Principles and Practice of Clinical Electrophysiology of Vision

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Chiasmal and Retrochiasmal Lesions

Graham E. Holder

CHIASMAL LESIONS

The principal cause of chiasmal dysfunction is a pituitary tumor, the anatomical relationship between the chiasm and the pituitary fossa making the chiasm readily susceptible to compression by lesions expanding from the pituitary fossa. Other tumors, aneurysms, inflammation, demyelination, and trauma can also affect the chiasm. Although often not present, a bitemporal hemianopia is the classic visual field defect due to a disturbance of the decussating fibers from the nasal retinas. This readily enables the use of hemifield stimulation in the visual evoked potential (VEP) assessment of chiasmal dysfunction. A large stimulating field is necessary to obtain reliable results with this type of stimulation, which is probably the most sensitive for detecting early chiasmal involvement (see below). However, patients with reduced visual acuity sometimes have difficulty in maintaining accurate fixation, and it may not be possible to perform hemifield stimulation adequately in all patients. Full-field stimulation can also give satisfactory localization of chiasmal lesions, but registration parameters must be given adequate consideration (see below). It is essential that an assessment of chiasmal function not be attempted with a single midline channel; multichannel recording must be used.

Prior to a discussion of the electrophysiological findings, it is important that the reader be familiar with the results of hemifield pattern stimulation in normal individuals. It should be remembered that use of a large stimulating field, e.g., greater than a 12-degree radius, will give the normal P100 component of the pattern-reversal VEP ipsilateral to the stimulated hemifield, the "paradoxical" lateralization of Halliday's group.[4] There is a contralateral P135 component. However, the P100 becomes contralateral with a progressive reduction in field size,[25] a widely spaced bipolar montage showing little difference from common reference recording.

Following the initial report by Muller[40] that the flash VEP (FVEP) could be of abnormal latency in chiasmal dysfunction, other workers[17, 31, 35, 52] noted that the maximum FVEP abnormality was localized contralateral to the visual field defect. The first reports using contrast stimuli appeared in 1976. Van Lith's group[54] used both full- and hemifield steady-state (8 Hz) stimulation in six patients with bitemporal hemianopia due to tumor and found both phase and amplitude abnormalities contralateral to the stimulated eye.

The first detailed report of transient pattern VEP (PVEP) was that of Halliday's group.[23] Using a 16-degree radius, 50-minute check stimulus, they found markedly asymmetrical scalp distribution in ten patients with chiasmal dysfunction. In particular, they described the "crossed" asymmetry typical of chiasmal lesions where the findings from one eye are more abnormal over one hemisphere but the findings from the fellow eye are more abnormal over the other hemisphere. Unexpectedly, the maximum abnormality was localized ipsilateral to the visual field defect, i.e., the "paradoxical" lateralization referred to above (Fig 70–1). They noted that PVEP abnormalities could be recorded from eyes with normal fields. Comment was also made that the findings differed from demyelination where preservation of waveform, a generally greater latency delay, and symmetry across the scalp were much more frequent observations. The use of hemifield stimulation was further elaborated in another publication by the

