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Pathology induced refraction and vision changes

By Andrew Watson BSc (Hons), MCOptom, DipTp (IP), Prof Cert (Glauc), FBDO CL

As dispensing opticians (DOs), we spend some of our working day talking to patients and discussing the small group of numbers written down on their spectacle prescription. We do it so much that sometimes it is easy to forget where these numbers come from, and what they can mean with regards to what is going on with our patients' eyes.

This article briefly revisits the basic principles of visual optics and looks at some of the pathologies that can cause or change a patient's spectacle prescription and alter their vision. Like an eye examination, we will work from the anterior segment to the posterior segment.

Whilst training to become DOs, we study visual optics and see schematics like **Figure 1** and solve equations to get an answer. It helps us understand the optical mechanics of why vision goes blurry if certain ocular anatomy parameters are changed, and whether this is a normal developmental change, physiological change or a pathology induced one.

Curvature, refractive index, and axial length are the modifiable elements in a reduced eye. From **Figure 1** we see that if F_e (the power of the reduced eye surface) is greater than +60.00, the eye becomes myopic – as F'_e would fall short of the macula. F_e would change if the corneal radius of curvature (r_e) were less than 5.55mm (this might happen in a condition like keratoconus, or KC). If the axial length is longer than +22.22mm then the eye becomes myopic; if it is less than +22.22mm, it is hyperopic.

If the refractive index of the reduced eye is greater than 1.3333, again the eye becomes myopic. This could well happen in cases of nuclear sclerotic cataract.

The refractive states of the eye are defined in simple terms as myopia, hyperopia and astigmatism. Obviously, there are also refractions where one meridian may be hyperopic and the other myopic, which is known as mixed astigmatism.

MYOPIA

Myopia is an increasingly prevalent vision deficit[1]. Its development is multi-factorial and consists of genetic inheritance risks (i.e. myopic parents), ethnicity,

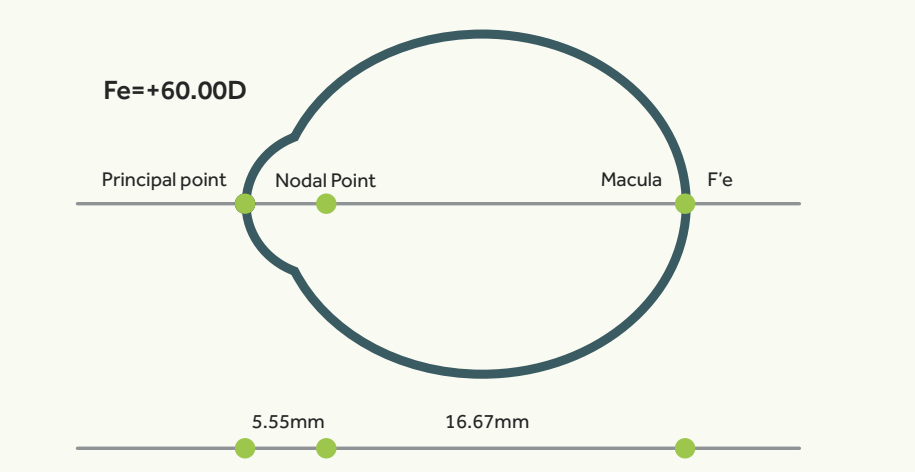

FIGURE 1: The standard +60D reduced eye

CPD CODE: C-102000

environment and prolonged near work[2]. As a child or adolescent grows, their eye grows. If the axial length of the eye exceeds 22.22mm (the normal reduced eye length f_e') the child becomes myopic.

We've all seen young children who never seem to be away from the practice, having their six-monthly eye examinations for their rapidly progressing myopia. It is this increasing axial globe length mechanism that brings them repeatedly to our practice, and one that new designs of spectacles and contact lenses are trying to retard the progression of.

If, however, the eyes are otherwise healthy – and provided the patient's refraction and spectacle dispensing is accurate – they will see very well with their new spectacles.

