment of hematologic and solid tumors, viral hepatitis, and autoimmune diseases, mainly multiple sclerosis. In addition evidence is growing of their role in the treatment of ocular diseases, especially for interferon-a in ocular Behcet disease and interferon-b in multiple sclerosis associated intermediate uveitis.

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Delhi Ophthalmological Society
Monthly Clinical Meeting, September 2010

Venue: Ayur Vigyan Auditorium, Army Hospital (R&R), Dhula Kuan, New Delhi
Date & Time: Sunday 26th September 2010, 10:30 a.m.

Clinical Cases:

1	Intra-Ocular Cysticercosis	:	Maj. A. Avasthi
2.	Spectrum of sports injuries	:	Dr. Rachna Pandey

Clinical Talk:

Issues with Multifocal and Accommodative Lenses	:	Col. J.K.S. Parihar

Symposium: ***Tube in Special Situations***
Chairpersons: *Ltd. Gen. D.P. Vats, Col. J.K.S. Parihar*

1.	Congenital Glaucoma	:	Maj. Jaya Kaushik
2.	Traumatic Glaucoma	:	Lt. Col. S. Mukherji
3.	Combined Procedures	:	Wg. Cdr. H.S. Trehan
4.	Posterior Segment Valves	:	Col. J.K.S. Parihar

Moderators: **Lt. General D.P. Vats, Gp. Capt. P. Shingal**

Practice Turnaround – Make It Possible

Vipin Sahni *MS*

In India most ophthalmologists are still in small, private, independent practice businesses, working for themselves, which, when you think about it, is remarkable. The regulatory oversight, competition, operational complexity and investment hurdles that ophthalmic business owners face—when added to the original educational challenges and ongoing liabilities—make it surprising that anyone is still in this business at all. Successful cataract surgeons whose incomes have softened in recent years have learned the hard way that it's very expensive being rich. We're now creating a whole new crop of LASIK surgeons who will have to learn this same painful lesson as refractive surgery goes through its inevitable peaks and valleys.

I hear of the same envious complain from Doctors with faltering practices. Looking at their more successful colleagues they ask, "Why isn't our practice as prosperous as theirs?" The answer is remarkably simple.

Some of the most successful practices are that way because they're located in charmed locations. I know one ophthalmologist in the north, who is the only eye surgeon for a service area with more than 20,00,000 people. The average ophthalmologist in India competes in a market that only yields about 65,000 people per eye surgeon. Attractive markets like Delhi only has about 15,000 people per eye surgeon. Some surgeons work in markets so rural that staffing and facility costs are a fraction of what their urban cousins' pay.

What about those practices whose locations aren't so fortunate? Every other flourishing ophthalmologist I know has had to get there the old fashioned way—they've worked hard and survived the pitfalls that can undermine success in the increasingly complex and demanding business side of eye care. What was once a very forgiving enterprise that could be run loosely with great success is now a business like most others. Practice expenses are rising each year in an essentially fixed or falling fee environment.

In reality, all practices periodically get into trouble. Even if you have a robust practice today, you will likely experience the some panic that is now common in the country's weakest clinics. Most agree we have not yet lived through the toughest times for ophthalmology. Here are some thoughts to help the strongest practices stay ahead, faltering practices turn their fortunes around, and the weakest practices at least hang on in the next difficult years.

India is a very special country

When I ask a practicing ophthalmologist, how much were they earning, they don't know the answer. Many of the eye specialists do not know how much they were actually earning. As they don't have any account. Whatever they have is just a cooked up account to satisfy the Government tax departments. They don't have OPD registers of a few years, to count actual OPD numbers. No receipt books as they do not give any receipt to any one until asked for. No staff salary logs, No expenditure books etc. When asked for they just have surgical consent forms from which they can know how many surgeries and what type of surgeries have been done by them. When they don't know how much they are earning, they are also unaware that their practice is improving or decreasing. They fail to change themselves according to rapidly changing environment. Slowly they get into the trouble and hence start a faltering practice.

There are another type of practices. After doing MS /DOMS ophthalmologists start their practice in a city where they belong to or just where their parents lived or in a city where they studied. They invest only few lacs buying only necessary basic equipment/ instruments. Usually they have no phaco or lasers etc. They do not even take a training in phaco or only short term phaco training. After few years they realize they are not growing by the pace they should grow. They don't have money to buy these equipments which are now must for every practice. They are in trouble, now they become a faltering practice.

Step One: Get the Real Picture

It is most important to know that when to call a practice a faltering one. From whatever I have learned over the past few years experience If -

- Your surgeries are not increasing or
- Have been decreasing for the past two consecutive years
- Your OPD is decreasing since two consecutive years
- Your revenue is decreasing since two consecutive years
- Your net profit is decreasing since two years, you may be in trouble.

