Original Paper

European Neurology

Eur Neurol 2015;74:171–177
DOI: 10.1159/000441056

Received: July 27, 2015
Accepted: September 13, 2015
Published online: October 23, 2015

Neuroanatomical, Clinical and Cognitive Correlates of Post-Stroke Dysphagia

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Key Words
Post-stroke dysphagia · NIHSS · Leukoaraiosis · Cognitive impairment

Abstract
Background and Purpose: About half of the dysphagic stroke patients have persistent swallowing dysfunction after 7 days from symptom onset. The aim of the study was to evaluate incidence, prognosis, clinical and neuroradiological correlates of post-stroke dysphagia. ***Methods:*** We prospectively examined consecutive patients with acute ischemic or hemorrhagic stroke. Patients' clinical and neuroradiological data were collected. Swallowing function was assessed by the water swallow test upon admission and after 14 days; patients were then classified as persistent dysphagic, non-persistent dysphagic or non-dysphagic. ***Results:*** We recruited 275 patients, 121 of whom were dysphagic upon admission and 254 patients attended follow-up at 14 days; 141 never presented dysphagia, 21 had a non-persistent pattern of dysphagia and 92 had a persistent one. Stroke type, leukoaraiosis degree, previous cognitive impairment and stroke severity upon admission independently predicted the occurrence of dysphagia after stroke and its persistence as well. At receiver operating characteristic (ROC) analysis, the National Institutes of Health Stroke Scale (NIHSS) score of 11.5 was the best predictive value of persistent dysphagia, with a specificity of 90.1% and a sensitivity of 72.4%. ***Conclusion:*** Stroke severity is an important predictor of a persistent pattern of dysphagia, with a suggested NIHSS cutoff value of ≥12. An independent correlation was observed with leukoaraiosis and with previous cognitive impairment.

Introduction

Dysphagia is a common consequence after stroke, presenting in up to 50% [1] of all acute stroke patients. It has been reported that the incidence of dysphagia ranges from 25 to 81% [2] depending on the timing of the assessment, diagnostic methods and criteria [1].

The clinical and scientific relevance of post-stroke dysphagia is demonstrated by its high incidence and negative effect on clinical outcome, with increased morbidity and mortality mainly because it dramatically increases the risk of aspiration and pneumonia [1, 3–6]. Nevertheless, there is also strong evidence that dysphagia significantly contributes to malnutrition and dehydration regardless of aspiration [7]. Moreover, dysphagia has been associ-

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ated with increased likelihood of residential placement and significantly increases estimated lifetime costs in stroke survivors [8]. About half of the stroke patients remain dysphagic after 7 days from symptom onset, and approximately 13–15% [9] of patients has persistent swallowing dysfunction after 6 months.

Formal dysphagia screening protocols significantly reduce the rate of pneumonia and improve the general outcome after stroke [10]. Furthermore, early behavioral swallowing interventions are associated with a more favorable outcome in dysphagic stroke patients [11]. Therefore, early detection and timely treatment of dysphagia play an important role in acute stroke management.

In this view, many efforts have been made in order to identify those patients who are at high risk for developing dysphagia after stroke. For example, it has been suggested that disruption of cortical–cortical and cortical–subcortical white matter connections may decrease the threshold of input to the medullary swallowing center. Notwithstanding, clinical and neuroradiological correlates of post-stroke dysphagia still remain unclear. Even fewer data are available on predictors of persistent dysphagia (PD). Therefore, the main purpose of the study was to investigate a sample of consecutive patients with ischemic and hemorrhagic stroke to evaluate incidence, prognosis, clinical and neuroradiological correlates of post-stroke dysphagia. The role of leukoaraiosis in the pathogenesis, persistence and prognosis of post-stroke dysphagia is also discussed.

Materials and Methods

Subjects

We prospectively examined consecutive patients with acute ischemic or hemorrhagic stroke, detected by MRI. Patients were recruited, within 72 h of symptom onset, from the stroke units of the Policlinico Umberto I, Rome, and 'L. Sacco' Hospital, Milan. We excluded patients younger than 18 years, those with TIA, subarachnoid hemorrhage, cerebral sinus venous thrombosis and those who were unable to undergo brain MRI. Moreover, patients with history of previous swallowing impairment or medical condition that may affect swallowing were excluded. For the purposes of this study, those patients who were unconscious or too drowsy (i.e. with a Glasgow Come Scale score <13) [12] were excluded as well. Written informed consent was obtained from all patients; the study was approved by the local ethics committee.

