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RESEARCH PAPER

The association between delays in screening for and assessing dysphagia after acute stroke, and the risk of stroke-associated pneumonia

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ABSTRACT

Background There is no robust evidence that screening patients with acute stroke for dysphagia reduces the risk of stroke-associated pneumonia (SAP), or of how quickly it should be done after admission. We aimed to identify if delays in bedside dysphagia screening and comprehensive dysphagia assessments by a speech and language therapist (SALT) were associated with patients' risk of SAP.

Methods Nationwide, registry-based, prospective cohort study of patients admitted with acute stroke in England and Wales. Multilevel multivariable logistic regression models were fitted, adjusting for patient variables and stroke severity. The exposures were time from (1) admission to bedside dysphagia screen, and (2) admission to comprehensive dysphagia assessment.

Results Of 63 650 patients admitted with acute stroke, 55 838 (88%) had a dysphagia screen, and 24 542 (39%) a comprehensive dysphagia assessment. Patients with the longest delays in dysphagia screening (4th quartile adjusted OR 1.14, 1.03 to 1.24) and SALT dysphagia assessment (4th quartile adjusted OR 2.01, 1.76 to 2.30) had a higher risk of SAP. The risk of SAP increased in a dose-response manner with delays in SALT dysphagia assessment, with an absolute increase of pneumonia incidence of 1% per day of delay.

Conclusions Delays in screening for and assessing dysphagia after stroke, are associated with higher risk of SAP. Since SAP is one of the main causes of mortality after acute stroke, early dysphagia assessment may contribute to preventing deaths from acute stroke and could be implemented even in settings without access to high-technology specialist stroke care.

INTRODUCTION

Stroke-associated pneumonia (SAP) is a common complication of acute stroke, affecting 6–10% of patients.[1] SAP independently increases the risk of mortality and is one of the main causes of death in the first few days and weeks after stroke.[2] It is also associated with worse functional outcomes, longer length of stay and increased healthcare costs.[3–8] One of the main risk factors for SAP is dysphagia, which affects 37–55% of patients[9] after stroke. Dysphagia screening using a brief bedside screening tool (such as a water swallow test), and comprehensive clinical assessments of aspiration risk by speech and language therapists (SALT) are, therefore, performed commonly in stroke care. Typically, all appropriate patients are screened for dysphagia, and those in whom dysphagia is suspected go on to receive a comprehensive assessment. Despite being well established in clinical practice, there is, however, very little evidence of effectiveness for these interventions. Previous studies have generally used weak designs and provided no information to guide recommendations on how quickly dysphagia assessments after stroke should occur.[10 11] Swallowing assessment prior to eating and drinking is recommended in European,[12] US[13] and UK clinical guidelines, but none recommend a specific approach to assessment or treatment of dysphagia.

As SAP develops most frequently within the first 7 days of stroke,[1] the timing of both dysphagia screening and SALT assessment after admission is likely to be of importance. In England and Wales, dysphagia screening is recommended within 4 hours of admission for acute stroke, and comprehensive assessment by a SALT (if required) within 72 hours of admission.[14] The aim of this study was to investigate the association between delays in dysphagia screening and SALT assessment, and the incidence of SAP within the first 7 days after admission. The hypothesis was that delays in these assessments would be associated with an increased incidence of SAP.

METHODS

Data were from the Sentinel Stroke National Audit Programme (SSNAP), the national register of stroke in England and Wales, of patients aged ≥16 years admitted with acute stroke (ischaemic stroke or primary intracerebral haemorrhage) between April 2013 and March 2014. SSNAP is a prospective continuous register with participation from all hospitals admitting adults with stroke in England and Wales, and is estimated to include 90–95% of all stroke admissions.[15] Ethical approval of SSNAP was granted by the Ethics and Confidentiality Committee of the National Information Governance Board. Mortality data were obtained through data linkage with the statutory register of deaths. Data linkage was carried out by a third party, and the investigators used an anonymised dataset with all patient identifiers removed.