If you have realized that your practice has not improved in all these years or actually decreasing then you think of a Turnaround. Taking decision of a turnaround is not easy; it involves a lot of efforts, time and money. It also leads to some drastic changes. Once you have decided for turnaround, get a broad, subjective impression of the state of your practice, and review the information you'll need to make some tough decisions. Call a meeting of everyone who matters. Your practice probably doesn't have a formal board or no outside directors, but you certainly have a "virtual board," composed of the doctor-owner (you and your spouse where appropriate), selected senior staff and a few relevant external advisors.

Before coming to this group meeting to frame up the problems, every person called to the meeting should separately write, in about 250 words or less, the history of the practice, the broad state of the market today, how is your practice doing today and the other important issues. Compare everyone's short essay, and agree on a consensus statement of the current position of the practice. Make this document as long or short as you desire, but it need not go any further than a page or two. Make sure that your consensus

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summary includes at least a sentence or two about the three to five year future of the practice. Next, mount the map of your service area (area from where you receive most of your patients) and the place for your current location/s, prospective or former locations, competitors, etc. Put this map in your normal meeting room to aid subsequent planning and discussions.

Examine what you are fighting for. Determine the total estimated population of your drainage area. This may be a few urban blocks, or a 100 Km radius. Divide the number of people by the number of full-time ophthalmologists serving this area. If the resulting number is over 200,000, you're in the "safe" zone. Fewer than 50,000 and it's going to be fighting uphill, unless your subspecialty area is under-represented, or there is an extremely high senior population ratio. Incorporate the facts and your impressions about these key population-to-provider ratios in your consensus summary, above. Collect all the data related to practice. This should include monthly OPD records, Surgical records (these should include from which geographical area), Earnings (facility wise) like from OPD, Retina practice and other sub specialties, fields, lasers, Cataract surgeries, other surgeries, insurers, corporate tie ups, opticals, medical etc. Expenses like facility rent, electricity, maintenance, staff wages, and supplies also are noted.

Step Two: Examine the Details

Examine all the details. There are no standards data available to compare as India is a very big country and all type of practices exist. But I think if you are an Ophthalmologist (invested ten golden years in studying), have invested around 20 lacs in practice and your practice is 3-5 year old; you should have

- OPD – 40+
- New/old ratio – 75/25
- Surgeries – 10-30 per patient encounter
- Facility expenses – less than 10%
- Staff wages – 10-30%
- Earnings per patient encounter – Rs. 500/- to Rs. 5000/-
- Earnings- above Rs. 50 lacs (per annum)

Assemble up-to-date financial records of the past three years, along with monthly procedure counts, patient visit statistics, staff payouts and new patient sourcing data. Use these to establish your own current benchmarks and future goals. From this list of ideal standards, you may find one or more areas where you are grossly mis-aligned. Focusing your major efforts on just one or two problem areas may accelerate your turnaround.

Step Three: Decide what's missing…and Replace It

Let's now compare the available resources, in all dimensions, with those needed to turn your enterprise around. This exercise, while still largely subjective, will get you thinking about the critical bottlenecks or choke points in the practice. Has your turnaround team reviewed the following general list of resources, which are critical for the survival and success of any modern practice.

- Your clinical and surgical skills.
- Your behavior with the patients.
- Your facility's looks and equipments
- Your boldness and willingness to take reasonable business risks and avoidance of unnecessary risks.
- Your leadership skills—the ability to set fair policies and inspire staff.

If most of your customers are satisfied:

- Your business plan (even if brief and informal) describing the realistic goals.
- Your discipline to stick to the business plan, and not to be distracted by trivial issues and opportunities.
- Your managerial abilities—the ability to turn policies into smooth daily functions.
- Your staff performance and motivation.
- Your adequate physical facility for current and anticipated operations.
- Your knowledge of the local marketplace, competitors, trends, etc., and proper management of external relationships (hospitals, referral sources, etc.).
- A proper team of external advisors (accountants, Lawyers, management consultants, etc.).
- Your marketing efforts, internally and externally.
- Your Staff recruitment, training and supervision protocols.
- Your Access to appropriate levels of capital, either borrowed externally or from ongoing operations.
- Your favorable reputation among the patients and in the wider community.

Now prioritize and estimate the Rupee cost of the top three needed resources. Note that these are the most needed in your practice at this time. Discuss the practicality, timing and logistics of securing these three most critical resources. If one or all of these key missing resources can be feasibly secured, decide a deadline for each item and the person responsible. Some practice owners will chronically—and perplexingly—starve certain critical departments of the resources they need to function. For example, lacking just one part-time staff member or a computer software you miss out forever on some account details or by just not having an optometrist you spend most of your time in refraction not paying attention to the patient's queries which leads to an unsatisfied patient. Does anything akin to this happen in your practice, or are available resources spread appropriately based on practice needs? If there is a significant gap between the practice's needs and potential resources, why have the needed people, capital, external expertise, etc. not been applied in this practice? Is your management neglectful? Don't you want to come out of your comfort zone? Is there simply no money available? Is a lack of decisiveness holding everything up? Or have resource gaps simply not been recognized until now?

