Differentiating transient and permanent negative dysphotopsia

I would like to comment on the case report about negative dysphotopsia by Cooke. It is disconcerting to point out that the author’s premise is incorrect.

Throughout my article, including the title, the discussion, and the conclusion, I emphasized that the negative dysphotopsia caused by corneal edema is **transient**. When I reviewed a consecutive series of 250 patients, I was astonished to find that 15.2% of patients were able to perceive a crescent-shaped temporal shadow immediately after surgery. Even more surprising was the finding that most of these patients became asymptomatic within the first several weeks after surgery. I hypothesized that these transient symptoms could be due to the broad-based clear or near-clear corneal incisional edema that interferes with oblique light projected into the far peripheral field known as the monocular temporal crescent. As the corneal edema resolves, the patient no longer perceives the shadow and the negative dysphotopsia disappears.

In contrast to the proposed incisional role in causing transient negative dysphotopsia, I clearly stated that **permanent** negative dysphotopsia “seems related to the contour of the lens optic, primarily its truncated square edge or its edge reflectivity. That there are patients who have had superior incisions yet are bothered by temporal shadows confirms that the incision is not the primary cause of permanent negative dysphotopsia.” I provided an extensive review of the literature and concluded that in patients whose symptoms rapidly disappear within the first several weeks after surgery, the associated edema of a clear or near-clear corneal incision seems to be a plausible explanation. I apologize if words such as “early postoperative period” were confusing. I clearly stated that “[i]n contrast, the etiology of the shadow in the group with long-standing or permanent symptoms must be related to the IOL.” In the final paragraph of the article, I again concluded that “[i]n contrast, permanent symptoms are clearly the result of a complex interaction between the optics of the IOL edge and an eye (or patient) with a unique anatomic disposition.”

Cooke fails to differentiate between transient and permanent negative dysphotopsia in his case report, and he erroneously states that I incriminated the temporal incision in both transient and chronic symptoms. His patient had a “shadow crescent” for 6 months before the intraocular lens (IOL) was exchanged through a temporal incision. Because the symptoms disappeared, Cooke concludes that “[t]he case reported here shows that not all cases of negative dysphotopsia are due to temporal incisions because the symptoms occurred with scleral tunnel incisions and resolved immediately after IOL exchange with temporal incisions.” Quite frankly, I would not have been surprised that the long-standing symptoms resolved since the patient’s complaints were not transient, hence not related to the incision. Nor would I have been surprised if the symptoms were unchanged after the IOL exchange, which has also been reported.

Cooke also states that “it has been proposed that long-lasting negative dysphotopsia is more likely to occur in combination with a prominent globe, brown irides, and shallow orbital anatomy,” again, he is incorrect and should have read my article more carefully. These findings were merely noted to have been shared in common by the patients with long-term dysphotopsia in this study. There was no proposal for causation, merely an observation intended to raise unanswered questions. I apologize if the word “perhaps” was confusing.

I realize that my theory to explain the common postoperative observation of negative dysphotopsia has been highly controversial. I am certainly willing to defend my hypothesis, but only when it is clearly understood and limited to transient rather than long-standing negative dysphotopsia.

Robert H. Osher, MD
Cincinnati, Ohio, USA

REFERENCES


REPLY: I appreciate Osher’s research and the contribution he has made to our understanding of this rather perplexing issue. In my case report, I intended to make a distinction between transient and permanent symptoms by stating, “Osher divided symptomatic patients by how long the symptoms lasted—short term or long term.” Later, I stated, “He divided his findings into those with short-term symptoms and long-term symptoms.”

In both his paper and his letter, Osher recognized there are patients who have had superior incisions yet are bothered by permanent negative dysphotopsia. I accept that in his discussion he states the cause of permanent symptoms is “difficult to understand” and is likely to be multifactorial. Yet in his series of 250 procedures, it is difficult to escape the fact that every occurrence of negative dysphotopsia, whether transient or permanent, was associated with exposure of
a temporal incision. In all 84 procedures in which the lid completely covered the superior incision, the patients had no symptoms. It seemed reasonable to infer there was an association between incision location and permanent symptoms. This is why I said the incision “appeared to be incriminated in the long-term symptoms because no dysphotopsia symptoms occurred in patients with incisions that were completely covered.” I did not mean to imply Osher thought the incision was incriminated.

I did think Osher was presenting a theory about long-term negative dysphotopsia. In the title, it is clear he was offering an explanation for transient symptoms. Yet in the body of the article, he devoted much space, most of the figures, and all 4 tables to permanent dysphotopsia.

Osher’s study required a large amount of work, and I apologize that my case report appeared to minimize his contribution. It was intended as a counterexample to show some of the anatomic associations observed and discussed by Osher may be absent. It is also a demonstration that permanent negative dysphotopsia occurs with incisions covered by the upper lid.

It seems we fundamentally agree that long-term negative dysphotopsia is better explained by IOL and perhaps other biometric parameters than by corneal edema or incision site. We all look forward to a better understanding of the precise causes of negative dysphotopsia, and I hope my case report will add to the collective knowledge in this regard.—David L. Cooke, MD

Optical phenomena causing negative dysphotopsia

In his article, Cooke rightly ruled out temporal incisions and other purported causes as a source of negative dysphotopsia. The association of unique anatomic predisposition resulting in prominent globes, dark irides, blue irides is incidental and cannot be implicated as a cause as these features are found in persons who have not had cataract surgery. What differentiates persons with negative dysphotopsia from normal persons is cataract surgery. Since the superior temporal incisions are also associated with such experiences, one must try to understand the comment “by putting hands to the side of each eye, the shadow went away” in terms of optical phenomena.

One optical phenomenon that might explain this is “negative afterimages,” which occur when the eye’s photoreceptors, primarily cone cells, adapt from overstimulation and lose sensitivity. The eye normally deals with this problem by microsaccades, rapid small amounts of movement, which filter out the negative images and make them unnoticeable. Although the mechanism of microsaccades nullifying negative afterimages primarily comes into play in cases of large color images (a cone cell function), large enough that the small movements are not enough to change the color under one area of the retina, rod cells can be affected in a similar manner. That the negative dysphotopsia are in the peripheral field of vision could explain and strengthen the argument that rods are equally affected by changes in microsaccades movements. In normal circumstances, cones will eventually tire or adapt and stop responding. As rods are primarily meant for mesopic vision, they do not come into play in photopic conditions and may not tire and could not adapt as efficiently as cones, causing negative afterimages even after a passage of time. The disappearance after intraocular lens (IOL) exchange is due to the larger size of the second IOL, which might have dampened/restored the microsaccades, or adaptation by the brain, which occurs in other conditions of palinopsia.

Further research into these phenomena is required as the number of negative dysphotopsia cases are on the increase, especially among literate and discerning individuals.

Seshubabu Gosala, DOMS
Andhra Pradesh, India

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REPLY: Gosala presents an interesting concept that negative dysphotopsia might represent negative afterimages. In the Science article that Gosala cites, subjects fixated on and were adapted for 20 seconds to an image. They then observed the afterimages on a blank screen in a semidark room. Negative afterimages typically occur after the stimulus is removed. Curiously, the opposite occurred in the patient in my case report. He did not have an adaptation period followed by an image. The image was constant until he put his hands to the side of each eye, presumably blocking the stimulus.

Gosala offers another explanation for the disappearance of negative dysphotopsia after IOL exchange, which is neuroadaptation (“adaptation by the brain as occurs in other conditions of palinopsia”). However, I don’t understand why the brain would neuroadapt the day after IOL exchange when it had not neuroadapted for the prior 6 months.