Dysphagia screening was defined as use of a bedside swallow screening test by an appropriately

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trained clinician (typically a trained nurse). The exact dysphagia screening protocol was not specified by SSNAP. The times from admission to documented dysphagia screen and comprehensive assessment by SALT were recorded to the nearest minute for all patients in whom these were carried out. For patients who had a stroke as an inpatient, the time from stroke onset was used instead of time of admission. All patients without clinical exclusions (eg, being treated palliatively only) were eligible for dysphagia screening, and eligibility for comprehensive dysphagia assessment was determined by clinical indications, such as a positive dysphagia screen or clinical suspicion of dysphagia. Patients (n=965) admitted directly to an intensive care unit (ICU) on admission were excluded from the primary analysis, since most of these patients would have been intubated.

SAP was defined as the administration of antibiotics for a new clinical diagnosis of pneumonia in the first 7 days after admission, and was determined by the treating physician.

Statistical analysis

The adjusted odds of SAP were estimated by fitting multivariable logistic regression models. Time from admission to dysphagia screen and SALT dysphagia assessment were analysed both as continuous variables and by division into quartiles. When included as a continuous variable, they were fitted as restricted cubic regression splines using the POSTRCSPLINE module.[16] Spline coefficients cannot be interpreted directly, and so, the models were displayed graphically, showing the modelled association (and 95% CI) between time to dysphagia screen or comprehensive dysphagia assessment and estimated adjusted SAP incidence. Multilevel multivariable logistic regression models were also fitted using quartiles of these times to enable quantification of the study results into ORs and also to account for clustering at the hospital level. These models were specified as two-level models, with hospital-level random intercepts.

All models included age, sex, stroke subtype (ischaemic, primary intracererbral haemorrhage, or undetermined), prestroke functional level using the modified Rankin Scale, place of stroke (out of hospital or inpatient), vascular comorbidity (heart failure, hypertension, atrial fibrillation, diabetes mellitus, previous stroke or transient ischaemic attack), and either NIH Stroke Scale (NIHSS) or level of consciousness on admission.

Data were complete for all data items apart from the NIHSS on admission, which was available for 73% of patients. Models were therefore also fitted using level of consciousness on admission as a proxy for severity (available for 100% of patients), and the results of the models compared in order to explore the effects of these missing data on the results.

We carried out several sensitivity analyses. First, competing risk from early mortality was explored by excluding patients dying or starting palliative care in the first 3 days. Second, models were fitted including a variable, indicating change (increase, no change, decrease) in the level of consciousness in the first 7 days after stroke, to explore the possible confounding effect of changing consciousness level. Third, ICU patients excluded in the main analysis were included in a complete dataset analysis. Fourth, time from admission to stroke unit was included in the models as a possible confounder. Finally, we fitted models of 30-day all-cause mortality, excluding patients dying or starting palliative care in the first 3 days (on the grounds that death in these latter patients is more likely due directly to brain injury from the stroke rather than SAP). These models explored whether delays in dysphagia screening and assessment were associated with mortality after stroke.

RESULTS

There were 63 650 patients with acute stroke included in the cohort, admitted to 199 hospitals. Of these, 55 838 (87.7%) had a dysphagia screen, and 24 542 (38.6%) proceeded to a comprehensive assessment. The characteristics of the whole cohort, and the subgroups of patients according to receipt of dysphagia screening and comprehensive dysphagia assessment, are described in table 1. Patients in whom a dysphagia screen was not performed had a greater incidence of inpatient stroke and primary intracerebral haemorrhage, lower level of consciousness on admission, and were less likely to have NIHSS on admission completed.

The overall incidence of SAP was 8.7%. SAP incidence was highest in the dysphagic group referred for comprehensive dysphagia assessment (14.6%). Thirty-day mortality was 13.2% overall, 10.2% in patients screened for dysphagia, 14.7% in patients referred for SALT assessment and 34.6% in patients in whom a dysphagia screen was not carried out.