Step Four: Make Sure You Have the Right Stuff to Succeed

Corporate planning consultants say, "Business failures are often preceded by personality dysfunction on the part of the executives

involved. More often than we think, the root causes of many business failures lie in the executives' own traits, which are mismatched with the requirements of the business." Could You be your faltering practice's chief problem? If you can't definitively answer, "No," look for improvements in this area before going any further.

Management skills and personality traits aside, the simple truth is that most practices that need a turnaround have as a companion problem, a frank lack of energy and time on the part of the doctor-owner. There comes a time when "working smarter" reaches its natural limits and "working harder" is the only solution. It's normal, even when the practice isn't facing an emergency, for an ophthalmic practitioner to be the first to arrive and/or the last to leave. It's normal for the managing partner of a group practice (or a solo practitioner) to spend more hours in an average week on purely practice management issues and tasks. The turnaround team should assess if the raw time commitment and work intensity of these team leaders is sufficient. The performance bar often needs to be raised much higher, at least during the early phases of an emergency turnaround, to get the job done.

Only the most fortunate surgeons can work in "easy" markets. For the rest, I've compiled a short priority list of "Ten Cardinal Rules" than can help to assure control over your business affairs and ultimate commercial success, even in the most demanding markets.

Ten Cardinal Rules

Make great decisions. It sounds easy, but it's not. After making sure that your decision is ethical and legal, most decisions should boil down to one simple test: Which option is more profitable, Option A or Option B? Gather the facts, and let the facts help you make decisions.

Don't make business decisions by yourself. Try and engage every individual likely to have an impact by your decision is good politics. Getting the counsel of people smarter or more experienced than you is more common sense. Even the most generous cannot do everything by himself. The sharpest clinical minds may often be frustratingly dull when applied to business affairs.

Strive daily to increase profitability without reducing quality, and to increase quality without decreasing profits. In mature practices, large profit and quality gains are rare. But even the most developed and refined practice can make small, incremental improvements.

Leave your options open. Think twice before abandoning any patient services, or dismissing a key staff member because of some misconceptions. Even if you don't dispense glasses today, leave room in your new office facility for a dispensary adjoining the reception area. Take an introductory Phaco or Medical Retina Training to stay current, even if you never plan to have Phaco or retina practice.

Get and stay diversified. "Don't put all the eggs in the same basket" Sharply falling practice profitability is just the latest in a long list of hard lessons in the business of ophthalmology. For safety, no single patient service and no single payer (with the possible exception of Medicare) should represent major share of your cash flow.

End each year with better relationships than you started with. Be balanced in your relationship building, including patients, payers, staff, local institutions, venders, lenders, and fellow doctors.

Be disciplined. Most practices that get into trouble are because their owner doctors are indisciplined in one way or another. They set performance standards for staff that they are unwilling to keep for themselves. In any area where you have a history of being poorly self-disciplined (financial, behavioral, exercise, etc.) hire an outside source of discipline.

Work harder, after you have exhausted all opportunities to work smarter. The most financially successful surgeons I know in the world are "B" students who did exceptionally well because they had simply worked harder than their peers.

Seize opportunity. The most successful people in business are rash and opportunistic. If you think there are no more opportunities left in eye care, you're not looking hard enough. And if the opportunities you can see aren't appealing, it may be time to find a new profession. When an opportunity knocks, seize it. Most importantly, have sufficient personal and business capital reserves on hand to be able to accept reasonable risks.

Pro-forma every major business decision. Using your current income statement as a baseline, review the impact of adding a new periphery, new doctors, or a new patient service to your practice. Have the intellectual integrity to believe on the exact numbers rather than relying on hunches and guess estimates. At the same time, trust your instinct. A bad feeling about a potential project should veto even the rosiest financial projections.

Remember winners don't do different things, they do it differently.

Author
Vipin Sahni MS

Vision 2020 - India

***Dr. Rajesh Noah**, Executive Director, Vision 2020 India*

Vision 2020 aims to Eliminate Avoidable Blindness from India. The National Forum (VISION 2020: The Right to Sight – INDIA) is a key driver of the World Health Organization-International Agency for the Prevention of Blindness (WHO - IAPB) joint global initiative for the elimination of avoidable blindness from India.

It aims to eliminate the main causes of avoidable blindness in India by facilitating the planning, development and implementation of a sustainable national eye care programme (NPCB) based on the three core strategies of disease control, human resource development and infrastructure and technology, incorporating the principles of Primary Health Care (PHC), integrated within the NRHM (National Rural Health Mission) in the National Health System.

Its key strengths are leadership, passion, knowledge, skills, experience and commitment brought together by like-minded member organizations to fulfill our vision and mission. 80% of blindness in India is because of cataract and uncorrected refractive errors. History has provided it (VISION 2020 INDIA Forum) with a unique privilege and opportunity – to work as a team in mission mode with a laser sharp focus - to eliminate avoidable blindness to a level that it ceases to be a public health problem for our citizens residing in our 626 districts in India.