Clinical Data Collection

Upon admission, the following clinical data were collected from all participants: basic demographic details, main cardiovascular risk factors, history of cerebrovascular events and ischemic heart diseases. Stroke severity was assessed by the National Institutes of Health Stroke Scale (NIHSS) [13]; previous cognitive impairment was evaluated with the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE) [14], which is a questionnaire completed by an informant, designed to assess change in functional performance secondary to cognitive change; it is used as a tool to identify those who may have dementia.

Bedside Assessment of Swallowing Function

Patients' swallowing function was assessed using a standardized bedside swallowing test, the water swallow test (WST). Patients were asked to drink 10 ml of water, and immediate coughing or delayed swallowing was monitored in the next 2 min. If coughing or choking occurred at this stage or the time taken was longer than 20 seconds, the test was considered failed. Otherwise, the procedure was repeated with 50 ml of water. Patients were defined as dysphagic if they failed the WST. Patients were revaluated every 24 h. We also defined as dysphagic those patients who were still insufficiently alert to perform the test 48 h after admission. Data collection and clinical examination were performed by a neurologist blinded to the swallowing evaluation, being the latter carried out by a speech pathologists blinded to the neurologist' results.

MRI Acquisition and Analysis

All patients underwent the same MRI protocol within 7 days from stroke onset. Both centers are equipped with a 1.5-T Philips Intera MR system. Apart from local specificities for the MR acquisition protocol, all examinations comprised a standard set of images obtained in the axial plane, parallel to the subcallosal line, using T2-FLAIR, T1 spin-echo, TSE double-echo T2 and diffusion-weighted imaging sequences. The slab covered the entire brain and the brain stem, from the cervicomedullary junction through the cranial vault. All examinations were evaluated without prior notice of any clinical parameters in collaboration with the neuroradiological section of our department.

The following neuroradiological parameters were collected: type of stroke (ischemic or hemorrhagic), size, number and site of acute lesions. Leukoaraiosis was evaluated by the Fazekas Scale [15], which is a rating scale designed to grade the severity of white matter changes, by visually evaluating both the periventricular and the deep white matter lesions, as detected by MRI.

Follow-Up

The aforementioned bedside assessment of swallowing was then repeated after 14 days (T1). According to the swallowing function, patients were then classified as the following: (a) persistent dysphagic, (b) non-persistent dysphagic or (c) non-dysphagic. Stroke severity was reassessed by means of NIHSS. All the complications occurring during this period (e.g. pneumonia and urinary infections) were recorded as well.

Statistical Analysis

Clinical, neuro-anatomical, demographic, anamnestic and neurological characteristics were compared between patients with and without dysphagia both at the baseline and after 14 days by using the chi-square, the Fisher exact or the Wilcoxon–Mann–Whitney test, as appropriate. A multivariable logistic model was applied to identify independent risk factors of dysphagia (T0 time point) and PD (T1 time point). Results are given as OR and 95% CI. The Spearman's rank correlation was used to measure the statistical dependence between the variables applied in this model (results are given as ρ and p value). The multivariable logistic model also al-

lowed us to represent receiver operating characteristic (ROC) curves and evaluate the predictivity of each significant variable using the area under the curve (AUC) value. An AUC of 0.5 means chance predictivity, and AUC of 1 means perfect predictivity.

The 3 different pattern of dysphagia (i.e. transient, persistent or absent) were longitudinally compared among the NIHSS score using the analysis of variance for repeated measures (ANOVARM) model. Post hoc analysis was delivered by means of Tukey HSD test.

All tests were 2-tailed with significance set to alpha = 0.05. Data were analyzed with SPSS software for Windows (version 18.0) and kept blind to the allocation.

Results

Admission

We consecutively recruited 275 patients, 138 (50.2%) men and 137 (49.8%) women. The mean age of the sample was 73 (SD 11.6) years and ranged from 36 to 95 years.

On admission, 121 (44%) patients had dysphagia. Among dysphagic patients, 81 (66.9%) were fed enterally and 40 (33%) were fed orally with limits of consistency of the food.

