

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.

We thank the Department of Health and Social Security and Picker International for their continuing support. We would also like to thank Downs Surgical Ltd for making the microsurgical slips available. These findings were initially presented by Mr J Firth at the 7th International Congress of the International Microsurgical Society, September 1982.

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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.

⁵ Wass JAH, Thorner MO, Morris DV, *et al*. Long term treatment of acromegaly with bromocriptine. *Br Med J* 1977;1:875–8.

Accepted 10 October 1984

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 shortcut 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

potential importance we have attempted to repeat this study using the traditional indirect immunofluorescence on cryostat sections of fresh human pituitary obtained from surgical operations. Undiluted sera were applied and sheep anti-human total Ig and specific IgA (Wellcome) were used as conjugates at the proper dilution. Normal human pituitary obtained from a patient with breast cancer was used throughout our study. The presence of prolactin, growth hormone, LH, FSH, TSH cells as well as the specific pituitary microsomal antigen was confirmed on every 30 sections with specific monoclonal antibodies and with prolactin cell positive serum as a positive control. Sera were collected from 20 patients with clinically diagnosed Alzheimer's type senile dementia and three patients with Down's syndrome who had had intellectual deterioration. Diagnosis was made on historical evidence of at least six months duration of symptoms and all patients had a cranial CT scan, were examined neurologically and had psychometry. Patients with a history of stroke, head injury, alcoholism, epilepsy, major psychiatric illness were excluded, as were those with focal neurological signs, hypertension, metabolic or endocrine illness. The Alzheimer group were aged between 63 and 95 (mean age 80 ± 9 yr), with a duration of illness of between 6 and 72 months (mean duration 33 ± 18 m). The Down's group were in their early sixties (mean age 61 ± 1 yr) with a duration of dementia of between 12 and 18 months.

None of the sera from either group contained autoantibodies to prolactin cells, although six had gastric parietal cell, five had thyroglobulin autoantibodies (titres 1:80–1:320) and five were positive for thyroid microsomal autoantibody (titres 1:400–1:1,600). The reproducibility of our results in relation to the use of different substrates was checked by testing serum randomly selected from patients affected by several endocrinopathies and normal controls on human, baboon and monkey pituitary sections. As shown in the table, sera tested on baboon and human pituitary gave virtually identical results for monkey endocrine cell autoantibody screening, whereas the same sera tested on monkey

pituitary showed a much higher number of positive reactions. We postulate that although rhesus monkey is close to the human, some human gamma-globulins could show a heterophile specificity with monkey pituitary. When in fact the 20 Alzheimer's and the three Down's syndrome sera were re-tested on monkey pituitary, 4/23 sera gave positive reactions on endocrine cells, two of which reacted with prolactin cells. Our failure to replicate Poupard's results could be due to substrate differences since the French group used post-mortem human glands. Further studies are warranted in this area before firm conclusions can be reached regarding the role of prolactin cell antibodies as markers for unexplained cerebral atrophies.

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Accepted 25 June 1984

Spinal angiomatosis presenting as a sensory neuropathy

Sir: Spinal angiomatosis¹ is a rare condition which usually presents with progressive symptoms suggesting a radiculomyelopathy. It may also cause subarachnoid haemorrhage.^{2,3} The unusual presentation of our patient with symptoms suggestive of a sensory neuropathy appears unique.

An Irish building labourer, aged 58 years, was admitted to hospital because of progressive tingling of his legs and unsteadiness in walking for ten days. He had not previously had any significant illnesses and there was no family history of neurological disease. There was no suggestion of any exposure to known neurotoxins. His alcohol consumption was considered moderate, but he smoked 80 cigarettes daily. He appeared unkempt, with markedly carious teeth. In other respects general medical examination was normal. His intellectual function had always been below average and he was vague about the details of his medical history. The cranial nerves and tone and power of his limb muscles were normal. All the tendon reflexes were reduced, while the right biceps, left knee and ankle reflexes were absent, but plantar responses were flexor. Modalities of sensation were impaired in the fingers as far as the metacarpophalangeal joints, and in the feet, as far as the ankles. Position sensation was impaired at the toes and ankles. He was unable to stand still with his eyes closed, and his gait was wide based and unsteady. Extensive haematological, biochemical and radiological investigation for a possible carcinoma gave normal findings. A CT scan of the brain was normal. Nerve conduction studies showed normal motor conduction velocities but reduced sensory action potentials (right radial $1 \mu\text{V}$; right sural $2 \mu\text{V}$; left sural absent). The F latency to the right thenar muscles was 34 ms and to the left abductor hallucis was 55 ms. Lumbar puncture revealed a clear fluid containing no cells

Table Unselected cases tested by IFL on three different pituitary substrates

Pituitary substrates	Total cases tested	Positive reactions
Normal human	100	17
Baboon	100	18
Rhesus monkey	51	28