Table Lumbar puncture results:

	Day 1	Day 2	Day 18
Appearance	turbid fluid	slightly xanthochromic	clear fluid
Pressure cm	23	23	—
Protein g/l	3.4	4.8	.9
Globulin	mod increase	mod increase	mod increase
Glucose mmol/l	—	—	—
C.S.F. (serum)	4.5 (14.2)	3.5 (13.3)	2.8 (9.0)
Chloride mmol/l	113	—	118
W.C.C. per mm ³	200	100	13
Polymorphs %	66%	—	95%
Lymphocytes %	34%	—	—
R.C.C. per mm ³	180	200	237
Microscopy (Gram and Z.N.)	Negative	Negative	—
Culture	<i>C. pyogenes</i>	<i>C. pyogenes</i>	No growth

scopy of the CSF. No improvement occurred in the next 18 hours and ampicillin and benzylpenicillin were added to the treatment. Twenty-four hours later a gram positive rod appeared on culture and after consultation with the microbiology department, Septrin was prescribed and chloramphenicol was withdrawn. Over the subsequent 24 hours, although he was pyrexial, his conscious level improved. The organism, *C. pyogenes* was shown to be sensitive to Septrin but resistant to benzylpenicillin and ampicillin. However, with the patient's improved clinical state no alteration was made to his drug treatment. He became apyrexial after a further 24 hours and by the ninth day on Septrin was fully conscious. All medication was withdrawn with the exception of Septrin, which was stopped after a negative lumbar puncture the following week (table). Throughout the infection blood cultures were repeatedly negative. The patient was discharged one month after admission following mobilisation.

Some samples of CSF were inoculated into Brewers medium and Robertsons meat broth. The remainder was spun down and streaked onto horse blood agar and chocolate agar kept in 5–10% CO₂. The culture plates were incubated at 37°C. After overnight incubation small pin-point, round, smooth colonies with zones of β haemolysis were isolated in pure culture on blood agar and growth was also seen on chocolate agar plates. The organism was also isolated from sub-culture from Robertsons meat broth. Microscopy of the cultured organisms showed straight gram-positive bacilli with few filamentous forms which stained uniformly and contained no spores. They were non-motile (by the hanging drop method, 22°C and 37°C) and the identification of *C. pyogenes* was based on biochemical tests. A relatively common cause of meningitis, by a gram-positive rod,

is *Listeria monocytogenes* which was excluded by the motility and biochemical tests. These were checked in duplicate in peptone-water sugars with added serum and serum agar sugars from the agar plates and Robertsons meat broth. Catalase, oxidase, indole production, Vogues-Proskauer and acid production from mannitol were negative. The oxidative-fermentative test was fermentation positive and acid production occurred from glucose, lactose, maltose and sucrose. Antibiotic sensitivity tests were carried out on Sensitest agar by paper disc technique. The organism was sensitive to vancomycin, rifampicin, gentamicin, netilmicin, Septrin, trimethoprim, sulphamethoxazole, tetracycline and erythromycin. The organism was resistant to penicillin, ampicillin, chloramphenicol, fucidin, cephaloridine and clindamycin. The MIC of trimethoprim was reported for veterinary strains of *C. pyogenes*¹ as 0.1–10 µg/ml. The concentration of trimethoprim in the CSF of this patient on day 18 was 2.3 µg/ml. *C. pyogenes* has been reported as the causative organism for an increasing list of human disease including subacute bacterial endocarditis, septic arthritis,² empyema,³ abscess formation, complication of frostbite and vulvo-vaginitis.⁴ A report from the National Collection of Type Cultures⁵ stated that six isolates of *C. pyogenes* had been received since 1965, but the sites of isolation were not stated of the species of *Corynebacteria* reported to the PHLS Communicable Disease Surveillance Centre, Colindale, between 1975 and 1982, this is the only case of *C. pyogenes* isolated from CSF.⁶ In this patient the portal of entry and source of infection remained obscure. However, the patient was a retired farmer who had worked with livestock. It was noteworthy that *C. pyogenes* was resistant to the first line antibiotics for meningitis although the organ-

ism has a wide range of sensitivity including Septrin, which was used in the treatment of this patient.

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Primary meningeal melanomatosis: limitations of current diagnostic techniques

Sir: Primary melanomata of the central nervous system are exceedingly rare except in association with giant pigmented cutaneous naevae in children.¹ Their origin is still debated although it is most likely that they arise from melanocytes in the leptomeninges.² However, even after careful post mortem examination, subclinical primary lesions of the skin, choroid, urinary or gastro-intestinal tract cannot be absolutely excluded.

