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Impaired cortico-bulbar tract function in dysarthria due to hemispheric stroke

Functional testing using transcranial magnetic stimulation

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Summary

We investigated cortico-lingual and cortico-orofacial tract function utilizing transcranial magnetic stimulation in 18 consecutive patients with dysarthria due to hemispheric stroke. Delayed responses (conduction time > mean + 2.5 SD of that of 43 controls) or absent responses were considered abnormal. In all patients, motor-cortex stimulation of the lesion side demonstrated absent (13 patients) or delayed (five patients) responses to the tongue bilaterally (17 patients) or unilaterally (one patient). In 14 patients the contralateral orofacial responses were either absent (13 patients) or delayed (one patient). According to the electrophysiological findings, all lesions revealed by CT or MRI, were located within the pyramidal tract at the lower motor cortex (n = 4), the corona radiata (n = *7), and the genu of the internal capsule (*n = *3) or its posterior limb (*n = *4). We conclude that interruption of the cortico-bulbar tract fibres to muscles involved in articulation is a frequent cause of dysarthria in hemispheric stroke.*

Keywords: dysarthria; hemispheric stroke; transcranial magnetic stimulation; cortico-bulbar tract

Abbreviations: CMAP = compound muscle action potential; PCT = peripheral conduction time; TCT = total conduction time; TMS = transcranial magnetic stimulation

Introduction

Despite the frequent occurrence of dysarthria in cerebral ischaemia, e.g. 24.5% in Arboix *et al*. (1990) and 29.0% in Melo *et al*. (1992), the underlying pathophysiology has not yet been ellucidated. In individual patients with dysarthria due to restricted supratentorial lesions, the lesions were located in or near the anterior limb of the internal capsule (Ozaki *et al*., 1986; Ichikawa and Kageyama, 1991), the genu (Ozaki *et al*., 1986; Bogousslavsky and Regli, 1990; Ichikawa and Kageyama, 1991), posterior limb (Decroix *et al*., 1986; Combarros *et al*., 1992), and corona radiata and lower motor cortex (Tonkonogy and Goodglass, 1981; Ichikawa and Kageyama, 1991). Stimulation experiments and anatomical investigations have demonstrated considerable inter-individual variability of the cortical representation areas (Penfield and Boldrey, 1937) and of the fibre tract localizations (Dejerine, 1914; Ross, 1980) in normal subjects. Even when the site of the lesion is obvious from imaging studies, it may not be possible to reach a definite conclusion concerning the involvement or sparing of individual cortico-bulbar or cortico-spinal fibre subpopulations. Moreover the full extent of their functional impairment is uncertain due to the close proximity of the fibre tracts and their variable location. This dilemma can only be overcome by functional testing of relevant pathways in the individual patients. Disturbed articulation is a prominent feature in dysarthric speech. Since the tongue and the orofacial muscles are the most important articulators (Harris 1976; Langmore and Lehman, 1994), we evaluated the function of the cortico-lingual and cortico-facial projections using transcranial magnetic stimulation (TMS) and correlated the findings with lesion topography.

Patients and methods

We report the findings in 18 consecutive patients with sudden onset of neurological deficits including dysarthria due to a small unifocal supratentorial ischaemic lesion. Dysarthria was diagnosed on the basis of auditory-perceptual presentation and confirmed by two experienced speech

therapists. Speech function was assessed using a neurophonetic test battery (modified from Ziegler *et al*., 1990). Articulation was evaluated thoroughly on the basis of various samples, i.e. spontaneous speech, repetition of sentences and words, reading of a short story and rapid iterations of syllables (/pa/,/ta/,/ka/). The examination of laryngeal function included laryngoscopy, stroboscopy and perceptual examination of voice quality, voice stability, pitch and loudness. Sustained vocalization of vowels and consonants and repetition of sentences of increasing length provided information as to respiratory support. Speech tempo was measured based on syllable repetition rate per second using a sound spectrograph (CSL 4300; Kay Elemetrics Corp., Pine Brook, NJ, USA).

