initiating and maintaining eyelid opening, resorting to separating her eyelids manually. At about the same time, she also developed unilateral tremor in her left hand.

The patient's initial neurological evaluation showed dysphasia, generalized bradykinesia, and difficulty with voluntary eyelid movement. Psychometric assessment in July 1982 showed mild impairment of language expression, with abnormal performance on the Boston naming and controlled oral word association tests. Reading and writing to dictation were intact. On a test of executive function (Wisconsin Card Sorting), she failed to generate correct categorisations and committed excessive perseverative errors. Although verbal memory could not be assessed because of language disintension, she did show visual reproduction tasks (Wechsler memory scale) in the low normal range.

Neuropsychological examination one year later disclosed a bradykinetic, essentially mute woman. Cranial nerve function was remarkable for bilateral ptosis; she could contract mimetic muscles voluntarily but lids would not remain raised even if displaced manually. The patient's reaction to light was normal. Gaze was conjugate but slightly restricted in upward pursuit. Moderate paratonic rigidity affected all limbs with a lesser degree of increased tone in axial muscles. Speech was slow. Despite mutism, she retained the ability to communicate in writing, although she rarely produced more than single word responses. Comprehension was intact to two step commands. She was oriented to time and place, and recalled two of three objects after brief delay. Tests of buccofacial praxis (for example, protrude tongue) produced unrecognisable responses despite multiple attempts. Transitive and intransitive gestures of the extremities were also flawed although recognisable.

Pertinent radiographic studies included cranial MRI, which showed only moderate, diffuse cortical atrophy without lobar or brainstem atrophy. Fluorodeoxyglucose PET showed relatively decreased tracer uptake in medial and lateral precentral regions on both sides with sparing of the frontal poles. A computed encephalogram revealed some increased cerebral activity, which resembles those described by Tyrrell et al., exhibiting initial disruption of speech output and pronounced orofacial dyspraxia associated with selective frontal lobe hypometabolism. Unlike the previous cases, however, our patient developed prominent apraxia of lid opening as well as ideomotor apraxia. Apraxia of eyelid opening characteristically occurs as part of extrapyramidal disorders such as Parkinson's disease, Huntington's chorea, progressive supranuclear palsy, and Shy-Drager syndrome. In rare cases where lid opening apraxia follows injury, however, the disorder resembled amnestic apraxia of the right hemisphere.1

On the basis of clinical and PET data, Tyrrell et al.2 proposed that the syndrome of reduced speech output and facial dyspraxia reflected a central degeneration of the inferior and lateral frontal lobe. The dominant frontal lobe contains cortical modules for both articulation and buccofacial praxis. Of two of three patients described by Tyrrell et al.1 shown asymmetric hypometabolism, worse in the left hemisphere. Accordingly, we speculate that our patient's initial symptoms reflect dominant function, although apraxia of eyelid movement supervened when homologous regions in the right hemisphere degenerated. The present finding extends the spectrum of the syndrome described by Tyrrell et al.2 More important, if focal cortical degeneration originates in the non-dominant hemisphere,3 patients with this syndrome might present with predominant apraxia of eyelid opening.

Clinicians confronted with eyelid apraxia must consider focal cortical degeneration in addition to extrapyramidal anomalies, with which it more commonly develops.

Correspondence to: Dr J C Adair, Department of Neurology, University of Florida, PO Box 100236, Gainesville, Florida 32610-0236, USA.


Unilateral hypotonic seizures successfully diagnosed by icatal SPECT with technetium-99m-HMPAO in a patient with a brain tumour

Among the simple partial seizures, motor seizures manifested by motor inhibition are rare1 and often misdiagnosed as transient ischaemic attacks.2 These icatal events are different from the more common postictal Todd's hemiparesis. Single photon emission computed tomography (SPECT) is being widely used as an adjunctive technique in the localisation of epileptogenic foci. We report a case of an extra-axial brain tumour associated with repeated transient left hemiparesis and stress the efficacy of using icatal SPECT for differentiating hypotonic seizure from transient ischaemic attacks.

A 52 year old man was admitted to our hospital after several episodes of acute transient left hemiparesis that each lasted for 20 to 30 minutes. There were no convulsive movements of the limbs, nor was there impairment of consciousness. Two years before this admission, a right frontal high grade astrocytoma had been totally removed (fig 1A) and he had been followed up as an outpatient on sodium valproate, which kept him free of seizures. On the cut-off date, he showed no static neurological deficits, but MRI showed recurrence of the right frontal astrocytoma (fig 1C). As transient ischaemic attacks were suspected after his reported episodes of transient hemiparesis, right carotid angiography was performed. A faint tumour stain was found in the late venous phase and the right frontal area appeared hypovascular, but no stenosis or occlusion of arteries was noted. Although a repeat right frontal craniotomy and gross total removal of the recurrent astrocytoma was performed (fig 1D), the same type of transient left hemiparesis continued. Interictal EEG showed slow waves in the right hemisphere but no periodic bursts of sharp or spike waves. To differentiate whether the transient hemiparesis was due to transient ischaemic attacks or epilepsy, interictal and ictal brain SPECT with technetium-99m-hexamethylpropylene amine oxime (99m-Tc-HMPAO) was performed.

