ISSN: (Online) 2410-1516, (Print) 2413-3183

A review of higher order aberrations of the human eye

Authors:
Ayesha Suliman[1]
Alan Rubin[1]

Affiliations:
[1]Department of Optometry, University of Johannesburg, Doornfontein Campus, Johannesburg, South Africa

Corresponding author:
Ayesha Suliman,
ayesha@cybersmart.co.za

Dates:
Received: 06 Feb. 2019
Accepted: 15 May 2019
Published: 24 Oct. 2019

How to cite this article:
Suliman A, Rubin A. A review of higher order aberrations of the human eye. Afr Vision Eye Health. 2019;78(1), a501. https://doi.org/10.4102/aveh.v78i1.501

Background: This literature review is part of a research study for aberration-correcting soft contact lenses, where wavefront aberrometry was utilised.

Aim: This study was conducted as part of a postgraduate research degree by the first author with particular relevance to spherical aberrations in relation to myopia and soft contact lenses, both aberration control and non-control types.

Setting: This study reports on a literature review of higher order aberrations.

Methods: A comprehensive review of various databases was performed, including PubMed and Google Scholar in terms of aberration control contact lenses and particular customised contact lenses for compensation of spherical aberration in myopia, was performed.

Results: Wavefront sensing and Zernike polynomials are increasingly used in optometry and ophthalmology to quantify the wavefronts for an optical system such as the eye, using either lower order (LOA) or higher order aberrations (HOA). Although other mathematical methods are available, zero, 1st and 2nd orders of the Zernike polynomial expansion are LOA. Defocus (Z_2^0) and astigmatism (Z_2^{-2} and Z_2^2) are 2nd-order modes that usually can be corrected by clinicians using ordinary sphero-cylindrical compensations such as spectacle lenses. Until recently, only LOA were easily correctable by clinicians in optometry and ophthalmology. Higher order aberrations are those modes in the third radial order, $n = 3$ and higher, which in the past were not correctable. However, HOA contribute to only about 7% of retinal image quality and often go unnoticed by individuals, although in some instances, for example, with keratoconus or after refractive surgery, such aberrations can become more problematic. Today, new treatments are available via specially designed or customised (to an individual) rigid or soft contact lenses that are claimed to reduce or eliminate HOA such as spherical aberration (Z_4^0).

Conclusion: Although such specially designed or customised contact lenses have some effect on HOA, there are conflicting reports and so further investigation of this intriguing aspect remains necessary.

Keywords: higher order aberrations; lower order aberrations; aberrometry; wavefront; aberration control contact lenses.

Introduction

The optics of the human eye and the potential correction or compensation of its errors of refraction have been studied for at least the past 700 years. Prior to modern-day refractive surgery, the wavefront aberrations (WA) of the eye were sometimes considered as relatively insignificant when thinking about the optics and image processing of the eye. The reason for this is that some, particularly higher order, WA do not necessarily cause any noticeable degradation in visual acuity (VA) below 20/20 vision, generally considered as the criterion standard for a healthy eye. But with the development of laser and other refractive surgeries (including cataract-related ones), significant amounts of higher order spherical aberrations (*SA*), coma and trefoil were sometimes induced in treated eyes, thereby increasing the interest in higher order monochromatic aberrations such as *SA*.[1,2] Traditional laser refractive surgery left some patients complaining of poor image quality owing to changes in ocular aberrations and sometimes *SA* of the eye. Aberrometry is useful in post-operative evaluation of both lower (LOA) and higher order aberrations (HOA) where asymmetry, decentration and irregularity of the corneal surface can be well described by aberration terms or modes. Preoperatively, aberrometry is useful in guiding wavefront error modification, especially in naturally aberrated eyes.[3] Modern wavefront-guided refractive surgery is claimed to also reduce sphero-cylindrical errors without inducing excessive HOA, thus reducing patient dissatisfaction or complaints.[4,5] The possible role of *SA* in terms of myopia development, progression and prevention has also become an interesting concern both clinically and in research. Often with small pupil sizes such HOA went unnoticed, but in low-lighting conditions or in patients with moderate to severe myopia and/or larger pupils, the induced aberrations sometimes caused discomfort such as glare or intolerance to bright lights from oncoming traffic.

In most young populations, provided samples are large enough, HOA average out to almost zero (instances of positive *SA* in a sense neutralises those for negative *SA*), with minor mean amounts of positive *SA* ($0.14 \pm 0.1\ \mu$m) for a 6 mm pupil.[6,7,8,9] The positive spin-off to this small amount of *SA* is an increase in depth of field. In addition, in young, healthy eyes, corneal aberrations are almost totally compensated for by the internal aberrations of the eye.[10,11,12,13] Instruments such as the KR-1W Wavefront Aberrometer are available these days that will allow both corneal and internal aberrations to be determined relatively easily and effectively and these measurements can have clinical and other diagnostic benefits.[14] With age, fourth-order aberrations in a population were shown to increase and mostly be positive and notably different from zero.[2,10] But, changes in other LOA and HOA in relation to ageing are also important and can be affected by physiological processes such as presbyopia or by disease such as diabetes.

