

Original article

Lesion location and other predictive factors of dysphagia and its complications in acute stroke



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SUMMARY

Background and aims: Early detection of dysphagia is crucial in stroke patients as a result of increased morbidity and mortality due to malnutrition and respiratory tract infections. The aim of this study was to identify possible predictors of the onset of dysphagia following stroke in order to be able to act precociously.

Methods: Observational, prospective study in which a Volume-Viscosity Swallow Test (V-VST) was carried out in the first 72 h following admission to assess dysphagia in acute stroke patients with a previous result of <3 in the Eating Assessment Tool-10. Lesions were analysed by computed tomography and/or magnetic resonance, using the ABC/2 formula to calculate their volume. Likewise, 3-month follow-up was carried out for the evaluation of the occurrence of respiratory tract infections and deaths.

Results: Out of 106 patients admitted for acute stroke, 60 (56.60%) presented dysphagia (44.40% showing alterations in the effectiveness of swallowing and 33.30% in its safety). The factors that were related to dysphagia were: older age (76.40 ± 11.50 vs 66.37 ± 13.85 years, $p = 0.0001$), stroke severity as measured on the National Institute of Health Stroke Scale (6.81 ± 5.83 vs 3.38 ± 3.46 , $p = 0.001$) and greater volume of the lesion (23.47 ± 47.15 vs 7.50 ± 14.53 ml, $p = 0.042$). The variables that were influenced by a greater lesion size were the presence of cough, oxygen desaturation and impaired labial seal. Dysphagia was not affected by the lateralization of the lesion or by the type of stroke (ischaemic/haemorrhagic). Despite the fact that 68.80% of the patients with a temporoparietal lesion presented dysphagia, no significant differences were observed regarding the location of the lesion in the regions studied. 27.3% of the patients with frontal lesions presented respiratory infections after discharge ($p = 0.018$), a condition which was also observed in 20.0% of patients with dysphagia ($p = 0.044$). Mortality during the 3-month follow-up period was 20.0% for patients with a positive V-VST ($p = 0.005$), due to respiratory infection in 66.6% of the cases ($p = 0.0001$).

Conclusions: Post-stroke dysphagia was associated with the occurrence of respiratory tract infection and mortality. Our study also provides more information about how certain demographic and clinical factors, as well as neuroimaging patterns, influence dysphagia. This fact may help to identify at an early stage those patients with a greater risk of developing swallowing alterations.

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1. Introduction

Oropharyngeal dysphagia is a swallowing disorder that frequently occurs after acute stroke, although its incidence varies in

the literature between 19% and 81% [1,2]. It has been associated with an increase in the morbidity and mortality of patients recovering from a stroke, as well as with an increase in hospital stay and health care costs due to malnutrition, dehydration and respiratory tract infections [3,4]. The early identification of patients at risk of suffering dysphagia is crucial in the management of acute stroke, since early treatment can improve the outcome [5].

The recognition of a brain lesion pattern associated with oropharyngeal dysphagia could help to distinguish those patients in need of more in-depth evaluation and the subsequent adoption of preventive measures. However, it is difficult to predict which patients are susceptible to developing swallowing alterations depending on neuroimaging findings. Several studies have attempted to identify patterns of brain lesions that predict dysphagia [6–9]. However, the findings have been inconsistent, mainly due to simplification in the classification of brain injuries into a small number of groups, or to the different methods employed in assessing swallowing function.

Although videofluoroscopy can be considered the gold standard in the diagnosis of dysphagia because of its ability to study the entire process of deglutition [10], it is not feasible to carry it out by default on all patients suspected of dysphagia after an acute stroke. In this regard, the Volume-Viscosity Swallow Test (V-VST) is a bedside clinical method that allows the existence of swallowing alterations to be evaluated, at a low cost and with a high sensitivity and specificity [11].

Therefore, the aim of this study was to identify possible predictors of oropharyngeal dysphagia in acute stroke using an early-detection bedside method.

