Latencies of the M-response plotted against the distance of the stimulation site from the wrist. Shortening of the latency by increasing the stimulus intensity is observed only with stimulation over the segment between 16-5 and 25-5 cm from the wrist. The points obtained by weak stimulation within this segment and at a more proximal site (28-5 cm) are on a straight line, and those obtained by stronger stimulation within this segment and at more distal sites (4-5-13-5 cm) are on another straight line. Inset: M-responses recorded from the thenar muscle. The median nerve is stimulated just below the elbow (19-5 cm from the wrist). The latency of the response shortens abruptly from 10-8 ms (upper trace) to 8-5 ms (lower trace) as the stimulus intensity is increased to a critical level.

against the distance of the stimulation site from the wrist, the points obtained by weak stimulation between 16-5 and 25-5 cm and at a more proximal site (28-5 cm) were on a straight line. The points obtained by stronger stimulation between 16-5 and 25-5 cm and those at more distal stimulation sites (4-5-13-5 cm) were on another straight line (fig).

Motor nerve conduction velocities calculated from the gradients of the two regression lines were 68-0 m/s and 56-5 m/s, respectively.

The motor unit potential obtained in this patient is in complete accordence with the description of the motor axon loop by Roth and Egloff-Baer.1 In their report, the motor units with a motor axon loop were recorded as a late potential preceded by the main M-response. The present case is unique in that the MUP with a motor axon loop is the only recordable motor response. The motor axon loop is considered congenital, and it appears that the last remaining motor unit in this case happened to have a looped axon. In this particular situation, the motor nerve conduction velocity of the segment containing the loop is calculated to be erroneously low. For example, motor nerve conduction velocity between 13-5 and 28-5 cm from the wrist is calculated as:

\[ \frac{28.5 - 13.5}{12.2 - 7.2} = \frac{15}{5} = 30 \text{ m/s} \]

which is obviously wrong, but one cannot know that it is wrong unless one gives weak and strong stimulations over the looped segment of the motor axon. It should be borne in mind that the motor axon loop can be a source of error in the measurement of motor nerve conduction velocity when the number of surviving motor units is extremely small.

**Reference**


Accepted 28 July 1988.

**Matters arising**

**Computed tomographic findings of brain and skull in myotonic dystrophy**

Sir: We read with great interest the article by Avrahami et al1 about computed tomography findings of brain and skull in myotonic dystrophy and the letter which appeared later on the same topic.2 We have seen a 30 year old patient with a family history of myotonic dystrophy (mother and sister) diagnosed on a clinical and EMG basis who had sudden right ear deafness with transitory vertigo. A round osteolytic lacuna of the upper end of the internal auditory canal and of the bony wall of the first cochlear turn was seen by radiographic tomography of the right petrous pyramid. CT of the petrous temporal bone confirmed this fig) and air-CT-cisternography excluded a small intracranialular tumor.

Other causes of sudden deafness, such as virus diseases, bacterial labyrinthitis or meningitis, endolymphatic hydrop, vascular diseases, head trauma, were excluded.

Calcium and phosphorus metabolism and parathormone level were normal. Audio- logical findings were: normal hearing in the left and a complete hearing loss in the right ear, a decreased response of the labyrinthine end organ to caloric tests on the right, stapedial reflexes present at normal intensity only on stimulation of the left ear, normal ABR amplitude and latency in the left ear, ABR absent in the other one. A 3 year clinical and CT of the petrous temporal bone follow-up showed the same picture.

To our knowledge an osteolytic lacuna, which could be the cause of the sudden deafness in our patient, has not been recognised in patients with myotonic dystrophy.

Fig CT of the petrous temporal bone showing the osteolytic lacuna of the upper end of the internal auditory canal and of the bone wall of the cochlear basal turn (arrow).
Although the precise cause of this lesion remains unknown we suspect that it may be a part of the process of bone resorption and deposition in myotonic dystrophy.

