Reply: We appreciate the opportunity to respond to the comments and queries by Masket and Fram and Hong et al. We are disappointed and saddened that they feel our ray-tracing methods are flawed and contradict previously published findings and clinical observations. Our work considered every article, case report, presentation, clinical observation, UBM, visual field, patient drawing, and slitlamp photograph that had been published or presented in English, Portuguese, French, and Spanish in the past 12 years, which were used to explain in fine detail every subtle feature and nuance of negative dysphotopsia (ND) from a type 3 shadow in our "treatise." We are extremely grateful to all these individuals for providing this information and making our work possible.

The comment by Hong et al. that the type 2 shadow would be outside the normal visual field for the nominal location (0.5 mm) behind the iris is true, as we state in the text and demonstrate in Figures 3, 4, and 6. We also state that the type 2 shadow (not ND) would be seen in the visual field only if the IOL were 2.3 mm or more behind the iris and this extreme depth would be very apparent to a clinician. Hence the type 2 shadow is not ND.

In addition to the preponderance of acrylic IOLs, our article provided an optical explanation for the higher incidence of ND with acrylic IOLs and that was supported by our ray tracing in the text and Figure 13. In Figure 13, the lower panels (*C* and *D*), the retinal field angle is on the y-axis and the axial space behind the iris (pupil) is on the *x*-axis. Due to the higher index of refraction of acrylic, the region in which ND can occur clinically (the shaded area) is 4 to 5 times larger, which would be directly related to the incidence of ND. Of note, in their UBM study, Vámosi et al.[1] reported a range of 0.00 mm to 1.10 mm in their control group for this distance. The shadow for an acrylic IOL can be formed 0.06 to 1.23 mm behind the iris, while a silicone IOL can form a shadow only when located 0.06 to 0.62 mm behind the iris. The nominal space for both is approximately 0.5 mm, but the shadow width of acrylic is 14.0 degrees wide and of silicone, only 2.3 degrees. In addition, the posterior border of the silicone shadow is 7.5 degrees more anterior. The more anterior the shadow, the less likely it will fall on functional nasal retina. To reiterate, ND is possible with silicone, but the probability would be much lower due to the 6 times smaller width of the shadow and because the shadow is more anterior and therefore less likely to fall on functional retina. Our ray tracing provides unequivocal optical data showing ND would be much more common with acrylic than silicone, even when adjusted for usage.

We agree that the space behind the iris has been reduced from 0.58 to 0.31 mm on 1 side and 0.66 to 0.28 mm on the other side in the preoperative and postoperative UBMs provided by Masket and Fram. As shown in our Figure 13, however, when the postoperative space behind the iris exceeds 0.06 mm, ND is still possible and is the reason for the failure for iris suture fixation to fully alleviate the symptoms.

Regarding Hong et al.'s concern about the pupil sizes used in our article: We chose a 2.5 mm pupil for photopic conditions because it matches the pupil size outside or in a photopically illuminated examination room (overhead lights on) with a penlight. The mean pupil diameter in elderly and pseudophakic eyes under photopic conditions has been reported to be 3.2 to 3.8 mm.[2–6] These are apparent pupil sizes as seen by the clinician, which are magnified by 14% due to the converging power of the cornea. When one accounts for the difference in the apparent pupil size and the actual pupil size within the eye, the photopic pupil would be from 2.8 to 3.4 mm. Since ND is exaggerated with a penlight, the pupil would be even smaller, so we chose the 2.5 mm for the photopic pupil with a penlight. We chose a 5.0 mm pupil diameter to match the pupil size for evaluating the reports of disappearance of ND with pharmacologic dilation, which would normally be even larger. As we stated in the article, the sharpness of the shadow would gradually decrease with increasing pupil sizes above 2.5 mm and be unrecognizable by 5.0 mm, as shown in Figures 6 and 7 of our study.