2. Materials and methods

2.1. Subjects

This is an observational, prospective study involving patients who were admitted to the Neurology Department with the diagnosis of acute ischaemic or haemorrhagic stroke between August 2017 and April 2018. Of the 389 patients hospitalized due to stroke in this period of time, 106 subjects who met the inclusion criteria were enrolled in this study. Fifty-eight were male (54.7%), with an average age of 72.05 ± 13.47 years. The assessment of dysphagia was carried out in all cases in the first 72 h after admission. The exclusion criteria were: presence of oropharyngeal dysphagia prior to acute stroke, previous history of stroke, transient ischaemic attack, admission to intensive care unit, neurosurgical intervention, history of other neurologic disorders other than cerebrovascular disease, history of head and neck damage, low level of consciousness, lack of patient collaboration and lack of patient/family consent.

2.2. Methods

Screening of dysphagia prior to stroke was carried out using the Eating Assessment Tool - 10 (EAT-10), and those patients with a score of 3 or higher were discarded. Stroke severity was assessed by the National Institutes of Health Stroke Scale (NIHSS). Presence of aphasia and dysarthria was recorded for each patient. The brain lesions were analysed by computed tomography and/or magnetic resonance, and classified according to their location in: frontal, temporoparietal, occipital, brainstem, cerebellum, basal ganglia and spinal cord. The ABC/2 method was used to calculate the volume of the brain lesions [12].

The assessment of oropharyngeal dysphagia was carried out according to the V-VST. This bedside method allows for the identification of both alterations in the safety of swallowing (cough,

changes in voice quality, oxygen desaturation) and alterations in its effectiveness (impaired labial seal, oral residue, fractional swallowing, pharyngeal residue), by the administration of fluids with different viscosities (liquid, nectar and pudding) and with different volumes (5, 10 and 20 ml) [11].

Those patients diagnosed with dysphagia secondary to stroke were provided with the required nutritional support. In addition, they were followed closely during the 3 months following stroke in order to assess possible complications related to dysphagia (respiratory infection and death).

2.3. Statistical analysis

All statistical analyses were performed with the SPSS 22.0 program (Chicago, Illinois; USA). Data are expressed as the mean \pm SD or % values. For each continuous variable, the hypothesis of a normal distribution was verified by the Kolmogorov–Smirnov test. T-test or Mann–Whitney test were used to compare a quantitative variable in two groups. The χ^2 test and Fisher's exact test were used to compare qualitative variables. The level of significance was set at $p < 0.05$.

2.4. Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Ethics Review Panel of the Consellería de Sanidade, Xunta de Galicia, and with the Helsinki declaration and its later amendments or comparable ethical standards. All subjects provided informed consent for participation in the study and for the publication of their clinical information. For those patients with communication impairments, informed consent was obtained from their legal guardian.

3. Results

Of the 106 patients admitted to the Neurology Department with the diagnosis of acute stroke, 60 (56.6%) presented dysphagia. Alterations were found in the effectiveness of swallowing in 44.4% of these patients, and in its safety in 33.3%.

The demographic and clinical data of these patients are shown in Table 1. The average age of the patients with dysphagia was higher than that of those who did not present alterations in swallowing, without finding differences regarding sex. Likewise, patients with dysphagia also presented higher severity of stroke according to the NIHSS score. Although no relationship was found between the presence of aphasia or dysarthria and the fact of presenting dysphagia globally, the influence of dysarthria on the alteration of the safety of swallowing was observed. Specifically, 80.0% of the patients who presented a change in voice quality during the V-VST ($p = 0.020$) and 65.6% of the patients with cough ($p = 0.020$) had dysarthria. In addition, 76.9% of patients who showed impaired labial seal also had dysarthria ($p = 0.010$).

Neither was there any relationship with dysphagia regarding the type of stroke (ischaemic vs haemorrhagic) nor the location of the brain lesion in the right vs the left hemisphere (Table 1). Of the patients with temporoparietal lesion, 68.8% presented dysphagia. However, this result did not reach statistical significance ($p = 0.052$) (Fig. 1). There were also no differences in the location of the lesion at the frontal region, occipital region, brainstem, basal ganglia, cerebellum or spinal cord. In addition, 21 of the patients showed lesions in more than one region, 71.4% of them presenting dysphagia. Regarding the possible association between respiratory tract infection and the location of the lesion, it was observed that 27.3% of the patients with frontal lesion developed respiratory infection ($p = 0.018$) (Fig. 1).

Table 1
Demographic and clinical data of the patients with and without dysphagia.