Vision 2020 is focusing on six key strategic areas to produce an impact, i.e.,

- Advocacy for Eye Health
- Policy & Program Development
- Quality in Eye Care
- Resource Mobilization & Sustainability
- Resource Center
- Organizational Development

Vision

"An India free of avoidable blindness, where every citizen enjoys the gift of sight and the visually challenged have enhanced quality of life as a right"

Mission

"To work with eye care organizations in India for the elimination of avoidable blindness by provision of equitable and affordable services as well as rehabilitation of visually challenged persons through development of appropriate policies, quality standards, advocacy, training, and promotion of best practices with a special emphasis on the poor and marginalized sections of society and underserved areas"

Core Values

VISION 2020: The Right to Sight – INDIA is committed to being a transparent, accountable, inclusive and sustainable organization that respects all its members and stakeholders whose participation is actively sought in democratic decision-making and organizational learning. It promotes quality and equity in eye care, with the highest ethical standards.

What is VISION 2020?

The global initiative (VISION 2020: The Right to Sight) was formed 10 years back by the World Health Organization (WHO) and the International Association for Prevention of Blindness (IAPB) to eliminate causes of avoidable blindness worldwide by the year 2020.

The National Body VISION 2020: The Right to Sight – INDIA, formed five years back in 2004, as a *member-based forum, represents the NGO Sector in Eye Care* and works with the Government and other stakeholders to eliminate avoidable blindness from India.

Why should your Organization be a member of VISION 2020 INDIA?

VISION 2020 INDIA provides an invaluable, historic opportunity to Eye Care Organizations and allied stakeholders to become part of a movement and team focused on eliminating causes of avoidable blindness from India, as was done for eradication of smallpox.

VISION 2020 INDIA is focusing on six key strategic areas to produce an impact, i.e.,

- Advocacy in Eye Care
- Policy & Program Development
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- Organizational Development

What have we achieved till date (2010?)

Policy and Programme Impact

- It has worked with the Government of India to formulate the XIth Five Year Plan (2007-12) for Blindness Control.
- This resulted in (1) inclusion of newer/emerging areas (Diabetic Retinopathy, glaucoma, childhood blindness) with focus on comprehensive eye care, and (2) an increased budgetary allocation of 1,250 crores over five years from 500 crores utilized for the Xth Five Year Plan.

Impact on service delivery

- It conducted thematic workshops to promote best practices and improve quality of service in the areas of Diabetic Retinopathy, Corneal Blindness, Childhood Blindness,

Establishing Vision Centres, use of IT in Eye Care & Comprehensive Eye Care & Resource Mobilization & Sustainability.

- Regional level workshops with Government participation to address the Grant-in-Aid issues and implementation of the National Program in a Public Private Partnership (PPP) mode.
- It worked with the States for the development of VISION 2020 plans in 5 States of India.
- In partnership with the NPCB, MOH, GOI, it has now conducted World Sight Day (WSD) Celebrations for the past six years. The 2009 Theme focused on "Gender and eye health – equal access to care"
- Undertaken publications for disseminating information & promoting best practices in Community Ophthalmology, Management of Diabetic Retinopathy & Setting up secondary level eye hospital.
- It has partnered with the Corporates to enhance the technical quality of our member organisations.
- Since the past two years, it has worked with the All India Ophthalmological Society (AIOS) to conduct sessions about elimination of avoidable blindness from India at its Annual Conference.

Work in Progress

- The development of HMIS for NPCB is in progress and it is closely working with NPCB for its implementation for better program management and administration, especially with relation to Grant-in-aid.

With a mandate to work closely with the Ministry of Health, Govt. of India, during these years VISION 2020 INDIA has forged a true partnership and good working relationship with the Government. This has been possible essentially because of the involvement, participation, support and commitment of the members.

To know more about VISION 2020 please look up at www.vision2020india.org

World Sight Day 2009

This national celebration was organized jointly with National Program for Control of Blindness (NPCB) Govt. of India successfully for the sixth time in a row.

WSD 2009 was based on the Gender equity theme: "Gender & eye health – equal access to care". As part of the national celebrations organized in New Delhi, a symposium was organized on the WSD theme on 7th October, 2009 followed by the Public function of WSD on 8th October, 2009. The event was attended by Govt. Officials, Delhi-based VISION 2020 INDIA member organizations, Ophthalmologists, Ophthalmic paramedics, Students & development NGO.

Global Theme for World Sight Day – October 14th 2010

"Countdown to 2020"

Brand Ambassador

Our Brand Ambassador Padmashree Smt. Hema Malini, Member of Parliament has prepared a short film to advocate for the prevention of avoidable blindness. This film can be previewed at http://www.vision2020india.org/homepage.asp

Dr. APJ Abdul Kalam at the 5th AGBM of VISION 2020 INDIA

Former President of India, Dr. APJ Abdul Kalam, was the chief guest at the 5th AGBM held in Patna on July 16, 2009 in the presence of Bihar State Health Minister. He motivated the audience which mainly consisted of Ophthalmologists, Govt. Officials and representatives from Development organization, Eye Care institutions, International NGOs etc. to take up the challenge to make Bihar free from avoidable blindness and pitch in with all possible support to the state Govt. to fulfill this mandate.

