DEJ
DiabeticEye Journal

ISSN 2055-1282
September 2014 / Issue 3

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What to consider in Cataract surgery?

Treatments: Current oral agents used in treatment of diabetes

Diabetic Eye Disease: When do we operate for diabetic retinopathy?

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DiabeticEyeJournal

EDITORIAL

Our third issue of DiabeticEyeJournal has made it! Thanks to the entirely voluntary, hard work of publishers (BARS), proof-readers (the Council members), designer (screener/grader) and all contributors. The Journal is a joint venture and we would like to say a big thank you to all who put their time and effort into it. Of course not forgetting advertisers, who support us financially, and without whom there would not be a hard copy of this Journal.

We want to make sure that we are able to bring you interesting and stimulating articles in each of our issues. Therefore we are always on the lookout for contributors who can present interesting articles to our readers. If you are one of them email us at *info@diabeticeyejournal.org*

As you may have noticed we aren't giving much away in regard of what will come out in our next Spring issue. This is mainly because the finances aren't in place yet. I would stress the word yet, becuse people are working to secure this essential part as we speak. So with the best of luck and a great deal of networking we will hopefully publish our Spring 2015 issue as well.
This Journal will strive to keep bringing you worthwhile articles, which we hope will enrich your professional life and help to keep our profession more stimulating and interesting. Happy reading.

IN THIS ISSUE

PUBLISHER

BARS Council

ONLINE VERSION

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COMMENTS CONTRIBUTIONS

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DiabeticEyeJournal is published and produced by BARS with support of North Central London Diabetic Eye Screening Programme from NMUH NHS Trust.

Courses

Diabetic Retinopathy Screening Training

Clinical Tutorial Centre (CTC)
Moorfields Eye Hospital, London EC1V 2PD
20th and 21st October 2014, London

To register:
http://www.readingcentre.org/Training/Default.aspx

Understanding Diabetic Retinopathy - diagnosis and management

Institute of Ophthalmology,
Bath Street, London EC1V 9EL
03 to 05 February 2015

http://www.moorfields.nhs.uk/list/events

Training Courses at Retinopathy Screening Centre, Heartlands Hospital, Birmingham

DR Screener Course: 23 to 24 October 2014
OCT Course: 03 to 04 December 2014
DR Grader Course: 12 to 16 January 2015

http://www.retinalscreening.co.uk

Diabetes UK – Diabetes Awareness Training

Various dates and in-house training available.
One-day course to provide participants with the knowledge and confidence when working with people with diabetes, accredited by the Royal College of Nursing (RCN)
Phone: 020 7424 1000

Contact enquiry: *commissioning@diabetes.org.uk*

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Diabetic Retinopathy Screening Training Alumni Day

Thursdayay 09 October 2014
Venue: UCL's Senate House, Malet Street, LONDON WC1E 7UH

To register:
http://www.readingcentre.org/Alumni/index.aspx

South West Professional Conference

Diabetes UK, aimed at Healthcare Professionals and Commissioners
Thursday 30 October 2014
Venue: Taunton Racecourse

Email: *south.west@diabetes.org.uk*
Phone 01823 448260

OIA 2014 Conference

14th & 15th November 2014,
Stratford Manor Hotel,
Stratford Upon Avon

To register:
http://www.oia.org.uk/pages/default.asp?id=2

Diabetic Screening Conference

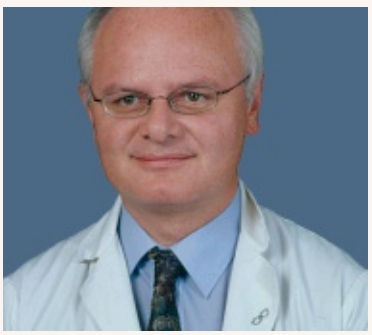
Royal Society of Medicine
1 Wimpole Street, London W1G 0AE
24th April 2015

To register: *ophthalmology@rsm.ac.uk*

Mr Mustafa R. Kadhim - Vitreoretinal Fellow at Moorfields Eye Hospital

Mr Paul Sullivan – Consultant Vitreoretinal Surgeon at Moorfields Eye Hospital

Surgical techniques in diabetic vitrectomy



Mr Paul Sullivan

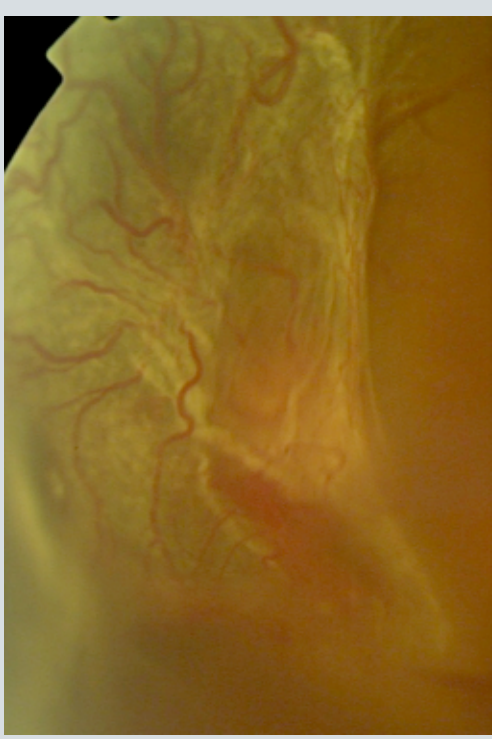
In this article we aim to delve into greater detail regarding the various surgical techniques involved in the treatment of diabetic eye disease. The pre-operative management, surgical preparation and then indications for surgery will be elucidated in turn. The aim is a comprehensive understanding of why, what and how the vitreo-retinal team deals with the various degrees of advanced diabetic retinopathy.

Preoperative management

Many diabetic patients are receiving some form of anticoagulant or anti-platelet therapy. The risk of postoperative haemorrhage may be reduced if these are discontinued in the perioperative period. This should only be done after consulting with the treating physician. Patients with drug eluting coronary stents are at particular risk and great caution should be exercised in stopping anti-platelet agents in this group. Many patients placed on long-term aspirin for other indications (e.g as prophylaxis after a previous myocardial infarction) are at much lower risk from temporary suspension of treatment. In the case of warfarin therapy the international normalized ratio (INR) is checked to ensure that it is within the therapeutic range.

If the retinopathy is poorly controlled preoperative supplementary photocoagulation should be considered. If there is limited vitreous haemorrhage an indirect laser deliver system may allow treatment to be given. Alternatively a preoperative injection of an anti-VEGF agent reduces perfusion of the neovascular complexes and makes surgery technically more straightforward. The timing of this injection is quite important. A delayed cicatricial response may cause progression of detachment in some cases. The beneficial effects may be apparent as soon as 48 hours after injection. Most surgeons perform surgery within one week of injection.

Figure 1 – effect of intravitreal anti-VEGF agent.

