



1 CET POINT

# OCT – Discover what lies beneath

Rachel Hiscox BSc (Hons), PhD, MCOptom

40

25/04/14 CET

The use of optical coherence tomography (OCT) in the consulting room is rapidly gaining popularity, with this technology now found in many practices around the UK. This article offers a clear understanding of the retinal landmarks highlighted in a normal OCT scan and the images observed in common retinal pathologies.

**Course code: C-36203 | Deadline: May 23, 2014**



## Learning objectives

To be able to explain to the patient about the implications of retinal disease (Group 1.2.4)  
To be able to recognise retinal abnormalities and refer appropriately (Group 6.1.5)



COMMUNICATION



OCULAR DISEASE



## Learning objectives

To be able to understand the implications of retinal disease (Group 8.1.5)



OCULAR ABNORMALITIES



## Learning objectives

To be able to understand the natural progress of retinal disease and assess severity of different presentations (Group 1.1.1)  
To be able to identify the nature, severity and significance of the different types of retinal disease that present in practice (Group 2.1.2)



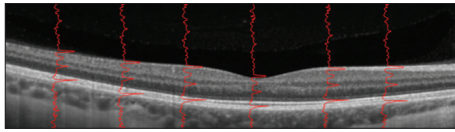
KNOWLEDGE



OPTIONS

## About the author

**Dr Rachel Hiscox** is an optometrist and clinical affairs specialist at Topcon. She has worked in both private practice and within a hospital environment. She has recently completed a PhD at Cardiff University investigating retinal structure in cystic fibrosis using optical coherence tomography.



**Figure 1** Representation of an OCT B-scan (grey-scale image) generated by multiple A-scans (red plot lines)

## Introduction

Optical coherence tomography (OCT) is a non-invasive, non-contact, trans-pupillary imaging technique able to produce high-resolution images detailing the structure of the eye in 3D, from the anterior segment to the posterior pole. With use of OCT growing in the primary eye care setting, this article explores the science behind OCT, discusses image interpretation and presents examples of common retinal abnormalities.

## Background – the science

Since OCT was first demonstrated in 1991,<sup>1</sup> it has rapidly evolved as the only non-invasive diagnostic technique able to provide images of the retinal microstructure, which directly relates to the histological structure of the retina. OCT generates cross-sectional or three-dimensional images by utilising low coherence interferometry to detect and measure the depth and magnitude of back scattered (reflected) light.<sup>2</sup> Low coherence, near-infrared light (~850nm) is emitted from a super-luminescent diode laser<sup>3</sup> and travels to the interferometer where it is split into two equal components by a semi-transparent mirror. One component is then directed towards the retina through the ocular media (the measurement beam), while the other is directed to a reference mirror (the reference beam). The distance between the beam splitter and the reference mirror is continuously varied until the distance between the light source and the retinal tissue is equal to the distance between the light source and reference mirror. When this occurs, the reflected light from the tissue being imaged and the light from the reference mirror interact to produce an interference pattern, which is detected by a photosensitive detector and processed into a signal.

A two-dimensional, cross-sectional retinal image is produced as the light source scans across the retina, stacking and aligning consecutive axial scans (A-scans) side by side to produce a two dimensional transverse scan (B-scan) (see Figure 1).<sup>4</sup> Eye movements are

corrected by digital processing (cross-correlation scan registration) to align the A-scans, and digital smoothing techniques are used to further reduce image noise.<sup>5</sup> The image produced resembles that of a histological section, with contrast produced by differences in the refractive index and scattering properties of the retinal layers.

OCT is an essential tool for the identification of the early stages of disease, before symptoms and irreversible vision loss occur. It can also be used to track disease progression and monitor treatment outcomes.

## Retinal image interpretation

A healthy retinal OCT B-scan is shown in Figure 2. As previously discussed, differentiation of the retinal layers is possible due to their varying scattering properties, and differences in optical densities. Large reflections are depicted by warm colours (yellow to red) with smaller reflections depicted by cooler colours (blue to green). Images in grey-scale utilise brighter shading in place of warmer colours. Where there is no reflection, the image appears black in both colour and grey-scale. As the vitreous is not very dense, it appears black. Similarly, if fluid is present, this will also appear black. Conversely, structures including the retinal nerve fibre layer (RNFL) and retinal pigment epithelium (RPE) are much more dense, and therefore appear bright/red.

