Matters arising

Visual evoked potentials and pattern electroretinograms in Parkinson's disease and control subjects

Sir: We read with interest the report by Nightingale et al on visual evoked potentials (VEPs) and electroretinograms in Parkinson's disease. In that study two checkerboard stimuli, respectively with checks of 25' and 50' angular subtense, over a central field (17' x 14' in size) were presented to 36 patients with idiopathic Parkinson's disease. Inter alia, it was found that “the latency of the P100 and N150 components were not significantly different among the Parkinson's disease patients and control subjects at either check size”. This result contributes to the controversy on the existence of VEP latency abnormalities in Parkinson's disease. In fact, while some authors refer to the occurrence of significantly delayed P100 to sine-wave gratings, 2-4 depending on spatial frequency, 4 others did not find changes in the latency of P100, generally to checkerboard presentation. 5-7

Nightingale et al quoting our suggestion 8 that checkerboard might be less effective than grating to identify visual changes due to Parkinson's disease, pointed out that our two stimuli differed not only in pattern form but also in angular subtense and retinal field stimulated. In particular, they argued that under these conditions it would be very difficult to establish whether the two pattern forms have a different diagnostic yield per se.

It should be noted that our goal was to demonstrate that the possibility of obtaining a delayed P100 in a Parkinsonian patient is contingent upon the stimulus employed. We were so aware of the experimental limitations indicated by Nightingale et al as to state that our data did not “presume to answer the general question as to whether gratings are more effective than checkerboard”. 8 Accordingly, the effectiveness of the two pattern forms was investigated in a further study 9 of which a brief account may be useful here.

Checkerboard (with checks of 55' angular subtense) and grating (with a spatial frequency of 2c/deg) were presented over different retinal areas, that is: (a) central area 10' wide; (b) central area 4' wide; (c) ring area with outer and inner diameters respectively of 10' and 4'. The average luminance was identical in all the stimuli, the dark elements presenting a luminance of 0.6 cd/m² and the light ones 30 cd/m². Stimuli had no light surround. VEPs were evoked monocularly by pattern reversal at a temporal frequency of 2 Hz. The investigation was carried out in nine patients with Parkinson's syndrome and in nine age matched controls; visual acuity was never less than 20/25. All the Parkinsonian patients were on medication with various combinations of drugs. Individual latencies of P100 obtained for each stimulus condition were entered into a mixed design of analysis of variance. VEP latencies of Parkinsonians to checkerboard stimuli (55') did not differ from those of controls, although a tendency for the central 4' stimulus to yield a longer latency in the former group was noted. On the contrary, sine-wave grating yielded significantly delayed P100 in Parkinsonians in all the stimulus conditions. In order to assess the incidence of abnormal P100 latencies in Parkinsonian patients as a function of stimuli, the original data 9 have been re-examined. Latencies were considered abnormal if they outweighed the cut-off value for each stimulus condition, expressed by the mean + 1.83 times the SD (that is 95% of control population determined by using a one tailed t distribution). Abnormal P100 latencies to checkerboard (55') occurred in one out of 18 eyes (5.5%) with either 10' or 4' stimuli and in no instance with the ring stimulus. Abnormal P100 latencies to sine-wave grating were found respectively in three eyes (16.7%) with the 10' stimulus, in nine eyes (50%) with the 4' stimulus and in seven (38.9%) with the ring.

Summing up, in Parkinsonian patients the grating has a greater diagnostic yield than checkerboard, no matter what the angular subtense and the retinal projection of the stimulus. However, foveal stimulation seems to be more effective in identifying the presence of functional damage, irrespective of the stimulus used.

We feel that the low sensitivity of checkerboard in identifying visual changes occurring in Parkinson's disease should be considered when evaluating the results obtained by Nightingale et al. 1 This applies to the 55' element and probably also to 25' element of checkerboard, although in this regard, the presence of a significant interaction of retinal field calls for further analysis. 9

As to the main problem whether Parkinson's disease is associated with electrophysiological signs of visual disfunction, it should be recalled that in multiple sclerosis the occurrence of VEP changes depend on the pattern employed 10-11 so that only the use of different types of stimuli in the same patient enables a satisfactory assessment of visual function. We believe that such approach should be applied also to Parkinson's disease, particularly if one considers that the disorder seems to involve selectively some aspects of visual function, as processing of spatial, 12 and temporal, 13 frequencies and four-verseifoveal relationships. 9 This would allow a better understanding of the visual impairment in Parkinsonians.

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Catatonia secondary to acute Chagas' encephalitis

SIR: We have read the contribution by MP Barnes, et al with great interest. It emphasi-
ses the need to view catatonia as merely a symptom and not a disease, and that full
neurological investigation is warranted to disclose any organic brain lesion. Our recent
experience with a patient suffering from Chagas' (American trypanosomiasis) encephalitis presenting with catatonia, encouraged us to report our findings.

This 25 year old man acutely developed abnormal behaviour and intermittent fever six months after renal transplantation. He had been treated with cyclosporin and methylprednisolone. When the psychiatric symptoms developed, he was on maintenance doses of both drugs and his renal function was normal. At age 12 years, he had been seen by a psychiatrist because of social withdrawal and a diagnosis of schiz-
 oid personality was made. On admission, he lay motionless and mute with an expression-
less face; he would reply occasionally and appropriately in monosyllables when ques-
tioned. He also had catalepsy and main-
tained a stereotyped posture. There were no abnormalities on neurological examination, except for reduced muscle tone. Initially, symptoms were attributed to a psychiatric cause. An EEG showed diffuse slowing. CSF was clear, with a protein content of 1.2 g/l and no abnormalities. The complement fixation (Machado Guerreiro) test for Chagas' disease was negative. A CT scan was normal.

There was progressive clouding of con-
sciousness, leading to stupor and coma within two weeks. A frontal brain biopsy showed necrotic parenchymal tissue with macrophages full of amastigotes positive for specific Trypanosoma cruzi antigen (Sternerberger's PAP Technique), and a few lymphocytes as well as reactive peripheral astrocytes. Despite treatment with Nif-
urtimox (15 mg/kg/day), there was no improvement and he died one month after onset of catatonia.

To the best of our knowledge, this is the first report of catatonia induced by Chagas' disease, and we suggest that it should be included in the list of neurological infections liable to cause catatonia. Chagas' encepha-
litis causes an acute non-supportive encephalomyelitis, with small inflam-
atory foci spread uniformly in the white and grey matter. Acute Chagas' disease is mainly restricted to children, but can occur in immunodeficient adults due to con-
taminated blood transfusion. In our patient, the negative complement fixation test seemed attributable to immunosuppressive treatment.

Since catatonia is often caused by an underlying brain disease, every effort should be made to exclude an organic lesion. A brain biopsy may be the only means of diagnosing a life-threatening condition.

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References


Matters arising

Behavioural manifestations of third ventricular colloid cysts

SIR: Dr Arnold Goran, a Diplomate of the American Board of Neurological Surgery, recently referred me to the section entitled "Matters Arising" in the April 1986 issue of J Neurol Neurosurg Psychiatry. In Dr Winer's reply to Dr Backlund, he makes the statement "unfortunately, in the United States there is difficulty finding a neurosurgical centre which performs CT guided biopsy." I would like to strongly disagree with Dr...