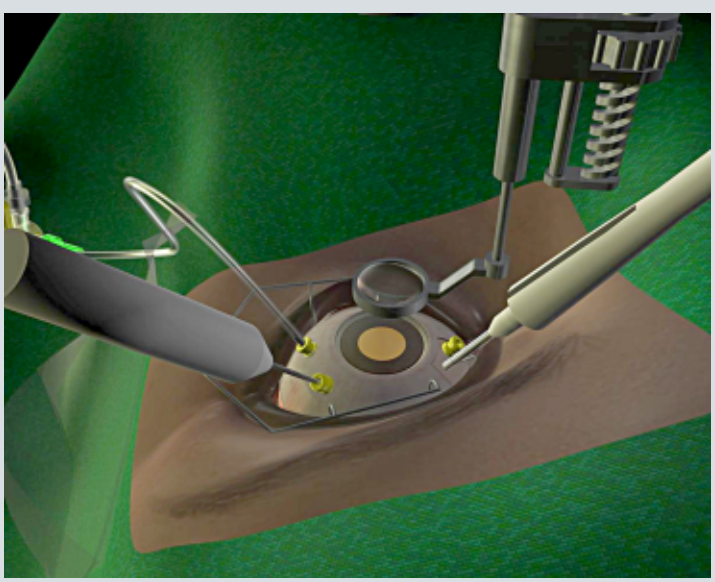


Surgical Preparation

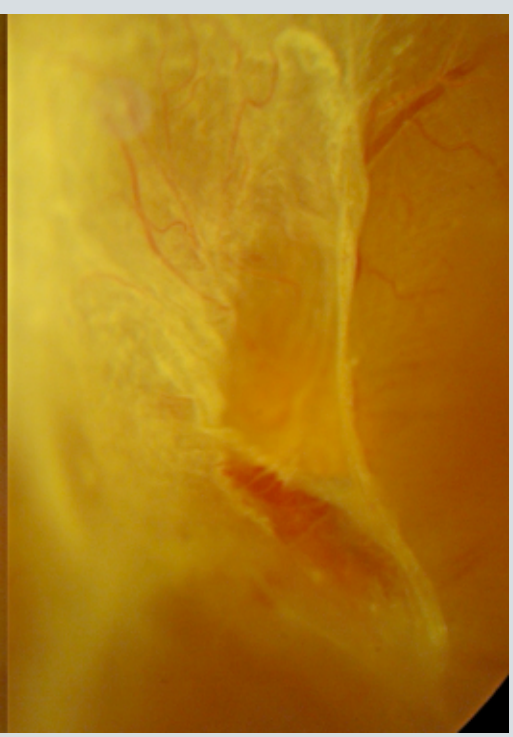
Most cases of diabetic vitrectomy, including complex delamination surgery, can now be performed using micro-incision vitreoretinal surgery (MIVS). The refined fluidics and proximity of the aspiration port to the tips of vitrectomy instruments allows controlled and precise excision of fibrovascular complexes. The use of cannula entry site systems has considerably reduced the incidence of entry site breaks that were frequent when performing 20-gauge surgery. If a 20-gauge instrument such as a horizontal delamination scissors is required a single separate sclerotomy may be made rather than attempting to enlarge a 23- or 25- gauge sclerotomy. This 20-gauge sclerotomy is then closed at the end of delamination, allowing the rest of the case to be performed with watertight seals around the MIVS instruments.

Diabetic patients are prone to corneal epithelial oedema during delamination surgery. The use of non-contact viewing systems reduces the probability of corneal abrasion. Switching from a wide field to a 90 Dioptre lens provides a good balance between width of field and depth of focus when dissecting posterior membranes.

Figure 2 – 23G and 20G entry combined, non-contact lens



Notice the reduced calibre of vascularity of the neovascular complex.



The next section will deal with the indications of surgery and the surgical particulars of each shall be considered.

Vitreous Haemorrhage

In cases with fundus obscuring vitreous haemorrhage it may be difficult the check that the tip of the infusion is correctly sited in the vitreous cavity. If there is any doubt a light pipe may be passed through the infusion cannula - the light can be seen easily even in dense vitreous haemorrhages.

The vitrectomy should proceed cautiously initially. In time the view will start to clear and fundal details will start to appear. It will then be possible to cut more peripherally and posteriorly. The vitreous in the superior mid-periphery should be cleared first so that, if a break is inadvertently created, it is superior and easier to deal with. Another advantage of starting in the mid-periphery is that there are less likely to be strong vitreoretinal adhesion in this area. The way the vitreous moves should be observed. Detached vitreous moves freely while being cut and the impression that the vitreous movement is tethered may be a clue to the presence and location of adherent membranes behind the blood. Cutting should be performed initially where the vitreous seems most mobile.

If a posterior vitreous detachment is present the liquefied puddle of retrohyaloid blood forms a plume to the cutter when a hole is made in the posterior hyaloid. When this is seen the cutter should be kept in place to aspirate the blood. It is usually safest to stay in cut mode initially rather than switch to active aspiration in order to avoid traction and any residual vitreous. The liquefied retrohyaloid blood is denser than saline and pools on the retinal surface from which it may be aspirated once the hyaloid has been cleared sufficiently. If fibrovascular membranes are present reflux can be used to displace it. The jet of fluid blows blood away from the retina so that it can be safely aspirated in the central vitreous cavity. Areas of blood that are immobile during reflux indicate the presence of overlying membranes or attached hyaloid.

Trapped blood in the anterior hyaloid may degrade the fundal view. It can be stripped posteriorly off the lens capsule - this may be difficult to achieve in younger patients. Combining vitrectomy with cataract surgery allows this to be performed without fear of lens touch.

Once the view is sufficiently clear indented shaving of the vitreous base is performed to reduce early vitreous cavity haemorrhage. This may be deferred to the end of the procedure in phakic patients to avoid the possibility of inadvertent lens touch degrading the fundal view.

Relief of traction in diabetic eyes – general principles

Relief of traction on the retina allows tractional detachments to resolve and reduces the risk of post vitrectomy haemorrhage. Even cases with an apparently complete vitreous detachment may have areas of adherent schitic vitreous. Intravitreal triamcinolone allows these to be seen. The triamcinolone is in the form of an almost chalky white suspension that sticks to the residual gel, allowing it to be clearly visualized.

In the early days of vitrectomy only anteroposterior traction could be relieved. The development of segmentation - division of membranes with vertical scissors - was a significant advance that allowed the relief of bridging traction. The residual islands of fibrovascular tissue were particularly likely to cause postoperative haemorrhage however. This problem was solved by the introduction of techniques to completely remove all the fibrovascular membranes. This relieves all traction (anteroposterior, bridging and tangential). This was initially performed with horizontal scissors. This surgical goal can now often be achieved with ultrahigh speed vitreous cutters. These can be used as multifunction tools capable of segmentation, delamination and gentle elevation of non fibrovascular membranes.

Segmentation

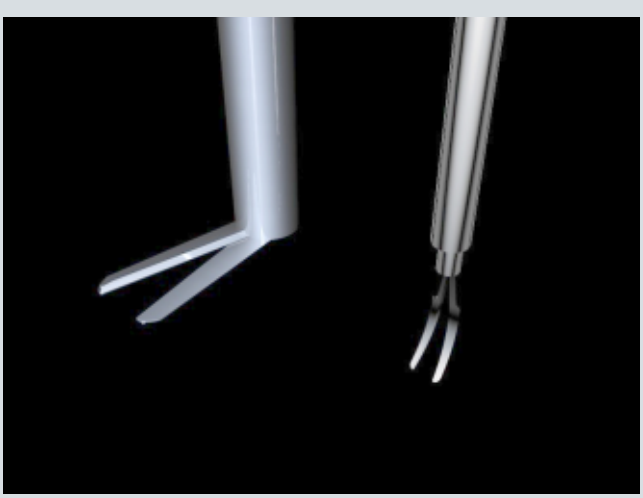
Segmentation still has a role in providing access to fibrovascular pegs. It may be carried out with scissors or a vitrectomy cutter. It is not usually an end in itself, merely a means to the end of complete excision of the fibrovascular membrane ('access segmentation').

Scissors designed for segmentation have blades which are not aligned with the shaft and so cannot be introduced through MIVS cannulas. The curved blades designed for segmentation may be used for access segmentation by tilting them slightly. Segmentation may also be achieved with MIVS cutters due to the proximity of the port to the end of the shaft.

Delamination – general principles

Fibrovascular pegs securely anchor the hyaloid to the retina. If these are stripped away there is a significant risk of retinal breaks and haemorrhage due to avulsion of a small plug from wall of the vein. Although the pegs may bleed slightly when transected the bleeding is far less and easier to control than the haemorrhage resulting from avulsion of the wall of the retinal vein. The fibrous tissue that surrounds the new vessels within the fibrovascular membrane impedes the physiological vasospasm that normally follows vessel injury. The bleeding, although slow, is prolonged and may be more difficult to control than the more isolated bleeding from a divided peg. The peg should therefore be transected between its origin in the vessel wall and its entry point into the fibrovascular membrane. This is relatively straightforward in eyes with partial vitreous separation around the peg as there is a clearly visible plane between the hyaloid/fibrovascular membrane and the retina. The challenge of complex delamination surgery is that frequently no such plane exists. It has to be created without undue traction on the peg (as bleeding makes the correct plane for dissection even harder to define).

Figure 3 – 20G (right), 23G (left) delamination scissors. Note the curve on the 23G design to allow entry via the cannula.



Delamination Techniques

The en bloc technique involves leaving much of the hyaloid in place initially. Vitreous around the sclerotomies is removed to avoid entry site breaks. Dissection starts at the edge of the fibrovascular membrane. The residual hyaloid elevates the fibrovascular membrane as it is dissected off the retina. Traction may be transmitted by the posterior hyaloid to the peripheral retina causing peripheral retinal breaks. For this reason many surgeons prefer to perform delamination 'inside out'. Dissection starts over the posterior pole working outwards. When starting dissection over the optic disc the fibrovascular tissue is first avulsed from the disc with forceps. This usually causes haemorrhage from the veins in the optic nerve head.

