

Professor Weale must be fully aware that no writer is ever consulted about publication price. Weale admits his opinion may be 'a little on the churlish side.' Agreed. Of course, I would have phrased it differently.

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Contrast sensitivity testing

SIR, A recent article¹ reports an important methodological improvement for contrast sensitivity testing with printed gratings derived from the Arden plates.² Vaegan and Halliday¹ used a 4-alternative forced-choice procedure to reduce the influence of response bias, and presented results obtained in glaucoma. However, in discussing these results they inaccurately characterised our previous findings on contrast sensitivity defects in glaucoma.³⁻⁵ The inaccuracies have implications which concern not only testing methodology but also the pathophysiology of vision changes in glaucoma.

Vaegan and Halliday¹ found that 'glaucoma patients . . . have a contrast sensitivity deficit of about 6 db in relation to aged matched normal persons at all spatial frequencies when tested with a conventional oscilloscope display of static gratings' (p. 481).¹ They erroneously conclude (p. 481)¹ that their results 'disagree with the findings of Atkin *et al.*³, and speculate (p. 490) that 'It is possible that the sample was too small or unrepresentative in many studies which do not find significant differences' (citing Atkin *et al.*^{3,5}). There actually is no disagreement. Our results with static patterns (unpublished) were similar to Vaegan and Halliday's.¹ The static stimuli, which were similar to those used in conventional contrast sensitivity methods,^{1,2} had in fact yielded significant differences between *group* means but had not shown a high enough ability to discriminate *individual* patients from normals to offer promise of a clinically useful test. Therefore in our original abstract we had said that 'sensitivity to the (non-flickering) grating . . . did not distinguish between patients and controls',³ meaning that the groups showed such a large amount of overlap that the static-target method provided little useful information about *individual* patients. Consequently our subsequent papers^{4,5} reported only the results with flickering targets.

With targets flickering at 8 Hz (a method not used by Vaegan and Halliday¹), our studies revealed dynamic components of early glaucomatous vision changes^{4,5} which appeared to be more clinically useful than the static response data. Vaegan and Halliday¹ seemed in fact to recognise this possibility, for they suggested (on p. 490) that 'Atkin *et al.*^{4,5} may . . . have had greater success with phase-alternating stimuli because responses to them are more reliable . . .' Exactly so! With dynamic testing there had been nearly perfect discrimination between glaucoma patients and age-matched control subjects.⁴ We had therefore concluded that the dynamic method might be useful to characterise individuals, not just groups.

It is important to call attention to this methodological difference—the use of *dynamic* rather than static contrast

sensitivity measures. Since our original publications³⁻⁵ there have been several studies corroborating our emphasis on the importance of temporal modulation for discerning early effects in glaucoma, all suggesting that an abnormality in glaucomatous visual responses is apparent with particular clarity at moderate frequencies, e.g. 8 Hz,⁶⁻¹¹ as well as at higher frequencies.⁶

Disparities in test sensitivity between dynamic and static methods may have pathophysiological implications. The higher sensitivity found for tests using flickering targets tends to support 2 of the several pathophysiological hypotheses that have been proposed to account for vision changes in glaucoma. (1) The first hypothesis was that there might be an early differential vulnerability of one component of retinal organisation (perhaps a subclass of retinal ganglion cells) which is especially sensitive when stimulated with fast temporal frequencies and less sensitive when stimulated with static gratings.^{2,8} (2) A subsequent suggestion was that vision changes might result in part from reduced synaptic efficacy within the CNS, due to depression of the rapid component of axoplasmic flow in optic nerve axons.¹² The resulting neurotransmitter depletion could lead to a generalised reduction of visual sensitivity¹² that would be exacerbated by temporal modulation, therefore showing greater effects with dynamic than with static testing.

We offer the foregoing comments to set the record straight about statements in the Vaegan and Halliday paper¹ which could cause confusion.

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SIR, Drs. Atkin *et al.* should not complain that their work has been inaccurately reported. In their letter they quote their report precisely and then tell us that it is neither what they meant nor what their (unpublished) data show. We would like to thank them for now confirming our result. They also stress the importance of flicker in glaucoma testing. Because we did not test patients with flicker we did not discuss their work beyond commenting on their stimulus. We now feel called upon to do so.

All clinical tests have a dual role. Those which rapidly and efficiently discriminate between affected and normal individuals come into general clinical use even when evaluated on purely pragmatic criteria. Others are normally used experimentally to tell us something about the functional or structural abnormality characterising the disorder. Such tests should be as accurate and unambiguous as possible.

We agree that tests with flickering targets can detect glaucoma better than those with static gratings. However, a maximally efficient test target should be placed in the midperiphery and need not be a grating. For example, C. Tyler measured the full *temporal* modulation transfer function for a defocused spot, subtending 4°, centered 20° above the fixation point. He reported hit rates better than 80% and few false alarms.¹ The success of flickering stimuli might reflect retinal organisation in that area, the type, number, or relative density of affected to unaffected ganglion cells, cortical synaptic efficiency, or the saliency of the stimulus. It may simply be that observers, especially naive patients, respond more reliably to flickering targets, where they must repeatedly distinguish between target presence and absence, than to static ones, where they decide only once.

Atkin *et al.*, in all 3 reports, specifically concentrated on the *central* visual field loss in glaucoma patients. They suggested that they had evidence of a specific loss of the Y cell group, mainly because their results with flicker were the only ones which were significant. We believe that we have not misinterpreted them or misunderstood them because of the importance they gave to this interpretation.

However, their theory has little support. Only a small percentage of optic nerve fibres come from Y cells. Their diameter spectrum overlaps that of X cells, so that they are unlikely to be specifically first affected by pressure damage. Although full spectra are still not available, optic fibre damage appears to be general within regions and bundles, not restricted to specific diameter groups.² Finally, X and Y cells may only respond differentially to flicker well above threshold, but not at it.³ With static stimuli we could not show an interaction between contrast sensitivity loss in

glaucoma and spatial frequency. Atkin *et al.* had to average over thresholds in structured and unstructured fields to get any significant effect. They have never shown that their effect is greater with the unstructured field, as it should be if it increases with decreasing spatial frequency. Other reasons for the loss of flicker sensitivity in disease are given above. In their letter, too, Atkin *et al.* present us with a second model of visual loss in glaucoma, which they had not previously advanced, but which is quite general and without implications for spatial or temporal frequency or field specific loss.

In our report⁴ we were concerned to develop a clinically acceptable test of the full contrast sensitivity function which could be used with any patient group. Glaucoma was discussed only as an example. We confined our discussion mainly to the many different patterns of loss which had been reported with static test stimuli.

If Drs. Atkin *et al.* aimed only at developing a maximally effective test of early glaucoma deficit they should have put their stimulus in the midperiphery, where the first detectable damage occurs. If they did want to say something about the underlying structural deficit in central vision in glaucoma, they have not established their point and are not misunderstood. In now stressing the importance of flicker they misrepresent the primary purpose of their work as well as our own.

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Book reviews

Current Management in Ophthalmology. By DOUGLAS D. KOCH, DAVID W. PARKE II, DAVID PATON. Pp. 338. £28.50. Churchill Livingstone: Edinburgh. 1982.

This is an informative, readable, well-researched book, always entertaining, sometimes controversial, but, like any



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