

Left-hemispheric dominance for articulation: a prospective study on acute ischaemic dysarthria at different localizations

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Dysarthria is a frequent symptom in cerebral ischaemia. However, speech characteristics of these patients have not previously been investigated in relation to lesion site in a prospective study. We investigated the auditory perceptual features in 62 consecutive patients with dysarthria due to a single, non-space-occupying cerebral infarction confirmed by MRI. Standardized speech samples of all patients were stored within 72 h after stroke onset using a digital tape recorder. Speech samples were assessed independently by two experienced speech therapists, who were unaware of the clinical and neuroradiological findings, using an interval scale ranging from 0 to 6. Separately assessed were features of articulation, phonation, prosody, and the global severity for a total of 31 items. Extracerebellar infarctions (85.5%) were located in the lower motor cortex (14.5%), striatocapsular region (46.8%) and base of the pons (24.2%). Isolated cerebellar infarctions were present in 14.5% of patients. There was a strong correlation between the findings of both examiners, showing identical scores, or only minor differences (<1 on the assessment scale) for 80% of all items. The average severity of dysarthria was 2.9 ± 1.3 . Articulatory abnormalities were the predominant deviation characteristics, affecting in particular the production of consonants. However, phonatory and prosodic abnormalities were also frequently observed speech characteristics. As revealed by factor analysis of speech characteristics the total severity of dysarthria was mainly influenced by the impairment of articulation. Speech parameters describing characteristics of articulation and prosody showed significant side-to-side and area differences, while this effect was lacking for any voice parameter. Left cerebral lesions showed a more severe overall impairment of speech and articulation, independent of lesion topography. Thirty-eight of 62 patients were available for follow-up. Speech evaluation showed normal speech within weeks in 15 out of 38 patients (39.5%). In the other 23 patients overall severity of dysarthria was mild. This is the first prospective study which describes speech characteristics of dysarthria due to acute unilateral cerebral infarctions. We could demonstrate that dysarthria in extracerebellar infarctions was more frequently caused by left-sided lesions and that the severity of dysarthria was more pronounced in left-sided lesions independent from lesion topography. All extracerebellar lesions were located along the course of the cortico-bulbar tract fibres. Compatible with a common pathophysiological basis of dysarthria in these patients, none of the 31 speech items differed significantly between subcortical and brainstem lesions.

Keywords: dysarthria; cerebral ischaemia; stroke; lesion topography

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Introduction

Dysarthria is a speech disorder characterized by dysfunction in the initiation, control and coordination of the articulatory structures involved in speech output (Peacher, 1950; Stamoulis *et al.*, 1995). Dysarthria has been observed in 8–30% of all patients included in a number of large stroke

series (Bogousslavsky *et al.*, 1988; Ghika *et al.*, 1989; Arboix *et al.*, 1990; Melo *et al.*, 1992; Kumral *et al.*, 1998), and may be the first and only clinical manifestation of cerebral ischaemia. For example, several lacunar syndromes, such as ‘pure motor hemiparesis’, ‘ataxic hemiparesis’, ‘dysarthria-clumsy hand

syndrome' and 'pure dysarthria' include dysarthria among the defining clinical characteristics (Fisher, 1982). Dysarthria has been reported in 25% of patients with lacunar stroke (Arboix *et al.*, 1990) and 30% of patients with stroke in the internal capsule (Fries *et al.*, 1993). Unfortunately, terms used for the description of dysarthria found in the literature are usually vague as e.g. 'slow', 'slurred' and 'thick' speech.

Despite the common occurrence of dysarthria in acute stroke, there have been no prospective studies investigating auditory comprehension in relation to the lesion sites to date. Previous perceptual analyses were performed retrospectively, which included only a small number of patients, did not determine the exact lesion topography, or excluded multiple lesions and enrolled patients up to 3 months after the onset of stroke (Ackermann *et al.*, 1992; Hartmann and Abbs, 1992; Thompson and Murdoch, 1995; Duffy and Folger, 1996).

We present the data of 62 consecutive patients with dysarthria due to a single cerebral infarction. The purpose of this study was to characterize the most salient deviant speech dimensions, and their association with neurological findings as well as lesion topography.

Patients and methods

Inclusion criteria

We report on 62 consecutive patients (18 females, 44 males, mean age: 64.7 ± 10.8 years, range: 34–87 years) with sudden onset of dysarthria due to a single, non-space-occupying cerebral infarction, and the absence of a history of previous stroke or transient cerebral ischaemia. All patients were native German speakers. About two-thirds of patients were initially admitted to our stroke unit, which provides all current diagnostic tools and treatment options (Krespi *et al.*, 2003). At the time of speech evaluation no patient was treated by sedative drugs. Dysarthria was diagnosed on the basis of auditory comprehension during spontaneous speech, expository speech, and oral reading. In the presence of only slight speech deviation, the subjective impression of the patients and their relatives was also considered. Not included were patients with stroke in the subacute or chronic stage, and patients with disturbance of consciousness, dementia, aphasia, and anarthria. Handedness was assessed by the Edinburgh handedness inventory (Oldfield, 1971): 92.2% of patients were right-handed and 7.8% left-handed. Informed consent for this study was obtained from all participants and the study was approved by the local ethics committee (LÄK Rheinland-Pfalz, Mainz).

