The vitamin B12 level was greater than 1000 µmol/ml (normal: 140–900 µmol/ml), 25 hydroxy calciferol was 3.8 µg/ml (normal: 3–30 µg/ml), red blood cell cyanate was 1.4 µmol/l (normal: <3 µmol/l), plasma cyanate was 0.1 µmol/l (normal: <0.2 µmol/l) and plasma thiocyanate was 35 µmol/l (normal: <250 µmol/l in smokers). Tests of dark adaptation were not performed.

She was treated with intramuscular vitamin A (3000,000 units monthly), strong vitamin B and C injections (thiamine 250mg, riboflavin 4mg, pyridoxine 50mg, nicotinamide 160mg and ascorbic acid 500mg) intramuscularly, initially daily for a week then biweekly till discharge. The azathioprine and prednisolone were discontinued. On such a regime the vision improved over the one month hospital stay, so that she was able to get about the ward at night without bumping into furniture and her visual acuity at discharge had returned almost to normal in both eyes (right eye N8, 6/9; left eye N6, 6/9). Colour vision was still impaired and the centro-caecal scotomata had all but disappeared in the left eye but was still present in the right eye, although reduced in size. Prior to discharge she had reverted to reading books with conventional print rather than extra large print.

This case illustrates the nutritional basis of bilateral visual failure with centro-caecal scotomata. The patient had proved vitamin A deficiency with night blindness. She had a recognised basis for malnutrition of 9 years standing. The visual failure developed in the setting of regular hydroxycobalamin injections and the serum level testified to the adequate administration. The serum and red blood cell cyanate levels were in the normal range and there was no attempt at dietary alteration during her hospital stay other than intramuscular vitamin administration.

Early reports^{1,2} of this condition relating malnutrition as a causative factor, invariably also included an improved general diet in addition to the vitamin B preparations. The importance of general improvement in dietary intake is pertinent to the fact that cyanide detoxification requires an adequate supply of sulphur-containing amino acids and normal liver function.¹ Furthermore such studies, which included patients with heavy alcohol and tobacco intake,^{3–5} failed to measure cyanide levels of such patients.

The possible extra cyanide load in prisoner-of-war camps may have been due to alternative food substitutes with high

cyanide content, such as cassava,² combined with inadequate sulphur containing amino acids in the diet rather than purely vitamin B deficiency. Similarly in the group of patients in whom cyanide levels were measured and were elevated,^{1,6} hospitalisation invariably would have involved improved diet and vitamins. In such a situation it is again difficult to be sure which factor was important in clinical improvement. In our patient there was no doubt that cyanide was not the responsible agent and that dietary intake was not changed except by the administration of multi B group vitamins. We considered the possibility that azathioprine was a toxic agent in this patient; however, extensive search of the literature failed to reveal an instance of toxic amblyopia during azathioprine administration. We can only conclude that visual improvement in our patient was due to the vitamin A and the B group vitamin replacement.

ROBERT IANSEK

CHRISTOPHER J EDGE

Department of Neurology,
The Middlesex Hospital,
Mortimer Street,
London W1N 8AA, UK

Address for reprint requests: Dr R Iansek, Dept of Neurology, Prince Henry's Hospital, St Kilda Rd, Melbourne, Australia 3004.

References

- McLean DS. *Nutritional Ophthalmology*. Academic Press 1980:150–89.
- Denny-Brown D. Neurological conditions resulting from prolonged and severe dietary restriction. *Medicine (Baltimore)* 1947;26:41–113.
- Victor M, Mancall EL, Dreyfus PM. Deficiency amblyopia in the alcoholic patient. *Arch Ophthalmol* 1960;64:1–33.
- Victor M, Tobacco: alcohol amblyopia. *Arch Ophthalmol* 1963;70:313–8.
- Victor M, Dreyfus PM. Tobacco: alcohol amblyopia. *Arch Ophthalmol* 1965;74:649–57.
- Jestico JV, O'Brien MD, Teoh R, Toseland PA, Wong HC. Whole blood cyanide levels in patients with tobacco amblyopia. *J Neurosurg Psychiatry* 1984;47:573–8.

Accepted 22 April 1985

Prolactin cell autoantibodies and Alzheimer's disease

Sir: Evidence of autoimmune dysfunction in Alzheimer's disease has been suggested by the excess of autoimmune disorders observed in families with the disorder^{1,2} and by the presence of immunoglobulins in amyloid fibrils of senile plaques.³ In a

report from Angers,⁴ France, an incidence of 96% of prolactin cell autoantibodies was described in 27 cases of Alzheimer's disease and of 90% in 11 cases of Down's syndrome. In view of the potential importance of this finding for the understanding of the disorder and the initiation of therapeutic measures we attempted to duplicate this study.

Three normal human hypophyses were obtained within 3 hours post mortem. Patient and control sera were diluted 1/10 and stored at 4°C for a maximum of 48 hours. Fluorescein anti-human (total) IgG (Kallestad) and peroxidase labelled anti-prolactin (Dako) were used. Reproducibility studies showed that storage below –20°C abolishes serum activity, that storage at 4°C during two weeks diminishes but not abolishes serum activity. Duplo studies with an interval of one day did lead to identical results in a blind control study. Patients and controls were selected between the inmates of Psychiatric Hospital Rosenberg. A diagnosis of Alzheimer's disease was made on clinical grounds including psychometry. CT scanning was performed whenever possible. Diagnostic criteria for multiple infarct dementia included CT scanning. Patients suffering from chronic psychiatric illness (schizophrenia, cyclic psychosis, depression) above age 65 years served as controls. CT scans showing infarcts or gross atrophy leading to exclusion from the control group. All histological investigations were performed without any knowledge of the names of the patients or their diseases. Serum batches were delivered to the laboratory under codenumbers. Decoding took place at the end of the study.

