tinued tuberculostatic treatment he has remained well. In order to attend hospital for follow-up examination almost 2 years after original presentation, he drove his motor car through city traffic. His only disability is mild dysarthria from spasticity of the tongue. All the other neurological abnormalities have resolved. At the recent attendance CT showed only some cerebral atrophy, most pronounced in the Sylvian areas of both hemispheres, more on the right side.

The operculum syndrome illustrates that cortical innervation of muscles supplied by the 5th, 7th, 10th, and 12th cranial nerves is variably bilateral. Most cases result from lesions in the operculum of both hemispheres.[1] The earlier lesion may pass unnoticed, the syndrome only manifesting when a second lesion develops in the opposite hemisphere.

In our patient the syndrome presented when he developed an abscess in the opercular area of his right hemisphere. We must presume that the relative atrophy in the left opercular area represents a pre-existing vascular or congenital lesion. The retained ability to wrinkle the forehead despite biolateral lower facial paralysis must illustrate the supranuclear control of upper facial movements is relatively remote from those of the lower face. The ptosis must have been an example of "cortical ptosis" which has been described in pseudobulbar palsy.[3]

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Serum prolactin evaluation after "minor" generalised seizures monitored by EEG

Sir: Serum prolactin levels have been widely investigated after generalised tonic-clonic and partial seizures.[1–8] It is now well known that a significant increase of prolactin occurs after both generalised tonic-clonic and complex partial seizures, while in the majority of patients with simple partial seizures (that is, without loss of consciousness) no significant alterations in postictal prolactin levels are found.

At the present time there is little information about the effect on prolactin release of generalised seizures as distinct from tonic-clonic ones ("minor" generalised seizures). In particular, postictal prolactin levels have been investigated only after three myoclonic seizures,[5–7] two simple absence attacks[6 8] and one akinetic seizure;[7] in none of these seizures was any significant rise of postictal prolactin blood levels found.

We examined prolactin levels after seven "minor" generalised seizures (three myoclonic seizures, four absence attacks) in seven female patients affected by primary generalised epilepsy. Mean age of the group was 22 years (range 11–36). Patients 1, 2 and 3 had myoclonic seizures and tonic-clonic seizures. Patients 4, 5 and 6 had simple absence attacks and rare tonic-clonic seizures. Patient 7 had complex absences and very rare tonic-clonic seizures. Informed consent was obtained in all cases.

In each patient the study was performed in the morning, after overnight fasting, under continuous EEG monitoring. A heparin lock was placed in an accessible vein. After having obtained a 30-minute EEG recording devoid of any epileptiform activity, a blood sample for baseline prolactin values was obtained. Successively, in each patient a specific stimulation was used to trigger a seizure.

Patients 1, 2 and 3 received 10 short runs of photic stimulation at 15 flashes/s, each lasting 4 seconds and separated from the others by a 2 second interval. In every patient each run constantly evoked a generalised discharge of polyspike-waves, accompanied by myoclonic jerks of the limbs, without loss of consciousness. In each patient the total duration of myoclonic jerks was 40 seconds.

In patients 4, 5 and 6 eye closure was followed after a few minutes by a simple absence attack, lasting about 10 seconds, and accompanied by a generalised discharge of spike-and-waves at 3/Hz.

In patient 7 hyperventilation evoked an atonic absence which lasted 60 seconds and was accompanied by a generalised discharge of polyspikes and spike-and-waves at about 3/Hz.

In patients 1, 2 and 3 the postictal samples were obtained 15 minutes after the last photic-induced myoclonic jerk. In patients 4, 5, 6 and 7 the postictal samples were obtained 15 minutes after cessation of the absence. Postictal levels were compared with the values obtained in basal conditions. Statistical analysis was carried out using the paired Student's t test. No significant changes from the baseline values were seen in any of the postictal samples.

Our preliminary results indicate that there is no evidence for "minor" generalised seizures having postictal prolactin elevations. This finding has been only occasionally reported in the literature,[5–8] without any attempt to give a physiopathological explanation.

On the other hand, even the mechanisms underlying the well documented prolactin elevations which follow generalised tonic-clonic and complex partial seizures are still unclear. In a recent report[7] it is suggested that once the entire brain is involved by a paroxysmal discharge, regardless of how the neuronal pathways have been activated, there is a rise of serum prolactin. However, in the same paper no postictal hyperprolactinaemia is found after two generalised seizures, one myoclonic and the other akinetic: the authors attributed the lack of prolactin rise to the brief duration of these attacks. Our observations do not agree with this interpretation since no rise of postictal prolactin level was found although in four cases (patients 1, 2, 3 and 7) the seizure was of long duration. Other authors have invoked loss of consciousness as a possible contributing factor leading to postictal prolactin rise; however, this possibility also seems to be ruled out by our observations, since four patients (Nos 4, 5, 6 and 7) suffered loss of consciousness (in one case of long duration) without any significant prolactin rise.

It seems most likely that in "minor" generalised seizures paroxysmal activity, although generalised, does not spread within hypothalamic neurons which regulate prolactin release and thus is not accompanied by prolactin rise. This feature seems to be independent of the duration of the seizure and of the occurrence of loss of consciousness. Further work obviously needs to be done before these conclusions can be accepted.

