General Optical Council Approved CET For Optometrists
General Optical Council Approved CET For Dispensing Opticians
General Optical Council Approved CET For Specialist Optician Contact Lenses

Basic contact lens course

Part 12 - Complications and management

Andy Franklin and **Ngaire Franklin** describe what to look out for at the aftercare appointment. Module C15274, one general CET point for optometrists and dispensing opticians, one specialist point for contact lens opticians

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Most aftercare appointments are fairly routine affairs, and as lens materials, lens designs, and the care systems used with them have improved, some of the complications that were common have all but disappeared. Nevertheless, from time to time intervention is necessary to resolve problems which have arisen, and we can save time and money for both patient and practitioner by adopting a systematic approach. The strategy for effective management of complications involves the following five steps:

1) Know your enemy. Correct identification of the root of the problem will save time and inconvenience. With that in mind, the families are as follows, though there is some overlap between them:

- Hypoxia
- Drying
- Mechanical insult
- Toxic and hypersensitivity reactions
- Sterile inflammation of the cornea
- Microbial keratitis.

2) Change one thing at a time, and see if it resolves the situation before making any more changes. A scattergun approach may solve the problem at least as fast, but you won't know why. Should the problem recur, you will be none the wiser about what you need to do to tackle the problem.

3) Always keep in mind a worse case scenario for the sign and symptoms you have collected, and an idea of the likely timescale involved. If this involves serious risk to the patient, as it will if microbial keratitis is suspected, make sure that you see the patient again before events can take their course. Bacterial ulcers become serious over hours rather than days, so seeing the patient in a week or so might be considered a little casual by m'learned friends.

4) Unless the worse case considerations are overriding, allow enough time for the changes to take effect. If the oxygen transmission is improved to eliminate microcysts, it makes little sense to see the patient in a fortnight, when the microcysts are certain to be more numerous. If seen in three months, you will be able to tell if your management has worked.

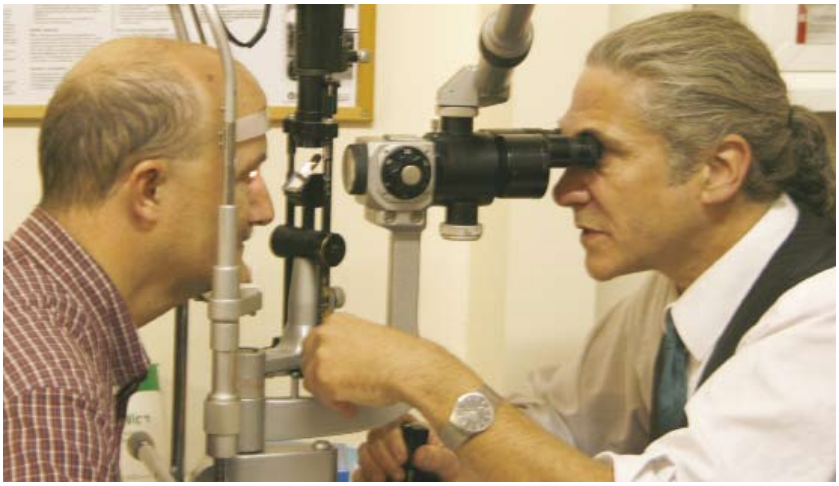

Figure 1 Use of the slit lamp during the aftercare appointment

5) Try not to 'bench-rest' patients. If a finding is not serious enough to do anything about, it will probably not be in a fortnight either. If the problem is too serious to ignore, it will probably not improve on its own, though there are always exceptions to every rule. Patients can usually tell when the practitioner is faffing about, and a proactive approach with objectives that are clear to both parties is generally more reassuring. Recording these objectives on the clinical record is essential and if instructions are lengthy then it is worth considering writing them down for the patient.

Hypoxia

With the increasing introduction of silicone hydrogel materials, hypoxia is increasingly less of an issue. Symptoms tend to be non-specific if mild, and patients will often complain of dryness when the actual cause is hypoxia. In more severe cases, the cornea may become oedematous, leading to a loss of contrast and light scattering which may cause photophobia towards the end of the wearing period. On the slit lamp, the first sign may be hyperaemia around the limbus. Corneal signs will depend on the severity of the condition. Visible areas of localised oedema are rare in soft lens wearers and more subtle signs should be sought. The signs are most likely to appear beneath the part of the lens that transmits the least oxygen, but they are often more easily detected in the central area against the dark background provided by the pupil. The degree of oedema present at the time of examination may be indicated by the presence of striae and folds.

