Optical Biometry Editorial

I enjoyed the recent editorial regarding optical biometry.1 However, in it, it is stated, “Since the advent of the first commercial device in 2001...” In reality, we received the first IOLMaster 500 in the United States in December 1999, and it became commercially available in 2000.

It is also stated the importance of the ability of PCI “to measure AL more accurately to resolutions of approximately 0.02 mm compared with the 0.15 mm obtained with contact US. This is because the laser light source from PCI is reflected from the retinal pigment epithelial layer in contrast to the US waves, which are reflected from the internal limiting membrane.” This unfortunately is not correct. According to Haigis,2 the IOLMaster optical biometer made up for this retinal pigment epithelium (RPE) distance discrepancy by adjusting the axial length (AL) obtained by the instrument to match that obtained with immersion ultrasound (US). This was also used by all subsequent optical biometers, including those based on PCI, optical low-coherence reflectometry (OLCR), and swept-source optical coherence tomography. The reason for the 0.02 mm versus 0.15 mm AL difference between the IOLMaster and contact US is the unpredictable shortening caused by the contact method,3 which pushes on the cornea, thus shortening the eye. This is not the case with immersion US.

It is also mentioned, “Measurements with OLCR have been shown to be consistent and reproducible and to correlate well with those obtained with the PCI device.” However, when referencing this statement, the first JCRS paper on OLCR to prove this4 was not listed.

Kenneth J. Hoffer, MD
Santa Monica, California, USA

REFERENCES


Reply: Thank you, Dr. Hoffer, for your queries and concerns regarding my editorial in the July 2015 issue of JCRS. Let me try to address your concerns.

You had stated that my statement “Since the advent of the first commercial device [optical biometer] in 2001” was inaccurate and that you had the first device in December 1999 in your office. A literature review led me to believe the first commercial device was launched in 2001.1 I am not disputing that you might have had the first optical biometer in your office in 1999. Unfortunately, I was not privy to this information and I went by the information that I found during the literature review for the editorial. You said that my statement regarding the accuracy of PCI in measuring AL with reference to the RPE later was inaccurate. Although I agree with you that unpredictable corneal indentation during contact biometry might also contribute to the inaccuracy between contact US and optical biometry, my understanding from the literature2 is that laser light is reflected from the RPE, in contrast to US waves, which are reflected from the internal limiting membrane of retina. I am indeed aware that the PCI device makes up for this RPE distance by adjusting the AL to match that of immersion US; however, this might not be entirely true when comparing PCI with contact biometry (nonimmersion). You were also concerned that your manuscript on OLCR was not quoted in my editorial. As you might know, with an editorial article we try to keep the references to the bare minimum in the interest of space.

The reference on the OLCR I quoted was in fact published a year before your paper. My intention with the editorial reference was not to provide an extensive list of papers but to quote the earliest work. I am aware of your contribution to the literature on this subject.—Sathish Srinivasan, FRCSEd, FRCOphth, FACS

REFERENCES

Dexamethasone implant as an effective treatment option for macular edema in Irvine-Gass syndrome

I read with interest the article by Mayer et al.1 The authors studied a cohort of 23 patients diagnosed with pseudophakic cystoid macular edema (CME) and managed it successfully with an intravitreal dexamethasone implant. However, certain aspects of this interesting study need further evaluation.

In Figure 4, the authors compare visual gain to the duration of the CME. It appears as though the final visual acuity is independent of the duration of the CME, although the visual gain might be slow initially in longstanding cases. This might be explained by the fact that most of the patients with a disease duration of more than 6 months had a baseline macular thickness of more than 500 μm (Table 1). It would be interesting to know how baseline macular thickness correlated with the duration of the CME.

Overall, 9 patients required retreatment, mostly after 3 or 4 months of therapy (Table 2), when the effect of the implant starts declining. However, all the retreated patients were stable at 1 year from the start of the study, with a macular thickness below 300 μm in all 23 patients. Hence, unlike the vascular endothelial growth factor–dependent macular edema, where delayed onset treatment might lead to poor response to therapy,2 visual gain in pseudophakic CME appears to be independent of treatment delay. This reflects the favored hypothesis for the cause of Irvine-Gass syndrome being inflammatory cytokines and it being self-limiting usually, as also mentioned by the authors. To further strengthen this argument, I suggest a separate analysis for patients requiring retreatment, especially in terms of their baseline characteristics. I hope this discussion adds to the valuable results in the study.

Brijesh Takkar, MD
New Delhi, India

REFERENCES

Safety of sleep regulation after cataract surgery and complexity of the photoentrainment of circadian rhythm

This letter is in response to the review by Erichsen et al.1 that primarily details the effect of cataract surgery on sleep regulation by circadian rhythm. Its generalized title does not convey lack of findings on many other circadian rhythm–regulated effects on core body temperature, plasma level of cortisol, glucose tolerance, and so forth.

The interest in the effect of cataract surgery on the circadian rhythm was triggered by the discovery in 2002 of melatonin photopigment–containing intrinsically photosensitive retinal ganglion cells and their role in circadian rhythm. This led to search of its action spectrum, and a concern was raised for blue light–filtering intraocular lenses (IOLs) based on erroneous action spectrum,2 even though blue light–filtering IOLs clearly do not pass less blue light than any adult phakic IOL or the elderly cataractous lens. Computations in our 2009 publication2 with more rigorously derived action spectra removed this concern and concluded that both blue light–filtering IOLs and clear IOLs are expected to be effective for melatonin suppression under average household illumination. Three comparative sleep studies reported in the review1 as well as a recent double-masked block-randomized clinical trial measuring post-illumination pupil response,3 actigraphy and salivary melatonin measurements, found no difference between the 2 types of IOLs, thus validating our computations and conclusion.

One of the newer action spectra that we used was based on the combined role of intrinsically photosensitive retinal ganglion cells and input from middle-wavelength cones resulting in a broader action spectrum with a peak at 492 nm. Since our 2009 publication,4 further progress in understanding basic science shows a far more complex inner retina.4,5 In eyes without rods and cones, intrinsically photosensitive retinal ganglion cells alone can achieve...