

Pneumonia in acute stroke patients fed by nasogastric tube

R Dziewas, M Ritter, M Schilling, C Konrad, S Oelenberg, D G Nabavi, F Stögbauer, E B Ringelstein, P Lüdemann

J Neurol Neurosurg Psychiatry 2004;**75**:852–856. doi: 10.1136/jnnp.2003.019075

See end of article for authors' affiliations

Correspondence to:
Dr Rainer Dziewas,
Department of Neurology,
University Hospital of
Münster, Albert-
Schweitzer-Strasse 33,
48129 Münster, Germany;
dziewas@uni-muenster.de

Received 19 May 2003
In revised form
25 August 2003
Accepted
10 September 2003

Background: Aspiration pneumonia is the most important acute complication of stroke related dysphagia. Tube feeding is usually recommended as an effective and safe way to supply nutrition in dysphagic stroke patients.

Objective: To estimate the frequency of pneumonia in acute stroke patients fed by nasogastric tube, to determine risk factors for this complication, and to examine whether the occurrence of pneumonia is related to outcome.

Methods: Over an 18 month period a prospective study was done on 100 consecutive patients with acute stroke who were given tube feeding because of dysphagia. Intermediate outcomes were pneumonia and artificial ventilation. Functional outcome was assessed at three months. Logistic regression and multivariate regression analyses were used, respectively, to identify variables significantly associated with the occurrence of pneumonia and those related to a poor outcome.

Results: Pneumonia was diagnosed in 44% of the tube fed patients. Most patients acquired pneumonia on the second or third day after stroke onset. Patients with pneumonia more often required endotracheal intubation and mechanical ventilation than those without pneumonia. Independent predictors for the occurrence of pneumonia were a decreased level of consciousness and severe facial palsy. The NIH stroke scale score on admission was the only independent predictor of a poor outcome.

Conclusions: Nasogastric tubes offer only limited protection against aspiration pneumonia in patients with dysphagia from acute stroke. Pneumonia occurs mainly in the first days of the illness and patients with decreased consciousness and a severe facial palsy are especially endangered.

Dysphagia is an important complication of acute stroke. Abnormal lip closure, lingual incoordination, and delayed or absent triggering of the swallowing reflex may lead to a disturbance of both the oral and the pharyngeal stage of swallowing. Frequently encountered consequences are incomplete oral clearance, pharyngeal pooling, regurgitation, and aspiration.^{1–5} In the acute stage of the illness, dysphagia is found in up to 50% of stroke patients, depending on the timing of the assessment, the diagnostic methods used, and the case mix.^{4–8} Dysphagic symptoms resolve in most patients within a week to a month and persist in only a small number of subjects beyond six months.^{6–8,9}

Aspiration with the possible consequence of aspiration pneumonia is the most important acute complication of dysphagia, affecting up to one third of dysphagic patients.^{4–6} Besides a proper diagnostic assessment and an early start to rehabilitative efforts, tube feeding is usually recommended as an effective and safe way of supplying nutrition in these patients during the first two weeks.^{10–13} In a recent study on patients who had suffered a stroke more than six months earlier, Nakajoh *et al* found that the frequency of pneumonia was significantly higher in dysphagic patients with oral feeding than with tube feeding.¹⁴ However, owing to fundamental differences of the clinical features in acute and chronic stroke, observations in post-acute stroke patients should not be extrapolated to patients in the acute stage of the disease.

The present investigation was conducted with the following aims: first, to estimate the frequency of pneumonia in acute stroke patients fed by nasogastric tubes; second, to determine risk factors for this complication; and third, to examine whether the occurrence of pneumonia is related to outcome.