When new to interpreting OCT images, a couple of normal anatomical features can cause the viewer concern. Firstly, because blood vessels are highly reflective, they cause a shadow to fall underneath them in the OCT scan by blocking the infrared OCT signal (see Figure 2). This can also occur with dense vitreous floaters, which will cast a shadow across all retinal layers of the OCT scan. Secondly, as the viewer moves over the foveal region of an OCT scan, the outer-segments of photoreceptors appear to become oedematous. This

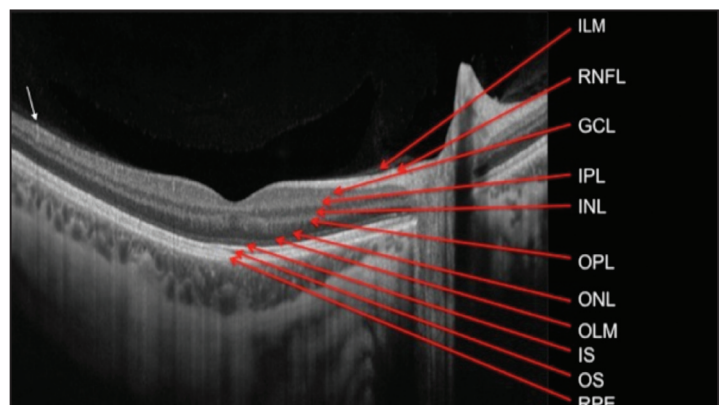
will cause obvious concern to those new to OCT interpretation – however, this is a normal feature of the fovea, representing the elongation of cone photoreceptors, which enables closer packing and hence provides high visual acuity.

## Common ocular conditions

### Age-related macular degeneration (AMD)

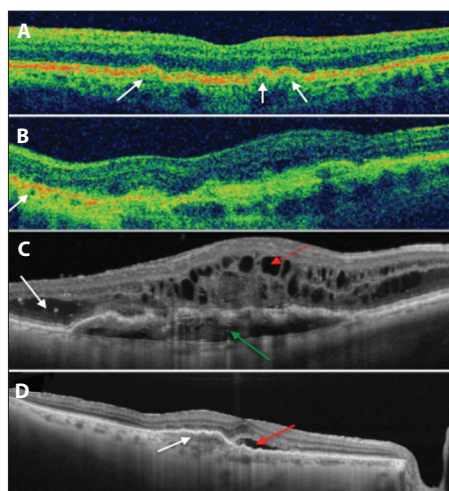
Age-related macular degeneration (AMD) is the principle cause of irreversible blindness among those aged over 65 years in the western world.<sup>6-8</sup> Currently, the prevalence of AMD in adults is approximately 3%,<sup>9</sup> however, this is likely to rise with increasing longevity, and the shift towards an ageing population.<sup>10</sup> AMD can be broadly classified into two states: wet and dry. Dry AMD, characterised by drusen within the macular region, is the most common type, accounting for up to 90% of all cases of AMD.<sup>11</sup> It is generally accepted that dry AMD precedes wet AMD,<sup>12</sup> which is characterised by growth of new vessels from the choroid (choroidal neovascularisation).<sup>13</sup> Dry AMD causes gradual deterioration of central vision as a result of retinal and RPE atrophy, whereas visual loss is sudden in wet AMD due to neovascularisation and subsequent leakage from the new, weak vessels.<sup>14,15</sup> While both forms can cause significant visual loss, wet AMD accounts for approximately 75% of cases with severe vision loss.<sup>16</sup>

One of the clearest benefits of OCT in practice is to differentiate between those patients with wet and dry AMD; therefore, avoiding unnecessary referrals and also giving practitioners the ability to detect dry AMD changes before drusen become visible at the



**Figure 2** A healthy retinal OCT scan showing the macula and optic nerve. The white arrow shows hyper-reflectivity of a blood vessel and a shadow behind it. Key: ILM (inner-limiting membrane); RNFL (retinal nerve fibre layer); GCL (ganglion cell layer); IPL (inner-plexiform layer); INL (inner-nuclear layer); OPL (outer-plexiform layer); ONL (outer-nuclear layer); OLM (outer-limiting membrane); IS (photoreceptor inner-segment); OS (photoreceptor outer-segment); RPE (retinal pigment epithelium)