- **Striae** are seen as fine, usually vertical grey-white lines in the posterior stroma. They are best observed with indirect illumination using a parallelepiped beam at about 16-20X magnification, against the background of the pupil area. Striae begin to appear when the level of oedema reaches about 5 per cent. In RGP wearers they tend to appear in clusters rather than singly, if the level of oedema is sufficiently high. They are probably caused by ▶

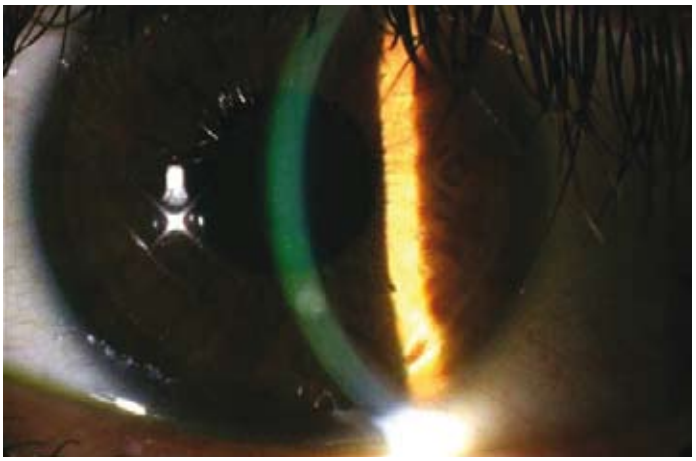

Figure 2 An old scar from a previous inflammatory event

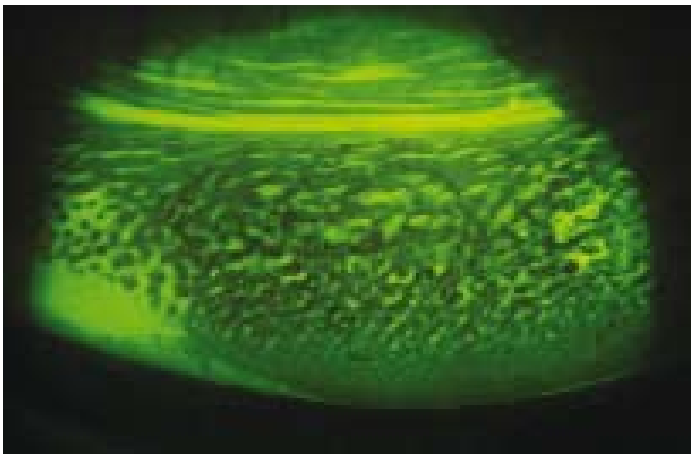

Figure 3 Fluorescein helps the assessment of the cornea and other surfaces, such as lower palpebral conjunctiva as seen here

fluid separation of the collagen fibrils in the posterior stroma, which are predominantly vertically arranged.

● Folds can be observed in the endothelial mosaic using specular reflection, appearing as grooves and ridges. If severe, they may appear as dark branching lines in direct illumination. They are caused by buckling of the posterior stroma with high levels of oedema. They appear when the level of oedema is about 15 per cent and the cornea is likely to be somewhat hazy when in this state.

Acute hypoxia is probably less significant than chronic hypoxia, and the latter may be detected by the following clinical signs:

● Epithelial microcysts and vacuoles appear as small grey dots in the epithelium in direct illumination. Initially they may be difficult to tell from dust particles in the tear film, but if the patient blinks, they are the ones that don't move. The best way to identify them is to use an angled parallelepiped beam at high (40 times) magnification. The area of cornea to observe is that where the indirectly illuminated and retro-illuminated areas meet, that is in marginal retro-illumination

● Microcysts, because they have a higher refractive index than the surrounding tissue, show reversed illumination under these conditions and may appear like tiny pin-pricks. Vacuoles are generally slightly bigger and do not show reversed illumination, so they appear as small bubbles. They represent fluid collected in the intracellular spaces and are therefore indicative of oedema, which maybe caused by hypoxia or hypertonic ocular exposure. It is possible that these intercellular spaces are exploited by *Acanthamoeba* to gain access to the cornea, so the presence of significant numbers should not be tolerated. Some patients may have a few of these independent of contact lens wear. Microcysts are probably apoptotic (ie dead) cells that are either ingested by phagocytes or encapsulated by material from the basement membrane and eventually expelled after travelling through the corneal layers. They are probably created by a combination of hypoxia, which produces lactic acid and hypercapnia (increase in carbon dioxide levels) which creates carbonic acid. They may also be induced by mechanical trauma in some cases. They can be eliminated by improving the level of oxygen available, but the recovery process is unusual. Initially, the number of microcysts will increase, as the corneal metabolism speeds up and cellular debris is removed more efficiently. There is then a gradual decrease in the number until they are finally eliminated. This can take 3-5 months. The point at which microcysts become significant is subjective, but generally if staining is also present intervention is required

