intact lateral branch of the nerve was found which was now resected. The main nerve trunk was cut as proximally as possible (several cm). After this new operation the hyperaesthetic area was considerably smaller although still present. However, within a few weeks the size of the hyperaesthetic area again grew and reached the original size. Three months after the reoperation the area of the left lateral femoral cutaneous nerve was so painful that the patient had much trouble wearing clothes. Detection thresholds tested with a piece of cotton were higher on the affected side; but even a light touch with fingers produced considerable pain. The patient did not discriminate between warm and cold in the affected region. There was no false localisation.

There remained a small oval area (diameter about 2 cm) in the middle of the affected region which was totally anaesthetic. In addition, the patient had another, qualitatively different, lancinating pain which disappeared after injection of local anaesthetics at the inguinal level; therefore this pain was considered to be due to a neuroma. Even a massive injection of local anaesthetic at the inguinal level did not affect the hyperaesthesia in the left thigh. Analgesics, carbamazepine, amitriptyline, propranolol and guanethidine proved ineffective. Transcutaneous electrical nerve stimulation applied to the right (normal) thigh made the pain and hyperaesthesia worse, even when it was applied at low intensities. Sympathetic blockade at the L2-4 level produced temporary relief of the lancinating component of the pain.

Regeneration of the resected nerve does not explain the present findings. At reoperation it was found that the resected nerve branch did not reach the periphery and, moreover, it was sectioned again. A plausible explanation could be the phenomenon described by Devor and Wall in rats and cats:^{1,2} transection of a peripheral nerve produces a reorganisation of the receptive fields of the spinal dorsal horn neurons. This reorganisation is based on unmasking of normally present but ineffective afferent terminals, through which the dorsal horn neurons receive impulses from the neighbouring intact skin nerves after the transection. Trophic mechanisms most probably have a major role in this unmasking,^{1,2} although other kinds of mechanisms, such as the inhibition by the lateral division of Lissauer's tract,³ may contribute. The central connections of the corresponding dorsal horn neurons remain intact if the transection is made distally to the soma of the primary afferent neurons.⁴

In the present case a nerve lesion at the peripheral level had first produced meralgia paraesthetica. After nerve resection, the dorsal horn neurons which normally mediated signals from the resected nerve began to mediate signals from the neighbouring intact skin nerves which either had pre-existing overlap of innervation areas covering the denervated region or, more probably, had sprouted to the denervated area.⁵ Small diameter afferents presumably are a dominant group in the sprouting nerves (cf ref. 5) which could explain the higher detection thresholds in the hyperaesthetic region. The production of pain by light touch could have been caused by lack of afferent inhibitory control at the spinal level. Lack of false localisation supports the theory that signals from the hyperaesthetic region are mediated via second order neurons of the affected nerve. Our case resembles the cases described by Noordenbos and Wall⁶ and reconfirms their recommendation that a resection should not be done in this kind of condition.

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Cryptococcal meningitis for 15 years

Sir: A case of cryptococcal meningitis is reported where the diagnosis was made, after 14 years of a chronic relapsing illness, by the isolation of *Cryptococcus neoformans* from the CSF. The organism was non-encapsulated, a finding that may be related to the relatively benign clinical course.

Mr C was an Indian, born in Bombay in 1934, who came to this country at the age of 26 years. He first presented in September 1966 with headaches and a left lateral rectus palsy which resolved after three weeks. At that time chest and skull radiographs, ESR, WR and full blood count were all normal. In June 1967 he was seen with a right hemiparesis of sudden onset which subsequently improved. Routine biochemical and haematological tests were again normal but his cerebrospinal fluid showed a protein of 1.6 gm/l, WBC 70/mm³ mainly lymphocytes, and a glucose of 1.0 mmol/l. An air encephalogram, EEG and isotope brain scan were normal. CSF culture was negative. No firm diagnosis was made at this time but he remained generally well; he was seen once in 1971 with sacro-iliac pain which settled spontaneously, but was then lost to follow-up until 1978. In 1978 he presented with an episode of vertigo related to head posture which improved over four weeks. At that time he had mild residual right upper motor neurone signs with bilaterally brisk reflexes. In August 1979 he had some perioral and right arm paraesthesiae which lasted for a few days. Following this a CT scan showed a communicating obstructive hydrocephalus with symmetrical enlargement of the third, fourth and lateral ventricles but no other lesion. Again routine biochemistry, haematology, skull and chest radiographs were normal, but the CSF showed a pressure of 14 cm of CSF, protein 1.3 g/l, WBC 20/mm³ mainly lymphocytes and a glucose of 0.6 mmol/l (blood glucose 4.3 mmol/l); culture, cryptococcal antigen and India ink stain were all negative. A Mantoux test was positive and a Kveim test negative, making sarcoid unlikely. He was then started on anti-tuberculous chemotherapy comprising streptomycin 1G, ethambutol 1G, rifampicin 600 mg, isoniazid 300 mg and pyridoxine 50 mg daily. However, by January 1980 all TB cultures had proved negative and he had developed visual impairment, numbness of his feet and vertigo possibly related to the ethambutol, isoniazid and streptomycin respectively. The anti-tuberculous chemotherapy therefore was stopped. In February 1980 he relapsed with malaise and headaches, so was started on prednisolone 60 mg a day