The localization of the lesion was characterized by CT in 18 patients and additional MRI in 11 patients. CT was performed with a Siemens Somatom (ART) using a slice thickness of 10 mm. Only images that showed well demarcated lesions were used. MRI images in the horizontal and coronal or sagittal planes were obtained with conventional spin-echo techniques using a 0.5 or 1.5 Tesla tomograph (Philips T5/ACS). All images were T_1- and T_2-weighted and gadolinium-enhanced. Slice thickness was 5 mm.

The atlases of Matsui and Hirano (1978) and Nieuwenhuys *et al*. (1988) were used as anatomical references. The size of the lesion was transferred for each patient to the appropriate reference section, irrespective of whether the lesion was located on the left or right side (Figs 1 and 2).

The cortico-facial projections were investigated by activating the orofacial muscles using TMS and recording the compound muscle action potentials (CMAP) of the buccinator muscles on either side of the face. We used pairs of Ag/AgCl-surface disc electrodes embedded 18 mm apart in a specially designed fork-shaped methacrylate device which was adapted to the oral vestibulum. The electrodes were in contact with the insides of the cheeks. Slight preinnervation was achieved by pursing the lips.

The cortico-lingual projections were examined by activating the tongue muscles using TMS and recording the CMAP on either half of the tongue. Two pairs of Ag/AgCl-surface disc electrodes at an interelectrode distance of 18 mm were mounted on a spoon-shaped metacrylate device adapted to the oral cavity. The electrodes were placed above the lateral dorsum of the tongue. Preinnervation was achieved by slightly pressing the dorsum of the tongue against the mouthpiece.

Filter settings for CMAP-recordings were 20–2000 Hz. A Magstim 200S (Novametrix, Whitland, Dyfed, UK) and a circular coil (mean diameter 9 cm) with a peak magnetic field of 2.0 Tesla were used for TMS.

For cortical stimulation the centre of the coil was positioned tangentially, 4–6 cm (tongue) and 1–2 cm (buccinator muscle) lateral to the vertex. For stimulation of the left (or right) hemisphere, side ‘A’ (or ‘B’) of the coil was viewed from above. Stimulation strength was increased stepwise until stable onset latencies were achieved. Out of four recorded responses the shortest onset latency (total conduction time, TCT) and largest amplitude (peak-to-peak) of the CMAP were measured.

The proximal peripheral facial and hypoglossal nerves were stimulated magnetically at their extra-axial intracranial segments. The circular coil was positioned at the ipsilateral parieto-occipital skull which is known to be appropriate for measuring the peripheral conduction time (PCT) (Muellbacher *et al*., 1994; Urban *et al*., 1997). For stimulation of the left (or right) peripheral nerve, side ‘B’ (or ‘A’) of the coil was visible outside. All responses were recorded at least twice to ensure reproducibility.

A detailed description of facial and lingual recording techniques and normative data has been published elsewhere (Urban *et al*., 1994, 1996, 1997). Magnetic stimulation of the peripheral hypoglossal nerve in 43 healthy subjects evoked responses in only 65 out of 86 nerves (75.6%) at a PCT of 3.6 ± 0.5 ms (Urban and Hopf, 1997). The irregular occurrence of CMAPs following TMS of the intact hypoglossal nerve has also been reported by other authors (Muellbacher *et al*., 1994; Campos *et al*., 1995).

The technique allows selective stimulation of either hemisphere and separate recording from either side of the tongue and the buccinator muscles, respectively, as demonstrated in patients with middle cerebral artery infarction, unilateral hypoglossal nerve section, and unilateral facial palsy (Urban *et al*., 1994, 1996). In controls, cortico-lingual fibres always project bilaterally from either hemisphere to the hypoglossal nuclei (Urban *et al*., 1994). Thus, involvement of the ipsi-and contralateral connections were evaluated separately. However, all cortico-orofacial fibres project to the contralateral facial subnuclei, but only 58–67% to the ipsilateral subnuclei. Thus, only the contralateral connections can be used reliably for an evaluation of the central pathways (Urban *et al*., 1997).