● Changes to the endothelium may also occur in response to the acidosis caused by hypoxia and hypercapnia. An acute response is observed in all contact lens wearers within a few minutes of lens insertion. When the endothelium is observed by the specular reflection, a number of dark areas can be observed within the endothelial mosaic. These are blebs, and represent swollen cells that disrupt the smooth mirror-like surface of the endothelium/aqueous interface. After 20-30 minutes the number of blebs peaks and thereafter falls over the next hour, though some blebs will be visible throughout the wearing period. The chronic response to acidosis is polymegathism, where the cells of the endothelial mosaic appear to vary markedly in size. Polymegathism occurs naturally with age, so the endothelium should be judged against expectations for a given age group. It is not easy to assess the endothelium accurately with a slit lamp. The highest magnification available is usually 40X or less, and even at 40X with a good slit lamp the best that can be seen is a textured area, and only the more advanced degrees of polymegathism may be detected with any reliability. This is most likely to be seen with low-Dk lenses, especially 38 per cent HEMA, and in hydrogel extended wear. Polymegathism is a response to significant metabolic stress and remedial action should be taken if it is detected. Recovery is at best very slow, and may not occur at all

● Neovascularisation is a common finding in conventional hydrogel soft lens wearers. Up to half a millimetre of vessel growth is commonly seen as a physiological response to lens wear and need not necessarily lead to greater vessel growth. To those unused to contact lens aftercare, half a millimetre can look quite scary under high magnification, and it is useful to measure the true extent of the vessels either by reference to a graticule in the eyepiece of the slit lamp or by comparing the vessels to the overlap of the lens beyond the limbus. This is typically 1-1.5mm, depending upon the HVID of the patient and TD of the lens. It is important to determine whether we are looking at a condition that is active, static or receding. The presence of limbal hyperaemia is a useful clue, as an eye with undilated limbal arcades is less likely to be under hypoxic stress. Neovascularisation is an inflammatory process, and the presence of diffuse infiltrates around the leading vessels is not a good sign. Conversely, the presence of 'ghost vessels' would suggest that the cornea is currently receiving adequate oxygen.

Significant chronic hypoxia is known to increase the risk of microbial keratitis, and it is now easy to address. The management of hypoxia consists, not surprisingly, of arranging for greater oxygen availability. This may be achieved by:

- Wearing the lenses less. This may be effective in the short term, but rarely in the longer term. The wearing pattern that a patient adopts is largely dictated by convenience to that individual and the patient will probably return to their previous wearing pattern sooner rather than later
- Improving the flow of tears under the lens may improve oxygen levels to a limited extent. A smaller total diameter, greater edge lift and smoother transitions may bring some improvement
- By far the most effective strategy is to use a more permeable material. There are materials available now that have sufficient permeability to eliminate the sign of hypoxia in any normal cornea. It should be borne in mind that if microcysts are being used as an indicator, that refitting with a high-Dk material will initially increase the number, so an aftercare interval of about three months is useful unless contraindicated by other clinical concerns.

Drying

Many patients complain of symptoms of dryness, but not all of them are actually due to drying. Considerable research has been undertaken with a view to improve the wetting performance of contact lenses in recent years, but the improvement in patient satisfaction, while considerable, has been less than perhaps anticipated. What patients are actually complaining of is persistent, progressive mild irritation and lens awareness, and this could be due to hypoxia or mild inflammation instead of drying per se.

Drying can cause lens awareness or discomfort, and this usually worsens progressively throughout the wearing time. The vision is often variable due to the accumulation of deposits on the lens, and again this gets worse towards the end of the wearing period. The other frequent patient complaint is of red eyes, and characteristically this is associated with hyperaemia of the bulbar conjunctiva in the area exposed between the lid margins.