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Nocardia asteroides infection of the cauda equina

Sir: Nocardiosis is most often localised to the lungs and skin, but frequently disseminates to involve the central nervous system. Primary extrapulmonary nocardiosis is extremely rare[1–5] and nocardiosis of the nervous system most commonly presents as a cerebral abscess metastasising from a primary pulmonary focus.[6–8] Purulent meningitis following rupture of a cerebral abscess into a ventricle is well recognised,[9] but primary nocardial meningitis has only been reported twice to our knowledge.[10 11] We describe a patient who initially presented with a rapidly progressive glomerulonephritis in the context of systemic vasculitis. Following successful treatment with prednisolone, cyclophosphamide and plasma exchange, she developed signs initially suggesting a multifocal peripheral neuropathy but then rapidly deteriorated with development of an obvious cauda equina syndrome. *Nocardia asteroides* was recovered from purulent cerebrospinal fluid and a diagnosis of primary nocardial meningitis was made. No extrameningeal focus could be identified and there was no evidence of previous pulmonary involvement. In vitro susceptibility studies demonstrated resistance to sulphonamides, but subsequent treatment with fucidin, amikacin, rifampicin and imipenem also proved ineffective, despite in vitro sensitivity to these agents. The site of infection, absence of an extrameningeal focus, myelographic appearances and antimicrobial sensitivities make this a unique case.

A 53 year old woman with previous good health was admitted to hospital in March 1985 with a short history of malaise, ankle swelling, oral ulceration and polyarthralgia. Shortly before admission she also noted a nasal discharge, sore red eyes, an erythematous rash over her ankles, and decreased urine volume. On examination she was apyrexial and pale. A resolving episcleritis was noted and vasculitic skin lesions were present over the lower legs and feet. The pulse was 80 beats per minute, blood pressure 130/70 mm Hg and a soft systolic murmur was present. The chest was clear and abdominal examination unremarkable. An area of decreased sensation to pinprick and light touch was present over the dorsum of the left foot. Initial laboratory findings revealed a haemoglobin of 10·1 g/dl, white blood cell count of 14,500/mm^3 with 88% neutrophils, and platelet count of 999,000/mm^3. The serum urea was 28·2 mmol/l and creatinine 980 μmol/l. A skin biopsy revealed leucocytoclastic vasculitis and renal biopsy showed focal and segmental glomerulonephritis with fresh crescents in four of the 13 glomeruli present. A diagnosis of rapidly progressive glomerulonephritis, secondary to a systemic vasculitis within the spectrum of polyarteritis, was made. Peritoneal dialysis was commenced, and the patient was treated with prednisolone 60 mg daily, cyclophosphamide 2 mg/kg daily and plasma exchange (five 4 litre exchanges for plasma protein fraction). Her clinical condition rapidly improved and renal function returned to normal. She was discharged on cyclophosphamide (later converted to azathioprine) and reducing doses of steroids, and remained well until August 1985.

She was readmitted with severe lancinating pain in both legs and an increased sensory deficit over the left lower leg. She had weakness of ankle and toe dorsiflexion and plantarflexion on the right and absent ankle jerks bilaterally. Cutaneous sensation was decreased over the dorsum of the left foot, the lateral side of the left lower leg, and the dorsum of the right hallux. Vibration sense was absent below the knees, and proprioception defective at the toes. Sensory nerve conduction studies in the right median and ulnar nerves were normal. The right median motor nerve conduction velocity (MNCV) was slightly reduced at 46 m/s, with a distal motor latency of 2·2 ms and evoked muscle action potentials (MAPs) of only 2·0 and 1·5 mV stimulating at the wrist and elbow. The left sural sensory nerve action potential was absent. Evoked MAPs stimulating the peroneal nerves were reduced in amplitude at 35 (right) and 350 (left) μV, with a MNCV on the left of 35 m/s. A diagnosis of a multifocal peripheral neuropathy secondary to vasculitis was made, and she was treated with increased doses of steroids and reintroduction of cyclophosphamide.

Three days later her condition deteriorated; she developed a moderately severe paraparesis and there was loss of all sensory modalities below T8. The tendon reflexes were absent in the lower limbs, and urinary incontinence and a lax anal sphincter were noted. A histamine flare test showed absent flares in affected dermatomes, suggesting that the site of the lesion was distal to the dorsal root ganglia. A myelogram was normal; the cerebrospinal fluid (CSF) white cell count was 104/mm^3 and glucose concentration was 2·7 mmol/l with a blood glucose of 3·7 mmol/l. Plasma exchange was reintroduced to the treatment regimen without effect.

One week later the CSF white cell count had risen to 1675/mm^3 and the glucose concentration was 1·1 mmol/l. Intrathecal infection was strongly suspected, but no organism was seen on gram stain or cultured. After a further 5 days, despite having commenced broad spectrum antibiotics, she became pyrexial, experienced rigors and developed neck stiffness. A myelogram was performed via cervical puncture which revealed poor filling of the cauda equina with multiple intradural extramedullary filling defects (fig, a). This appearance, together with the cerebrospinal fluid findings (lumbar: white cell count 3000/mm^3, glucose undetectable; cervical: white cell count 204/mm^3, glucose 3·7 mmol/l) suggested a focus of infection in the lumbar region. Under radiographic screening, pus was aspirated from the T12/L1 space. Gram stain revealed gram positive branching filamentous organisms, which were identified as *N. aster-*