Analysis of LOA and HOA are also important both for pre- and post-refractive surgery to evaluate optical image quality and determine baseline values for comparative purposes; accordingly, aberrometry is commonly used today as an important clinical tool in ophthalmology to assess the outcomes of refractive surgery. But the methods are no less important in optometric practice, especially in relation to contact lens treatment for keratoconus or orthokeratology, proper diagnosis of unexplained vision fluctuations or ocular discomfort. Customised contact lenses (CL) and intra-ocular lenses (IOL) have also been developed to remedy HOA and their vision benefits are the more pronounced improvement in contrast sensitivity when compared with more conventional VA measures.[15]

Both LOA and HOA are utilised in the advancement of the design of ophthalmic, contact and IOL as well as other lenses such as those used in cameras.[11] To use a spectacle lens to reduce *SA*, the lens would have to have a specific pantoscopic tilt, facial wrap and vertex distance, which is usually difficult to achieve.[12] Simple spherical refractive errors (related to the LOA of defocus) have been corrected by spherical lenses from as early as the 17th century. But in principle it should be possible to correct HOA of an optical system by making adjustments to the optical paths of rays entering all points in the pupil. Cervino et al.[12] refer to Hershel as apparently the first scientist to suggest that an eye that suffered from aberrations could perhaps be corrected with a contact lens. Hershel proposed that a lens could be fitted that would have its posterior '*surface next to the eye*' with a spherical front surface to correct aberrations of the eye. It was Smirnov who also realised that spectacle lenses do not move when the eye moves, therefore proposing that CL could be the solution to wave aberration compensation.[12] With the advancement and increasing knowledge of CL in the 20th and 21st centuries, it was discovered that rigid lenses could negate irregular aberrations and small amounts of astigmatism of the anterior cornea.[1] Contact lenses also have numerous advantages in correcting HOA over refractive surgery, with the most important probably being the fact that corneal surface CL are a reversible or relatively easily modifiable option.[12] In recent times, rapid progress has been made in the investigation of *SA* and other HOA and the possibility of complete rectification of HOA.

Wavefront-guided ablations, aberration control contact lenses (ACCL) and aberration control IOL have become standard practice and WA can be measured, both before, during and after refractive surgery to hopefully provide an optimal outcome. They can also be used to optimise design and fit of soft or rigid CL to reduce LOA as well as HOA.[16]

Factors affecting higher order aberrations

This review will emphasise these factors:

- Anatomical factors
  - Tear film
  - Pupil
  - Cornea and crystalline lens
  - Retina
- Refractive error
- Accommodation
- Age
- ACCL

Anatomical factors

Anatomical factors that affect HOA include the tear film, pupil, cornea, crystalline lens and retina. The role of the tear layer is a very significant one as it is vital in guaranteeing the excellent quality of the anterior corneal surface and eventually that of the retinal image.[17] The tear film ensures a healthy and smooth corneal surface, thus minimising light scatter from the anterior corneal surface.[16] When the tear film breaks up, there is irregularity of the air-tear optical surface, resulting in additional aberrations of the optical system.[18] Larger and fluctuating optical aberrations are more commonly seen in dry eyes when compared with normal, healthy eyes and, owing to this irregularity, causes a profound alteration and often a decline in optical imaging quality.[18,19] Any abnormality in the tear film causes changes and overall, an increase in the optical aberrations of the eye, while the insertion of artificial tears causes a temporary change and decline in aberrations.[18] Stabilisation of the tear layer brings about regularity of the tears, thereby reducing short-term variation of aberrations and distortions of retinal imaging. In several studies, tear film characteristics have been shown to affect HOA[18,20,21] and, of course, also LOA.

Over the years, the possible relationships between pupil diameter and HOA (and LOA) have been investigated by several researchers. Pupil diameter is important in determining the size or amount of the central and/or peripheral blur in an out-of-focus image. In an eye that is either compensated or requires no spectacle-compensating lenses, VA is best with pupil diameters of 2 mm – 5 mm. For pupils smaller than 2 mm, diffraction will cause a decrease in retinal image quality and results in a decrease in light transmission, which adversely affects retinal image quality. However, smaller pupils result in an increase in depth of focus, which improves image quality. For pupils larger than 5 mm, *SA* may reduce VA. Charman[1] stated that for smaller pupils, diffraction was responsible for reduction in optical performance. However, as the diameter of the pupil increases above 2.5 mm, aberrations become responsible for degradation of the retinal image. During low light levels, cone-dominated vision is overtaken by rod-dominated vision. In such an instance, the quality of the ocular image may still be good but not better than a smaller pupil in light conditions.[22]

Therefore, with an increase in pupil diameter, LOA (but also HOA) have greater consequences.[23] Patients with large pupils in mesopic conditions may complain of glare, halos around lights, night-driving problems and poor contrast sensitivity. Zernike polynomial coefficients are dependent on pupil diameter; therefore, larger pupils generally mean greater magnitudes (moduli) for coefficients. Changes in wavefront aberrations in the outer parts of the larger or dilated pupil result in HOA, contributing to the total root mean square (*RMS*) aberrations of the dilated pupil.[1] Aberrations thus can be specified in terms of raw coefficients (having positive or negative values) directly or using *RMS* or moduli (absolute values) of coefficients, thus always positive in value. Paquin et al.[24] found most dilated myopic eyes presented with positive *SA* and other studies support the absolute relationship between pupil size and total *RMS*.[24]

Although both the cornea and crystalline lens affect HOA (and LOA) of the eye, almost 90% of HOA are the result of the cornea (or more truly tears) in normal eyes.[25] The anterior corneal surface has a stronger central power than the posterior corneal surface, approximately 48 dioptre (D) *versus* –6 D, thus about 43 D in terms of thin lens theory. This, together with the fact that it is easier to take measurements of the anterior corneal surface, makes the anterior cornea the focus of many studies. The crystalline lens contributes about one-third of the total power of the eye (between 21 D and 30 D), depending on the accommodative state of the eye. Studies of the human crystalline lens usually need to be conducted *in vitro*, also making it a difficult structure to study. The ocular lens also varies short-term during ocular accommodation and long-term with age. This, together with intra- and inter-subject variation and *in vitro* study, makes data collection for the crystalline lens a challenge.[17]

The most powerful refractive component of the eye is the front corneal surface and therefore a principal contributor to the WA of the eye.[26] Mrochen et al.[27] suggest that 2nd-order astigmatism and 3rd-order aberrations originate from the cornea, while 4th- to 6th-orders mostly do not arise from the anterior cornea. Complicating matters is that HOA on the corneal front surface are shown to be compensated by the internal structures in normal myopic eyes.[28,29]

As mentioned earlier, data about LOA and HOA of the eye, anterior cornea and internal optics of the eye are critical in understanding the physiological optics and retinal imaging of the eye.[10] Of course, the cornea is not the only refractive component of the eye, and therefore the total ocular aberrations are not equal to the corneal aberrations alone. Spherical aberration, for instance, is reliant on both the corneal and crystalline lens curvatures and shapes for the individual concerned.[2] Variability across individuals, even of a similar age, between wave aberrations of the cornea and crystalline lens is a common finding.[30] Internal aberrometry cannot be directly measured with an aberrometer and we attain the aberration data of the internal optics by negating the data obtained for the complete eye and that of the anterior cornea: $WA_{eye} = WA_{internal} + WA_{cornea}$.