Management of dryness can involve a number of strategies:

- Elimination of causative factors, especially the various forms of blepharitis, should be pursued. Lid scrubs and hot compresses will be useful for MGD and seborrhoeic anterior blepharitis, while staphylococcal blepharitis will need antimicrobial treatment which may require routine referral to a general medical practitioner. However, if the dryness of the eyes is caused by systemic medication or a medical condition, elimination of the cause may be outside the control of the contact lens practitioner
- Careful selection of care systems may help to reduce or remove deposits. Lipid deposits are often seen with solutions containing chlorhexidene. Protein deposition may require the use of an enzyme cleaner. Compliance with cleaning regimes should also be encouraged
- Old lenses tend to deposit more, so planned replacement may help considerably, particularly as modern materials tend to scratch more easily
- The use of surface-treated lenses or hybrid materials may improve comfort considerably as well as reducing deposition
- If all else fails, rewetting or 'comfort' drops may relieve symptoms. However, they are rarely a long-term solution as patients eventually either stop using them or use them so infrequently that they make little difference. In the past, a few patients went to the other extreme, instilling preserved comfort drops by the bucket-load and eventually becoming allergic to them.

Mechanical insult

A number of findings caused by mechanical insult may be seen:

- Steep lenses may trap small bubbles under the optic zone. These can cause small circular depressions in the cornea, which may retain fluorescein, though the epithelial surface is intact. With the major slit lamp it looks like rather coarse, and neatly circular, punctate staining. This dimple veiling is usually asymptomatic, though if severe some visual degradation may be noticed. A flatter fit, increased edge lift or smoother transitions will generally eliminate it. Dimple veil can also be associated with mucin balls. These are created by sheer forces acting on the mucin component of the tears, and with the lens in place appear as small grey bodies between the back surface of the lens and the cornea. They rarely cause problems but there are reports of a correlation between mucin balls and an increased frequency of inflammatory events
- SEALs (superior epithelial arcuate lesions) (Figure 4) may appear in the upper, and occasionally lower, cornea as arcuate areas of confluent staining. They appear to more common in patients wearing the 'stiffer' silicone hydrogels than those wearing hydrogels, leading to the conclusion that most SEALs are caused by mechanical forces under the lens. However, many other factors have been associated with SEALs over the years, including tight lids, hypoxia and solution reactions. The patient may be asymptomatic or have mild discomfort. If lens wear is suspended for two weeks, about half will resolve and are

unlikely to recur on resumption of wear. However, the rest will need refitting, often with a 'softer' lens. Unfortunately only about half of them will resolve, and suspension of lens wear may be necessary, given that SEALs represent a significant epithelial defect

- Mechanical irritation may also induce papillary conjunctivitis in silicone hydrogel wearers, usually of the upper lid. The earliest sign is hyperaemia of the upper palpebral conjunctiva relative to conjunctiva of the lower lid, though this may be asymptomatic. Later papillae will appear, and may coalesce to form giant papillae with diameters over 1mm. The papillae themselves can are hyperplastic vascular tissue and have a central vascular core. Should the papillae become inflamed, the lens may adhere to them and decentre, creeping up under the lid. It may also be caused by an immune response to deposits on the lens, and this is thought to be the more common aetiology in hydrogel wearers. Management will depend on the perceived cause. Mechanical papillary conjunctivitis may be tackled by improving the edge profile, reducing edge stand-off by changing the total diameter or reducing edge lift in one or more meridians. That due to deposits may be improved by more effective cleaning and frequent lens replacement. The use of non-preserved care systems may also useful in these cases
- Patients may present at aftercare appointments with staining in the 4 and 8 o'clock positions which sometimes can extend across the whole lower cornea, thus mimicking drying stain. This may indicate mechanical abrasion caused by a clumsy lens removal technique, whereby the lens is pinched straight off the cornea rather than moving down on to the sclera first.

Toxic and hypersensitivity reactions

Intolerance to solutions is considerably less common than was once the case, due to improvements in their formulation, particularly to the preservative elements. However, they are still encountered from time to time. Some become apparent within a day or two of first use of the solution, but there are occasions when the symptoms gradually build up over time before they become severe enough for the patient to consult their practitioner. Early clinical signs may be apparent in an asymptomatic patient. The symptoms associated with solution intolerance are typically noticed immediately after the lenses are inserted, and may resolve if the lenses are kept in and the solutions diluted by the tear film. Lens awareness, itching, burning and sensations of dryness are all common. In severe cases photophobia may be reported.