Publications

Community Eye Health Journal with Indian Supplement (CEHJ-IS)

- Published quarterly
- Circulated free of cost to more than 5000 readers
- Soft copy available at http://www.vision2020india.org/ceh.asp
- Please write to cehjindia@vision2020india.org for related enquiries.

Priced Publications

a. Manual on Equipping Secondary Eye Hospital

b. Guidelines for Management of Diabetic Retinopathy in India

- Hard copy: contact Secretariat at info@vision2020india.org
- Soft copy available at http://www.vision2020india.org/publications.asp

Membership Information

The strength of VISION 2020 INDIA is its member organizations committed to elimination of Avoidable Blindness in India.

Organizations desirous of contributing their knowledge, skills and experience to make its vision a reality can request the Board to provide them with this unique opportunity.

Please download the membership application package from www.vision2020india.org.

Together, we will eliminate blindness from India.

Zone Legend

- North
- East
- West
- South

Opportunities provided to Members

Make your Voices heard.

Network and share experiential learning and best practices by

- Participating in Zonal meetings & Workshops
- Presentation by Member Organizations in AGBM coordinated by Zonal representative on the Board

- Participate in meetings organized by NPCB (Central & State level) whenever possible.
- Access to publications and reports on a wide range of subjects.
- Directory of Members
- Community Eye Health journal with Indian Supplement
- Information on important events organized BY VISION 2020 INDIA and other activities, press releases, membership etc.
- Govt. of India Circulars related to Ophthalmology & rehabilitation of the visually challenged

Contribute to Policy development

- Participate & contribute in National & State level Consultations on planning & policy matters.
- Get expert advice on Government programs and policies of eye care.
- Representations to Central & state Governments and INGOs.

Advocacy Roadmap – Issues

Prioritized Critical Issues for Advocacy

- HMIS
- Diploma in Ophthalmic techniques (MLOP) Training – Curriculum and Accreditation
- Cataract Quality / Cluster Outbreak guidelines/ directive
- Eye Health Mass Media Awareness Campaign
- Inclusive and integrated services : Especially with ICDS and SSA
- Equity and accessibility mainly related to gender as well as rural/urban issues/low HDI districts
- Public Private Partnership at Primary Health Centres for Eye Care

Management Board (2010-11)

Office Bearers

- Dr. GV Rao
  ORBIS International
  President
- Dr. Supriyo Ghose
  RP Centre (AIIMS)
  Vice President
- Dr. Taraprasad Das
  LVPEI
  Secretary
- Ms. Elizabeth Kurian
  Sightsavers International
  Treasurer

Six Zonal Representatives

- Ms. Tanuja Joshi
  Venu Charitable Society
- Rtn. Paritosh Das
  JPM Rotary Eye Hospital
- Dr. Jennifer Basaiawmoit
  SPECS
- Col. (Dr.) M. Deshpande
  PBMA's HV Desai Eye Hospital
- Mrs. K. Mani Mala
  Sankar Foundation
- Dr. BK Jain
  Sadguru Netra Chikitsalaya

Ex-Officio Member

- Dr. Rajesh Noah
  Executive Director

Vision 2020 India Technical Strategic Areas

Strategic Area 1: Advocacy for Eye Health

Strategic Area 2: Policy & Program Development

Strategic Area 3: Quality in Eye Care

Strategic Area 4: Resource Mobilization & Sustainability

Strategic Area 5: Resource Center

Strategic Area 6: Organizational Development

Editorial comments: The vision 2020 overview Global and Indian perspective has been published in DOS Times March, 2006.

Dr. Rajesh Noah
Executive Director, Vision 2020 India

Dr. Harbansh Lal

Comparison of Macular Thickness Between Cirrus HD-OCT And Stratus OCT

Masashi Kakinoki, MD; Osamu Sawada, MD; Tomoko Sawada, MD; Hajime Kawamura, MD; Masahito Ohji, MD

[Ophthalmic Surg Lasers Imaging 2009;40:135-140.]

Ever since the invention of Optical Coherence Tomography (OCT) by Huang et al in 1991, it has become an essential tool for diagnosing and monitoring retinal diseases. While Time Domain-OCT (TD-OCT) measures the echo delay time of light that is reflected back from the retinal microstructures, Spectral Domain OCT (SD-OCT) uses the spectrometer to resolve the relative amplitude and phases of the spectral components of light using Fourier transformation. The latter is hence 50 times faster than TD-OCT and this substantial increase in the speed helps one to acquire 3D datasets.

In the current study, both the machines were found to give reproducible results although mean CV (coffiecient of variation) of SD-OCT was significantly smaller than that of TD-OCT.

One important finding was that the mean macular thickness with SD-OCT was 60 µm thicker that TD-OCT, and the difference was significant (p value< 0.001).

The difference in the definition of the retinal thickness between the two OCT machines explains this difference. While TD-OCT defines retinal thickness as the distance from the surface of the internal limiting membrane to the boundary between the inner and outer segments of the photoreceptors, the SD-OCT defines the retinal thickness as the distance from the surface of the internal limiting membrane to the surface of the retinal pigment epithelium.