Cutter delamination

Micro-incision vitrectomy cutters offer improved fluidics, faster cut rates and cutting apertures closer to the end of the end of the shaft. These properties have considerably extended the scope of the surgeon to perform surgery in diabetic eyes with the cutter alone. Steve Charles has coined the terms 'conformal delamination' and 'foldback delamination' to describe techniques used to excise fibrovascular membranes with the cutter. Both techniques rely on the membrane around the vascular pegs being mobile.

Once the 'business-end' of the surgery is completed successfully (vitrectomy, segmention/delamination), then the following must be considered.

Endolaser

If the retina has ischaemic features supplementary panretinal photocoagulation (PRP) is performed. This reduces postoperative posterior reproliferation (around any residual membranes) and anterior vasoproliferation (rubeosis and anterior hyaloid proliferation).

When performing PRP:

Preretinal blood is removed prior to laser. Laser to preretinal haemorrhages results in inner retinal burns. Nerve fibre layer loss causes extensive arcuate scotomas.

A curved endoprobe is used to treat the equatorial retina from the opposite side (e.g. treatment of nasal retina with the laser in a temporal port). The angle of incidence of the laser light is more perpendicular and the laser spots less oblique.

Whenever possible the laser probe is swung in an arc at a constant distance from the retina giving uniform burn size and intensity. This is also easier when treating the opposite side of the eye.

Only attached retina should be treated. Attempts to perform PRP in the presence of subretinal fluid lead to intense burns (as the power has to be very high to get a visible reaction) which causes energy propagation to the choroid. Chronic tractional detachments have viscous subretinal fluid which cannot be removed sufficiently to perform PRP safely. Areas of detached retina may be treated postoperatively when the tractional detachment has resolved.

If any eye has not had previous laser a methodical approach treating posteriorly first and than in arcs outwards may be employed to reduce the risk of losing orientation and accidentally treating the fovea.

Laser should also be performed around retinal breaks (after fluid gas exchange if there is associated subretinal fluid).

Tamponade

No tamponade agent is required unless a retinal break is present, in which case air or a short acting gas is usually sufficient.

Silicone oil is reserved for the more complex cases with anterior hyaloid proliferation or large retinectomies with unrelieved traction. The silicone-fluid interface seems to provide a scaffold for reproliferation. The threshold for using silicone oil in diabetic patients should be very high indeed.

Complications

Finally we shall look at the intraoperative complications of diabetic vitreo-retinal surgery.

The major intraoperative complications are haemorrhage and retinal breaks. These are far more likely in complex cases with little vitreous separation or in which uncontrolled proliferation is present. The two complications often go together. Uncontrolled bleeding under a fibrovascular membrane makes it more likely that a break will be made. Breaks may lead to a traction detachment becoming more mobile which makes delamination more difficult and increases the likelihood of haemorrhage.

Preoperative administration of anti-VEGF agents reduces the incidence of preoperative haemorrhage and complexity of surgery. The combination of preoperative administration of anti-VEGF agents and MIVS seems to improve visual outcomes in the most complex cases.

Haemorrhage

Intraoperative bleeding may arise from:

Transected pegs. This is usually self limiting.

Avulsed pegs. Here the traction is irising directly from the wall of the vein and may be more persistent. It may be managed by temporarily increasing the infusion pressure. Direct pressure may be applied to the bleeding point with the cutter tip for 30 seconds. Bleeding of this sort is particularly likely to occur if a fibrovascular membrane is avulsed from the disc. This may be avoided by leaving a nubbin of tissue over the disc. Bleeding from this may be managed with unimanual bipolar diathermy.

Bleeding from the cut edges of a fibrovascular membrane. This may be managed with diathermy. The fibrovascular membrane seems to hold vessels open and, paradoxically, bleeding may stop or be easier to control if the whole fibrovascular membrane has been removed.

Solid clots are probably best left if bleeding has stopped. Stripping these off the bleeding point may cause haemorrhage to restart. If very large clots are present their edges may be aspirated free of the retina and trimmed with the cutter, leaving the centre undisturbed.

Endolaser can be applied to a persistent bleeding point on the retinal surface - this is preferable to diathermy which may stick to the surface of the retina. Quite low laser power levels are used as the blood absorbs laser energy effectively.

Retinal Breaks

Posterior retinal breaks usually arise from:

Applying undue traction to the retina.

Failure to isolate a single peg before cutting with scissors.

Uncontrolled scissor cutting under a fibrovascular membrane, especially when the underlying retina is convoluted.

Poor vitrectomy cutter delamination technique.

Posterior breaks are managed by relieving all the traction on them, reattaching the retina with air-fluid exchange and applying retinopexy.

Peripheral retinal breaks arise from traction on a detached posterior hyaloid, especially around the instrument entry sites. These occur much less frequently with MIVS surgery. They are managed by retinopexy and tamponade. Scleral buckling is no longer used.

Postoperative vitreous cavity haemorrhage

Post vitrectomy haemorrhages may occur due to:

Leaching of residual blood from within the vitreous base.

Residual membranes causing traction on posterior fibrovascular complexes.

Anterior vascular proliferation.

These problems typically present at different times

after surgery. Early haemorrhage is typically due to residual blood from the vitreous base or bleeding from residual membranes. Anterior hyaloid proliferation typically presents as delayed vitreous cavity haemorrhage.

Measures to prevent postoperative haemorrhage include:

Indented trimming of the vitreous base.
Panretinal photocoagulation or even cryotherapy to the anterior retina, particularly around the sclerotomies.
Use of triamcinolone to exclude the presence of residual posterior membranes.
The presence of erythrocytes in the anterior chamber indicates a high probability that haemorrhages will clear spontaneously. Failure to clear may be managed by vitreous cavity washout, with vitreous base trimming and excision of residual membranes as required.

Anterior Proliferation

Following vitrectomy VEGF release may drive proliferation of new vessels on the residual anterior vitreous base if the retina is ischemic. In many cases the vessels seem to arise from the extraocular ciliary vessels via the sclerotomies. These entry site new vessels may simply cause recurrent vitreous cavity haemorrhages. Occasionally they follow the same sequence of fibrosis, contraction and bleeding seen with posterior retinal neovascularisation, progressing eventually to tractional detachment of the ciliary body and phthisis.
Surgical excision of advanced anterior hyaloidal proliferation is very difficult. A clean delamination plane cannot be created because of the tight vitreoretinal adhesions present in the vitreous base. Untreated it has a terrible prognosis but some cases respond to lensectomy, dissection of anterior membranes around the ciliary body and anterior photocoagulation. Retinectomies are often required as is silicone oil tamponade. Any case requiring a delayed vitreous cavity washout should be inspected for fibrovascular tissue in the vitreous base and extensive anterior photocoagulation delivered if this is present.