Electrophysiological investigations and examination of speech function were performed within the first 5 days after stroke.

Informed consent for this study was obtained from all participants and the study was approved by the local ethical commitee (LÄK Rhlol–Pfalz, Mainz).

Results

The clinical findings for each patient, including risk factors for stroke are summarized in Table 1. All patients complained of dysarthria. Dysarthria was characterized by slurring with imprecise articulation and the patients reported a ‘thick’ tongue. Articulatory movements and speech rate were mildly slowed, showing a mean syllable repetition rate of 4.5 syllables per second (normal rate $\geqslant$6 syllables per second). Modulation of pitch and intensity were reduced. Speech was not scanning, explosive, or dysprosodic. In the patient without accompanying aphasia the syntactic structure remained intact. The degree of dysarthria was usually mild to moderate; no patient had unintelligible speech. The voice was breathy, sometimes pressed, and slightly hoarse. Laryngoscopy

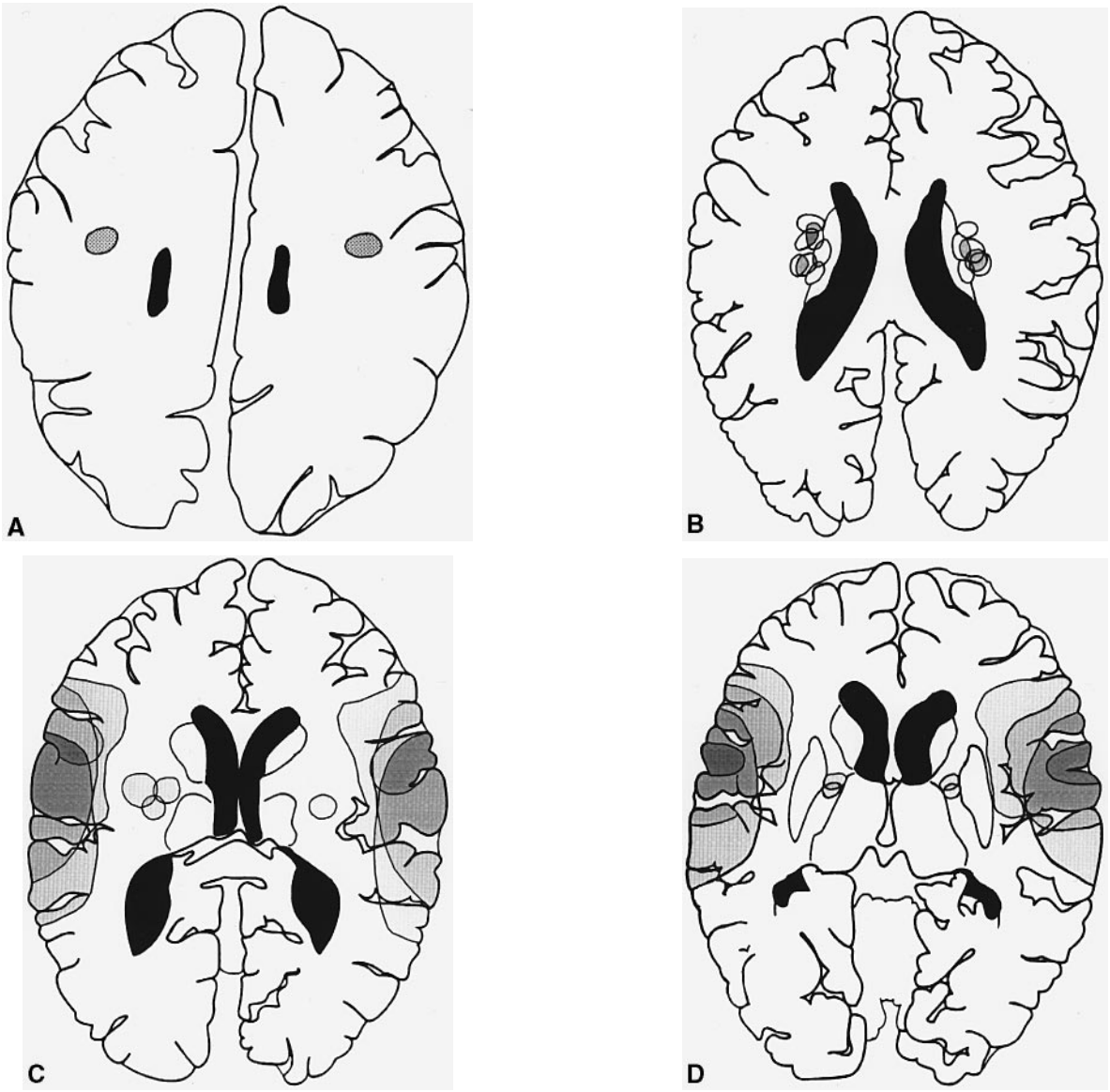

Fig. 1 Horizontal sections of the brain in cranio-caudal direction. The sections are oriented at an angle of 15° from the canthomeatal line. Locations of structural defects as seen on CT and MRI are plotted on templates derived from the atlas of Matsui and Hirano (1978) irrespective of whether the lesions were left- or right-sided. Lesions with functional impairment of the cortico-lingual (n = 18) and cortico-orofacial (n = 14) projections are shown on the left and right side of the figure, respectively.

