Table CSF and antitoxoplasmic antibody studies

	Date of sample					
	20 Jan, 1982	5 Feb, 1982	26 Apr, 1982	26 Aug, 1982	26 Oct, 1983	13 Jan, 1984
Serum						
Dye test titre	1:51 200	1:51 200	1:51 200	1:51 200	1:800	1:800
IgM-IFA test	<0	<0	<0	<0	<0	<0
IgM-ELISA test	30	—	<20	<20	<20	<20
CSF						
White cells/mm ³	70	24	<2	<2	3	—
Total proteins (mg/l)	370	260	300	310	320	—
Immunoglobulin G (mg/l)	27	19	18	16	20	—
PAGE	N	N	N	N	N	—
Dye test titre	—	1:50	—	<0	<0	—
IgM-IFA test	—	<0	—	<0	<0	—

IFA = Immuno Fluorescent Antibody

PAGE = Poly Acrylamide Gel Electrophoresis

N = Normal

For the IgM-ELISA test, results are expressed in enzymatic units (<20 U = negative; 20–40 U = doubtful; >40 U = positive).

patients with a very active synthesis of specific IgG.⁶ Negative results in the IgM-IFA test have also been attributed to steric competition from high levels of specific IgG⁷ as in our case. This bias may be avoided by IgM-ELISA tests⁷ and, when the original sera of our patients were retested with the IgM-ELISA assay, we found slightly positive results for the first sample only (table). Cultures from CSF were negative for *T gondii*, but they were made after the course of antibiotics. Diagnosis was confirmed by the close correlation between acute symptoms and high dye test titres, the clinical recovery obtained with corticoids and specific antibiotic therapy, and the sixfold decrease in dye test titres and the IgM-ELISA assay. A two-year follow-up did not afford an explanation other than toxoplasmosis for the initial symptoms and signs. The acquired nature of toxoplasmosis in this case seemed probable, in view of the clinico-serological correlations, lack of the classical features of congenital toxoplasmosis and positivity of IgM-ELISA test.

Loss of vision in *acquired* toxoplasmosis is usually due to chorioretinitis or changes in the media of the eye, associated with inflammation.¹ Only two cases of cerebral blindness with involvement of central visual pathways have been reported to date.⁸ Optic neuritis is exceedingly rare. Single cases have been reported in association with overt ocular lesions.^{9–11} We are aware of only three reports with normal ocular fundi.^{3–5} In Hogan's case 5,⁵ there was concurrent definite multiple sclerosis. In Wood's case 1,³ the dye test was positive at 1:256 only, and there were no physical signs other than optic neuritis. In Wood's case 2,³ a classical picture of optic neuritis evolved in a 25-year-old patient with acute

febrile illness, cervical enlarged glands and mononucleosis. Toxoplasmosis was diagnosed by a dye test positive at 1:1024 and complement fixation plus four. In the case of Rieger,⁴ chills, left acute optic neuritis, right eye upward paresis were observed in a 49-year-old patient. Dye test was positive at 1:64 and, three months later, at 1:1024. There was a canine source of infection. Such a low incidence of optic neuritis in acquired toxoplasmosis is probably due to the obligatory intracellular parasitism of *T gondii*, although the presence of the parasite in the optic nerve has been mentioned.¹ It is important to consider acquired toxoplasmosis in the diagnosis of acute meningo-encephalitis even in immunocompetent hosts. Acute optic neuritis is not an argument for discarding the diagnosis. Prompt confirmation of *T gondii* infection by laboratory investigations is critical for this potentially curable disease.

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"True" cystic meningioma.

Sir: It has long been known that a meningioma may be associated with cysts which increase its compressive effect.^{1–4} These tumours often have been described as cystic meningiomas, although this is an unsatisfactory term. The case we present

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may contribute to the definition of the cystic meningioma, which is an extremely rare tumour.

A 58-year-old female was admitted with a two months history of Jacksonian seizures in the right limbs, with no loss of consciousness. Examination revealed a mild right hemiparesis with no other abnormality. An isotopic brain scan showed abnormal uptake in the left fronto-parietal parasagittal region. Subsequently, a CT scan revealed a low-density lesion in the left parietal parasagittal area, with peripheral contrast enhancement (fig). Left carotid angiography revealed a large parietal avascular mass with no meningeal blood supply and a depression of the pericallosal artery. At subsequent craniotomy the neoplasm was apparently totally removed. There were no adhesions between the tumour and the falx. The tumour was, histologically, a meningothelial meningioma with degenerative changes and no aberrant cells. The patient received radiotherapy. The residual disability was a leg paresis.

A review of reported cases of cystic meningioma, shows that this term has been used to describe two kinds of cysts related to a meningioma: intra and peritumoral cysts. Intratumoral cysts are very rare^{2,5-8} and arise from secretory or degenerative changes within the neoplasm or even from an increased fat content.⁹ It is generally accepted⁶ that they represent ischaemic areas of necrosis, usually located in the part of the tumour which is far from the peripheral blood supply. In the walls of such cysts there are neoplastic (meningeal) cells. There are only few reported cases of this type of meningioma^{4,10-14} and they are not always completely documented.

