Further investigations including full blood count, ESR, VDRL, blood sugar, chest and cervical spine radiographs, myelography, CSF examinations done were all normal. All the patients showed normal sensory nerve conduction study. Six patients involving seven nerves showed prolongation of motor distal latency greater than $2\frac{1}{2}$ SD. The range for the median nerve from wrist to APB was 3.5 to 5.9 ms. ($N \pm SD = 3.5 \pm 0.5$ ms); and for the ulnar nerve from wrist to ADM, it was 3.2 to 4.9 ms ($N \pm SD = 3.1 \pm 0.7$ ms). All the motor nerve conduction studies were normal. The range for the elbow to wrist segment of the median nerve was 48 to 67 m/s ($N \pm SD = 58.6 \pm 4.2$); and 52 to 71 ms ($N \pm SD = 56.8 \pm 4.9$) for the same segment of the ulnar nerve. Electromyography showed a neuropathic picture in all with varying combinations of fibrillation, positive sharp wave, giant motor units, and isolated interference pattern.

The total duration of illness from date of first symptom to last date of follow up was 15 years in one patient, 8 years in two patients, 6 years in four patients, 5 years in two patients, 3 years in one patient, 2 years in four patients and less than 1 year in five patients. No progression of the illness was seen in those with total duration of illness for 2 years or more.

We agree with Loong *et al*⁹ and O'Sullivan *et al*¹⁰ that the anterior horn cell is the most likely site of pathology. This is supported by the segmental distribution seen clinically, the absence of sensory abnormality, the relatively normal motor nerve conduction study and the presence of "giant motor units" which may sometimes be seen in the EMG examination. The aetiology of this condition remains an enigma. The predominance of cases reported from Japan and India, suggest an ethnic predisposition to development of the disease. Malaysia is a multiracial country, the main racial group in the peninsular are Malay, immigrant Chinese and Indian. The three races are ethnically distinct. It is interesting to note that when they are exposed to the same environment, all three groups showed the same apparent prevalence of the disease. The predominance of right hand involvement was noted also by Hirayama *et al*⁶ and Hashimoto *et al*.⁵ It was suggested that the use of the limb is a factor in the development of the disease.

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The effects of magnetic resonance imaging on different types of microsurgical clips

Sir: Magnetic resonance imaging (MRI) is a very attractive modality since it does not use ionising radiation and is sensitive to a wide range of pathology. However, a hazard may exist when a microsurgical clip is exposed to strong magnetic fields since these may induce heating or cause a force sufficient to move the clip or even dislodge it from a vessel.

We have investigated these hazards and have tested 47 microsurgical clips in a 0.5 Tesla MR system based in the Queen's Medical Centre, Nottingham. Each clip was suspended on a length of cotton 40 cm from the magnet bore and any movement caused by the magnetic field measured in degrees. Each clip was then detached, placed within the head coil and subjected to 10 minutes of radiofrequency energy thus simulating the conditions used in human head scanning. Temperature measurements were made before and after radiofrequency exposure. The results are displayed in table. No measurable temperature rise was observed.

The potential hazard posed by the movement of a microsurgical clip especially when attached to an aneurysm is obvious and this risk increases proportionately with

Table Magnetic properties of various microsurgical clips

Clip	Numbers tested	Ferromagnetic (that is moved within the magnetic field)
Scoville Lewis	6	Yes
Schwarz	2	Yes
Mayfield	13	Yes
Drakes	2	Yes
Sundt Kees	4	Yes
Heifetz	4	Yes
Kerr	1	Yes
McFaddens	2	Yes
Liga	2	No
Yasargil	2	No
Sugita	2	No
Olivecrona-Norlen	3	No
Cushing-McKenzie	2	No
Silver Brain	2	No
Weck	2	No
Ferromagnetic (moved within magnetic field) = 34		
Non-ferromagnetic (did not move within magnetic field) = 13		

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increasing magnetic field strengths. Neurosurgeons should try to remember this risk when using such clips and should use non-ferromagnetic clips whenever possible. At present we exclude from magnetic resonance imaging any patient who is known to have any aneurysm clip since as well as the risk of detachment the image is severely degraded.

