A recent report from the UK-TIA Study Group² presented 11 cases of intracranial tumours among 2449 patients with transient ischaemic attacks or minor strokes. Occasional cases of small cerebral haematomas have been found in patients with minor strokes,³ but only exceptionally in patients with transient ischaemic attacks.⁴ In a CT scan study of 284 cases with transient ischaemic attacks⁵ five patients had a mass lesion; none a brain haematoma.

We present the results of a prospective CT scan study of 175 patients (63 with transient ischaemic attacks and 112 with minor deficits lasting longer than 24 hours) recruited in the emergency rooms of two general hospitals. In every case, the CT scan (CX Tomoscan, Philips) was performed within the first week of the clinical event (with a mean delay of 13 (SD 24) hours). The mean age of the patients was 68 (8.5) years; 132 events were located in the carotid artery territory, 38 in the vertebrobasilar territory and five were of uncertain location. The CT scan was normal in 114 patients. Low density areas compatible with infarction were present in 56. Non-ischaemic causes of the presenting symptoms were found in three minor cases of stroke—namely, a brain tumour, compatible with an extensive hemispheric malignant glioma by MRI and biopsy features; a small occipital haematoma; and a medium sized basal ganglia haematoma (associated with an ipsilateral subdural haematoma). All three patients were over 60 and had at least one vascular risk factor. In one of the cases of transient ischaemic attack a mass located on the clivus (compatible by CT features with a meningioma) was considered to have caused cerebral symptoms through compression of the basilar artery. Also aetiologically related to the symptoms could have been a minor stroke case with a thrombosed middle cerebral artery bifurcation aneurysm, demonstrated by MRI angiography.

The number of cases in our study is insufficient to support definite conclusions. The yield of CT scan for the detection of non-ischaemic causes (such as cerebral and subdural haematoma and brain tumour) in minor strokes (cases with symptoms lasting longer than 24 hours) was 2.7% (95% CI 0 to 5.7) and in transient ischaemic attacks the yield was 1.6% (95% CI 0 to 4.7).

M GARCIA-ESCRIG
A PEREZ-SEMPERE
L CALANDRE
F VILLAVEDE
M DE LA FUENTE
E CLAVERIA

Neurology and Neuroradiology Sections,
12 de Octubre Hospital, Madrid,
and Neurology Section,
Segovia General Hospital,
Segovia, Spain

Correspondence to: Dr L Calandre, Servicio de Neurología, Hospital 12 de Octubre, Ctra Andalucía, km 5,400, 28041 Madrid, Spain.

- Hankey GJ, Warlow CP. Cost-effective investigation of patients with suspected transient ischaemic attacks. *J Neurol Neurosurg Psychiatry* 1992;55:171–6.
- UK-TIA Study Group. Intracranial tumours that mimic transient cerebral ischaemia: lessons from a large multicentre trial. *J Neurol Neurosurg Psychiatry* 1993;56:563–6.
- Dennis MS, Bamford JM, Molyneux AJ, Warlow CP. Rapid resolution of signs of primary intracerebral haemorrhage in computed tomograms of the brain. *BMJ* 1987;295:379–81.
- Tsuda Y, Ayada Y, Takahashi T, et al. Semiquantitative regional cerebral blood flow evaluation using 123 I-IMP SPECT in

a case showing transient ischemic attack caused by putaminal hemorrhage. *Acta Neurol Scand* 1991;94:448–51.

5 Murros KE, Evans GW, Toole JE, Howard G, Rose LA. Cerebral infarction in patients with transient ischemic attacks. *J Neurol* 1989;236:182–4.

Sumatriptan and giant cell arteritis

We have proposed a *unified theory* that suggests that migraine is essentially driven from the central nervous system and entrains the trigeminal innervation of the cranial vessels to form one of the clinical expressions of the disease.¹ The introduction into clinical practice of the novel antimigraine compound sumatriptan, a serotonin (5-HT) agonist, has provided a tool to understand further the underlying mechanisms of the disease. Its action as a vasoconstrictor and its inhibition of neurogenic inflammation in experimental animals has been cited by various groups as evidence for either the vascular or neurogenic inflammatory theories of migraine respectively. A patient was recently admitted to our institution with giant cell arteritis and headache not responsive to sumatriptan. Her lack of response casts some doubt on the neurogenic inflammatory theory of migraine.