Speech examination

The evaluation of dysarthria was performed by an experienced speech pathologist within the first 72 h after stroke onset. Speech function was assessed using a neurophonetic test battery (modified from Ziegler *et al.*, 1990). A Token-test was carried out to exclude aphasia. Articulation was evaluated on the basis of various samples, i.e. spontaneous speech, repetition of sentences and words, reading of a short story, and rapid iteration of syllables (/pa/, /ta/, /ka/). The examination of laryngeal function included the perceptual examination of voice quality, voice stability, pitch, and loudness. Sustained realization of vowels and fricatives and repetition of sentences of increasing length provided information on respiratory support. In all patients a standardized speech sample of 25 min was tape recorded

(DAT-recorder, Sony TCT-D7) including spontaneous speech, repetition of sentences, sustained vowels, alternate motion rates (/pa/, /ta/, /ka/), and reading of a text passage ('the northwind and the sun'). All recordings were performed in a quiet room with a constant mouth-to-microphone distance of 10 cm. The tape recordings were assessed independently by two other experienced speech pathologists, who were unaware of the clinical and neuroradiological findings. The perceptual analysis of the recordings followed a standardized protocol comprising 31 items including articulation, voice, prosody and a total severity score (Darley *et al.*, 1975; Ziegler *et al.*, 1990). The speech pathologists were asked to rate each item using a 7-point equal-appearing interval scale. On this scale, 0 represents normal and 6 very severe deviation from normal. Each speech pathologist was then allowed unlimited time to listen to the speech samples, and to rate each speech dimension.

Follow-up examinations

All 62 patients were contacted after an interval of at least 6 months (6–38 months, median: 10.5 months). A minimum interval of 6 months was considered, since the recovery is nearly complete after this period of time, and only little functional improvement may be expected (Wade *et al.*, 1985). The follow-up visit included a neurological and a speech examination with the same protocol as described for the acute stage.

Neuroimaging

Location of the lesion was identified by CT ($n = 62$) and MRI ($n = 59$) scans. MRI was not done in three patients because of claustrophobia ($n = 1$), weight >120 kg ($n = 1$) or cardiac pacemaker ($n = 1$). CT was performed with a Picker PQ 5000 (slice thickness of 5 mm infra-tentorial and 10 mm supratentorial). MRI was carried out with conventional spin echo techniques on two 1.5 T systems (Siemens Vision and Philips S15). Transverse and coronal and sagittal T1- and T2-weighted slices were measured before and after i.v. administration of contrast medium (slice thickness 5 and 3 mm supra- and infra-tentorial, respectively, without gaps). Additional EPI (echo-planar imaging)-based diffusion-weighted images were obtained in the last 33 patients of our series to confirm recent ischaemic lesions (Fitzek *et al.*, 1998). The area of infarction was identified independently by two experienced neuroradiologists unaware of the clinical findings. Patients with multiple lesions or diffuse white matter lesions (grade 1 and 2 abnormalities, according to van Swieten *et al.*, 1990) were excluded from the study. Infarctions were assessed as lacunes if they appeared on CT and MRI as sharply marginated, round, ovoid, or linear lesions without mass effect of <1.5 cm in diameter, and were located in the territory of deep perforators irrespective of clinical presentation (Fisher, 1965; Sacco *et al.*, 1991; Bogousslavsky *et al.*, 1992; Adams *et al.*, 1993).

Statistical analysis

All statistical calculations were performed using 'Statistica' software for Windows (StatSoft Inc., USA). Differences between areas of infarction (cortical, subcortical, pontine and cerebellar), and side (right versus left) were compared using a two-way analysis of variance (ANOVA). This procedure was repeated for all speech parameters as described elsewhere in this paper. For each parameter *post hoc*-comparisons were calculated using LSD *post hoc*-tests (LSD = least significant difference). Affinities of different speech characteristics were compared using a principal component factorial

analysis of raw data. This calculation was performed using the standard varimax algorithm.

Results

History and clinical findings

Isolated dysarthria ('D') occurred in one out of 62 patients (1.6%). All other patients presented with additional signs. The majority of patients (85.0%) in the extracerebellar infarction group showed pyramidal tract signs, affecting the face (79.6%), tongue (24.1%), upper limb (66.7%) and lower limb (48.1%). In patients with cerebellar infarction (with and without brainstem involvement) ataxia of stance and gait dominated (88%). Dysphagia was observed in 6.5% of patients. Dysarthria as part of a 'classic lacunar syndrome' (Fisher, 1967) was encountered in 53.1%; 'dysarthria-clumsy hand syndrome' (D-CHS) in 14.1%, 'dysarthria and pure motor hemiparesis' (D-PMH) and/or 'ataxic hemiparesis' (D-AH) in 23.4%, and dysarthria associated with central facial (D-F) or lingual (D-L) paresis in 10.9 and 3.1% of patients, respectively.

Lesion location

The lesion topography is presented in Table 1. All extracerebellar lesions were located along the course of the corticobulbar tract. Isolated cerebellar infarctions were present in nine patients, in all of whom the paravermal region within the territory of the superior cerebellar artery (SCA) was affected. Dysarthria due to extracerebellar infarctions more often affected the left hemisphere (89.5 versus 10.5%), while in cerebellar infarctions the right side was more frequently involved (77.7 versus 22.3%). There was no association with handedness.

Deviant speech characteristics

All patients complained of a sudden onset of speech difficulties, and reported a 'thick' and 'heavy' tongue, difficulties to articulate words or 'speaking like an inebriated person'.

Ratings of both listeners showed a significant correlation ($r = 0.54$; $P < 0.001$) and the rating scores between both listeners were either identical or differed only by one point

Table 1 Localization of infarctions in ischaemic dysarthria ($n = 62$)

Localization	Patients	Percentage	Side of infarction	
			Right	Left
Supratentorial	38	61.3%	4 (10.5%)	34 (89.5%)
Motor cortex	9	14.5%	2	7
Striatocapsular	29	46.8%	2	27
Brainstem/pons	15	24.2%	2 (13.3%)	13 (86.7%)
Cerebellum	9	14.5%	7 (77.7%)	2 (22.3%)

Lesions causing dysarthria were predominately located in the left brain with the exception of more frequent right-sided infarctions in the cerebellum.

on the rating scale in 80% of all items. For statistical analysis (ANOVA, factor analysis) each rating of each listener was used resulting in two judgements per patient and item. Lower agreements (>1 point) were found for the items 'total impairment of voice' (1.1 ± 1.2), 'low pitch' (1.9 ± 1.1), voice qualities ('harsh': 2.3 ± 1.2 , 'breathy': 1.5 ± 1.9 and 'pressed': 2.2 ± 2.0), and for 'speech pauses' (2.0 ± 1.2).