The internal optics of the human eye would take into consideration the posterior corneal surface and both surfaces of the crystalline lens and the ocular media such as the aqueous. The anterior and posterior corneal surfaces are dissimilar in optical form. Therefore, to make sense of the WA optics of the eye, we theoretically detach the posterior corneal surface from the internal optics of the eye. The discrepancy between the refractive index of the cornea and aqueous is minimal; therefore, we deduce that the posterior cornea contributes only a minor proportion of total aberrations of the eye. Internal HOA are essentially produced then by the front and back surfaces of the crystalline lens and its gradient variation in refractive index.[14,30] In younger subjects, the positive corneal aberration (for example, for *SA*) is balanced out by the internal aberrations of the eye.[13] As we age, coma and *SA* are induced by changes in the cornea and crystalline lens, causing a shift in the balance, resulting in an increase in total ocular HOA.[28]

Although the shape of the corneal surface has the slightest change with age and accommodation, the shape of the crystalline lens deviates greatly with both accommodation and ageing.[31] For specific aberrations such as spherical aberration (*SA*), eventual retinal image quality does not depend on the cornea or lens separately, but depends on the combined effect of the two structures because *SA* induced by the cornea can be reduced by that of the crystalline lens. In young eyes, the lens and cornea have spherical aberration of opposite signs, thereby reducing the total *SA* of the whole system.[1] In a study by Wang et al.,[32] which was conducted to assess the aberrations of the cornea and complete eye, it was concluded that in some eyes, the aberrations of the cornea and lens were compensatory (subtractive), yet in other eyes they were additive.

Young crystalline lenses are normally transparent, but there is a reduction in the transparency of the lens with age, pathology, drugs and exposure to ultraviolet and infrared light. Cataract formation will cause an increase in *RMS* HOA of the eye.[32] Cataract is one of the main sources of scatter or misdirected light within the eye and, for example, Hashemi et al.[33] found that nuclear cataract caused an increase in *RMS* HOA. Maeda[34] agreed that after the age of 50 years, ocular aberrations become more prominent owing to an increase in lenticular HOA.

Retinal functioning and health are important factors in image reception and interpretation. If the optics of the eye are essentially good but the retinal photoreceptors are not functioning properly, or if the eye and photoreceptors are working smoothly but there is damage to the post-retinal neural pathway, there will be degradation in the retino-cortical image quality. Image quality is dependent on the optics of the eye, together with healthy functioning of the photoreceptor and neural pathway as well as the ability of the individual to express the perception.[4] Adding to the complexity is the phenomenon such as the Stiles-Crawford effect, stating that central light rays passing through the ocular pupil and reaching the photoreceptors are most effective compared with peripheral rays. The retinal image and its interpretation are also influenced by neural factors such as cone photoreceptor density, location of photoreceptors, visual memory and blur interpretation. Retinal and/or cortical factors could also limit VA[32] and aberrations of the eye and cone directionality also influence image interpretation or perception.[31] Initial stages of neural processes involved in the formation of the retinal image, when taken into account, are generally designed or organised in healthy organisms to improve the ability to produce satisfactory or good image quality.[32]

Refractive error

Refractive error is mostly caused by either the axial length of the eye being too long or too short or the refractive components of the eye being too strong or weak, causing retinal blur. Genetic and environmental factors also play a role in processes such as emmetropisation and in deviations such as the development of myopia or astigmatism that degrade retinal image quality.[33] Some studies on animals have shown that it is the sign of defocus (positive or negative), myopic or hyperopic blur, that adapts eye growth.[26,33] According to Tian et al.,[35] myopic growth is triggered by marked image degradation, while subtle degradation has no effect. Humans have the ability of distinguishing unconsciously between hyperopic and myopic defocus, which increases as aberrations increases.[36]

The sign of defocus is instrumental in triggering accommodation as well as regulating the growth of the eye in emmetropisation.[33] Thus, in the early stages of ocular development, the growth of an eye is guided by the vision of that eye and a blurred point spread function (PSF; basically the distribution of light over the retinal fovea and surrounds) of a specific type can possibly encourage the development of myopia.[37] This process is currently under intense research to try and facilitate new methods of reducing the increasing scourge and prevalence of myopia on a worldwide basis. The length of an eye is regulated by the focal length of its optics to facilitate the image of the object to fall in focus on the retina.[38] The *Wallman hypothesis* states that retinal activity and high image-contrast restrains eye growth but low image-contrast encourages growth. Emmetropisation in young children will cause growth of the eye to continue until high-contrast images stop it. Excessive accommodative lag and negative *SA* during near tasks produce a low-contrast retinal image, producing eye growth. Uncorrected myopia with positive *SA* leads to further myopic advancement because the eye is habitually overpowered. In the case of the eye having a negative *SA*, hyperopic blur could also cause myopic progression.[33] In a normal eye, positive *SA* prevents myopia progression.[39] Differences in central *versus* peripheral blur of retinal images are also believed to be a part of this adaptive system of the human and animal kingdoms.