Peritumoral cysts are not uncommon and are variously located in relation to the meningioma. They usually contain xanthochromic fluid with a high protein content. The origin of these cysts has been a matter of speculation. Some authors suggest that such lesions might represent a loculated widened subarachnoid space or oedema of the surrounding brain.¹⁵⁻¹⁷ Other causes may be demyelination near the tumour or haemorrhage.¹⁸⁻²⁰ The walls of these peritumoral cysts often consist of gliotic proliferation and glial fibrillary acidic protein has been found in them.⁶⁻⁸ In other cases the cyst wall arises from a fibroblastic reaction or consists of the arachnoid space itself.^{8,9,16} Sato²¹ stated that head injury or convulsions do not play a part in the cyst formation. In our opinion, the various possible mechanisms of cyst formation make these two groups of cystic lesions non-

Fig CT scan showing a low-density left parietal parasagittal area with a peripheral contrast enhancement.

homogeneous and it is better to classify them separately, both histologically and as regards the clinical behaviour. We suggest that cystic meningioma should be defined as tumour with an intratumoral cyst whose walls are composed of meningeal cells; the other kind of cysts can simply be considered as associated with a meningioma.

Both types of neoplasms are diagnostically confusing. Cerebral angiography may reveal a meningeal blood supply to the tumour as well as an avascular mass (as in our case). The CT differential diagnosis may also be difficult, particularly as regards gliomas, glioblastomas or hemangioblastomas.^{10,11,22}

The CT features that can lead to a misdiagnosis are the presence of irregular areas of non-enhancing lesions and of regions of persistent low density.²² Russel *et al*²³ stated that a CT misdiagnosis occurs in 7% of cases of meningioma.

There are few features that can lead to the diagnosis of meningioma, such as the clinical history, the site of the tumour and the possible evidence of a meningeal blood supply.

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Stroke due to atrial myxoma in a young woman with co-existing acoustic neuroma and pituitary adenoma

Sir: Cardiac myxomas are uncommon benign tumours with a gelatinous friable consistency. They may present in a wide variety of ways and are difficult to diagnose clinically owing to the lack of specific signs.¹ The majority present with features of atrioventricular valve obstruction, systemic embolism, or constitutional disorder such as fever, malaise, leucocytosis, and an elevated erythrocyte sedimentation rate. Seventy-five per cent occur within the cavity of the left atrium, when systemic embolism is a major risk.² Emboli to a wide variety of sites have been reported, some being massive, and complications include infarction and mycotic aneurysm formation.³ Cerebrovascular embolism is a relatively common mode of presentation.⁴

A 28-year-old woman was referred three weeks after suddenly developing slurred speech, diplopia, paraesthesiae on the left side of the face, and weakness of the left arm. She had vomited initially and developed a left sided headache later. Poor hearing in the left ear was noticed following a head injury three years before this presentation. Otherwise she had recently been well, was on no medication and did not smoke. Examination revealed acromegalic facies and hands. There were splinter haemorrhages in the fingers. She was normotensive and in sinus rhythm. Neurological examination revealed nerve deafness in the left ear, and mild left finger-nose ataxia. Power was reduced in the left leg, and the left calf was wasted following childhood poliomyelitis. Clinically a brainstem stroke was the initial

diagnosis, but plain and computed tomography showed a large left acoustic neuroma complicated by hydrocephalus, and enlargement of the pituitary fossa. Following a decompressive shunt the acoustic neuroma was removed, and transthemoidal hypophysectomy was performed for a chromophobe adenoma during a later admission. Her subsequent progress was good and she returned to her job in Greece.

Two years later she was again referred, after suffering a major dominant hemisphere stroke, with dysphasia, facial weakness and a dense right hemiplegia. The heart was clinically normal, and the electrocardiograph within normal limits. Computed tomography showed a left hemisphere infarct, and angiography showed complete occlusion of the left internal carotid artery. In view of the probable embolic nature of the stroke and the history of splinter haemorrhages, echocardiography was performed and demonstrated a large left atrial myxoma. This was removed under cardiopulmonary bypass, and since then she has been making a slow but encouraging recovery from her stroke.

The initial clinical diagnosis in this case was brainstem cerebrovascular accident, and retrospectively this is still possible although the acoustic neuroma and hydrocephalus were the likely cause. Because of the rarity of cardiac myxoma little is known about associations with other conditions. In addition it does not qualify for registration in cancer registries which are gathering valuable epidemiological information on malignant tumours, including the coincidence of multiple primaries.⁵ Pituitary adenomas and acoustic neuromas occur as elements of multiple tumour complexes, for example, in the multiple endocrine adenopathy syndrome and neurofibromatosis respectively. However, although there is an association between cardiac myxoma, pigmented skin lesions and myxoid neurofibromata,^{6,7} there is no recognised association of these myxomas with other primary tumours.

When a cardiac cause for systemic or cerebral emboli is suspected, two dimensional echocardiography provides a reliable and noninvasive investigation which may demonstrate treatable causes, including endocarditis, mural thrombus, mitral stenosis and cardiac myxoma. However the yield is small unless there is already clinical evidence of heart disease.^{8,9} Even in this selected group, few patients have a myxoma; one case was found in 138 patients with suspected focal cerebral ischaemia,⁸

and one in 110 patients referred with suspected systemic embolism.¹⁰ Maroon and Campbell discussed features which should alert the physician to the presence of a cardiac tumour in patients with cerebral embolism.¹¹ (1) a patient aged under 30 years in whom endocarditis has been excluded; (2) normal cardiac rhythm and no history of cardiac disease; (3) a cardiac dysrhythmia or murmur which varies with time and posture; (4) hyperglobulinaemia, elevated sedimentation rate, anaemia and leucocytosis with constitutional symptoms; (5) myxomatous tissue in an excised embolus; and (6) the finding of a filling defect or pseudoaneurysm on cerebral angiography. Now that surgery provides a potential cure for cardiac myxoma,¹² although follow up is advisable, it is vital that the diagnosis is considered and confirmed as early as possible.

The neurosurgical operations in this case were performed by Mr MJ Torrens, and the cardiac surgery by Mr G Keen.

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