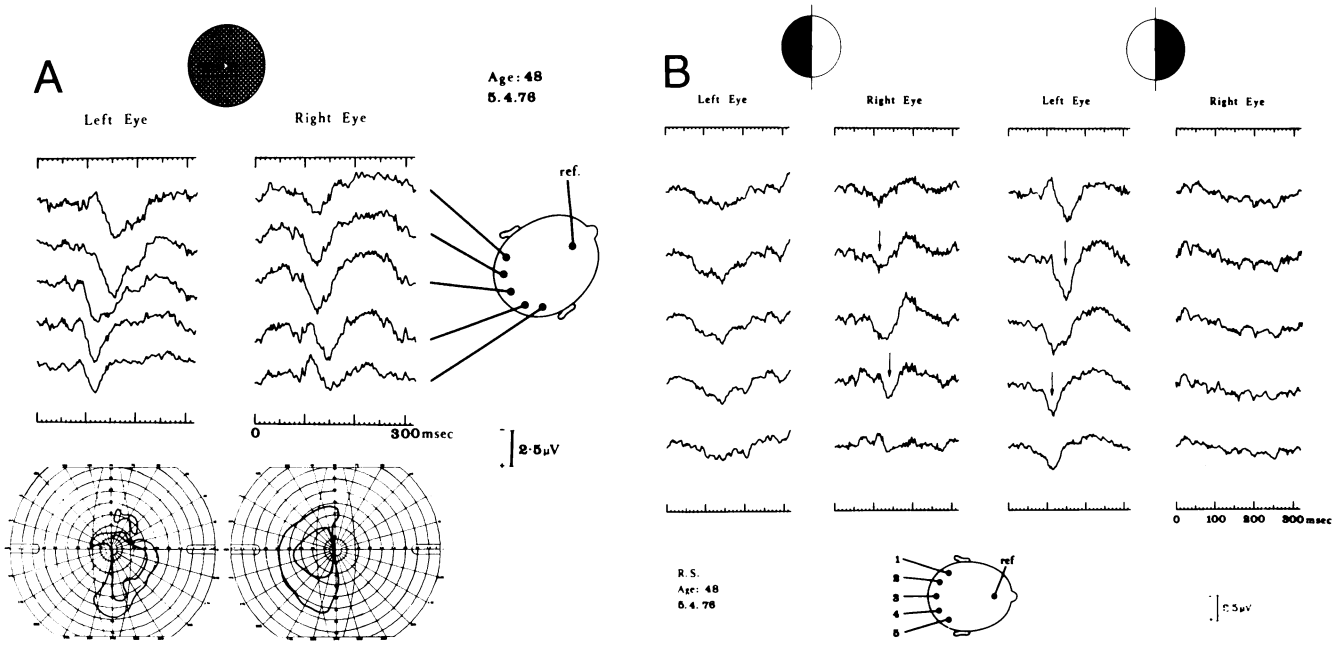

FIG 70–1.

A, crossed PVEP asymmetry in a 48-year-old man with bitemporal hemianopia from a suprasellar mass (16-degree radius, 50-minute checks). The P100 component is seen ipsilateral to the preserved nasal field from each eye. Note the later positivity recorded from the two channels on the other side of the head. **B,** hemifield stimulation in the same patient shows that the major features of the full-field response are similar to stimulation of the nasal half field of each eye. In particular, the later contralateral positivity is shown to be the contralateral P135 component of the half-field response. (From Halliday AM, Barrett G, Blumhardt LD, et al: The macular and paramacular subcomponents of the pattern evoked response, in Lehmann D, Callaway E (eds): *Human Evoked Potentials. Applications and Problems.* New York, Plenum Publishing Corp, 1975, pp 135–151. Used by permission.)

same group.[6] Holder[29] confirmed this "crossed" asymmetry in ten patients, but when using full-field stimulation (5.5-degree radius, 26-minute checks, bipolar recording), the PVEP abnormality was always contralateral to the field defect (Fig 70–2). Although apparently contradictory, these findings are in fact consistent with those of Halliday's group,[6, 23] the alternate abnormality lateralization reflecting the use of a smaller stimulating field/check size (see above). The abnormality lateralization was enhanced with a 4-degree radius, 13-minute check stimulus. It was confirmed that the asymmetrical scalp distribution was atypical for demyelination and that abnormal VEPs could occur in eyes with full visual fields. Equally, normal PVEPs could occur in eyes with field defects. Latency delays were a frequent occurrence. These findings have recently been extended[30] in a study of 34 patients with histologically confirmed nonfunctioning chromophobe adenomas. The PVEP results were compared with clinical, radiological, and surgical findings. There were four eyes with normal PVEPs: one had a full field, one had a paracentral scotoma, and two had superior temporal quadrant defects. It is of interest that FVEPs in the latter two eyes were abnormal. Full fields but abnormal PVEPs occurred in two eyes. The PVEPs often indicated marked functional asymmetry when the computed tomographic (CT) scan suggested symmetrical midline suprasellar extension. The PVEPs were usually more sensitive than the conventional clinical tests of visual acuity and visual fields.