The median time from admission to dysphagia screening was 2.9 hours (IQR 1.3–5.7 hours), and for comprehensive dysphagia assessment was 22.9 hours (IQR 6.2–49.4 hours). In unadjusted analyses, there was a strong association between time from admission to dysphagia screen and incidence of SAP, rising from 7% to 8% from 0 to 8 hours, and increasing to 15% by 72 hours after admission. Although the association was attenuated after adjusting for patient characteristics, there was still a modest association (equating to approximately 1% absolute increase in the incidence of SAP) between delays in dysphagia screening and incidence of SAP (figure 1). After adjustment, patients in the fourth quartile (ie, those with the longest delays in dysphagia screening) had 36% higher odds of SAP compared with those in the first quartile (aOR 1.36, 1.20 to 1.53) (table 2).

There was a strong relationship between delays in comprehensive dysphagia assessment and incidence of SAP, and delays in comprehensive dysphagia assessment were associated with an absolute increase in the risk of SAP of 3% over the first 24 hours (figure 2). Delays in SALT dysphagia assessment beyond 24 hours were associated with an additional 4% absolute increase in the incidence of SAP (approximately threefold increase in the relative incidence). Patients in the slowest quartile had 1.98 (1.67–2.35) the odds of SAP compared with patients receiving the quickest SALT dysphagia assessments (table 3). Findings were similar in the sensitivity analyses (see online supplementary material). The secondary analysis of 30-day mortality broadly supported these findings: there was very weak evidence that delays in dysphagia assessment were associated with an increase in 30-day mortality (aOR 1.14, 0.99 to 1.30 in the slowest quarter). There was moderately strong evidence that delays in comprehensive SLT assessment were associated with an increase in mortality risk in the second (aOR 1.22, 1.02 to 1.47), third (aOR 1.55, 1.29 to 1.85) and fourth (aOR 1.35, 1.12 to 1.63) quarters of time to assessment, respectively. Unlike pneumonia, a dose-response relationship was not demonstrated for the association with mortality (see online supplementary material).

DISCUSSION

In this national cohort of unselected stroke patients, we found that there was evidence of a modest association between delays in performing dysphagia screening and the risk of SAP. There was stronger evidence for an association between the risk of SAP and delays in carrying out a comprehensive dysphagia assessment. Although limited by the risk of residual