Myopes typically are considered to have higher *RMS* WA than emmetropes,[40] but in contradiction to this, Hazel et al.[40] found that myopic eyes were not more aberrated than emmetropic eyes. According to Atchison,[41] a change in ocular refraction brings about a change in *SA* at a rate of about 0.007 μm per dioptre of myopia, and it is the anterior cornea that makes the largest contribution to the HOA of the eye in relation to the total HOA of the eye (*SA* being one of the most important components of the HOA of the normal eye). Anterior corneal aberration is higher than total ocular aberration, changing more gradually with an increase in myopia. As myopia increases, the vertex curvature of the cornea increases but the cornea remains unchanged in asphericity, suggesting that the elevated *SA* of the eye is caused by an increase in axial length of the eye.[42]

Myopes have increased negative *SA* during near tasks, causing a central hyperopic blur and a peripheral hyperopic defocus compared with emmetropes and hyperopes. This generates growth of the eye, resulting in myopia progression.[43] Thus, the use of CL or other methods to correct retinal hyperopic blur by reducing negative *SA* during near tasks could potentially play a role in reducing myopia progression in children. This could reduce the incidence and prevalence of high myopia in adults, which is a major concern currently. Slowing the progression of myopia is also vital as this can positively influence reducing the prevalence of possible complications (such as retinal tears, detachment and even myopic maculopathy) with moderate to severe myopia that might affect retinal image quality and life quality. Besides the detrimental effect on individuals, some of these complications can also have very significant societal and economic disadvantages.

Thibos et al.[44] concluded the following: Even though myopia is uncommon in young children, near work results in accommodative lag, which – in combination with negative

SA – causes eye growth. Furthermore, a high positive *SA* in the unaccommodating eye of a child would serve as a protector from myopic regression as too much accommodation is required for reversal to negative *SA*. Outdoor daytime activity for children will cause a reduction in pupil size (from improved general lighting conditions), a reduction in accommodation (during play rather than near-oriented activities) and an increase in retinal contrast, which will reduce eye growth. These researchers concluded that in our modern world, children are mostly starting school or pre-school at a younger age when they are more hyperopic, forcing them to be indoors more of the time to accommodate more, and they are often spending excessive hours viewing electronic gadgets such as cell phones or tablets, thus doing a much greater proportion of near-oriented activities than for previous generations. Accommodative lag combined with negative *SA* and possibly diminished natural light (with mainly sedentary activities) indoors reduces image-contrast, accommodative variability and encourages eye growth and myopia progression.

The vital question here is: Does WA cause myopia or is WA caused by myopia? The wavefront aberrations is an underlying factor in the development of myopia. The genetic nature of some types of myopia has always been known and a common risk factor is having both parents with myopia. Image degradation caused by irregularity of the cornea or lens may be inherited, although the effect of near work (environmental factor) and time and activities outdoors should also be considered. Also, an accommodating eye during near work has higher aberrations along with inferior image quality, causing myopia progression. We cannot say absolutely that WA is caused by myopia because most myopes have the same averaged aberrations as applies to emmetropes, but they do have more than for hyperopes.[39] But, because HOA and *SA* specifically causes degradation of the retinal image, it is said to be of importance in myopia advancement.[43] There are very complicated interactions between LOA and HOA and the operations or processes of the optical imaging system of the eye that have only recently begun to be studied in greater detail. Although some ideas are in place, there remains a lot to be understood. For example, corneal primary spherical aberration is positive for all refractive error groups (that is, myopes, hyperopes and emmetropes).[14] Carkeet et al.[45] and Paquin et al.[24] indicate that myopic eyes display higher aberrations than hyperopic eyes. He et al.[42] agreed and added that with an increase in refractive error, especially more than 6 D, there was an increase in *RMS* WA.

In contrast to this, Philip et al.[14] found that low myopes and emmetropes exhibited less total ocular HOA *RMS*, showed less total HOA *RMS* 4th-order aberrations and less *SA* than hyperopes (fifth and sixth-order aberrations were similar in both groups). In the same study, it was found that hyperopes exhibit lower negative *SA* with regard to internal aberrations. These studies explained that the lower negative lenticular *SA* in hyperopic eyes was due to lesser axial growth, resulting in the curvature of both lens surfaces to have smaller radii of curvatures. Philip et al.[14] concluded that hyperopic eyes have greater positive total *SA* than myopic or emmetropic eyes. Hashemi et al.[33] found that hyperopes have the highest levels of aberrations in an age group between 40 and 64 years, during which there is a hyperopic shift owing to lens changes, explaining the change in aberrations.

Ocular accommodation

If the eye has too much power, as in myopia, light rays will focus in front of the retina and the image will suffer a positive myopic blur. In hyperopia, rays would focus behind the retina (in its absence) and the retina suffers a negative hyperopic blur. A negative hyperopic blur results in an increase in power of the crystalline lens and conversely, a positive myopic blur will cause a drop in refractive power. The signs of defocus, blur, chromatic aberrations and proximity controls the accommodative mechanism of the eye.[32,42] Binocularly amplitude of accommodation is assumed to be greater than monocularly, owing to the support of convergence.[43]

Stimulus to accommodation is either blur, chromatic aberration or awareness of target proximity.[42] Visual sensing of the sign of defocus is provided by *SA*, and accommodation of the eye will reverse this sign.[32] It is inevitable that accommodation will bring about a change in *SA* as there is a change in shape and position of the crystalline lens.[22]

With increasing levels of accommodation, for example, with reading, the HOA (and *SA*) of the eyes alters significantly.[26] Our eyes generally present with a positive *SA* when in a relaxed accommodative state and exhibit stronger refracting power for marginal rays than paraxial ones.[32,44] As accommodation increases, *SA* goes from a positive to negative value, owing to lenticular changes.[1,32,45,46] There are also changes in 3rd-order aberrations[44] and *SA* and coma change as an eye accommodates.[8] Wavefront aberrations vary from an unaccommodated to an accommodated eye, and from subject to subject. Ocular accommodation plays a significant role in regulating WA within the eyes, thereby affecting the optical image quality.[30,44]