METHODS

Over an 18 month period we included in this study 100 consecutive patients referred to our stroke unit. Our department provides a neurological service for about 500 000 inhabitants aged over 16 years in the area around Münster. All patients presented within 24 hours of the onset of acute stroke and were provided with a nasogastric tube because of dysphagia (for definition see below). Exclusion criteria were admission more than 24 hours after stroke onset, oral feeding from day 0, coma on admission, and endotracheal intubation on admission. The study protocol was approved by the local ethics committee. All patients underwent unenhanced computed tomography or magnetic resonance imaging of the brain.

The clinical severity of the stroke was assessed on the day of admission using the National Institutes of Health stroke scale (NIHSS). For further analysis, the items "level of consciousness," "facial palsy," "dysarthria," "best language," "extinction or inattention (neglect)," and a sum score of "motor arm" and "motor leg," reflecting the degree of a hemiparesis, were selected a priori. These variables were dichotomised into severe deficits, corresponding to individual NIHSS scores of ≥ 2 , and non-severe deficits, corresponding to NIHSS scores of ≤ 1 . In case of the sum score for hemiparesis we chose a cut off value of ≥ 6 to indicate a severe deficit. All the clinicians involved had been trained in applying this scale with the help of a suitable teaching video.

The patients' ability to swallow was assessed clinically on the day of admission by an experienced speech pathologist

Abbreviations: MRS, modified Rankin scale; NIHSS, National Institutes of Health stroke scale

(SO) or by the specially trained physicians involved in this study, using the following two standardised tests:

The swallowing provocation test was applied in all patients. This test is generally used to evaluate the swallowing reflex. The test requires the injection of 0.4 ml (first step) and, if necessary, 2.0 ml (second step) of distilled water into the suprapharynx through a small nasal catheter. As suggested by Teramoto and colleagues, this test was judged to be normal if the latency of swallowing after either of the water injections was less than three seconds.^{15 16}

The water swallowing test assessed the patient's ability to drink 5 ml (first step) and 50 ml (second step) of water.¹⁷ Stuporose patients (that is, those with an NIHSS score of 2 for the item "level of consciousness") were not evaluated with this test. Subjects who drank the water without cough or wet/hoarse voice were considered normal.

On the day of admission, a nasogastric tube (flexible silicon tube with inner diameter of either 14 mm or 16 mm) was inserted in all patients in whom either of the above tests yielded abnormal results. Patients who were initially stuporose received a nasogastric tube if they had a pathological swallowing provocation test. Tube positioning was checked by bubbling, aspiration, and chest radiograph.

During the first 24 hours the nasogastric tube was used for gastric emptying. Tube feeding was started not earlier than 24 hours after admission. The indication for tube feeding was re-evaluated daily. We noted the time when the nasogastric tube was removed and oral feeding was started.

Intermediate outcomes were pneumonia and artificial ventilation. The patients were examined daily throughout their hospital stay (stroke unit and normal ward) for the presence of pneumonia. The diagnosis was based on three or more of the following variables: fever ($>38^{\circ}\text{C}$), productive cough with purulent sputum, abnormal respiratory examination (tachypnoea (>22 breaths/min), tachycardia, inspiratory crackles, bronchial breathing), abnormal chest radiograph, arterial hypoxaemia ($\text{PO}_2 <9.3$ kPa), and isolation of a relevant pathogen (positive Gram stain or culture).⁴ If endotracheal intubation and artificial ventilation became necessary, the suspected reason was noted. Pneumonia acquired after endotracheal intubation was not considered for further analysis.

Functional outcome was assessed at three months by a standardised interview, using the modified Rankin scale (mRS).¹⁸ An mRS score of ≤ 1 was classified as a good outcome, a score of 2 or 3 as a moderate outcome, and a score of ≥ 4 as a poor outcome.

Crude associations between the occurrence of pneumonia and each of the epidemiological and clinical variables were assessed with either the χ^2 test (for categorical variables) or the Mann–Whitney U test (for quantitative data). Time to the occurrence of pneumonia was analysed by the Kaplan–Meier product limit technique. Logistic regression analysis was applied to identify clinical variables significantly associated with the occurrence of pneumonia. To identify predictors of a poor outcome, multivariate regression analysis was carried out, which included the variables age, sex, NIHSS score, endotracheal intubation, and duration of nasogastric tube feeding.

