Correspondence

The optic disc in glaucoma

Sir, The article by Hitchings and Wheeler\(^1\) demonstrates the difficulty in recognising nerve fibre layer loss in glaucoma. Except for a single case they noted no difference in the visibility of the nerve fibre layer between normotensive eyes and hypertensive mates of 10 patients with uniocular hypertension. Yet another case, illustrated in Fig. 4 of their article, clearly shows this difference. Nerve fibre layer striations are far more apparent in the normotensive than the hypertensive eye. The loss of nerve fibre layer is confirmed by the sharper definition of major vessels in the hypertensive eye. These changes are most apparent in the area of the superior temporal vein.

Nerve fibre layer loss can be recognised by slit-like defects,\(^2\) but also by thinning of the nerve fibre layer as it crosses the temporal rim and as a general loss of striations and prominence of the major vessels near the disc.\(^2,4\) Unfortunately these changes are often difficult to recognise. Better, more reproducible techniques for visualising the nerve fibre layer and its abnormalities are clearly needed.

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References


Sir, We thank Dr Sommer for his interest in our article and fully endorse his view that slit-like defects in the retinal nerve fibre layer are important evidence of neuronal loss. Caution must be used, however, when interpreting general loss of visibility of the nerve fibre layer as seen on photographs, for this appearance may be altered by refocusing the camera. The 2 photographs illustrated are of the same eye seen in Fig. 4 of our article, taken at the same visit but at different foci. A difference in visibility in the retinal nerve fibre layer is apparent on comparing the 2. Thus while intra-photography differences in neuronal visibility may accurately reflect neuronal loss, inter-photograph differences may reflect different focusing levels; we consider that this reason accounts for the change noted by Dr Sommer.

A second major problem arises when interpreting the lack of visibility in the retinal nerve fibre layer as evidence for neuronal loss in glaucoma patients, for many patients will have minor degrees of opacification of the lens. This opacification combined with an inability to dilate the pupil fully will suffice to prevent clear visualisation of the nerve fibre layer.

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The measurement of cyclofusional response

Sir, In their recent paper Sen et al.\(^1\) claimed to have measured torsional fusional vergence by a synoptophore
method. It has been generally accepted that human fusional response to retinal image disparity contains two components: a motor component in the form of compensatory, vergence eye movements and a sensory or nonmotor component described by the extent of Panum’s fusional areas. Objective measurements of cyclofusional response, which utilised binocular eye movement monitoring devices, revealed a substantial sensory component in cyclofusional response.  

The magnitude of this sensory component is a function of stimulus complexity and of the visual angle subtended by the stimulus. Subjective methods such as the one used by Sen et al. may be used to measure the overall amplitude of cyclofusional response, but cannot be used to measure the amplitude of either the motor or the sensory components individually, because the method is unable to distinguish between the two components. Therefore Sen et al. could not have measured torsional vergence (a term reserved exclusively for the description of the motor component) as was indicated in their paper, but only the overall cyclofusional response. I think that the interesting results of Sen et al. may be better appreciated subsequent to this correction in terminology.

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References


Oculocardiac reflex

Sir. A recent article by Drs Apt and Isenberg described the usefulness of the oculocardiac reflex in identifying and recovering a lost muscle. The authors make the observation that a chronically lost muscle may be difficult to identify visually, ‘often blending into the surrounding tissues.’ They also correctly point out that the ‘area to be explored often has adhesions and extraneous tissue.’

It is not clear to me how the surgeon can be certain that he is not pulling on nonmuscular scar tissue which has adhered to the true muscle, thereby producing a vagal response. This false muscle identification would lead to surgery (recession, resection, advancement) on nonmuscular tissue, which would not correct the patient’s strabismus. Also, in a recent paper by Drs Parks and Bloom they demonstrated that in certain cases the muscle capsule may remain attached to the surgical insertion site on the sclera, while the muscle itself slipped back within the capsule; surgery performed on the nonmuscular capsular tissue was ineffective in correcting these patients’ motility problems. The oculocardiac reflex was not investigated in this study. However, it is reasonable to expect that, since the capsule remained intimately attached to its muscle, pulling on the capsule would stretch the muscle and produce the oculocardiac reflex.

Drs Apt and Isenberg have made an important observation in the treatment of both the lost and the slipped muscles. However, the surgeon must be aware that production of the oculocardiac reflex may represent a false-positive result in the identification of a lost or slipped muscle.

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