Electrophysiological assessment of central and peripheral motor routes to the lingual muscles

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Abstract

Compound muscle action potentials (CMAPs) of the lingual muscles were recorded by especially devised bipolar surface electrodes placed on the tongue. Distinct responses were evoked in the tongue muscles by peripheral electrical stimulation of the hypoglossal nerve medial to the angle of the jaw and by transcranial magnetic stimulation of the motor cortex. With cortical stimulation during voluntary activation of the tongue muscles it proved easy to obtain responses with the characteristics of centrally evoked responses allowing reliable measurements of latencies and amplitudes. By contrast, responses from magnetic stimulation of the intracranial segment of the hypoglossal nerve were more difficult to obtain and the reproducibility was often not satisfactory. In a group of 20 healthy subjects the average distal motor latency of both sides from peripheral stimulation was 2·4 ms and the corresponding amplitude was 9·3 mV on the left and 8·6 mV on the right side (range 5·1–16·0 mV). Cortical stimulation gave responses with an average onset latency of 8·6 ms and 8·8 ms and an average amplitude of 1·8 mV and 2·6 mV on the left and right sides of the tongue respectively (range 0·7–5·6 mV). From this mean conduction times of 6·2 ms on the left and 6·4 ms on the right side (SD 1·0 ms) between cortex and mandibular angle and relative amplitudes from cortical stimulation as compared with the peripheral CMAP of 29% on the left and 21% on the right side (range 7%–66%) were calculated. In 16 patients it was possible to differentiate between a central (supranuclear) and a peripheral (infranuclear) site for the lesions of the motor routes to the lingual muscles and to show subclinical lesions in some cases. With a recording arrangement allowing selective unilateral recording of muscle activity from both sides of the tongue the assumed bihemispheric motor representation of the lingual muscles was confirmed.

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Since the first description by Barker *et al* in 1985, transcranial magnetic stimulation of the human motor cortex has become a non-invasive and hence widely used method to assess the functional integrity of the central motor pathways.[1–7] Cortical stimulation not only evokes twitches in limb muscles, but also in muscles supplied by cranial nerves. The facial muscles have been shown to be easily excited by magnetic stimulation of the motor cortex as well as the intracranial segment of the facial nerve.[8 9] Weakness of the lingual muscles is found in various diseases involving either the hypoglossal nerve, the hypoglossal nuclei, or the corresponding corticobulbar tracts. Electrophysiological assessment of the motor routes to the lingual muscles has not been widely established so far, except for conventional needle myography, to show axonal lesions of the peripheral motor neuron and neurography of the most distal segment of the hypoglossal nerve.[10–12] The central and proximal peripheral segments of the motor pathways to the tongue have been inaccessible by routine diagnostic studies. Here we describe a simple technique to assess the central and peripheral motor routes to the lingual muscles by transcranial magnetic stimulation of the motor cortex and electrical stimulation of the hypoglossal nerve.

Subjects, patients, and methods

HEALTHY CONTROL SUBJECTS AND PATIENTS

Twenty nine healthy subjects (14 men) with a mean age of 30 (range 20–53) years, volunteered with informed consent for the experiments. In addition, two patients with myotonic dystrophy, five with Guillain-Barré Syndrome, four with brainstem lesions, three with amyotrophic lateral sclerosis, and two with hemispheric cerebrovascular infarction were investigated. The patients underwent physical, conventional electrophysiological, and neuroradiological examination at the time of the motor evoked potential study in the scope of the usual diagnostic procedure. All normal subjects and patients were right handed. The study complied with the standards of the local ethics committee. Subjects or patients with a history of sinus-caroticus syndrome, epilepsy, or after neurological, ophthalmological, or otological surgery, as well as patients with a cardiac pacemaker were excluded from the study.

STIMULATION AND RECORDING PROCEDURE

To stimulate the hypoglossal nerve medial to the angle of the jaw, a high voltage low output impedance stimulator (Digitimer D180, maximal output 750V, decay time of 50 μs)

and bipolar saline-soaked pad electrodes with an interelectrode distance of 1·5 cm were used. Adhesive ground electrodes were placed on both cheeks. The stimulus strength was increased stepwise up to a supramaximal intensity. The motor cortex was excited by a magnetic stimulator with a circular coil of 8 cm mean diameter as described previously.[13] The stimulator had a total capacitance of 800 μF and a total charging energy of 2500 joules with a maximum output voltage of 2·5 kV.