showed normal vocal fold motility during phonation. On stroboscopy the mucosal waves and amplitudes of the vocal folds were slightly larger than normal with irregular vibrations and complete closure at medium voice intensity. Three patients (Patients 12, 14 and 18) showed additional non-fluent aphasia with severe phonematic paraphasia, neologisms, dysprosody, reduced spontaneous speech output with effortful initiation, severely impaired writing and moderate word-finding difficulties. Moderate buccofacial apraxia was present in response to command or imitation. However, simple tongue movements (sticking out the tongue, freely moving tongue around) were not affected.

Comparably large infarcts were found in the three patients with additional aphasia (Patients 12, 14 and 18) including the left lower motor cortex, the frontal operculum and the adjacent white matter. Another patient (Patient 16) with aphemia (loss of articulation; *see* Discussion) showed infarction of the left precentral gyrus and the adjacent subcortical white matter. In the remaining 14 patients infarctions were small and located in the corona radiata (seven patients), in the genu (three patients), or the posterior limb (four patients) of the internal capsule (*see* Table 1 and Figs 1 and 2).

In all 18 patients, stimulation of the motor cortex on the lesion side revealed absent (13 patients) or delayed (five patients) cortico-lingual responses of both halves (17 patients) or of one half (Patient 3) of the tongue. On clinical examination, tongue deviation was apparent in only six cases (Table 1). Thus, all patients with clinical weakness of the tongue were dysarthric and demonstrated TMS abnormalities

(Table 2). We have never encountered any patients with dysarthria due to hemispheric stroke without cortico-lingual tract involvement on TMS.

TMS-evoked orofacial responses contralateral to the lesion side were absent in 13 of the 18 patients and delayed in one (Patient 12). Clinically, central facial paresis was observed in 16 patients. Two patients (Patients 2 and 16) with central facial paresis showed normal electrophysiological results on TMS.