The effect of ocular accommodation on 3rd- and 4th-order aberrations differs from individual to individual.[45] The eye's short-term WA instability is because of fluctuations in accommodation or defocus. Dynamic variations in leads and lags in accommodation make it difficult to correct aberrations via simple approaches such as non-adapting spectacle or CL. The accommodative ability, with change in pupil size, varying thickness of tear film during blinking, diurnal changes in structures such as the tears and cornea all make diffraction-limited vision impossible. With changes in illumination comes a change in pupil size, accommodation and movement of the pupil centre by up to 0.4 mm.[47]

In the Zernike pyramid, the horizontal and vertical tilts (the first two odd [n = 1] order aberrations) are compensated for by eye rotation, making it tedious to gauge their role in accommodation. In López-Gil et al.[46] and Chin et al.,[47] it was concluded that odd-numbered, higher order aberration terms such as coma and trefoil play a relatively small role in accommodation control. Conversely, even-order terms like astigmatism and *SA* perform a more important role in accommodation control.

Increased and contrasting HOA have been seen in accommodative spasms when compared to normal accommodative processes, suggesting that wavefront analysis may be used in the determination of accommodative anomalies. In Ninomiya et al.,[48] eyes with accommodative spasms were shown to have negative *SA* values and resolving the spasm of accommodation with cycloplegic agents did not substantially change the *SA*.

Age

With a change in age, there are numerous changes to the crystalline lens of the eye, which include changes to the outer curvatures; gradient refractive indices; and centre, para-central and peripheral thicknesses of the lens with the addition of lens fibres throughout life. An increase in the thickness of lens fibres with age causes the aberrations of the crystalline lens to increase as well.[41]

There is a linear increase of HOA with age.[49] Age is also associated with pupil centre shift as well as a reduction in pupil size, which also influences most HOA coefficients. The effect of age on aberration was also investigated in the aforementioned study by Hartwig and Atchison,[50] where the introduction of a near addition affected all third-order aberrations, spherical aberration and secondary astigmatism. In Radhakrishnan and Charman,[51] it was found in pre-presbyopes that *SA* went from positive to negative with an increase in accommodation, but in the early presbyopes *SA* was the only HOA that became more positive during accommodation, which was welcomed as it increased the depth of focus and hence produced a better near-vision. Wang and Koch[32] reported a significant increase in the total HOA for third to sixth orders, including 4th-order *SA* coefficients, while there was only a slight increase in coma with an increase in age. Brunette et al.[52] analysed the effect of age on monochromatic aberration and found that *RMS* 3rd to 7th orders decreased until adulthood and then increased after the age of 40, suggesting that HOA may play a role in emmetropisation and presbyopic-related changes in the refractive state.

Total HOA also increase with the formation of cataract.[31] In Wang et al.,[10] the increase in 4th-order *SA* started only after the age of 50 years. They suggested in their study that the approach to treatment by the clinician should be varied as a function of age. In patients much younger than 50 years, refractive compensations should be based on current ocular wavefront measurements, but for patients nearing 50 years, it would be desirable to induce a more negative shift in *SA* to compensate for the imminent ageing changes.

Age-related miosis causes a decrease in *SA* of the eye[49] with compensation of corneal aberrations by the internal aberrations of the eye declining with age. As mentioned, age-related retinal disease and cataract also increase ocular aberrations.[50] Age-related neural changes, worsening of visual performance and decline in contrast sensitivity are all factors that cause an increase in *SA*.[4] In presbyopic patients, refractive surgery should be carefully thought through, as changes in aberrations in the eyes are still taking place.[51]

Aberration control contact lenses

The use of CL creates additional refractive surfaces (tears both in front and behind the contact lens, that itself has two surfaces) in front of the cornea, potentially increasing reflections and light scattering, thereby reducing the contrast of retinal images and subsequently visual performance. Scattering can have similar effects as some aberrations in terms of creating disturbances or distortions of visual performance,[53] which are important considerations when discussing the effects of CL on the eye and its aberrations.

The introduction of a contact lens to the eye can bring about dynamic and complicated changes in the wavefronts of light entering the eye, affecting LOA and possibly increasing or decreasing HOA, owing to factors including lens centration, movement, rotation and flexure and perhaps increasing the *SA* of the eye.[9,54,55] Even small decentration movements of soft CL produce HOA, which are significant and need to be accounted for in the design of soft CL. [9,63,64,65] For example, to correct *SA*, some manufacturers have incorporated aspheric optics into the design of soft CL.[9,64] True customised aberration-control CL are tailor-made, requiring specially designed lenses for each eye, which can be expensive and not readily available.[58] Alternatively, lenses are mass-produced with the identical generic *SA* correction for a population.[55] Mass-produced aberration-control CL (ACCL) assume that most subjects have a standard amount of aberration, which is untrue.[66]

Atchison[41] found that aspheric CLs eliminate myopic shift, but spherical soft CLs produce myopic shift. Ocular wavefront tomography (OWT) is a new technique used to improve the design of CLs to optimise peripheral optical quality of the eye and is expected to become important in the future in this regard.[66]

The constituent material and the type of contact lens (that is, whether rigid or soft) and its overall diameter in relation to the corneal diameter are other factors that are important in designing ACCL. Semi-sclerals and other CL that may be larger than the corneal diameter may interact differently with the eye with regard to its aberrations than, say, a rigid lens that has a diameter that is smaller than the corneal diameter; thus there are many factors that impact the ability of CL to influence the aberrations of a specific eye.

The clinical relevance of aspheric over non-aspheric CL remains controversial. Rather than trying to totally eliminate

HOA, a more realistic goal may be to reduce aberrations only in eyes where visual imaging is degraded below normal levels. The best correction of HOA will not take us beyond the levels of photopic acuity achieved by young, natural eyes. There is a scope for the use of aberrometers to distinguish those patients who have degraded visual performance by high levels of aberrations, in order to assist them to improve their vision. Although research is somewhat limited, there seems to be a place for positive *SA* CL in the prevention of myopia progression.