# 1 CET POINT



**Figure 3** A) Drusen in dry AMD appear as focal elevations of the RPE (white arrows). (B) Atrophy causes increased penetration of the measurement beam to the choroid, which appears hyper-reflective (white arrow). (C) Wet AMD showing cystoid macular oedema (red arrow), sub-retinal fluid (white arrow) and PED (green arrow). (D) Wet AMD showing sub-retinal fluid (red arrow) and choroidal neovascularisation (white arrow)

retinal surface.

Drusen, the hallmark of dry AMD,<sup>17,18</sup> consist of extracellular deposits of material, collected between the basal lamina of the RPE and the inner layer of Bruch's membrane.<sup>11</sup> Common constituents of drusen include RPE remnants, complement, lipids, lipoproteins, dendritic cell processes, cholesterol esters, fibrinogen, Class II antigens and immunoglobulins.<sup>12,19-21</sup> On OCT examination, drusen appear as focal, hyper-reflective elevations of the RPE, disrupting the typically straight and smooth RPE (see Figure 3A).

Atrophic AMD accounts for approximately 25% of cases with severe central visual loss.<sup>16</sup> It is characterised by RPE dysfunction and death, leading to loss of photoreceptors which are unable to survive without the nutritional and metabolic support of the RPE.<sup>22</sup> Large areas ( $\geq 175\mu\text{m}$  diameter) of atrophy are termed 'geographic atrophy',<sup>22,23</sup> representing end-stage dry AMD and accompanied by severe visual loss. Geographic atrophy can develop following fading of drusen, involution of choroidal neovascularisation (CNV) or following a resolving pigment epithelial detachment.<sup>11</sup> Atrophy is easily discernable with OCT as retinal thinning due to loss of retinal structures, accompanied by increased hyper-reflectivity of the choroid due to lack of attenuation of the OCT signal by the

absent RPE (see Figure 3B).

Development of CNV is the hallmark of wet AMD, a stage found in approximately 10% of all AMD cases. CNV refers to the growth of new blood vessels from the choroid, which may remain beneath the RPE ('occult' CNV) or breach the RPE and enter the sub-retinal space ('classic' CNV).<sup>11</sup> CNV on OCT examination typically presents as increased reflectivity of the RPE, often associated with irregular RPE elevation (see Figure 3D). Leakage of these new blood vessels causes development of intra-retinal fluid, which appears as dark spaces within the retina (see Figure 3C and 3D). In cystoid macular oedema, a condition that is also associated with diabetes and branch retinal vein occlusion, the fluid forms characteristic cystic spaces (see Figure 3C).

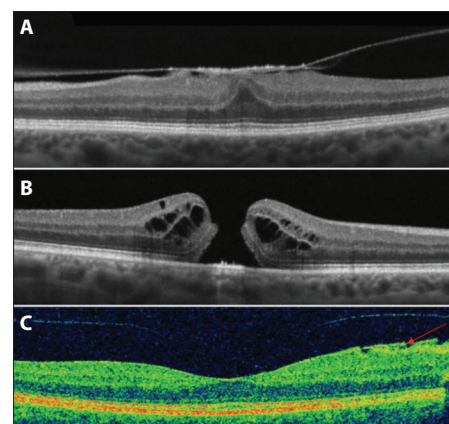
Retinal pigment epithelial detachment (PED) is a common finding in both dry and wet AMD, formed by separation of the RPE from Bruch's membrane due to the presence of sub-RPE fluid, blood, fibrovascular membrane or drusenoid material. Figure 3C shows a serous PED associated with wet AMD. Small PEDs can be distinguished from drusen as they lack hyper-reflectivity under the RPE elevation.