The overall severity of dysarthria was mild-to-moderate (2.9 ± 1.3 , range: 0.5–6.0) (Fig. 1) and the intelligibility was not or only mildly affected. No patient had unintelligible speech. The severity scores, distribution and frequency of deviant speech characteristics are presented in Table 2.

Deviant speech characteristics due to lesion topography

Regarding the total severity of dysarthria, left-sided lesions, irrespective of the lesion topography, were associated with a more severe speech impairment than right-sided lesions (ANOVA, $P < 0.001$, Fig. 2, Table 3). Global articulation was more severely affected in all patients with left-sided lesions, while global prosody was significantly more pronounced in left-sided pontine and cerebellar lesions. Total impairment of voice did not differ significantly depending on the lesion topography.

Regarding the lesion side, speech items as listed in Table 3 showed significant differences with more severe deviations in left-sided lesions for the items articulation and prosody, affecting 'vowel articulation', 'speaking rate variable over time', 'prolonged phonemes and syllables', 'speech intonation reduced', 'articulatory inaccuracy', 'reduction or elision of phonemes and syllables', 'repetition of phonemes and syllables', 'speech tempo slowed'. Only one item ('hyponasality') was significantly more pronounced in right-sided lesions of the cortex.

Regarding the lesion areas, differences were significant only for cerebellar and cortical lesions. A significantly more

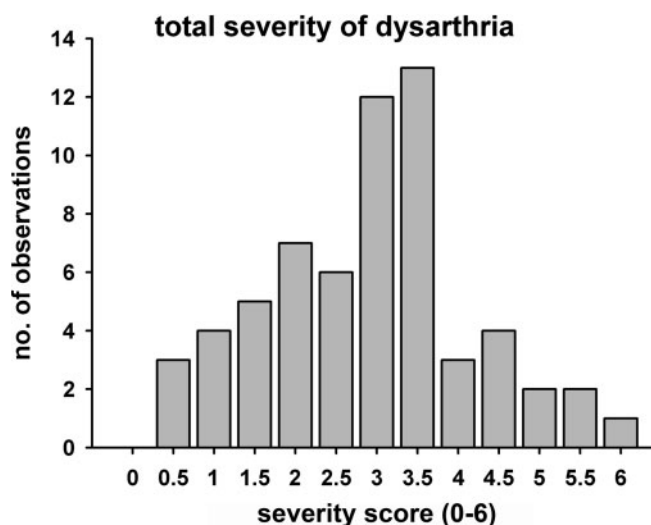


Fig. 1 Distribution of the total severity of dysarthria in 62 patients with acute unilateral ischaemic stroke.

Table 2 Perceptual speech characteristics in dysarthria

Perceptual speech characteristics	Severity (score 0–6) Mean ± SD	Distribution of severity (score 0–6)			Frequency of deviant speech characteristics Percentage
		≤1.5	2–4	4.5–6	
Global scores					
Total severity of dysarthria	2.9 ± 1.3	10	43	9	100
Total severity of articulation	2.6 ± 1.4	17	36	9	95.2
Total severity of voice	2.9 ± 1.1	6	48	8	100
Total severity of prosody	2.1 ± 1.2	20	40	2	93.5
Articulation					
Articulatory inaccuracy	2.8 ± 1.4	13	40	9	95.2
Consonant articulation imprecise	2.4 ± 1.4	18	37	7	95.2
Reduction or elision of phonemes and syllables	1.9 ± 1.1	26	34	2	90.3
Articulatory precision variable/ Irregular articulatory breakdown	1.6 ± 1.0	34	28	0	88.7
Vowels imprecise and/or distorted	1.1 ± 1.0	54	6	2	83.9
Hypernasality	1.0 ± 1.0	43	19	0	67.7
Repetition of phonemes and syllables	0.5 ± 0.8	55	7	0	48.4
Hyponasality	0.5 ± 0.9	57	5	0	35.5
Voice					
Voice quality 'harsh'	3.0 ± 1.3	13	40	9	100
Voice quality 'breathy'	2.1 ± 1.0	19	40	3	100
Voice quality 'pressed'	2.1 ± 1.2	25	34	3	96.8
Voice irregularity, pitch breaks	1.9 ± 1.0	28	32	2	96.8
Variable voice quality	1.2 ± 0.7	50	12	0	93.5
Uncontrolled changes of pitch	1.3 ± 0.9	42	20	0	87.1
Loudness decreased	1.7 ± 1.1	33	39	0	78.2
Pitch too low	1.6 ± 1.1	40	22	0	63.2
Voice tremor	0.8 ± 0.9	51	11	0	56.5
Uncontrolled changes of loudness	0.5 ± 0.6	58	4	0	46.8
Pitch too high	0.7 ± 1.2	51	10	1	25.0
Prosody					
Speech pauses	2.9 ± 1.2	12	40	10	100
Speech intonation reduced	2.0 ± 1.1	26	35	1	91.9
Speaking rate variable over time	1.4 ± 1.0	43	18	1	85.5
Prolonged phonemes or syllables	1.1 ± 0.8	50	12	0	82.3
Speech tempo slowed	1.9 ± 1.3	23	35	4	77.0

Severity, distribution and frequency of perceptual speech characteristics in dysarthria ($n = 62$) due to acute unilateral ischaemic stroke (rating score: 0; normal function to 6; severe deviation).

severe involvement of cerebellar lesions as compared to extracerebellar lesions was found for the items 'consonant articulation', 'speaking rate slowed over time', 'prolonged phonemes and syllables', 'articulatory inaccuracy', 'reduction or elision of phonemes and syllables', 'repetition of phonemes and syllables', and 'vowels imprecise and/or distorted', indicating that cerebellar lesions affect parameters of articulation and prosody more pronounced than voice characteristics.