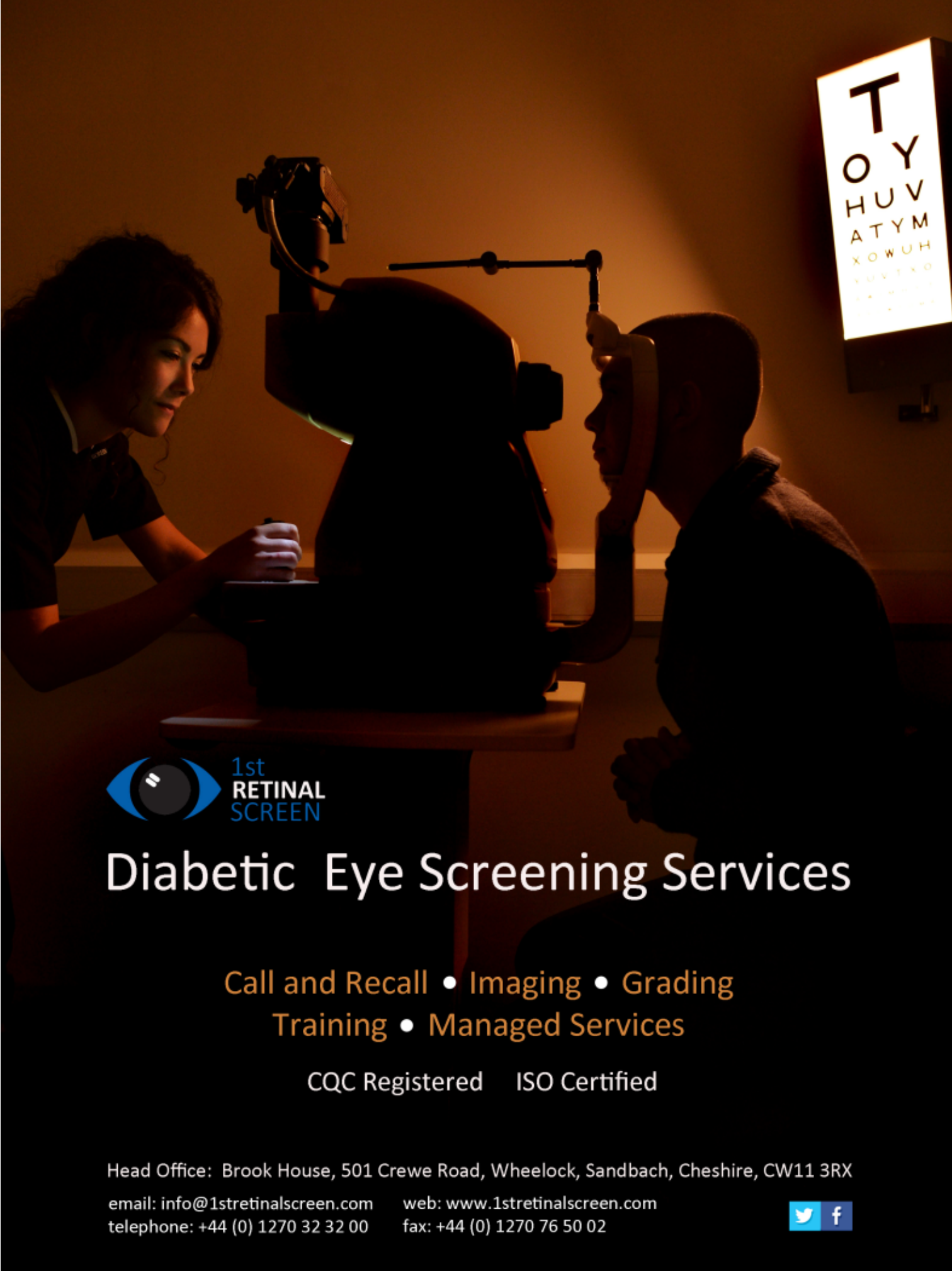
In summary

Vitreo-retinal surgery for the complications of diabetic retinopathy can aid visual rehabilitation.

Combined pharmaceutical, laser and surgical techniques are the mainstay of care.

Pre-operative, intraoperative and postoperative considerations are vital for a good outcome.

Complications exist and patients must be counseled about these prior to surgery.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Oxfordshire Diabetic Eye Screening Programme Based at Oxford University Hospitals NHS Trust

Oxford University Hospitals (OUH) NHS Trust has been providing the Diabetic Eye Screening (DES) using digital photography to the population of Oxfordshire since 2006. It was started by Dr Peter Scanlon, who remains the Clinical Lead for the service.

Prior to 2006 Diabetic Eye Screening was provided to patients of the Oxford Centre for Diabetes, Endocrinology & Metabolism (OCDEM) by accredited optometrists, this service was co-ordinated by OCDEM but was only provided to patients who were registered with them and not to the whole population of Oxfordshire.

When the service was commissioned in 2006, GP surgeries were given two screening modality options:

• Screening to take place at the GP surgery. All patients of that surgery would be screened on their premises (mobile based).

• Screening to take place in the community. All patients of that surgery would be given the option of attending one of 16 contracted optometrists (optometry based).

The surgeries who opted to have their patients screened at their premises tended to be rural and those who opted to be community based, tended to be nearer Oxford. Figure 1 shows the location of each GP surgery and the mode of screening.

In 2013 the number of contracted optometrists was reduced to 15 and, after consultation with the GP surgeries that it affected, ODESP took over screening in that area.

The contracted optometrists screen patients but do not carry out the grading of the images.

ODESP have two mobile cameras which are rotated around the GP surgeries on an annual basis; each surgery is visited twice in a screening year. We also have a camera based at the John Radcliffe Hospital and run clinics during the week, including our Digital Surveillance Clinic, Monday and Saturday clinics. We also screen once a month in the Paediatric Diabetic Clinic, screening young people aged 12-18 and at the Silver Star clinic for pregnant ladies. We also run a number of drop-in clinics at OCDEM and provide screening at two prisons within Oxfordshire.

Our Team

The programme provides screening to over 27,000 diabetic patients; we have a team of 16 including the Clinical Lead, Programme Manager, Administration Manager, Failsafe Officer in addition to the 15 contracted optometrists.

The department is split into two. The administration team oversees the call/recall of patients, setting up the mobile clinics, booking all the appointments for mobile patients, referring patients to HES, etc. The screening/grading team take the images and assess the images. The Failsafe officer works across both teams and all members of the team undertake failsafe work.

We are fortunate that the majority of our referrals to HES are made to the Oxford Eye Hospital which is situated in the same building as the screening programme and we have easy access to Electronic Patient Records and patient notes etc.

Unlike some other DESP, when we refer a patient to the HES the ODESP Admin team book the first HES appointment. We started doing this a number of years ago as we found, like many programmes, patient were not being seen in the correct time frames as appointments were not being booked in a timely manner.

As a team we are always looking at ways of improving our service to our patients. Our Failsafe Officer is currently undertaking an audit of patients referred to the Slit Lamp Biomicroscopy Clinic. As with lots of other hospital departments, demand for appointments far outweighs the supply of appointments and we thought if we only refer patients who were willing to attend appointments this would:

1. Ensure that patients were seen appropriately

2. Cut down the number of patients who DNA

Another audit being undertaken in conjunction with the other Adult screening programmes hosted by the OUH Trust is how people with learning disabilities are accessing our services and what can be done to improve access to this group of patients.

In addition Oxford DESP in conjunction with BARS, have held two successful Failsafe workshops lead by our Failsafe Officer Alyson Jaycock.

This feature was contributed to and composed by ODESP Programme Manager *Helen Lipinski* and Administration Manager *Hazel Benjamin*.

Cataract Surgery: **considerations in diabetic patients**

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Introduction

Diabetes mellitus has a major prevalence in the UK population (England 6.0%, increased incidence in patients of Indian or Chinese background (1)). As diabetes mellitus is a recognised risk factor for development of cataract ophthalmic surgeons undertaking cataract surgery in large hospitals in the UK will have up to 50% incidence of diabetes in their cataract patients. Diagnosis of cataract in diabetics has increased with the national retinopathy screening programme where cataract causes poor quality fundus photographs.

This article sets out some considerations for ophthalmologists and healthcare professionals dealing with diabetic patients undergoing cataract surgery.

Preoperative Considerations

The decision to undertake surgery for cataract causing visual symptoms is made after discussion with the patient of likely benefits in visual function and possible complications during and after surgery. Comorbidities such as existing diabetic retinopathy/maculopathy, amblyopia, glaucoma (over-represented in the diabetic population (2)) and age-related macular degeneration must be considered in such discussions. Sometimes medical staff will encourage patients towards cataract surgery to improve views of the retina for fundus imaging or retinal treatment. In addition, cataract surgery now has an established place in control of intraocular pressure in glaucoma patients (3) and should be considered where risk of postoperative macular problems is considered low.

Attention to detail in the outpatient clinic can smooth the path of diabetic patients through cataract surgery.

Figure 1 - Cataract obstructing view of the Retina

Blood glucose

Should be assessed in outpatients. The author has used a level of 20mmol/L as unacceptably poor control on the day of surgery indicating cancellation of surgery. This level is an indicator of increased risk of infection (postoperative and general), suggests increase risk of diabetic complications with time (4) and risk of life threatening events including diabetic ketoacidosis. If random blood glucose in outpatients indicates poor control, referral to the relevant diabetic service for improvement in control is required before surgery can be undertaken.

Advice regarding starvation prior to daycase GA surgery or locally-devised light diet advice before local anaesthesia surgery must be accompanied by advice about changes to insulin and/or oral agents on the day of surgery.

Blood pressure

Although there is evidence that raised systemic blood pressure and use of cardiovascular medications are risk factors for suprachoroidal haemorrhage during cataract surgery (5), the main concern is to avoid last minute cancellation of patients who are found to have unacceptable blood pressure control on the day of cataract surgery. Some patients with raised blood pressure (example 200/120mm Hg) may still be taken through surgery where anxiety is reduced with intravenous sedation resulting in lower blood pressure. The blood pressure is measured in outpatients at the time of the decision to proceed to cataract surgery and blood pressure treatment optimised by appropriate referral when required.

Anticoagulant/antiplatelet therapy

These treatments can be stopped before surgery where this does not present risk to welfare or survival of patients. Cataract surgery with injectable local anaesthesia (peribulbar sharp needle anaesthesia) can be undertaken with INRs up to 3.5 with safety in patients on warfarin (6). Patients on new oral anticoagulants seem to do well with non-injection technique topical anaesthesia clear corneal surgery with intracameral antibiotic prophylaxis (cefuroxime 1mg or moxifloxacin 0.1mg) and topical postoperative steroid/anti-inflammatory agents.