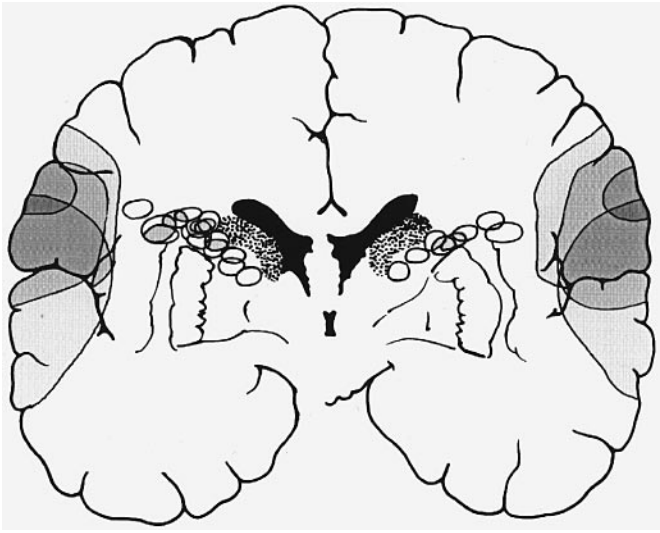

Fig. 2 Coronal section of the brain at the precentral gyrus level. Locations of structural defects as seen on CT and MRI are plotted on a template derived from the atlas of Nieuwenhuys *et al.* (1988) irrespective of whether the lesions are left-or right-sided. Lesions with functional impairment of the cortico-lingual (n = 18) and cortico-orofacial (n = 14) projections are shown on the left and right side of the figure, respectively.

TMS of the peripheral facial nerve elicited biphasic CMAPs with PCTs within the normal range in all patients. In contrast, magnetic stimulation of the hypoglossal nerve evoked CMAPs in only 26 out of 36 nerves (72.2%). PCT of the evoked responses was within the normal range.

Apart from the patients with additional aphasia, no differences in dysarthria were noted with regard to the location and size of the lesion or the side of the affected hemisphere. We found no correlation between the neurophysiological abnormalities (absence versus delay of responses, isolated cortico-lingual versus combined cortico-lingual and cortico-orofacial involvement) and the severity or auditory form of dysarthria.

Discussion

The characteristics of dysarthria observed in our patients were almost uniform. Dysarthria was mild to moderate. No differences in dysarthria were noted with regard to the location and size of the lesion and the side of the affected hemisphere. The most common features were imprecise articulation, mildly slowed speech rate and a slightly monotonous voice. These auditory findings are similar to those reported previously with hemispheric infarction (Ropper, 1987; Hartman and Abbs, 1992). The uniform

Table 1 *Clinical and magnetic resonance imaging data from patients with dysarthria due to hemisperic stroke*

Patient (sex)	Age (years)	Lesion/diameter (cm)	Facial paresis	Lingual paresis	Upper limb	Lower limb	Other	Risk factors
1 (M)	64	R-corona radiata/2.0	+	+	Central paresis (4–5)	(5)	Slight hemiataxia	SM
2 (M)	59	R-internal capsule/1.0	+	−	(5)	(5)	–	HT
3 (M)	65	R-corona radiata/1.5	−	−	Central paresis (3)	Central paresis(4–5)	–	HT, SM, HL
4 (F)	76	L-corona radiata/1.0	+	+	Central paresis (0)	Central paresis (4)	Dysphagia, hemihypesthesia	HT
5 (F)	75	R-internal capsule/1.0	+	_	Central paresis (0–1)	Central paresis (4)	–	HT, DM
6 (M)	59	L-internal capsule/0.5	+	−	Central paresis (3–4)	Central paresis (4)	–	HT, DM
7 (M)	63	L-corona radiata/1.0	+	−	Central paresis (0)	Central paresis (1–2)	–	HT, DM
8 (M)	71	L-internal capsule/1.5	+	−	Central paresis (4–5)	Central paresis(4–5)	–	DM
9 (F)	57	R-corona radiata/1.0	(+)	+	(5)	(5)	–	HT
10 (M)	59	L-corona radiata/1.5	+	(+)	Central paresis (4–5)	(5)	–	–
11 (M)	55	L-internal capsule/2.5	+	−	Central paresis (4–5)	Central paresis (4–5)	Hemihypesthesia	HL
12 (M)	60	L-bulbar motor cortex/3.0 frontal operculum, SWM	+	−	(5)	(5)	Non-fluent aphasia	HT, DM
13 (M)	82	L-corona radiata/1.0	+	−	Central paresis (4–5)	Central paresis (4–5)	–	–
14 (F)	80	L-bulbar motor cortex/3.0 frontal operculum, SWM	+	−	Central paresis (0)	Central paresis (0)	Non–fluent aphasia	HL
15 (M)	48	L–internal capsule/1.0	−	+	(5)	(5)	–	HL
16 (M)	60	L-bulbar motor cortex/1.0	+	−	Central paresis (4–5)	(5)	Aphemia	HT
17 (F)	74	R-internal capsule/1.5	+	−	Central paresis (4–5)	Central paresis (4)	Hemihypesthesia	HL, AF
18 (M)	45	L-bulbar motor cortex/3.0 frontal operculum, SWM	+	(+)	(5)	(5)	Non-fluent aphasia	–