The patient is a 68 year old woman who had a 10 day history of right sided temporal and frontal headache. The headache had spread from a small region above the eye to involve most of the right side of the head and she had noticed some increasing tenderness of the scalp muscles. The headache became more severe with time and had some pounding exacerbations but no associated features of migraine. Eight days into the illness she attended her general practitioner and was given sumatriptan (100 mg) orally as a single dose, which did not alter the headache. She had no other history, particularly of regular headaches, and there was no relevant family history. Physical examination was unremarkable except for tenderness of the temporal arteries bilaterally. The erythrocyte sedimentation rate (ESR) at this time was 110 mm/h. She was treated with high dose steroids with complete remission of the headache and general malaise and a drop in the ESR by the next day. A temporal artery biopsy showed pronounced inflammatory changes.

The patient had a classical presentation of temporal arteritis that responded to steroids and she has remained well on steroids. She had no response to sumatriptan despite some side effects from the drug, notably nausea and mild neck and arm discomfort typical of that reported in trials.² Practitioners should be watchful for secondary headache and in the elderly temporal arteritis must **always** be considered. Administration of a cranial vasoconstrictor to patients with inflamed narrowed vessels with the propensity to thrombose must be avoided absolutely. Sumatriptan is not a bedside test for migraine; it must not replace the careful history and should only be given to patients with a positive diagnosis in appropriate circumstances.

P J GOADSBY
Department of Neurology,
The Prince Henry Hospital,
Little Bay, NSW 2036
Australia

- Lance JW, Lambert GA, Goadsby PJ, Duckworth JW. Brainstem influences on cephalic circulation: experimental data

from cat and monkey of relevance to the mechanism of migraine. *Headache* 1983;23:258–65.

- Goadsby PJ, Zagami AS, Donnan GA, et al. A double blind placebo controlled crossover study of sumatriptan in the treatment of acute migraine attacks. *Lancet* 1991;iii:782–3.

Isolated lingual myoclonus associated with an Arnold-Chiari malformation

Reports on isolated rhythmic tongue movements are infrequent and may contribute to the understanding of rhythmic hyperkinesias in general. Isolated rhythmic movements of the tongue led to the diagnosis of an Arnold-Chiari malformation.

Case report

A 61-year-old man noted continuous jerking movements of the tongue for the first time 15 days before he was admitted to Sant Pau Hospital in 1984. The jerks, which were not preceded by any illness, persisted all day, were not accompanied by a clicking noise, and were not stopped or influenced by any action attempted by the patient. In 1974 he had a reactive mental depression that was treated with amitriptyline (75 mg/day) for nine months. No other medication was taken regularly in the nine years before the beginning of the abnormal tongue movements. There were no antecedents of head trauma or other relevant personal or family history. Neurological examination was normal apart from the lingual jerks. They consisted of continuous rhythmic 3 Hz, low amplitude, symmetrical contractions of both lateral edges of the tongue affecting mainly its middle and posterior parts and causing a midline depression of the tongue. The soft palate and other muscles innervated by the brainstem were not involved. The jerks persisted during sleep. Although he did not complain about phonation disturbances, speech was mildly affected. A forced palatalisation of some words was evident. He spontaneously produced vowel prolongation with the tongue pulled against the palate; when he was asked to let the tongue free in the mouth, most sounds had a quivering quality. Swallowing was normal. Protrusion, intrusion, passive depression, touch, or taps to the tongue did not influence the amplitude or the rhythm of the jerks. After hours of continuous activity, the movements would unexpectedly stop, only to start once again after a few minutes.