	Total (n = 106)	Dysphagia (n = 60)	No Dysphagia (n = 46)	P value
Sex (% male/female)	53.7/46.3	47.5/52.5	60.9/39.1	0.171
Age (years)	72.05 ± 13.47	76.40 ± 11.50	66.37 ± 13.85	0.0001
Type of stroke (% ischaemic/haemorrhagic)	77.8/22.2	79.3/20.7	82.2/17.8	0.711
Hemisphere (% right/left)	50.9/49.1	55.2/44.8	34.9/65.1	0.129
NIHSS score	5.49 ± 5.44	6.81 ± 5.83	3.38 ± 3.46	0.001
Aphasia (%)	14.8	15.3	15.6	0.588
Dysarthria (%)	41.7	45.8	40.0	0.350

Data are mean ± SD or % values.

NIHSS, National Institutes of Health Stroke Scale.

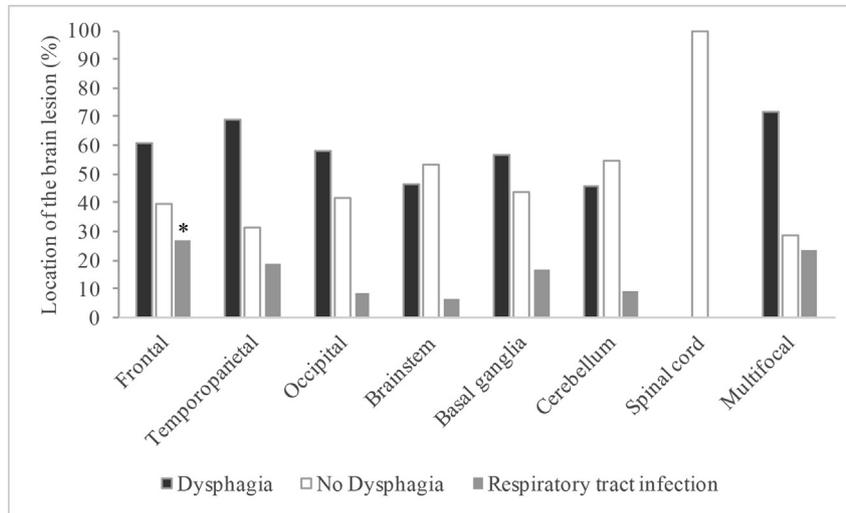


Fig. 1. Presence of dysphagia and respiratory tract infection depending on the location of the lesion. Data are % values. *p < 0.05.

As far as the volume of the lesion is concerned (Table 2), we have observed its influence in the global presentation of dysphagia, and in particular in the appearance of alterations in the safety of swallowing (28.93 ± 47.39 ml in patients with impaired safety of swallowing vs 10.37 ± 29.82 ml in patients without this impairment), showing statistical significance. The variables that were influenced by a greater volume of brain lesion were the presence of cough, oxygen desaturation and impaired labial seal. However, no association was found with regard to respiratory infection.

In the 3-month follow-up period after hospital discharge (Table 3), among those patients who had been diagnosed with dysphagia, 12 (20%) developed respiratory infection and 12 (20%) died (66.6% in the context of respiratory infection and 33.3% due to

other causes). Three patients (6.5%) who did not receive a diagnosis of dysphagia during admission had a respiratory infection, one of whom died due to this cause.

4. Discussion

Dysphagia is a well known complication of stroke. Its incidence varies in the literature between 19% and 81% [1,2]. This is mainly due to the use of different evaluation methods. Thus, using a bedside clinical assessment, the incidence is 51%–55% [3], which agrees approximately with the percentage of patients who presented dysphagia after stroke in our study (56.6%).

As previously mentioned in the literature, we have observed the influence of some demographic (age) and clinical factors (severity of stroke, dysarthria) in the presentation of alterations in swallowing. Several studies agree that age, severity of stroke and a worse functional and nutritional status are factors that are associated with dysphagia after a stroke [6,13,14]. Moreover, the association between dysphagia and aphasia or dysarthria has also been described [1,6].

Regarding the location of the brain lesion according to neuroimaging, much work has been done in an attempt to correlate it with resulting deficits, in order to predict the type of difficulties in swallowing that patients may present with [6–9]. However, the results obtained have been inconsistent, with quite variable conclusions depending on the study and the methodology used. Traditionally, post-stroke alterations were associated with brainstem lesions [15] but recently, dysphagia has also been related to hemispheric lesions. In our study, we have not identified any parameter related to the location of the lesion and alterations in

Table 2
Volume of the lesion (ml) according to alterations in the safety and efficacy of swallowing and respiratory tract infection.