A number of other studies have reported PVEP findings in chiasmal dysfunction,[11, 18, 21, 22, 38, 41, 49, 51] mostly (those using multichannel recording techniques) confirming the "crossed" PVEP asymmetry to be pathognomonic of chiasmal dysfunction but describing clinical and electrophysiological findings in varying degrees of detail. Gott and her colleagues[21] examined 83 patients with tomographically demonstrated pituitary tumors. Most were intrasellar and had normal fields and PVEPs. Suprasellar extension was radiologically demonstrated in 12 cases, all of which had abnormal PVEPs, 8 with normal fields. The abnormality was usually an increased P100 latency, but asymmetrical scalp amplitude dis-

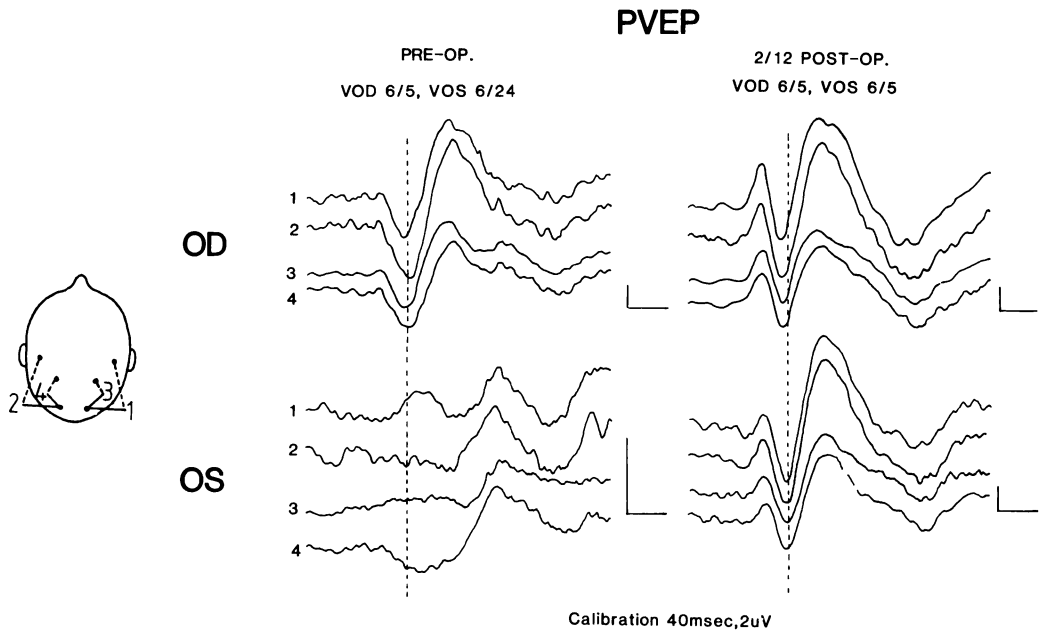

FIG 70–2.
Preoperative and postoperative findings in a 38-year-old male with a nonfunctioning chromophobe adenoma illustrate the use of small full-field stimulation. The preoperative right eye findings are of abnormal latency in the left hemisphere traces, but the right hemisphere traces fall within the normal range. The preoperative left eye findings are grossly abnormal over both hemispheres, possibly worse on the right hemisphere. The findings indicate left optic nerve and chiasmal dysfunction. Note that the use of a small-field, small-check stimulus does not give "paradoxical" lateralization. The postoperative right eye findings are normal with no significant interhemispheric asymmetry. The postoperative left eye findings show a marked improvement; the interhemispheric asymmetry has resolved, and the P100 component latency falls just within the normal range. The magnitude of the interocular latency asymmetry does, however, fall 3 SD outside the normal range, thus suggesting mild residual left optic nerve dysfunction. The *dotted vertical line* indicates the upper limit of normal latency. Positivity is downward. Note the differences in calibration. (From Holder GE, Bullock PR: *J Neurol Neurosurg Psychiatry* 1989; 52:31–37. Used by permission.)