## Vitreo-retinal traction

The prevalence of posterior vitreous detachment (PVD) increases with age, as the vitreous liquefies. While uncomplicated PVD occurs in the majority of cases, the incidence of retinal complications in symptomatic PVD has been reported to be as high as 24%<sup>24</sup> and include retinal tears, tractional retinal detachments, macular holes and epiretinal membrane (ERM) formation.<sup>25</sup> These complications are secondary to abnormally strong adhesions between the vitreous and the retina. While this strong attraction cannot be imaged with standard fundus examination techniques, it is easily observed with OCT (see Figure 4A), where vitreo-retinal traction is seen as a thin, moderately reflective band, pulling on the retina in an incomplete v-shaped PVD.

## Macular hole

If vitreo-retinal traction persists it can lead to the formation of a macular hole, which is typically seen in females in the sixth or seventh decade of life, with a prevalence of 1/3300.<sup>26</sup> It is now accepted that the early event leading to



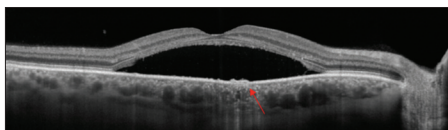
**Figure 4** Vitreo-macular traction (A) which could progress to a full thickness macular hole (B), or cause development of an ERM (C) (red arrow)

macular hole formation is persistent vitreoretinal traction. The resultant traction on the fovea causes foveal detachment or an intraretinal space – termed pseudocyst – that appears on OCT as a dark cystic space. Continued traction leads to dehiscence of foveal tissue, resulting in full-thickness macular hole formation as shown in Figure 4B.<sup>27,28</sup> Observation of an OCT image of a full thickness macular hole makes it easy to understand why patients experience a central scotoma and distortion due to absence of photoreceptors in the centre of the hole and the presence of a cystic fluid in the cuffs, respectively.

## Epiretinal membrane (ERM)

ERM – also known as cellophane maculopathy and macular pucker – occur in 2.2% to 18.5%<sup>28</sup> of the population and, while it may be idiopathic, it can occur secondary to intraocular inflammatory conditions, retinal vascular disease, after surgical intervention or following a complete or incomplete PVD. ERM typically forms between the ILM and the vitreous body interface<sup>29</sup> and consists of proliferation of retinal glial cells which gain access to the retinal surface through defects in the inner-limiting membrane. Visual symptoms can range from mild to severe, with blurred vision and metamorphopsia. On OCT examination, ERM appears as a highly reflective layer on the inner retinal surface that is either totally adherent or partially adherent to the retina (see Figure 4C). ERMs are often accompanied by retinal folds and are distinguished from the posterior vitreous base by the formation of a thicker reflective band.





**Figure 5** Serous detachment of the sensory retina in CSR with RPE disruption (red arrow)

### Central serous retinopathy

Central serous retinopathy (CSR) is a sporadic disorder of the outer blood-retinal barrier, typically affecting young to middle-aged men with Type A personality.<sup>30</sup> CSR is reported to be aggravated by stress, untreated hypertension, high alcohol intake and corticosteroid use.<sup>31</sup> Symptoms often include metamorphopsia, blurred vision and relative positive scotoma. OCT examination of CSR is characterised by a well-defined round or oval serous detachment of the sensory retina in the macula area (see Figure 5). An accompanying RPE defect can often be seen. Research using indocyanine green angiography (ICG) have shown multiple areas of choroidal vascular hyperpermeability, vascular congestion and venous dilation<sup>32</sup> suggesting that CSR is associated with a generalised choroidal vascular disturbance.<sup>33</sup> Spontaneous resolution and absorption of fluid occurs in most cases within three-to-six months, with return to normal or near-normal vision, however, recurrence does occur in up to 50% of patients. A small percentage of patients may experience chronic disease which is associated with permanent visual loss following atrophy, scarring and secondary choroidal neovascularization,<sup>34</sup> therefore, careful follow-up and patient education is advised.<sup>35</sup> Treatment of prolonged or chronic CSR is typically with laser or photodynamic therapy (PDT).<sup>35</sup>

### Diabetic retinopathy

Diabetes currently affects approximately three million people in the UK alone, with diabetic eye disease one of the leading causes of visual