The first author's dissertation involves a detailed study of ACCL and their efforts on HOA, especially the 4th-order *RMS*, including *SA*.[67]

Ethical considerations

The ethical clearance to conduct this study was obtained from the Faculty of Health Sciences Ethical Committee at the University of Johannesburg (ethical clearance number: REC-241112-035).

Conclusion

Understanding WA (including HOA and especially *SA*) are important for many reasons, especially in dealing with patients' complaints and problems such as progressive myopia. Although the best correction of HOA may not take us beyond the levels of photopic acuity achieved by young, natural eyes, there is scope for the use of aberrometers to distinguish those patients who have degraded visual performance by high or different levels of aberrations, in order to assist them to improve their vision. Understanding both LOA and HOA may also be used to prevent myopia progression. The introduction of modern innovations in the exciting field of *adaptive optics* (AO) has seen the use of HOA involved in the composition of aberration control systems for the study of microscopic structures including retinal structures[62] and such developments may lead to *adaptive* (rapidly variable) spectacles or CL that may be useful in clinical situations such as myopia, presbyopia or keratoconus.

Acknowledgements

The authors thank Prof. Wayne Gillan, who assisted with ideas on the topic and initially supervised the dissertation before his retirement from the university.

This article is based on research towards a master's degree at the Department of Optometry, University of Johannesburg, by A.S. with the guidance from the second author (A.R.). The research is based on the dissertation 'The effect of conventional and aberration control soft contact lenses on 4th order aberrations, especially spherical aberrations of myopic eyes'.

Competing interests

The authors have declared that no competing interests exist.

Authors' contributions

A.S. wrote the research under the guidance and supervision of A.R.

Funding information

This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Data availaibility statement

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Disclaimer

The views and opinions expressed in this article are those of the authors and do not necessarily reflect the official policy or position of any affiliated agency of the authors.

References

1. Charman WN. Wavefront technology: Past, present and future. Contact Lens Anterior Eye. 2005;28(2):75–92. https://doi.org/10.1016/j.clae.2005.02.003
2. Porter J, Guiro A, Cox GI, Williams DR. Monochromatic aberrations of the human eye in a large population. Opt Soc Am. 2001;18(8):1793–1803. https://doi.org/10.1364/JOSAA.18.001793
3. Smolek MK. Method for expressing clinical and statistical significance of ocular and corneal wavefront error aberrations. Cornea. 2012;31(3):212–221. https://doi.org/10.1097/ICO.0b013e318221ce7d
4. Applegate RA. Glenn Fry Award Lecture 2002: Wavefront sensing, ideal corrections, and visual performance. Optom Vis Sci. 2004;81(3):167–177. https://doi.org/10.1097/00006324-200403000-00008
5. Pepose JS, Applegate RA. Making sense out of wavefront sensing. Am J Ophthalmol. 2005;139(2):335–343. https://doi.org/10.1016/j.ajo.2004.11.010
6. Bao J, Le R, Wu J, Shen Y, Lu F, He JC. Higher-order wavefront aberrations for populations of young emmetropes and myopes. J Optom. 2009;2(1):51–58. https://doi.org/10.3921/joptom.2009.51
7. Martin J, Vasudevan B, Himebaugh N, Bradley A, Thibos L. Unbiased estimation of refractive state of aberrated eyes. Vis Res. 2011;51:1932–1940. https://doi.org/10.1016/j.visres.2011.07.006
8. Lindskoog Petterson A, Martensson L, Salkic J, Unsbo P, Brautaset R. Spherical aberration in relation to visual performance in contact lens wear. Contact Lens Anterior Eye. 2011;34:12–16. https://doi.org/10.1016/j.clae.2010.08.008
9. McAlinden C, Moore JE, McGillan VE, Moore TCB. Spherical aberration and higher order aberrations with Bafilcon A (pure vision) and Comfilcon A (Biofinity). Graefes Arch Clin Exp Ophthalmol. 2011;249:607–612. https://doi.org/10.1007/s00417-010-1476-9
10. Wang L, Santaella RM, Booth M, Kock DD. Higher-order aberrations from the internal optics of the eye. J Cataract Refract Surg. 2005;31(8):1896–1903. https://doi.org/10.1016/j.jcrs.2004.01.048
11. Comastri SA, Martin G, Pfortner T. Analysis of pupil and corneal wave aberration data supplied by the SN CT 1000 Topography System. Optik. 2006;117:537–545. https://doi.org/10.1016/j.ijleo.2005.11.018
12. Cervino A, Hosking SL, Montes-Mico R, Bates K. Clinical ocular wavefront analyzers. J Refract Surg. 2007;23(6):603–615. https://doi.org/10.3928/1081-597X-20070601-12
13. Keir N, Simpson T, Fonn D. Visual and optical performance of silicon hydrogel contact lenses for moderate myopia. J Optom. 2010;3(3):149–157. https://doi.org/10.1016/S1888-4296(10)70021-2
14. Philip K, Martinez A, Ho A, et al. Total ocular, anterior corneal and lenticular higher order aberrations in hyperopic, myopic and emmetropic eyes. Vis Res. 2012;52:31–37. https://doi.org/10.1016/j.visres.2011.10.018
15. Legras R, Rouger H. Calculations and measurements of the visual benefit of correcting higher-order aberrations using adaptive optics technology. J Optom. 2008;1(1):22–29. https://doi.org/10.3921/joptom.2008.22
16. Lopez-Gil N, Castejon-Mochon JF, Fernandez-Sanchez V. Limitations of the ocular wavefront correction with contact lenses. Vis Res. 2009;49:1729–1737. https://doi.org/10.1016/j.visres.2009.04.016
17. Navarro N. The optical design of the human eye: A critical review. J Optom. 2009;2(1):3–18. https://doi.org/10.3921/joptom.2009.3
18. Lu N, Lin F, Huang Z, He Q, Han W. Changes of corneal wavefront aberrations in dry eye patients after treatment with artificial lubricant drops. J Ophthalmol. 2016;3:1–11. https://doi.org/10.1155/2016/1342056