tribution was also observed (22-degree full field, abnormality ipsilateral to the field defect). The ability of the PVEP to influence management was noted by Stark and Lenton,[49] who cite one case with a radiologically confirmed pituitary tumor but totally unreliable clinical testing where an abnormal PVEP prompted surgical intervention.

Haimovic and Pedley[22] found a delayed P100 (19 × 13.5-degree hemifield, 31-minute checks, abnormality ipsilateral to the field defect) in 1 of 15 patients with hemifield stimulation but in 4 patients when using full-field stimulation; this illustrates the possible difficulties in component identification with large-field stimuli which can lead to spurious "delays." This point has been forcefully argued by Blumhardt.[5] Maitland et al.[38] felt that the VEP was not a suitable means of detecting subtle field defects following a study of eight patients (5-degree hemifield, 50-minute checks; two patients normal, four with ipsilateral abnormality, two with no lateralization), but this may be related to the use of inappropriate stimulus parameters. The two patients with normal PVEPs were presumably postoperative because the visual field defects had "resolved." There was, however, subjective desaturation to red. Flanagan and Harding[18] carefully examined the effects of various stimulus parameters in nine patients with pituitary tumor and concluded that hemifield stimulation with a large-check, large-field stimulus is best at detecting early chiasmal dysfunction.

With the introduction of bromocriptine therapy for pituitary lesions, there is a need for sensitive, objective assessment of chiasmal function. Wass et al. [54] first described PVEP improvement during bromocriptine therapy in patients with large pituitary tumours, but did not give full details. Pullan and colleagues[44] examined hemifield PVEPs in five nonfunctioning and five functioning tumors before and after bromocriptine treatment. Suprasellar extension on CT scan was a criterion for patient selection. All patients with radiological evidence of tumor shrinkage showed PVEP improvement, as well as one patient without radiological change. In our laboratory (unpublished data) we have also monitored patients

with intrasellar lesions. Changes in the VEP may be the first indicator of functional involvement of the chiasm; these may precede field loss and suggest a change from medical to surgical management.

RETROCHIASMAL LESIONS

Unilateral Dysfunction

The typical VEP appearance in unilateral retrochiasmal dysfunction is an "uncrossed" asymmetry where there is an abnormal scalp distribution that is similar for each eye. The comments in the previous section regarding the influence of registration parameters on PVEP abnormality lateralization and component identification are equally applicable to retrochiasmal dysfunction and should be borne in mind when considering the findings. Although there are many reports of VEP changes, the development of improved neuroradiological techniques such as high-resolution CT scanning and magnetic resonance imaging (MRI) has greatly reduced any role that electrodiagnostic evaluation may have played in the diagnosis and management of these patients.

The PVEP is more sensitive than the FVEP in most conditions but needs a cooperative patient able and willing to fixate and concentrate. If this is not possible, the FVEP may give useful information. Equally, the two techniques can provide complementary information about the intracranial visual pathways (Fig 70–3). A brief review of FVEP reports is therefore presented.

