Correspondence

Corneal arcus and coronary heart disease mortality

Sir. Previous studies in western countries reported an association between corneal arcus and coronary heart disease (CHD). In-hospital studies have shown that corneal arcus is a useful discriminator between CHD patients and controls. The mechanism of the statistical association between arcus and CHD is unclear, and several studies suggest that the two are not directly related but rather that both are associated (independently) with elevated cholesterol levels.

Evans County, Georgia, a biracial, rural community located in the high stroke region of the south-eastern United States, has been the subject of a longitudinal study of cardiovascular disease since its inception in 1960. All county residents aged 40 and older and 50% of those less than age 40 were invited to participate in the study. 3102 (92%) agreed and were examined during 1960–2. The race and sex composition of the cohort is as follows: 947 white males, 972 white females, 537 black males, and 646 black females. Visual inspection of corneal arcus was included in the baseline physical examination and was diagnosed in 625 persons. Mortality surveillance for this cohort has been maintained. Causes of death extracted from death certificates were classified according to the eighth revised edition of the International Classification of Disease (ICD). As of mid-1980 64% of the original cohort were known to be alive, 34% deceased, and 2% were lost to follow-up. The present data included examinees aged 40 and older at baseline. Of these 2216 subjects 60 have been excluded because their arcus status was unknown, and an additional 90 are excluded because of prevalent heart disease at baseline examination. An additional 35 people are excluded because, although deceased, their death certificates were not available, and exact date of death is unknown. The age, sex, and race specific means of serum cholesterol, blood pressure (systolic and diastolic) and weight/height (Quetelet index) were compared between subjects with and without arcus. Cholesterol and blood pressure (systolic and diastolic) were slightly higher in subjects with arcus than in others.

The possible association between arcus and CHD mortality (ICD 410–414) was examined by comparison of age-race-sex specific Kaplan-Meier survival curves and tested by Mantel-Haenszel statistics (Table 1). The differences in CHD mortality between subjects with and without arcus were small and inconsistent across the age, sex, and race groups. The only significant differences (0.01 \( \leq p < 0.05 \)) found in the white male group aged 40–59 are based on a small number of observations. The lack of significant association between arcus and CHD mortality in our data supports an earlier report based on Evans County data in which corneal arcus was not predictive of the incidence of CHD.

In a previous study using slit-lamp examination it was shown that the location and density of corneal arcus were important determinants of the arcus-serum cholesterol association. It is possible that there are differences in the association between CHD mortality and various types of arcus that cannot be differentiated by visual inspection of the eyes. If corneal arcus is to be used for prognostic purposes, we recommend the use of more definitive examination procedures for diagnosis.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>White males, arcus</th>
<th>White females, arcus</th>
<th>Black males, arcus</th>
<th>Black females, arcus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>40–49</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>23*</td>
<td>25</td>
<td>217</td>
<td>13</td>
</tr>
<tr>
<td>50–59</td>
<td>35</td>
<td>15</td>
<td>164</td>
<td>17</td>
</tr>
<tr>
<td>60–69</td>
<td>38</td>
<td>38</td>
<td>69</td>
<td>34</td>
</tr>
<tr>
<td>70+</td>
<td>25</td>
<td>52</td>
<td>16</td>
<td>46</td>
</tr>
<tr>
<td>Total</td>
<td>123**</td>
<td>31</td>
<td>466</td>
<td>18</td>
</tr>
</tbody>
</table>

*\( \chi^2 \) significance 0.01 < p < 0.05.
**\( \chi^2 \) significance p < 0.001.
†Kaplan-Meier derived.
‡Examines free of coronary heart disease in 1960.
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Sir. With reference to Dr Bronte-Stewart’s letter I do not propose that the City University Colour Vision Test be used for clinical diagnoses on the basis of its classification of colour defects. The paper under discussion suggested that under conditions of reduced viewing time the response of control subjects, with no ocular pathology or inherent colour defect, is different from the responses of subjects suffering from ocular pathology. A change in colour vision with reduction in viewing time, in control subjects, has been found by the use of more sophisticated techniques of measurement of colour performance than the 'City' test. The results of these tests do not confirm the ‘City’ diagnosis of a tritan defect. The ‘City’ colour test was in fact chosen for its ease of administration and not for its diagnostic ability.

I would agree that the ‘City’ test is not an adequate screening test, as has been suggested. It is designed from the colours of the Farnsworth panel D-15 with additional chips to aid classification of defects, and on this basis alone it is simply a colour test to screen suitability for specific occupations where a moderate to severe defect would be a hindrance. It does not provide clear cut classifications and, even with the improvements in scoring technique that have been suggested, it does not reach the screening standards of other tests. The above does not, however, preclude the use of the ‘City’ test as an indicator of change in colour vision.

Department of Optometry,
UWIST,
Colum Drive,
Cardiff CF1 3EU.

S. P. TAYLOR

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S T Halfon, C G Hames and S Heyden

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