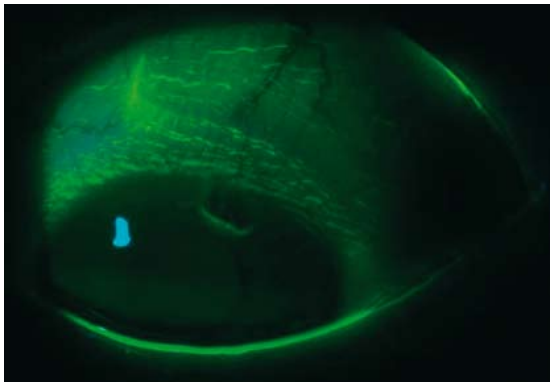

Figure 4 Superior epithelial arcuate lesion

The clinical signs may include the following in patients with mild or no symptoms:

- Hyperaemia of the palpebral and bulbar conjunctiva. The latter may be in a diffuse pattern, more marked around the limbus
- Superficial punctate keratitis (SPK). This may favour the lower cornea, but is typically diffused over the corneal surface
- Occasionally, infiltrates may be observed. These are usually intra-epithelial or sub-epithelial. They may be discrete or diffuse, and tend to favour the area just inside the limbus.

More severe reactions will have symptoms, sometimes quite marked, and the following signs may be present:

- The lids may be swollen
- The tear film may be unstable, and mucus strands may be visible. Reflex lacrimation is common
- Conjunctival hyperaemia
- Diffuse SPK
- In hypersensitivity reactions, infiltrates may be observed, but there is usually a delay of about 24 hours between the initial signs and symptoms and the appearance of infiltrates.

The management of solution reactions will depend on their severity. If the symptoms and signs are severe, it is wise to suspend contact lens wear until the eye returns to normal, especially as the signs of microbial infection may be rather similar. If there is any question of infection, the patient should be seen the next day. Once the initial reaction has subsided, or if the clinical signs are mild to begin with, management involves identifying likely triggering agents and avoiding them. The prime suspect in these cases is usually the preservative in the conditioning solution. However, a surprising number of patients clean their lenses before insertion, and may not rinse them thoroughly before insertion, so the cleaning solution may also be a factor to consider. Buffering agents and residues from enzymatic systems may also be suspected on occasion. Careful questioning of the patient is necessary to establish precisely what solutions are in use, and how they are used. It may not even be the solutions used for the lenses, as self-prescribed or GMP-prescribed eye-drops can cause the same reactions. Where more than one potential suspect is present, eliminate them one at a time, in descending order of probability, until the signs and symptoms are eliminated. Strictly speaking, a causal relationship can only be proven by reintroducing the suspected agent and observing the return of clinical signs. However, this may be a test too far for most contact lens wearers, so this step is usually omitted.

STERILE INFLAMMATION OF THE CORNEA

Inflammation of the cornea is not specific to one causative agent. The same response will occur whatever the initial trigger. The trigger can be trauma, toxicity or immune response and the common factor is that corneal cells, usually in the epithelium, become distressed and release chemical agents which initiate the inflammatory response. Contact lens wear tends to potentiate all of the likely triggers:

- Trauma is more likely as the cornea may be hypoxic, which makes the epithelium more fragile and slow to repair. Insertion and removal of the lenses may induce mechanical insult
- Contact lens solutions, deposits and bacterial toxins are all capable of inducing a toxic response. In extended wear, the products of dead epithelial cells may also be a source, as they may be trapped under the lens for some time. Immobile lenses may keep the dead cells in one location which will increase the likelihood of localised epithelial distress
- Solutions deposits and bacterial toxins may also cause immune responses, and the cornea may also react to chemicals released by adjacent inflamed tissues, such as the palpebral conjunctiva. Infiltrates are sometimes noted as an 'innocent bystander' effect of CLPC.

In the anterior eye these signs are often fairly subtle unless the response is severe, and a severe response always suggests the possibility of infection. ▶

In the cornea, the most useful sign of inflammation is the presence of infiltrates, which are collections of white blood cells. These may form discrete patches or diffuse areas in the epithelium and anterior stroma. Generally speaking the more serious the cause, the deeper they are and the more likely they are to be central, but this is only a rough guide.

Clinically, corneal infiltrates can be divided into the following categories, in ascending order of seriousness.

- Asymptomatic infiltrates (AI)
- Asymptomatic infiltrative keratitis (AIK)
- Infiltrative keratitis (IK)
- Contact lens associated red eye (CLARE)
- Contact lens peripheral ulcer CLPU).

Most serious of all is infective or microbial keratitis and this will be dealt with in a future article.