Aspirin, clopidogrel and other antiplatelet agents can be managed as for non-diabetics.

Systemic alpha blockers

During the author's time at University College Hospital, London, in the period 2012-14 the use of alpha 1 antagonists for urinary outflow symptoms reached virtually 50% in males undergoing cataract surgery. Intraoperative floppy iris syndrome (IFIS) (7) will be an unpredictable risk in patients taking these drugs, especially tamsulosin. This source of poorly dilating pupils will be added to the poorly dilating pupils of diabetics with any degree of autonomic neuropathy. Stopping tamsulosin prior to cataract surgery is of questionable effectiveness (7)

Risk stratification for cataract surgery with poorly dilating pupils is advised at the time of scheduling patients for cataract surgery. These patients are not suited to surgery by junior trainees on Friday afternoons (8).

Severity of diabetic eye disease

Proliferative diabetic retinopathy should be controlled wherever possible before cataract surgery is undertaken. Neovascularisation after cataract surgery may result in rubeosis iridis which will require prompt management by antiVEGF agents and/or argon laser treatment. Where severe retinopathy coexists with cataract combined phacovitrectomy/lens implant, possibly with endolaser, should be considered.

Operative Techniques

Cataract surgery seeks to restore visual quality that has been lost when cataract develops. Diabetics with retinopathy require continued surveillance after cataract surgery and operations should be designed to restore vision for the patient and optimise fundus view for subsequent retinal examinations.

Pupil size

This article does not seek to cover all aspects of cataract surgery technique in detail. However, the small pupil in diabetes and in male patients on alpha blockers may require specific techniques such as high viscosity viscoelastics, intracameral phenylephrine (9, 10) and iris hooks or pupil enlarging devices to increase pupil size to allow a 5mm capsulorhexis to be fashioned and safe cataract surgery.

Capsulorhexis size

It is desirable to make a large capsulorhexis to enhance fundus view and allow a large posterior capsulotomy if required later. Capsulorhexis size will be related to diameter of lens implant, bearing in mind that optimal posterior capsule opacification (PCO) rate and achievement of the postoperative refractive target require the capsulorhexis to be sitting on the periphery of the intraocular lens (IOL) throughout 360 degrees. Use of an 5.6mm corneal optical zone marker will guide the surgeon to produce a capsulorhexis of 5mm, well suited to a 6mm optic lens implant allowing for minor physiological decentration of capsulorhexis versus IOL position.

In diabetics with pseudexfoliation of the lens capsule, insertion of a capsule tension ring at the time of surgery will reduce the risk of phimosis of the capsulorhexis resulting from the slack zonules that are part of this condition.

Lens implant design

Lens implants should be chosen for optical quality, sustained optical clarity (a number of designs have shown loss of clarity with time), ease of insertion, good centration and low posterior capsule opacification rates (11, 12, 13). As mentioned above, 6mm lens optics are the standard size presently although larger sizes are offered by some manufacturers.

PCO rates have been shown to be better in lenses with a square posterior edge which inhibit lens cell migration across the posterior capsule. In general, three piece designs ensure a 360 degree square posterior edge as well as posterior vaulting to press the square lens edge against the posterior capsule. Manufacturers are moving towards a 360 degree square edge in one piece designs to avoid haptic-optic junction PCO (14) although these lenses tend to be single plane (producing less pressure between square posterior optic edge and capsule). Surgeons are advised to be aware of the design features of the lens implants they are using; technical drawings will show lens edge design and are available on manufacturer's websites.

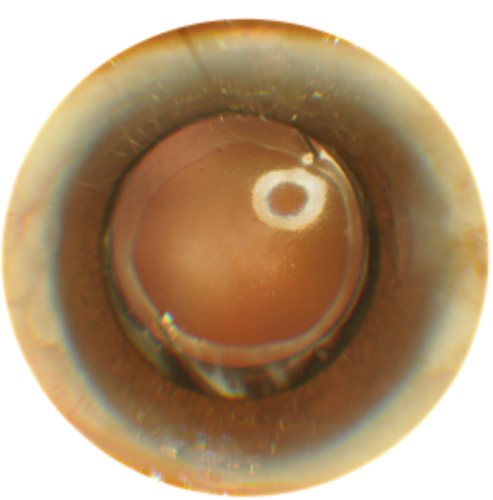

Figure 2 - A generous size of capsulorhexis optimises fundus views after cataract surgery

Prophylactic drug therapies

All patients undergoing cataract surgery should receive antibiotic prophylaxis at the time of surgery (15). Where postoperative inflammation or worsening of maculopathy are anticipated, other therapies including intracameral (16) or intravitreal steroid or antiVEGF drugs may be required. Standard postoperative regimens of topical steroid and topical antibiotics for 4-6 weeks are usually followed unless the clinical situation indicates otherwise.

Postoperative Management

Postoperative management of the majority of diabetic cataract surgery is identical to that of their non-diabetic counterparts. However there are three areas of special consideration:

Inflammation

Diabetic patients are prone to an increased postoperative inflammatory response (15) and more prolonged inflammatory response after cataract surgery (17). These responses can be managed by more intensive and/or prolonged postoperative topical steroid, possibly with addition of topical NSAIDs especially for the second eye where control of inflammation has been difficult for the first eye.

Occasionally, diabetic patients will suffer fibrinous post operative uveitis due to severe breakdown in the blood aqueous barrier. Experience suggests that this can be managed by intensive topical steroid therapy with adequate allowance of time for the fibrin to clear from the anterior ocular segment. Where recovery is delayed, perhaps for three weeks or more, use of intracameral recombinant tissue plasminogen activator (r-TPA) could be considered (18).

Diabetic maculopathy

Studies have shown that severity of diabetic maculopathy prior to cataract surgery influences visual outcome and maculopathy developing after surgery is more likely to resolve (19, 20). Management of these events requires assessment of the macula prior to surgery (OCT images providing retinal thickness measurements) and increased medical retinal follow up with active management of the maculopathy by anti-inflammatories (21), anti VEGF drugs and laser where appropriate.

Diabetic subjects without retinopathy may develop pseudophakic macular oedema which can be assessed by OCT imaging/fluorescein angiography as indicated and treated initially with topical non-steroidals. Evidence exists for improvement of pseudophakic cystoid macular oedema with topical NSAIDs (22) and also some evidence that prophylactic use may prevent the condition (for example in the second eye)(21, 23).

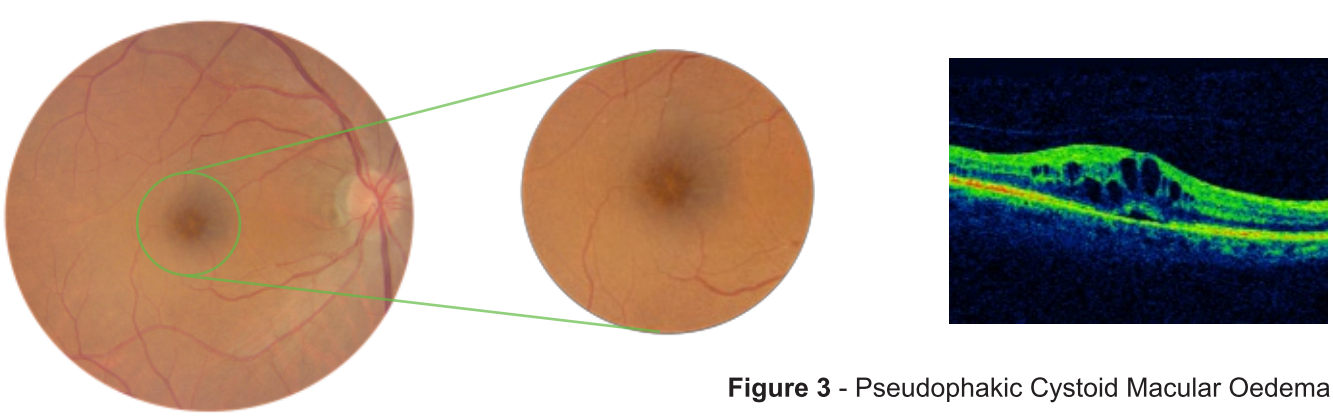

Figure 3 - Pseudophakic Cystoid Macular Oedema

Postoperative endophthalmitis

Presently the incidence of post cataract surgery endophthalmitis in all patients is thought to be around 0.05% (24). Diabetic patients may be at increased risk of postoperative endophthalmitis and are more likely to be culture positive (25). Endophthalmitis has a worse outlook in diabetic patients (26, 27).