Simultaneous surface recordings from both sides of the lingual muscles were taken with silver-silver chloride electrodes of 0·3 cm diameter placed in various positions on the tongue. The responses were amplified and recorded with a Medelec ER94a sensor system with bandpass filter from 3 Hz to 6 kHz. At least three subsequent recordings were stored on a microcomputer for further analysis. The latencies were measured to the first reproducible negative deflection from the baseline, and the amplitudes were evaluated peak to peak.

Results

EVALUATION OF RECORDING ELECTRODE

In preliminary experiments two separate conventional superficial silver-silver chloride disc electrodes were arranged over each side of the tongue and connected in bipolar fashion to the amplifier. A considerable variation in shape, latency, and amplitude of the peripherally elicited compound muscle action potentials (CMAPs) was found in the same subject, requiring a painstaking search for the optimal position of the electrodes on the tongue each time. Therefore, the electrodes were fixed in an arrangement that was found to be optimal, imbedded in a polyvinyl chloride mouthpiece as shown in fig 1 (montage A) and tested in 20 normal subjects. The subjects were asked to hold the mouthpiece tightly between the teeth while closing the mouth and to push the tongue against the electrodes and the lower teeth. The electrodes had to be repeatedly removed to allow swallowing because of the induced salivation. This was important to avoid water bridges. This recording arrangement provided CMAPs with an initial negative deflection in all control subjects for peripheral as well as for cortical stimulation (fig 2). A second arrangement with a separate active and reference electrode placed on either side of the tongue (montage B) was tested in nine normal subjects (see later). This recording arrangement allowed a selective recording of unilateral muscle activity, whereas the common reference of montage A electrode introduced contralateral activity.

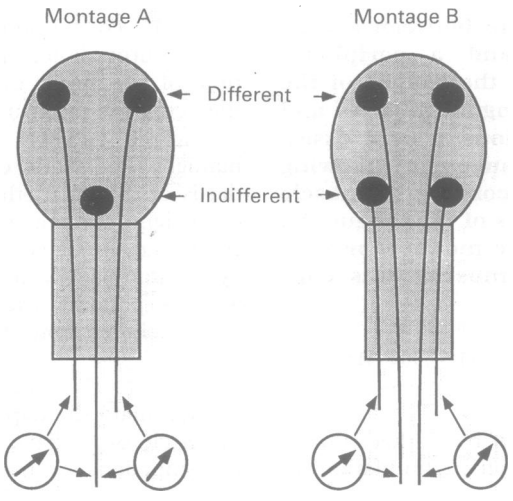

Figure 1 Mouthpiece with montages A and B of the surface recording electrodes for lingual muscles.

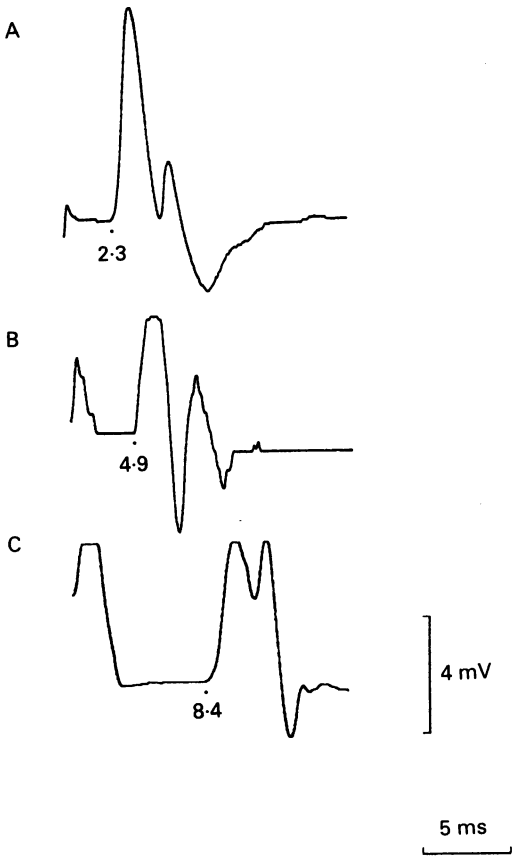

Figure 2 Motor evoked potentials recorded from the right sided lingual muscles elicited at different stimulation sites. (A) The hypoglossal nerve medial to the angle of the jaw; (B) transcranial magnetic stimulation of the intracranial segment of the nerve; and (C) transcranial magnetic stimulation of the contralateral motor cortex. The numbers indicate the latencies of the CMAP onset in ms.