Asymptomatic infiltrates are sometimes seen in non-contact lens wearers (about 5 per cent), and are probably unrelated to contact lens wear, as they have a similar incidence in contact lens wearers. They may be induced by environmental factors such as air pollution. Typically there is one or more small (up to 0.2mm) discrete, greyish-white patch anywhere on the cornea. These are intra-epithelial or occasionally sub-epithelial (an optical section at high magnification will indicate how deep the infiltrate is). There are no symptoms or other signs of inflammation. No action is required.

In asymptomatic infiltrative keratitis there is a diffuse infiltrate in the peripheral parts of the cornea, sometimes with some discrete infiltrates as well. It doesn't appear to cause any problems in itself, but it may be a mild form of CLARE, with similar causes, so the patient's care of the lenses should be under scrutiny.

Infiltrative keratitis can present as a diffuse or focal infiltrate, but here it is accompanied by symptoms of discomfort or pain, and by bulbar conjunctival hyperaemia, especially around the limbal area. The focal form is probably a response to local epithelial trauma caused by a foreign body trapped under an immobile lens. The diffuse form may be a mild form of CLARE response, with similar aetiology.

CLARE itself is a complication of extended wear, and typically the onset is in the early hours of the morning, after a period when the eyes have been closed. It is less common with RGP lenses than soft, but it has been reported with bound, immobile lenses. There is an association with gram-negative bacteria, as about a third of CLARE cases have contaminated lenses. The symptoms vary from mild discomfort to pain, and there is marked bulbar hyperaemia, especially around the limbus. Feint diffuse infiltrates are seen arising from the limbal arcades, though there may be focal infiltrates present as well. Following a CLARE reaction, fine keratic precipitates (bedewing) may appear on the corneal endothelium. These are the result of mild anterior chamber activity and persist for about six months, so they can be a useful clue to past inflammatory events that the patient may not report.

CLPU presents as a round or oval greyish-white infiltrate with an overlying full-thickness epithelial defect. It is generally located near the periphery of the cornea, though there is a band of clear cornea between the infiltrate and the limbus. They may be asymptomatic or painful. Hyperaemia may be generalised or limited to an area adjacent to the lesion. They are culture negative, though a correlation has been found with high levels of gram-positive bacteria. If lens wear is suspended, signs and symptoms will resolve in 48 hours, except for the infiltrate that can persist up to three months, though it usually resolves within one month. It is likely that the infiltrate represents a response to localised trauma or toxicity and that the epithelial loss is a result of leukocyte action. However, given the epithelial defect and the bacterial correlation, caution is advisable, and the patient should be seen the next morning if they are not referred.

The management of sterile inflammation depends on its severity, and upon the chances that it might be an infection. The asymptomatic and white-eyed forms generally need no intervention, though CLARE's smaller sisters might be regarded as a warning shot from the bacteria, and lens hygiene might be worth some scrutiny. The symptomatic forms will require suspension of lens wear. This should ideally be until infiltrates have resolved, unless we are sure that the cause is unthreatening. The time required will vary with the location and depth of the infiltrate. Intra-epithelial infiltrates resolve within 2-3 weeks, but sub-epithelial and anterior stromal infiltrates take longer, up to three months in some cases. Anything that persists longer than that in the absence of active inflammation is probably a scar, and these tend to have a 'bull's-eye' appearance, with a fainter centre.

● **Ngaire Franklin** and **Andy Franklin** are contact lens specialist optometrists practising in the South West

MULTIPLE-CHOICE QUESTIONS – take part at opticianonline.net

1 Where might striae appear?
A Epithelium
B Anterior stroma
C Posterior stroma
D Endothelium

2 Where might folds appear?
A Epithelium
B Anterior stroma
C Posterior stroma
D Endothelium

3 Where might microcysts appear?
A Epithelium
B Anterior stroma
C Posterior stroma
D Endothelium

4 Where might blebs appear?
A Epithelium
B Anterior stroma
C Posterior stroma
D Endothelium

5 In what percentage of non-contact lens wearers are asymptomatic infiltrates found?
A Zero
B 5 per cent
C 10 per cent
D 35 per cent

6 How long do sub-epithelial infiltrates take to resolve?
A 48 hours
B One week
C Four weeks
D Three months

Successful participation in this module counts as one credit towards the GOC CET scheme administered by Vantage and one towards the Association of Optometrists Ireland's scheme.
The deadline for responses is December 30 2010