SWM = subcortical white matter; + = marked paresis, (+) = slight paresis, – = no paresis; limb paresis was graded on a five-point scale (5 = full strength, 0 = plegia); HL = hyperlipidemia; SM = cigarette smoker; HT = arterial hypertension; DM = diabetes mellitus; AF = atrial fibrillation.

speech abnormality, as associated with the above-mentioned lesions, is consistent with a common pathophysiological basis.

Impaired articulation is one of the most prominent features of dysarthria. The tongue and orofacial muscles are the most important articulators (Harris, 1976; Langmore and Lehman, 1994). Cortico-lingual fibres project bilaterally from either hemisphere to the hypoglossal nuclei (Brodal, 1981; Urban *et al*., 1994) and the cortico-orofacial fibres project predominantely to the contralateral subnuclei (Brodal, 1981; Urban *et al*., 1997). Involvement of the ipsi-and contralateral connections can be separately evaluated (Urban *et al*., 1994). In ischaemic cerebral lesions, the degree of limb-muscle paresis correlates with an increase in latency, and decrease in amplitude, of the muscle response to TMS (Abbruzzese *et al*., 1991; Ferbert *et al*., 1992). These parameters reflect the degree of functional impairment of the fast conducting large diameter pyramidal fibres (Eisen *et al*., 1990) and absence of a response indicates a more severe effect than a delayed response. Since amplitudes of the potentials evoked by TMS show a wide interindividual variation (Amassian *et al*., 1989; Eisen *et al*., 1991), which also applies to tongue muscle responses (Urban *et al*., 1994), only absent or delayed ($>$mean + 2.5 SD) responses were considered abnormal. Control values were obtained from 43 healthy subjects (Urban *et al*., 1994, 1997). The recorded values were within the same range as values reported by Muellbacher *et al*. (1994), who investigated cortico-lingual projections in nine subjects with a comparable recording technique, and by Benecke *et al*. (1988), who investigated the cortico-facial projections to the mentalis muscle using needle recording electrodes.

Clinical examination revealed cortico-bulbar tract involvement in 17 of 18 patients (impaired tongue motility in six and central type facial paresis in 16), and cortico-spinal tract involvement in 13 out of 18 patients (increased tendon reflexes, Babinski's sign or paresis of the upper limb in 13 patients and of the lower limb in 10). Electrophysiological testing, however, demonstrated cortico-bulbar tract impairment in all patients. The abnormalities of the cortico-lingual fibres were bilateral in 17 patients and ipsilateral in one (Patient 3). Cortico-orofacial fibre disorder was found in 14 patients (Table 2). Thus, as has also been found for multiple sclerosis patients (Urban *et al*., 1994), and in stroke patients, functional testing of cortico-lingual projections utilizing TMS shows a considerably higher sensitivity than clinical examination (18 patients were shown to have abnormalities with TMS, whereas only six patients were shown to have impaired tongue motility by clinical examination. However, concerning the cortico-facial pathway, TMS has a lower sensitivity in detecting impairment of cortico-facial projections compared with clinical examination (14 patients with abnormalities revealed by TMS versus 16 patients with lower facial paresis revealed by clinical examination). The reason for detecting more subclinical cortico-lingual tract lesions than subclinical cortico-facial tract lesions is probably associated with the