	Presence	Absence	P value
Dysphagia	23.47 ± 47.15	7.50 ± 14.53	0.042
- Impaired safety of swallowing	28.93 ± 47.39	10.37 ± 29.82	0.017
• Cough	29.73 ± 48.85	10.80 ± 29.81	0.018
• Changes in voice quality	24.72 ± 40.73	15.37 ± 37.07	0.389
• Oxygen desaturation	41.85 ± 62.14	12.83 ± 31.18	0.008
- Impaired efficacy of swallowing	21.17 ± 41.92	12.87 ± 33.35	0.265
• Labial seal	45.91 ± 71.24	21.61 ± 28.84	0.003
• Oral residue	7.70 ± 2.40	16.67 ± 37.72	0.739
• Fractional swallowing	20.69 ± 43.10	13.61 ± 32.92	0.347
• Pharyngeal residue	13.42 ± 20.07	16.79 ± 38.68	0.797
Respiratory tract infection	30.64 ± 59.91	13.94 ± 31.77	0.108

Data are mean ± SD values.

Table 3
Occurrence of respiratory tract infection and death in the 3 months after stroke.

	Total (n = 106)	Dysphagia (n = 60)	No Dysphagia (n = 46)	P value
Respiratory tract infection (n)	15 (14.15%)	12 (20.00%)	3 (6.52%)	0.044
Death (n)	13 (12.26%)	12 (20.00%)	1 (2.17%)	0.005
- Due to respiratory tract infection (n)	9 (69.23%)	8 (66.66%)	1 (100%)	0.0001
- Due to other causes (n)	4 (30.76%)	4 (33.33%)	0 (0.00%)	

Data are n and % values.

swallowing evaluated by V-VST. Although 68.8% of the patients with temporoparietal lesion presented dysphagia, this result was not statistically significant. In this regard, a recent study carried out on 200 patients with acute stroke applying fibre optic endoscopic evaluation of swallowing, showed that right hemispheric temporoparietal regions were related to penetration and aspiration, and were also significantly associated with impaired swallow response and oropharyngeal residue [16,17]. Although their role in swallowing processing has not yet been clarified, activation during swallowing tasks has previously been found in the temporopolar cortex [18]. Other studies have shown that lesions in the frontal and insular cortex predict prolonged dysphagia and pneumonia after stroke [9,19]. It has also been suggested that the increased attributable risk of right peri-insular infarction in relation to developing pneumonia may be due more to impairments in host immunity than to the increased likelihood of aspiration [20]. In our study, respiratory tract infections were observed in 14.1% of the patients, which is consistent with the data reported in the literature, where the incidence of pneumonia after stroke has been reported to range widely between 1% and 44% [21,22], with 27.3% of patients with frontal lesions developing it after hospital admission.

As far as laterality is concerned, some studies have associated impairment of the oral phase with left hemispheric lesions and pharyngeal phase problems to right hemispheric lesions [23,24], whereas others have found no relation between lateralization and swallowing characteristics, as is the case of our study, suggesting that control is more complex [17,25–27].

Regarding the volume of the brain lesion, the variables that were influenced by a greater lesion size in our study were the presence of cough, oxygen desaturation and impaired labial seal during the V-VST. However, paradoxically, no association was found with regard to respiratory infection. Several studies have related larger lesion size with higher risk of developing swallowing problems and respiratory infection [6,14,25,26]. Specifically, stroke lesion volume has been linked to the efficacy of the cough reflex [17,28]. However, to the best of our knowledge, there are no other trials relating oxygen saturation or other alterations in the effectiveness of swallowing, such as impaired labial seal, with the volume of the brain lesion.

On the other hand, the goal in dysphagia therapy is to reduce the associated morbidity and mortality. The mortality incidence in our study is slightly lower (20%) than in previous studies, where it stands at between 27% and 37% for dysphagic stroke patients [29,30]. The presentation of pneumonia post-stroke has been shown to increase mortality threefold as well as overall hospital care costs [21]; in fact, 66.6% of deaths among patients with dysphagia in our study were due to respiratory tract infection. Poor nutritional status has also certainly been correlated with increased mortality after admission with acute stroke [31]. Dysphagia does therefore have prognostic implications, which is why its early detection is extremely important.

