stray light also spreads horizontally in the retina, the size of the blind spot depends to some extent on the stray light generating properties of the disc, i.e., due to its pallor. One can argue, therefore, that any decrease of the size of the blind spot in time, or its disappearance, may be related to changes in its pallor, provided other sources generating stray light can be shown to be constant.

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Reply

T. Bek

We wish to thank Dr. Fankhauser for his important supplementary remarks on the effects of stray light as a contributory factor to the findings described in our paper.

Our study explored some of the methodological considerations involved in developing a technique for the correlation of localized retinal morphology with localized light sensitivity in diabetic retinopathy. We demonstrated that when the detection of small scotomata is attempted using dense stimulus patterns within the existing dynamic range of the perimeter, the stimulus size is a very important variable to be taken into account. Hitherto, most automated perimetry studies in diabetic retinopathy have been carried out employing the Goldmann III stimulus size, probably because on many perimeters this stimulus size has no value. With this target size, smaller scotomata may easily escape detection, however, a fact which we have confirmed in our practice.

In our routine procedure the employing Goldmann I stimulus we test grids with a stimulus spacing of 1 degree. With this procedure, only small parts of the visual field can be tested within an acceptable testing time. We, therefore, specifically choose the relevant part of the visual field to be tested – corresponding to morphological lesions in the retina – on the basis of the funduscopic examination prior to perimetry. The visual field data are accurately correlated with the corresponding fundus morphology as represented on fundus photographs, according to an algorithm we have described elsewhere [1].

Reference

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Oculogyric crisis after the Tensilon test

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Sir,

A 21-year-old white girl, with no history of post-encephalitic parkinsonism, epilepsy or drug dependency, presented with bilateral ptosis, which was minimal in the morning and became more severe as the day progressed. She was examined in the Neurophthalmology Unit of San Raffaele Hospital. Levator fatigue was demonstrated as slowly progressive ptosis, which was provoked by a sustained gaze in the upward direction. In order to corroborate a preliminary diagnosis of myastenia gravis, we decided to carry out a Tensilon (edrophonium hydrocloride) test [1].

We injected 0.2 ml Tensilon and flushed with saline. When after 1 min no change had occurred, we injected another 0.4 ml Tensilon. After a few seconds, significant improvement in the ptosis occurred.

While we were recording the results of the test, the patient underwent an oculogyric crisis, with spasmodic upward deviation of the eyes. The first attempt to resolve the oculogyric crisis was to inject 0.4 mg atropine sulfate. Since there was no resolution of the crisis we decided to administer diphenhydramine IV [3], after which complete resolution of the crisis was obtained. The day afterward an EEG disclosed no abnormalities.

The causal mechanisms of the oculogyric crisis are still unclear. Sometimes an epileptic attack can be confused with an oculogyric crisis. The normal EEG obtained, for our subject, few hours after the crisis allowed us to rule out this hypothesis.

Fraunfelder [2] listed approximately 50 drugs reported to cause oculogyric crisis, but he did not mention edrophonium hydrocloride, and none of the drugs reported was being used by our patient. Our case report

is the first one describing oculogyric crisis after the Tensilon test and is aimed at making ophthalmologists aware that this test could have this complication. Moreover, it shows that taking precautions when administering the Tensilon is of no use in predicting or avoiding the crisis.

As papers are stressing the safety and feasibility of this diagnostic procedure [4], our case report is important.

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