For peripheral stimulation of the hypoglossal nerve, the optimal positioning of the stimulating cathode requiring minimal stimulus intensities was found to be 2 cm anterior and 1 cm medial to the angle of the jaw with the anode more posterior. To investigate possible interference from neighbouring facial muscles, simultaneous recordings from the nasalis and tongue muscles were performed. By stimulating the facial nerve at the mastoid and transcranially as described earlier, responses were recorded from the nasalis but not from the lingual muscles.[8] By stimulating the hypoglossal nerve, responses were recorded from the lingual but not from the nasalis muscle.

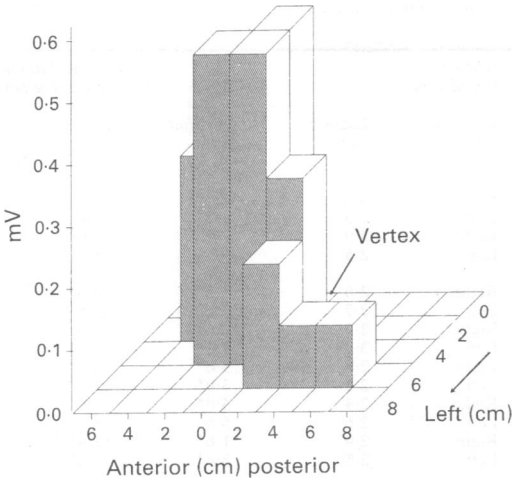

Figure 3 Amplitudes of the CMAPs elicited in the right-sided lingual muscles by transcranial magnetic stimulation of the contralateral hemisphere are related to the stimulus site. Each bar represents the average value of eight subsequent CMAP amplitudes. The position of the coil's centre relative to the vertex is indicated.

In an attempt to stimulate the intracranial segment of the hypoglossal nerve by the magnetic stimulator transcranially, the coil was placed over the back of the head with the centre 3 cm lateral to the inion and the current in the coil flowing anticlockwise as viewed from behind for the right side and vice versa. The precise positioning of the stimulating coil, however, proved to be very critical and it was rarely possible to obtain a clearcut response (fig 2B).

For motor cortex stimulation, the optimal positioning of the coil was evaluated by mapping 40 stimulation points over the scalp (fig 3). The coil's centre was moved in steps of 2 cm over the contralateral hemisphere so as to make the 8 cm diameter coil roughly cover the primary motor fields of the tongue.[14] With a constant stimulus intensity of 0·8 kV with the current in the coil flowing anticlockwise as viewed from above for the left hemisphere and vice versa, a mean amplitude from eight responses was determined for each stimulation point. The optimal position with the greatest mean amplitude was found when the coil was centred between 2 cm anterior to 2 cm posterior and 2 to 4 cm laterally of the vertex. The stimulus intensity was then increased stepwise to about 1·5 kV. A slight steady voluntary background innervation of the tongue muscles was sufficient to facilitate the responses and produce CMAPs with sizeable amplitudes. As these CMAPs showed the inherent variation typical for cortically evoked responses, the shortest reproducible latency and the greatest amplitude out of at least three responses were taken for evaluation.

Table 1 Motor evoked potentials from lingual muscles: recording with montage A electrode from 20 control subjects

	Stimulus site			
	Angular right	*Angular left*	*Cortical left*	*Cortical right*
Recording site	Right	Left	Right	Left
Latency (ms)	1·9–2·9 2·4 (0·3)	1·9–3·0 2·4 (0·3)	7·4–10·8 8·8 (0·9)	7·3–10·2 8·6 (0·9)
Side difference (ms)	0·0–0·6 0·2 (0·2)		0·1–2·0 0·8 (0·6)	
Amplitude (mV)	5·1–16·0 8·6 (2·7)	5·7–14·5 9·3 (2·7)	0·7–4·6 1·8 (1·1)	0·7–5·6 2·6 (1·3)
Side difference (mV)	0–3·3 1·3 (0·9)		0–2·6 0·8 (0·7)	
Crossed CACT (ms)			4·7–7·9 6·2 (0·9)	4·4–8·6 6·4 (1·0)
Side difference (ms)			0·1–2·3 1·0 (0·7)	
Relative amplitudes (%)			8·3–65·9 29·2 (16·0)	7·8–49·5 21·0 (12·0)
Side difference (%)			0–38·4 11·1 (9·1)	

Values are above, range; below, mean (SD); CACT = Corticoangular conduction time.