In conclusion, this study shows how dysphagia is clearly associated with clinical outcome and mortality, and it also provides information about how certain demographic and clinical factors, as well as neuroimaging patterns, influence dysphagia, which may

help to identify those patients at greater risk of developing swallowing alterations at an early stage.

Statement of authorship

Fernández-Pombo A, Seijo-Raposo IM, Martínez-Olmos M and Cantón-Blanco A; were responsible for the construction and design of the study. Fernández-Pombo A, López-Osorio N and Seijo-Raposo IM; were responsible for setting up the methodology of the study, as well as data acquisition, analysis and interpretation. Santamaría-Nieto A, Díaz-Ortega C and Gómez-Vázquez E; were also responsible for data acquisition and literature review. Fernández-Pombo A; was responsible for drafting the article. Martínez-Olmos M, Cantón-Blanco A, González-Rodríguez M, Arias-Rivas S and Rodríguez-Yáñez M; have made important contribution revising the article for important intellectual content and were also responsible for the final approval of the version submitted.

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Conflict of interest

The authors have nothing to disclose.

References

- Barer DH. The natural history and functional consequences of dysphagia after hemispheric stroke. *J Neurol Neurosurg Psychiatry* 1989;52:236–41.
- Meng NH, Wang TG, Lien IN. Dysphagia in patients with brainstem stroke: incidence and outcome. *Am J Phys Med Rehabil* 2000;79:170–5.
- Martino R, Foley N, Bhogal S, Diamant N, Speechley M, Teasell R. Dysphagia after stroke: incidence, diagnosis, and pulmonary complications. *Stroke* 2005;36:2756–63.
- Crary MA, Carnaby-Mann GD, Miller L, Antonios N, Silliman S. Dysphagia and nutritional status at the time of hospital admission for ischemic stroke. *J Stroke Cerebrovasc Dis* 2006;15:164–71.
- Elmstahl S, Bulow M, Ekberg O, Petersson M, Tegner H. Treatment of dysphagia improves nutritional conditions in stroke patients. *Dysphagia* 1999;14:61–6.
- Falsetti P, Acciaci C, Palilla R, Bosi M, Carpinteri F, Zingarelli A, et al. Oropharyngeal dysphagia after stroke: incidence, diagnosis, and clinical predictors admitted to a neurorehabilitation unit. *J Stroke Cerebrovasc Dis* 2009;18(5):329–35.
- Galovic M, Leisi N, Muller N, Weber J, Abela E, Kägi G, et al. Lesion location predicts transient and extended risk of aspiration after supratentorial ischemic stroke. *Stroke* 2013;44:2760–7.
- Flowers HL, Skoretz SA, Streiner DL, Silver FL, Martino R. MRI-based neuro-anatomical predictors of dysphagia after acute ischemic stroke: a systematic review and meta-analysis. *Cerebrovasc Dis* 2011;32:1–10.
- Steinhagen V, Grossmann A, Benecke R, Walter U. Swallowing disturbance pattern relates to brain lesion location in acute stroke patients. *Stroke* 2009;40(5):1903–6.
- Costa MM. Videofluoroscopy: the gold standard exam for studying swallowing and its dysfunction. *Arq Gastroenterol* 2010;47(4):327–8.
- Rofes L, Arreola V, Mukherjee R, Clavé P. Sensitivity and specificity of the eating assessment tool and the volume-viscosity swallow test for clinical evaluation of oropharyngeal dysphagia. *Neuro Gastroenterol Motil* 2014;26(9):1256–65.

