The vitamin B12 level was greater than 1000μg/ml (normal: 140–900μg/ml), 25 hydroxy calcerol was 3–8μg/ml (normal: 3–30μg/ml), red blood cell cyanate was 1.4μmol/l (normal: <2μmol/l), plasma cyanate was 0.1μmol/l (normal: <0.2μmol/l) and plasma thiocyanate was 35μmol/l (normal: <250μmol/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 centrocaecal 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 centrocaecal 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 hydroxycoobalamin 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 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. Furthermore such studies, which included patients with heavy alcohol and tobacco intake, 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, 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, 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.

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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 disorder4 and by the presence of immunoglobulins in amyloid fibrils of senile plaques. In a report from Angers, 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. Fluorescin anti-human (total) Ig (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 lead 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 codenames. 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.

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Letters

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

<table>
<thead>
<tr>
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<th>N</th>
<th>+</th>
<th>±</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer's Dementia</td>
<td>15</td>
<td>5</td>
<td>1</td>
<td>33</td>
</tr>
<tr>
<td>Multiple infarct dementia</td>
<td>10</td>
<td>1</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>Psychosis</td>
<td>19</td>
<td>4</td>
<td>3</td>
<td>21</td>
</tr>
</tbody>
</table>

+: positive in a large number of cells.
±: positive in scattered cells.

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Accepted 10 May 1985

Note: the author had intended this letter to 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 polymyositis and has been reported only in the childhood form of the disorder. We report a case of bilateral quadriiceps 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 quadriiceps were hypertrophied and strong. 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. 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.

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.

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Prolactin cell autoantibodies and Alzheimer's disease.

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