Table 2 Motor evoked potentials from lingual muscles: recording with montage B electrode from nine control subjects

Recording side	*Right*	*Right*	*Left*	*Left*
Stimulated hemisphere	Left	Right	Right	Left
Corticomuscular latency (ms)	6·1–10·8 8·1 (1·3)	7·4–10·0 8·3 (0·8)	7·4–10·3 8·4 (1·1)	7·2–10·4 8·1 (1·1)
Amplitude (mV)	1·6–8·3 4·3** (1·9)	1·3–5·2 3·1** (2·0)	2·2–7·3 4·1 (1·5)	1·4–10·6 3·0 (2·7)

** $p < 0·01$.
Values are above, range; below, mean (SD).

NORMAL SUBJECTS

Figures 2 and 4A show typical responses evoked by electrical and cortical stimulation in two normal subjects. Table 1 summarises the latencies and amplitudes including side to side differences of the motor evoked potentials obtained after motor cortex and peripheral hypoglossal nerve stimulation in 20 control subjects (40 sides). For peripheral stimulation only ipsilateral and for cortical stimulation only contralateral responses were analysed when using the more simple montage A recording electrode. These values showed no significant differences with respect to age, sex, or recording side ($p > 0·05$). The upper limit of normal for the distal latency defined as the average plus 2SD was 3·0 ms. Likewise, for the corticomuscular latency an upper limit of 10·6 ms and for the corticoangular conduction time an upper limit of 8·4 ms was calculated. The normal range for differences between the right and left side was 0·1 to 2·0 ms for the corticomuscular conduction time and 0·1 to 2·3 ms for the corticoangular conduction time. The corresponding CMAP amplitudes with peripheral stimulation ranged from 5·1 to 16·0 mV and with cortical stimulation from 0·7 to 5·6 mV. Cortically evoked CMAP amplitudes expressed as a percentage of the amplitude from peripheral stimulation (relative amplitude) ranged from 7% to 66%. Side to side differences in CMAP amplitudes were between 0 to 3·3 mV for peripheral and between 0 and 2·6 mV or 0 and 38% for cortical stimuli, respectively.

With a common reference electrode mounted at the midline of the tongue (fig 1; montage A) the recorded activity could not be attributed to a single side of the tongue. When stimulating the peripheral nerve we also obtained a contralateral CMAP with an amplitude of up to 100% of the ipsilateral response. The arrangement with two separate bipolar electrode pairs for each side of the tongue (fig 1; montage B) allowed separate recording without major interference from

Table 3 Motor evoked potentials from lingual muscles: recording with montage A electrode from 15 patients (case 15 was examined with montage B)

Patient no. (Sex, age)	Angular stimulation: Recording side	Angular stimulation: Latency (ms)	Angular stimulation: Amplitude (mV)	Cortical stimulation (crossed responses): Latency (ms)	Cortical stimulation (crossed responses): Amplitude (mV)	Cortical stimulation (crossed responses): CACT (ms)
Myotonic dystrophy						
1	Right	2·5	2·4*	7·7	0·8	5·2
(M 34)	Left	2·5	3·2*	8·0	0·7	5·5
2	Right	2·9	7·2	8·0	2·7	5·1
(M 54)	Left	2·5	9·5	8·2	2·3	5·7
Guillain-Barré syndrome:						
3	Right	2·6	3·2*	8·5	0·7	5·9
(M 63)	Left	2·7	3·6*	7·8	0·5*	5·1
4	Right	2·9	2·6*	9·6	1·3	6·7
(M 70)	Left	3·4*	1·2*	11·2*	0·8	7·8
5	Right	2·8	3·6*	14·2*	2·3	11·4*
(M 25)	Left	2·4	3·9*	13·0*	3·2	10·6*
6	Right	2·4	4·8*	8·9	0·8	6·5
(M 45)	Left	2·6	5·5	8·9	0·9	6·3
7	Right	2·9	1·6*	11·4*	1·2	8·5*
(F 23)	Left	2·5	3·8*	11·4*	0·9	8·9*
Brainstem lesion:						
8	Right	2·3	9·0	NR*	NR*	NR*
(M 44)	Left	2·2	8·4	NR*	NR*	NR*
9	Right	2·4	7·3	8·0	5·6	5·6
(M 48)	Left	2·3	8·6	10·8*	2·5	8·6*
10	Right	2·0	9·2	9·6	4·0	7·6
(F 35)	Left	1·9	9·8	12·0*	2·3	10·1*
11	Right	2·4	5·3	7·8	1·9	5·4
(M 51)	Left	2·5	6·5	NR*	NR*	NR*
Amyotrophic lateral sclerosis:						
12	Right	2·9	8·7	9·8	1·3	6·8
(F 72)	Left	2·6	7·0	NR*	NR*	NR*
13	Right	2·3	4·9*	NR*	NR*	NR*
(F 65)	Left	2·4	4·8*	11·4*	0·3*	9·0*
14	Right	2·4	6·0	10·0	0·6*	7·6
(F 71)	Left	2·2	4·8*	11·5*	0·7	9·3*
Cerebrovascular ischemia:						
15	Right	2·8	6·8	7·7	3·5	4·9
(F 31)	Left	2·3	5·8	NR*	NR*	NR*
16	Right	2·0	14·0	8·0	0·7	6·0
(F 47)	Left	2·3	11·0	NR*	NR*	NR*