RESULTS

During the study period, 527 patients (216 women, 311 men; mean (SD) age, 63.0 (15.8) years) were treated on our stroke unit. Of these, 100 were included in the study. In two patients with dysphagia the placement of a nasogastric tube proved to be impossible, and they were not included in the study. Pneumonia was diagnosed in 44 of the 100 patients. Pneumonia occurred in all these patients while the tube was in situ. The mean age of the study patients was 68.8 years,

and there were no significant differences between the pneumonia and non-pneumonia groups. Both groups featured more men than women (table 1). Mean follow up during the initial hospital stay (stroke unit and normal ward) was not significantly longer for the pneumonia group than for the non-pneumonia group. Four patients in the non-pneumonia group and three in the pneumonia group had a follow up of less than 10 days (three patients for five days, one for six days, two for seven days, and one for eight days). Lesion location was similar in both groups. Overall, more than twice as many patients had a left hemisphere stroke than a right hemisphere stroke (57 v 25 patients); brain stem stroke was present in 12 patients, and six had suffered from an intracerebral haemorrhage.

Twelve patients received 0.9 mg of recombinant tissue plasminogen activator/kg body weight. Ninety four patients were treated with intravenous heparin to prevent secondary stroke by doubling the apparent partial thrombin time for at least 24 hours, and generally until a source of cardiac embolism had been ruled out. Decompressive hemicraniectomy was undertaken in seven patients.

Most patients acquired pneumonia on the second or third day after the onset of stroke (median 2 days, mean 2.4, range 0 to 9). The resulting Kaplan–Meier curve (fig 1) was therefore censored for a follow up period shorter than 10 days in the group of patients who had not acquired pneumonia already.

As measured by the NIHSS score, patients who acquired pneumonia were significantly more severely affected than patients without pneumonia. Univariate analysis established an association between the occurrence of pneumonia and the clinical variables "decreased consciousness," "facial palsy," "hemiparesis," and "aphasia" (table 1).

As inferred from logistic regression analysis, independent factors in the occurrence of pneumonia were the NIHSS items "consciousness" and "facial palsy" (table 2). The duration of nasogastric tube placement varied greatly between the two groups. While patients without pneumonia had a nasogastric tube inserted for only two to three days (median 2 days, mean 4.9 (4.8)), the nasogastric tube was removed in patients with intercurrent pneumonia only after a median of 15 days (mean 16.3 (7.2) days; $p < 0.001$).

Endotracheal intubation and mechanical ventilation became necessary in three patients without pneumonia and in 15 with pneumonia ($p < 0.001$). The indications for endotracheal intubation were deterioration of consciousness ($n = 11$), primary respiratory failure ($n = 6$), and severe congestive heart failure ($n = 1$).

Outcome was evaluated in 87 patients; 13 patients could not be contacted. Seventeen patients made a good recovery, 30 had a moderate outcome, and 40 had a poor outcome, seven of whom had died (table 3). Patients with a good or moderate outcome more often had brain stem infarction than patients with a poor outcome. Patients with a poor outcome had a lower NIHSS score on admission, more often acquired intercurrent pneumonia, and more often needed endotracheal intubation and artificial ventilation. The duration of nasogastric tube placement was longer in patients with a poor outcome than in either of the other outcome groups. Multivariate regression analysis showed the NIHSS score on admission to be the only independent predictor of a poor outcome.