- [12] Kothari RU, Brott T, Broderick JP, Barsan WG, Sauerbeck LR, Zuccarello M, et al. The ABCs of measuring intracerebral hemorrhage volumes. *Stroke* 1996 Aug;27(8):1304–5.
- [13] Rofes L, Muriana D, Palomeras E, Vilardell N, Palomera E, Alvarez-Berdugo D, et al. Prevalence, risk factors and complications of oropharyngeal dysphagia in stroke patients: a cohort study. *Neuro Gastroenterol Motil* 2018;23:e13338.
- [14] Paciaroni M, Mazzotta G, Corea F, Caso V, Venti M, Milia P, et al. Dysphagia following stroke. *Eur Neurol* 2004;51(3):162–7.
- [15] Daniels SK, Foundas AL. Lesion localization in acute stroke patients with risk of aspiration. *J Neuroimaging* 1999;9(2):91–8.
- [16] Suntrup S, Kemmling A, Warnecke T, Hamacher C, Oelenberg S, Niederstadt T, et al. The impact of lesion location on dysphagia incidence, pattern and complications in acute stroke. Part 1: dysphagia incidence, severity and aspiration. *Eur J Neurol* 2015;22:832–8.
- [17] Suntrup-Krueger S, Kemmling A, Warnecke T, Hamacher C, Oelenberg S, Niederstadt T, et al. The impact of lesion location on dysphagia incidence, pattern and complications in acute stroke. Part 2: oropharyngeal residue, swallow and cough response, and pneumonia. *Eur J Neurol* 2017;24(6):867–74.
- [18] Hamdy S, Rothwell JC, Brooks DJ, Bailey D, Aziz Q, Thompson DG. Identification of the cerebral loci processing human swallowing with H₂ ¹⁵O PET activation. *J Neurophysiol* 1999;81(4):1917–26.
- [19] Broadley S, Croser D, Cottrell J, Creevy M, Teo E, Yiu D, et al. Predictors of prolonged dysphagia following acute stroke. *J Clin Neurosci* 2003;10:300–5.
- [20] Kemmling A, Lev MH, Payabvash S, Betensky RA, Qian J, Masrur S, et al. Hospital acquired pneumonia is linked to right hemispheric peri-insular stroke. *PLoS One* 2013;7:8889. e71141.
- [21] Papavasileiou V, Milionis H, Smith CJ, Makaritsis K, Bray BD, Michel P, et al. External validation of the prestroke independence, sex, age, National Institutes of health stroke Scale (ISAN) score for predicting stroke-associated pneumonia in the athens stroke registry. *J Stroke Cerebrovasc Dis* 2015;24(11):2619–24.
- [22] Emsley HC, Hopkins SJ. Acute ischaemic stroke and infection: recent and emerging concepts. *Lancet Neurol* 2008;7(4):341–53.
- [23] Robbins J, Levine RL, Maser A, Rosenbek JC, Kempster GB. Swallowing after unilateral stroke of the cerebral cortex. *Arch Phys Med Rehabil* 1993;74(12):1295–300.
- [24] Daniels SK, Foundas AL, Iglesia GC. Lesion site in unilateral stroke patients with dysphagia. *J Stroke Cerebrovasc Dis* 1996;6:30–4.
- [25] Sharma JC, Fletcher S, Vassallo M, Ross I. Prognostic value of CT scan features in acute ischaemic stroke and relationship with clinical stroke syndromes. *Int J Clin Pract* 2000 Oct;54(8):514–8.
- [26] Minnerup J, Wersching H, Brokinkel B, Dziewas R, Heuschmann PU, Nabavi DG, et al. The impact of lesion location and lesion size on poststroke infection frequency. *J Neurol Neurosurg Psychiatry* 2010;81:198–202.
- [27] Hamdy S. Role of cerebral cortex in the control of swallowing. *GI Motil Online* 2006. <https://doi.org/10.1038/gimo8>.
- [28] Vilardell N, Rofes L, Nascimento WV, Muriana D, Palomeras E, Clave P. Cough reflex attenuation and swallowing dysfunction in sub-acute post-stroke patients: prevalence, risk factors, and clinical outcome. *Neuro Gastroenterol Motil* 2017;29(1).
- [29] Smithard DG, O'Neill PA, Park C, Morris J, Wyatt R, England R, et al. Complications and outcome after acute stroke: does dysphagia matter? *Stroke* 1996;27:1200–4.
- [30] Katzan IL, Cebul RD, Husak SH, Dawson NV, Baker DW. The effect of pneumonia on mortality among patients hospitalized for acute stroke. *Neurology* 2003;60:620–5.
- [31] FOOD Trial Collaboration. Poor nutritional status on admission predicts poor outcomes after stroke: observational data from the FOOD trial. *Stroke* 2003;34:1450–6.