Table 1 Characteristics of the study cohort

	Cohort	Dysphagia screening not performed	Dysphagia screening performed	Comprehensive dysphagia assessment
n (%)	63 650	7812 (12.3)	55 838 (87.8)	24 542 (38.6)
Median age, years (IQR)	77 (67–85)	80 (70–86)	77 (67–85)	80 (70–87)
Female (n, %)	32 054 (50.4)	4264 (54.5)	27 790 (49.8)	13 160 (53.6)
Stroke type (n, %)				
Ischaemic	56 167 (88.2)	5948 (76.1)	50 219 (91.0)	21 751 (89.6)
Primary intracerebral haemorrhage	6575 (10.3)	1592 (20.4)	4983 (8.9)	2523 (10.4)
Undetermined	908 (1.4)	272 (3.5)	636 (1.1)	268 (1.1)
Inpatient stroke (n, %)	3155 (5.0)	974 (12.5)	2181 (3.9)	1599 (6.5)
Prestroke mRS (n, %)				
0	36 208 (57.9)	3808 (48.8)	32 400 (58.0)	12 174 (49.6)
1	9726 (15.3)	1109 (14.2)	8617 (15.4)	3919 (16.0)
2	6036 (9.5)	851 (10.9)	5185 (9.3)	2614 (10.7)
3	6708 (10.5)	1073 (13.7)	5635 (10.1)	3163 (12.9)
4	3734 (5.9)	670 (8.6)	3064 (5.5)	1988 (8.1)
5	1248 (2.0)	301 (3.9)	937 (1.7)	684 (2.8)
Admission NIHSS complete (n, %)	46 447 (73.0)	3792 (48.4)	42 655 (76.4)	17 041 (69.4)
Median admission NIHSS (IQR)	4 (2–9)	5 (2–19)	4 (2–9)	7 (3–14)
Level of consciousness on admission				
0 (alert)	53 433 (84.0)	4961 (63.5)	48 472 (86.8)	19 156 (78.1)
1 (Not alert: responds to voice)	6032 (9.5)	1069 (13.7)	4963 (8.9)	3697 (15.1)
2 (Not alert: responds to pain)	2498 (3.9)	828 (10.6)	1670 (3.0)	1267 (5.2)
3 (Totally unresponsive)	1687 (2.7)	954 (12.2)	733 (1.3)	422 (1.7)
Comorbidity (n, %)				
Heart failure	3463 (5.4)	491 (6.3)	2972 (5.3)	1625 (6.6)
Hypertension	34 212 (53.9)	3930 (50.3)	30 382 (54.4)	13 323 (54.3)
Atrial fibrillation	13 159 (20.7)	1801 (23.1)	11 358 (20.3)	5929 (24.2)
Diabetes mellitus	12 372 (19.4)	1482 (18.9)	10 890 (19.5)	4720 (19.2)
Previous stroke/TIA	17 626 (27.7)	2144 (27.4)	15 482 (27.7)	7109 (29.0)
Time from onset to admission (n, %)				
Unknown (eg, wake up stroke)	24 668 (38.8)	4233 (54.2)	20 435 (36.6)	9631 (39.2)
0–179 min	21 504 (33.8)	2113 (27.1)	19 391 (34.7)	9086 (37.0)
180–359 min	6144 (9.7)	539 (6.9)	5605 (10.0)	2289 (85.6)
360+ min	11 334 (17.8)	927 (11.9)	10 407 (18.6)	3536 (14.4)
Thrombolysis (n, %)	7087 (11.1)	417 (5.3)	6670 (12.0)	3415 (3.1)
SAP (n, %)	5533 (8.7)	1077 (13.8)	4456 (8.0)	3592 (14.6)
30-day mortality (n, %)	8397 (13.2)	2701 (34.6)	5696 (10.2)	3599 (14.7)

mRS, modified Rankin Scale; NIHSS, NIH Stroke Scale; SAP, stroke-associated pneumonia; TIA, transient ischaemic attack

confounding, these findings provide the first evidence from a large multicentre cohort that prompts dysphagia screening and comprehensive dysphagia assessment stroke are associated with clinically significant reductions in the risk of SAP, one of the principal causes of early death after stroke.

Detecting dysphagia through the use of bedside screening assessments and comprehensive dysphagia assessments carried out by a SALT is widely recommended in clinical guidelines.[12–14] However, these recommendations are largely based on consensus, and there is little direct evidence for dysphagia screening or assessment after stroke.[17] Previous studies have been limited to ecological studies demonstrating an association between site-level rates of screening assessment and SAP rates after stroke.[8 10 11] Several observational studies have described an association between dysphagia screening at any time after stroke and reduced poststroke mortality,[10 18–20] and dysphagia screening was a component of a stroke care bundle found in a cluster randomised controlled trial to reduce death and dependency after stroke.[21] By contrast, an analysis of the Get with the Guidelines—Stroke registry data from the USA found that dysphagia screening was associated with a higher risk of SAP, although the results suggest that confounding by stroke severity contributed to the observed association.[22] There is no current evidence of how quickly dysphagia assessment should occur after stroke, or good quality evidence of whether dysphagia assessment reduces the risk of SAP. As a result of this lack of evidence, dysphagia screening has been dropped from the list of stroke quality indicators used in the USA,[23] although they remain part of quality indicators used in the UK.[24]

