

PAPERS AND SHORT REPORTS

Serial visual evoked potential recordings in Alzheimer's disease

A ORWIN, C E WRIGHT, G F A HARDING, D C ROWAN, E B ROLFE

Abstract

Primary presenile dementia slows the major positive component of the visual evoked potential to flash stimulation but does not affect the visual evoked potential to patterned stimulation. The progressive effect of Alzheimer's disease was followed in a 58 year old woman over three and a half years from the development of the earliest symptoms to complete mental incapacity. The pattern reversal visual evoked potential remained normal, but the flash visual evoked potential gradually slowed from 129 ms in 1981 to 153 ms in 1984. The severity of the abnormality of the flash visual evoked potential thus reflected the severity of the dementia. Electroencephalography, computed tomography, and psychometric tests indicated generalised cortical disease, but the results were not specific to dementia.

The combination of a slowed flash and normal pattern visual evoked potential seems to be specific to Alzheimer's disease and supports the use of flash and pattern visual evoked potentials in routine diagnostic testing for this condition.

Introduction

Primary dementia of the Alzheimer type is a growing problem in our society as life expectancy continues to increase. We have previously reported that primary presenile dementia slows the major positive component of the visual evoked potential to flash stimulation but does not affect the visual evoked potential to patterned stimulation.^{1,2} We present the results of different methods of assessment of a patient with progressive dementia who was followed up from the development of the earliest symptoms to complete mental incapacity.

Woodbourne Clinic, Edgbaston, Birmingham

A ORWIN, FRCPsych, DPM, medical director

Department of Vision Sciences, Aston University, Birmingham B4 7ET

C E WRIGHT, PHD, FBCO, research fellow

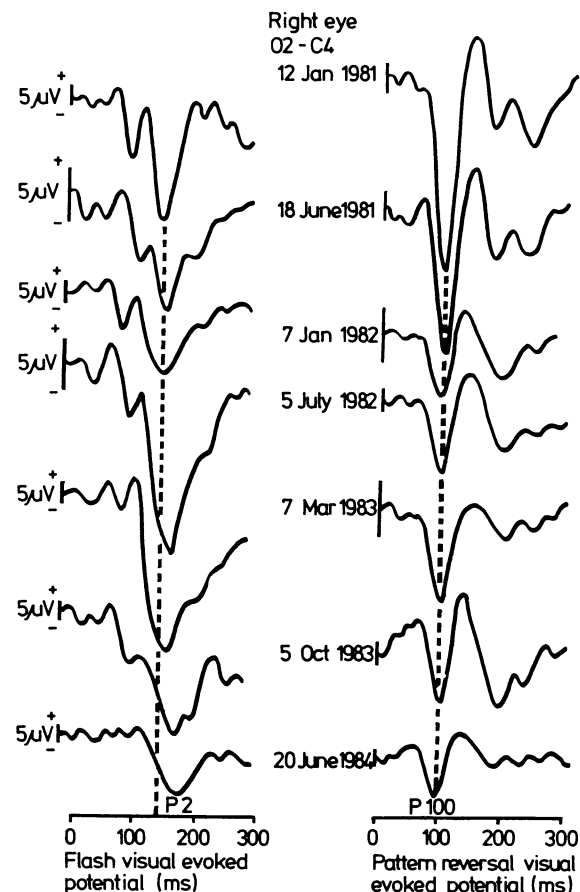
G F A HARDING, PHD, ABPSS, professor of clinical neurophysiology

Midland Nerve Hospital, Edgbaston, Birmingham

D C ROWAN, PHD, ABPSS, principal clinical psychologist

Queen Elizabeth Hospital, Edgbaston, Birmingham

E B ROLFE, MB, FRCR, consultant neuroradiologist



Serial flash and pattern reversal visual evoked potentials over three and a half years in patient with Alzheimer's disease.

Case report

The patient was previously a nursing officer and tutor, and on her first admission her former pupils were able to describe her previous character and help assess the extent of deterioration. She had used her formidable personality to compensate for the early symptoms for about a year. The death of a close friend precipitated symptoms of severe depression, for which