Factor analysis: influence of the impairment of articulation and voice on the total severity of dysarthria

A principal component factorial analysis was computed using varimax rotated raw data. Using a two-factorial model with factor 1 representing impairment of articulation and factor 2 representing impairment of voice altogether 50.4% of the

total variance in dysarthria was explained. This statistical approach allows to judge the influence of different speech characteristics on global severity of dysarthria by comparing factor loadings. Figure 3 illustrates the different factor loadings of the global severity scores for dysarthria, articulation, voice, and prosody—accompanied by the single speech parameters referring to each global score. Factor 1 explained the largest part of variance (37%), indicating that impairment of articulation has the most prominent effect on global dysarthria. The influence of the impairment of voice (factor 2) seemed to play a minor role. Prosody takes an intermediate position without any significant factor loading.

Follow-up examinations

Three of the 62 patients included in the study had died, 12 patients were not available for follow-up and six patients were immobile due to residual impairments as a result of

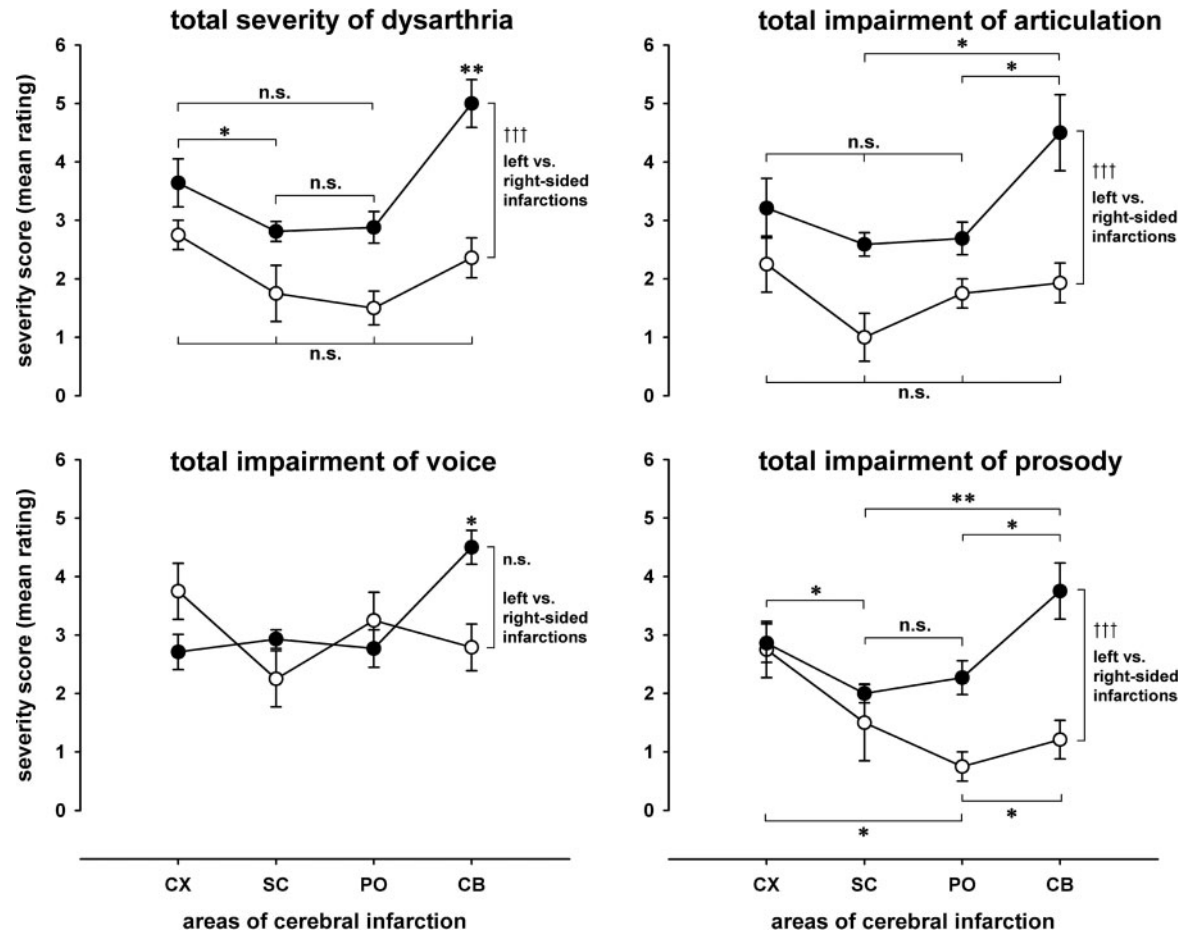


Fig. 2 Total impairment of dysarthria, articulation, voice, and prosody over different localizations of cerebral infarctions. Severity scores (0–6) are separately shown for left-sided (black circles) and right-sided (open circles) lesions (CX: motor cortex involvement, SC: striatocapsular infarctions, PO: pontine infarctions, CB: cerebellar infarctions). Stars denote the level of significance for differences across cortical areas (* $P < 0.05$; ** $P < 0.01$; ANOVA; LSD *post hoc*-test), while crosses denote the level of significance right versus left within areas collapsed for each side (††† $P < 0.001$; ANOVA).

the stroke, five patients had suffered recurrence of stroke. Thus, 38 patients (61.3%) were available for follow-up. All patients had received speech therapy over the first 2–4 weeks after the onset of stroke. Seven patients complained of residual speech difficulties, in the others, subjective speech had returned to normal function between 1 week and 12 months (median: 3 weeks) after stroke. The speech evaluation showed normal speech in 15 out of 38 patients (39.5%) in their subjective judgement. In the other 23 patients overall severity of dysarthria was mild (1.6 ± 1.1). Analogous to the subjective impression, the formal speech evaluation showed a significant improvement ($P < 0.001$) for 24 out of 31 speech characteristics. The items ‘pitch too high’, ‘loudness increased’, ‘uncontrolled changes of loudness’, ‘hypernasality’, ‘hyponasality’, ‘speech tempo increased’, and ‘speech intonation increased’ did not differ significantly.