Among the demographic and clinical variables (online suppl. table 1; for all online suppl. material, see www.karger.com/doi/10.1159/000441056), univariate analysis showed that only older age was associated with dysphagia. Mean age was 75.4 (SD 11.4) years in dysphagic patients and 71.3 (SD 11.6) years in non-dysphagic ones ($p = 0.0023$).

Data from univariate analysis also showed that, regarding neurological and neuroradiological characteristics (online suppl. table 2), patients with dysphagia had more severe stroke. The mean NIHSS score was significantly higher in dysphagic patients compared to non-dysphagic ones (15.3 ± 8.3 vs. 5.5 ± 4.1, $p < 0.0001$). Dysphagic patients had also a higher previous cognitive impairment, as assessed by IQCODE score (84.0 ± 11.1 vs. 81.3 ± 7.6, $p < 0.0033$).

Moreover, dysphagia was more frequent in patients with hemorrhagic lesions ($p = 0.0002$), single lesions ($p = 0.0066$), larger lesions ($p < 0.0001$), lesion involving frontal lobe ($p = 0.0128$), basal ganglia ($p = 0.004$), insula ($p = 0.0002$) and temporal lobe ($p < 0.0001$); furthermore, patients with dysphagia had a significant higher Fazekas score ($p = 0.0030$).

Multiple logistic regression analysis (table 1) showed that stroke type (OR 2.5 (1.3–4.8)), leukoaraiosis degree (OR 1.3 (1.1–1.5)), IQCODE (OR 1.05 (1.01–1.09)) and NIHSS score (OR 1.34 (1.25–1.44)) independently predict the occurrence of dysphagia after stroke.

Table 1. Multiple logistic regression analysis: independent risk factors for dysphagia (T0) (n = 275)

	OR (95% CI)
Hemorrhagic lesions	2.5 (1.3–4.8)
Fazekas Scale	1.3 (1.1–1.5)
IQCODE	1.05 (1.01–1.09)
NIHSS (T0)	1.34 (1.25–1.44)

Follow-Up

Among the 275 patients recruited, 254 patients attended follow-up at 14 days (10 dropped out and 11 died). Out of 254 patients, 141 (55.5%) never presented dysphagia, 21 (8.3%) had a transient dysphagia (TD; <14 days) and 92 (36.2%) a PD.

We analyzed demographic data, medical history, clinical and neuro-anatomical characteristics according to the different patterns of dysphagia (i.e. transient, persistent or absent). Univariate analysis showed that the following data were significantly correlated with the pattern of dysphagia (detailed data are shown in table 2): age ($p < 0.0001$), education ($p = 0.0239$), mean IQCODE ($p = 0.0119$), coronary artery disease ($p = 0.0388$), mean NIHSS score at baseline (i.e. T0 NIHSS score; $p < 0.0001$), pneumonia ($p < 0.0001$) and urinary infections ($p < 0.0001$). Among neuroradiological features, hemorrhagic strokes ($p = 0.0032$), single lesions ($p = 0.007$), larger lesions ($p < 0.0001$), Fazekas score ($p = 0.0076$) and lesions involving the frontal lobe ($p = 0.0089$), the basal ganglia ($p = 0.0182$), the insula ($p < 0.0001$) and the temporal lobe ($p = 0.0006$) were found to be significantly related to the pattern of dysphagia.

Multiple logistic regression analysis revealed that T0 NIHSS score (OR 1.5 (1.34–1.69)), Fazekas score (OR 1.63 (1.25–2.12)), IQCODE (OR 1.06 (1.01–1.1)) and hemorrhagic stroke (OR 3.03 (1.17–7.85)) are independent predictors of a persistent pattern of dysphagia at 14 days (table 3).

The Spearman's rank correlation between variables (online suppl. fig. 1) revealed a statistical dependence between T0 NIHSS score and hemorrhagic stroke ($\rho = 0.169$, $p = 0.005$) and between leukoaraiosis and IQCODE ($\rho = 0.288$, $p < 0.001$). A weaker dependence was also found between hemorrhagic stroke and leukoaraiosis ($\rho = 0.142$, $p = 0.022$).

