the bowel should be surgical and followed by total abdominal radiotherapy. Spinal metastases causing a paraparesis should be in most cases decompressed, biopsied and treated with radio-therapy.

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Periodic EEG pattern in meningovascular syphilis

Sir: The electroencephalographic abnormality of periodic complexes occurs in a heterogenous group of disorders, such as subacute sclerosing panencephalitis, subacute vascular encephalopathy, Creutzfeldt–Jakob disease, herpes simplex encephalitis, anoxic encephalopathy, metabolic encephalopathy, drug intoxication, subcortical arteriosclerotic encephalopathy, human rabies encephalitis and infectious mononucleosis encephalitis.[1–4] The following report of periodic EEG activity in association with meningovascular syphilis is believed to be unique.

The patient, a 32-year-old right handed black Libyan soldier was hospitalised on 22 January 1984 with a six week history of headache and impairment of memory and intellectual capacity. He became inefficient at his work and exhibited nervousness and aggression. A week prior to admission he had two episodes of generalised convulsions after which he lapsed into a state of semi-stupor. He was married and had four children. On examination at the time of admission, he was drowsy, mute and failed to respond to verbal commands. He had neck stiffness. The pupils, ocular movements and the optic fundi were normal. There was a partial right facial paresis. He moved the right upper and lower limbs less, the right sided deep tendon jerks were brisk and the right plantar response was extensor. His temperature was normal; the general physical examination and the rest of the systemic examination revealed no abnormality. Normal investigations included routine haematology, urine analysis, blood biochemistry, radiographs of chest and skull, ECG and echocardiogram. Lumbar puncture yielded clear cerebrospinal fluid (CSF) with a protein content of 1·9 g/l. It contained 23 × 10^6 lymphocytes/l, but no organisms were seen on Gram and Ziehl-Nielson stains. The serum and CSF VDRL was positive at titres of 1:160 and 1:80, respectively. Serum fluorescent treponemal antibody—absorption (FTA-ABS) test using Bio Merieux kit with serum diluted in sorbent in serial dilution was positive at 1:320. The first EEG recording obtained on the second day of admission showed generalised slow wave complexes having a duration of 400–500 ms and an amplitude of 50–150 μV (fig. (A)). They occurred in a periodic fashion at the intervals varying between 1·6 and 2·2 seconds. The discharges were larger in the left temporal region, where they appeared to have the earliest origin, and were least evident in the right temporal region. This implied that they are a variant of periodic lateralised epileptiform discharges (PLEDs). The background activity between the periodic transients showed 4–5 Hz slowing and burst suppression. The EEG repeated three days later still demonstrated the periodic discharges, but at a reduced amplitude. The last EEG recorded on 7 February 1984 did not show the periodic activity. It disclosed alpha rhythm in the background and 6–7 Hz slowing and suppressed voltage over the parieto-temporal region of the left hemisphere. A CT scan done on 2 February 1984 showed an area of decreased density involving left parietal cortex and slight ventricular dilation (fig. (B)). The lesion exhibited no enhancement with contrast.

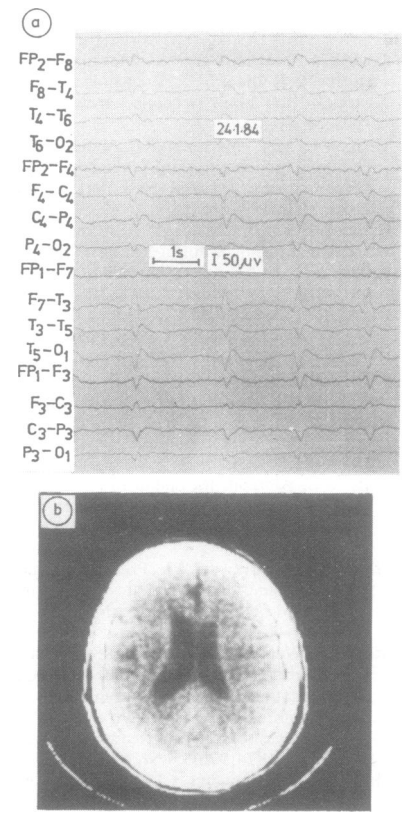

Fig (A) *EEG shows left temporal dominant periodic slow wave discharges.* (B) *CT scan demonstrates ventricular enlargement and left parietal low-density lesion.*

A diagnosis of meningovascular syphilis was made and the patient was treated with high doses of penicillin administered parenterally starting from the fourth day of admission. The patient's progress was satisfactory. He became conscious within a week; however, he remained apathetic and his mentation was slow. During the next two weeks his conversational speech became fluent but empty, with almost no substantive words. Language comprehension was limited to following elementary

commands; repetition and naming were markedly disturbed. When examined one month later his hemiplegia had recovered. His mood was highly labile, he remained confused; the profound loss of memory and the posterior aphasia persisted. Because of the mental and speech disturbances, past history could not be obtained.

The clinical features, CSF findings, positive serological tests and the response to penicillin therapy are diagnostic of meningovascular syphilis in our patient.[5,6] The essential pathological changes in cerebral syphilis are subacute meningitis, vascular and perivascular inflammation involving large and medium sized arteries and gumma formation.[5] The middle cerebral artery or its branches are most frequently the site of syphilitic thrombosis.[5] The CT scan in the present patient showed an infarct in left parietal lobe.

PLEDs are a much more common phenomenon than true bisynchronous generalised discharges and occur most often with acute unilateral lesions such as infarcts or tumours.[7-9] In the present instance their origin correlated with the lesion evident clinically and confirmed in the CT scan. The evolution described coincides with the usual natural history of PLEDs.[8] Pathophysiologic mechanisms responsible for the periodicity in the EEG are unknown. Recent studies favour the concept of a disturbance involving both cortical and subcortical structures.[9,10] The EEG pattern seen would appear to be dependent on the extent and location of the insult rather than the type.

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Notices

Sixth Prague International Symposium of Child Neurology. This Symposium will be held 2–4 July 1985. Further information may be obtained from The Secretariat, PO Box 88, tr. Vitezneho unora 31, 120 26 Praha 2, Czechoslovakia.

4th International Child Neurology Congress of the **International Child Neurology Association** Jerusalem, Israel, 16–20 March, 1986.

Further information may be obtained from Congress Secretariat—ICNA, P.O. Box 29313, Tel Aviv 61292, Israel.

Tenth International Congress of Neuropathology will take place in Stockholm, Sweden, 7–12 September, 1986. For further information contact Tenth International Congress of Neuropathology, c/o Stockholm Convention Bureau, P.O. Box 1617, S-111 86 Stockholm, Sweden.