Regarding the comment that our "theory would not explain that ND has never been reported with anterior chamber IOLs (AC IOLs) or sulcus-placed IOLs" is not true and highlights a fundamental concept underlying how ND occurs. Negative dysphotopsia cannot occur with AC IOLs because they cannot be 0.06 mm behind the iris (Figure 13). Regarding the sulcus-placed IOLs, it is possible to produce ND because they can be 0.06 mm behind the iris and still be in the sulcus. Masket and Fram[7] confirm this observation in their article in cases 4 and 5, in which 3-piece silicone IOLs were placed in the sulcus with powers of 0.0 diopters (D) and +4.0 D and the ND "was improved" but not eliminated. The improvement was a result of the nominal type 3 shadow for acrylic in the bag (0.5 mm behind iris) having a retinal field angle of 86 to 100 degrees (14 degrees wide), whereas the silicone in the sulcus (0.06 mm behind iris) is 92 to 101 degrees (9 degrees wide). The new shadow is two-thirds the width and 6 degrees more anterior. The extremely low front-surface powers of the secondary piggyback IOLs (0 and +4.0 D total power) were also important to

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allow the pencil of peripheral rays to be incident on the nasal edge and nasal posterior surface of the IOL. For a single primary sulcus IOL, this low front-surface power would be even more improbable, but ND is still possible.

It is much more likely for the internal anatomy of the eye to be a contributing factor when the border of functional nasal retina is located more anteriorly than average. The location of the anterior border of functional nasal retina has a standard deviation of 0.8 mm for this border,[8,9] so patients complaining of ND after their primary surgery are likely to be among those with a more anterior border. Exchanging the posterior chamber IOL for an AC IOL or using a fully (not partially) rounded-edge IOL are the only 2 treatments that are sure to eliminate ND. Exchanges for a silicone material, secondary piggyback IOLs, and reverse optic capture usually will "improve" the symptoms but cannot guarantee elimination of ND.

As we stated in the section on "Additional Influences," the temporal clear corneal incision has nothing to do with ND, transient or permanent.[10,11] It is not conjugate with the peripheral retina, so it cannot be seen by the patient.

Masket and Fram's comment that they have observed ND despite opacification (actually translucification, since it is not opaque) of the anterior capsule overlying the IOL further supports our model. Opacification of the anterior capsule overlying the IOL would not affect the type 3 shadow because the light rays would undergo Lambertian scatter and be reflected or refracted and then pass through the front surface of the IOL, creating an alternative path for incident light to reach the truncated sharp posterior nasal edge of the IOL, creating or enhancing the type 3 shadow. In fact, it is possible that this may be the only path and removing a sector of opacified nasal anterior capsule overlying the IOL might eliminate the ND by removing this path of scattered light. It is the portion of the anterior and posterior capsule peripheral to the edge of the IOL that scatters light into the shadow and eliminates ND. The increased incidence of peripheral capsule opacification over time explains the complete resolution in the majority of cases. We also mentioned an alternative possibility that forward movement over time due to capsular bag contraction, could lead to resolution of ND by moving the shadow anterior to the border of functional retina, but that this would be associated with a myopic shift due to increased lens effectivity.

Neural adaptation can mitigate and reduce symptoms but not eliminate them. Just like halos with multifocal IOLs diminish with time but never disappear. When specifically questioned the patients with multifocal IOLs can always see the halo. If the ND shadow is still present and not eliminated by light scatter from the capsular fornix peripheral to the IOL, the patient would still comment that they could see the dark arcuate temporal shadow, but it is no longer noticeable or annoying.

Masket and Fram's comment that our article provides no correlation with clinical findings is certainly demonstrably inaccurate. We have correlated every clinical manifestation, observation, and finding related to true ND. To state that we do not think ND is multifactorial misreads the article and ignores the explanation provided. Our ray-tracing diagrams and Table 1 detail all the variables that contribute to ND. We explain that the temporal field must be fully illuminated, the temporal cornea must refract the rays from 10 to 17 degrees, these rays must pass through a small pupil, strike the front surface of the IOL with a specific front curvature that directs the rays to the nasal edge and posterior nasal surface of the optic, pass through a clear peripheral anterior/posterior capsule and finally fall posterior to the anterior border of functional nasal retina. In our original manuscript, we called this multifactorial combination the "perfect storm," but the reviewers thought this was too colloquial. Negative dysphotopsia is most certainly multifactorial. The necessary and critical missing link before the mid-1990s was the truncated sharp edge.