Routine blood analysis, surface EEG, cortical somatosensory evoked potentials, and brain stem auditory evoked responses were all normal. A CT scan showed a diffuse and symmetrical enlargement of the two lateral and third ventricles with mild periventricular oedema and a normal sized fourth ventricle. An Arnold-Chiari type I malformation was evident on an MRI study (figure).

After three days on 6 mg/day of clonazepam the movements were no longer continuous but occurred, at the same rate and amplitude, in random bursts lasting up to three minutes. These bursts totally disappeared in the second week. Over the next eight years, many attempts to discontinue clonazepam were followed by the re-emergence of the movements. During this period, other neurological and neuropsychological examinations, as well as repeated EEGs during and between the episodes,

Summary of clinical characteristics in reported cases of rhythmic isolated lingual myoclonus

Authors	Age	Sex	Related illness	Interval	Characteristics	Course	Other findings
Troupin and Kamm ²	25 years	M	Head trauma	8 days	Continuous	Spontaneous recovery	EEG slowing/left temporal focus
Keane ³	26 years	M	Head trauma	3 weeks	Episodic	Spontaneous recovery	Brainstem damage
(two patients)	19 years	M	Head trauma	1 month	Episodic	Spontaneous recovery	Brainstem damage
Sridharan ⁴ (case 4)	10 month	M	Subacute encephalitis	2 days	Continuous	?	Fever/EEG slowing
Gobernado <i>et al</i> ⁵	55 years	F	None		Continuous	Stop with valproate	—

Sagittal T1-weighted MRI scan showing herniation of the cerebellar tonsils through the foramen magnum and lack of enlargement of the fourth ventricle.

were normal. No additional signs of activity of the hydrocephalus or hypertrophy of the inferior olives were noted on CT or MRI. In March 1993 he was asymptomatic on a regimen of clonazepam (2 mg/day). The long-standing, monosymptomatic, and non-progressive movement disorder of our patient fits well with the characteristics of the focal rhythmic disorders that affect branchial muscles in various combinations with or without accompanying palatal myoclonus.¹ Both palatal and branchial myoclonus persist during sleep; their rhythms are especially resistant to external influences and respond poorly to medica-

tion. They usually arise from brainstem and cerebellar lesions involving the pathway from the dentate nucleus to the contralateral inferior olive, which, in many cases of palatal myoclonus and less often in branchial myoclonus, shows hypertrophic degeneration and is presumed to be the pacemaker of the movement disorder.¹ A distortion of the brainstem due to an Arnold-Chiari malformation was previously associated with palatal myoclonus, and at least two other patients have been successfully treated with clonazepam.¹ Nevertheless, few patients with branchial or palatal myoclonus have lingual involvement,¹ and isolated lingual myoclonus seems infrequent.²⁻⁵ Similar isolated rhythmic 3 to 5 Hz movements of the tongue were seen in a continuous mode in two patients—one reported by Troupin and Kamm,² and the other by Gobernado *et al*⁵—and as brief repetitive episodes in three other patients—two reported by Keane,³ who termed the condition “galloping tongue”, and the third by Sridharan (table).⁴ No possible aetiology was evident in the patient of Gobernado *et al*, whose lingual myoclonus responded to sodium valproate.⁵ Sridharan’s patient exhibited transient isolated lingual myoclonus in the context of a presumed subacute encephalitis.⁴ In the other three patients the movements occurred as a transient sequel to head trauma with brainstem damage.^{2,3} Brainstem damage, in our case owing to a downward elongation of the medulla and cerebellum, seems to be relat-

ed to the isolated lingual myoclonus. Troupin and Kamm considered the lingual movements a form of branchial myoclonus without palatal participation.² Keane, however, was more reluctant to use this term for the episodic occurrence of the lingual movements in his two patients.³ A categorisation into episodic and continuous forms for isolated lingual myoclonus was also suggested by Gobernado *et al*.⁵ The myoclonic activity of our patient was initially continuous and later became episodic, which challenges the pathophysiological value of distinguishing episodic and continuous forms of isolated lingual myoclonus. JAIME KULISEVSKY ASUNCIÓN AVILA JOSEP M GRAU-VECIANA Department of Neurology, Sant Pau Hospital, Autonomous University of Barcelona, Barcelona, Spain

