at that level. The physical characteristics and the type of food ingested are identified as important factors in activating this form of eating epilepsy. Attacks may recur even when temporal structures and those anterior or posterior to the sensory motor strip have been resected. In addition to reflex seizures, spontaneous attacks may occur. Thalamic sensory afferents to the damaged cortex seem to play a predominant part in the genesis of seizures in these patients. By contrast, eating epilepsy of the opercular origin is dependent more on the context of a meal and there are often additional clinical features suggesting temporal localisation. Such patients have no features of a contralateral temporal resection usually leads to cessation of the attacks. Our patient clearly showed an AOS. He had no spontaneous seizures and we were not able to confirm that the characteristics of the focal paroxysmal aura is a part in triggering the attacks. He is exceptional because of the association of two infrequent but related neurological syndromes. We stress the value of careful imaging and of inquiring about the circumstances of occurrence of attacks in diagnosing this syndrome and the associated reflex epilepsy.

Patient with acquired bilateral opercular syndrome and eating epilepsy: coronal T1 weighted MRI showing focal atrophy in both Rolandic opercula.

ground activity with focal interictal slow spikes and waves over the right centrotemporal region, not increased during sleep. No ictal recording was obtained.

Voluntary control of facial muscles is dependent on the normal function of the motor cortex and of the corresponding pyramidal tract. Subcortical structures, particularly the basal ganglia, thalamus, and subthalamic nuclei, are involved in automatic or emotional movement. A bilateral lesion involving the anterior opercular region is manifested by facial diplegia with dissociation of voluntary and involuntary movements.1 Pyramidal signs, cheiro-oral paresthesiae, and severe speech disturbance or even mutism with unimpaired language comprehension may be due to or associated with bilateral opercular lesions depending on their symmetry and extent. Symmetric lesions of the posterior limbs of both internal capsules may produce a picture similar to AOS because the projections from the anterior opercular cortex are represented there. The differential diagnosis of AOS, therefore, is based not only on bulbar dysfunction but also on other clinical features of pseudobulbar palsy, such as those due to striaton pontine lesions.3 The absence of automatic-voluntary dissociation and affective liability point to the presence of AOS.1 Bilateral stroke is the main aetiology of AOS and the two events may be separated in time. A unilateral lesion may lead to AOS in a patient who already has contralateral opercular dysfunction on the other side.

The occurrence of AOS in childhood is exceptional. In most cases the syndrome is of developmental origin with polymicrogyria most likely due to prenatal vascular lesions. Infections of the CNS are also an important cause of the syndrome in children.4 There are well documented cases with transient bilateral Rolandic dysfunction and features resembling AOS due to an epileptic disturbance.5

In this patient, reflex seizures were triggered by a specific stimulus, the act of eating. Eating epilepsies have been subclassified into two distinct groups with either temporal or opercular onset, according to a recent anatomo-clinical review.6 In the opercular subgroup electroclinical studies pointed to the presence of an epileptogenic focus in the postcentral gyrus, frequently with evidence of a cortical structural lesion this condition is endemic. In Japan, Osame et al called it HTLV 1 associated myelopa thy (HAM).7 The HTLV 1 has previously been isolated and identified in 1980 by Poizot et al as the aetiological agent of adult T cell leukaemia-lymphoma (ATLL).8 Epidemiological studies have since shown the high prevalence of both ATLL and HAM/TSP in areas with a high HTLV 1 carrier rate. The virus has been isolated from patients with both the conditions and has been thought to be transmitted by the retrovirus. It is not known why some carriers develop chronic progressive myelopathy whereas others develop the haemato-malignancy. The coincidence of both conditions occurring together as in our patient is extremely rare.

A 61 year old black Afro-Caribbean woman who had migrated to the United States from the Republic of Martinique was seen with a four year history of progressive difficulty in walking and backache. She had also noticed a progressive deterioration in her vision and difficulty in micturition. Two and a half years later she returned to have a positive syphils serology. A CSF examination was normal.

Neurological examination on presentation disclosed diminution of visual acuity in both eyes (20/200 vision) and the left side was more affected. Hemiparesis of the right side was present with mild ataxia of the left arm. The tendon reflexes were brisk and were symmetrical except for the right biceps which was absent. The plantar responses were flexor. The tone and power in her arms were normal, although the reflexes were brisk. Sensory examination was normal to all modalities and there were no cerebellar signs. She was able to walk only with the help of a stick and she had a spastic gait.

Haemoglobin concentration was 10 g/dl and the white cell count was 10 2 x 109. Magnetic resonance imaging of the cervical and dorsal spine was normal but MRI of the brain showed hypertense signals in the periventricular white matter on T2 weighted and a half year later some of those signals had delayed bilaterally and the somatosensory evoked responses showed delay of N19 on stimulation of the median nerves at the levels of the thalamus. Asymmetrical T2 signals were detected in the serum and a diagnosis of HTLV 1 associated myelopathy (HAM) was made.

Four months after the diagnosis of HAM, the patient had a rapid deterioration of power in her legs. She was febrile and delirious. She became progressively obtunded and her level of consciousness rapidly worsened. There was considerable tenderness in the long bones. The liver and spleen could not be palpated. Haemoglobin was 10 g/dl, and the white cell count was 66 x 109. The peripheral smear showed a pronounced leucopenia and presence of abnormal lymphocytes with multilobulated nuclei with clefted and cleaved forms. About 20% of the lymphocytes showed abnormal multilobulated nuclei. Leukaemia typing confirmed that the abnormal T lymphocytes (CD-2 count 98%, CD-3 count 97%, CD-4 count 76%, and CD-5 count 0%) and a diagnosis of adult T cell leukaemia was made. Examination of the peripheral blood film, skeletal survey, and serum calcium concentrations were normal. The patient received supportive treatment but she soon deteriorated and died.

HTLV 1 associated myelopathy and adult T cell leukaemia-lymphoma in the same patient: report of a case

Tropical spastic paraplegia (TSP) is a chronic progressive myelopathy occurring in endemic areas around the world with particularly high prevalence in Japan, the Caribbean, parts of Africa, and South America. In 1985, Gessain and colleagues working on the island of Martinique identified the presence of anti-HTLV 1 antibodies in patients with TSP. This finding was later confirmed in other tropical regions where

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560 Letters to the Editor

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MATTERS ARISING

Magnetic resonance spectroscopic study of parkinsonism related to boxing

We read with interest the paper by Davie et al reporting a study of proton magnetic resonance spectroscopy (MRS) in three ex-boxers with parkinsonism. They report a significant reduction in the absolute concentration of N-acetyl-aspartate (NAA) in the putamen and globus pallidus in the boxes with striatonigral dopamine...
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K Rajamani, F P Rugman, C S Savant and S D Vakil

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