A recent report from the UK-TIA Study Group1 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,1 but only exceptionally in patients with transient ischaemic attacks.2 In the CT scans of 284 cases with transient ischaemic attacks3 five patients had a mass lesion; none a brain haematoma.

We present the results of a prospective CT scan study of 175 patients (63 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 verteobasilar 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 CT 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 meningoima) was considered to have caused cerebral symptoms through compression of the basilar artery. Also pathologically 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) in transient ischaemic attacks the yield was 1-6% (95% CI 0 to 4-7).

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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.3 The rise to 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 the vascular or neurogenic (neuroinflammatory) 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 inflammation 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 at night 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 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 second 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.

Practitioners should be watchful for secondary headache and in the elderly tempo- ral arteritis should 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.

Letters to the Editor

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 hyperkine- sias 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 swallowing noise, and would 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 rele- vant personal or family history. Neuro- logical examination was normal apart from the lingual jerks. They consisted of continu- ous rhythmic 3 Hz, low amplitude, symmet- ric contractions of both lateral edges of the tongue affecting more the anterior and posterior parts and causing a midline depression of the tongue. The soft palate and other muscles innervated by the brain- stem 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, bringing the tongue pulled against the palate; when he was asked to let the tongue free in the mouth, most sounds had a quevering quality. Snoring decreased, and voicing, intrusion, passive depression, touch on 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 dif- fuse 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 clon- azepam the movements were no longer con- tinuous 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-emer- gence of the movements. During this period, other neurological and neuropsy- chological examinations, as well as repeated EEGs during and between the episodes,
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. 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. 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. Nevertheless, few patients with branchial or palatal myoclonus show an isolated movement disorder, and isolated lingual myoclonus seems infrequent.

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, and the other by Gobenardo et al.—and as brief repetitive episodes in three other patients—two reported by Keane, who termed the condition "galloping tongue", and the third by Sridharan (table). No possible aetiology was evident in the patient of Gobernardo et al, whose lingual myoclonus responded to sodium valproate. Sridharan's patient exhibited transient isolated lingual myoclonus in the context of a presumed subacute encephalitis. In the other three patients the movements occurred as a transient sequel to head trauma with 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. Keane, however, was more reluctant to use this term for the episodic occurrence of the lingual movements in his two patients. A categorisation into episodic and continuous forms for isolated lingual myoclonus was also suggested by Gobenardo et al. 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.