HYPEROPIA

It is a natural state of full-term new-born eyes to be hyperopic[3]. Obviously there are variations, but it is hoped that the process of emmetropisation[4] occurs and leaves the child with either no prescription (Rx) or a small Rx with good unaided vision.

If a child is hyperopic by a similar amount in both eyes, accommodation will occur to allow for clear vision and development of both eyes. This, of course, depends on the amount of hyperopia to overcome and the absence of any binocular vision anomaly.

If a child has anisometropic hyperopia, the child will accommodate in both eyes to give clear vision in the least hyperopic eye, leaving the other eye blurry and with the potential for the development of amblyopia if not corrected by spectacles[6].

ASTIGMATISM

This condition arises from the principal meridians of either the cornea or crystalline lens (or both) having different radii of curvature. If we have the Rx -3.00/-2.00 x 180 (compound myopic astigmatism), then on our schematic eye we have principal power meridians of:

F_{e180} = +63.00D and F_{e90} = +65.00D

From the schematic eye, cross-cyl diagrams and associated mathematics, it is easy to think of the condition in this black and white orthogonal sense. If you have ever looked at a computerised corneal topography plot of a patient with astigmatism, you will find a 'bow-tie' appearance to the plot, as shown in **Figure 2**. The 'warmer' colours indicate a steepening of the corneal curvature, while the 'cooler' colours indicate a flatter radius of curvature.

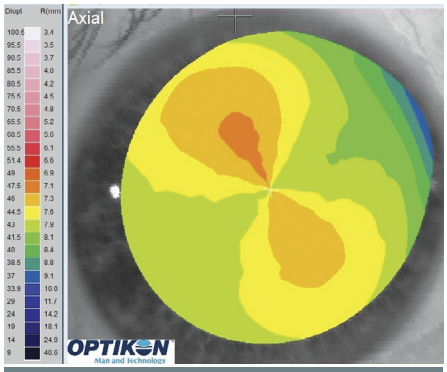

FIGURE 2: Topography image showing astigmatism

The astigmatism in **Figure 2** is that of regular astigmatism, as the principal meridians are perpendicular to each other and centred over the visual axis.

This type of astigmatism is relatively easy to correct with spectacles and contact lenses which, in the absence of other pathology, give a good best corrected visual acuity (BCVA) level. We will see later an example of irregular astigmatism.

PRESBYOPIA

Whilst technically not a pathology, presbyopia is worthy of note due to it being a common vision problem. Presbyopia is a natural age-related degeneration of near vision due to structural changes in the crystalline lens.

When we are born, the crystalline lens weighs approximately 140mg (wet weight) and by the time we reach 85 years old, it can weigh 280mg[5]. This increase in weight is because the crystalline lens continuously has new cells added to the outer layers, which over time make the lens less elastic and thus less able to provide accommodation for near visual tasks.

It is often stipulated that presbyopia occurs in your 40s. This means it can have a considerably variable age of onset. Near ability stems from your amplitude of accommodation, your distance ametropia and visual tasks performed.

For those who have been emmetropic throughout their lives, becoming presbyopic can be a real blow as it may be the first indication that their body's normal abilities are changing due to age. For those who have worn spectacles throughout their lives, it is just another visual hurdle to jump over as they switch from single vision lenses to multifocals.

As presbyopia continues into a patient's 50s, they become increasingly aware that mid-range tasks are not addressed by reading spectacles, or that the intermediate zone of their progressive is in the wrong place for comfortable sustained VDU use. Occupational lenses work well in these instances, normally allowing the full reading addition at the bottom of the lens and a reduced addition in the top of the lens to facilitate mid-range focusing.

There is a very good correlation between age and amplitude of accommodation (**Figure 3**) and thus the patient's age can normally be a very accurate indicator of reading addition required (considering specific working distance requirements).