*Abnormal response; NR = no response; CACT = corticoangular conduction time.

the other side but this montage required careful positioning of the mouthpiece to obtain negative onsets of the CMAPs. In a series of nine normal subjects the contralateral amplitude from peripheral stimulation remained below 20% except for two tongue sides where it reached 30% of the ipsilateral CMAP amplitude. Cortical stimulation of both hemispheres invariably produced an ipsilateral response averaging roughly 75% compared with the response on the contralateral side of the tongue (table 2). This difference in amplitude reached significance for the right-sided tongue muscles ($p < 0.01$) but not for the left side. In two normal subjects the maximal amplitude from the left-sided muscles was even greater on the ipsilateral side. For latency no significant difference (two-tailed paired t test) was found between the responses from the left and the right tongue side elicited simultaneously through crossed and non-crossed projections from either hemisphere ($p > 0.05$). Coexcitation of the contralateral hemisphere was ruled out by moving the stimulus coil gradually from one side to the contralateral hemisphere. Stimulation near the midline did not induce CMAPs on either side of the tongue (see discussion). A bilateral central representation of tongue muscles was additionally confirmed in two right-handed subjects with a more focal figure 8 stimulus coil.

Table 4 Motor evoked potentials from lingual muscles: summary of data recorded from 16 patients (15 with montage A)

Diagnosis	MD	GBS	BSL	ALS	CVI
	Unilateral or bilateral involvement (No. of patients)				
Total patients	2	5	4	3	2
One side abnormal	0	1	3	1	2
Both sides abnormal	1	4	1	2	0
Both sides normal	1	0	0	0	0
	Peripheral and/or central involvement (No. of sides)				
Total sides	4	10	8	6	4
Only PES abnormal	2	3	0	0	0
PES and TMS abnormal	0	6	0	3	0
Only TMS abnormal	0	0	5	2	2
PES and TMS normal	2	1	3	1	2

MD = Myotonic dystrophy; GBS = Guillain-Barré syndrome; ALS = amyotrophic lateral sclerosis; CVL = cerebrovascular ischaemia; BSL = brainstem lesion.

PATIENT GROUP

Table 3 gives the latencies, amplitudes, and the corticoangular conduction times of the CMAPs elicited from the lingual muscles by peripheral nerve stimulation and by cortical stimulation of the contralateral hemisphere for each of the 16 patients. In table 4 the frequency of bilateral involvement (upper part) and the preferred localisation of the pathologies within the motor route (lower part) are summarised for each patient group. Patients with a Guillain-Barré syndrome most often showed bilateral reduced amplitudes from peripheral stimulation. Often these patients also had an abnormal result from cortical