DISCUSSION

In our cohort of acute stroke patients needing tube feeding because of dysphagia, the incidence of pneumonia was 44%. At first glance, this number seems surprisingly large compared with other studies. Thus Mann *et al.*,⁴ Gordon *et al.*,⁸ and Hilker *et al.*¹⁹ found evidence of pneumonia in only

Table 1 Epidemiological features, lesion location, clinical features, and outcome data stratified by the occurrence of pneumonia

	No pneumonia (n = 56)	Pneumonia (n = 44)	p Value
Epidemiological features			
Age (years)	67.9 (12.9)	70.0 (10.5)	NS
M/F	33/23	28/16	NS
Follow up during initial hospital stay (days)	18.1 (9.2)	19.1 (9.2)	NS
Lesion location			
Middle cerebral artery, left	30 (54%)	23 (52%)	NS
Middle cerebral artery, right	15 (27%)	10 (23%)	NS
Middle cerebral artery + anterior cerebral artery, left	1 (2%)	3 (7%)	NS
Brain stem	8 (14%)	4 (9%)	NS
Internal capsule haemorrhage, left	1 (2%)	2 (3%)	NS
Internal capsule haemorrhage, right (%)	1 (2%)	2 (3%)	NS
Clinical features			
NIHSS score	10.3 (5.1)	17.4 (4.9)	p<0.001
Decreased consciousness	2 (4%)	19 (43%)	p<0.001
Facial palsy	18 (30%)	29 (66%)	p<0.001
Hemiparesis	21 (38%)	26 (59%)	p<0.05
Dysarthria	11 (20%)	14 (32%)	NS
Aphasia	14 (25%)	24 (55%)	p<0.01
Neglect	8 (14%)	8 (18%)	NS
Endotracheal intubation	3 (5%)	15 (34%)	p<0.001
Nasogastric tube duration (days)	4.9 (4.8)	16.3 (7.2)	p<0.001
Outcome			
	n = 48	n = 39	
Good	13 (27%)	4 (10%)	
Moderate	23 (48%)	7 (18%)	p<0.001
Bad	12 (25%)	28 (72%)	

Data are given as mean (SD) or n (%).

Numbers of NIHSS items refer to severe clinical findings (corresponding to individual NIHSS scores of 2 or more). In case of "hemiparesis," numbers of the NIHSS items "motor arm" and "motor leg" were added; hemiparesis was counted as severe if the sum score was ≥ 6 .

F, female; M, male; NIHSS, National Institutes of Health stroke scale.

20%, 13%, and 21% of patients, respectively, and Smithard *et al* observed pneumonia in 33% of their patients.⁶ However, the discrepancies between these numbers are clearly explained by selection bias, as the frequencies given in the first three studies referred to unselected stroke patients, and Smithard *et al*—although referring to dysphagic patients—excluded those with a decreased level of consciousness.

In our study, a decreased level of consciousness and facial palsy turned out to be independent clinical variables predictive of pneumonia. The first of these has already been identified as a major risk factor for pneumonia by others, and is not specific for stroke patients.^{20–22} Pathophysiologically, a decreased level of consciousness is known to lead to attenuation of protective reflexes and to worsening of the coordination of breathing and swallowing,^{23–25} thereby predisposing to aspiration independent of the underlying disease. In contrast, the correlation between a severe facial palsy and pneumonia is a new and stroke specific observation. We hypothesise that it is not the facial palsy itself that

increases the risk of aspiration. More probably, a severe facial palsy indicates concomitant paresis of the tongue and other oropharyngeal muscles involved in swallowing. Although this point has to be examined more closely in future investigations, we suggest that a severe facial palsy is interpreted as a marker for a substantially increased risk of aspiration.

In our study, patients with a poor functional outcome after three months had acquired an intercurrent pneumonia significantly more often than those with a good or moderate outcome (70%, *v* 23% and 24%, respectively). Despite this obvious correlation, only the NIHSS score on admission proved to be an independent predictor of outcome. This reflects the limited sample size, the investigation of only a small number of possible risk factors, and the preselection of stroke patients with dysphagia. Other studies of unselected stroke patients have identified a broader range of independent prognosticators, such as dysphagia, impaired consciousness, urinary incontinence, and raised body temperature.^{6, 26–27}

