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	1	33
Multiple infarct dementia	10	1		6
Psychosis	19	4		10
			3	21
				16

+: positive in a large number of cells.

±: positive in scattered cells.

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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.

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