Table 2 Neuropsychological test results and SLI in three study groups mean (SD) scores

Score		Full Scale IQ	Verbal IQ	Performance IQ	Block Design max=42	Digit Symbol max=6	Logical Memory (MQ)	Associate Learning (MQ)	Wordlist genera- tion	SLI (fmol/ml)
PD without dementia (N=11)	$\bar{x}$ SD	100.9 (9.7)	103.0 (9.7)	97.9 (10.7)	7.8 (2.8)	6.8 (3.8)	91.4 (10.8)	99.1 (11.4)	50.4 (14.1)	49.1 (14.4)
PD with dementia (N=11)	$\bar{x}$ SD	82.5** (7.5)	91.6 (11.9)	76.6** (7.1)	3.0† (2.0)	2.9† (1.8)	81.0† (5.7)	70.6 (36.4)	25.6‡ (8.0)	56.4 ns (24.6)
Controls (N=11)	$\bar{x}$ SD	101.4 (15.8)	101.1 (11.0)	99.1 (16.9)	9.8 (2.9)	10.0 (4.4)	90.4 (4.5)	94.8 (18.2)	56.0 (17.1)	50.2 (17.7)

MQ = Memory Quotient (Wechsler Memory Scale)

** = $p \leq 0.001$ One-Way ANOVA† $p \leq 0.05$ Kruskal-Wallis‡ $p \leq 0.001$ Kruskal-Wallis

SLI = somatostatin-like immunoreactivity.

measured using the verbal and performance subtests of the HAWIE, a German equivalent of the Wechsler Adult Intelligence Scale. The sum of all scaled scores yielded the full scale IQ (FSIQ). A German version of the Wechsler Memory Scale (WMS) was used to test verbal learning, while semantic memory and associative verbal production were tested by time limited (60s) word list generation for categories (supermarket, animals) and letters.

Lumbar punctures were performed in the morning after an overnight fast before the patients' arose. Three aliquots of 3ml each were collected into glass tubes and the third was immediately frozen and stored at -70° until assessment of SLI by radioimmunoassay as described previously.³ The anti-serum used equally detected somatostatin-14, somatostatin-28 and prosomatostatin.

No significant differences were found in an analysis of variance between mean SLI-concentrations of the three groups ($F=0.435$, $P=0.65$, $df=2$).

Table 2 summarises the mean SLI values and the most relevant neuropsychological test results. Highly significant differences existed between the Parkinson-dementia group and the non-demented and control groups in several cognitive measures and the overall test pattern in the demented group was compatible with subcortical dementia. There

were no significant differences between the non-demented group of Parkinson's disease patients and controls in any of the cognitive functions tested in this study (table 2).

Spearman rank correlation coefficients for possible relationships between CSF-SLI levels and scores of any of the neuropsychological test parameters did not reach significant values in any of the three groups.

This study thus failed to confirm previous findings of lowered CSF-SLI in Parkinson's disease⁴ and agrees with the findings of Beal *et al*⁵ in a smaller sample of Parkinsonian patients. Based on the finding of lowered cortical somatostatin concentrations in demented but not in non-demented Parkinsonian patients² it has been suggested that discrepant results concerning CSF-SLI in Parkinson's disease might be due to differences in the prevalence of dementia among the patient populations studied. This is not supported by the results of our study where there was no difference in mean CSF-SLI values between patients with and without dementia as assessed by formal neuropsychological testing. This does not exclude a role for cortical somatostatin depletion in the dementia of Parkinson's disease but suggests that it is less significant than in Alzheimer's disease. Cortical somatostatin depletion is apparently not reliably reflected in the CSF and CSF-

SLI does not appear a useful biochemical marker for dementia in these patients.

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Multifocal astrocytoma presenting as action myoclonus

Action myoclonus is an uncommon presentation of intracranial tumour.¹ We have recently seen a multifocal astrocytoma present in this way.

