Principles and Practice of Clinical Electrophysiology of Vision

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Electrodiagnostic Testing in Malingering and Hysteria

Graham E. Holder

The usual role of electrodiagnostic testing in the diagnosis of nonorganic visual loss is to provide objective evidence of normal retinal and/or intracranial visual pathway function in the presence of subjective reports that suggest otherwise. In some cases it is difficult if not impossible for the electrophysiologist to distinguish between malingering and hysteria; in others there is little doubt. A recent clinical review of functional visual loss discusses the diagnostic distinctions.[16] The term *nonorganic visual loss* may be preferable if volitional aspects are uncertain. In view of the well-recognized value of electrophysiology, there are remarkably few reports in the literature.

The first study appears to be that of Potts and Nagaya,[20] who reported that patients with hysterical amblyopia displayed completely normal foveal visual evoked potentials (VEPs) (0.06-degree flashing red stimulus) whereas strabismic amblyopes showed diminished or absent foveal VEPs. The use of conventional flash stimulation was described by Arden's group,[1] but these authors also warned against the unequivocal acceptance of electrophysiological criteria and cited one case of almost certain hysterical amblyopia in which scotopic flash VEP (FVEP) anomalies were observed. Subsequent reports,[2, 3, 12, 18] although mostly of only a few cases, confirmed the finding of normal FVEPs in nonorganic visual loss. The use of the FVEP in disclosing the nonorganic basis of suspect symptoms following trauma was also described.[9, 17]

However, although the use of a luminance stimulus may be satisfactory in the evaluation of hysterical total blindness, the use of a contrast stimulus is essential to evaluate less severe nonorganic visual deficit. Halliday[10] first described the use of the pattern VEP (PVEP) in hysterical visual loss. Normal, symmetrical PVEPs were recorded from both eyes in patients with markedly asymmetrical visual acuities. The technique was thought to be most useful with unilateral visual loss where the good eye acts as a control. He also stressed that a normal PVEP, although strongly suggesting nonorganic visual loss, does not preclude the existence of some organic disease.

The early studies using flashed PVEPs[13, 14, 21] found that the maximum response amplitude occurred with check sizes of 10 to 30 minutes. More recent studies with PVEPs have come to similar conclusions for small fields and also suggested an interrelationship between check size and field size[8, 19, 28]; small checks and fields are optimal for foveal stimulation, large checks and large fields for the more peripheral retina. The diagnosis of nonorganic visual loss would be facilitated if a simple relationship between VEP measurements and visual acuity existed so as to enable an accurate objective assessment of acuity. Unfortunately, most scaling methods perform poorly as predictors of acuity.[7] Halliday and McDonald[11] suggested that a well-formed PVEP is incompatible with an acuity of some 6/36 or less.

Despite these problems, there have been attempts to objectively assess acuity in patients suspected of nonorganic visual loss. Wildberger[27] reported the findings in two groups of patients: 17 "malingerers" (mostly schoolgirls) with no signs of an organic lesion and 10 patients (accident or disease) with signs

but marked overlay. The findings in the patients were compared with those obtained in normal subjects to the same four check sizes in relation to insertion of graded orthoptic filters intended to reduce the acuity. The VEPs were easily able to detect malingering in the second group of patients, where acuities were usually claimed to be markedly reduced, but were not sensitive in the first group with milder claimed reductions. The VEP was assessed with the offset amplitude of the P100 component; in our laboratory the onset amplitude may be more sensitive—both should routinely be measured.

Technical factors in the recording of patients suspected of hysteria or malingering are of paramount importance but have received little attention. The patient may fail to fixate, may attempt to defocus, may attempt prolonged eye closure during blinking, etc. PVEP changes have been reported under such conditions.[5, 24] Direct observation of the patient, with the patient aware of such observation, will often result in improved compliance (Fig 72–1). Careful observation of both the raw electroencephalographic (EEG) input and also the developing average is advisable. The appearance of an alpha rhythm in the ongoing EEG may indicate a failure in concentration; in the acquired average, a tendency of the P100 component to broaden or increase in latency will also suggest that accommodation or fixation is unsatisfactory. Verbal commands to the patient to concentrate and attend to the fixation mark (or the center of the screen if perception of the fixation mark is denied) may be beneficial. If all perception is denied with one eye, fixation can be obtained with the good eye, and an instruction to "try and keep your eyes still" will surprisingly often produce good results following occlusion of this eye. It may be necessary to stop averaging after fewer sweeps than usual to prevent waveform deterioration. A tendency for the P100 component to sharpen and remain of stable or slightly reducing latency during acquisition of the average will only occur with good patient compliance. The use of pattern-onset stimulation rather than the more commonly used reversal may sometimes yield improved results (Arden, personal communication). In all cases a subjective report of stimulus perception should be obtained from the patient. Marked discrepancies between the subjective reports and the objective electrophysiological findings can be useful in alerting the examiner to nonorganic visual loss, particularly when marked interocular per-

