Principles and Practice of Clinical Electrophysiology of Vision

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Parkinsonism

Irene Gottlob

Parkinson's disease (PD) is generally believed to be caused by a dopamine deficiency in the nigrostrial pathway that produces motor dysfunction. However, there is evidence that, in addition, widespread areas of the brain are affected[2] and that the disorder is generalized.[1] Therefore not only motor but also sensory pathways are involved. In the human retina a high dopamine density has been detected in amacrine and interplexiform cells,[6, 13] and it is conceivable that other sites of the visual system also contain this neurotransmitter.[8] Thus in patients with PD, various abnormalities in the visual system have been found.

VISUAL EVOKED POTENTIALS

Bodis-Wollner and Yahr found in 1978 that patients with PD had abnormal visual evoked potentials (VEPs).[3] Since that time several studies have been conducted on VEPs of PD patients. A significant delay of the major positive component (P100) of the VEP has been found. The changes seem to be stimulus dependent. When using checkerboard stimuli controversial results have been reported,[3–5, 9, 11, 14–16] while sine wave gratings produce a high diagnostic yield of abnormalities.[24] The VEP changes are contrast[15] and temporal frequency dependent, becoming more pronounced at lower stimulation rates.[21] Figure 114–1 shows the VEP of a PD patient in response to a sinusoidal grating modulated at the rate of 1 Hz; increased VEP latencies, are clearly shown. Furthermore, correlations between the duration and severity of the disease and the VEP latency delay have been found.[5] Abnormal delays in VEP responses in PD patients could be normalized after levodopa or carbidopa treatments.[4]

ELECTRORETINOGRAMS

It has been shown in animal experiments that dopamine agonists and antagonists influence the electroretinogram (ERG).[10, 18, 19] This and the fact that the retina has a high dopamine receptor density caused us to investigate whether changes in the visual system occur at the retinal level. Thus, significantly reduced amplitudes of scotopic b-waves and photopic a- and b-waves and oscillatory potentials were recently found in the flash ERG.[12, 15, 17] Although no consistent latency increase was found in a- and b-waves, a subtle increase was found in the latency of the photopic ERG recorded from the retina ipsilateral from the more symptomatic side of the patients.[17] Figure 114–2 shows an example of the ERG of a patient with PD. The amplitudes of the scotopic and photopic a- and b-waves and of the photopic oscillatory potentials were reduced at all light intensities that were compared with normal subjects. Increases in the amplitudes and reductions in the implicit time of the ERG from patients with PD have been found after the administration of levodopa.[12] A reduction in pattern ERG amplitudes has been reported in monkeys with a drug MPTP (1-methyl 4 phenyl-1,2,3,6-tetra-tetrahydropyridine) that induced a Parkinson-like syndrome[22] and in patients with PD.[15] These findings suggest that in PD retinal dopamine deficiency may be involved and that the changes in VEP are not caused by alterations of the visual cortex alone.

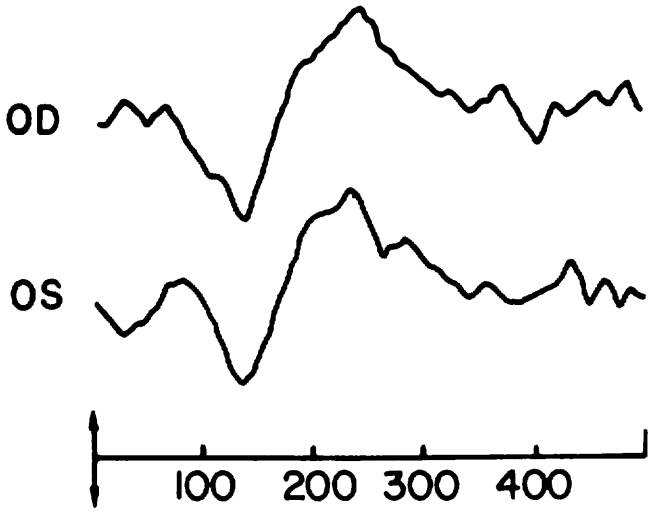

FIG 114–1.
Increased VEP latency in the right eye (*OD*, 140 ms) and the left eye (*OS*, 142 ms) of the major positive wave (downward deflection) of a 75-year-old patient with PD. (From Bodis-Wollner I, Yahr MD: *Brain* 1978; 101:661–671. Used by permission.)