The results are listed in the table. Clear cut positive reaction was encountered in 10 instances, slight reaction in four. Differences between the three groups are not significant. The high frequency of antibodies reacting to prolactin cells in Alzheimer's disease, reported by Pouplard *et al* was not confirmed in this study. The positive reactions shown in the table may indicate the presence of antibodies cross-reacting to prolactin cells related to Alzheimer's disease or senescence.

TA VAN DORE

LJ ENDT

J TE VELDE

JPJ SLAETS

J REICHGEL

Departments of Neurology and Pathology
Municipal Hospitals of The Hague and
Psychiatric Hospital Rosenberg,
The Hague, The Netherlands

Letters

Table Antibodies (cross-)reacting to prolactin cells in Alzheimer's disease, multiple infarct dementia, and controls.

	N	Antibodies (cross-)reacting to prolactin cells		
		+	±	%
Alzheimer's Dementia	15	5		33
			1	6
Multiple infarct dementia	10	1		10
Psychosis	19	4		21
			3	16

+: positive in a large number of cells.

±: positive in scattered cells.

References

- Heston LL, Mastri AR, Anderson VE, White J. Dementia of the Alzheimer type: clinical genetics, natural history and associated conditions. *Arch Gen Psychiatry* 1981; **38**:1085-90.
- Heyman A, Wilkinson WE, Hurwitz BJ, et al. Alzheimer's disease: genetic aspects and associated clinical disorders. *Ann Neurol* 1983; **14**:507-15.
- Ishii T, Haga S. Immuno-electron microscopic localisation of immuno globulin in amyloid fibrils of senile plaques. *Acta Neuropathol (Berl)* 1976; **36**:243-7.
- Pouplard A, Emile J, Vincent-Pineau F. Auto anticorps circulants anti cellules à prolactine de l'hypophyse humaine et maladie d'Alzheimer. *Rev Neurol (Paris)* 1983; **139**:187-91.

Accepted 10 May 1985

Note: the author had intended this letter to be published with that of Philpot et al (*J Neurol Neurosurg Psychiatry* 1985; **48**:287.)

Muscle hypertrophy in chronic polymyositis.

Sir: Muscle hypertrophy is rare in polymyositis¹ and has been reported only in the childhood form of the disorder.² We report a case of bilateral quadriceps hypertrophy in an adult with chronic polymyositis.

A 26-year-old man was admitted in July 1984 with the complaint of slowly progressive weakness of insidious onset of both pelvic girdle muscles for 5 years, of the shoulder girdle for 4 years and of leg muscles for 3 years. There was no history of similar illness in the family.

Examination revealed bilateral atrophy of spinati, biceps, triceps, deltoids, glutei, gastrocnemii and anterior tibial muscles. Both quadriceps were hypertrophied and strong (fig). There was weakness of neck flexors, proximal muscles of upper limbs

and hip flexors. The gait was waddling. Sensation, reflexes and cerebellar function were normal. There were no fasciculations.

Routine haematological investigations, blood chemistry, chest radiograph and electrocardiogram were normal. Creatine kinase values were 1052 IU/L (normal up to 55 IU/L). Thigh radiographs for soft tissue showed no calcified lesions. EMG revealed a myopathic pattern. The muscle biopsy specimen taken from left deltoid showed necrosis, phagocytosis, internal migration of nuclei, fibre-size variations and a mononuclear inflammatory infiltrate, often most prominent in a perivascular location suggestive of polymyositis. There was no fatty infiltration.

Muscular hypertrophy results from an increase in the number of myofibrils.³ The basic mechanisms involved in the laying down of new myofibrils are incompletely understood. Longitudinal splitting of myofibrils, once they reach a certain stage may be one of the ways in which the numbers of myofibrils are increased.⁴

In pathological states, muscular hypertrophy has been encountered in myotonia congenita, Becker's and limb-girdle muscular dystrophy, long standing hypothyroidism, acromegaly, slowly progressive forms of spinal muscular atrophy, childhood form of polymyositis and cysticercosis. As far as we know, the present case is unique because of muscular hypertrophy occurring in a chronic form of polymyositis in an adult.

K PRASAD

M BEHARI

MC MAHESHWARI

Department of Neurology,
All India Institute of Medical
Sciences, New Delhi-110029, India

References

- Walton JN. Clinical examination of the neuromuscular system. In: Walton JN ed.

Disorders of Voluntary Muscle, Edinburgh Churchill Livingstone: 1983:448-80.

- Thompson CE. Polymyositis in children. *Clin Pediatr*. 1968; **7**:24-28.

- Cullen MJ, Mastaglia FL. Pathological reactions of skeletal muscle. In: Mastaglia CL, Walton JN eds. *Skeletal Muscle Pathology*, ed: Edinburgh Churchill Livingstone 1982:88-139.

- Golaspink D. Post embryonic growth and differentiation of striated muscle: In: Bourne GH (ed). *Structure and function of Muscle*. New York Academic Press, 1972; **1**:179.

Accepted 22 April 1985