If our findings represent causal effects, then they imply that dysphagia screening and assessment is effective in reducing the risk of SAP. Since SAP is one of the main causes of death in acute stroke, reducing the risk of SAP would be expected to lead to reduced mortality after stroke. The secondary analyses of mortality in this study provide supporting evidence that this might be the case. We would, however, emphasise caution in interpreting the mortality findings—there are many causes of death in acute stroke, and so reductions in SAP will only prevent a proportion of deaths (in keeping with the reduced effect sizes for mortality we observed), and we did not observe

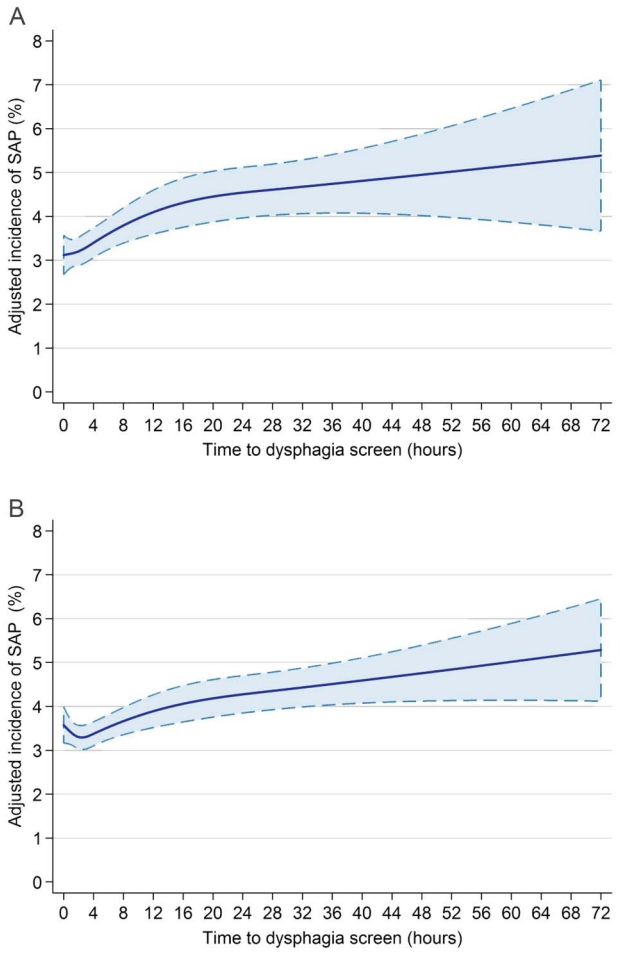

Figure 1 Modelled relationship between estimated incidence of SAP in the first 7 days of admission and time to dysphagia screening. (A) Multivariable model including NIHSS. (B) Multivariable model including level on consciousness. NIHSS, NIH Stroke Scale; SAP, stroke-associated pneumonia.