Comparison of results from different methods of assessment

	Clinical assessment	Psychometric assessment	Electroencephalography	Visual evoked potential (milliseconds)	Computed tomography*
January 1981	Occasionally forgetful or confused. Cares for herself		Too slow for age but not typical of primary presenile dementia. Background alpha 8-10 cycles/s, intermittent theta 6-7 cycles/s	Within normal limits Flash P2 129 Pattern 96 Difference 33	Sulci 1 Sylvian fissures 0 Interhemispheric fissure 0 Ventricle: brain 9
January 1982	Memory worse. Relies heavily on timetables and calendars. Went on holiday alone	Organic impairment not specific to primary presenile dementia. WAIS IQ: V=70, P=74, F=70. Kendrick: 0=59, D=80. Benton: score=1, errors=21		Borderline. Flash P2 139 Pattern 100.5 Difference 38.5	
July 1982	Disorientated. Friend who moved in does everything except washing, feeding, and dressing	Deterioration. Organic impairment not specific to primary presenile dementia. WAIS IQ: V=62, P=65, F=61. Kendrick: 0=63, D=69. Benton: score=0, errors=32	Abnormal but not consistent with primary presenile dementia. Very little alpha activity. Large amount of theta at 7 cycles/s	Abnormal, consistent with primary presenile dementia. Flash P2 149 Pattern 100 Difference 49	
August-October 1983	September: can only feed herself. Dependent on friend for everything else, including dressing and cleanliness	August: Rapid deterioration. Confusion and incomprehension. Response could not be scored	October: very abnormal, consistent with primary presenile dementia. Excess theta and some delta activity, especially over posterior regions	October: very abnormal, consistent with primary presenile dementia. Flash P2 161 Pattern 104.5 Difference 56.5	
July-August 1984	Totally inaccessible. Barely continent. Follows friend all the time. Has doll for company		Grossly abnormal, further deterioration. Theta activity dominating entire record	Very abnormal, as before Flash P2 153 Pattern 98 Difference 55	Sulci 2 Sylvian fissures 1 Interhemispheric fissure 1 Ventricle: brain 18.7

WAIS=Wechsler adult intelligence scale: V=verbal, P=performance, F=full score.
Kendrick=Kendrick battery for detection of dementia: O=object learning, D=digit copying.
Benton=Benton visual retention test.

P2=Major positive component.
*Lishman grading scale.⁶

she was admitted in December 1980, aged 58. The depression was successfully treated, but an underlying primary dementia of the Alzheimer type was unmasked. Her memory and intellect progressively deteriorated, but her ability to cope socially was preserved for a year after discharge (table).

We were fortunate in being able to follow the progressive effect of Alzheimer's disease on the visual evoked potential. The visual signals from the brain were recorded between scalp electrodes over the occipital cortex (O2 and O1, international 10-20 system) and ipsilateral central reference electrodes (C4 and C3). The flash stimulus was produced twice a second by a Grass stroboscope (intensity 2, 68 candelas/m²/s). The pattern stimulus was an optically projected checkerboard of 28° diameter containing checks of 56 minutes of arc in 76% contrast that abruptly changed place (or reversed) twice a second.

The unusual combination of a slowed flash visual evoked potential and a normal pattern visual evoked potential in Alzheimer's disease is specified by the difference in latency between the two (where latency is the time in milliseconds from the onset of the stimulus to the peak of the major positive component). The difference in latency in this patient exceeded the upper normal limit of 40 ms for her age group in July 1982 (table).² The figure shows the gradual slowing of the flash visual evoked potential over three and a half years. It is remarkable that, despite this, the pattern reversal visual evoked potential remained normal throughout.

Interestingly, the amplitude of the pattern reversal visual evoked potential decreased with time (figure). The amplitudes of the flash visual evoked potentials varied but showed no consistent trend. Our previous results in patients with primary presenile dementia, however, have shown that dementia has a significant effect only on the latency of the major positive component of the flash visual evoked potential and not on flash or pattern amplitude. The changes in amplitude in this patient are therefore unlikely to have been due to dementia.

Electroencephalograms in patients with Alzheimer's disease have shown a reduction of background alpha activity (8-13 cycles/s) and an increase of slow theta (4-7 cycles/s) and delta (less than 4 cycles/s) activity.³ Electroencephalography in this patient first showed this complete picture in October 1983 (table). Psychometric and radiological investigations (table) both indicated organic impairment but did not confirm a specific diagnosis.

Discussion

We think this case is unique in showing the progressive increase in latency of the major positive component of the flash visual evoked potential from normal to grossly abnormal values. This provides

clear evidence that the severity of the abnormality of the flash visual evoked potential is related to the severity of the dementia. The flash visual evoked potential seemed to mirror the social ability of the patient, the difference in latency reaching abnormal values when she could no longer cope alone (table).

The normal pattern visual evoked potential indicates that the visual pathways up to the primary visual cortex are unaffected and that ophthalmic disease is not influencing the results. The slowed flash major positive component seems to reflect disease of the visual association regions of the brain. Sparing of the primary visual cortex with widespread posterior cortical disease has also been shown in Alzheimer's disease by positron emission tomography⁴ and post-mortem histological studies.⁵

This combination of a slowed flash and normal pattern visual evoked potential seems, therefore, to be specific to Alzheimer's disease. The table shows that electroencephalography, computed tomography, and psychometric tests all indicated generalised cortical disease in this patient, but, as several diseases could have produced the same results, these techniques are non-specific. Our findings provide further support for the use of flash and pattern visual evoked potentials in routine diagnostic testing for Alzheimer's disease.

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