Regarding the ray-tracing model used in the Hong et al. article,[12] we reference the comments made on the last page of our article. As we explained, our main objection to their approach is not with the human eye model, but the point source object at 75 degrees used for the simulation. Positive dysphotopsias are best simulated by illuminated objects in a dark surrounding and NDs by the presence of dark shadows in an illuminated surrounding. From looking at the pictures drawn by patients in our Figure 14, it is clear that with ND the entire temporal field (in fact the entire field) is illuminated; no one drew a point source in a dark surrounding. In fact, Osher[10] demonstrated in his Figure 3 that "shielding the eye with hand [over the temporal field] eliminates temporal shadow," illustrating that ND cannot be perceived if the temporal field is dark. Negative dysphotopsia occurs in photopic conditions, in an illuminated examination room, or outside. If one were to place a patient with ND in a dark room and hold a penlight at 75 degrees, the patient would not perceive a shadow because the dark band or intensity gap is not visible against a black background. Furthermore, if the temporal field were fully illuminated in their simulation, the intensity gaps (areas of darkness between the lighted secondary images) would be filled with light,

whether higher-order aberrations were present or not (Figure 1, *B*, in the Hong et al. letter).

From the new figures in the Hong et al. letter, it can be seen that the primary and secondary images as well as the intensity gaps would move with the object and not be fixed in the extreme temporal field, but movement of the temporal shadow has never been reported by any clinician or patient. They are fixed in the extreme temporal field as in our ray-tracing simulation. Also, the positive dysphotopic images that Hong et al. provided would be depicted in patient drawings that would look like their Figures with primary and secondary images, spread out like coma, with dark spaces between. Patients would not simply draw the dark spaces and omit the light areas. Finally, portions of their images are at intensities 10^{10} (10 billion, 10^{3} to 10^{-7}) times less intense than the primary image, making them unperceivable. For reference, our sun appears 1.4×10^{10} times brighter than Sirius, the brightest star in our celestial sphere.[13] Even without earth's atmosphere, Sirius would not be visible if the sun were at 75 degrees in our peripheral vision.

We appreciate the thoughtful comments provided in response to our article. We have carefully responded to every comment and criticism raised by the letters, not only to satisfy their concerns but also to amplify concepts that are critical to understanding ND for all readers. A more detailed version of this response is also available at http://jcrsjournal.org. We believe our ray-tracing methods have fully explained all the clinical manifestations, subtleties, and nuances of what has been called negative dysphotopsia. The type 3 penumbra is no longer an enigma.—*Jack T. Holladay, MD, MSEE*

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Reply: We appreciate the opportunity to respond to the comments and queries by Masket and Fram and Hong et al. We are disappointed and saddened that they feel our ray-tracing methods are flawed and contradict previously published findings and clinical observations. Our work[1] considered every article, case report, presentation, clinical observation, UBM, visual field, patient drawing, and slitlamp photograph that had been published or presented for the past 12 years in English, Portuguese, French, and Spanish which were used to explain in fine detail every subtle feature and nuance of negative dysphotopsia (ND) from a type 3 shadow in our "treatise." We are extremely grateful to all of these individuals for providing this information and making our work possible.

Davison's original conclusions as an observant clinician and investigator preceded our scientific proof by over a decade.[2] He recognized that the critical change in IOL design when ND began to emerge was a result of the truncated sharp edge and that a higher index of refraction material increased the incidence. His conclusions in 2000 are identical to our ray-tracing findings in 2012.

The stimulus that led to the truncated sharp edge becoming the predominant design of posterior chamber IOLs (PC IOLs) implanted in the United States was Nishi et al.'s pioneering work[3,4] that unequivocally confirmed that the sharp posterior edge was instrumental in preventing posterior capsule opacification and that the material had little or no effect.[3] As manufacturers adopted the truncated sharp edge design, reports of positive dysphotopsias became more common and it was postulated by many observant surgeons that these phenomena were likely due to the truncated sharp edge and higher index of refraction material. These clinicians were correct. Addressing these observations, we published our ray-tracing results demonstrating why patients saw a reflected arc in the opposite field from a peripheral light source (such as an automobile head light) which appeared brightest and was most noticeable at an angle of 35 degrees (range from 29 to 41 degrees) and demonstrated how this was due to internal reflection of the light from the truncated sharp edge and higher index of refraction IOL material.[5] Of note, Masket et al.[6] had made the same observation in their laboratory experiment using a laser and an ovoid IOL (with a truncated sharp edge) 6 years earlier.

Although some cataract surgeons had noted reports from their patients of a shadow temporally (ND), symptoms were rare and disappeared with time in the majority of patients, so ND was rarely discussed. It was not until Davison's original article[2] that heightened awareness of ND was universally recognized and reports flourished. Narvaez et al.,[7] Trattler et al.,[8] Osher,[9] Vámosi et al.,[10] Mamalis,[11] Cooke,[12] Wei et al.,[13] and Masket[14] all followed Davison,[2] suggesting the same mechanism of the truncated sharp edge as the cause. Our work provides the optical explanation for ND and proves their suspicions were correct.