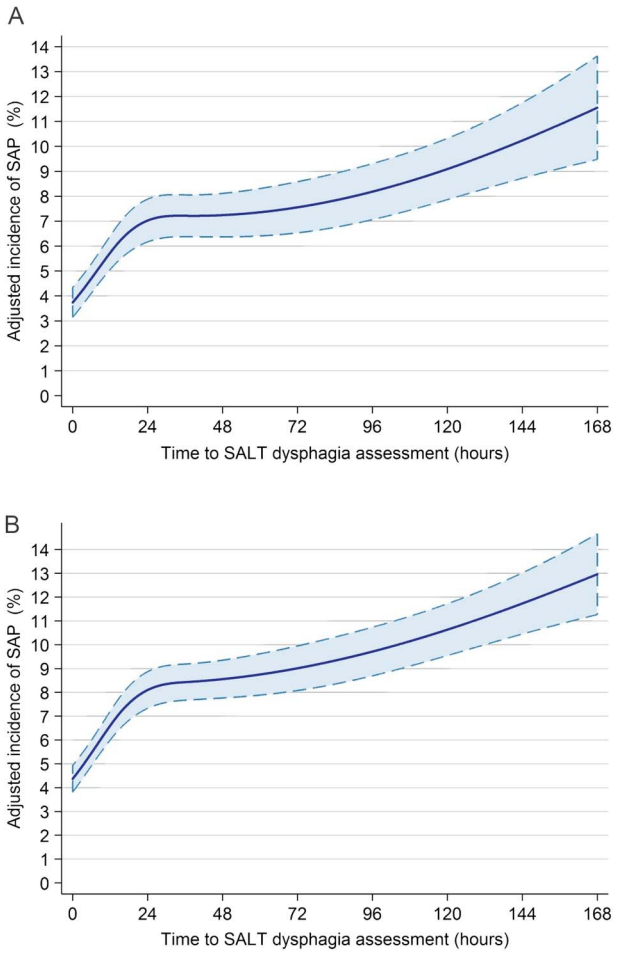

Figure 2 Modelled relationship between estimated incidence of SAP in the first 7 days of admission and the time to SALT dysphagia assessment. (A) Multivariable model including NIHSS. (B) Multivariable model including level on consciousness. NIHSS, NIH Stroke Scale; SALT, speech and language therapist; SAP, stroke-associated pneumonia.

Table 2 OR for SAP in univariable and multivariable models of time from admission to dysphagia screening

	Time (min)	OR	95% CI	p Value
Univariable (n=55 838)				
1st quartile	0–79	REF		
2nd quartile	80–176	0.89	0.81 to 0.98	0.016
3rd quartile	177–344	0.85	0.77 to 0.94	0.001
4th quartile	≥345	1.33	1.21 to 1.46	<0.0001
Multivariable, including NIHSS (n=42 655)				
1st quartile	0–79	REF		
2nd quartile	80–176	0.94	0.83 to 1.05	0.27
3rd quartile	177–344	1.06	0.94 to 1.20	0.36
4th quartile	≥345	1.36	1.20 to 1.53	<0.0001
Multivariable, including level of consciousness (n=55 838)				
1st quartile	0–79	REF		
2nd quartile	80–176	0.92	0.83 to 1.01	0.08
3rd quartile	177–344	0.89	0.81 to 0.99	0.03
4th quartile	≥345	1.14	1.03 to 1.24	0.008

All multivariable models were also adjusted for age, sex, stroke type, prestroke functional level, place of stroke and comorbidity, and measure of stroke severity (NIHSS or level of consciousness).
SAP, stroke-associated pneumonia; NIHSS, NIH Stroke Scale

a dose-response relationship between delays in comprehensive dysphagia assessment and mortality, suggesting that there might be additional confounding or bias not accounted for in the analysis.

There are several possible mechanisms for why delays in dysphagia assessment might lead to an increased risk of SAP, and further studies would be required to test these hypotheses and confirm (or refute) a causal relationship: early screening may reduce the risk of inappropriate administration of oral fluid or food, prompt measures to reduce risk of aspiration through positioning, nursing care and appropriate feeding strategies, and avoid unnecessary nasogastric tube insertions. As well as exploring mechanisms, further research might usefully also explore organisational aspects of dysphagia assessment, such as the use of specific assessment and treatment protocols and the relationship between specialist SALT provision on stroke units and patient outcomes.

These data are strengthened by being drawn from a national register of unselected patients, reducing the risk of selection bias. Similarly, the study used clinical rather than administrative data, providing more detail than would be available from routine administrative data alone. There are, however, a number of limitations. First, SAP was not defined by specific criteria but was based on the judgement of the treating physician, and we

Table 3 OR for SAP in univariable and multivariable models of time from admission to SALT dysphagia assessment

