Floaters and the Quality of Life

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N 1976, DR ERNST WYNDER, FOUNDING PRESIDENT OF THE American Health Foundation and founding editor of the journal Preventive Medicine, stated that "It should be the function of medicine to help people die young . . . as late in life as possible." However, medicine in general, and ophthalmology in specific, are disease-oriented disciplines. In the former case, this is due to the fact that "Disease is a living experience for the man of flesh and bone. In contrast, health is a disembodied concept. It stimulates no emotional response because it is an inhuman, fleshless abstraction" (René Jules Dubos, The Silliman Lectures, Yale, 1965). As ophthalmologists, we are trained to evaluate eyes by seeking evidence of disease using biomicroscopy and ophthalmoscopy, as well as with diagnostic testing. When we rule out pathology by these modalities, we advise patients that they are free of disease. Such advice is apparently a frustrating and unfulfilling experience for patients with "floaters." From their point of view, the consulting ophthalmologist who sought evidence of disease and found none has nonetheless failed to address their health and quality-of-life issues.

In youth, vitreous is a solid and clear gel filling the center of the eye, firmly attached to the retina. The exquisite transparency of vitreous allows unhindered transmission of light to the retina for photoreception.² Other than "exogenous" sources, such as hemorrhage and inflammation, there are 2 main causes of the entopic phenomenon called "floaters," which result from light scattering by structures within the vitreous body and/or at the posterior vitreous cortex. Although devoid of liquid during infancy, the vitreous body begins to liquefy in childhood due to molecular rearrangement of the constituent macromolecules hyaluronan and collagen. Part of this molecular alteration involves cross-linking and aggregation of vitreous collagen fibrils. If advanced, fibrillar aggregation can cause sufficient interference of photon transmission to induce chronic and progressive floaters. In myopia, the process of vitreous gel liquefaction and fibrillar aggregation appears to be accelerated, a manifestation of myopic vitreopathy.3

Concurrent with vitreous gel liquefaction, there is weakening of vitreoretinal adhesion. When both processes advance in tandem to a critical threshold, 2,3 there is separation of the posterior vitreous cortex from the retina, perhaps initially only in the perifoveal region. 4 Displace-

See Accompanying Article on page 60. Accepted for publication Feb 11, 2011.

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ment of liquefied vitreous into the developing cleavage plane between the posterior vitreous cortex and the internal limiting lamina of the retina collapses the posterior vitreous away from the retina, an event called posterior vitreous detachment (PVD). While it has long been taught that PVD is abnormal, it may well be that PVD is the salubrious result of evolutionary progress. This concept arises from the growing awareness that in a variety of diseases, such as diabetic retinopathy⁵ and age-related macular degeneration^{6,7} (AMD), PVD is a far safer condition. That notwithstanding, entopic phenomena resulting from this event induce acute floaters. These arise from the posterior vitreous cortex itself as well as tissue that is sometimes adherent to the posterior vitreous cortex, typically parapapillary fibro-glial in origin. When attributable to myopic vitreopathy,³ PVD occurs 10 to 15 years earlier in life.

The subjective experience of sudden floaters is very common after PVD. While many patients complain that this is bothersome, ophthalmologists tend to pay little heed to these symptoms other than to rule out anomalous PVD^{3,8} manifesting as either peripheral or posterior retinal pathology. Once the absence of disease has been assured, the typical eye care professional ceases to be concerned about the issue of floaters. While the Hippocratic principle of "primum non nocere" has guided our approach to date, it may well be time to reexamine our perception that floaters are simply an innocuous, indeed curiously desirable, manifestation of the "normal" aging process.

In this issue of the Journal, Wagle and associates⁹ present fascinating new information concerning the utility value of floaters, as expressed by patients. Utility values allow an objective quantification of the functional quality of life associated with a specific "disease" state. A utility value of 1.0 implies a perfect "health" state, while death has a utility value of 0.0. The findings of this study indicate that the utility values of floaters are equal to AMD and lower than diabetic retinopathy and glaucoma. According to this study, floaters have lower utility values than mild angina, mild stroke, colon cancer, and asymptomatic HIV infection. This indicates that floaters have a significant negative impact on the quality of life as compared to ocular as well as systemic diseases. It is interesting to note that there was no difference between acute (less than 1 month) and chronic (mean duration of more than 1 year) floaters. This finding throws into question our long-held belief and oft-offered counsel to patients that their symptoms will lessen in severity, either due to settling of vitreous opacities below the optical axis or because of neuro-psychological adaptation. Surprisingly, the investigators claimed that

49.3% of the study group had no PVD. This is suspect, since subjects only underwent an examination and not diagnostic testing, such as ultrasound or optical coherence tomography. Furthermore, 56% of the subjects were women and 59.7% were myopic—both known to predispose to PVD. On the other hand, the authors appropriately point out that the retinal magnification of the images associated with myopia can make floaters seem more pronounced, perhaps explaining how a large number of subjects in this study complained of floaters in the absence of a PVD.

Most remarkably, the investigators of this study found that these patients were willing to take an 11% risk of death and a 7% risk of blindness to get rid of symptoms related to floaters. As the authors state, patients with floaters are willing to trade off 1.1 years out of every 10 years of their remaining lives to get rid of the symptoms of floaters. To some extent that explains the willingness of patients to undergo unproven attempts at mitigating their symptoms, such as YAG laser vitreolysis, for which there is no evidence of efficacy. Definitive treatment is available with vitrectomy, which has been rendered faster, less invasive, and safer by the advent of 25G instrumentation. Yet, there are small risks associated with this invasive procedure and in phakic patients there are lens-related considerations. To obviate the cost and risk (albeit small) of surgery, the

future will likely see the development of drug therapy for floaters, via pharmacologic vitreolysis. ¹⁰ Caution must be exercised, however, for some agents may induce or aggravate floaters as opposed to dissolve them. ^{10,11}

Future advances in our ability to promote health and not just treat disease will depend upon a paradigm shift in philosophy and the development of technologies for health evaluation. We should first accept health as its own diagnosis¹ and not just the absence of disease. Improving our understanding and management of conditions such as age-related vitreous degeneration, 2,3 diabetic vitreopathy, 12 and myopic vitreopathy will then depend upon developing new diagnostic nanotechnologies, such as dynamic light scattering (DLS). This noninvasive, laserbased nano-detector is able to quantitate particle sizes in the cornea, lens, aqueous, and vitreous¹³ as small as 3 nm in diameter. DLS has been used to determine an alphacrystallin index in 380 lenses of human eyes¹⁴ as well as demonstrate the effects of diabetic vitreopathy¹¹ and pharmacologic vitreolysis. 15,16 In the meantime, however, we need to be aware of and sensitive to the fact that there continues to be a proliferation of floater websites on the internet and the formation of international floater organizations, as expressions of patient frustration with our inability or unwillingness to help them die young, as late in life as possible.

THE AUTHOR INDICATES NO FUNDING SUPPORT. THE AUTHOR HAS BEEN A CONSULTANT TO PFIZER, SHIRE, STORZ, ALCON, ISTA, Vitreo-Retinal Technologies, and ThromboGenics, and is a shareholder in ThromboGenics, Ltd. The author (J.S.) is solely responsible for the conceptualization of this article; collection, management, analysis, and interpretation of data; and preparation, review, and approval of the manuscript.

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Biosketch

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