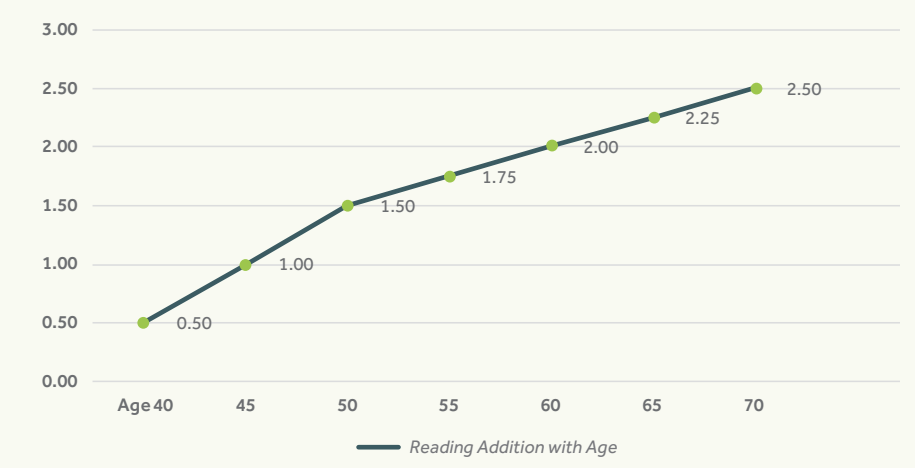

FIGURE 3: Reading addition with age

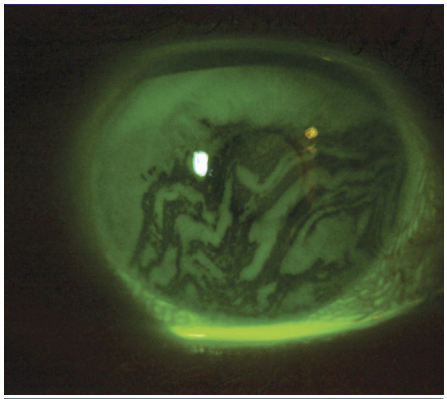

FIGURE 4:
Tear film assessment with fluorescein

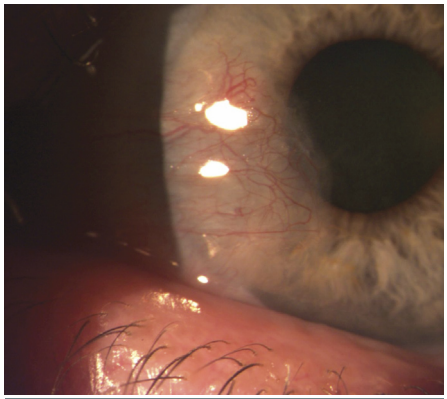

FIGURE 5:
Pterygium

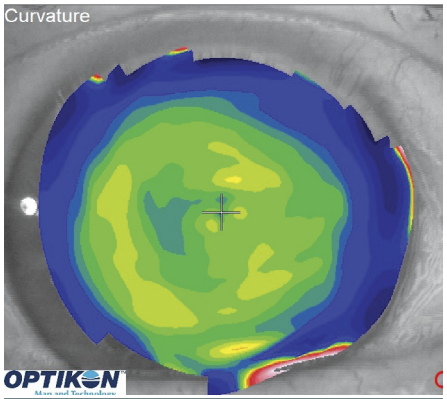

FIGURE 7:
Chalazion topography

DRY EYE DISEASE

As light reaches us, the very first part of our anatomy to start the process of refraction is the tear film. This thin soup of chemicals has many functions, and one is to provide a smooth refracting surface[7].

If you have tried to refract a patient with dry eye disease, you will be aware of the inherent difficulty of trying to reach the end point of the refraction. Just as you think you have reached 6/6, the patient blinks and suddenly they can only see 6/9 or worse. It is frustrating for patient and practitioner alike, but it is a good indicator of what you are going to find during slit lamp examination.

In **Figure 4**, we can see a tear film stained with sodium fluorescein. The tear prism height is low, and the black areas seen in the image are where the tears have evaporated away: reduced tear break-up time (TBUT). This is due to a reduction of aqueous production and poor lipid secretion from the meibomian glands.