Conclusion

As the prevalence of diabetes mellitus in the UK increases along with a progressive rise in life expectancy, the burden of diabetic cataract surgery is certain to increase in the coming years. Close liaison between anterior segment surgeons and medical retina specialists will optimise outcomes.

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Taking a fresh look at diabetes education

Diabetes education is a key element in the supported self-management of the condition. Yet only a small minority of patients are offered validated structured diabetes education and fewer still take up the offer. Diabetes UK argues that a new approach is needed, to ensure that more people with diabetes will have access to education and information that meets their needs. ***Dr Susan Aldridge***, Editor of *Diabetes Update*, introduces the charity's new position statement on diabetes education

How many of your patients mention diabetes education when they attend for their digital retinal screen? Have they ever been offered a place on one of the formal courses, like DAFNE or DESMOND (see box)? Did they attend, and did it meet their needs? You may be surprised to learn that only 12 per cent of those newly diagnosed with diabetes were offered structured education in 2011/12. Moreover, of those with both Type 1 and Type 2 diabetes, only 25 per cent of those who are offered structured education actually chose to take it up. This is why Diabetes UK is now calling for a fresh look at diabetes education, with new initiatives to be set up to meet the needs of people at all stages and levels of the diabetes journey.

The role of education

Diabetes UK believes that supported self-management that puts the person with diabetes at the centre of their care is the key to long-term success in dealing with the condition. Self-management has to include personalised care planning, emotional and psychological support and easy access to information and education.

'The National Institute for Health and Care Excellence has been recommending access to structured diabetes education since 2003.' The original NICE technology assessment (CG87) specifies that:

• Educational interventions should reflect established principles of adult learning.

• Education should be provided by an appropriately trained multidisciplinary team (MDT) to groups of people with diabetes.

• The MDT should include, as a minimum, a diabetes specialist nurse (or a practice nurse with experience in diabetes) with knowledge of patient education and a dietitian.

• Sessions should be accessible to the broadest range of people (taking into account culture, ethnicity, disability and location) and could be held either in the community or at a local diabetes centre.

• Programmes should use a variety of techniques to promote active learning and be adapted wherever possible to meet the different needs, personal choices and learning styles of people with diabetes, and should be integrated into routine diabetes care over the longer term.

Meanwhile, NICE Quality Standard 6 lays down the following five key criteria for a patient education programme.

• The programme should be evidence-based and meet the needs of the individual. It should have specific aims and learning objectives. It should also support the learner and his or her family in developing attitudes, beliefs, knowledge and skills to self-manage diabetes.

• The programme should have a structured curriculum that is theory-driven, evidence-based and resource-effective, has supporting materials and is written down.

• The programme should be delivered by trained educators who have an understanding of educational theory appropriate to the age and needs of the learners, and who are trained and competent to deliver the principles and content of the programme.

• The programme should be quality assured, and be reviewed by trained, competent, independent assessors who measure it against criteria that ensure consistency.

• The outcomes from the programme should be regularly audited.

Diabetes UK continues to support the above approach, believing that structured education meeting these standards should be made available to all people with diabetes – not just at diagnosis, but at any stage of their journey with the condition.

However, ten years on from the introduction of these recommendations, access to structured diabetes education remains poor. Data from the National Diabetes Audit shows that there were no referrals to structured education in eight clinical commissioning group (CCG) areas. Almost half of CCG areas had referral rates of less than 10 per cent. The national referral rate is only 14 per cent. Where courses are offered to the newly diagnosed, take-up rates are only 25 per cent. This is despite one survey showing that 76 per cent of people with diabetes want access to ongoing learning.

A new approach to diabetes education

The poor take-up of diabetes education suggests that many people may not see the need, or are put off by the prospect of 'education' in a group setting. It is not enough for people to be referred to a course – the clear benefits need to be explained. It may also be hard to find the time to attend, or the location may be inconvenient. A recent study revealed that as many as 76 per cent of those referred to one X-PERT programme did not attend, with the main reason given being working hours and lack of transport.

For some, the word 'education' brings back bad memories of school, so it may be better to talk about 'skills training', 'diabetes awareness training', 'diabetes support sessions' or 'diabetes information exchange'.

Diabetes UK now believes it is time for a different approach which meets the education and information needs with more people with diabetes. This would be to supplement, rather than replace, current structured education programmes. Diabetes Education Scotland refers to three levels of diabetes education, forming a 'spiral curriculum' and Diabetes UK generally supports this idea, for there is indeed a need for different approaches at different stages of the education journey.

Three levels of diabetes learning

The three components of the spiral curriculum can be described as follows:

• Introductory Diabetes Education. This is given at the time of diagnosis and may be in the form of information, signposting to relevant educational tools, or one-to-one discussion, often delivered by the healthcare team that is responsible for the person's diabetes care. It may include specific skills education, such as carbohydrate counting in Type 1 diabetes.

• Ongoing Diabetes Learning. This is ongoing, throughout the diabetes journey, relating to those aspects a person needs to know more about as they learn to live with the condition. This might include gaining a deeper knowledge of diet or learning how to manage a specific complication, such as an eye condition. Ongoing learning could also include peer-led support and education.

• Standard Education. This is delivered to a group of patients and would include the NICE-approved structured education programmes.

The first two levels should act as a catalyst for people to work towards Standard Education. However, attendance at Standard Education need not be dependent on someone having attended previous courses. Diabetes learning opportunities should be provided as and when the patient needs them, particularly when they are newly diagnosed. If an individual is reluctant to attend Standard Education, then they should be offered other educational opportunities which may be more appropriate.

Diabetes UK is calling for appropriate educational initiatives to be funded and delivered to meet the needs of people at all stages. Should a need be identified, then a new educational initiative should be developed to meet this. Education sessions should be as accessible as possible with regard to timing, physical access, meeting the learning abilities of the individual and including carers and family.

The NICE recommendations call for the involvement of a specialist nurse and dietitian. However, research shows that peer education can be equally effective. Therefore, peer-led initiatives should be a part of the diabetes education armoury.

What about quality assurance?

It is essential that new educational initiatives be developed with the involvement of people with diabetes, to ensure that they are appropriate and meet the needs of those they are aimed at. It would not be appropriate for all

approaches to have to demonstrate effectiveness through clinical trials and research. However, they should be able to show what people with diabetes will achieve if they take part and should have some measure of validation to demonstrate these outcomes. Improved clinical outcomes are, of course, the gold standard, but there are other relevant and valid outcomes. These could include Quality of Life indicators, Patient Reported Outcome Measures, improved knowledge or improved confidence in self-management. Furthermore, all educational approaches should consider NICE standards when they are being developed.

Some examples

There are already a number of examples of diabetes education approaches at each of the three levels.

Introductory Diabetes Education

- One-to-one discussion from a diabetes-trained healthcare professional
- Provision of Diabetes UK Companion Guides, with follow-up discussion
- Signposting to a Type 2 Diabetes and Me online learning programme, with follow-up discussion
- Signposting to Diabetes UK Living with Diabetes Day

Ongoing Diabetes Learning

- Group work with Healthy Conversation Maps about specific areas of diabetes management
- Open University online diabetes course
- Carbohydrate counting tool from Bournemouth Education Centre online
- Diabetes UK's Peer Support Programme, currently being piloted
- My Diabetes My Way online resources in Scotland

Standard Education

- Group education meeting NICE standards (DESMOND, X-PERT, DAFNE – see box)

Diabetes UK recommendations

Diabetes learning should be lifelong and offer a number of different ways in which people can learn about their condition. The existing structured education courses are an important part of this.

People with diabetes need:

- High-quality courses that are appropriately quality assured, offered both on diagnosis and throughout the diabetes journey
- Clear and compelling explaining of the benefits of attendance
- Courses designed around their needs (conveniently timed and located, and tailored to different learning styles, personal circumstances and cultural backgrounds)

New, shorter and more informal ways of learning about diabetes are needed, as well as current structured education. All healthcare professionals need to raise awareness of the importance of learning as part of managing diabetes and the importance of engaging more people with diabetes in their own care.

Diabetes UK will:

• Develop and deliver new services, like peer-based support and learning and Living with Diabetes Days to help meet this need
• Campaign for the needs of people with diabetes to have access to quality education and raise awareness of the importance of people with diabetes accessing learning throughout their life with diabetes.

Commissioners need to ensure that learning opportunities at all stages of diabetes education are available and that people with diabetes are encouraged to take up these opportunities. They should also put systems in place to check the uptake and effectiveness of programmes. Finally, national decision makers should put metrics in place to measure how well people are having their diabetes education needs met.

