Letters to the Editor

Dear Editor:

We thank Dr. Nehmad for taking an interest in our article, in which we were careful to provide the reader with the actual data so that an unbiased opinion could easily be formed. Although Dr. Nehmad justifiably states that a significant proportion of young children do not perform a very reliable frequency-doubling technology (FDT) visual field (VF) task, we had an opposite impression—namely, that a significant proportion of children as young as 4 were perfectly capable of performing a VF task reliably the very first time they sat to the task. With repeated testing, as seen with adults, the proportion of unreliable tests is likely to decline even further. The significance of our article might be 2-fold: (1) device manufacturers may eventually decide to include a normative database for the younger population, and (2) far more importantly, a physician sending a 4- or 5-year-old child to perform a VF task (regardless of the test chosen) might not be viewed as wasting taxpayer dollars but, rather, using reasonable clinical judgment.

Regarding the reliability indices for FDT, because the number of trials (the denominator) for reliability scores in the FDT full-threshold program used is usually around 6, meaningful cutoffs include 16.7% (1/6) and 33% (2/6). We believe that 33% is an acceptable and common cutoff for many (if not most) FDT VF studies.

We look forward to future studies analyzing the Swedish interactive threshold algorithm as well as other automated perimetry programs as they pertain to young children, in the

is accentuated with overnight wear despite improved oxygen permeability of newer lens materials. There should be no dispute regarding the increased risk of corneal ulceration when overnight wear is compared with daily-wear contact lenses. There have been a number of cases reported in the literature where patients wore highly oxygen-permeable orthokeratology lenses and were meticulous with their contact lens hygiene.1-3

Last but not least, we believe that prudence is especially important if the lens wearers are children, as they may not have the maturity or understanding to volunteer and verbalize early unusual events experienced.

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References


Frequency-Doubling Perimetry in Young Children

Dear Editor:

Due to the clinical challenge of obtaining meaningful, reliable visual fields (VFs) for children and the relative lack of information on the subject in the literature, I read with interest the study on the reliability of frequency-doubling perimetry (FDP) in young children by Blumenthal et al.1 The authors are to be commended for their investigation of a population for which there is currently no normative perimetric database. But although the finding that FDP performance begins improving toward a relatively constant level starting around age 8 is of interest, the authors conclude that this is clinically meaningful. Some of their data, however, might suggest other interpretations.

Twenty-six and nine-tenths percent of the fields were abnormal for children 8 years and older. Because these were considered normal subjects, these results would seem to indicate a low specificity for the test. Twenty-three and one-tenth percent of VFs for children 8 and older were unreliable. This number is based on a somewhat broad definition of reliability: 33% false positives, false negatives, or fixation losses. Although some perimeters have used these values to flag the field as unreliable, in clinical prac-


Author reply

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We look forward to future studies analyzing the Swedish interactive threshold algorithm as well as other automated perimetry programs as they pertain to young children, in the
hope that they too will prove useful in the setting of this very young age group.

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Protective Eyewear for Young Athletes

Dear Editor:
I read with great interest the Joint Policy Statement about protective eyewear for young athletes.1 The publication considers mainly racket sports, baseball, basketball, women’s lacrosse, and field hockey. The need for protective eyewear in soccer was given far less attention than for these selected sports.2 Our study confirmed that the soccer ball causes eye injury by protruding into the orbit. In addition, we concluded that protectors that pass American Society for Testing and Materials Standard F803 and have polycarbonate lenses would prevent orbital intrusion. Therefore, and in agreement with Table 2 of the Policy Statement, we recommended the wearing of eye protectors that comply with the requirements of American Society for Testing and Materials Standard F803 for soccer and the writing of a specific standard for soccer.

Soccer ocular injury is an important eye health problem in Europe and South America and, probably, worldwide. Severe ocular injuries with potential long-term effects can be sustained on the soccer field, according to an analysis of soccer-related eye injuries at our sports ophthalmology unit at the University of Porto School of Medicine.3,4 As a result of these studies, we strongly recommend that soccer protective eyewear be worn, particularly by athletes who require prescription lenses, functionally one-eyed athletes, and those who have had refractive surgical procedures that weaken the eye. Reducing the number of injuries by encouraging players to use readily available protective eyewear that conforms to American Society for Testing and Materials Standard F803 would be in the best interests of public health.5

Injuries are predictable and, for the most part, preventable if all ophthalmologists make an appropriate eye safety prescription part of our routine. I look to the American Academy of Pediatrics’ Committee on Sports Medicine and Fitness and the American Academy of Ophthalmology’s Eye Health and Public Information Task Force for guidance on this issue, and I thank them for presenting this important policy statement.

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References


VSX1 Mutation and Corneal Dystrophies

Dear Editor:
I am greatly concerned about conclusions drawn in an article recently published in Ophthalmology that related a mutation in the VSX1 gene to the development of both posterior polymorphous corneal dystrophy (PPCD) and keratoconus.1 My concerns deal primarily with flawed criteria used to define the affected phenotypes of PPCD and keratoconus, internal inaccuracies within the article and in the authors’ interpretation of the relevant literature, and assumptions made by the authors in the interpretation of the results of molecular genetic analysis.

The most troublesome aspect of this article is the fact that the presented patients do not meet the standard, accepted criteria that are used to diagnose either PPCD or keratoconus. The diagnosis of keratoconus was made based solely on asphericity quotients obtained with the EyeSys topographer. To use this as the sole means of diagnosing keratoconus is inaccurate and not standard. Traditionally, the diagnosis of PPCD has been based on the observation of characteristic corneal endothelial changes, such as bands, vesicles, and gray–white geographic opacifications. The vesicles commonly appear in clusters, surrounded by a gray halo, and are best visualized with direct illumination. The authors present images of “vesicles” in the endothelium that are not consistent with the endothelial changes in PPCD. Additionally, the authors describe “holes” in the endothelial mosaic noted on specular microscopy that are not part of the standard criteria used to diagnose PPCD, and may simply correspond to guttae rather than to the endothelial vesicles of PPCD. Additionally, although peripheral iridocorneal adhesions may be seen in PPCD, the 360° adhesions that are depicted in this article are certainly not characteristic of PPCD, contrary to the authors’ statements.

Of concern also are apparent internal inconsistencies in the article. In Figure 1, the D144E change previously identified in the VSX1 gene2 is incorrectly listed as D114E. Additionally, it should be marked with an asterisk (according to the authors’ conventions) as the corresponding amino acid at codon 256 is highly conserved across the species, but this is not demonstrated in Figure 2A, as the corresponding amino acid at this position is not shown for the other species listed in the multiple sequence alignment. Additionally, the authors’ statement that the “A256S amino acid substitution will result in a change of conformation and, therefore, activity of the protein” is unfounded. Although the secondary structure of the VSX1 gene protein product, like other proteins, may be predicted by analyzing the primary amino acid sequence, it is not possible to