Data from the ANOVARM after Tukey HSD post hoc analysis show (see also fig. 1) the following:

(1) Patients with PD versus patients with TD:

Table 2. Univariate analysis: significant demographic data, medical history, clinical and neuro-anatomical characteristics according to the pattern of dysphagia at follow-up

	ND (n = 141)	TD (n = 21)	PD (n = 92)	p value
Age	70.90±11.51	68.66±12.38	76.77±11.05	<0.0001
Education, years				
0–4	12 (8.5)	1 (4.8)	10 (10.9)	0.0239
5–7	54 (38.3)	9 (42.9)	52 (56.5)	
8–12	52 (36.9)	6 (28.6)	14 (15.2)	
13+	23 (16.3)	5 (23.8)	16 (17.4)	
IQCODE	81.09±7.15	82.33±5.96	84.55±11.98	0.0119
Coronary artery disease	114 (80.9)	21 (100)	82 (89.1)	0.0388
NIHSS (T0)	4.98±3.50	9.86±6.09	15.77±7.79	<0.0001
Lesion type (hemorrhagic)	27 (19.1)	7 (33.3)	36 (39.1)	0.0032
Number of lesions (single lesion)	114 (80.9)	21 (100)	85 (92.4)	0.0070
Lesion size (>3 cm)	41 (29.1)	11 (52.4)	60 (65.2)	<0.0001
Frontal lobe	34 (24.1)	5 (23.8)	39 (42.4)	0.0089
Basal ganglia	14 (9.9)	2 (9.5)	21 (22.8)	0.0182
Insula	11 (7.8)	2 (9.5)	29 (31.5)	<0.0001
Temporal lobe	28 (19.9)	10 (47.6)	37 (40.2)	0.0006
Fazekas Scale, median value	1.0	3.0	2.0	0.0076
Pneumonia	3 (2.1)	2 (9.5)	36 (39.1)	<0.0001
Urinary infections	10 (7.1)	8 (38.1)	32 (34.8)	<0.0001

Values are mean ± SD or n (%).

Table 3. Multiple logistic regression analysis: PD vs. ND (T1) (n = 254)

	OR (95% CI)
Hemorrhagic lesions	3.03 (1.17–7.85)
Fazekas Scale	1.63 (1.25–2.12)
IQCODE	1.06 (1.01–1.1)
NIHSS (T0)	1.5 (1.34–1.69)

– PD showed a significantly higher T0 and T1 NIHSS score with respect to both T0 ($p < 0.001$ and $p = 0.023$, respectively) and to T1 ($p < 0.001$ and $p < 0.001$, respectively) NIHSS scores of TD.

(2) Patients with PD versus no dysphagic (ND) patients:

– PD showed a significantly higher T0 and T1 NIHSS score with respect to both T0 ($p < 0.001$ and $p < 0.001$, respectively) and to T1 ($p < 0.001$ and $p < 0.001$, respectively) NIHSS scores of ND.

(3) Patients with TD versus ND patients:

– TD showed a significantly higher T0 NIHSS score with respect to both T0 ($p = 0.002$) and to T1 ($p < 0.001$) NIHSS scores of ND, whereas T1 NIHSS of TD did not differ from both T0 ($p = 0.95$) and to T1 ($p = 0.38$) NIHSS scores of ND.

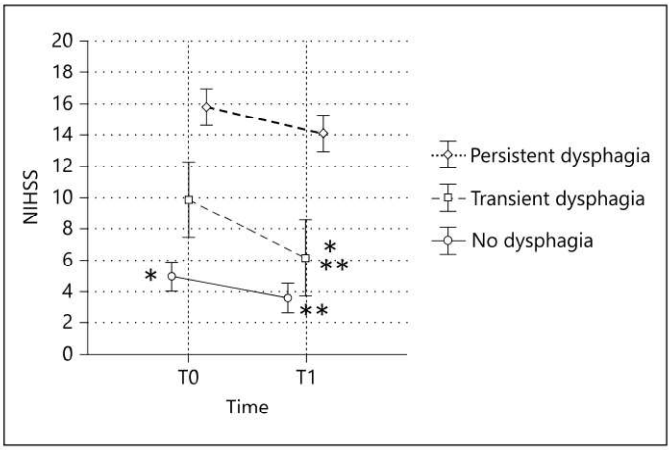

Fig. 1. ANOVARM Tukey post hoc analysis showed a significant difference between all groups (longitudinally compared among NIHSS) but: * $p = 0.95$, ** $p = 0.38$. See the text for the details.

