interesting part of this patients' clinical presentation was the circling movements. Rotational movements occurred as a transient epileptic phenomenon in the patients described by Schneider et al.\(^1\)\(^{14}\) In the present case they persisted for nearly one month, abated slowly, and were not associated with EEG evidence of seizure activity. In these respects, our patient probably is unique. The most effective lesion in monkeys to cause circling is in the neighbourhood of the vestibular area in the temporal operculum.\(^1\)\(^{17}\) Irritation of the vestibular cortical area, releasing the vestibular cortex from the inhibitory effects of other cortical regions which have been destroyed, or lesions at the association pathways have been shown to cause circling movements in animals.\(^1\)\(^{17}\) The circling towards the side of the lesion exhibited by the monkeys following unilateral ablation of the frontal cortex is presumed to be due to the removal of frontal lobe inhibition over the homolateral vestibular cortical area.\(^8\) In our patient with predominant left anterior temporal lesion, the persistent circling to the left may be due to a similar mechanism.

**Figure (B)** CT scan shows bitemporal non-enhancing low-density lesion, much more on the supero-anterior part of the left temporal lobe.

**Opsoclonus in a confirmed case of St Louis Encephalitis**

Sir: During the 1980 outbreak of St Louis Encephalitis, there were 56 confirmed and 17 suspected cases in Harris County, Texas. One of the confirmed cases had opsoclonus.

A 27-year-old white male was admitted to hospital on July 22, 1980, after being found comatose in a city park. In retrospect later, the patient reported moving to Houston from Illinois in late June, 1980. He remembered a few days history of headache and sleepiness prior to admission. The patient had a temperature of 104°F. He was unconscious with only symmetrical withdrawal of the extremities to painful stimuli. No abnormality of the cranial nerves was found. The deep tendon reflexes were normal, but both plantar responses were extensor. Lumbar puncture on the day of admission revealed an opening pressure of 36 cm water. The cerebrospinal fluid (CSF) glucose was 0·8 g/l (simultaneous blood glucose, 112), and the protein was 1·01 g/l. There were 32 white cells (13 neutrophils and 19 lymphocytes) and 7 red cells. Routine, AFB and fungal stains and cultures were negative. Counterimmune electrophoresis and CSF cryptococcal antigen were negative. A CT scan of the head was normal, but a chest radiograph showed a right lower lobe pneumonia. The patient initially was felt to have an aspiration pneumonia and a viral encephalitis. He was treated with gentamicin and penicillin G for the pneumonia, but later was switched to nafcillin when sputum cultures grew *Staphylococcus aureus*. Two days after admission, the patient was lethargic and oriented to person only. Extraocular movements showed opsoclonus. The plantar reflexes were flexor. Repeat lumbar puncture gave an opening pressure of 22 cm of water. CSF showed a glucose of 0·62 g/l, protein 0·57 g/l, 70 white cells (64 lymphocytes and 6 neutrophils), and 35 red cells. Routine, fungal, and AFB cultures again were negative. An EEG was diffusely slow and poorly organised. Titres for St Louis Encephalitis rose fourfold or more. On July 25, the haemagglutination inhibition (HI) titre was 1/160, and the comple ment fixation (CF) titre was 1/64. On August 1, the HI titre was 1/640, and the CF titre was 1/512. Western blotting for Equine Encephalitis and Dengue Type serum titres were negative. The pneumonia was resolved and nafcillin was discontinued after completing a ten day course. The patient's mental state gradually cleared. By August 6, the patient's mental state had returned to normal. The opsoclonus had disappeared, but ocular flutter was observed. Also noted was mild ataxia of right heel-to-shin testing and unsteadiness on tandem gait. The patient was discharged on August 7. Follow-up clinic visit at the end of August revealed a few beats of horizontal gaze jerk nystagmus bilaterally, occasional ocular flutter, and ocular dysmetria with both eyes. Opsoclonus was not present. The patient returned to Illinois and was lost to further follow-up.

Opsoclonus has been described in the following cases: the opsoclonus-patalanysoclonus syndrome; with acute cerebellar ataxia of childhood; in association with neuroblastoma; as a remote effect of carcinomas of the uterus, lung, and breast; in association with an intracranial tumour; and in neonatal opsoclonus.\(^1\)\(^{13}\) Although opsoclonus has long been associated with encephalitis, a specific viral aetiology has only rarely been documented. There is a case report of a 14-year-old girl with benign encephalitis, opsoclonus, and myoclonic jerks of the neck and trunk where the mumps culture was positive.

**References**

Serologically confirmed St Louis Encephalitis was diagnosed in a 28-year-old man with the syndrome of benign encephalitis, opsoclonus and body tremulousness. Our case gives further evidence for a specific viral aetiology of opsoclonus.

Opsoconus in a Confirmed Case of St Louis Encephalitis

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**References**


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**Matters arising**

**Asymmetry of the aura and pain in migraine**

Sir: I was very interested in the article of RC Peatfield and others on the “Asymmetry of the aura and pain in migraine” (*J Neurol Neurosurg Psychiatry* 1981; 44:846-8). However, I feel it is crucial to know how the information with regard to location of the headache and the symptoms of paraesthesiae and numbness was collected. In my experience patients’ recall for the site of the headache in particular is very poor and the only reliable way to collect this sort of information is to ask the patients to prospectively fill in a form recording the details of their headache actually during or shortly after they have had their episode. If this has been done it would add greatly to the reliability of your most interesting findings.

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**F Clifford Rose replies:**

Information on the lateralisation of headache in our migraine patients was collected when they first attended the clinic, but on the lateralisation of the aura symptoms by later questioning. Both were retrospective in the sense that questions were not asked during the acute attack, and so the answers were dependent on the patient’s memory. We would re-emphasise that the statistical analysis was based only on those reporting that the majority of their headaches were on the same side. We know of no objective data that casts doubt on the reliability of patients who report headaches almost exclusively on one particular side.

**Multiple Sclerosis and Rheumatoid Arthritis**

Sir: During my 21 years in rheumatology at this Unit, I have come across only one example of a combination of multiple sclerosis and rheumatoid arthritis. My colleagues share my impression concerning the infrequency of this association based on no cases being observed over a combined rheumatological experience of over forty years.

I would be interested to hear through your readership if others have noted any particular infrequency of association between these two diseases.

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