together with isoniazid and pyridoxine. The dose of steroids was gradually reduced and there was little symptomatic or objective change. Four further CSF specimens before July 1980 were abnormal with raised lymphocyte count and low sugar but, again, no organism was grown, cryptococcal antigen and India ink stains were negative as were tests for cryptococcal antigen using latex particles coated with immune globulin. He then had an episode of loss of consciousness and an electroencephalogram at that time showed generalised slowing with a few episodic forms over the temporal areas; he was started on phenytoin when his prednisolone had been tailed down to 4 mg a day. On 22nd July 1980 a repeat CSF grew one colony of a non-encapsulated *Cryptococcus neoformans*. Two subsequent CSF's grew several colonies of the same organism, and cryptococcal antibody against this organism was present in the patient's serum at a titre of 1 in 8 using a fluorescent antibody technique. Cryptococcal antigen tests remained negative because the organism was non-encapsulated. He was then started on a six week course of anti-cryptococcal chemotherapy consisting of amphotericin B 0.3 mg/day as an intravenous infusion and 5-fluorocytosine 150 mg/kg/day orally in divided doses, while continuing on 15 mg prednisolone. Further specimens of CSF showed growth of cryptococci two weeks after starting chemotherapy, but not at four weeks or subsequently, and his CSF glucose levels began to rise. The most recent CSF (in February 1982) showed a pressure of 110 mm of CSF, protein 0.69 gm/l no WBC's and a glucose of 2.9 mmol/l (plasma 5.3 mmol/l). At that time he was off steroids, had no headache and felt that his general health was much improved. He still had mild signs of his old right hemiparesis.

This case illustrates some of the problems involved in diagnosing cryptococcal meningitis. Mr C had an abnormal cerebrospinal fluid for 14 years before a culture diagnosis was made. The response to treatment (in particular the rise in glucose and disappearance of white cells from the cerebrospinal fluid) provides strong evidence that the entire illness was due to chronic cryptococcal meningitis. The prompt response to antifungal chemotherapy, but not to therapy with steroids, make it unlikely that the cryptococcal infection was secondary to some other underlying disease; in particular, sarcoid^{1,2} seems unlikely in view of the negative Kveim test and positive Mantoux.

It is interesting that this man had a

number of CSF examinations over the years which gave not only negative results for cryptococcal antigen and India ink stains (explicable by the absence of capsule from the organism eventually isolated) but also negative cryptococcal culture. Persistent³ and intermittent^{4,5} negative CSF culture in patients with active cryptococcal meningitis has been reported previously. It seems reasonable to assume that here it was the steroids which enabled the organism to grow in culture in view of the temporal relationship between starting the steroids and the first positive culture.

The case is also unusual in the long duration of the untreated disease with only slow deterioration. Reports before any specific treatment was available suggested that up to three quarters of patients died in the first year of their illness,⁶ although there were very occasional cases with intermittent symptoms and persistent meningeal reaction for up to 30 years.^{4,7} It is interesting to speculate that in Mr C's case this may be related to the fact that the organism was non-encapsulated. It is thought that pathogenicity of cryptococci may be related to the capsule; non-encapsulated strains have been shown to be less pathogenic in mice⁸ and studies suggest that a thick capsule impairs killing by neutrophils.⁹ The low level of cryptococcal antibodies may be related either to the lack of capsule or to the chronicity of the disease.

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The reliability of clinical assessment of Parkinson's disease

Sir: The subjective assessment of disability of patients with Parkinson's disease has generally been based on a variety of rating scales of symptoms and signs, or of functional disability.¹⁻³ These clinical rating scales have often formed the basis of both single centre and multicentre clinical trials of new drugs for this disease. They permit the conversion of clinical features to numerical scores for mathematical purposes. Since the statistical evaluation of any such trial depends on the reliability of the scorers, it is important that there should be inter- and intra-observer consistency.

We recently had the opportunity to evaluate this point using the commonly performed Webster rating scale.¹ The performance of seven patients with idiopathic Parkinson's disease on seven of the ten listed items (bradykinesia of hands; posture; upper extremity swing; gait; tremor; facies; speech) was prerecorded on video tape. Nineteen practising consultant neurologists, all of whom had some experience using the Webster rating scale, participated. Each neurologist had available a full description of the scale. For each patient a video recording illustrating the seven items was presented and then scored in the order described above. The first session took place in the morning (Session 1) and the same procedure was repeated some eight hours later (Session 2). The Parkinsonian patients were mildly or moderately affected, the mean total score ranging from 2.4-11.1 out of a possible maximum of 21. There was considerable observer variability, three