Kevin Miller[15] was the first to provide an accurate illustration of a shadow, but Miller's figure depicts a type 1 shadow, the result of the internal reflection from the truncated sharp edge that had been documented using Goldmann visual fields by Osher[9] (Osher's Figure 6) and using Humphrey visual fields by Marques and Marques[16] (their Figure 2). These shadows correspond to the visual field angles from 29 degrees to 41 degrees in our article on positive dysphotopsia.[5] In contrast, the type 3 shadow (ND) that forms nominally from retinal visual field angles between 86 degrees and 100 degrees is much further in the periphery than the type 1 shadow. Also, Miller[15] suggested using pilocarpine as a treatment, which does reduce the type 1 shadow, but actually enhances the contrast of the type 3 shadow (ND). Miller also provided the accurate clinical observation that the [fully] "rounded edge completely solves the problem," 7 years before our article.

The comment by Hong et al. that the type 2 shadow would be outside the normal visual field for the nominal location (0.5 mm) behind the iris is true, as we state in the text and demonstrate in Figures 3, 4, and 6. We also state that the type 2 shadow (not ND) would only be seen in the visual field if the IOL were 2.3 mm or more behind the iris and this extreme depth would be very apparent to a clinician. Hence the type 2 shadow is not ND.

The criticism by Masket and Fram that we said ND is more likely with acrylic than silicone and that we offer no data or reference for this statement and that the higher incidence is due to the higher usage of acrylic is not true. Our actual statement was "[n]egative dysphotopsia is possible with silicone; however, the probability would be much lower because of the smaller [width] and more anterior location of shadow on the retina, as well as the reduced range of distances behind the iris." There is no question that acrylic IOLs were used more frequently than silicone or poly(methyl methacrylate) around 2000. According to Market Scope in the third quarter of 2001, acrylic was 52.2%, silicone 40.5%, and "other" was 7.3%. Davison[2] was an early adopter of acrylic and by 2000, silicone IOLs were implanted only 7% of the time when he reported that all 14 of the 6668 cases were acrylic. In the 2 eyes that were exchanged with silicone IOLs, the ND disappeared and this finding led Davison to the second conclusion that a higher index of refraction material must also be a factor.

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In addition to the preponderance of acrylic IOLs, our article provided an optical explanation for the higher incidence of ND with acrylic IOLs, and that was supported in the text and by our ray tracing in Figure 13. In Figure 13, in the lower panels (*C* and *D*), the retinal field angle is on the *y*-axis and the axial space behind the iris (pupil) on the *x*-axis. Due to the higher index of refraction of acrylic, the region in which ND can occur clinically (the shaded area) is 4 to 5 times larger, which would be directly related to the incidence of ND. Of note, in his UBM study,[10] Vámosi et al. reported a range of 0.00 to 1.10 mm in his control group for this distance. The shadow for an acrylic IOL can be formed 0.06 to 1.23 mm behind the iris, while a silicone IOL can form a shadow only when located 0.06 to 0.62 mm behind the iris. The nominal space for both is approximately 0.5 mm, but the shadow width of acrylic is 14.0 degrees wide and of silicone, only 2.3 degrees. In addition, the posterior border of the silicone shadow is 7.5 degrees more anterior. The more anterior the shadow, the less likely it will fall on functional nasal retina. To reiterate, ND is possible with silicone, but the probability would be much lower due to the 6 times smaller width of the shadow and because the shadow is more anterior and therefore less likely to fall on functional retina. Our ray tracing provides unequivocal optical data showing ND would be much more common with acrylic than silicone, even when adjusted for usage.

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Regarding Hong et al.'s concern about the pupil sizes used in our article: We chose a 2.5 mm pupil for photopic conditions because it matches the pupil size outside or in a photopically illuminated examination room (overhead lights on) with a penlight. The average pupil diameter in elderly and pseudophakic eyes under photopic conditions has been reported to be 3.2 to 3.8 mm.[17–21] These are apparent pupil sizes as seen by the clinician, which are magnified by 14% due to the converging power of the cornea. When one accounts for the difference in the apparent pupil size and the actual pupil size within the eye, the actual photopic pupil would be from 2.8 to 3.4 mm. Since ND is exaggerated with a penlight, the pupil would be even smaller, so we chose the 2.5 mm for the photopic pupil with a penlight. We chose a 5.0 mm pupil diameter to match the pupil size for evaluating the reports of disappearance of ND with pharmacologic dilation, which would normally be even larger. As we stated in the article, the sharpness of the shadow would gradually decrease with increasing pupil sizes above 2.5 mm and be unrecognizable by 5.0 mm or more as shown in Figures 6 and 7 of our study.