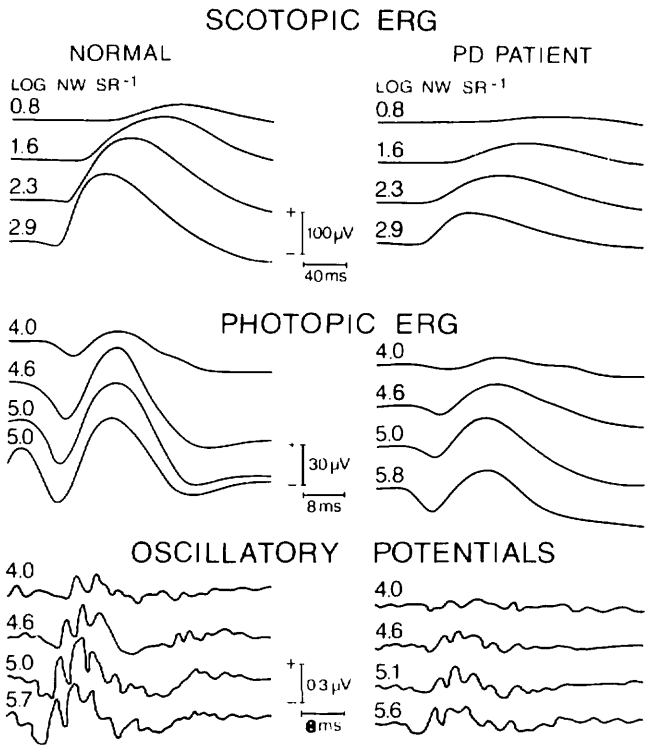

FIG 114–2.
ERG of a normal subject *(left column)* and a patient with PD *(right column)*. The ERGs were recorded at four light intensities as indicated. The oscillatory potentials were filtered with a band pass from 100 to 300 Hz from the photopic ERGs. (From Gottlob I, Schneider E, Heider W, Skrandies W: *Electroencephalalogr Clin Neurophysiol* 1987; 66: 349–357. Used by permission.)

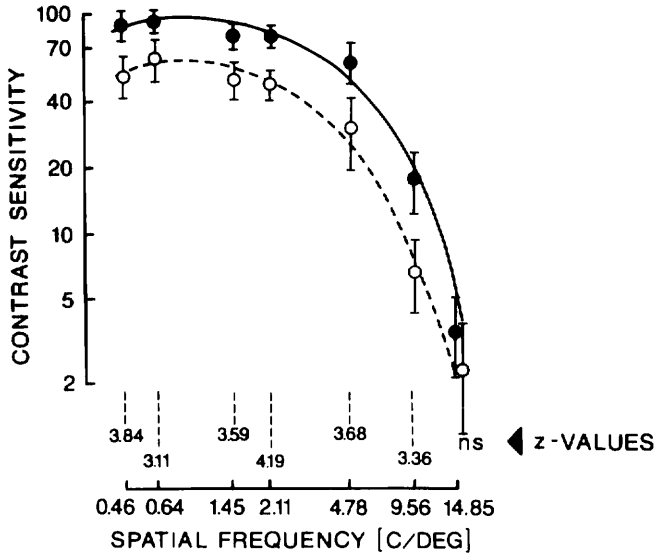

FIG 114–3.
Mean contrast sensitivity curves computed from the data of 18 patients *(open circles)* or the 18 controls *(filled circles)*. *Bars* indicate 2 SE; numbers refer to z-values obtained from Wilcoxon rank tests. The differences in sensitivity are highly significant at all spatial frequencies tested. The nonsignificant finding at 14.85 cycles/degree is due to the small amount of patient data. (From Skrandies W, Gottlob I: *Hum Neurobiol* 1986; 5:255–259. Used by permission.)

PSYCHOPHYSICS

Conventional measures of visual acuity generally are normal in patients with PD. In opposition, a determination of the contrast sensitivity function, which permits a more complete assessment of visual sensitivity, shows alterations.[7, 20, 23] While we found a sensitivity decrease at all spatial frequencies (Fig 114–3),[23] Bulens et al. found sensitivity loss at intermediate frequencies in most of their patients.[7] The sensitivity loss is not correlated to the severity of the disease, and no sensitivity differences between patients with or without cerebral atrophy were seen.[23]

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