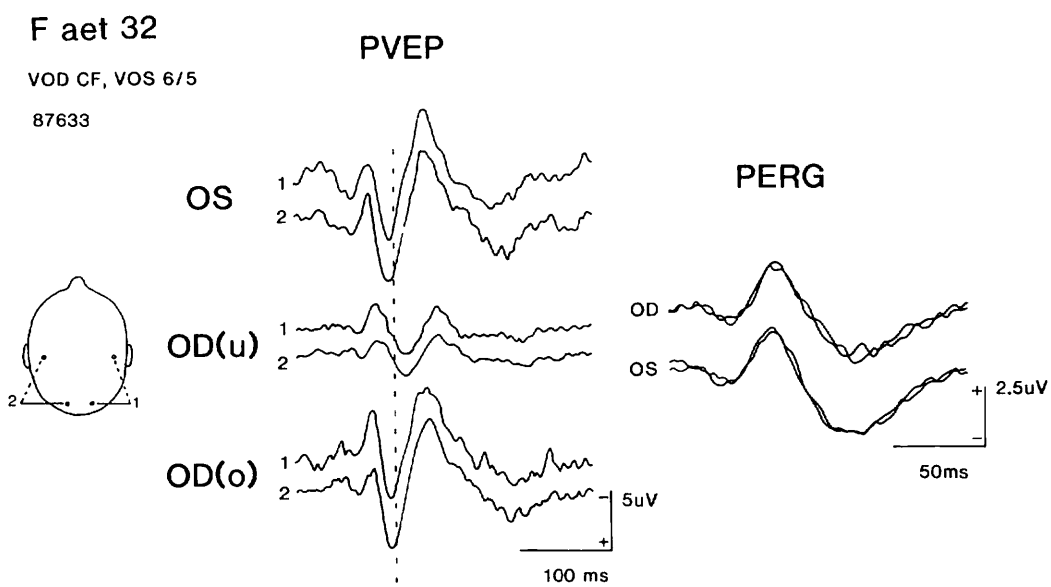

FIG 72–1.
PVEP and pattern electroretinographic (PERG) findings in a woman with nonorganic visual loss in the right eye; note the severely reduced subjective acuity in this eye. Left eye findings are normal. When the right eye was stimulated without observation of the patient *[OD(u)]* the PVEP was of abnormal latency and reduced amplitude. When the patient was aware that she was being directly observed and commands were given to modify the direction of gaze if she appeared to fixate away from the screen, the right eye PVEP was normal *[OD(o)]*, and there was no significant interocular asymmetry. Binocular registration is used for the PERGs that are bilaterally normal and show no interocular asymmetry in either P50 or N95. Fixation under these circumstances is maintained by the "good" eye. The *dotted vertical line* indicates the upper limit of normal PVEP latency (mean + 3 SD).

ceptual asymmetries are unaccompanied by PVEP asymmetries.

PERG recording must also be performed. The PERG is very susceptible to deterioration with poor compliance, and a normal PERG can only be obtained with satisfactory fixation, accommodation, etc. The effects of defocus, etc., on the normal PERG are fully discussed elsewhere in this volume (see Chapter 38). Equally, this author has observed cases of mild maculopathy where the PVEP has fallen within the normal range but the PERG was unequivocally abnormal; a normal PVEP cannot necessarily be assumed to preclude mild anterior visual pathway dysfunction. Rover and Bach[22] have successfully used simultaneous recording of the PERG and PVEP to reveal malingering. Their patients were complaining of marked acuity loss, but only a few representative cases were discussed and no quantitative patient data presented. In particular, no mention was made as to whether their cases were bilateral or unilateral. A normal PERG and PVEP indicated malingering, a normal PERG and an abnormal PVEP indicated a "lesion of the visual pathway," and an abnormal PERG and an abnormal PVEP indicated blurred image, poor cooperation, or retinal dysfunction.

