Delayed visual evoked potentials with bilateral disease of the posterior visual pathway

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SUMMARY A man of 51 years presented with sudden onset of a confusional state and marked visual deterioration. The computerised transaxial tomogram showed a mass in the corpus callosum which had spread to involve both cerebral hemispheres. The visual evoked potential to pattern stimulus was delayed by about 20 ms. Necropsy confirmed the presence of an astrocytoma of the corpus callosum involving the optic radiation and occipital cortex on both sides. The anterior visual pathway was normal.

In patients with disease of the optic nerve the visual evoked potential to a pattern stimulus (VEP) often shows increased latency. This test is now widely used in diagnosis and may be abnormal in patients who are symptom-free, and have normal visual acuity, fundi, and visual fields. The delayed response is a common finding in multiple sclerosis (Halliday *et al.*, 1972, 1973).

The VEP is also delayed in eye disease such as glaucoma (Arden, 1973), and in other diseases of the visual pathway. We report here the case of a man aged 51 years who showed increased latency of the response due to an astrocytoma of the corpus callosum which had spread to involve both occipital lobes.

Method

The method was that described by Halliday *et al.* (1972) with minor modifications. An image of a checkerboard slide was back-projected on to a translucent screen via a rotatable mirror controlled by a small electromechanical transducer. The image was rapidly (less than 10 ms) displaced laterally through a distance of one square by rotating the mirror to provide the stimulus. Half a second later the image was rapidly returned to its previous position (commercial apparatus, Digi-

timer Ltd). The occipital electrode was placed in the midline 50 mm above the inion with a reference electrode on one mastoid process. The other mastoid process was connected to earth.

The pattern was viewed by the patient, who was seated in an adjustable chair, from a distance of 850 mm. The screen displays 22×22 checks in a square aperture 140×140 mm. Thus each square subtends a visual angle of approximately 30'' and the entire pattern approximately 10 degrees. One eye was tested at a time, the other being covered by a soft pad. The room was quiet and darkened. Responses to 100 successive stimuli, presented at intervals of one second, were averaged using a general purpose digital computer system. Four trials were made with the eyes stimulated alternately in the order R–L–R–L. The results have been superimposed by the plotter (Fig. 1).

No suitable normative data are at present available for this particular examination in this laboratory, but Halliday (1973) using a very similar apparatus quotes a latency of 103.9 ± 4.5 ms in a group of normal subjects, and this corresponds with our experience.

Case history

The patient was admitted as an emergency because of a change in his mental state. During the previous week he had worked normally as a lorry driver. At the weekend he did some home decorating, but became confused and was unable to recognise what he had done. He had difficulty in

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finding his way about the house, but was unaware of his illness.

When admitted to the ward he was unable to give any history of his condition, but he was alert and speech was normal. Physical examination revealed little. The blood pressure was 160/90 mmHg. There was a left homonymous hemianopia to confrontation tests, but he could not concentrate sufficiently well to allow more detailed recording. Tendon jerks were symmetrical and plantar responses flexor. The remainder of the neurological examination was normal. Psychometric testing revealed poor retention and defective verbal repetition. Visual design reproduction was impaired to a greater extent than could be attributed to the field defect.

Plain radiographs of the chest and skull were normal. The computerised transaxial tomogram showed an area of increased density over the corpus callosum with extension into the right and left parieto-occipital areas. There was no evidence of hydrocephalus.

His condition deteriorated, and several generalised convulsions occurred. He died six weeks after the onset of the psychiatric disturbance.

Results

ELECTROPHYSIOLOGICAL TESTS

The EEG showed alpha rhythm at 9–10 Hz in

spindles of 50 μ V amplitude on the left side, but only traces of similar rhythm on the right. The rhythm was almost completely attenuated by eye opening. During light sleep arousal responses were symmetrical. There was no excess of slow activity or focus of delta waves.

The visual evoked potentials were symmetrical and showed a prominent positive (P^{100}) wave with a latency of 120 ms (Fig. 1).

NECROPSY

There was bronchopneumonia. The heart was normal and the arteries showed only minimal atheroma. A cyst was found in the left adrenal gland. The remainder of the general examination was negative. The brain was diffusely swollen and weighed 1620 g.

Examination of brain after fixation

The cerebral hemispheres were swollen, and there was herniation of the hippocampal gyrus on both sides. Coronal sections showed a partly necrotic, partly cystic, tumour centred on the splenium of the corpus callosum and extending into the medial portion of each occipital lobe. The tumour infiltrated the cortex of the superior margin of the right calcarine fissure and extended forward in the hippocampal gyrus as far as the red nucleus (Figs. 2 and 3).

Histological examination showed a malignant

Fig. 1 Visual evoked potential. Four traces are recorded—two from each eye. Activity seen to the left of zero time was recorded before stimulation began. Latency was measured to the peak of the large positive potential which is directed downwards on the trace.

Fig. 2 Coronal section of occipital lobes. Tumour has spread from the corpus callosum to involve the visual pathway on both sides.

Fig. 3 Coronal section of occipital lobes. There is infiltration of tumour around the visual cortex on both sides and haemorrhage.

astrocytoma. The tumour was highly cellular, pleomorphic, vascular, and, in places, necrotic and haemorrhagic.

Temporal and occipital lobes on both sides showed extensive tumour infiltration more marked on the right. Tumour was found in the white matter around the occipital horns, and had spread to the cortical grey matter in the region of the calcarine sulci.

The optic nerves, chiasma, and optic tracts were normal. The right lateral geniculate body showed slight microglial proliferation. The left lateral geniculate body was normal.

Discussion

The visual evoked potential is commonly delayed and reduced in amplitude when there is disease of the anterior visual pathway. Our patient showed visual disorientation as well as homonymous hemianopia. Necropsy confirmed the presence of diffuse infiltration with tumour in both occipital lobes as well as the optic radiations and temporal lobes. There was no evidence of disease of the eye or anterior visual pathway.

Delay in the VEP has been reported most commonly in patients with demyelinating disease involving the optic nerves. Demyelination of fibres reduces the velocity of nerve conduction. It is possible that synaptic delay and destruction of fibres also contribute to the increased latency.

In our patient there was no evidence of demyelination but the tumour involved the cortex and the optic radiation over lengths of several centimetres on both sides. We suggest that tumour infiltration may interfere with nerve fibre conduction, and this could account for the delay in VEP.

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