This patient may need regular application of lubricant drops, treatment of underlying meibomian gland dysfunction or liposomal therapy to achieve BCVA, both in their everyday life and during refraction.

PTERYGIUM

In this condition, the corneal topography is altered due to an intrusion of collagen and vascular tissue into the corneal structure. This can in some cases cause an increase in regular astigmatism, and in severe cases cause a significant reduction in BCVA due to irregular astigmatism and opacification of the visual axis area of the cornea.

The case demonstrated in **Figures 5 and 6** still achieves good BCVA of 6/6 with spectacles, however, her refraction has become progressively more astigmatic over time. She uses a lubricant drop four times daily and is aware of UV protection when outside. It is also worth making the patient aware of the potential future need for surgery should it cause vision loss.

CHALAZION

A blocked meibomian gland continues to produce meibum, which as it stagnates can become a granulomatous mass causing a visible lump on the eyelid both anteriorly and posteriorly. If the chalazion has a horizontally central position within the lid, then during the hours of sleep with the lid closed the lump can cause a flattening of the corneal profile – as shown in **Figure 7**.

This has a similar effect to orthokeratology, in as much as it is changing the corneal profile and will lead to a change in refraction. In this instance, the patient's refraction changed from -2.00DS to +0.50DS in the space of just a month. An explanation of the condition, along with heat therapy and lid massage were instigated as first line conservative measures.

The topography plots help explain the condition to the patient, but also allows monitoring of the condition and if therapy is working. If no resolution occurs with conservative measures, then referral for excision surgery may be required.

KERATOCONUS

Keratoconus (KC) arises from a thinning of the corneal stroma and resultant weakness in the collagen matrix that gives the cornea structure. There are different sub-categories of KC depending on the position and type of cone, but they all essentially cause the same problems of increasing myopia and irregular astigmatism.

From the plot shown in **Figure 8**, there is an area of very steep curvature (keratometry readings, or Ks, can be in the region of 5-6mm) and this is inferior to the visual axis. The overall steepening of the corneal contour increases F_e –

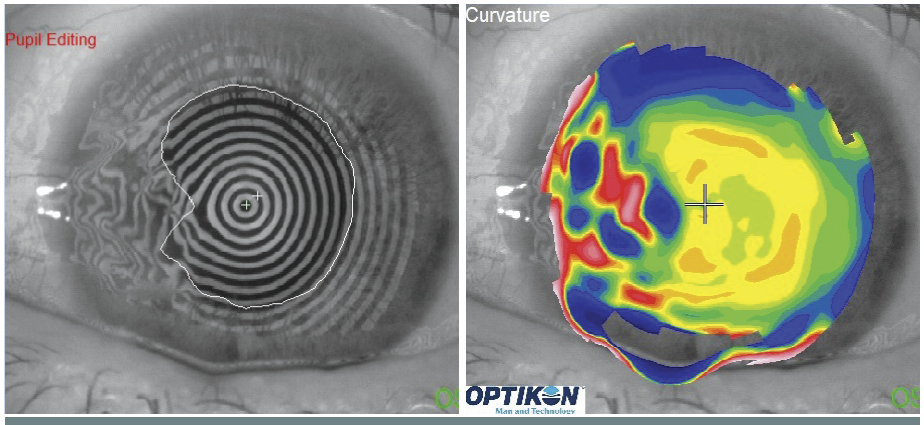

FIGURE 6: Pterygium topography

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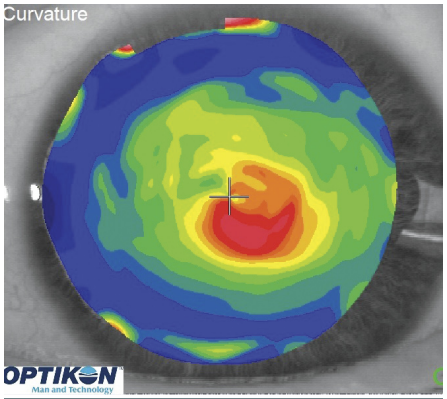

