precise guidelines must await the outcome of further controlled studies in this small subgroup of patients.

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SHORT REPORTS

Idiopathic hypopituitarism in the elderly

The prevalence of pituitary failure in the elderly is unknown.[1] Equally uncharted are the clinical picture and the aetiology of hypopituitarism when it does occur in this age group. Five patients are presented who showed several common features, raising the possibility that hypopituitarism may be commoner than hitherto suspected.

Case reports

Case 1—A 90 year old man was admitted with bronchopneumonia. He showed postural hypotension and was clinically hypothyroid. Thyroid function tests showed low serum thyroxine concentrations but thyroid stimulating hormone within the normal range. He was mildly hyponatraemic and had low plasma cortisol concentrations (table). Plain skull films showed a normal pituitary fossa. He responded dramatically to replacement treatment with corticosteroids and thyroxine.

Case 2—A 76 year old woman presented with features of hypothyroidism, disproportionate pallor, and absent body hair. She was found to have subnormal serum thyroxine without a raised thyroid stimulating hormone concentration, mild hyponatraemia, and hypocortisolaemia. Further investigations showed panhypopituitarism and a radiologically normal pituitary fossa (table). Replacement treatment with hydrocortisone and thyroxine produced a pronounced improvement, but she subsequently developed congestive cardiac failure and died from myocardial infarction.

Case 3—An 84 year old woman presented with fainting episodes and severe postural hypotension. She had disproportionate pallor and cool, thin skin. Investigation showed low cortisol, sodium, and thyroxine concentrations with normal thyroid stimulating hormone and adrenals responsive to exogenous adrenocorticotrophic hormone. Further investigations showed anterior pituitary insufficiency and normal skull films. Hydrocortisone and thyroxine improved her clinical state and the postural hypotension resolved, but she died suddenly three months later.

Case 4—An 86 year old congenitally deaf and dumb woman was admitted with diarrhoea. Thyroid function tests disclosed slightly low serum thyroxine concentration but normal thyroid stimulating hormone. She had disproportionate pallor, periorbital oedema, myotonic jerks, thin skin, little pubic hair, and postural hypotension. She was found to have non-insulin dependent diabetes mellitus.

Case 5—This patient first presented in 1976 with a history of blackouts for several years and longstanding pallor. Initial investigations showed low serum thyroxine and plasma sodium concentrations and low urinary sodium excretion. Skull *x* ray films were normal. Great improvement followed treatment with cortisone acetate and thyroxine. Eight years later he developed a rapidly growing carcinoma of bronchus, confirmed at necropsy.

Comment

All patients reported on had subnormal serum thyroxine concentrations and, in the four in whom it was measured thyroid stimulating hormone was within the normal range. All had low or low normal plasma sodium concentrations and cortisol concentrations at 0900 h. Adrenal response to Synacthen was deficient when tested, while the use of glucagon in the three women (a more appropriate stress test of secretion of adrenocorticotrophic hormone and growth hormone in this age group than insulin induced hypoglycaemia) showed partial insufficiency of the cortisol response and more profound deficiency of the growth hormone response. Hyperprolactinaemia was not seen. Despite low gonadotrophin secretion at diagnosis all three women had borne children, and two had undergone the menopause aged about 50. None of the patients was short—one (case 1) was over 180 cm tall. In no case was aetiology established, but the two pituitary glands examined post mortem showed atrophy. There is little evidence for pituitary atrophy with increasing age.[2] The data do not permit differentiation between primary pituitary disorder and dysfunction secondary to hypothalamic disease. Vascular insufficiency might have been responsible.[3] In the context of Sheehan's syndrome, the association of severe pituitary failure and widespread atheroma is well recognised.[4]

Hypopituitarism may occur at all ages. Simple replacement treatment with glucocorticoids and thyroxine may not guarantee prolonged

Clinical and endocrine findings in five cases of hypopituitarism

	Case 1	Case 2	Case 3	Case 4	Case 5	Normal values
Sex	M	F	F	F	M	
Age (years)	90	76	84	86	70	
Thyroxine (nmol/l)	20	18	14	61	28	70-140
Thyroid stimulating hormone (mU/l)	1·0	3·0	6·7	6·5		0-7
Peak thyroid stimulating hormone after thyrotrophin releasing hormone (mU/l)	6·0	3·5	7·7	14·5		△>2
Plasma sodium (mmol/l)	133	133	128	130	119	135-145
Cortisol at 0900 h (nmol/l)	60	217	141	170	109	>180
Cortisol 60 minutes after Synacthen (nmol/l)	203				213	>500
Peak cortisol after glucagon (nmol/l)		483	419	460		>550
Basal follicle stimulating hormone, luteinising hormone (U/l)	2·4, 4·6	0·6, 1·2	12, 6·9	1·2, 1·3		>28, >29*
Peak follicle stimulating hormone, luteinising hormone after gonadotrophin releasing hormone (U/l)	2·4, 5·1	0·6, 1·6	14, 12	2·0, 2·1		
Peak growth hormone after glucagon (mU/l)		0·8	1·1	4·4		>20
Basal prolactin (mU/l)		162	145	540		<550
Peak prolactin after thyrotrophin releasing hormone (mU/l)		198	188	1166		>315

*Postmenopausal women.
Conversion: SI to traditional units—Thyroxine: 1 nmol/l ≈ 0·08 μg/100 ml. Plasma sodium: 1 mmol/l = 1 mEq/l. Cortisol: 1 nmol/l ≈ 0·036 μg/100 ml.

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.

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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 maculopapillary 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.

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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 aerosol 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