Letters to the Editor

Lens-Equivalent Age Controls for Diabetics

To the Editor:

Many psychophysical tests show a dependence on age, and good practice in clinical studies employing them requires test replication on age-matched controls. A number of physiological factors change with age, and the one that concerns us here is the absorption spectrum of the crystalline lens and its effect on the spectral composition of light reaching the retina.

In an analytical review of psychophysical and physical data for more than 900 normal eyes, Pokorny et al\textsuperscript{1} derived two linear relations describing the lens absorbance spectrum for the age ranges 20–60 yr and 60–80 yr. These may well be optimized approximations to some continuous function for which polynomial\textsuperscript{2} and exponential\textsuperscript{3} alternatives have been suggested.

Recently, Lutze and Bresnick\textsuperscript{4} assessed lens absorbances for diabetic patients and normal controls by the van Norren and Vos\textsuperscript{5} method, and confirmed the acceleration of lens “yellowing” in diabetes. By relating their results to the linear model of lens aging, they demonstrated that the absorbance increment of diabetics over age-matched normals was linearly related to diabetes duration over 4–34 yr. Lutze and Bresnick reported a regression slope of 0.018 absorbance units per year with the line passing very close to the origin.

This result provides a convenient basis for defining controls for psychophysical population studies on diabetics where lens-matching is more appropriate than age-matching (and where patient contact-time is short or where facilities for the van Norren and Vos method of lens absorbance assessment are not available).

Lens-matched age controls may be readily determined by simple calculation using equations (4) and (5), which are deduced as follows.

The linear model\textsuperscript{1} equations relevant to the Lutze and Bresnick study are:

\begin{align*}
D_v &= 0.4224 + 0.0068A \\
D_o &= -0.5303 + 0.02268A
\end{align*}

where \(D_v\) and \(D_o\) are, respectively, the normal “young” (20–60 yr) and “old” (60–80 yr) lens absorbance differences for the 420 and 550 nm stimuli employed and \(A\) is the age in years.

The absorbance increment relation for young diabetic lenses adopted here is:

\[ B - D_v = 0.0173T - 0.0260 \] (3)

where \(B\) is the diabetic lens absorbance and \(T\) is the diabetes duration in years. This is the regression of \((B - D_v)\) on \(T\) using mean B data, provided by Dr Lutze (as distinct from the original regression that used medians).

If these three equations are regarded as functional relations, then it is legitimate to find the age relationship between equi-absorbant normal and diabetic lenses by eliminating the wavelength dependent absorbances.

Thus, equations (1), (2), and (3) yield

\[ E = A + 2.54T - 3.8 \]

for \(20 < E < 60\) (4)

\[ E = 0.30A + 0.76T + 40.9 \]

for \(60 < E < 80\) (5)

where \(E\) is the age of a normal lens having the same absorbance spectrum as that of a diabetic patient of age \(A\) and with a diabetes duration \(T > 1.5\). For durations up to 1.5 yr, \(E = A\). (\(E, A,\) and \(T\) in yr)

Consider, for example, two diabetic patients, both 30 yr old but with different diabetes durations: 7 yr and 20 yr. Equation (4) applies to the former and predicts a lens-equivalent control of 44 yr, whereas equation (5) applies to the latter (because \(E\) predicted by equation (4) is greater than 60) and predicts a lens-equivalent control of 65 yr.

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References

2. Coren S, Girgus JS. Density of human lens pigmenta-
5. van Norren D, Vos JJ. Spectral transmission of the human ocular media. Vision Res. 1974;14:1237–.

Congenital Nystagmus Mechanism

To the Editor:

The recent report by Waugh and Bedell1 presents an interesting description of the threshold sensitivity to large-field temporal flicker of individuals with congenital nystagmus (CN). Although the enhanced sensitivity to low temporal frequency is of interest by itself, the inferences drawn by the authors concerning the suppression of oscillopsia in such patients seem unwarranted.

Chief among these is the claim to have disproved the idea that CN patients sample their visual world only during foveation periods, suppressing visual input during other portions of their slow phase waveforms. The concept that vision in CN patients only occurs during foveation periods would imply that these individuals perceive a flickering world, with darkness filling in the times of high velocity retinal slip. Because no one has ever advanced such a hypothesis, and a conversation with anyone with CN would disprove it, the present attack upon it seems superfluous. The authors’ inferences about the relevance of their findings to suppression of oscillopsia assume homogeneous processing of the entire visual field, with signals from the peripheral retina and fovea being treated identically. Since peripheral retina, with its higher concentration of M cells,2 is preferentially sensitive to change but not involved in fine spatial resolution, it would be quite plausible for its output to be processed with constant sensitivity at different points in the CN slow phase, while foveal signals were attenuated during high velocity portions. This would be consistent with the results of Waugh and Bedell as well as with both the perceptual continuity and absence of oscillopsia that characterize the visual experience of individuals with CN.

The foveation periods in idiopathic CN (ie, when the retinal image of the target is both in the foveal area and has a low retinal slip velocity) remain the only periods of time when clear, high resolution vision is possible. Furthermore, the retinal image and ocular motor stability criteria3–5 that are required for the suppression of oscillopsia are only satisfied during these foveation periods. The reduced acuity or perception of oscillopsia in individuals whose afferent defects or acquired neurological conditions result in poor foveation support rather than detract from the importance of well-developed foveation periods for both high acuity and oscillopsia suppression.

The findings of this study are of inherent interest for what they say about the perceptual modifications occurring in individuals with CN. Stretching them to serve as an attack upon a “straw man” hypothesis about oscillopsia suppression requires making unwarranted assumptions about the existing literature.

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References


Reply

To the Editor:

The primary purpose of our report1 was to describe the temporal contrast sensitivity function for a large, uniform field stimulus, in an attempt to more fully understand the visual sensitivity of persons with congenital nystagmus (CN) to such stimuli. Although the sensitivity of these persons to various spatial stimuli had been previously described, little was known about their sensitivity to temporally modulating stimuli.

We believe that our results also bear upon the question of why persons with CN do not report oscillopsia, despite the almost constant to and fro movement of their eyes. Because the temporal contrast sen-