Discussion

The results of this study provide new insights into the basic demographics and general neurologic and speech

characteristics of patients who become dysarthric as a result of unilateral ischaemic stroke. An association between dysarthria and lesions of the language-dominant hemisphere has been postulated already by Weisenburg and McBride (1935) without presenting detailed lesion studies. However, this matter has not been further investigated applying adequate selection criteria for patients and adequate methods of data analysis. In the present study we clearly demonstrated that dysarthria due to ischaemic stroke is not only more frequently caused by left-sided extracerebellar lesions, but also that severity of dysarthria is more pronounced in left-sided infarctions, independent from lesion topography. Because the left-sided dominance was demonstrated at different lesion levels (cortex, striatocapsular region and brainstem), we conclude on an asymmetry of descending cortico-bulbar projections relevant for speech articulation.

At present, it is not possible to determine as to whether these findings apply to individuals who also have aphasia, which limits verbal output, or apraxia of speech, due to the fact that patients with these disorders were not included in this study.

Table 3 ANOVA for the comparison of speech parameters in dysarthria over different areas and sides of cerebral ischaemia.

Speech parameter	Side		Area		Side × area	
	F-value	P-value	F-value	P-value	F-value	P-value
Global scores						
Total severity of dysarthria	19.665	<0.001	4.373	0.006	1.251	n.s.
Total severity of articulation	14.947	<0.001	2.490	n.s.	0.904	n.s.
Total severity of voice	0.371	n.s.	1.537	n.s.	2.787	0.044
Total severity of prosody	11.518	0.001	3.102	0.029	2.417	n.s.
Articulation						
Vowels imprecise and/or distorted	23.055	<0.001	6.602	<0.001	3.766	0.013
Repetition of phonemes and syllables	16.874	<0.001	3.582	0.016	1.691	n.s.
Consonant articulation imprecise	13.778	<0.001	3.687	0.014	1.809	n.s.
Articulatory inaccuracy	13.540	<0.001	3.356	0.021	1.188	n.s.
Reduction or elision of phonemes and syllables	8.461	0.004	3.300	0.023	1.111	n.s.
Hypernasality	2.761	n.s.	0.061	n.s.	1.228	n.s.
Hyponasality	0.844	n.s.	2.751	0.046	6.421	<0.001
Articulatory precision variable/irregular articulatory breakdown	0.545	n.s.	2.533	n.s.	1.217	n.s.
Voice						
Voice irregularity, pitch breaks	0.190	n.s.	1.022	n.s.	1.583	n.s.
Voice quality 'harsh'	0.340	n.s.	1.343	n.s.	0.617	n.s.
Voice quality 'breathy'	0.018	n.s.	0.625	n.s.	0.160	n.s.
Voice quality 'pressed'	0.905	n.s.	1.504	n.s.	2.516	n.s.
Voice tremor	0.610	n.s.	2.199	n.s.	1.669	n.s.
Variable voice quality	0.048	n.s.	1.136	n.s.	1.181	n.s.
Pitch too low	0.156	n.s.	1.142	n.s.	2.084	n.s.
Pitch too high	0.616	n.s.	0.688	n.s.	0.613	n.s.
Uncontrolled changes of pitch	0.414	n.s.	2.121	n.s.	0.761	n.s.
Uncontrolled changes of loudness	0.125	n.s.	1.313	n.s.	2.387	n.s.
Loudness decreased	1.664	n.s.	2.181	n.s.	4.878	0.003
Prosody						
Speech tempo slowed	8.409	0.005	3.760	0.013	2.385	n.s.
Speaking rate variable over time	6.466	0.012	3.385	0.021	1.723	n.s.
Prolonged phonemes or syllables	5.795	0.018	5.675	0.001	1.416	n.s.
Speech intonation reduced	4.510	0.036	1.822	n.s.	2.493	n.s.
Speech pauses	2.897	n.s.	1.527	n.s.	0.942	n.s.

The two-way analysis of variance (F- and P-values) shows highly significant differences comparing side and area of cerebral ischaemia ($n = 62$), while interactions (side × area) are almost absent. Differences over side or area are present for global scores, articulation, and prosody, but not for voice parameters. For detailed description of the speech parameters see also Table 2. n.s. = not significant.

In previous studies, also the term 'aphemia' coined by Broca (1865) was used to describe patients with initial mutism evolving to impaired articulation showing a speech which is effortful, slow and dysprosodic but preserved comprehension of spoken language, written language function and bucco-facial coordination. The pathology of 'aphemia' was located in or immediately inferior to Broca's area in the dominant hemisphere (Bastian, 1887; Alexander *et al.*, 1989). However, other authors deny the existence of 'aphemia' as a separate speech disorder and consider it as apraxia of speech (Mohr *et al.*, 1978; Schiff *et al.*, 1983; Ziegler, 2003). Apraxia of speech is characterized by inconsistent articulatory errors approximating the target word, articulatory groping and attempts of self-correction, and associated disruption in prosody and rate of speech (Wertz *et al.*, 1991). This concept is further

supported by recent findings that ischaemic lesions of Broca's area (left posterior inferior frontal gyrus) are also responsible for apraxia of speech (Hillis *et al.*, 2004). Using the criteria of Wertz *et al.* (1991) apraxia of speech can be differentiated from dysarthria, although both speech disorders may be present in the same patient (Dronkers, 1996). Regarding the ancient literature (Bastian, 1887) it is not clear if 'aphemia' was apraxia of speech or severe dysarthria or both since relevant information on some language features was not provided.