FIGURE 8:
Keratoconus topography

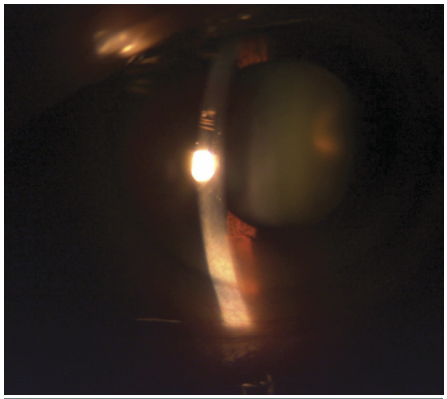

FIGURE 9:
Nuclear sclerotic cataract

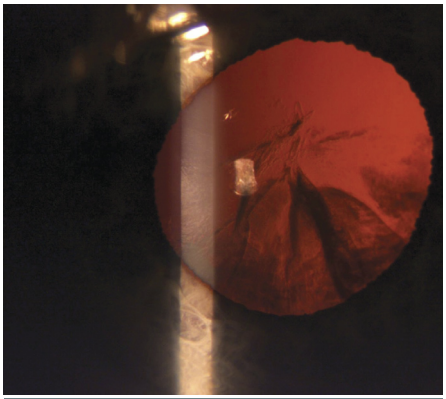

FIGURE 10:
Cortical cataract

thus inducing myopia. Unlike regular astigmatism (as seen earlier) the inferior zone of steepening causes increasing astigmatism, and this is irregular in nature. As spectacle lenses cannot take this into consideration, the patient often ends up with reduced BCVA.

It is easy to see why refracting this patient is difficult and why it is nearly impossible to correct the patient effectively with spectacle lenses – although a specialist contact lens fitting could be undertaken.

The spectacle refraction of this eye is -7.00 / -13.00 x 77, which gives a spectacle BCVA of 6/24. The same eye fitted with a Synergeyes Ultrahealth hybrid contact lens improves to a VA of 6/6.

If the KC is a new development and appears to be progressive, then referral to an ophthalmologist with a corneal sub-speciality should be recommended to see if collagen cross-linking procedures would be of benefit to the patient.

CATARACTS

A cataract is an opacity within the crystalline lens. Like a lot of medical terminology, it is an umbrella term for many different forms of opacity within the lens.

Nuclear sclerosis is where the entire lens becomes progressively and uniformly thicker (as part of the natural ageing process) but as the axial length and refractive index increases – so does its refractive nature. Thus, we see hyperopic patients start to lose their plus prescriptions at every visit; some can become emmetropic and gladly announce they no longer need their spectacles for driving.

We also see our myopic patients become increasingly more myopic. **Figure 9** shows an image of nuclear sclerotic cataract; note its uniformity thus producing changes in spherical power of the patient's prescription. As the thickening increases, short-wave blue light cannot penetrate the lens and it takes on a characteristic yellow-brown appearance. This also affects the patient's colour vision perception, and quite often patients are more ecstatic about the "wonderful colours" post cataract surgery rather than the sharpness of vision.

Cortical cataract is shown in **Figure 10**, with distinct areas of opacity within the lens cortex, which tend to appear as opaque 'spokes on a wheel'. Here patients' refractions yield increasingly astigmatic prescriptions. These can be difficult for patients to tolerate if they've either had no cylinders, or small cylindrical elements in their prescriptions previously, and suddenly find themselves with a moderate cylindrical component (>2.00DC) to achieve the BCVA.

DIABETES

In either undiagnosed or uncontrolled diabetes, changes in blood sugar chemistry from hypo- to hyperglycaemia can cause changes to the cornea and crystalline lens structures. The actual mechanism for these changes related to blood plasma glucose levels is still not understood[8]. The changes, however, can lead to an increase in hyperopia or myopia and there is no correlation of ametropia with blood plasma glucose levels[8].

Suspected mechanisms include corneal thickening, increased lenticular axis length, refractive index changes in the cornea and crystalline lens, osmotic pressure gradients from aqueous, lens and vitreous[8]. **Figure 11** shows diagrammatic versions of hypoglycaemia, normal blood glucose levels and hyperglycaemia.