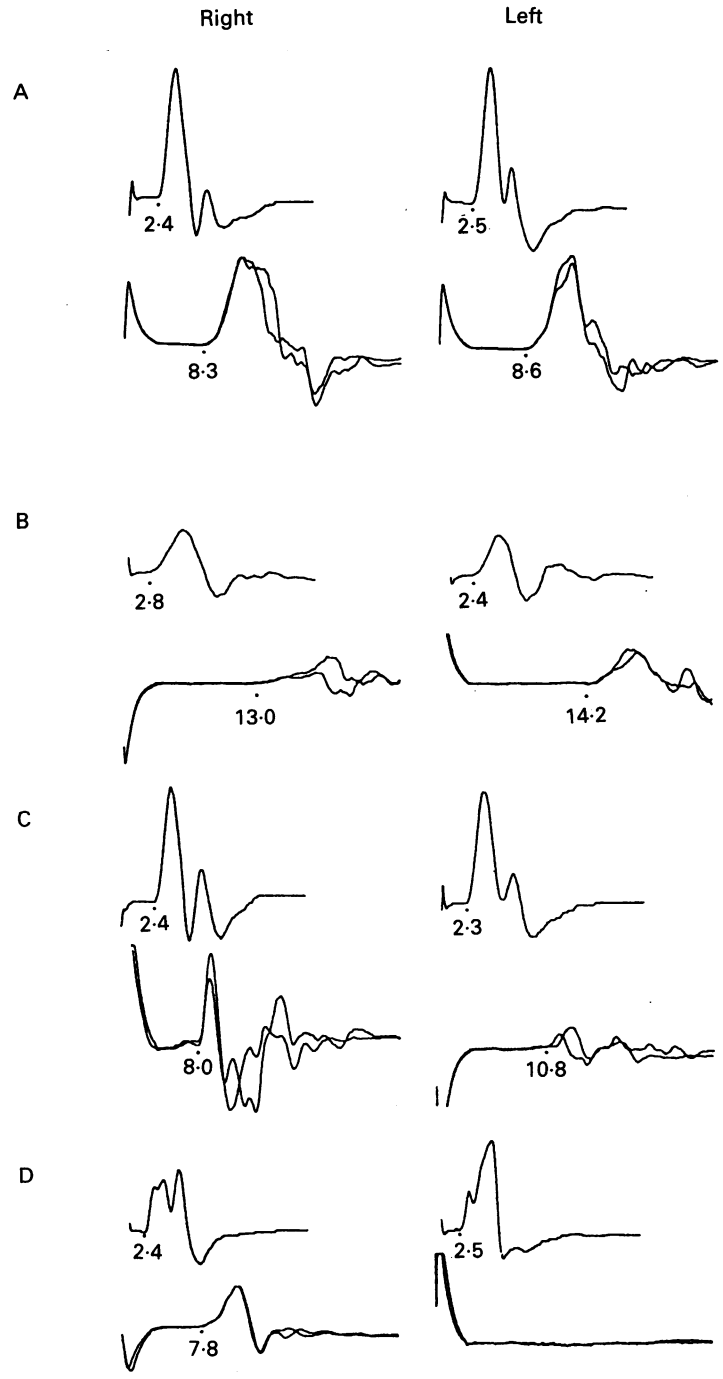

Figure 4 Compound muscle action potentials evoked by electrical stimulation of the peripheral nerve (upper traces) and by cortical stimulation (lower traces with two responses superimposed) in a control subject (A), in a patient with Guillain-Barré syndrome (B; case 5), in a patient with brainstem infarction (C; case 9), and in a patient with a pontine lesion (D; case 11). The numbers indicate the latencies of the CMAP onset in ms.

stimulation. We did not find a proximal abnormality in the three sides when the responses from peripheral stimulation were normal (case 2 bilateral; case 6 left). As expected, we found normal latencies in both patients with myotonic dystrophy, and the amplitude was bilaterally reduced in one of them. In patients with a suspected lesion in the CNS, responses were most often unilaterally absent or delayed, but responses from peripheral stimulation were normal on both sides in all six cases. In one case with a brainstem lesion the responses from cortical stimulation were bilaterally absent (case 8). In three patients with amyotrophic lateral sclerosis (six sides) for three sides the abnormalities were found in the proximal as well as in the distal segment, for two sides only a proximal abnormality was revealed and in no side was a distal abnormality alone found. Clinically an involvement of the lingual muscles with a deviation of the tongue to the weak side was seen in both patients with hemispheric infarction and in one patient (case 11) with a brainstem lesion.

Discussion

We describe an easy to perform and non-invasive technique for assessing the central and peripheral motor routes to the lingual muscles. Two different bipolar electrodes for surface recordings of CMAPs from the tongue muscles were designed. Only low stimulus intensities and slight voluntary activation of the lingual muscle were required to produce CMAPs of sizeable amplitudes and with sharp onsets. As opposed to the facial nerve, transcranial stimulation of the intracranial segment of the hypoglossal nerve was not reliably possible.