Ischaemic speech abnormalities without other neurological findings, however, are most frequently due to isolated dysarthria (Fisher, 1982; Kim, 1994; Urban *et al.*, 1999; Kim *et al.*, 2003), while apraxia of speech is nearly always associated with other neurological abnormalities such as Broca's aphasia (52%), limb apraxia (52%), bucco-facial apraxia (48%),

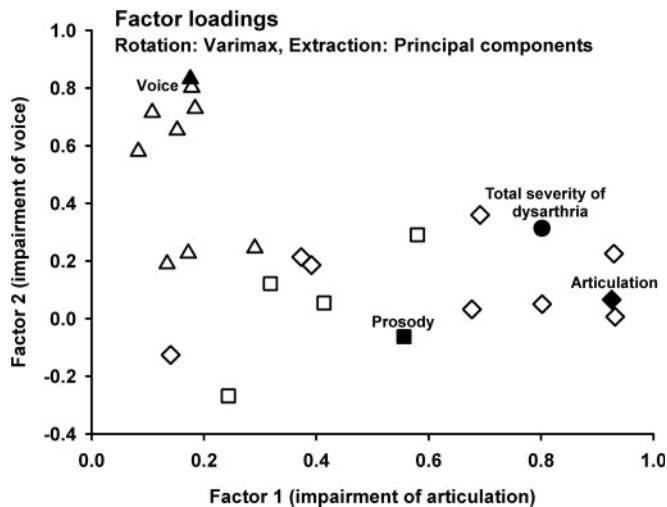


Fig. 3 Factor analysis of speech characteristics in dysarthria. Total severity scores for dysarthria (filled circle), articulation (filled diamond), voice (filled triangle), and prosody (filled square) are accompanied by the referring impaired speech characteristics (articulation parameters: open diamonds, voice parameters: open triangles, prosody parameters: open squares). The total severity score of articulation and referring speech parameters show significant loadings on factor 1 with almost no loading on factor 2. In opposite the total severity score of voice and referring speech parameters show significant loadings on factor 2 with almost no loading on factor 1, indicating that factor 1 represents impairment of articulation, and factor 2 representing the impairment of voice. The total impairment of dysarthria presents with a significant loading on factor 1 but not factor 2, indicating that dysarthria after ischaemic stroke is more influenced by the impairment of articulation than voice. The total severity score of prosody takes an intermediate position without any significant loading on factor 1 or 2.

dysarthria (40%), anomia (28%) and global aphasia (12%) (Dronkers, 1996).

In our study we carefully excluded patients with apraxia of speech and patients presenting with initial mutism so that we are sure that only patients with dysarthria have been included.

Lesion topography

The location of lesions associated with dysarthria ranged from the cortex to the brainstem. The majority of infarctions were located in the supratentorial region (61.3%), followed by the brainstem (24.2%) and cerebellum (14.5%). As reported previously, all extracerebellar infarctions causing dysarthria were located along the course of the pyramidal tract through the brain which correlates with the frequent association of pyramidal tract signs and the high incidence of cortico-bulbar tract lesions demonstrated by transcranial magnetic stimulation (Urban *et al.*, 1996, 1997, 1999, 2001). Thus, we can assume the presence of a lesion with an extracerebellar location along the course of the pyramidal tract in a patient with dysarthria and associated pyramidal tract signs. However, in the absence of other clinical signs indicating the lesion level, it may be located between the lower part of the primary motor cortex and the pontomedullary junction.

Although dysarthria has been reported in about 50% of patients with dysphagia following stroke (Mann *et al.*, 1994), dysphagia was present in only 6.5% of our dysarthric patients. The low coincidence of dysarthria and dysphagia in the present series might have several reasons. In our study swallowing function was only clinically assessed (asking for swallowing difficulties and observation of food intake) and fibre-endoscopy or videofluoroscopy have been performed in single patients only, thus probably underestimating the incidence of dysphagia. Although speech and swallowing tasks are performed in part by the same muscles (Larson, 1985), it is possible that impaired central motor control primarily affects speech function which probably requires a higher degree of coordination of different muscles than swallowing. In the present study only patients with single lesions were included, while other studies also considered patients with multiple infarctions (Mann *et al.*, 1999) which increases the risk of dysphagia (Daniels and Foundas, 1999). Furthermore, some authors found that dysphagia seems to be clinically more significant in patients with right-hemispheric stroke (Daniels *et al.*, 1996), while in our patients left-sided lesions predominated.

The cerebellar region involved in dysarthria has not yet been definitely identified. Holmes (1917) observed dysarthria following gunshot wounds of the cerebellar vermis and the hemispheres. Lechtenberg and Gilman (1978) reported a significantly higher incidence of dysarthria with left-sided paravermal lesions in 122 patients with cerebellar tumours. The retrospective study design and the possibility of perifocal oedema with possible brainstem involvement may, however, have biased the result of their investigation. A paravermal location of the lesions has also been reported in two patients with isolated dysarthria due to superior cerebellar artery (SCA) infarction (Amarenco *et al.*, 1991; Gironell *et al.*, 1997). Out of 12 patients with cerebellar infarctions, only the patients with SCA involvement ($n = 4$) exhibited dysarthria, while no patient with involvement of the posterior inferior cerebellar artery (PICA) ($n = 8$) showed the disorder (Ackermann *et al.*, 1992). Similarly, isolated cerebellar infarctions (10.9%) were always located within the SCA-territory in our series. Dysarthria was also reported in individual patients with infarctions in the anterior inferior cerebellar artery (AICA)- and PICA-territory. These studies report four of 13 patients with AICA-infarction, who showed brainstem involvement at autopsy (Amarenco and Hauw, 1990). In PICA-infarctions, dysarthria was reported in 0% (Amarenco *et al.*, 1990; Ackermann *et al.*, 1992), 10% (Amarenco *et al.*, 1990), 20% (Barth *et al.*, 1994), and 39% (Sacco *et al.*, 1993) of patients. These differences are most likely due to the retrospective study design, different frequencies of brainstem involvement, and the absence of a standardized evaluation of dysarthria.

