neurologists tending to give higher scores in Session 1 than Session 2 and two neurologists the converse. There was also inter-observer variability, two neurologists tending to be high scorers while two other neurologists were low scorers. The standard deviation of the scores of individual patients was quite high ranging between 1·3 and 2·2 for any single set of scores.

The Kendall coefficient of concordance among the observers was 0·633 in Session 1 and 0·624 in Session 2. An analysis of variance of the data from neurologists was performed and the total variance of the scores was 2·75 corresponding to a standard deviation of ±1·7. The inter-observer variance of the neurologist's score at the same session was 1·38 corresponding to a standard deviation of ±1·2. The inter-observer variance was an average of the scores by one neurologist at the two sessions was 1·71 corresponding to a standard deviation of ±1·3.

Although only seven of the 10 items in the Webster scale were scored, seborrhoea, rigidity and self care being omitted, we think that they were representative of clinical scales in general.

The number of patients scored was small and more severely affected patients were not included. However, we do not think this invalidates our observations since in our experience one of the major difficulties in using such a clinical rating scale in which each major sign or symptom is rated between 0–3 is determining the value for the milder abnormalities. The small number of patients did not permit a conclusive analysis of variance on individual items of the scale. With a bigger patient group it would be possible to identify individual items of the scale in which ambiguities of description make an excessive contribution to the overall variance.

The group of neurologists ranged in their familiarity with rating Parkinsonian patients using the Webster scale from those who performed it frequently to those performing it only occasionally. Some neurologists complained that some of the procedures described by Webster used in the video recording were not their standard technique for assessment. For example Webster rates bradykinesia by assessment of pronation-supination movements of the hand placed on the thigh whereas a common clinical test used by some of the neurologists was rapid sequential opposition of thumb to fingers. Even those most used to the scale were not entirely consistent over the two sessions.

We conclude, therefore, that there is a considerable variance in the Webster rating scores performed by a number of different neurologists on the same group of patients. We suggest that doctors taking part in clinical trials of drugs for Parkinson's disease using such clinical rating scales spend some time familiarising themselves with the scale preferably practising the ratings on patients prior to the commencement of the trial. This should ensure a higher degree of internal consistency. In addition, in multicentre trials the trials should be brought together prior to commencement to reach agreement on the various ratings and we would recommend the use of video recordings of patients for this purpose.

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Painful ophthalmoplegia in systemic lupus erythematosus

SIR: Painful ophthalmoplegia is a combination of ocular pain, ipsilateral ocular palsies, and sensory loss in the ophthalmic and sometimes maxillary distributions of the fifth nerve. Vision can be impaired. The syndrome may be caused by lesions in the cavernous sinus or superior orbital fissure, including idiopathic inflammation, neoplasms, infections, endocrinopathies, thrombosis of the orbital veins or cavernous sinus, and vascular malformations. We report here a patient with a painful ophthalmoplegia as an initial manifestation of systemic lupus erythematosus.

In May 1978, a 38-year-old woman was admitted with severe pain behind the right eye, blurred vision, diplopia and nausea. Physical examination revealed a right third nerve palsy with a dilated and fixed pupil, a right fourth nerve palsy, and sensory loss in the right ophthalmic and maxillary distributions of the right fifth nerve with diminished corneal reflex. The ocular fundi were normal. Routine laboratory studies showed an erythrocyte sedimentation rate (ESR) of 25 and 52 mm, and serologic tests for syphilis were negative. Roentgenograms of the skull and chest, orbital tomograms, bilateral carotid and vertebral angiograms, and electromyographic studies were normal. Visual evoked potentials showed amplitude and latency abnormalities in the response from the right eye, consistent with a prechiasmatic lesion in the right side. Steroid therapy was initiated. Eye pain and the muscle ocular palsies disappeared over the following week, and ten days later, visual evoked potentials became normal. In December 1979 and October 1980 she suffered generalised tonic-clonic seizures. In May 1981 she returned after the sudden development of a left hemiplegia. Physical examination showed Raynaud's phenomenon and facial erythema. Laboratory investigations revealed an ESR of 106 mm, thrombocytopenia, a positive antinuclear factor native DNA antibodies and antibody-type circulating anticoagulant. Cranial CT scan showed a right temporoparietal infarct. The diagnosis of systemic lupus erythematosus was established and the patient was started on steroid therapy. Ten months later she developed right hemiplegia and aphasia. The patient subsequently died but no authorization for necropsy was obtained.

We report a patient with painful ophthalmoplegia believed to the an initial manifestation of systemic lupus erythematosus. The sensory disturbance was confined to the ophthalmic and maxillary divisions, and the sixth cranial nerve was unimpaired. This supports the diagnosis of a lesion involving the lateral wall of the cavernous sinus, and distinguishes it from lesions confined to the superior orbital fissure.1 Mathew and Chandy2 suggested that the syndrome of painful ophthalmoplegia may be a manifestation of a more generalised autoimmune disease, since half of their patients had positive test for LE cells and raised ESR. Only one patient affected by systemic lupus erythematosus with painful ophthalmoplegia has been reported, but not as a primarily manifestation.3 Painful
ophthalmoplegia in our patient might be the result of an immune reaction located in the wall of the cavernous sinus. Tests for lupus should be systematically done in every patient affected by painful ophthalmoplegia.