The average amplitude of 1·8 mV on the left side and 2·6 mV on the right side of the tongue from transcranial magnetic stimulation of the motor cortex found in 20 healthy subjects (table 1) is comparable with the values obtained from muscles innervated by the facial and trigeminal nerve.[8 9] The average corticomuscular latency of 8·6 ms on the left side and 8·8 ms on the right side is in the same range as the values obtained for facial muscles but longer than those supplied by the trigeminal nerve. Rösler found a corticomuscular latency of 10 ms for the facial routes when recording from nasalis muscle, and Cruccu *et al* obtained a corticomuscular latency of 5·6 ms for the trigeminal routes when recording from masseter muscle.[8 15] A direct corticomotoneuronal connection was assumed for the second but a polysynaptic pathway for the facial route. Our finding of comparatively long latencies and considerable variations in amplitude and latency of the centrally evoked CMAPs from the tongue in successive trials also suggests a polysynaptic pathway.

To investigate the pattern of crossed and uncrossed central innervation of the lingual muscles, an electrode arrangement with two separate electrode pairs on each side of the tongue was also used (montage B). In all subjects important ipsilateral innervation was found, and in two subjects even greater responses were evoked on the left side of the tongue by stimulating the ipsilateral hemisphere. Nevertheless, the average amplitudes of the CMAPs from the right side of the tongue were significantly greater when evoked from the left hemisphere compared with ipsilateral excitation. On the left side of the tongue, however, contralateral stimulation evoked only slightly greater amplitudes than ipsilateral stimulation. The corticomuscular latencies did not differ when stimulating over

the dominant or non-dominant hemisphere and no differences in latency between crossed and uncrossed projections were found either. These findings are in good agreement with the clinical and anatomical evidence of a bilateral central representation of the lingual muscles. Because no response could be evoked when stimulating at the midline with a suprathreshold stimulus strength as defined for an optimal coil position, relevant coexcitation of the contralateral hemisphere could be ruled out.

The results obtained in a small group of patients showed the diagnostic value of the method in differentiating between central and peripheral lesions and in detecting subclinical lesions. In five patients (10 sides) with a Gullain-Barré syndrome, three sides with abnormal responses from peripheral stimulation alone, and six sides with abnormal responses from peripheral as well as cortical stimulation were found. Cortical stimulation produced prolonged latencies more often than reduced amplitudes, whereas peripheral stimulation revealed reduced amplitudes of the CMAPs bilaterally in four patients and unilaterally in the fifth. Only on one side was a prolonged distal motor latency found. An abnormal response from cortical stimulation never occurred when responses from peripheral stimulation were normal. We therefore suggest that the abnormal corticoangular conduction time in the presence of abnormal peripheral responses was due to a peripheral lesion of the proximal segment of the hypoglossal nerve within its intracranial or extracranial portion. One patient with a myotonic dystrophy had bilaterally reduced responses from peripheral stimulation, whereas the other showed normal results. In no patient with a generalised peripheral nervous or muscular system disease was a tongue deviation seen. This finding could be explained either by a less severe lesion or by bilateral involvement. The second was neurophysiologically confirmed in five of the seven patients. A unilateral abnormality was found in only one patient, with a Guillain-Barré syndrome (case 6) and a patient with myotonic dystrophy (case 2) had normal results on both sides. These results indicate a large proportion of subclinical lesions in the proximal segment of the hypoglossal nerve in Guillain-Barré syndrome.

In all six patients with a brainstem or hemispheric lesion, abnormal responses from cortical stimulation and normal results from peripheral stimulation were obtained. Most often the response from cortical stimulation was absent on the affected side whereas the result on the not affected side was normal. A bilateral abnormality only occurred in one patient (case 8) with a brainstem concussion presenting as a "one and a half syndrome". In a patient (case 10) with a bilateral pontomesencephalic syndrome (laboratory supported probable multiple sclerosis) and a right-sided facial palsy, cortical stimulation showed an abnormality on the clinically less affected left side despite symmetrical tongue movements. In the remaining four patients, including two cases with hemispheric infarction, cortical stimulation confirmed the abnormality on the affected side. Interestingly, in three out of the six patients with a central lesion, a tongue deviation was clinically obvious. In the two patients with hemispheric infarction the tongue deviated to the side of the hemiparesis, a finding which has been described by others.[16] In these two patients, not only the contralateral but also the ipsilateral responses were absent when stimulating over the affected hemisphere. This gives additional evidence that magnetic stimuli applied over one hemisphere did not lead to relevant coexcitation of the contralateral hemisphere, and the same has been shown for facial muscles.[9] By contrast, in these patients, responses were readily obtained from both sides of the tongue by stimulation of the unsevered hemisphere, and this was also true when using the montage B electrode in one patient. The preserved cortical response on the paretic side of the tongue after stimulation of the unaffected ipsilateral hemisphere is evidence for the existence of uncrossed pathways that are voluntarily inaccessible during the acute stage of the disease. Likewise, the rapid clinical improvement of a tongue deviation after the hemispheric lesion could be explained by the early compensatory use of these uncrossed pathways. Similar discrepancies between voluntarily and magnetically evoked muscle activation have also been found in traumatic spinal lesions where no voluntary motor activity was possible but muscle activity could be evoked either by reinforcement manoeuvres or by transcranial electrical stimulation.[17 18] We have observed preserved responses initially from transcranial magnetic cortex stimulation in spite of tetraplegia in a patient with locked-in syndrome, who had a good clinical outcome.