Table 2 *Results of transcranial magnetic stimulation in patients with dysarthria due to hemispheric stroke*

Patients (affected hemisphere)	Total conduction time at different recording sites (ms)					
	R tongue (L cortex stimulus)	L tongue (L cortex stimulus)	R tongue (R cortex stimulus)	L tongue (R cortex stimulus)	R buccinator (L cortex stimulus)	L buccinator (R cortex stimulus)
1 (R)	11.1	9.4	NR	15.8*	NR	9.3
2 (R)	10.4	10.9	12.3*	12.4*	9.9	10.4
3 (R)	8.1	8.4	12.2*	11.0	10.0	10.1
4 (L)	NR	NR	9.3	10.2	NR	9.6
5 (R)	8.1	7.4	NR	NR	9.1	NR
6 (L)	17.4*	18.9*	8.5	9.2	NR	12.3
7 (L)	NR	NR	9.5	9.6	NR	11.9
8 (L)	NR	NR	9.8	9.9	NR	10.4
9 (R)	8.4	8.1	13.4*	12.0*	9.5	NR
10 (L)	NR	NR	10.2	10.1	NR	11.3
11 (L)	NR	NR	9.3	10.9	NR	11.1
12 (L)	NR	NR	8.3	12.9*	14.8*	11.8
13 (L)	NR	NR	10.3	8.8	NR	11.7
14 (L)	NR	NR	9.8	8.3	NR	9.3
15 (L)	11.9*	17.3*	8.9	10.3	9.7	11.1
16 (L)	8.6	8.2	NR	NR	9.7	9.6
17 (R)	NR	NR	9.9	8.7	12.7	NR
18 (L)	NR	NR	9.1	8.9	NR	11.1
Control subjects						
Mean ± SD	8.9 ± 0.9	8.8 ± 1.1	8.8 ± 1.2	8.8 ± 1.1	10.3 ± 1.0	9.8 ± 1.0
Upper limit	11.1	11.6	11.8	11.6	12.8	12.3

Total conduction times for the cortico-lingual (tongue) and cortico-orofacial (buccinator muscle) projections (*see* methods). R = right; L = left. Abnormal results are shown by NR (no response) and *(delayed response, i.e. time $>$ upper limit of control subjects, defined as mean + 2.5 SD).

bilateral symmetric tongue innervation by which the sequels of a lesion of one hemisphere are clinically masked, whereas the predominantly unilateral (contralateral) innervation of the lower facial muscles leads more often to a clinically apparent paresis.

TMS of the peripheral facial nerve showed normal results in all patients. TMS of the peripheral hypoglossal nerve evoked CMAPs of normal latency in 72.2% of the stimulated nerves, which is identical to the results obtained in controls (75.6%, *see* Patients and methods). The irregular occurrence of responses following TMS of the intact hypoglossal nerve has also been reported by other authors (Muellbacher *et al*., 1994; Campos *et al*., 1995) and is probably due to the short course of the hypoglossal nerve through the CSF and to its deep location at the base of the skull (Benecke *et al*., 1988). Therefore, a delayed response, not only the absence of a response, following TMS of the peripheral hypoglossal nerve may be considered abnormal. Together with absent clinical signs of peripheral facial and hypoglossal nerve lesions, the observed conduction abnormalities in our patients, following cortical stimulation, must be attributed to central lesions, as seen in their CT and MRI scans.

