survival in elderly patients but enormously enhances the quality of remaining life. How often hypopituitarism is responsible for postural hypotension is unclear, but pointers seen in our patients included low serum thyroxine concentrations without the expected raised thyroid stimulating hormone, hyponatraemia, and disproportionately pale facies.

I am grateful to Dr R J Machell, Penzance, Dr J E Earis, Liverpool, Dr N G Dey, Southport, Dr J Playfer, Liverpool and Dr G Phillips, Liverpool, for permission to report on these patients.

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(*Accepted 19 February 1985*)

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Visual evoked potential and contrast sensitivity function in diabetic retinopathy

Retinopathy is a major complication of diabetes, but clinically obvious optic neuropathy is uncommon. Recent reports of abnormal visual evoked potentials in diabetics without retinopathy have been conflicting.[1 2] We therefore studied the function of the visual pathway using the visual evoked potential and contrast sensitivity function in diabetics with and without retinopathy. In the absence of ocular disease a delayed visual evoked potential indicates abnormal transmission of nerve impulses from the retina to the visual cortex. The contrast sensitivity function is a psychophysical method of detecting subtle disturbances of the visual system such as early glaucoma and lesions of the optic nerve.[3 4]

Subjects, methods, and results

We studied 22 insulin dependent diabetics aged 20-35, of whom five did not have retinopathy, 11 had background retinopathy, and six had proliferative retinopathy. The table shows the clinical details. The three groups were comparable for age and glycosylated haemoglobin concentration. All patients had a corrected visual acuity of 6/9 and J6 or better.

The visual evoked potential was recorded using a standard technique. In each patient 128 responses were averaged and the latency of the major positive peak calculated. The contrast sensitivity function was determined using a standard method based on previous work.[4] All testing was performed uniocularly. Reference values for both tests were obtained in non-diabetic controls matched for age and sex, and results were considered to be abnormal if they exceeded the mean + 2 SD in the control group. Statistical analysis was by the permutation t test.

All patients with proliferative retinopathy showed delayed visual evoked potentials, compared with only one patient without retinopathy ($p < 0.001$) and five with background retinopathy ($p < 0.01$) (table). There was no significant difference, however, between the group with background retinopathy and the group without retinopathy. There were no differences between any of the groups in the prevalence of abnormal contrast sensitivity.

Comment

Although clinically manifest optic neuropathy is uncommon in diabetes, subclinical disease might be relatively common. Our findings show that in the absence of retinopathy there is no significant increase in the proportion of diabetic patients with either an abnormal visual evoked potential or abnormal contrast sensitivity function. Although the range of severity of background retinopathy was wide, this was likewise not associated with abnormalities in these tests. There was, however, a strong correlation between prolifertive retinopathy and an abnormal evoked potential.

Neuronal degeneration in the ganglion cell and layers of nerve fibre is one of the earliest changes in diabetic retinopathy,[5] and presumably patients with proliferative changes have more extensive neuronal and vascular retinal damage than those with background retinopathy. Our findings could reflect either damage to the maculo-papillary fibres in the retina or subclinical optic neuropathy. Our observations are unlikely to have been related to coagulation treatment with argon laser light as none of the patients had undergone this treatment within six months of testing. None had received retrobulbar anaesthesia or sustained vitreous haemorrhages.

These results do not agree with the previous findings of abnormal visual evoked potentials in patients without clinical diabetic retinopathy[1] and abnormalities of contrast sensitivity in patients with minimal retinopathy.[3] Our findings imply subclinical neuronal damage in the visual pathway in diabetes, affecting either the retina or the optic nerve. This seems, however, to be a feature only of patients with proliferative retinopathy.

We thank Miss Joan Lennie for preparing the manuscript, Dr R J Prescott for statistical advice, and Dr E H Jellinek and Dr B Ashworth for their helpful comments.

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(*Accepted 29 April 1985*)

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Effect of inhalation of corticosteroids on exercise induced asthma: randomised double blind crossover study of budesonide in asthmatic children

Corticosteroids are well established in the management of bronchial asthma and are thought to act by inhibiting the late asthmatic reaction. Whether they have any effect on immediate reactions like exercise induced asthma is controversial. Generally, they are thought to be ineffective in exercise induced asthma, whether given short or long term and orally or by inhalation,[1 2] but attenuation of exercise induced asthma has been shown during regular treatment with inhaled steroids.[3 4] I report the results of a double blind placebo controlled study in which children with exercise induced asthma received budesonide acrosol for three weeks.

Clinical details of patients and results of tests of visual function

Retinopathy	No of patients	Mean (SD) age (years)	Mean (SD) duration of diabetes (years)	Mean (SD) glycosylated haemoglobin (%)	No of eyes tested	No with abnormal visual evoked potential	No with abnormal contrast sensitivity function
None	5 (4M, 1F)	25·4 (3·9)	8·8 (1·0)	10·8 (1·8)	10	1	
Background	11 (6M, 5F)	27·9 (2·8)	15·0 (4·1)	10·5 (1·5)	22	5	
Proliferative	6 (4M, 2F)	27·8 (5·0)	15·2 (4·4)	12·5 (2·5)	12	12	3