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References

The frequency, characteristics and prognosis of epileptic seizures at the onset of stroke

Sir: The report by Shinton et al of a high mortality over the first two days after admission in stroke with epileptic seizures at onset, prompted us to analyse our results in a series of 421 patients with acute stroke studied prospectively between January 1986 and March 1988 by means of a stroke data bank. All patients underwent cranial computed tomography (CT). Transient ischaemic attacks were excluded. There were 267 males and 145 females with a median age of 65 years. Atherothrombotic cerebral infarct was diagnosed in 271 cases, cardioembolic infarcts in 63 and intracerebral haemorrhage in 66. In the remaining 21 cases, all with ischaemic strokes, the cause could not be established. Subarachnoid haemorrhages were sent to another hospital and have not been included. Age, sex, hypertension, diabetes, alcohol consumption (> 80 g/day), previous stroke or TIA events, heart diseases, neurological signs and symptoms at admission, vascular territory (carotid or vertebrobasilar), type of stroke, CT results, neurological deterioration, hospital stay, in-hospital mortality and morbidity at discharge were analysed. Extension of CT lesions was graded as 0: normal CT or deep lesions, 1: lesion limited to one lobe, 2: two lobes affected, 3: three lobes affected. Neurological deterioration was considered when impairment of level of consciousness or signs of transtentorial brain herniation appeared after admission. Twenty-three patients (5-6%) suffered epileptic seizures at onset or within 48 hours after stroke. Chi-square test with Yates correction showed a significant relation between seizures and in-hospital mortality, headache, loss of consciousness, extension of the cerebral infarction or haemorrhage and neurological deterioration. Age and hospital stay were similar in both groups (Mann-Whitney-Wilcoxon rank test) (table 1).

Table 1  Results of univariate analysis

<table>
<thead>
<tr>
<th></th>
<th>Cases without seizures (n = 389)</th>
<th>Cases with seizures (n = 23)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>46 (11.8)</td>
<td>7 (30.4)</td>
<td>0.023</td>
</tr>
<tr>
<td>Headache</td>
<td>66 (17.0)</td>
<td>10 (43.5)</td>
<td>0.003</td>
</tr>
<tr>
<td>Loss of conscience</td>
<td>97 (24.9)</td>
<td>13 (56.5)</td>
<td>0.002</td>
</tr>
<tr>
<td>Neurological deterioration</td>
<td>40 (10.3)</td>
<td>7 (30.4)</td>
<td>0.008</td>
</tr>
<tr>
<td>Extension of CT lesions</td>
<td>0 (0)</td>
<td>1 (4.3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 (0.2)</td>
<td>12 (52.2)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 (0.5)</td>
<td>6 (26.1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (0.8)</td>
<td>4 (17.4)</td>
<td></td>
</tr>
<tr>
<td>Type of stroke</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atherothrombotic</td>
<td>259 (65.8)</td>
<td>12 (54.5)</td>
<td></td>
</tr>
<tr>
<td>Cardioembolic</td>
<td>58 (15.3)</td>
<td>5 (22.7)</td>
<td></td>
</tr>
<tr>
<td>Cerebral hemorrhage</td>
<td>61 (16.1)</td>
<td>5 (22.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Age</td>
<td>65·1 ± 11x</td>
<td>64·8 ± 15</td>
<td></td>
</tr>
<tr>
<td>Hospital stay</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Survivors</td>
<td>13·3 ± 9</td>
<td>14·4 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>Deceased</td>
<td>9·8 ± 11</td>
<td>6·3 ± 6</td>
<td>NS</td>
</tr>
</tbody>
</table>

S: number (%); x: media ± SD

Table 2  Multiple logistic regression. Dependent variable: mortality

<table>
<thead>
<tr>
<th></th>
<th>Beta</th>
<th>T</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological deterioration</td>
<td>0.38704</td>
<td>9.044</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Loss of conscience</td>
<td>0.26232</td>
<td>6.141</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Age</td>
<td>0.12003</td>
<td>2.925</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Extension of CT lesions</td>
<td>0.08576</td>
<td>2.051</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Epileptic seizures at onset</td>
<td>0.01411</td>
<td>0.335</td>
<td>NS</td>
</tr>
<tr>
<td>Headache</td>
<td>0.01174</td>
<td>0.282</td>
<td>NS</td>
</tr>
</tbody>
</table>

Our data suggest that epileptic seizures are present frequently in patients with a large infarct or haemorrhage that involves cortical and subcortical regions and produce loss of consciousness or neurological deterioration after admission. These factors are predictors of a poorer prognosis but not epileptic seizures by themselves.

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Updated information and services can be found at:
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