	Time (min)	OR	95% CI	p Value
Univariable (n=24 542)				
1st quartile	0–369	REF		
2nd quartile	370–1371	1.53	1.34 to 1.74	<0.0001
3rd quartile	1372–2961	1.95	1.71 to 2.22	<0.0001
4th quartile	≥2962	2.65	2.33 to 3.01	<0.0001
Multivariable, including NIHSS (n=17 041)				
1st quartile	0–369	REF		
2nd quartile	370–1371	1.35	1.15 to 1.60	<0.0001
3rd quartile	1372–2961	1.61	1.37 to 1.91	<0.0001
4th quartile	≥2962	1.98	1.67 to 2.35	<0.0001
Multivariable, including level of consciousness (n=24 542)				
1st quartile	0–369	REF		
2nd quartile	370–1371	1.40	1.22 to 1.60	<0.0001
3rd quartile	1372–2961	1.60	1.41 to 1.84	<0.0001
4th quartile	≥2962	2.01	1.76 to 2.30	<0.0001

All multivariable models were also adjusted for age, sex, stroke type, prestroke functional level, place of stroke and comorbidity, and measure of stroke severity (NIHSS or level of consciousness).
NIHSS, NIH Stroke Scale; SALT, speech and language therapist; SAP, stroke-associated pneumonia

did not have information on the date of diagnosis of SAP and whether it occurred before or after dysphagia screening and assessment. Nonetheless, the overall rate of SAP observed in this study is consistent with other studies, suggesting that differences in ascertainment between centres were not significant sources of bias. In addition, although data completeness was high, NIHSS data were not available for one-quarter of patients. We used level of consciousness as a proxy for this and found that the findings were similar, but having complete data on stroke severity may have strengthened the study. The dataset lacked information on the nature of the bedside dysphagia screening tools used, the details of the comprehensive assessment (eg, videofluroscopy or fibre-optic evaluation of swallowing), and the results of these assessments. Further studies should aim to capture in more detail the components of these interventions. The main limitation of this study is the risk of residual confounding. The hypothesis that early dysphagia screening and SALT assessment reduce the risk of SAP could be tested in a cluster-randomised controlled trial of a protocol of expedited comprehensive dysphagia assessment, and this would help guide clinical practice in an important area of stroke care which currently has a poor evidence base.

Implementing faster dysphagia assessments in clinical practice is principally a matter of training healthcare professionals appropriately, and in most instances does not require expensive medical equipment. Dysphagia screening has been identified by the World Stroke Organisation as being achievable even in health economies with the lowest level of resources;[25] ensuring that all stroke patients receiving rapid dysphagia assessments could, therefore, be a part of global efforts to improve the outcomes of acute stroke, even in settings without advanced specialist stroke care.

Summary

Delays in screening for dysphagia and carrying out SALT dysphagia assessments after stroke are associated with an increased risk of SAP. This hypothesis that expedited dysphagia screening and assessments reduce the risk of SAP, would be testable in an appropriately designed trial or controlled evaluation. In the meantime, these findings suggest that reducing delays in screening and assessing for dysphagia in people with acute stroke should be a focus of quality improvement in stroke care.