There is agreement among FVEP studies that any abnormality detected is lateralized to the side of the lesion (contralateral to the field defect) in unilateral hemisphere dysfunction, most differences relating to the incidence of abnormality in relation to the visual field defect. After summarizing the results of a number of studies with unilateral lesions,[20, 31–33, 42, 52, 53] it is clear that some 70% to 75% of patients with homonymous hemianopic defects have abnormal FVEPs, an abnormality being more likely in complete homonymous hemianopia than in quadrantanopia. Some patients have FVEP abnormalities with lesions that do not produce a field defect.[15, 32, 42] Abnormalities have also been reported to occur ipsilateral to the lesion with flashed pattern stimulation.[46]

The first report of contrast stimulation is that of Regan and Heron.[45] By using a technique involving Fourier analysis they found that the response to sine wave–modulated light was reduced but that to pattern stimulation was normal in a patient with a macular-sparing homonymous hemianopia. Wildberger et al.[55] studied steady-state VEPs, both full- and hemifield, in six patients with homonymous hemianopia and found abnormalities contralateral to the

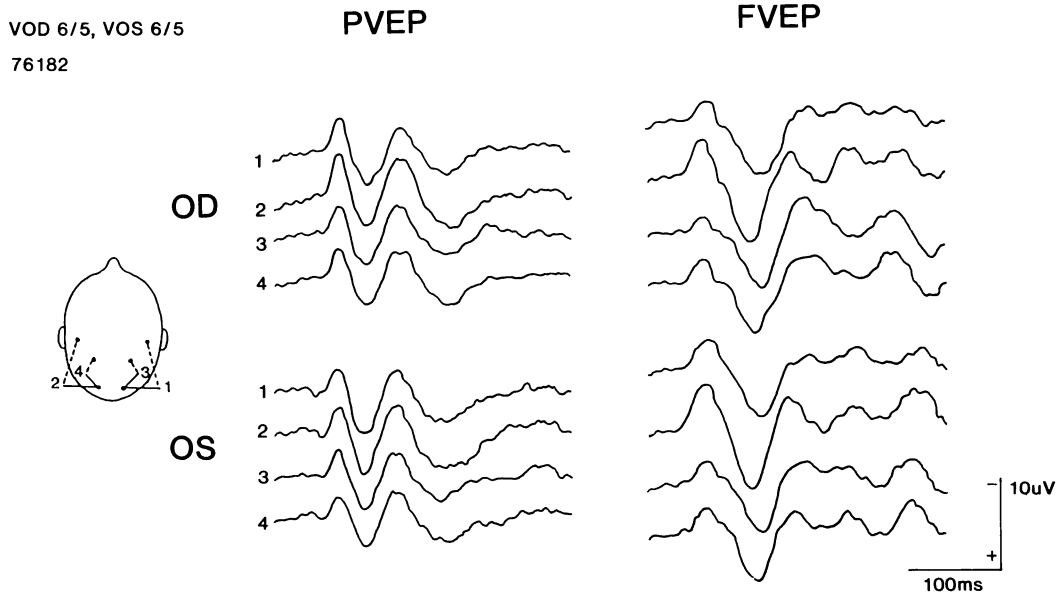

FIG 70–3.
PVEP (11-degree full field, 26-minute checks, 1.95 reversals per second) and FVEP in a patient with a macular-sparing, left homonymous hemianopia. CT showed right occipital infarction. PVEPs are normal, but FVEPs show a significant increase in major positive-component latency ipsilateral to the lesion.

