few encephalitis slow waves, less than usually up to pattern.2

The disease was strongly positive. Paul-Bunnell was positive twice in a titre of 1:112. VDRL and Weil-Felix tests were negative. Initial EEG showed diffuse disturbance with theta waves and some bursts of high amplitude slow waves, compatible with encephalitis. The patient recovered within a few days. The fever subsided and liver function tests returned to their normal levels. The seizures did not recur; however, in a repeat EEG, although no longer a slow-high activity could be detected, two episodes of short bursts of spike and wave pattern were recorded on hyperventilation. In view of the obvious diagnosis and the quick recovery, spinal tap was not performed. The patient was discharged with instructions to take phenytoin 300 mg/day.

Although the incidence of neurological manifestations in infectious mononucleosis ranges from 0-37% to more than 20%, the largest series showed less than 1%.1-3 The main types of neurological complications are: meningo-encephalitis, encephalitis, peripheral neuritis, Guillain-Barré syndrome and spinal cord disease.1-3 The neurological involvement may precede or follow the common manifestations of fever, lymphadenopathy and splenomegaly by as much as several weeks. In a few cases of infectious mononucleosis the only clinical signs that appeared were related to the nervous system. The pathological changes in the brain consist of inflammatory lesions with dense perivascular cuffing and diffuse infiltration of the parenchyma mainly with typical mononuclear cells.4

Infectious mononucleosis is the cause of up to 5% of all cases of acute viral encephalitis.5,6 It is not yet clear whether the encephalitis of infectious mononucleosis is due to direct invasion of the brain or represents a remote effect of the viral infection. The occurrence of seizures during the course of infectious mononucleosis encephalitis is uncommon, and in fact only less than 20 cases have been recorded so far in the English literature.1,4,7 Only in a few of these cases were the seizures the major presenting sign of infectious mononucleosis, as it was in our case. The EEG usually demonstrated increased activity of slow waves, predominantly theta waves, appearing either in paroxysms or in a more continuous pattern.7 The disease is usually self limited. The exact prognosis of the neurological complications is difficult to ascertain. Nevertheless, some reviews indicate a mortality rate of 8% and residual neurological deficits in 12% of the patients.8 We advised the patient to take phenytoin, a therapeutic regime which was also recommended by others.7 The possibility that the seizures which appear during the course of infectious mononucleosis actually express a quiescent tendency of epilepsy that was activated by the viral disease cannot be ruled out. The focal spike and wave pattern which was demonstrated in EEG after the clinical recovery of our patient may hint at that possibility. On the other hand, there could be of course a focal lesion induced by the virus itself. In another encephalitis case presented by Ruutu et al, a transient localised lesion was demonstrated in CT scan.10

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CT scan evidence of postero-lateral thalamic infarction in pure sensory stroke

Sir: Pathological studies have shown that the postero-lateral thalamic nucleus is the most frequent site of infarction in pure sensory stroke.1 The CT scan is commonly normal in these patients,2 and none of the few reported cases with positive CT findings had a single hypodense lesion limited to the thalamus.3 In the following case of pure sensory stroke, CT scan revealed a small infarction in a site believed to be that of postero-lateral thalamic nucleus.

A hypertensive right-handed man, aged 51 years, with no previous history of cerebrovascular disease, experienced the sudden onset of numbness and paraesthesias involving the whole left side of the body. Neurological examination revealed distal hypaesthesia of the left limbs without motor or visual impairment, dysphasia or dyspraxia; tendon reflexes were increased on the left side. A CT scan showed a small hypodense lesion in the lateral part of the right thalamus, 5 mm in diameter (fig.). Cerebral angiography showed tortuosity of intracerebral vessels without significant stenosis. Symptoms gradually disappeared within four days. A second CT scan, performed three weeks later, was unchanged.

Fig. CT scan showing small lacune in the right postero-lateral thalamus.
According to a recent review by Fisher, pure sensory stroke is "the most common lacunar manifestation". However, few cases have been reported with positive CT findings. This discordance may be explained by the small size of these lacunes, which are unresolved by CT scanners. On the other hand, although larger lesions may be limited to the posterolateral part of the thalamus, as in our patient, adjacent structures are probably involved as well in most cases, giving rise to more than purely sensory symptoms.

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The Montreal Neurological Institute and Hospital. The 50th Anniversary celebrations will be held 23-26 September, 1984. Information concerning the symposia, lectures, films, etc., can be obtained from: Synapse-50, Montreal Neurological Institute and Hospital, 3801 University Street, Room 638, Montreal, Quebec, H3A 2B4 Canada.

Correction

Spinal blastomycosis—case report

It is regretted that in this letter (J Neurol Neurosurg Psychiatry 1984;47:217) the figure legend was incorrect. It should be: "Thickly encapsulated PAS positive budding yeasts with broad based attachment between daughter yeasts typical of blastomycosis."

Notices

The Volvo awards for low back pain research

In order to encourage research in low back pain, the Volvo Company of Göteborg, Sweden, also this year has sponsored three prizes of US $6000.00 each. Awards will be made competitively on the basis of scientific merit in the following three areas: (1) Clinical studies, (2) Bioengineering studies, (3) Studies in other basic science areas. Enquiries should be addressed to Professor Alf L Nachemson, Department of Orthopaedic Surgery I, Sahlgren Hospital, S-413 45 Göteborg, Sweden.