Interictal SPECT showed hyperperfusion of the right frontal lobe where the recurrent astrocytoma had been removed (fig 2 left). Except for that area, there was no appreciable difference in signal between right and left hemisphere (fig 2 left). Ictal SPECT, however, showed an increased signal involving a wide area of the right hemisphere (fig 2 right), thus confirming the transient hemiparetic attacks as unilateral hypotonic seizures and not transient ischaemic attacks. The patient received carbamazepine as an additional anticonvulsant and no more seizures occurred.

In this patient, unilateral hypotonic seizures, which presented as transient ischaemic attacks, were clearly diagnosed by SPECT with 99m-Tc-HMPAO by documenting a change from interictal hypoxia to ictal hyperactivity in the involved brain area. Although postictal paralysis, usually called Todd's paralysis, is well recognised, unilateral hypotonic seizure is less commonly diagnosed, as it does not follow convulsive movements, whereas Todd's paralysis usually does. This seizure has been described as the "paralytic equivalent of genuine epilepsy", "negative seizures", "focal inhibitory seizures", and "ictal hemiparesis".1,3 Unilateral hypotonic seizures often resemble the clinical picture of transient ischaemic attacks and are often misdiagnosed. For the diagnosis of unilateral hypotonic seizures, an ictal EEG has been used and showed either episodic bursts of sharp waves with focal activity or focal bursts of slow activity with high amplitude.2

Figure 1 Sagittal T1 weighted MRI with gadolinium before (A) and after (B) the initial operation and before (C) and after (D) the second operation. Homogeneously enhanced astrocytoma in the right frontal lobe (A) was totally removed at the first operation (B). Two years later, recurrence of the tumour was seen at the same position (C) and the tumour was successfully removed by surgery (D).
Cervical extradural abscess complicating discitis and associated disc prolapse, secondary to a long line infection

Spinal epidural abscess is rare, accounting for only one or two cases per 10,000 hospital admissions. Trauma and surgery (20% each) are the two commonest causes. Dental, skin and soft tissue infections, and endocarditis account for a further 20%. In most of the remaining cases no source is found. *Staphylococcus aureus* is the commonest organism isolated (60%).

Although discitis or osteomyelitis are reported in 18-80% of cases, disc prolapse has not been mentioned as a comorbid condition.

We report a patient (a 53 year old woman) who underwent total thyroidectomy for carcinoma on 20 October 1993. A right antecubital long line was inserted in theatre. Postoperatively she became hypocalcaemic and calcium was given via the long line. Ten days later she developed a clinical thrombophlebitis affecting the right arm although ultrasound showed no axillary vein thrombosis. The long line was removed and venous blood cultures were taken on 1 and 2 November 1993. Cultures and the long line tip grew *Staphylococcus aureus* that was resistant to penicillin but sensitive to gentamicin, erythromycin, and flucloxacinilin. After a single dose of erythromycin she was changed to regular intravenous flucloxacillin on the advice of the microbiologists.

On 4 November 1993 she complained of "electric shocks" in the shoulders followed by weakness of the right arm, which progressed over the next five days to involve the left arm and both legs. There were no other sensory symptoms, no sphincter disturbance, and no respiratory difficulties. She was apyreal, with no meningism, cranial nerve, or fundal abnormality. The neck wound was well healed, and neither it nor the long line site was clinically infected. There was flaccid weakness of her right arm, grade 3/5 proximally and 4/5 distally, and a milder degree of weakness of her left arm. Both legs showed very mild spastic weakness. There were no sensory abnormalities. Biceps and triceps jerks were absent, knee and ankle jerks brisk, and plantars equivocal.

Contrast enhanced cervical MRI showed high signal in the C4-5 disc and in both the anterior and posterior longitudinal ligaments. There was thickening and elevation of the posterior ligament over both of the vertebral levels, and a low intensity mass at the level of the disc space compressing the theca of the cord and C5 nerve root. This was thought to be either pus or a sequestered disc prolapse (figs 1 and 2).

She was started on intravenous cefuroxime and metronidazole and operated on immediately for anterior cervical decompression and fusion.

At operation through the right half of the thyroidectomy scar the tissues were found to be extremely stuck down. No overt
Unilateral hypotonic seizures successfully diagnosed by ictal SPECT with technetium-99m-HMPAO in a patient with a brain tumour.

I Date, M Kamitani and T Ohmoto

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