A 46 year old right handed housewife presented with a three month history of abnormal movements of her left arm. Soon after waking one morning she was aware of jerking movements of the left arm when she reached out to touch her husband. Jerking persisted for a few seconds only and she thought no more of it. One week later she had several similar attacks, all following voluntary movements of the left arm. Over the next two months similar attacks occurred at least five times daily but often up to 100 times per day. Attacks would last between 10 and 15 s. They usually occurred during and interfered with eating, dressing and bathing. Her attacks consisted of coarse and irregular jerky movements involving the left shoulder and to a lesser degree the elbow and wrist. Attacks were consistently triggered by voluntary movement but not by the intention to move,

nor by touch, pressure or startle. She had noticed a refractory period of about five minutes after an attack during which time she could use the left arm quite normally. She did not have attacks during sleep.

Examination revealed spasticity and exaggerated tendon reflexes in the left arm. All routine blood tests, examination of the cerebrospinal fluid (CSF), unenhanced cranial computed tomography (CT), somatosensory evoked potentials, and an electroencephalogram (EEG) performed during jerking were normal. Carbamazepine failed to control her abnormal movements and was stopped after 10 days because of a generalised rash. Subsequently the attacks became less frequent and stopped completely about a month later without any further treatment.

One month after her jerking had stopped, she developed mild pyramidal weakness of the left arm and slight clumsiness of her right hand. Magnetic resonance imaging (MRI) of the brain showed multiple lesions of different sizes involving both grey and white matter (figure). The largest lesions were in the left striatum, left frontal white matter, and right frontal and parietal cortex. None of the

lesions produced mass effect. Subsequent cranial CT now showed ill-defined non-enhancing low density changes in the same places. All evoked potentials, EEG, re-examination of CSF, autoantibody screen, echocardiogram and tests for *Borrelia burgdorferi* and human immunodeficiency virus were normal or negative.

It was thought that she was probably suffering from some sort of inflammatory encephalopathy. She returned to her home abroad with arrangements for repeat cerebral MRI. After remaining well and asymptomatic for the following six weeks she developed generalised seizures which were treated with phenytoin. After the occurrence of a number of fits in rapid succession she did not recover consciousness.

She was readmitted to the National Hospital, six months from the onset of her symptoms. She had marked neck stiffness, swelling of both optic discs, her eyes were divergent and her pupils were semi-dilated and unreactive. She could flex her limbs to painful stimuli and her left arm and right leg were spastic. Both plantar responses were extensor and there were Cheyne-Stokes res-

Figure T2-weighted MR images (TR/TE 2000/80 at 0.5T). Multiple areas of increased signal are shown. The largest lesions are in the left striatum, left frontal white matter and right frontal and parietal cortex.

pirations. She was ventilated and treated with mannitol and steroids.

Cranial CT confirmed a swollen brain with tonsillar herniation. The right parietal and left frontal lesions had become more prominent. Biopsy of the right parietal lobe lesion was performed through a burr hole and histological examination showed the appearances of a grade IV astrocytoma. She died three weeks after her operation.

At necropsy the brain was diffusely swollen (fixed brain weight—1449g) with flattened gyri and narrowed sulci. An area of grey discoloration was seen on the left posterior frontal lobe adjacent to the midline. There was a similar area on the right posterior frontal lobe 5 cm from the midline. A needle tract on the right anterior parietal lobe was present.

Coronal slices showed small asymmetrical ventricles, the left being larger than the right. Several areas of partly necrotic tumour involving the left frontal, the right parietal lobes and both caudate nuclei were prominent. The left anterior corpus callosum was enlarged and the left pericallosal gyrus showed pale grey discoloration. The brain stem and cerebellum were macroscopically normal.

Histological examination showed a multifocal glioma. The tumour, which invaded the grey matter, varied in the degree of cell density and pleomorphism. In the most malignant-looking areas numerous mitoses per low power field could be seen as well as newly formed capillaries with swollen endothelial cells. The cerebellum and brain stem were free of tumour. However, in the mid pons there was a small area of myelin loss with preserved nerve cells, the appearances of which suggested central pontine myelinolysis which may have been related to an incidental electrolyte disturbance.

There are three points of note in this case: 1) intracranial tumour is a rare cause of myoclonus.¹ Symptomatic action myoclonus may often be a manifestation of anoxic encephalopathy,² or may occur in the course of encephalitic illness such as Creutzfeldt-Jakob disease,³ 2) this is an unusual presentation of glioma, 7.5% of malignant tumours are multicentric, and just 2.9% show different histological appearances,⁴ 3) it was most surpris-

ing to find normal EEG records in the presence of multiple grey and white matter lesions, and in the presence of myoclonus. Even in retrospect, the records were within normal limits; from time to time there were short runs of paroxysmal theta activity in the left frontal and fronto-central regions, but these changes were far from compelling.