Recent reports have stressed the usefulness of computed tomography (CT) in the diagnosis of tumours involving the leptomeninges and in particular in raising the possibility of melanomatosis.³ Likewise cerebrospinal fluid (CSF) cytology is extensively reported to be of use in the early diagnosis of meningeal malignant infiltrations.⁴ The present case illustrates the difficulties of making an early diagnosis despite the use of both the above mentioned diagnostic techniques.

A 68-year-old retired English milkman presented in January 1982, with a one year history of numbness initially in the right foot which spread to involve the whole leg and weakness in the right leg for two

Figure Metrizamide radiculogram showing numerous rounded filling defects in close association with the roots of the cauda equina.

months prior to presentation. Hearing in the right ear had deteriorated in association with progressive double vision on looking to the right as well as intermittent right sided facial paraesthesiae. Initial examination revealed a partial 3rd and complete 6th and 8th cranial nerve lesions on the right, in addition to a mild pyramidal distribution paraparesis with bilateral extensor plantar responses. Sensation in the right leg was diminished to all sensory modalities. Investigations at that time including an enhanced CT scan were unremarkable apart from an elevated CSF protein of 4.83 g/l which contained no malignant cells. A clinical diagnosis of an intrinsic brain stem glioma was made, and he was treated with prednisolone for four weeks. The patient continued to deteriorate to the point where he was wheelchair bound and had developed a complete right ptosis. On readmission the principal new findings were the complete loss of all lower limb reflexes and a sensory level to pin prick in the lower dorsal region. A repeat CT scan revealed obstructive hydrocephalus but no other definite pathology even after contrast enhancement. Two further CSF examinations failed to detect malignant cells but a metrizamide myelogram showed multiple intradural, extramedullary, rounded lesions, in close association with the roots of the cauda equina. These were thought initially to be metastases or neurofibromata (fig). At this stage, a review of the 3rd CT scan revealed a small area of pathological enhancement in the vermis, indenting the roof of the 4th ventricle. Therefore a posterior fossa exploration was performed, which revealed numerous black streaks on the arachnoid, biopsies of which confirmed a diagnosis of

malignant melanomatosis. Tumour mass was seen on the surface of the right cerebellar hemisphere. Despite careful fundoscopy and clinical examination no evidence of other melanotic lesions were found. Melanuria was not detected. The patient died two months later despite craniospinal irradiation. There was no post mortem examination.

It is likely that this tumour originated in the posterior fossa leptomeninges and spread into the substance of the brain stem and the cerebellum as well as via the CSF to the cauda equina. It is surprising that despite clear evidence of multiple meningeal lesions and of supposed CSF tumour spread, that repeated examinations of the CSF for malignant cells were negative. Although in isolated cases melanoma cells have been detected in CSF,⁸ no information is available on the detection rate, as has been published for gliomas and secondary carcinomatosis.⁹ "Coal black" CSF was obtained in one case of spinal cord primary melanoma. Post mortem examination of this case revealed numerous tumour nodules throughout the cauda equina region.¹⁰ Abnormal CT findings have been reported in four cases of primary leptomeningeal melanomatosis.^{6,11,12} In three cases dense meningeal enhancement was reported, whilst in the fourth case CT revealed a low density frontal lesion without meningeal enhancement despite subsequent surgical evidence of melanotic meningeal invasion.¹² In the present case, in spite of extensive posterior fossa meningeal involvement with tumour, only a small area of contrast enhancement was seen in the vermis on review of the final CT scan.

The diffuse pattern of meningeal melanomatosis reported in the current case and seen in various other cases of primary nervous system melanomas is in sharp contrast to that found in metastatic melanomata. In a recent series of 33 cases of cerebral metastatic melanomatosis, 20 showed a solitary space occupying lesion, 12 multiple lesions and in only one case was diffuse meningeal disease observed.¹³ When melanomata exclusive to the nervous system present with spinal cord or root symptoms an early preoperative diagnosis is usually made.³ However, intracranial presentations as in our case are difficult to diagnose and often remain undiagnosed pre-mortem¹⁻² despite modern techniques. We thank Dr JN Blau for permission to report this case.

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Subarachnoid haemorrhage and nasal vasoconstrictor abuse.

Sir: Recently, several arterial abnormalities attributed to abuse of nasal sympathomimetic drugs have been described. Fendler *et al*¹ reported the case of a woman with arteriographic changes in renal arteries and severe hypertension after taking fenoxazoline HCL in nasal spray for