Our findings demonstrate that cortico-lingual projections (and to a lesser degree cortico-orofacial projections) are impaired in patients with dysarthria. The hypoglossal and facial nerves are responsible for the motor innervation of the tongue and orofacial muscles, whose precise interactions are required for producing different sounds (MacNeilage *et al*., 1964; Bole and Lessler, 1966). The anatomical bases are monosynaptic connections between the motor cortex and hypoglossal nucleus/orofacial subnucleus (Kuypers, 1958; Jenny and Saper, 1987; Iwatsubo *et al*., 1990) and the high innervation density of the tongue and the lips with small motor units (Bowman, 1971; Happak *et al*., 1988). Since the tongue and orofacial muscles are the most important articulators (Harris, 1976; Langmore and Lehman, 1994), we conclude that impairment of the central lingual and orofacial motor subsystems account for the imprecise articulation observed in dysarthric speakers.

The origin of the pyramidal tract is not known in detail. However, the present consensus is that in humans ~60% of pyramidal tract axons arise from the primary motor cortex (Brodmann's area 4) (Davidoff, 1990). Stimulation experiments have demonstrated that the cortico-bulbar fibres emerge from the lower part of the precentral gyrus (Foerster, 1936; Penfield and Boldrey, 1937). They are part of the corona radiata following a 'screw-like' course through the centrum ovale (Foerster, 1936) and converging into the internal capsule. The pyramidal tract enters the rostral capsule in the anterior half of the posterior limb and progressively shifts to the posterior half of the posterior limb at a more caudal level (Ross, 1980). Apart from this shift there is also some variability within the posterior limb (Ross, 1980). Within the pyramidal tract, the fibres are somatotopically arranged (Foerster, 1936), but overlapping may occur (Brodal, 1981). Fibres to the cranial nerve nuclei are located most anteriorly near the genu, followed by the cervical, thoracic, lumbar and sacral segments located successively more dorsally. Thus, the high coincidence of cortico-lingual and cortico-facial tract involvement in dysarthric patients is probably due to the close proximity of the two tracts during their entire course from the motor cortex to the brainstem.

Lesions demonstrated by CT and MRI have been located within the pyramidal tract (Brodal, 1981; Nieuwenhuys *et al*., 1988), between the lower motor cortex and the genu and the posterior limb of the internal capsule. The interindividual variability of the pyramidal tract localization within the internal capsule (Ross, 1980) has been confirmed by our findings (Fig. 1B). In the four patients with additional non-fluent aphasia (Patients 12, 14 ,16 and 18), the lesions extended from the left lower motor cortex into the frontal operculum and the adjacent white matter, which is different from the classical Broca's aphasia area (Mohr *et al*., 1978). Apart from the classical form, other subtypes of non-fluent aphasia are (i) aphemia with impaired articulation and prosody associated with lesions of the left lower motor cortex and the posterior portion of the inferior frontal gyrus (Schiff *et al*., 1983), (ii) transcortical motor aphasia with delayed initiation of language, sparse utterances, semantic paraphasias, normal articulation and preserved repetition with lesions of the white matter anterolateral to the left frontal horn of the lateral ventricle, sparing the opercular and precentral structures (Freedman *et al*., 1984), and (iii) buccofacial apraxia with impaired articulation and prosody, phonemic errors in all output, with lesions of the lower motor cortex, as well as in the subcortical and paraventricular white matter. Aphemia was the condition in patient 16 showing dysarthria and disturbed fine finger movements of the right hand. MRI demonstrated a small lesion confined to the left precentral gyrus and the adjacent subcortical white matter. The speech and lesion pattern recorded in Patient 14 corresponds to subtype (iii) described above. Classical Broca's aphasia was present in Patients 12 and 18, whose infarction areas showed the greatest extension into the medial and frontal subcortical white matter (Figs 1C and D, and 2). This demonstrates that the lower motor cortex or its efferent pathways are involved in different subtypes of non-fluent aphasia which can be diagnosed by TMS of the cortico-bulbar tract.

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