1 Deuschl G, Mischke G, Schenck E, Schulte-Mönting J, Lücking CH. Symptomatic and essential rhythmic palatal myoclonus. *Brain* 1990;113:1645-72.
2 Troupin AS, Kamm RF. Lingual myoclonus: case report and review. *Dis Nerv Syst* 1974;35:378-80.
3 Keane JR. Galloping tongue: post-traumatic, episodic, rhythmic, movements. *Neurology* 1984;34:251-2.
4 Sridharan R. Rhythmic involuntary lingual movements (lingual myoclonus): study of 4 cases. *Indian J Pediatr* 1984;51:733-7.
5 Gobernado JM, Galarreta M, De Blas G, Jimenez-Escrig A, Hernandez A. Isolated continuous rhythmic lingual myoclonus. *Mov Disord* 1992;7:367-9.

Ankylosing spondylitis associated with myositis

Ankylosing spondylitis is a systemic rheumatic disorder characterised by inflammation of the axial skeleton and a host of systemic manifestations such as acute uveitis or iritis, aortitis, cardiac conduction abnormality, and fibrosis of the lung.¹ Muscle wasting is often a feature of ankylosing spondylitis, and has usually been ascribed to disuse of the muscles or nerve root compression secondary to axial skeleton lesions.² The case is reported of a man with ankylosing spondylitis associated with muscular atrophy and weakness in whom a muscle biopsy sample showed myositis. A 21-year-old man presented to Chiba University Hospital on 11 December 1990, with a dull pain around his buttocks and lumbar spine and weakness in his legs. At the age of 20 years in April 1990 he had noticed stiffness in his lower back on bending over to pick up an object on the ground. He subsequently developed a dull pain in his buttocks which gradually extended to affect his lower back in August 1990. He did not notice any abnormalities in his legs until a colleague pointed out muscle wasting in his right thigh. He began to have difficulty in climbing stairs in September 1990 because of weakness in his legs, in addition to lower back pain. On examination his

axial movements were greatly reduced. There was marked symmetrical atrophy of his legs, more conspicuous in the right quadriceps and hamstrings. There was mild weakness (MRC grade 4/5) in the upper limb girdle muscle, both iliopsoas, and the right quadriceps and hamstring. There was no fasciculation or pain in the muscles. Tendon reflexes were brisk in his arms and legs. There was no sensory deficit. A complete blood count and biochemical screening examinations, including plasma creatine kinase, were normal. The erythrocyte sedimentation rate was increased at 75 mm/h. He was positive for antibodies to HLA-B27. Plain film radiographs showed bilateral erosive arthritis of the sacroiliac joints consistent with the modified New York criteria for ankylosing spondylitis.¹ Myelography and MRI of his spinal cord did not show any abnormality in the nerve roots or spinal cord. Needle EMG showed fibrillation potentials in his left arm and leg and paraspinal muscles. Muscle biopsy samples were taken from his right biceps and right rectus femoris. A specimen obtained from the right biceps showed focal, mild mononuclear cell infiltrates, especially around endomysial blood vessels, and increased variability in fibre size on haematoxylin and eosin staining (figure). There was no necrotic fibre, but some scattered regenerating fibres were observed.

Biopsy sample from the right biceps showing mononuclear cell infiltrates around blood vessels and increased variability in fibre size. Haematoxylin and eosin stain.

Selective type 2B fibre atrophy was seen without conspicuous change in distribution on ATPase staining. A specimen obtained from the right rectus femoris showed a reduction and variability in fibre size without inflammatory cell infiltrates. It has been reported that neuropathic changes such as small angular fibres, target fibres, or non-specific myopathic changes such as central nucleation and variation in fibre size are often observed in muscle spec-