Figure 2 shows the ROC curves and the respective AUC. T0 NIHSS score of 11.5 was found to be the best predictive value of PD, with a specificity of 90.1% and a sensitivity of 72.4%, with a reported AUC of 0.898.

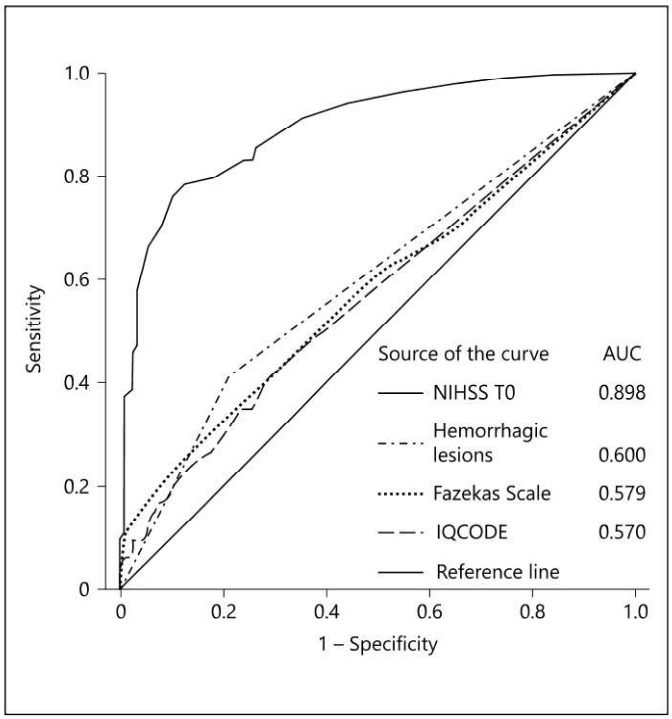

Fig. 2. ROC analysis: PD.

Discussion

Dysphagia is extremely common in the acute phase of stroke, with a reported incidence ranging from 25 to 81% [2], depending on the timing of the assessment, diagnostic methods and criteria. In the present study, dysphagia was observed in 44% patients at baseline.

Patients who presented dysphagia were significantly older than those without; thus, elderly patients with stroke may have more swallowing alterations due to the reduced cough reflex and alteration of swallowing/breathing coordination [16].

Dysphagia was influenced by the lesion size, but no differences were identified in the involvement of right or left hemisphere. In this view, the majority of studies performed on stroke patients did not observe any relation between the presence of dysphagia and side of hemispheric location [17–19]. However, it is known that lesions located in the right hemisphere may cause more pharyngeal changes and lesions in the left hemisphere cause more changes in the oral phase of swallowing [20].

The NIHSS score at admission was higher in dysphagic patients. Comparing the 3 different patterns of dysphagia over time regarding the stroke severity at admission, stroke severity was significantly higher in patients with PD with respect to those with TD or without dysphagia, both at admission and at T1. Moreover, those patients with a transient pattern of dysphagia showed a sensibly improved NIHSS score at T1, which became similar, on average, to that of patients without swallowing impairment upon admission.

In the multivariable logistic analysis, stroke severity at admission was found to be an independent predictor of a persistent pattern of post-stroke dysphagia. In this view, looking at ROC analysis, the T0 NIHSS score of 11.5 was found to be the best predictive value of PD; we also found an AUC of 0.898, which means that stroke severity at admission allowed the correct classification of about 90 of 100 dysphagic patients in a persistent or non-persistent pattern. Hence, a NIHSS score of ≥12 would be suggested as cutoff value in order to predict, upon admission, those patients who will probably remain dysphagic after 14 days follow-up.

In our sample, dysphagia was more frequent in patients with hemorrhagic stroke; there are little available data in the literature reporting a different risk factor for dysphagia depending on stroke type. Paciaroni et al. [18] found, in a univariate comparison, an increased frequency of dysphagia in hemorrhagic stroke patients compared to ischemic ones. They suppose that it could be related to stroke severity. Our results confirm this datum, since the Spearman's rank correlation revealed a strong statistical dependence between stroke severity at admission and hemorrhagic stroke. Looking at logistic regression analysis, the stroke type also appears to be an independent predictor for post-stroke dysphagia (OR 2.5); nonetheless, hemorrhagic patients present a more than 3-fold risk of PD. Hence, further research is needed in order to investigate a possible intrinsic mechanism linking dysphagia and hemorrhagic stroke.

