A recent report from the U.K. Study Group on Intracranial Hemorrhages among Patients with Transient Ischemic Attacks (TIAs) has been published. The study involved 235 patients with TIAs, of whom 11% had intracranial hemorrhages. Patients with hemorrhages were more likely to have a history of hypertension or diabetes.

We have proposed a unified theory that suggests that the clinical presentation of migraine with aura is due to the activation of the central nervous system and the release of neurotransmitters. In cases of migraine with aura, the central nervous system is activated and the release of neurotransmitters causes the aura and the headache.

The number of cases in our study is insufficient to support definite conclusions. Further studies are required to confirm the findings of this study.

The mechanism of action of Sumatriptan in migraine is not fully understood. However, it is believed that Sumatriptan acts on the serotonin 1B receptor, which results in vasoconstriction and a decrease in the severity of the headache.

In conclusion, Sumatriptan is an effective treatment for migraine with aura. Further research is needed to fully understand its mechanism of action and to develop more effective treatments for this condition.
Summary of clinical characteristics in reported cases of rhythmic isolated lingual myoclonus

<table>
<thead>
<tr>
<th>Authors</th>
<th>Age</th>
<th>Sex</th>
<th>Related illness</th>
<th>Internal</th>
<th>Characteristics</th>
<th>Course</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troupin and Kamm¹</td>
<td>25 years</td>
<td>M</td>
<td>Head trauma</td>
<td>8 days</td>
<td>Continuous</td>
<td>Spontaneous recovery</td>
<td>EGG slowing/left temporal focus</td>
</tr>
<tr>
<td>Keane¹</td>
<td>20 years</td>
<td>M</td>
<td>Head trauma</td>
<td>3 weeks</td>
<td>Episodic</td>
<td>Spontaneous recovery</td>
<td>Brainstem damage</td>
</tr>
<tr>
<td>(two patients)</td>
<td></td>
<td></td>
<td>Head trauma</td>
<td>1 month</td>
<td></td>
<td></td>
<td>Fever/EEG slowing</td>
</tr>
<tr>
<td>Sridharan⁴ (case 4)</td>
<td>10 months</td>
<td>M</td>
<td>Subacute encephalitis</td>
<td>2 days</td>
<td>Continuous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gobernado et al</td>
<td>55 years</td>
<td>F</td>
<td>None</td>
<td></td>
<td>Continuous</td>
<td>Stop with valproate</td>
<td></td>
</tr>
</tbody>
</table>

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 hypotrophic 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 an initial movement,¹ and isolated lingual myoclonus seems infrequent.²³

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,¹ and the other by Gobernado et al—and as brief repetitive episodes in three other patients—two reported by Keane,¹ who termed the condition "galloping tongue," and the third by Sridharan (table).³ No possible aetiology was evident in the patient of Gobernado et al, whose lingual myoclonus responded to sodium valproate.⁴ 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 brain damage.¹ 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.

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 hip girdle muscles, 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.

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 neculation and variation in fibre size are often observed in muscle spec-

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

Haematoxylin and eosin stain.