Clinical implication

Although both the machines give reproducible results, the SD-OCT has better reproducibility. The mean retinal thickness measured with SD OCT is around 60 µm thicker than TD –OCT .So, if a patient comes with prior scans of macular thickness using TD-OCT, one can not directly compare it with macular thickness achieved with SD-OCT and vice versa. That is why one must be cautious while comparing retinal thickness between the two OCT machines.

Dr. Nidhi Panwar

Clinical Factors Related to Visual Outcome in Central Serous Retinopathy (CSR)

Aggio, Fabio B; Roisman, Luiz; Melo, Gustavo B et al. Retina.

30(7):1128-1134, July/August 2010.

CSR is one of the most common retinal diseases encountered in our clinics. In the present study the authors have attempted to describe clinical, angiographic and tomographic prognostic factors of CSR (prospectively). In this study 46 eyes of 43 patients with active CSR were included and followed from March 2003 to July 2007. Best Corrected Visual Acuity (BCVA), FFA, ICG Angiography (if doubtful lesions on FFA) and OCT were done in all the patients. Laser treatment was done in 5 out of 46 eyes. The investigators found significant correlation with final BCVA was noted for the following factors: Baseline BCVA, duration of symptoms, focal leak pattern on FFA (On FFA lesions were graded as focal leak/ multifocal leak/ multiple window defects), shorter period of subfoveal fluid on OCT (Duration of neurosensory detachment involving fovea on OCT), and central macular thickness on OCT. Laser treatment assisted in early resolution of neurosensory detachment howver, did not result in better final visual acuity when compared with the observation group.

Clinical Implication

The following guidelines could be followed based on the above article:

(a) If BCVA is ≥ 6/18, and the duration of symptoms is less than 6 weeks the patient can be followed up with serial fundus photographs and OCT scans every 6 weeks. The prognosis for this group of patients is good.

(b) If BCVA is ≤ 6/18, and the duration of symptoms is more than 6 weeks, the patient should be advised FFA and OCT at the first visit. The BCVA, duration of symptoms and pattern of leakage guide the prognosis of the patient.

The Laser treatment in form of laser photocoagulation/ fomentation is usually indicated for non resolving CSR, chronic CSR and recurrent CSR. The laser treatment help in early resolution of the fluid but doesn't result in better BCVA.

Dr. Naginder Vashisht

Dose of Intravitreal Bevacizumab (Avastin) used as Preoperative Adjunct Therapy for Proliferative Diabetic Retinopathy

***Takayuki Hattori** et al. Retina 30:761-64, 2010*

Bevacizumab (Avastin) is currently being used not only as therapeutic agent for the treatment of Diffuse Diabetic Macular Edema but also as a preoperative adjunctive therapy in patients undergoing vitrectomy for proliferative diabetic retinopathy (PDR). The dose used in both of the above modalities is currently 1.25mg with therapeutic effect lasting upto 3 months.

The effect of Bevacizumab in case of PDR is required for only shorter duration as a measure to decrease intraoperative bleeding. Studies have shown occurrence of retinal artery occlusion, enlargement of foveal avascular zone and onset or progression of tractional retinal detachment which may be dose related.

Therefore through this study author has tried to find out the minimal effective dose in case of PDR as preoperative adjunct therapy, by diluting the dose of 1.25 mg with normal saline to form lesser doses 0.63mg, 0.31mg and 0.16 mg and administered 3 days prior to surgery. A total of twelve eyes were included in bevacizumab group out of which 2 eyes got 1.25 mg dose, 4 eyes got 0.63 mg, and 3 eyes got 0.31mg and 3 eyes received 0.16mg dose.

The current study utilizes a reduced dose of 0.16mg to achieve significantly lower vitreous VEGF concentration along with decrease in intraoperative bleeding as measured by reduced number of coagulation spots administered for hemostasis as compared to patients who did not receive adjunctive Bevacizumab therapy. There was no correlation between the number of intraoperative coagulation spots and the bevacizumab dose where as number of intraoperative coagulation spots differed significantly between bevacizumab and non bevacizumab group.

Clinical Impression

Though the results of the study are encouraging as lesser dose will also prevent rise in IOP following intravitreal injection of bevacizumab, yet the small number of eyes treated (twelve eyes) preclude the derivation of a standard dose for the adjunct therapy in PDR. Further studies with larger number of subjects will be required for standardization of treatment dose.

Dr. Rohini Grover

Descemet's Stripping Automated Endothelial Keratoplasty (DSAEK) Outcomes Compared with Penetrating Keratoplasty (PKP) from the Cornea Donor Study

Ophthalmology 2010; 117: 438–444

Until recently, penetrating keratoplasty (PKP) was the standard procedure for the surgical treatment of Fuchs' dystrophy or pseudophakic/aphakic corneal edema. Nowadays, the newer technique of endothelial keratoplasty i.e, DSAEK has become quite popular. However, as a clinician, what concerns one most is what would be the graft survival and the endothelial status after DSAEK as compared to the time proven technique of PKP. Should one shift to DSAEK or rather when should one prefer DSAEK?