Patients with high IQCODE score, which means a pre-stroke cognitive impairment, had more chances to present swallowing dysfunction; moreover, logistic regression analysis showed that the IQCODE score is an independent risk factor for post-stroke dysphagia and PD as well. These results are in agreement with previous reports that stated that brain lesions that cause decline in cognitive function, such as concentration and attention, may impair the control of swallowing [21].

Dysphagia was more frequent in patients with insular stroke, as well as in those with stroke involving frontal and temporal lobes and basal ganglia. As a matter of fact, all the aforementioned structures are involved in the neu-

ral control of swallowing; in particular, insula is known to be closely connected both with cortical and subcortical pathways involved in the neural control of swallowing. Nevertheless, none of those structures was found to be an independent risk factor for dysphagia. In the case of insular stroke, this seems mostly due to the small number of pure insular stroke.

Otherwise, concerning the other aforementioned structures that failed to reach statistical significance once logistic regression analysis has been performed, this datum could suggest that cortical–subcortical pathways involved in the swallowing function are probably more important in causing dysphagia after stroke than a single structure 'per se'.

This hypothesis could be corroborated by analyzing the contributions of white matter to swallowing behavior. Interestingly, we observed an independent correlation between post-stroke dysphagia and leukoaraiosis severity as well as a strong association between leukoaraiosis and previous cognitive impairment. Recent studies reported that subcortical white matter connections play an important role in the pathogenesis of dysphagia: disruption of cortical–cortical and cortical–subcortical white matter connections seems to increase the risk of dysphagia and aspiration pneumonia by lowering the threshold of input to the medullary swallowing center.

These data, as well as those showing the role of a decline in cognitive function, suggest that swallowing impairment after stroke might be not just a simple pure motor disturbance but also a more complex phenomenon. In this context, one might speculate that dysphagia may result from the combined effects of an acute focal damage and pre-existent destruction of the subcortical white matter connecting pathways. In other words, we might think of dysphagia as a kind of deficit in executive functions, which we know to be particularly associated with leukoaraiosis and cognitive disorders [22], and of course age [16].

Finally, pneumonia is among the most common medical complication after stroke, affecting up to one-third of patients and accounting for nearly 35% of post-stroke deaths [4, 23]. Our results suggest that stroke and pneumonia are associated with 2-fold: on the one hand, pneumonia appears to be strongly linked to the presence of dysphagia at admission. In fact, as shown in table 2, among those patients who developed pneumonia during the entire hospitalization, 38 (93%) patients were dysphagic at admission. On the other hand, the persistence of dysphagia after 14 days was closely linked with the occurrence of pneumonia ($p < 0.001$), with 36 patients (88%) with pneumonia who showed a persistent pattern of dysphagia.

We found a relatively high rate of pneumonia among PD patients (39.1%), in comparison to the whole sample of patients (14.9%); this may be due to the fact that some of the risk factors we found to be related to post-stroke dysphagia (i.e. older age, high NIHSS and cognitive impairment) are also known to be independent predictors of pneumonia in acute stroke patients [23].

The small percentage of our patients who died does not allow any firm conclusion on the correlations between pneumonia and mortality. Another limitation of this study is the use of WST for dysphagia detection, which may be therefore underestimated. Nevertheless, although the most important international guidelines stress the importance of screening of swallowing in patients with stroke, they do not recommend a specific screening method [24]. There is not even consensus regarding the best type of swallowing assessment to use, that is, clinical or instrumental [25]. Hence, WST appears as a simple tool, quick to perform at bedside and sensitive enough to identify stroke patients with risk of swallowing impairment.

Conclusions

Our results suggest that stroke severity, assessed using NIHSS, is an important predictor of post-stroke dysphagia; it is also predictive of a persistent pattern of dysphagia, with a suggested NIHSS cutoff value of ≥12 (90.1% specificity, 72.4% sensitivity). An independent correlation was observed with leukoaraiosis and with previous cognitive impairment. These data might suggest that subcortical white matter connections are important in swallowing and that the disruption of cortical–subcortical white matter connections plays an important role in the pathogenesis of dysphagia after stroke.

Acknowledgments

We would like to thank Prof. A. Giuliani for the statistical advice and B. Viganò for the graphic support.

Disclosure Statement

The authors state no conflicts of interest. There was no funding for this work.

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