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### LETTER

Response to: WaveLight<sup>®</sup> Contoura topographyguided planning: contribution of anterior corneal higher-order aberrations and posterior corneal astigmatism to manifest refractive astigmatism

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## **Dear editor**

I would like to thank Wallerstein et al<sup>1</sup> for his thoughtful research letter, but I do have some issue with the analysis. The paper references only Part 1 of the LYRA Protocol series, and not the other two parts. Part 3 provides the data for treatment with Contoura-measured astigmatism using the LYRA Protocol (termed anterior corneal astigmatism [ACA]). The concept of anterior corneal higher-order aberrations (CHOA) modifying the manifest refractive astigmatism (RA) was illustrated in an ovalization form to demonstrate why the LYRA Protocol works. These interactions are three-dimensional (3D) in nature, and we are demonstrating with 2D CHOA maps from Contoura. These maps are impacted by user/technical error, and also by epithelial compensation of the CHOA, adding a layer of inaccuracy in these comparisons. In fact, the concept of epithelial compensation affecting the ability to measure and treat CHOA is not part of any current refractive system, Contoura, Ray tracing, etc. The static Gullstrand model has mainly been used, ignoring the fact that epithelial compensation causes variability.

Prior studies looking at posterior corneal astigmatism (PCA) did not fully consider that ACA could be modified by CHOA. If their findings were correct, then we simply could not achieve the level of accuracy that we did in Part 3 of the LYRA Protocol, nor in 2.5 years of performing Contoura with LYRA Protocol on primary eyes in our clinic. If 9% of eyes had -0.5 D of astigmatism caused by PCA, then at least 9% of eyes would have residual astigmatism immediately after treatment with LYRA Protocol, and that is not the case. If anterior/posterior lenticular astigmatism, lens decentration, and retinal astigmatism were also clinically significant, an even higher number of patients have incorrect outcomes post-Contoura with LYRA Protocol. These patients would have residual RA, but no ACA on Contoura processing, and have astigmatism present on Wavefront analysis. We have documented two types of cases where residual RA is present after laser correction utilizing Contoura with LYRA Protocol- residual RA immediately post-op (very uncommon), and change in clinical refraction from plano to RA over 3-6 months. In virtually all of these cases measurable ACA has been found. This was confirmed by using Wavefront analysis to look for astigmatism sources other than ACA. For this reason, a study is currently being conducted to examine

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Clinical Ophthalmology 2018:12 2001-2004 Commercial use of this work, is published and licensed by Dowe Medical Press Limited. The full terms of this license are available at https://www.dovepress.com/terms.php and incorporate the Crative Commons Attribution — Non Commercial (unported, v3.0) License (http://crativecommons.org/license/by-nd/3.0). By accessing the work you hereby accept the Terms. Non-commercial uses of the work are permitted without any further permission from Dove Medical Press Limited, provided the work is properly attributed. For permission for commercial use of this work, please see paragraphs 4.2 and 5 of our Terms (https://www.dovepress.com/terms.php). epithelial thickness compensation of CHOA as the reason for the uncommon eyes that have significant RA after ACA treatment, or for the 6%–7% of eyes that develop RA over 3–6 months postop.

Finally, it is important to note that PCA is usually antagonistic to ACA. PCA is against the rule in most eyes, and ACA is with the rule in most eyes; therefore, it is not additive as is stated by Wallerstein et al.<sup>1</sup>

The reason the ovalization theory was initially postulated was due to the outcome data not being explained by prior theories on the source of ocular residual astigmatism. The outcome data overwhelmingly shows that ocular astigmatism is anterior corneal in origin. The most important part of any theory is that it explains the observed outcome data.<sup>2,3</sup>

# Disclosure

The author reports no conflicts of interest in this communication.

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## Authors' reply Avi Wallerstein<sup>1,2</sup> Mathieu Gauvin<sup>2</sup> Mark Cohen<sup>2,3</sup>

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# **Dear editor**

Dr Motwani states that the purpose of his paper is to "demonstrate how HOAs can cancel out modify or induce lower order corneal astigmatism."1 He hypothesizes that anterior corneal higher-order aberrations (CHOAs) and anterior corneal astigmatism (ACA) interact, and for illustrative purposes he describes them as creating 2D ovalizations in the central cornea, "depending on how the two ovals of higher-order and lower order line up, it will either increase or decrease the manifest measurement of astigmatism."<sup>1</sup> He acknowledges that his simplification is "attempting to break down complex three-dimensional interactions between HOA and lower order astigmatism."1 He mentions the possibility of specifically quantifying the axes of the ovals and "creating a vector diagram that can accurately predict the axis."1 We did precisely that.<sup>2</sup> To objectively investigate his theory based on approximations, we developed an advanced algorithm that analyzed the Contoura ablation profile in 3D, accurately detailing the CHOAs ellipse.<sup>2</sup> We then studied the vectorial relationship between the calculated CHOAs and ACA ellipses. Once mathematically quantified, our data shows that his notion of ovals interacting does not hold true for 50% of eyes.<sup>2</sup> He also gives an example of a case that is not explained by his theory. Dr Motwani's response that CHOAs are undermeasured because of epithelial compensation is another interesting topic to explore but cannot explain our empirical analysis that does not support his theory.<sup>1</sup> His logic that the ovalization hypothesis is valid and that ocular residual astigmatism (ORA) is caused mainly by CHOAs because treating on the ACA has "not worsened patients' vision" – is not scientific.<sup>1</sup> Furthermore, his Part 3 paper (which is not the subject of our study) only presents 50 eyes, no control group, with a small mean RA to ACA magnitude discrepancy of 0.55 D (not high ORA), and does not include the standard reporting refractive surgery outcome graphs, nor astigmatism vector analyses. One cannot make definitive

conclusions of accuracy and "astigmatism elimination,"<sup>1</sup> nor make a recommendation for treating solely the ACA, especially in high ORA cases, based on such preliminary data and incomplete analysis.

Dr Motwani claims that posterior corneal astigmatism (PCA) does not contribute significantly to RA.<sup>1</sup> That claim negates many previous studies that are not referenced in his paper,<sup>3–5</sup> and his observations from clinical practice that few ACA treated eyes have postoperative astigmatism has not been substantiated with rigorous outcomes data. Dr Motwani misunderstood our term "additive" as meaning PCA increases RA, but it refers to vectoral addition in which the cornea is subtractive most of the time. Published data shows that 87% of posterior corneas produce against-the-rule (ATR) refractive astigmatism by acting as a negative lens.<sup>4</sup> This finding, with some contribution from CHOAs and cerebral preference for with-the-rule (WTR), likely contributes to the decreased subjective RA seen in most WTR corneas, and the increased subjective RA in most ATR corneas, which we described in 5,403 eyes.<sup>2</sup> PCA magnitude and its impact is frequently small, but in certain cases it can be significant and therefore cannot just be ignored. There is a complex interplay of compensatory mechanisms optimizing subjective vision. Postoperatively, these include biomechanical corneal shifting, epithelial remodeling, internal compensation of induced CHOAs, and as Dr Motwani describes, cerebral processing. Rigorous studies of thousands of eyes are needed to truly understand the interaction of CHOAs, ACA, PCA, and the effect of a combined CHOA and lower-order astigmatism excimer ablation. We thank Dr Motwani for his comments and contribution to the discussion surrounding this important topic.

# Disclosure

The authors report no conflicts of interest in this communication.

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