19. Denoyer A, Rabut G, Baudouin C. Tear film aberration dynamics and vision-related quality of life in patients with dry eye disease. Ophthalmology. 2012;119(9): 1811–1818. https://doi.org/10.1016/j.ophtha.2012.03.004

20. Montés-Micó R, Alio' JL, Charman N. Dynamic changes in the tear film in dry eyes. Investig Ophthalmol Vis Sci. 2005;46(5):1615–1619. https://doi.org/10.1167/iovs.05-0017

21. Atchison DA. Wavefront aberrations and their clinical application. Clin Exp Optom. 2009;92(3):171–172. https://doi.org/10.1111/j.1444-0938.2009.00380.x

22. Charman WN. The Charles F. Prentice Award Lecture: Optics of the human eye: Progress and problems. Optom Vis Sci. 2006;83(6):335–345. https://doi.org/10.1097/01.opx.0000221389.36278.d0

23. Castejón-Mochón JF, Lopez-Gil N, Benito A, Artal P. Ocular wave-front aberration statistics in a normal young population. Vis Res. 2002;42(13):1611–1671. https://doi.org/10.1016/S0042-6989(02)00085-8

24. Paquin MP, Hamam H, Simonet P. Objective measurement of optical aberrations in myopic eyes. Optom Vis Sci. 2002;79(5):285–291. https://doi.org/10.1097/00006324-200205000-00007

25. Wei S, Song H, Tang X. Characterization of anterior corneal high-order wavefront aberrations and correlations with astigmatism in Chinese elderly subjects. Optik. 2016;127:3969–3975. https://doi.org/10.1016/j.ijleo.2016.01.044

26. Buehren T, Collins MJ, Carney L. Corneal aberrations and reading. Optom Vis Sci. 2003;80(2):159–166. https://doi.org/10.1097/00006324-200302000-00012

27. Mrochen M, Jankov M, Bueeler M, Seiler T. Correlation between corneal and total wavefront aberrations in myopic eyes. J Refract Surg. 2003;19:104–112. https://doi.org/10.1117/12.470593

28. Birkenfeld J, De Castro A, Oritz S, Pascual D, Marcos S. Contribution of the gradient refractive index and shape to the crystalline lens spherical aberration and astigmatism. Vis Res. 2013;86:27–34. https://doi.org/10.1016/j.visres.2013.04.004

29. Lopez-Miguel A, Martinez-Almeida L, Gonzalez-Garcia MJ, Coco-Martin MB, Sobrado-Calvo P, Maldonado MJ. Precision of higher-order aberration measurements with a new Placido-disk topographer and Hartmann-Shack wavefront sensor. J Cataract Refract Surg. 2013;39:242–249. https://doi.org/10.1016/j.jcrs.2012.08.061

30. Wang W, Wang Z-Q, Wang Y, Zuo T. Optical aberrations of the cornea and crystalline lens. Optik. 2006;117(9):399–404. https://doi.org/10.1016/j.ijleo.2005.10.009

31. Wang Y, Wang Z-Q, Guo H, Wang, Y. Wavefront aberrations in the accommodated human eye based on the individual eye model. Optik. 2007;118:271–277. https://doi.org/10.1016/j.ijleo.2006.03.019

32. Wang L, Koch DD. Ocular higher-order aberrations in individuals screened for refractive surgery. J Cataract Refract Surg. 2003;29:1896–1903. https://doi.org/10.1016/S0886-3350(03)00643-6

33. Hashemi H, Khabazkhoob M, Jafarzadehpur E, et al. Higher order aberrations in a normal adult population. J Curr Ophthalmol. 2015;27:115–124. https://doi.org/10.1016/j.joco.2015.11.002

34. Maeda N. Clinical applications of wavefront aberrometry – a review. Clinical and Experimental Ophthalmology, 2009;37:118–129. https://doi.org/10.1111/j.1442-9071.2009.02005.x

35. Tian Y, Tarrant J, Wildsoet CF. Optical and biometric characteristics of anisomyopia in human adults. Ophthalmic Physiol Opt. 2011;31:540–549. https://doi.org/10.1097/OPX.0000000000000040

36. Cooper J, Tkatchenko AV. A review of current concepts of the etiology and treatment of myopia. Eye Contact Lens. 2018;50(2):200–212. https://doi.org/10.1016/S0042-6989(00)00103-6

37. Ramamirtham R, Kee C, Hung LF, Qiao-Grider Y, Roorda A, Smith EL. Monochromatic ocular wave aberrations in young monkeys. Vis Res. 2006;46:3616–3633. https://doi.org/10.1016/j.visres.2006.04.006

38. Cervino A, Hosking SL, Ferrer-Blasco T, Montes-Mico R, Gonzales-Meijome JM. A pilot study on the differences in wavefront aberrations between two ethnic groups of young generally myopic subjects. Ophthalmic Physiol Opt. 2008;28:532–537. https://doi.org/10.1111/j.1475-1313.2008.00592.x

39. Wallman J, Winawer J. Homeostatis of eye growth and the question of myopia. Neuron. 2004;43:447–468. https://doi.org/10.1016/j.neuron.2004.08.008

40. Hazel CA, Cox MJ, Strang NC. Wavefront aberration and its relationship to the accommodative stimulus-response function in myopic subjects. Optom Vis Sci. 2003;80(2):151–158. https://doi.org/10.1097/00006324-200302000-00011

41. Atchison DA. Optical models for human myopic eyes. Vis Res. 2006;46:2236–2250. https://doi.org/10.1016/j.visres.2006.01.004