Any patient who is having variable prescription and acuity, in the absence of other causes, may be either an undiagnosed diabetic (ask about other risk factors/symptoms) or a diabetic patient who has poor control of their condition.

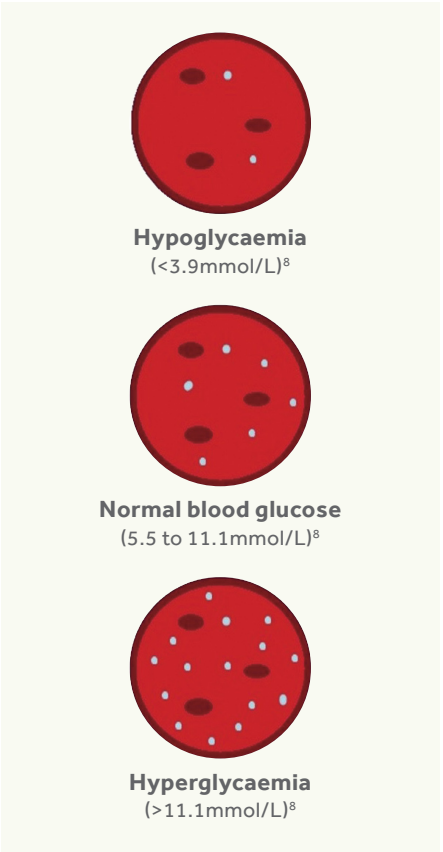

FIGURE 11: Blood glucose levels[9]

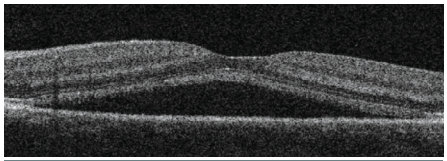

FIGURE 12: OCT image of central serous retinopathy

CENTRAL SEROUS RETINOPATHY

Central serous retinopathy (CSR) normally involves an idiopathic accumulation of fluid underneath the sensory retina, which causes a forward displacement of the foveal area which induces a hyperopic shift of up to +1.25DS.

Figure 12 shows an ocular coherence tomography (OCT) image of CSR; the dark area under the sensory retina but anterior to the retinal pigment epithelium (RPE) is free fluid arising from the choriocapillaris.

The patient demographic for this condition tends to be a 30-50-year-old male with a type-A personality (anxious/highly strung). They present with symptoms of progressive blur and or metamorphopsia (distortion) in the affected eye. The photoreceptor layer of the retina is still intact and functional but displaced anteriorly due to the sub-retinal fluid.

The patient's BCVA may still be normal or near to normal with their modified prescription, but the hyperopic shift should alert the practitioner to the potential of CSR. The condition can be self-limiting, resolving in months, monitored in the Hospital Eye Service, but occasionally it may require treatment.

CONCLUSION

The spectacle prescription is realistically 'just a bunch of numbers' telling us the power of lens to order. It tells us nothing of the patient's visual acuity or eye health status. Should it be a concern that, with the possibility of online refraction tools, patients will think nothing of the reason their prescription has changed? The author feels the answer must be yes.

A refraction leading to a spectacle prescription, without taking history and symptoms and a thorough examination of the eyes themselves, could lead to missed pathology and potentially long-lasting harm to the patient.

The aim of this article was to highlight some of the reasons for spectacle prescription changes – and make us consider what's behind the numbers of the prescription.

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LEARNING OUTCOMES FOR THIS CPD ARTICLE

DOMAIN: Communication

2.1: *Utilise appropriate lay-term language and communication approaches when discussing conditions with patients who have pathology induced refraction changes.*

DOMAIN: Clinical Practice

5.4: *Understand how vision may be affected by a range of pathologies and reflect on areas where a greater level of knowledge may be required for your professional practice.*

7.7 : *Develop an understanding that there may be a need to consult with professional colleagues when patients present with changes to their vision.*

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