In the three patients (six sides) with amyotrophic lateral sclerosis abnormal responses from both peripheral and cortical stimulation were recorded on three sides. For two sides only cortical but not peripheral stimulation elicited abnormal responses; a finding that has not previously been reported in Guillain-Barré syndrome or myotonic dystrophy.

We conclude that this technique is a non-invasive, safe, painless, simple, and clinically valuable method, and allows recognition, localisation, and quantification of clinically apparent and unapparent lesions of the central and peripheral motor routes to the lingual muscles.

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Robert Bentley Todd (1809-60) and Todd's paralysis

Habitués of King's College, London, are familiar with the Todd Prize and with Todd ward. One of 16 siblings, Todd's father's (Charles Hawkes Todd) other occupation was that of a well-known surgeon in Dublin. Bentley Todd, pupil of Robert J Graves, was author of many publications devoted to the anatomy and physiology of the nervous system. He published *Diseases of the brain and other affections of the nervous system* (1854); and edited the 6000 page *Cyclopaedia of anatomy and physiology* in five volumes (1835–59) celebrated for its scope and his scientific originality.[1]

Todd distinguished "three kinds of convulsions"—the jactitating or choreic, tetanic or tonic, and clonic or epileptiform, but he is best known for his account of postepileptic paralysis described in the Lumleian lecture:

> A paralytic state remains sometimes after the epileptic convulsion. This is more particularly the case when the convulsion has affected only one side or one limb: that limb or limbs will remain paralytic for some hours, or even days, after the cessation of the paroxysm, but it will ultimately perfectly recover.[2]

Hughlings Jackson acknowledged the Todd and Robertson theory that the local paralysis after an epileptic seizure was due to exhaustion. He worked on peripheral neuritis, physiology of the afferent and efferent pathways of the cord. He helped the foundation of the first school of nursing with Florence Nightingale at St John's House, Queen Square, London.

Born in Dublin, Todd graduated with a BA at Trinity College and LRCSI, then moved to Pembroke College, Oxford, graduating in 1833. He was anatomist, physiologist, and physician, lectured at the Aldersgate Medical school and replaced Herbert Mayo as Professor of Physiology at King's College (1836–53), and became its first Dean.[3] He was a Fellow of the Royal Society and a founder of King's College Hospital in 1840. Amongst many distinguished pupils was his friend Sir William Bowman, FRS whose work *On the structure and uses of the Malpighian bodies of the kidney* disclosed the capsule and basement membrane of the renal tubules; Bowman's membrane in the eye and his account of the ciliary muscle are well recognised.

Censor at the Royal College of Physicians, Todd gave the Goulstonian lectures (1839), Croonian lectures (1842) on "Practical remarks on gout, rheumatic fever and chronic rheumatism of the joints", and the Lumleian lectures (1849–50)[2] "On the pathology and treatment of convulsive disease."

His seminal work on spinal cord disease, in volume 3 of the *Cyclopaedia* (1847) is almost certainly the first description of locomotor ataxy (tabes dorsalis) four years before Romberg and 11 years before Duchenne.[4]

He believed in the efficacy of hard liquor which he prescribed indiscriminately, and sadly, died of alcoholic cirrhosis, when leaving his consulting room in Brook street. Had he lived longer he would almost certainly have been a founder member of staff of the National Hospital, Queen Square, London which started in 1860, the year of his premature death.

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See also p. 359.

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