field defect but no difference between those with and without macular involvement. Halliday's group[6] described the typical "uncrossed" asymmetry in homonymous hemianopia. When using full-field stimulation (50-minute checks, 16-degree radius), they found a markedly asymmetrical scalp distribution, the normal P100 component being recorded only over the damaged hemisphere, which is in keeping with the "paradoxical lateralization" originally described by the same group.[4] Hemifield stimulation confirmed that the responses obtained with the full-field stimulus were due to preservation of the normal responses from the residual hemifield. Holder[27] confirmed the "uncrossed" asymmetry in homonymous hemianopia but found the abnormality ipsilateral to the lesion when using small full-field stimulation (26- or 13-minute checks, 5.5- or 4-degree radius, see Fig 70–4). Although the cause of some controversy at the time, the apparently contradictory findings reflect the different registration parameters[24, 25] (see the previous section), in particular, the size of the stimulating field. The lateralization of Halliday's group was also demonstrated in a patient following unilateral occipital lobectomy for glioma by using similar techniques. Subsequent reports[7, 8, 11, 13, 14, 22, 26, 28, 36–38, 41, 50] have confirmed the "uncrossed" asymmetry in retrochiasmal dysfunction, the main conclusions being that hemifield stimulation is more sensitive than full-field stimulation[13, 22, 36, 41]; that ear reference recording is unsatisfactory[26]; and that, in general, the more severe the extent of the hemianopic defect, the more likely the PVEP to be abnormal. Normal PVEPs will often be found in quadrantic field defects,[7, 13, 22, 28] but a dense, macular-splitting hemianopia can be expected to give an abnormal PVEP. The percentage of abnormal PVEPs in the presence of known field defects is in the region of 80% to 90%. Latency delays may be found, even with half-field stimulation, in up to 25% of cases,[36] but they do not approach the magnitude of those regularly seen in anterior visual pathway dysfunction. The problems of accurate component identification in the assessment of "delays" are again noted.

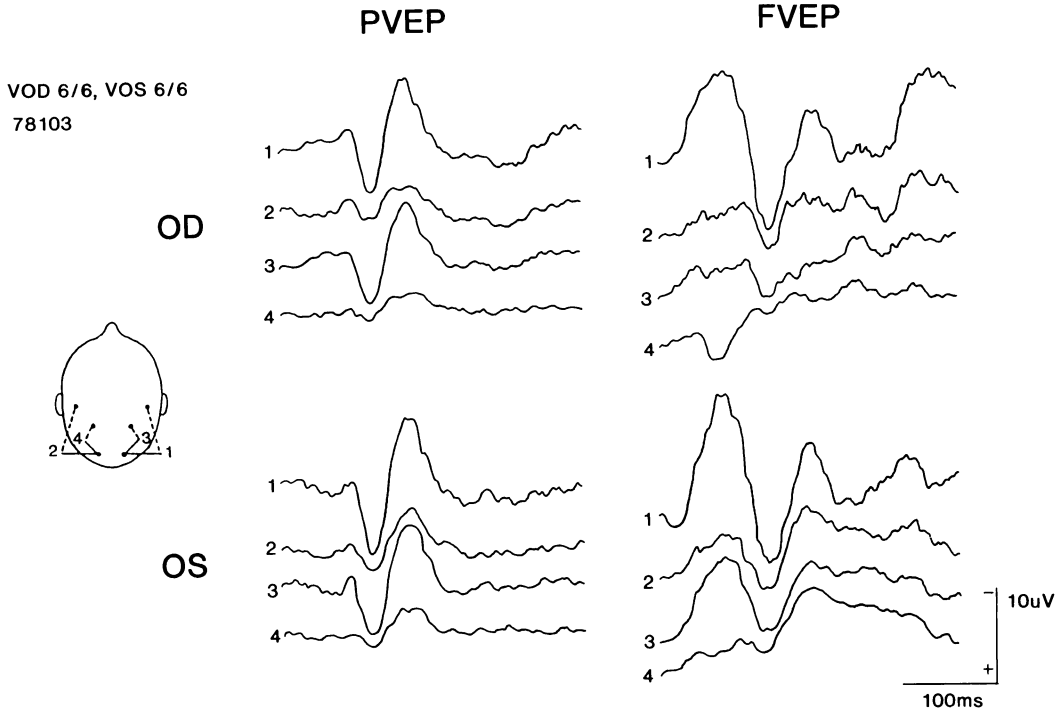

FIG 70–4.
PVEP (11-degree full field, 26-minute checks, 1.95 reversals per second) and FVEP in a patient with a macular-splitting, right homonymous hemianopia. CT showed left occipital infarction. PVEPs show an "uncrossed" asymmetry, with the recordings from both the right and left eyes showing significant amplitude reduction over the affected hemisphere. Left-hemisphere FVEPs also show significant changes when compared with the corresponding traces from the right hemisphere. Note that this PVEP lateralization is not that obtained with a large-field, large-check stimulus when the abnormality would be localized contralateral to the side of the lesion (calibration 10 μV, 100 ms).