Structured diabetes education
Diabetes Education for Self-Management for Ongoing and Newly Diagnosed (DESMOND) for people with Type 2 diabetes
Self-management education modules, toolkits and care pathways for people with, or at risk of, Type 2 diabetes
Dose Adjustment for Normal Eating (DAFNE) for adults with Type 1 diabetes
DAFNE is proven to improve blood glucose control and quality of life, while reducing severe hypoglycaemia.

X-PERT Diabetes Programme for people with Type 1 and Type 2 diabetes
Helps patients to increase their knowledge, skills and understanding of managing diabetes on a day-to-day basis and is shown to significantly improve health and wellbeing.

X-PERT Insulin Programme for people with Type 1 or Type 2 diabetes
A chance to explore the management of diabetes and learn all about the most up-to-date treatment options available to help patients match insulin to carb intake and physical activity levels.

Type 2 and me

Diabetes UK, in partnership with Bupa, has created an interactive online e-learning programme to help people with Type 2 diabetes understand and manage their diabetes successfully. This is not intended to take the place of a more formal patient education course.

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Oral Agents used in the treatment of Diabetes

Abstract:

The management of people with Type 2 Diabetes includes dietary and lifestyle modification to improve glycaemic control and modification of cardiovascular risk factors. A combination of insulin resistance and inadequate insulin secretion (pump failure) usually results in gradually escalating therapy being needed over time. This article outlines available oral agents used to help control blood glucose levels.

Introduction:

Diabetes mellitus (DM) is a metabolic disorder characterized by a chronic elevation in blood and tissue glucose levels / hyperglycaemia and is due to a combination of reduced insulin secretion compared to requirements, often termed 'pump failure' and reduced insulin action, often termed 'insulin resistance', where more insulin is needed to obtain the same blood glucose lowering effect so exacerbating this 'pump failure'.

Control of this elevation in blood glucose is important in reducing the long term complications of diabetes, and people with Type 2 Diabetes can require both oral and injectable therapies to achieve this. These treatments aim to reduce the absorption of glucose, increase the loss of glucose in the kidney, increase pancreatic insulin production (secretagogues) or improve insulin resistance (insulin sensitizing agents), with replacement of insulin by injection being the final resort. The need for combinations of drugs working in different ways is not unusual, with 50% of those on monotherapy with metformin or a sulphonylurea needing extra therapy after 3 years in the UKPDS trail).

The potential detrimental effects of these treatments should also not be forgotten, which include weight gain, lowering of blood glucose levels too much / hypoglycaemia and with tight glycaemic control an initial deterioration in some of the complications we are trying to reduce, such as diabetic retinopathy. The previous withdrawal of some blood glucose lowering agents due to potential liver damage (e.g. Troglitazone) or worsening of cardiovascular disease risk (e.g. Rosiglitazone) should also be remembered. This article will concentrate on the oral agents currently available for the treatment of this condition.

Treatment:

While concentrating on the oral therapies available we should not forget the importance of the non pharmacology treatment of Diabetes, in particular the role of dietary modification to reducing saturated fat, simple or refined sugars, salt and alcohol as well as the importance of an increase in aerobic exercise and, for many people, weight reduction.

The choice of agent to use also needs to be tailored to the individual patient with the typical over weight, more insulin resistant type 2 person, usually starting with an 'insulin sensitizing' agent such as Metformin, while a thinner person with pump failure may start with a 'secretagogue' such as Gliclazide, but combination therapy is often needed with time.

Blood glucose lowering therapy for those with diabetes can be broadly categorised into 2 groups i.e. Injectable and Oral therapy. There are 2 groups of injectable therapy: insulin and GLP1 (glucagon-like polypeptide-1) agonist.

We will discuss the oral blood glucose lowering therapies here, which can be broken into several groups based on their mode of action:

1. Altering insulin resistance, which can also be called 'insulin sensitizing' agents:
Biguanides (e.g Metformin), Thiazolidinediones or Glitazones (e.g. Pioglitazone and before its withdrawal Rosigltazone)

DPP-4 (dipeptidyl peptidase 4) inhibitors (e.g Sitagliptin, Vildagliptin, Saxagliptin, and Linagliptin)

2. Increasing insulin release / production, also termed 'secretagogues'
Sulphonylureas (e.g. Glibenclamide, Gliclazide, Glipizide, Glimepiride and Tolbutamide)
Meglitanides, also called prandial glucose regulators or prandial insulin releasers (e.g. Repaglinide and Nateglinide)

3. Reducing gastrointestinal absorption of glucose
α-Glucosidase inhibitors (e.g. Acabose)

4. Increasing renal / kidney glucose loss
SGLT-2 (sodium glucose co-transported-2) inhibitors (e.g. Dapagliflosin, Canagliflosin)

Insulin Sensitizing agents:

1. Biguanides (e.g. Metformin)

Metformin is the most widely used anti diabetic therapy worldwide. It is first line therapy in the overweight population (BMI / body mass index >25kg/m2) and when compared to other therapies has benefits including reduced mortality and complications. Start with 500mg daily and increase slowly.
In Medieval Europe Galega officinalis (French Lilac or Goat's Rue) was used as a herbal treatment for the symptoms of diabetes and was shown to contain guanidine, a substance with blood glucose lowering properties by the Clinical Pharmacologist Jean Stern in the 1950s who then helped develop di-methyl biguanide (Metformin), which he named 'Glucophage' (glucose eater). Metformin was introduced for the treatment of diabetes to the UK in 1957. In 1998 the United Kingdom Prospective Diabetes Study / UKPDS showed metformin improves microvascular and macrovascular outcomes, particularly retinopathy and reduced the need for laser therapy. In 2001 the Diabetes Prevention Program (DPP) showed metformin reduces the progression of impaired glucose tolerance to type 2 diabetes.

Metformin is rapidly absorbed (peak blood concentration at 1-3 hours for standard and 4-8 hours for the slow release preparation) and is excreted by the kidney. It requires the presence of insulin to work as it does not cause insulin release but increases insulin sensitivity by decreasing hepatic gluconeogenesis (liver glucose production) and fatty acid oxidation, as well as increasing muscle glucose uptake / usage. It can also improve lipids / cholesterol profiles by reducing LDL and produces weight loss by improving satiety, although it can give a metallic taste and causes bloating and some gastrointestinal upset in 20% of people but only 5% have to stop it due to these side effects. It reduces basal hyperglycaemia (fasting glucose typically falls 1-4mmol/L and post prandial by 2-4mmol/L) and does not cause hypoglycaemia.

A reduction in Vitamin B12 levels occurs in <10% with lactic acidosis a rarer but potentially more serious side effect with this drug, occurring in 3/100,000 patients per year, but is fatal in 50%.

Administration:

Should be taken with food or immediately before meals. GI side effects are worse if taken without food. Advised to be avoided with alcohol consumption due to the delayed hypoglycaemic effects of alcohol.

Contraindications:

1) Renal disease / dysfunction (Serum Creatinine >150µmol/L or eGFR <30mL/minute/1.73m2), or conditions which might induce these, such as the use of radiological contrast media

2) Any condition predisposing to hypoperfusion and low tissue oxygen levels / hypoxia eg. recent MI, cardiac insufficiency / failure, sepsis, acute shock

3) Significant liver disease

2. Thiazolidinediones

Also known as 'glitazones' these were introduced to the UK in 2000, with three drugs available so far but only (Pioglitazone) is still in use in the UK. Pioglitazone acts on a receptor in the nucleus of cells (peroxisome proliferator activated receptor - PPAR-γ), activating this , so increasing the production of glucose transporter molecules (such as Glut 1 and Glut 4), which allow any insulin molecule to have a greater effect, e.g. an insulin sensitizing effect.

This causes reduced liver glucose production, increased glucose uptake by muscle and fat cells. It is rapidly absorbed (peak concentration in <2 hour) and while dropping glucose (HbA1c reduced by 0.6-2.0%, fasting glucose by 2-3mmol/L), does not carry a risk of major hypoglycaemia. However as it alters a central nuclear pathway it takes some time to drop glucose levels and is likely to cause weight gain (2-4kg in first 6 months) and in 5-10% of people fluid retention. So should not be used in those with macular oedema or heart failure. There is also concern about osteoporosis in women and a weak association with an increased incidence of bladder cancer if used for more than 2 years, both of which are being investigated further.