Regarding the comment that our "theory would not explain that ND has never been reported with anterior chamber IOLs (AC IOLs) or sulcus-placed IOLs," is not true and highlights a fundamental concept underlying how ND occurs. Negative dysphotopsia cannot occur with AC IOLs because they cannot be 0.06 mm behind the iris (Figure 13). Regarding the sulcus-placed IOLs, it is possible to produce ND because they can be 0.06 mm behind the iris and still be in the sulcus. Masket and Fram[22] confirm these observations in their article in cases 4 and 5, in which silicone 3-piece IOLs were placed in the sulcus with powers of 0.0 diopters (D) and +4.0 D and the ND "was improved" but not eliminated. The improvement was a result of the nominal type 3 shadow for acrylic in the bag (0.5 mm behind iris) having a retinal field angle of 86 to 100 degrees (14 degrees wide), whereas the silicone in the sulcus (0.06 mm behind iris) is 92 to 101 degrees (9 degrees wide). The new shadow is two-thirds the width and 6 degrees more anterior. The extremely low front-surface powers of the secondary piggyback IOLs (0 and +4.0 D total power) were also important to allow the pencil of peripheral rays to be incident on the nasal edge and nasal posterior surface of the IOL. For a single primary sulcus IOL, this low front-surface power would be even more improbable, but ND is still possible.

Whether external anatomy, such as a recessed brow or proptosis has a role in ND is rare but can be confirmed by determining whether the cornea can be seen from the temporal side at an angle of 105 degrees when the patient is in primary gaze. It is much more likely for the internal anatomy of the eye to be a contributing factor when the border of functional nasal retina is located more anteriorly than average. The location of the anterior border of functional nasal retina has a standard deviation of 0.8 mm for this border,[23,24] so patients complaining of ND after their primary surgery are likely to be among those with a more anterior border. Exchanging the PC IOL for an AC IOL or using a *fully* (not partially) rounded edge IOL are the only 2 treatments that are sure to eliminate ND. Exchanges for a silicone material, secondary piggyback IOLs, and reverse optic capture usually will "improve" the symptoms, but cannot guarantee elimination of ND.

Our comments about Osher's article[9] and letter,[25] authored by someone we consider to be an astute clinician with exemplary observational skills, were meant to highlight his distinction between "permanent" and "transient" ND. Osher commented that

"permanent negative dysphotopsia seems to be related to the contour of the IOL optic, primarily its truncated square edge or its edge reflectivity." He was correct that the truncated sharp edge is a critical and essential factor for ND. We further explain that the location of the wound and crescent-shaped shadow that he saw on the iris has nothing to do with ND. The reviewer required that we explain the difficulty with Osher's "transient" ND explanation and the observation of the straight to curved image on the iris with rotation of the slit beam. We complied and explained in detail that the temporal clear corneal incision was not conjugate with the peripheral retina, so it could not be seen and the curvature of the slit-beam image was due to curvature of field that occurred when rotating the slitlamp. We also indicated that ND was still present with the same incidence when the incision was at other locations than temporal. As we stated in the article, the location of the incision and the image of the slit beam on the iris have nothing to do with ND.

Masket and Fram's comment that they have observed ND despite opacification (actually translucification, since it is not opaque) of the anterior capsule overlying the IOL further supports our model. Opacification of the anterior capsule overlying the IOL would not affect the type 3 shadow because the light rays would undergo Lambertian scatter and be reflected or refracted and then pass through the front surface of the IOL, creating an alternative path for incident light to reach the truncated sharp posterior nasal edge of the IOL creating or enhancing the type 3 shadow. In fact, it is possible that this may be the only path and removing a sector of opacified nasal anterior capsule overlying the IOL with a neodymium: YAG laser might eliminate the ND by removing this path of scattered light. It is the portion of the anterior and posterior capsule peripheral to the edge of the IOL that scatters light into the shadow and eliminates ND. The increased incidence of peripheral capsule opacification over time explains the complete resolution in the majority of cases. We also mentioned an alternative possibility—that forward movement over time due to capsular bag contraction could lead to spontaneous resolution of ND by moving the shadow anterior to the border of functional retina but that this would be associated with a myopic shift due to increased IOL effectivity.

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