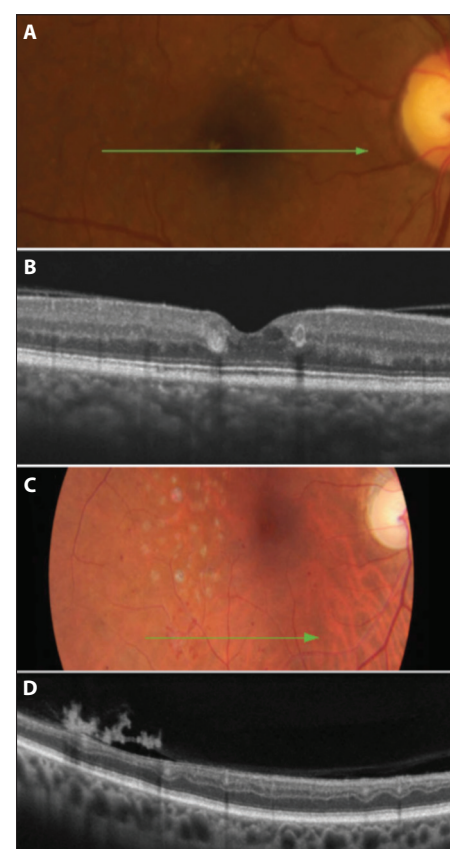
impairment in people of working age in the UK, along with hereditary retinal disorders.<sup>36</sup> Although the current diabetic retinopathy screening service only screens patients with fundus photography, OCT examination of diabetic patients can be incredibly beneficial in order to detect early signs of macular oedema and to clearly differentiate between exudates and drusen (see Figure 6A and 6B) or even confirm the presence of new vessels in proliferative disease (see Figure 6C and 6D). OCT examination of diabetic patients will allow earlier diagnosis and consequently result in prompt referral and treatment, thereby avoiding sight-threatening damage. Within the hospital eye service OCT is increasingly replacing fluorescein angiography as it provides a fast, non-invasive assessment of the level of macular oedema and allows easy monitoring of the effectiveness of treatment.<sup>37</sup>

While OCT is clearly not needed to locate microaneurysms and haemorrhages in diabetic retinopathy, it is useful to confirm the presence of exudates, which are a sign of chronic localised retinal oedema and which can be present in any stage of diabetic retinopathy. While on the surface, exudates may be mistaken for hard drusen, OCT is able to clearly differentiate between the two based on their differences in retinal location. While drusen form under the RPE, exudates are mainly located within the outer plexiform layer (see Figure 6A and B). Composed of lipoprotein and lipid-filled macrophages, exudates are highly reflective, appearing red on OCT imaging, and casting a shadow behind them. If exudates are noted within one disc diameter of the macula, the patient is classified as having diabetic maculopathy and referral is required for hospital follow up.<sup>37</sup>

### Conclusion

Never before has the optometrist had the

ability to detect disease before signs become visible at the ocular surface. Thanks to OCT, it is now possible to see what lies beneath the retinal surface. In the same way that fundus photography is now an accepted part of the routine eye examination, it is expected that OCT examination will soon become commonplace, allowing patients a higher level of care, and giving optometrists greater diagnostic abilities.



**Figure 6** (A) Fundus photograph of a diabetic patient showing exudates, which can be easily distinguished on (B) OCT examination. (C) Fundus photograph of an eye with proliferative retinopathy, with neovascularisation, which can be seen to lie on the retinal surface on OCT (D). (Green arrows on fundus photos indicate where the OCT B-scan is taken).

### MORE INFORMATION

**References** Visit [www.optometry.co.uk/clinical](http://www.optometry.co.uk/clinical), click on the article title and then on 'references' to download.

**Exam questions** Under the new enhanced CET rules of the GOC, MCQs for this exam appear online at [www.optometry.co.uk/cet/exams](http://www.optometry.co.uk/cet/exams).

Please complete online by midnight on May 23, 2014. You will be unable to submit exams after this date. Answers will be published on [www.optometry.co.uk/cet/exam-archive](http://www.optometry.co.uk/cet/exam-archive) and CET points will be uploaded to the GOC every two weeks. You will then need to log into your CET portfolio by clicking on 'MyGOC' on the GOC website ([www.optical.org](http://www.optical.org)) to confirm your points.

**Reflective learning** Having completed this CET exam, consider whether you feel more confident in your clinical skills – how will you change the way you practice? How will you use this information to improve your work for patient benefit?