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REFERENCES

1 Westendorp WF, Nederkoorn PJ, Vermeij JD, *et al*. Post-stroke infection: a systematic review and meta-analysis. *BMC Neurol* 2011;11:110.
2 Vernino S, Brown RD Jr, Sejvar JJ, *et al*. Cause-specific mortality after first cerebral infarction: a population-based study. *Stroke* 2003;34:1828–32.
3 Katzan IL, Cebul RD, Husak SH, *et al*. The effect of pneumonia on mortality among patients hospitalized for acute stroke. *Neurology* 2003;60:620–5.
4 Finlayson O, Kapral M, Hall R, *et al*., Canadian Stroke Network; Stroke Outcome Research Canada (SORCan) Working Group. Risk factors, inpatient care, and outcomes of pneumonia after ischemic stroke. *Neurology* 2011;77:1338–45.
5 Katzan IL, Dawson NV, Thomas CL, *et al*. The cost of pneumonia after acute stroke. *Neurology* 2007;68:1938–43.
6 Ingeman A, Andersen G, Hundborg HH, *et al*. In-hospital medical complications, length of stay, and mortality among stroke unit patients. *Stroke* 2011;42:3214–18.
7 Koennecke HC, Belz W, Berfelde D, *et al*. Factors influencing in-hospital mortality and morbidity in patients treated on a stroke unit. *Neurology* 2011;77:965–72.
8 Odderson IR, Keaton JC, McKenna BS. Swallow management in patients on an acute stroke pathway: quality is cost effective. *Arch Phys Med Rehabil* 1995;76:1130–3.
9 Martino R, Foley N, Bhogal S, *et al*. Dysphagia after stroke. Incidence, diagnosis, and pulmonary complications. *Stroke* 2005;36:2756–63.
10 Hinchey JA, Shephard T, Furie K, *et al*., Stroke Practice Improvement Network Investigators. Formal dysphagia screening protocols prevent pneumonia. *Stroke* 2005;36:1972–6.
11 Titsworth WL, Abram J, Fullerton A, *et al*. Prospective quality initiative to maximize dysphagia screening reduces hospital-acquired pneumonia prevalence in patients with stroke. *Stroke* 2013;44:3154–60.
12 European Stroke Organisation (ESO) Executive Committee; ESO Writing Committee. Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis* 2008;25:457–507.
13 Jauch EC, Saver JL, Adams HP Jr, *et al*., American Heart Association Stroke Council; Council on Cardiovascular Nursing; Council on Peripheral Vascular Disease; Council

on Clinical Cardiology. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2013;44:870–947.
14 *National clinical guideline for stroke*. 4th edn. Intercollegiate Stroke Working Party, 2012. http://www.rcplondon.ac.uk/sites/default/files/national-clinical-guidelines-for-stroke-fourth-edition.pdf (accessed 10 Jun 2015).
15 Sentinel Stroke National Audit Programme. http://www.strokeaudit.org (accessed 10 Jun 2015).
16 Buis M. POSTRCSPLINE: Stata module containing post-estimation commands for models using a restricted cubic spline. http://EconPapers.repec.org/RePEc:boc:bocode:s456928 (accessed 10 Jun 2015).
17 Luker JA, Wall K, Bernhardt J, *et al*. Measuring the quality of dysphagia management practices following stroke: a systematic review. *Int J Stroke* 2010;5:466–76.
18 Turner M, Barber M, Dodds H, *et al*., Scottish Stroke Care Audit. Implementing a simple care bundle is associated with improved outcomes in a national cohort of patients with ischemic stroke. *Stroke* 2015;46:1065–70.
19 Bravata DM, Wells CK, Lo AC, *et al*. Processes of care associated with acute stroke outcomes. *Arch Intern Med* 2010;170:804–10.
20 Bray BD, Ayis S, Campbell J, *et al*. Associations between the organisation of stroke services, process of care, and mortality in England: prospective cohort study. *BMJ* 2013;346:f2827.
21 Middleton S, McElduff P, Ward J, *et al*., QASC Trialists Group. Implementation of evidence-based treatment protocols to manage fever, hyperglycaemia, and swallowing dysfunction in acute stroke (QASC): a cluster randomised controlled trial. *Lancet* 2011;378:1699–706.
22 Masrur S, Smith EE, Saver JL, *et al*. Dysphagia screening and hospital-acquired pneumonia in patients with acute ischemic stroke: findings from Get with the Guidelines—Stroke. *J Stroke Cerebrovasc Dis* 2013;22:e301–9.
23 Donovan NJ, Daniels SK, Edmiaston J, *et al*., American Heart Association Council on Cardiovascular Nursing and Stroke Council. Dysphagia screening: state of the art. Invitational conference proceeding from the state-of-the-art nursing symposium, international stroke conference 2012. *Stroke* 2013;44:e24–31.
24 Stroke Quality Standards. National Institute for Health and Care Excellence 2010. http://www.nice.org.uk/guidance/qs2 URL (accessed 10 Jun 2015).
25 Lindsay P, Furie KL, Davis SM, *et al*. World Stroke Organization global stroke services guidelines and action plan. *Int J Stroke* 2014;9(Suppl A100):4–13.

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