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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³ Spark RF, Baker R, Bienfang DC, Bergland R. Bromocriptine reduces pituitary tumour size and hypersecretion. *JAMA* 1982; 247:311–6.

⁴ Wass JAH, Williams J, Charlesworth M, *et al*. Bromocriptine in management of large pituitary tumours. *Br Med J* 1982; 284:1908–11.

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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