The above mentioned article describes the results of a prospective study which examines the outcomes of DSAEK compared to the outcomes of PKP, in terms of graft survival rate and the endothelial cell loss. In the Specular Microscopy Ancillary Study (SMAS), PKP results of 410 patients were studied. These results were compared with the outcomes of 173 patients, included in this study, who underwent DSAEK.

The percent endothelial cell loss was 34 +/- 22% versus 11+/- 20% (6 months) and 38+/- 22% versus 20+/- 23% (12 months) in the DSAEK and PKP groups, respectively (both $P<0.001$). The regraft rate within 15 months was 2.3%(DSAEK group) and 1.3%(PKP group).

Overall, at one year post-transplantation, graft success was comparable for DSAEK and PKP procedures and endothelial cell loss was higher with DSAEK.

Clinical Relevance of this study

Even though the regraft rate and the endothelial cell loss is higher with DSAEK patients as compared to PKP patients, the problems associated with PKP like unpredictable astigmatic changes, risk of traumatic wound dehiscence and suture related problems are eliminated. Visual rehabilitation is also faster with DSAEK. So when the availability of corneas is not an issue, DSAEK would be a preferred surgery. PKP can also be performed after DSAEK if the endothelial graft fails. Thus, at least initially, the DSAEK procedure results in significantly greater loss of cells than PKP, particularly within the first 6 months of the procedure,principally related to greater surgical manipulation and trauma. Longer term, 5-year data on endothelial cell survival after DSAEK, would provide a more definitive determination of behavioral differences in the endothelial cell population of the DSAEK and PKP groups.

Dr. Swapna Parekh

Forthcoming Events: National

October 2010

1-3 HYDERABAD, ANDHRA PRADESH

Thirty-Fourth Annual Meeting of Andhra Pradesh Ophthalmological Society, 2010
Venue: Hyderabad International Convention Center (HICC), HICC - Novotel Complex, Cyberabad
Hyderabad 500081
Telephone: + 91-40-66134422,
E-mail: enquiries@hicc.com
http://www.hicc.com

22-24 UTTARAKHAND

NZOS & Uttara Eyecon 2010
(Combined Annual conference of North Zone Ophthalmological Society and Uttarakhand State Ophthalmological Society)
Venue: Hotel Park Plaza, Mall Road, Mussorie, Uttarakhand
Organizing Secretary:
Dr. B.K.Oli
57, Haridwar Road, Dehradun-248001
Phone: 0-99971-22222, 0-94123-19035
Email: dr.bkoli@gmail.com

28-31 NEW DELHI

10th Annual Meeting of Uveitis Society of India
Venue: Advanced Eye Centre, PGI Chandigarh
Contact Person & Address
Dr. Vishali Gupta
email : vishalisara@yahoo.co.in
www : http://www.usi2010.in

November, 2010

12-14 NEW DELHI

20th Annual Conference of Glaucoma Society of India & 5th International Congress on Glaucoma Surgery
Venue: Le Meridien Hotel, Janpath, New Delhi & India Habitat Centre, Lodhi Road, New Delhi
Conference Secretariat:
Dr. Harsh Kumar
D-8/8127, Vasant Kunj, New Delhi-70
(M): 9810442537, Tel.: 91-11-4519910, 25513051,
Fax: 91-11-26122053

14 NEW DELHI

Mid-term Conference
Delhi Ophthalmological Society
Venue: India Habitat Centre, Lodhi Road, New Delhi
Contact Person & Address
Dr. Amit Khosla, Secretary DOS
Room No. 2225, 2nd Floor, New Building,
Sir Ganga Ram Hospital,
Rajinder Nagar, New Delhi - 110 060
Ph.: 011-65705229, ***E-mail:*** dosrecords@gmail.com
Website: www.dosonline.org

December 2010

2-4 MYSORE

19th Annual Conference of Vitreo Retina Society - India 2010
Organizing Secretary,
Retina Institute of Karnataka
#122, 5th Main Road, (Next to Venlakh Hospital)
Chamarajpet, Bangalore - 18
Ph: +91-80-22410106 / 536 (Hospital)
Fax: +91-80-26607811
E-mail: retinainstitute@sify.com