Administration:

Can be taken without meals, usually once daily

Contraindication:

Avoid in congestive cardiac failure or liver disease, pregnancy, breast feeding

3. Gliptins / DPP4 Inhibitors

Insulin secretion is stimulated by the incretin hormones Glucagon like polypeptide-1 (GLP-1) and Gastrointestinal polypeptide (GIP), which are synthesized and released in response to a food load entering the gastrointestinal tract. The incretin hormones also suppress glucagon secretion and slow gastric emptying, with an accompanying improvement in insulin sensitivity. The breakdown of GIP and GLP-1 is by the enzyme Dipeptidyl peptidase-4 (DPP-4), Gliptins inhibit this enzyme. This class of agent was introduced to the UK in 2007 (sitagliptin) with several other drugs since then, including Linagliptin, which can be used freely in those with renal impairment.

These agents do not cause weight gain and are thought of as weight neutral, while the GLP1 therapies are weight reducing. They have a low risk of hypoglycaemia.

Administration:

Can be taken without meals as does not cause hypoglycaemia.

Contraindication:

Avoid in moderate to severe renal failure (Exception Linagliptin and Sitagliptin 25mg dose) and those with significant liver disease, pregnancy, breast feeding

Insulin Secretagogues

1. Sulfonylureas

Sulphonylureas were developed following the discovery of sulphonamide drugs having the potential to cause hypoglycaemia. This group of drugs were introduced to the UK in 1950s and for decades were the most commonly used treatment for type 2 diabetes together with metformin. Second generation agents released in the 1960s include Glibenclamide and Gliclazide.

Sulphonylureas act on the pancreas to stimulate insulin secretion from the functioning beta islet cells. They typically have a peak effect at 2-4 hours and drop HbA1c by 1-2%, fasting and post prandial glucose by 2-4mmol/L. The main limitation was the side effect of causing weight gain (1-4kg stabilizing after 6 months), which may also worsen insulin resistance and hypoglycaemia, 20% get 1 or mild episodes per year and 1% more severe episodes, with a mortality from these of 1 per 50,000 patients per year. Reliance on the patient's own pancreatic cells to make insulin also sees a 5-10% drug failure rate per year typically.

Administration:

Always take with / before meals to avoid hypoglycaemia, and those on this group of drugs should check capillary blood glucose prior to driving.

Contraindications:

Avoid with kidney and liver impairment, pregnancy, breast feeding

2. Meglitanides / Prandial glucose regulators

This group of drugs are similar to the sulfonylureas and so need functioning beta cells in the pancreas to work, but have a shorter duration of action, potentially reducing the risk of hypoglycaemia. Useful in people with erratic / viable eating patterns, the elderly or at times such as Ramadan. Repaglinide and Nateglinide have a peak blood level in <1 hour and a duration of action of 2-6 hours. Both drop HbA1c by 0.5-2%, fasting glucose by 1-3mmol/L and post prandial glucose by 1-4mmol/L. Side effects include weight gain (1-3kg stabilizing after 3-6 months) and hypoglycaemia, although much less frequently that the sulphonylureas.

Administration:

They are taken before each meal, in order to target post-prandial hyperglycaemia.

Contraindications:

Significant Liver Disease, pregnancy, breast feeding

Alpha-glucosidase inhibitor

Acarbose

Acarbose was introduced in the UK in 1993. It is taken with food and reduces post meal / prandial glucose peaks by inhibiting the gut enzyme α-glucosidase, thus slowing carbohydrate digestion. It delays digestion of sucrose, so glucose is the preferred treatment of hypoglycaemia (acarbose does not itself cause hypoglycaemia).
It drops HbA1c by 0.5-1.0% and post prandial glucose by 1-4mmol/L. Its main restriction is gastrointestinal side effects, especially flatulence.

Administration:
Should always be taken with meals rich in complex carbohydrate.

Contraindications:
Severe renal and liver disease, pregnancy, breast feeding and in chronic intestinal disease.

SGTL2 Inhibitors

The kidney gives us 25-30% of circulating glucose when we are fasting. Sodium glucose co-transporters (SGLTs) are responsible for stopping glucose being lost in the urine, re-absorbing about 180g of glucose from the glomerular filtrate each day, 90% of glucose is reabsorbed by transporters of the SGLT-2 type. SGLT-2 inhibitors act independently of insulin to increase renal glucose excretion (resulting in about 70g of glucose being lost per day) and so lower blood glucose, as well as causing a net calorie loss and in many people some weight loss. HbA1c drops by 0.6-3% but care is needed with genitor-urinary tract infections and potential electrolyte disturbances although both are uncommon (typically <1% of patients stop due to these).

Administration:
Daily independent of food

Contraindications:
Renal impairment (eGFR <60mL/minute/1.73m2, although Canagliflozin can still be used if eGFR fall into the 45-60ml/min/1.73m2 range)

Conclusion

Currently the management of people with Type 2 diabetes includes careful control of both blood glucose and modification of cardiovascular risk factors with lifestyle advice, dietary modification and the use of multiple therapies in most patients. A combination of insulin resistance and inadequate insulin secretion (pump failure) usually results in gradually escalating therapy over time.
Common practice is to commence oral anti diabetic therapy once non pharmacological approaches fail to achieve safe HbA1c targets. Metformin is generally the first agent of choice. A patient centred approach is essential for the selection of oral anti diabetic combination therapy. This may be done in a step wise manner and as beta cell failure progresses and multiple insulin dependent oral therapies fail to achieve target, insulin independent oral therapies can be needed prior to trying injectable therapies including insulin if needed.

Table 1.

Glucose lowering effect in oral hypoglycaemic agents used in the treatment of Type 2 diabetes.

Class of Agent	Expected reduction in HbA1c	Typical effect on weight	Potential adverse effects
Biguanides	0.8–2.0% (sustained)	Weight neutral or minimal weight loss	Gastrointestinal (common but usually transient), rarely skin rashes, lactic acidosis, vitamin B12 absorption can be reduced
Sulphonylureas	1-2% (sustained)	1-4kg weight gain (plateaus at six months)	Major episodes of hypoglycaemia Occasional skin reactions, alterations in liver function tests, minor gastrointestinal symptoms
Meglitanides	0.6–2%	1-3kg weight gain possible	Hypoglycaemia Skin reactions Hepatic dysfunction (nateglinide) Cardiovascular disease (repaglinide)
α-Glucosidase inhibitors	Extra 0.5% when added to other therapies	Weight neutral	Can precipitate/aggravate hypoglycaemic episodes Gastrointestinal (common, eg bloating), jaundice and elevated hepatic transaminase levels
Thazolidinediones	1 to 3% in combination with metformin or sulfonylurea	Weight gain (2-4kg)	Hepatic dysfunction possible Fluid retention Fractures in the elderly
DPP-4 inhibitors	0.6-1.2%	Weight neutral	Hypoglycemia when in combination with sulphonylurea or Insulin
SGLT-2 inhibitors	2-3%	Weight loss	Possible electrolyte disturbance, urinary tract infections, genital infections (e.g. ‘thrush’)

Table 2.

Dosages for commonly used oral hypoglycaemic agents.

Drug	Daily Doses
Glipizide Gliclazide Glimepiride	5mg – 20mg 40mg – 320mg MR 30mg -120mg 1mg -6mg
Metformin	500mg – 3,000mg Or Slow release formulation 500mg - 2,000mg daily
Repaglinide Nateglinide	0.5mg-4mg with meals (up to 16mg / day) 60mg -180mg per meals (up to 540mg / day)
Acarbose	50-200mg per main meal (up to 600mg daily)
Pioglitazone	15-45mg od
Sitagliptin	100mg (25mg, 50mg in renal impairment)
Linagliptin	5mg
Dapagliflosin Canagliflosin	5-10mg 100-300mg

Further reading / References

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3. CJ Bailey, C Day. Metformin: its botanical background. Practical Diabetes International. April 2004: 21(3):115-117

Since 2001, the British Association of Retinal Screening (BARS) has been the UK's professional organisation for those who provide retinal screening services for people with diabetes. We offer education, representation and support to a wide range of professionals involved in diabetic retinopathy screening, and our members include retinal screeners, graders, administrative and failsafe staff, programme managers, optometrists and ophthalmologists – in fact anyone with a professional interest or involvement in diabetic eye screening.

BARS is a not-for-profit organisation, run by an elected council of volunteers who are drawn from a range of roles within eye screening programmes across the UK, and we aim to support our members via regular conferences, meetings, training days and educational activities. The association has assisted those studying for the City & Guilds Diploma in Diabetic Retinopathy Screening by offering tutorial days for individual units, while administrative staff have benefitted from our popular Failsafe Discussion days. Programme managers and clinical leads can access our online support forum, and we organise regular programme managers' meetings, often with input from the national team.

BARS future plans include expanding our range of meetings and training days to include staff groups and topics not previously covered, and YOUR input will tailor these events to members' needs.

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