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Cryptococcal granuloma of the brain

Cryptococcus neoformans is a budding yeast, found in pigeon excreta, and widely distributed throughout the world.¹ Infection of the nervous system by *C. neoformans* most often causes meningitis, and immunocompromised patients are especially at risk. Rarely, a space-occupying mass, "cryptococcoma", may occur in the brain due to granulomatous inflammation around the organisms.² We report a case of intracerebral cryptococcal granuloma, in an immunologically normal patient.

A 47 year old man presented with a four month history of frontal headache, one month's loss of energy and weakness, and difficulty with memory and concentration for one week. There was no relevant past history. He was married with seven children, did not smoke and had a modest alcohol intake. On examination, he was afebrile, normotensive,

and well-oriented. He had nominal dysphasia, dyscalculia, constructional apraxia, early bilateral papilloedema and a mild right facial weakness. General examination was normal. Investigations: ESR 9 mm/hr, HB 15.1 g/dl, WBC 4.66 × 10⁹/ul, neutrophils 73%, lymphocytes 16%, serum electrolytes, CXR and urinalysis were all normal. IgM was slightly elevated at 3.7 g/l (N = 2.20 g/l), IgG and IgA were normal. Antinuclear factor and RA latex tests were negative. EEG showed left temporal slowing. CT scan demonstrated a low density left temporal mass, with ill-defined contrast-enhancing margins, and midline shift. Two needle biopsies, performed a week apart, yielded normal tissue. He was treated with dexamethasone 2 mg twice daily, which gave marked improvement. EEG recordings improved and a repeat scan six weeks later failed to demonstrate any lesion. We assumed he had either a steroid-responsive neoplasm, such as lymphoma, or an inflammatory mass. His symptoms, EEG and CT scan changes recurred three weeks after stopping steroids.

Six months following presentation a left temporal craniotomy was performed. A gelatinous yellow 5 cm gritty mass, adherent to the dura and extending into the left temporal lobe was widely excised. An intra-operative smear stained with Toluidine blue, PAS, and India ink showed numerous cryptococci, in a honeycomb pattern, in chains or as single organisms, some with buds. A portion was immediately sent for culture, but no growth was obtained. The excised mass showed many small so-called "soap bubbles" or cysts (fig a). Sections were stained with H/E, PAS, PAS/Alcian Blue, and Haematoxylin Van Gieson. Microscopy showed cryptococci arranged in groups or singly, easily seen with H/E and PAS (fig b). The mass formed a thick layer on the meningeal surface and had no inflammatory reaction in the centre, but a marked reaction at the margins. Lymphocytes, plasma cells and occasional multinucleate giant cells were present, together with fibrous strands and foci of dystrophic calcification. The lesion extended into the brain parenchyma, where there was proliferation of blood vessels, perivascular inflammation and astrocytic hyperplasia. Electronmicroscopy showed organisms ranging in size from 5050 to 15 833 nms, with capsules of 760-5000 nms. Many of the organisms appeared degenerate.

A screen for other "opportunistic" infections was performed. Toxoplasma, cytomegalovirus, herpes simplex, hepatitis B, and human immunodeficiency virus antibody tests were all negative. The ratio of helper to suppressor lymphocytes was also normal. His serum *Cryptococcus neoformans* antigen titre was one in 75. On further inquiry, he remembered that before the onset of symptoms he had worked in an attic heavily soiled with pigeon dirt. Although he did not have any respiratory illness, we believe he inhaled infected material, and had haematogenous spread to the brain from a pulmonary focus. Postoperatively, he developed a deep venous thrombosis in the leg, which required heparin and precluded anti-fungal chemotherapy. The repeat serum cryptococcal antigen titre showed a fall to one in five. He was entirely asymptomatic, so we decided to withhold anti-fungal treatment.

He was followed up regularly, and one year later, he remains asymptomatic, and has resumed work. His chest radiograph remains normal, CT brain scan does not show any new