The present analysis of the lesion side shows that 88.7% of all extracerebellar infarctions leading to dysarthria were located in the left, and 11.3% in the right hemisphere. Furthermore, the ANOVA clearly demonstrated that the severity of

dysarthria was more pronounced in left-sided infarctions, independent from lesion topography. These findings indicate a strong left hemisphere lateralization of speech motor control and confirm the early assumption of Geschwind (1969) who postulated a left hemisphere dominance of speech production beyond cognitive aspects of language processing. A recently published positron emission tomography study ($H_2^{15}O$ PET) demonstrated a strong hemispheric lateralization after contrasting voiced and unvoiced speech in health subjects supporting our findings in dysarthric patients (Shulz *et al.* 2005). In several small series of patients with dysarthria due to cortical infarction (Kim *et al.*, 2003: left: five patients, right: one patient), striatocapsular infarction (Ozaki *et al.* 1986: left: four patients, right: one patient) and pontine infarction (Toghi *et al.*, 1996: left: 21 patients, right: 11 patients) a predominance of left-sided infarctions, irrespective of the lesion site was observed. However, in these studies the speech characteristics were not described. In view of the fact that left-side dominance in extracerebellar infarctions applies to both supratentorial (89.5% versus 10.5%) and brainstem lesions (86.7% versus 13.3%), the side difference might be explained by a lesion of a common descending projection, e.g. a cortico-bulbar projection, to the articulatory muscles (Urban *et al.*, 1996, 1997, 1999). Since it has previously been shown, that a lesion of the cortico-lingual pathway is crucial in the pathogenesis of dysarthria in stroke, a possible explanation might be a more dominant descending pathway from the left motor cortex. In addition, dysarthria is more frequently associated with cerebellar infarctions on the right side (in our series seven as compared to two patients). In the series of Ackermann *et al.* (1992) the infarctions were also located on the right side in three patients, and bilaterally in one. The patients of Amarenco *et al.* (1991) and Gironell *et al.* (1997) had lesions equally distributed on the right and on the left side. Both right- and left-sided SCA-infarctions may therefore be assumed to lead to dysarthria. A right-side dominance of the cerebellum, however, has thus far not been proven (Urban *et al.*, 2003).

Correlation with lacunar infarctions and lacunar syndromes

Dysarthria as a part of a ‘classic lacunar syndrome’ (Fisher, 1982, 1991) was found in 53.1% of our patients, while a ‘classic lacunar syndrome’ is considerably less frequent in unselected series [Ege Stroke Registry: 13.0% (Kumral *et al.*, 1998), Besancon Stroke Registry: 19.2% (Moulin *et al.*, 1997)]. ‘Isolated dysarthria’ (Fisher, 1982) occurred in 1.6% of patients. In all other patients dysarthria was associated with additional symptoms and signs. Dysarthria was most frequently associated with ‘pure motor hemiparesis’ (Fisher and Curry, 1965) and/or ‘ataxic hemiparesis’ (Fisher and Cole, 1965). In ataxic hemiparesis, ataxia may be expected to be more pronounced in the limb least affected by the motor deficit (Fisher and Cole, 1965). However, hemiparesis per se may lead to uncoordinated movement, and the two

syndromes may sometimes be difficult to differentiate clinically (Landau, 1989; Gorman *et al.*, 1998). According to the criteria of Fisher and Curry (1965) and Melo *et al.* (1992), pure motor hemiparesis was only assumed when face, arm and leg were involved (proportional hemiparesis) in the absence of sensory deficit, visual field defect, aphasia, agnosia and apraxia. The ‘dysarthria-clumsy hand syndrome’ as originally described by Fisher (1967), included patients with dysarthria, clumsiness, awkwardness, and slowness of fine manipulations of the affected hand. However, central facial paresis and/or central lingual paresis may also be part of the syndrome (Fisher, 1967). In our study, seven patients showed dysarthria and additional central facial paresis, and two further patients had additional central lingual paresis.

Speech characteristics

Although the focus of the present study was not on inter-rater reliability, we found a good agreement between both listeners for most items which corresponds to previous studies (Darley *et al.*, 1969). The lowest agreement scores were observed for the speech characteristics ‘voice quality’, ‘low pitch’ and ‘speech pauses’, which were also found in previous studies (Sheard *et al.*, 1991; Kreiman and Gerratt, 1998). This finding lends support to the hypothesis that these items may still be poorly defined (Kreiman *et al.*, 1994). The relatively low interrater reliability of voice items may also be due to the large variability of voice characteristics already in healthy subjects and the unknown premorbid voice status (Ziegler *et al.*, 1990).

One of the main findings of the present study is that the articulatory abnormalities were the predominant deviant characteristics influencing total severity of dysarthria as revealed by factor analysis. Articulatory inaccuracy and imprecise articulation were the most frequent articulatory abnormalities noted (in 95.2%) and consonant articulation was more frequently and more severely affected than the vowel articulation. From a clinical point of view, so called ‘tongue twisters’, test sentences with a lot of consonants are still useful in revealing mild dysarthria, at least of vascular origin. The predominant disturbance of consonant articulation has previously also been described by Thompson and Murdoch (1995), who analysed the speech characteristics in 20 patients with cerebrovascular accidents. It is likely that the facial and tongue weakness observed in a large number of the patients with extracerebellar lesions exerted an effect on their imprecise articulation. In addition, face and tongue weakness undetected in the clinical examination, but revealed by transcranial magnetic stimulation of the tongue associated motor cortex (Urban *et al.*, 1999), may have played a similar role in a number of the remaining patients, who exhibited articulatory imprecision.

Several patients with supratentorial lesions present with signs like ‘irregular articulatory breakdowns’, ‘harsh voice’, ‘imprecise and/or distorted vowels’ which have previously been associated with cerebellar lesions (Darley *et al.*, 1969; Ackermann *et al.*, 1992). In our patients, we found no

significant differences between cerebellar and extracerebellar infarctions for the items ‘irregular articulatory breakdowns’ and ‘harsh voice’. This observation might be due to several reasons. In other studies, frequent irregular articulatory breakdowns and harshness of voice were observed in patients with upper motoneuron lesions due to unilateral cerebral ischaemia (Thompson and Murdoch, 1995; Duffy and Folger, 1996). It is possible that these ‘ataxic-like’ characteristics in upper motoneuron lesions might be an artefact of the unilateral weakness in the face and tongue of many patients, rather than a symptom of ataxia. It may further be possible that unilateral weakness leads to a degree of ‘clumsiness’ in articulation, particularly when it is asymmetric in bilaterally innervated structures, and when its effects vary in severity from one set of speech muscles to another. Another possibility is that the ataxic-like features reflect abnormalities in cerebellar influences on speech. Cerebellar-cortical and cortico-cerebellar pathways pass through a number of the structures that were frequently involved in the patients of our study (e.g. corona radiata, internal capsule, base of the pons). Although not universally accepted (Landau, 1989), interference in such pathways is a common explanation for the apparent ataxia frequently observed in the limbs in lacunar stroke syndromes such as ‘ataxic hemiparesis’ or ‘dysarthria-clumsy hand’ (Fisher, 1967; Arboix *et al.*, 1990).