42. He JC, Sun P, Held R, Thorn F, Sun X, Gwiazda E. Wavefront aberrations in eyes of emmetropic and moderately myopic school children and young adults. Vis Res. 2002;42(8):1063–1070. https://doi.org/10.1016/S0042-6989(02)00035-4

43. Oberholzer M, Gillan WDH, Rubin A. Higher order aberrations of the eye: Part two. Afr Vis Eye Health. 2016;75(1):1–6. https://doi.org/10.4102/aveh.v75i1.335

44. Thibos LN, Bradley A, Liu T, Lopez-Gil N. Spherical aberration and the sign of defocus. Optometry Vis Sci. 2013;90(11):1–8. https://doi.org/10.3921/joptom.2008.71

45. Carkeet A, Luo HD, Tong L, Saw SM and Tan DTH. Refractive error and monochromatic aberrations in Singaporean children. Vis Res, 2002;42:1809–1824. https://doi.org/10.1016/S0042-6989(02)00114-1

46. López-Gil N, Rucker FJ, Stark LR, et al. Effects of third-order aberrations on dynamic accommodation. Vis Res, 2007;47:755–765. https://doi.org/10.1016/j.visres.2006.08.010

47. Chin SS, Hampson KM, Mallen EAH. Role of ocular aberrations in dynamic accommodation control. Clin Exp Optom. 2009;92(3):227–237. https://doi.org/ó10.1111/j.1444-0938.2009.00361.x

48. Ninomiya S, Fujikado T, Kuroda T, et al. Wavefront analysis in eyes with accommodative spasm. Am J Ophthalmol. 2003;136(6):1161–1163. https://doi.org/10.1016/S0002-9394(03)00585-3

49. Cheng X, Xu J, Chehab K, Exford J, Brennan N. Soft contact lenses with positive spherical aberration for myopia control. Optom Vis Sci. 2016;93(4):353–366. https://doi.org/10.1097/OPX.0000000000000773

50. Hartwig A, Atchison DA. Analysis of higher-order aberrations in a large clinical population. Invest Ophthalmol Vis Sci. 2012;53(12):7862–7870. https://doi.org/10.1167/iovs.12-10610

51. Radhakrishnan H, Charman N. Age-related changes in ocular aberrations with accommodation. J Vis, 2007;7(11):1–21. https://doi.org/10.1167/7.7.11

52. Borish IM. Clinical refraction. 3rd ed. New York: Professional Press Books, 1970; p. 174.

53. Atchison DA, Collins MJ, Wildsoet CF, Christensen J, Waterworth MD. Measurement of monocular ocular aberrations of human eyes as a function of accommodation by the Howland Aberroscope Technique. Vis Res. 1995;35(3):313–323. https://doi.org/10.1016/0042-6989(94)00139-D

54. Collins MJ, Wildsoet CF, Atchison DA. Monochromatic aberrations and myopia. Vis Res. 1995;35(9):1157–1163. https://doi.org/10.1016/0042-6989(94)00236-F

55. Buehren T, Collins MJ. Accommodation stimulus-response function and retinal image quality. Vis Res. 2006;46:1633–1645. https://doi.org/10.1016/j.visres.2005.06.009

56. Atchison DA. Recent advances in measurements of monochromatic aberrations of human eyes. Clin Exp Optom. 2005;88(1):5–27. https://doi.org/10.1111/j.1444-0938.2005.tb06659.x

57. Brunette I, Bueno JM, Parent M, Hamam H, Simonet P. Monochromatic aberrations as a function of age, from childhood to advanced age. Investig Ophthalmol Vis Sci. 2003;44:5438–5446. https://doi.org/10.1167/iovs.02-1042

58. Fujikado T, Kuroda T, Ninomiya S, et al. Age-related changes in ocular and corneal aberrations. Am J Ophthalmol. 2004;138(1):143–146. https://doi.org/10.1016/j.ajo.2004.01.051

59. Jiang H, Wang D, Yang L, Xie P, He JC. A comparison of wavefront aberrations in eyes wearing different types of soft contact lenses. Optom Vis Sci. 2006;83(10):769–774. https://doi.org/10.1097/01.opx.0000236786.96023.9c

60. Legras R, Rouger H. Calculations and measurements of the visual benefit of correcting higher-order aberrations using adaptive optics technology. J Optom. 2008;1(1):22–29. https://doi.org/10.3921/joptom.2008.22

61. Lu F, Mao X, Qu J, Xu D, He JC. Monochromatic wavefront aberrations in the human eye with contact lenses. Optom Vis Sci. 2003;80(2):135–141. https://doi.org/10.1097/00006324-200302000-00009

62. Roberts B, Athappilly G, Naikoo H, Asbell P. Higher order aberrations induced by soft contact lenses in normal eyes with myopia. Eye Contact Lens. 2006;32:138–142. https://doi.org/10.1097/01.icl.0000195570.73454.a5

63. Efron S, Efron N, Morgan PB. Optical and visual performance of aspheric soft contact lenses. Optom Vis Sci. 2008;85(3):201–210. https://doi.org/10.1097/OPX.0b013e318165100a

64. Lindskoog Petterson A, Jarko C, Alvin A, Unsbo P, Brautaset R. Spherical aberration in contact lens wear. Contact Lens & Anterior Eye. 2008;31:189–193. https://doi.org/10.1016/j.clae.2008.05.005

65. Wei X, Thibos L. Designing contact lenses for a wide field of view via ocular wavefront tomography. J Optom. 2010;3(3):125–133. https://doi.org/10.1016/S1888-4296(10)70018-2

66. Suliman A. The effect of conventional and aberration control contact lenses on fourth order aberrations, especially spherical aberrations on myopic eyes. Master's Dissertation in the Department of Optometry. Johannesburg, South Africa: University of Johannesburg; 2018.

67. Marcos S, Burns SA. On the symmetry between eyes of wavefront aberration and cone directionality. Vis Res. 2000;40:2437–2447. https://doi.org/10.1016/S0042-6989(00)00103-6