Pattern ERG and glaucomatous visual field defects


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Abstract. In the past five years numerous reports have suggested that ganglion cell function can be tested by means of a specialized form of electroretinography, the so-called pattern electroretinogram (PERG). Because of the important potentials of a ganglion cell test for clinical use this technique has been applied by several investigators to patients with (presumed) ganglion cell dysfunction, especially glaucoma. On grounds of principle we had reason to question whether the reported positive results should be attributed to ganglion cell dysfunction or to other factors such as optical disturbances. We investigated in this study the PERG as a function of visual field loss in glaucoma patients with careful control of optical factors. We did not find changes in PERG as a function of field loss. So either field loss is not related to the mass behaviour of ganglion cells, or ganglion cells are not the prime basis of the PERG. We believe the latter to be true.

Introduction

The notion that electroretinographic responses elicited by pattern stimulation (PERG) are generated by the ganglion cells originates from Maffei and Fiorentini (1981). They experimentally sectioned the optic nerve in the cat and found subsequent regression of the PERG. Since sectioning of the optic nerve leads to retrograde degeneration of the ganglion cells, they concluded that the PERG must be of ganglion cell origin. This would make the PERG an excellent tool for studying the function of the ganglion cells, opening new vistas for the study of glaucoma. In fact several authors have subsequently reported that in glaucoma the PERG may be defective, starting with Fiorentini et al. (1981) for one uniocular case. The same has now been reported for a variety of other diseases affecting the optic nerve or the ganglion cells. Loss of PERG in glaucoma is reported also by May et al. (1982), Bobak et al. (1983), Wanger and Persson (1983), van Lith et al. (1984) and Papst et al. (1984a). The classical large field luminance ERG is reported to be unaffected. This corresponds with the classical notion that the ERG is not affected in glaucoma and also with the new studies by Maffei and associated on animal preparations, as well as with (P)ERG studies on other diseases of the optic nerve or ganglion cells. This independent behaviour of
the PERG as compared to the ERG supports the argument that the PERG is of ganglion cell origin, since the ERG is known to originate from earlier cell types.

On the other hand this independence of PERG from ERG poses a problem with the interpretation of the PERG, since it is on logical grounds highly probable that the PERG of the normal eye is at least partly the direct result of the local ERG's of the constituent elements of the stimulus pattern. An early study of this point was performed by Spekreijse et al. (1973). Just imagine what would be the fate of the ERG if instead of one large field a number of smaller fields of, say, 1 degree were presented. Since the retinal elements generating the ERG are sure to generate a comparable potential under these circumstances — since their receptive fields are not too large — one would expect that the PERG at least contains the sum of these local ERG's. Indeed the results of several authors, comparing the PERG of normal subjects to the luminance ERG recorded from the same retinal area, showed rather close correspondence (Arden and Vaegan, 1983; Korth, 1983; Riemslag et al., 1983; Hess and Baker, 1984; Riemslag et al., 1985). On the other hand some data reported in literature suggest that the PERG cannot in all cases be explained on the basis of the luminance ERG only. For intermediate spatial frequencies (element size around 60') a (relative) enhancement of the PERG is often found. A variable degree of this spatial selectivity of the PERG is seen depending on the stimulus conditions used, ranging from none (Riemslag et al., 1985) through mild (Alden and Vaegan, 1983; Korth, 1983; Sokol et al., 1983) to marked (Hess and Baker, 1984; Korth and Rix, 1984). From these reports it appears that stronger stimulus conditions, as are favoured in the clinic, result in little spatial selectivity of the PERG.

So we may conclude that the clinical PERG is derived for the greater part from the luminance ERG. This is however difficult to reconcile with PERG loss in glaucoma if it is believed that glaucoma does not affect the luminance ERG. Perhaps optical degradation of the retinal image, that can so readily be associated with glaucoma, was the reason for some of the reported PERG losses. It might be essential here to know how well matched the normal control group is to the patient group in age, miotics, occurrence of cataract, refraction and accomodation errors, etc. An easier approach would be to study interocular PERG differences in cases of asymmetric glaucoma. Wanger and Persson (1983) found for 11 unilateral cases systematic loss of PERG in the affected eye. A close look at their data shows, however, that optical factors might also have played a role. The visual acuity of their patients was asymmetric down to hand movements in the glaucoma eye and this correlated with the PERG amplitude. With the relatively small check of 24' the fact that there was no check on the retinal image may have been of special importance.

In this study we eliminated optical factors as far as possible. Patients were selected with strongly asymmetrical vision field loss but no visual acuity loss