In our study, several other articulatory and prosodic items were significantly more pronounced in cerebellar lesions compared to extracerebellar lesions including ‘consonant articulation’, ‘speaking rate slowed over time’, ‘prolonged phonemes and syllables’, ‘articulatory inaccuracy’, ‘reduction or elision of phonemes and syllables’, ‘repetition of phonemes and syllables’, and ‘vowels imprecise and/or distorted’. This finding underlines the relevance of the cerebellum for speech performance and its influence on articulatory and prosodic features. However, due to the relatively small number of patients with dysarthria due to cerebellar infarction, this finding awaits further confirmation.

The frequent perception of voice abnormalities has also been observed by previous studies in patients with upper motoneuron lesions due to unilateral cerebral ischaemia (Thompson and Murdoch, 1995; Duffy and Folger, 1996). Voice irregularities, pitch breaks, and a harsh, breathy, and pressed voice quality were frequent findings that represent laryngeal dysfunction. This finding is considered surprising in view of the fact that the laryngeal muscles have a bilateral upper motoneuron supply to confer sufficient redundancy to protect against dysphonia. However, the frequent phonatory perception abnormalities suggest that a unilateral upper motoneuron lesion may affect laryngeal function to a degree where dysphonia may result, possibly secondary to a central impairment of vocal cord function. This assumption is supported by the observation that both central motor dysfunctions of the larynx were frequently seen following brain injury and stroke in a large number of investigated subjects (Morasch *et al.*, 1987; Venketasubramanian *et al.*, 1999) and that intrinsic laryngeal muscle responses to transcranial

magnetic stimulation of the primary motor cortex are characterized by bilateral innervation with left hemispheric dominance (Ludlow *et al.*, 1989; Sims *et al.*, 1996) or without hemispheric dominance (Rödel *et al.*, 2004). Rasmussen and Penfield (1947) described vocalization by direct cortical stimulation throughout the lower face area of both the dominant and non-dominant hemisphere. Thus, interhemispheric differences in the cortical laryngeal representation are possible, although the phonatory abnormalities noted in the present study were not associated with significant differences between both sides. Out of all 31 items assessed, only loudness was more significantly decreased in left-sided cerebellar lesions.

The production of intelligible speech requires a controlled, sustained, and smooth flow of sufficient air supply. The decreased loudness noted in some patients might therefore be due to a disruption of the respiratory supply, lack of respiratory muscle control, or a combination of both. This view is supported by the facts, that respiratory abnormalities can be detected following unilateral stroke (Przedborski *et al.*, 1988), and that transcranial magnetic stimulation demonstrated frequent involvement of the central motor connections to respiratory muscles in hemiplegia (Urban *et al.*, 2002).

The perceptual analysis revealed a number of prosodic disturbances which were most prominent in speech pauses, reduced speech intonation, variability of speaking rate over time, and slowed speech tempo. Although dysarthria is a pure central motor disorder, impairment of the articulatory, velopharyngeal, laryngeal and respiratory subsystems may result in the perception of impaired prosody (Thompson and Murdoch, 1995). Impaired articulation is mainly responsible for a slow rate of speech, as shown by factor analysis. The item ‘slow rate of speech’ showed much higher loadings on factor 1 ‘impairment of articulation’ (0.64) than on factor 2 ‘impairment of voice’ (0.27). On the basis of the factorial structure the item ‘slow rate of speech’ was correlated with the total severity of dysarthria ($r = 0.6$), followed by ‘articulatory inaccuracy’ ($r = 0.5$), and ‘total impairment of articulation’ ($r = 0.45$). In contrast, the ‘slow rate of speech’ was only weakly correlated with the total impairment of voice ($r = 0.37$). These data indicate that altered laryngeal functions have only a minor importance for speech rate, probably as a result of reduced expiratory volume, or impaired maintenance of expiratory airflow necessary for speech production. Some prosodic items (‘speech tempo slowed’, ‘speaking rate variable over time’, ‘prolonged phonemes and syllables’ and ‘speech intonation reduced’) were more pronounced in left-sided lesions. A significant difference was demonstrated in pontine and cerebellar lesions but the difference was not significant in hemispheric lesions. These findings let us assume that a lack of speech muscle coordination due to a lesion of the cerebellum or pontocerebellar fibres in ventral pons infarctions may result in prosodic disturbances.

Follow-up examinations

A comparison of the scores from the speech therapists showed that deviant speech characteristics were generally

perceived as mild, or mild-to-moderate. This is consistent with clinical observations that dysarthria due to unilateral upper motoneuron lesions is usually mild, and that recovery is generally substantial when it results from stroke. However, this has never been substantiated by the findings of a clinical study. Our results of the follow-up examination confirm this hypothesis, showing a complete recovery in 39.5% of patients within 10 months (mean follow-up), and a significant improvement in the remaining patients.

In conclusion, the conversational speech of patients with dysarthria due to unilateral cerebral ischaemia is frequently characterized by articulatory imprecision, in particular regarding consonants, irregular articulatory breakdowns, reduced speech intonation leading to the impression of a monotonic voice, and harsh dysphonia with reduced loudness. The overall severity is usually in the mild-to-moderate range, but rarely severe. The recovery is often nearly complete, although mild dysarthria may persist.

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