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Rapid enlargement of non-functioning pituitary tumour following withdrawal of bromocriptine

Sir: Bromocriptine, an ergot derivative with dopamine receptor agonist properties, has been shown to reduce the size of prolactin secreting pituitary tumours.¹ Recent work has demonstrated involution of adenomatous prolactin cells with tumour shrinkage occurring within three weeks of commencing bromocriptine.² The effect of bromocriptine upon other pituitary adenomas is uncertain. Occasional reports have suggested that a reduction in tumour size may occur in non-functioning^{1,3,4} and growth hormone secreting tumours.⁵ We report a patient with a non-functioning chromophobe adenoma whose visual acuity deteriorated rapidly following withdrawal of bromocriptine.

The patient presented in October 1981 with visual impairment and lethargy. Bilateral optic atrophy was present with a bitemporal hemianopia and visual acuities of 6/24 bilaterally. Chiasmal compression was suspected and pituitary fossa tomograms and CT scan confirmed the presence of a pituitary macro-adenoma with suprasellar extension. Hypopituitarism was confirmed biochemically and serial prolactins in the range 530–630 mU/l (normal range for females less than 300 mU/l) were too low to represent a prolactinoma. The patient refused surgery and was com-

menced on replacement therapy with hydrocortisone 10 mg b.d. and thyroxine 0.1 mg a day. Bromocriptine 2.5 mg eight-hourly was added when an outpatient serum prolactin of 890 mU/l was reported three months later. During the next two years her visual acuity and field defect remained unchanged as did pituitary fossa tomography. Her serum prolactin was persistently less than 100 mU/l. She was readmitted in September 1983 with a chest infection. As she also complained of indigestion with reflux and was found to have a hiatus hernia with an iron deficiency anaemia, her bromocriptine was stopped. At outpatient review six weeks later she complained of marked visual loss and her acuities had deteriorated to 5/60 in both eyes with further field restriction. CT scan revealed enlargement of the tumour with marked suprasellar extension but no evidence of pituitary apoplexy. Her serum prolactin was 430 mU/l. Subfrontal decompression was carried out but the patient died after operation from a myocardial infarction. Histology revealed a chromophobe adenoma with no evidence of pituitary infarction. Stains for prolactin using the immunoperoxidase technique were negative.

We feel that the rapid deterioration in vision within six weeks of stopping bromocriptine was due to tumour enlargement, suggesting that bromocriptine may also have a suppressive action on some non-functioning pituitary tumours. The time course of enlargement was of the same scale noted by Barrow *et al* who observed prolactinoma re-expansion within 7–14 days of stopping bromocriptine in two patients.² When bromocriptine is discontinued in patients with pituitary macroadenomas, visual fields and tumour size should be monitored even if the tumour is non-functioning.

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Prolactin cell autoantibodies and Alzheimer's disease

Sir: Alzheimer's disease is the commonest type of dementia in old age.¹ Diagnosis is based on a history of gradual global intellectual deterioration, and the exclusion of other conditions producing a picture of dementia. To be complete the latter requires an extensive, and expensive, series of investigations including CT brain scan.² The search for readily identifiable markers of Alzheimer's disease which would short-cut this process and facilitate earlier and more accurate diagnosis has so far been unsuccessful.

The possibility that there may be immunological markers for Alzheimer's disease would be of clinical and theoretical importance. To date the association between Alzheimer's disease and certain histocompatibility antigens has been questioned³ and the finding of abnormally raised levels of immunoglobulins in cognitively impaired elderly⁴ has not been replicated.⁵ However, Pouplard *et al*⁶ using an indirect immunofluorescence test on cryostat sections of human post mortem pituitary (2–3 hours after death), recently reported that 26 of 27 cases of Alzheimer's disease, and 10 of all 11 cases of Down's syndrome with dementia (where the neuropathological changes parallel those of Alzheimer's), have been sera positive for prolactin cell autoantibodies. A control group of normal 80 year olds and patients with Parkinson's disease had much lower rates of positive reactions (three of 21 and two of 40 respectively). Three out of 10 patients with multi-infarct dementia had prolactin cell autoantibodies. These antibodies were first described in patients with autoimmune polyendocrine disease⁷ and rarely occur in the general population.^{8,9}

The unexpected findings of Pouplard have attracted world-wide interest and we know of several groups who are attempting to replicate these results. In view of its