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References


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Triphasic EEG discharges in metrizamide encephalopathy

Sir: The water-soluble contrast agent metrizamide has proved safe and effective in various neuroradiologic procedures. The adverse effects of this agent differ in some respects from those of other contrast media, and have been well described. Seizures and transient cerebral symptoms have been seen occasionally, but increasing attention has been focused on a prolonged toxic encephalopathy with stuttering, obtundation, asterixis, and myoclonic jerks, which occur at least on one occasion has relapsed after recovery from the initial episode. Various EEG changes have been described after metrizamide injection. Triphasic waves associated with metabolic encephalopathy have recently been considered. We encountered a patient with toxic encephalopathy after metrizamide myelography whose EEG showed triphasic waves on several occasions. Such patients may provide insight into the mechanisms of triphasic EEG discharges in metabolic encephalopathies.

A 65-year-old female had been healthy except for hypertension treated with salt restriction, but suffered left leg and foot pain since an automobile accident in 1968. She was referred for implantation of a transcutaneous nerve stimulator after failure of analgesics and antidepressants. Strengh was normal except for 4/5 proximal and 3/5 distal weakness in the left leg. Pain and touch sensation were decreased over the lateral and posterior aspects of the left leg, thigh, and buttocks, and the medial aspect of the left foot was hyperesthetic. Deep tendon reflexes were brisk and symmetric in the arms and hypoactive in the legs, with an encephalopathy accentuated by a flexor plantar reflex. Laboratory studies were normal. Metrizamide myelography of the thoraclolumbar region was carried out, and on the following morning the patient abruptly became confused and agitated. All medication given before or during the myelogram had been discontinued. Neurologic examination was unchanged except for disorientation, monosympathetic responses to stimulation, and asterixis. Complete blood count, blood glucose, serum electrolytes and calcium, liver function tests, arterial blood gases, and cerebrospinal fluid were normal. An electroencephalogram showed continuous triphasic sharp waves of 100–200 μV amplitude and 2–4 Hz frequency, which were maximal frontocortically and decreased in amplitude as they extended posteriorly. Occasional theta activity was present, with the flexor plantar and occipital areas, but alpha rhythm was absent and background activity was unchanged by stimulation or eye opening. The patient's mental state improved over the next several days, no further studies were undertaken, and she underwent implantation of a dorsal column stimulator. Subsequent EEGs have shown diffuse theta slowing, without triphasic waves of asymmetry. This patient developed an acute encephalopathy with asterixis and triphasic EEG discharges after metrizamide myelography. No underlying or precipitating medical or neurologic disorder was found, but the subsequent persistence of EEG slowing raises the possibility of a pre-existing mild encephalopathy placing her at risk for this complication. The persistent EEG slowing despite return of the mental state to normal could also reflect a mild but persistent encephalopathy afterward, analogous to the persistent head bobbing reported in two patients by Davis and coworkers. In these patients, the movement disorder was present for several months after myelography despite normal mentation, and the EEG was diffusely slow in one patient. The mechanism and generator sites of triphasic waves are unknown, but subcortical orthalamic generation has been suggested, and could be supported by evidence of subcortical origin for other rhythmic EEG patterns, and of the role of thalamic and brain stem centers in the generation of normal EEG rhythms.

Kaada8 studied 79 patients undergoing lumbar myelography, none of whom developed encephalopathy, and observed transient and diffuse slowing in 13 and epileptiform discharges in none. Ropper, Chiappa, and Young9 studied prospectively the effects of metrizamide myelography or cisternography in 61 patients, without one patient had a normal EEG, while the other patients had either paroxysmal discharges or slow activity with some sharp features. This suggested the possibility of diffuse cerebral uptake of metrizamide after lumbar injection, with both irritative and depressive effects.

Vincent and Zimmerman briefly reported a patient who developed confusion, agitation, tremor, and asterixis after lumbar myelography. The EEG demonstrated generalised slowing and bifrontal triphasic delta transients, and clinical and EEG findings resolved within 48 hours. The authors ascertained the previous occurrence of at least two similar cases, one patient showing triphasic EEG discharges. Two patients with transient confusion and asterixis after lumbar myelography were reported by Rubin, Horowitz, and Katz, one patient had a normal EEG, while the other tracing showed bilateral and independent paroxysmal sharp transients and slow waves in the posterior temporal, parietal, and occipital areas. Bertoni et al.7 reported two patients with similar features, one of whom had no EEG and the other a normal tracing when the encephalopathy had virtually resolved. A CT scan of the brain in the former patient demonstrated widespread cerebral uptake of metrizamide.

Two mechanisms for encephalopathic complications of metrizamide myelography were proposed by Bertoni et al.: interferenc with the sodium-potassium ATPase pump and competitive inhibition of glucose metabolism as a 2-deoxyglucose analogue. No in vitro inhibition of a partial reaction of the cation pump was found, but metrizamide in concentrations analogous to those after lumbar injection competitively inhibited microbial hexokinase. This finding suggests that the encephalopathy might be due to impaired glucose metabolism.

The clinical and EEG similarities between several recent cases of metrizamide encephalopathy and metabolic derangements typified by hepatic encephalopathy

Letters
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