Forthcoming Events: International

September, 2010

4-8 PARIS

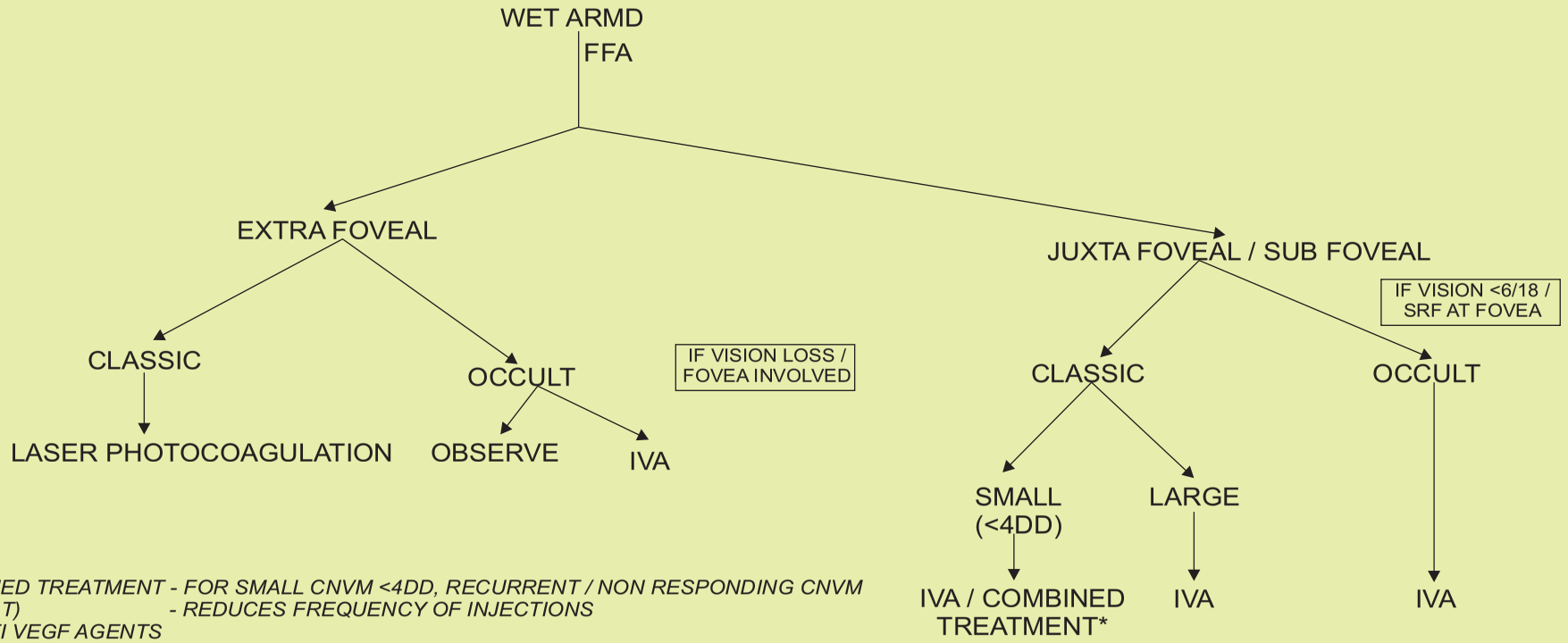
XXVIII Congress of the ESCRS
Venue: Le Palais des Congrès, Porte Maillot, Paris, France
Organiser Secretary
ESCRS, Temple House, Temple Road, Blackrock, Co. Dublin, Ireland.
Tel: +353 1 209 1100 Fax: +353 1 209 1112
Email: maria.crowley@escrs.org
Web: www.escrs.org | www.euretina.org

September, 2010

16-20 BEIJING, CHINA

APAO-AAO Joint Congress
China National Convention Centre, Beijing
APAO Central Secretariat:
Secretariat, Asia Pacific Academy of Ophthalmology
C/o. The Chinese University of Hong Kong,
Dept. of Ophthalmology & Visual Sciences, Hong Kong Eye Hospital, 3/F, 147K Argyle Street, Kowloon,
Hong Kong
Email: secretariat@apaophth.org, ***Tel:*** (852) 2762-3042,
Fax: (852) 2715-9490, ***Website:*** www.apao2010beijing.org

Management of CNVM (WET AMD)



**COMBINED TREATMENT - FOR SMALL CNVM <4DD, RECURRENT / NON RESPONDING CNVM (IVA+PDT) - REDUCES FREQUENCY OF INJECTIONS*
IVA - ANTI VEGF AGENTS

Followup: (I) Every 4 weeks (Lucentis) (ii) Every 6 weeks (Avastin / Macugen)
Visual Acuity
Serial OCT Scans for : - Membrane Activity
- Subretinal Fluid (SRF)
- Intra Retinal Edema
- Pigment Epithelial Detachment (PED)
Colour Photography
Repeat FFA: - If fresh hemorrhage (Suspected or Recurrent CNVM)
- Fresh CNVM

Treatment Schedule
Schedule - I
-4 weekly injection
(Irrespective of OCT)
(MARINA Study
ANCHOR Study)

Schedule - II
3 One Monthly Injections
↓
OCT Based Treatment
(Pronto Study) - **1 monthly** followup
- Repeat Injection if SRF ++
Increase in retinal thickness >100µ

**We recommend this schedule*

Schedule - III
3 One Monthly Injections
↓
OCT Monitoring (4-6 weekly)
↓
Repeat Injection Till macula is Dry
↓
Increase / Decrease visit by one week if macula remains Dry/Fluid Re-accumulates respectively

[1]***Naginder Vashisht*** *MD,* [1]***Amit Khosla*** *MD,* [2]***Sanjeev Gupta*** *MD,*
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[2]***Siri Fort Laser Eye Centre,*** *New Delhi*