In this author's experience simultaneous PERG and PVEP can be useful, but in unilateral cases binocular registration of the PERG, with the "good" eye fixating, will usually reveal whether the PERG from the "bad" eye is abnormal or not (see Fig 72–1). Significant macular dysfunction is excluded if the PERG from the "bad" eye is normal with binocular stimulation, and a unilateral PERG abnormality from the bad eye will indicate either optic nerve or more distal dysfunction depending on whether the N95 or P50 component of the PERG is affected.[15, 23] Knowledge of the PERG from the "bad" eye thus obtained can then be used to more meaningfully evaluate simultaneous PERG and PVEP if poor patient compliance is suspected during original PVEP recording. Routine ERG should also be performed; ERGs can be markedly abnormal in retinal dystrophies with no or minimal ophthalmoscopic change but constricted visual fields, a common symptom in nonorganic visual loss. PERG and PVEP findings may be normal in such conditions if the maculae are spared.

However, because normal PVEPs may be found in patients with cortical blindness, great care should be taken in making the diagnosis of nonorganic visual loss if the symptoms suggest that cortical dysfunction may be responsible. Celesia's group[6] studied a 72-year-old woman with bilateral destruction of area 17 and attributed the presence of normal VEPs to conduction in extrageniculocalcarine pathways. Bodis-Wollner et al.[4] reported normal VEPs in a

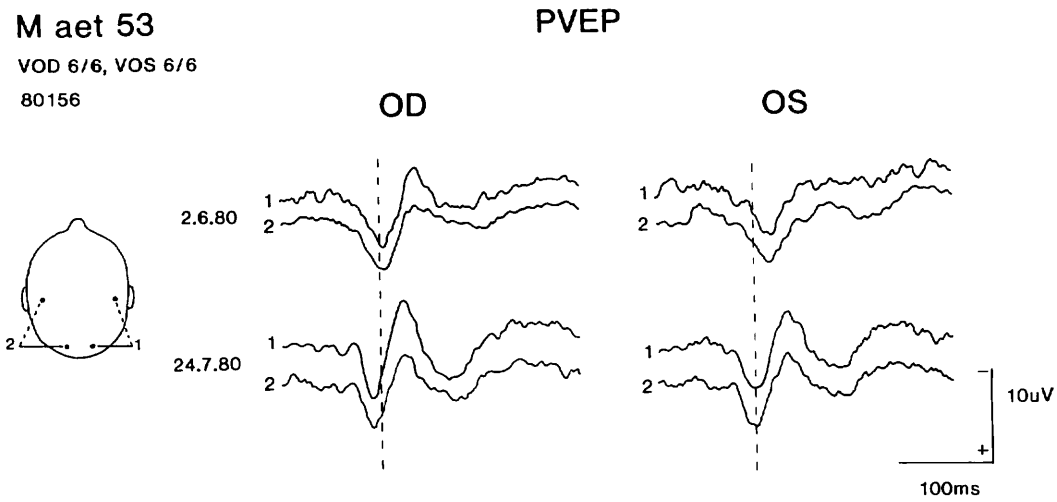

FIG 72–2.
PVEP findings in a 53-year-old male alcoholic admitted for detoxification. He complained that his vision was blurred but his acuity was unaffected, and the referring physician felt that his complaints did not have an organic basis. Initial PVEPs (11-degree full field, 26-minute checks, 1.95 reversals per second) show a definitely abnormal P100 latency from the left eye, that from the right being borderline abnormal. A history of excessive cigar smoking was also obtained and a presumed diagnosis made of tobacco-alcohol amblyopia. Treatment was commenced with hydroxocobalamin and the patient instructed to stop tobacco and alcohol consumption. Seven weeks later the right eye PVEP was normal and that from the left borderline had markedly improved. The *dotted vertical line* indicates the upper limit of normal PVEP latency (mean + 3 SD).

6-year-old boy with destruction of areas 18 and 19 but preservation of area 17. Similarly, retrochiasmal lesions may not give a PVEP abnormality even with a demonstrable field defect (see Chapter 71). Evaluation of the P300 component may circumvent such problems.[25]

To conclude, electrodiagnostic testing is invaluable in the detection or confirmation of nonorganic visual loss, particularly if unilateral so that one eye can be judged against the other. The reader is reminded of the warning of Halliday[10] that normal electrophysiology does not preclude the presence of some underlying organic disease. In the author's laboratory, by far the most difficult patients to assess are those with marked overlay superimposed on genuine dysfunction. Particular caution must be exercised if there is a possibility of cortical dysfunction. It is essential that an accurate history be taken and comprehensive ophthalmic/neurological examination performed. Particular techniques for revealing functional deficit are described in full elsewhere.[26] Electrophysiological examination is always advisable if there is *any* doubt; the objective nature of electrodiagnostic testing may not only demonstrate normal visual pathway function in patients whose symptoms suggest otherwise but may also reveal the presence of organic dysfunction in a patient with a presumed diagnosis of nonorganic visual loss (Fig 72–2).

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