A particularly interesting report recently appeared that examined both P100 and the late P3 component in four patients with homonymous hemianopia, including one with clinical evidence of "blindsight."[47] The P3 was well formed to target stimuli in the preserved field for all four patients, but it was additionally present for target stimuli in the hemianopic field of the patient with "blindsight." In contrast, the P100 component could only be recorded with stimulation of the preserved field in this patient. The authors suggested that cognitive processing could occur in the absence of subjective perception, presumably via a mechanism independent of the geniculostriate pathway.

Bilateral Dysfunction and Cortical Blindness

Bilateral occipital lobe disease will result in bilateral homonymous hemianopic defects of variable severity; in its most severe form there is complete cortical blindness. The patient may, however, deny blindness (Anton's syndrome).

There are few reports of PVEPs in patients with bilateral occipital infarction. Streletz et al.[50] describe the PVEPs in two cases as being of grossly abnormal waveform. Blumhardt et al.[7] cite two cases, one with low-amplitude PVEPs of normal latency. Three personal cases [27] all showed amplitude reductions, with latency changes seen in two of the three patients. Subsequent experience suggests that reduced amplitude responses are usually seen but that waveform abnormalities or mild latency changes can also occur depending on the degree of visual field preservation. A recent report[2] describes PVEP findings in nine cases, some with bilateral occipital infarction. No response was seen in five cases, an increased latency in two, and normal findings in two. These findings correlated poorly with outcome. FVEPs were studied in ten patients, some with bilateral occipital infarction; these also showed poor correlation with outcome.

There are fewer reports of PVEPs in complete cortical blindness. Bodis-Wollner's group[9] report the case of a 6-year-old boy with normal PVEPs to high-contrast gratings and preservation of area 17 but destruction of areas 18 and 19 (partial in one hemisphere, complete in the other). Celesia's group[12] describes one patient with normal PVEPs and bilateral destruction of area 17, with preservation of areas 18 (partial) and 19. They postulated that the PVEPs were mediated by extrageniculocalcarine pathways. A more recent report describes a case with complete cortical blindness and normal-latency PVEPs.[2] Bodis-Wollner and Mylin[10] studied two patients during recovery from cortical blindness with both monocular (gratings) and binocular (random-dot correlograms) VEPs. The recovery of binocular vision occurred later than that of monocular vision. One further case is reported[43] in which 1-degree, 20-minute checks "sometimes" gave a response over one hemisphere 2 to 4 months following cortical blindness, but the exact nature of the stimulus is not defined and may be flashed pattern.

Many more cases have been investigated using flash stimulation. Preserved FVEPs in cortical blindness have been described by some authors in adults,[1, 2, 34, 48] one group[1] concluding that the FVEP was of prognostic value in basilar artery occlusion. Others have examined childhood cortical blindness.[3, 16, 19, 39] In one series of 30 children[19] only 1 child with cortical blindness had extinguished FVEPs; some others had abnormal FVEPs but normal vision. The VEP was noted to be a poor method for diagnosing cortical blindness in children.

Electrophysiological examination is therefore of limited value in the clinical management of patients with retrochiasmal dysfunction, particularly with the advent of high-resolution CT and MRI scanning. However, the functional assessment provided by careful serial VEP recording can be valuable in the objective monitoring of disease progression or resolution, and may add considerably to the clinician's understanding. Also, as a recent report[47] demonstrates, valuable information can be obtained by using electrophysiology as a research tool in the investigation of higher visual function.

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