The question remains as to what conclusions should be drawn from this study for the care of patients with acute stroke. Obviously, a 44% incidence of pneumonia in acute stroke patients fed by nasogastric tube warrants changes to our present therapeutic strategy. According to current

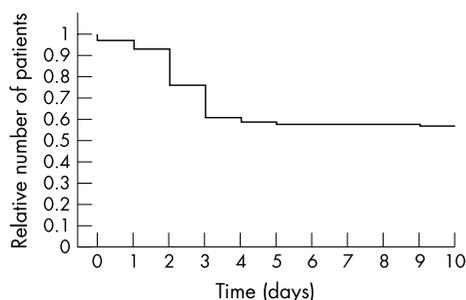


Figure 1 Kaplan–Meier survival curve showing relative number of patients (y axis) free of pneumonia. The x axis gives the time in days. The Kaplan–Meier curve was censored for a follow up of less than 10 days in patients without pneumonia.

Table 2 Clinical risk factors for the development of pneumonia: results of logistic regression analysis

	Odds ratio	p Value
Consciousness	7.4 (2.9 to 18.4)	<0.001
Facial palsy	3.1 (1.0 to 9.3)	<0.05
Hemiparesis	0.6 (0.4 to 1.1)	0.08
Aphasia	2.1 (0.7 to 6.1)	0.18
Dysarthria	1.4 (0.6 to 3.4)	0.47
Neglect	1.2 (0.4 to 3.8)	0.72

Table 3 Epidemiological features, lesion location, and clinical features, stratified by outcome

Feature	Good or moderate outcome (n = 47)	Poor outcome (n = 40)	p Value
<i>Epidemiological features</i>			
Age (years)	66.2 (13.6)	70.1 (9.1)	NS
M/F	32/15	23/17	NS
<i>Lesion location</i>			
Middle cerebral artery, left	25 (53%)	21 (53%)	NS
Middle cerebral artery, right	10 (21%)	12 (30%)	NS
Middle cerebral artery + anterior cerebral artery	0 (0%)	3 (8%)	NS
Brain stem	11 (23%)	0 (0%)	p<0.01
Internal capsule haemorrhage, left	0 (0%)	3 (8%)	NS
Internal capsule haemorrhage, right	1 (2%)	1 (3%)	NS
<i>Clinical features</i>			
Pneumonia	11 (23%)	28 (70%)	p<0.001
NIHSS (points)	9.4 (5.1)	18.0 (4.0)	p<0.001
Intubation	4 (9%)	11 (28%)	p<0.05
Nasogastric tube duration (days)	6.3 (6.2)	14.9 (7.0)	p<0.001

Values are mean (SD) or n (%).
F, female; M, male; NIHSS, National Institutes of Health stroke scale.

knowledge, the early insertion of a gastrostomy tube does not appear to be superior to feeding through a nasogastric tube. As with nasogastric tubes, gastrostomy tubes do not offer protection from colonised oral secretions²⁸ and are known to increase gastro-oesophageal reflux.^{29–30} Thus, with the exception of a study by Norton *et al.*,³¹ others have found similar aspiration rates with gastrostomy and nasogastric tube feeding.^{32–34}

To reduce the rate of aspiration pneumonia in acute stroke patients, one might consider the use of early protective endotracheal intubation in high risk patients—that is, those with severely decreased consciousness and severe facial palsy. There are at least three arguments in favour of such a strategy. First, according to our findings pneumonia is a complication of the acute stage of the illness, occurring as early as the second day after stroke onset. Thus one might reasonably expect that a short interval of endotracheal intubation and mechanical ventilation would be sufficient to get the patient past the most critical period. Second, even if one refrains from endotracheal intubation initially, it will anyway become necessary later in a substantial number of patients: in our study, more than one third of patients with pneumonia required endotracheal intubation and mechanical ventilation, 40% of them because of pneumonia related respiratory failure. Finally, although the outcome of stroke patients needing mechanical ventilation is generally considered to be unfavourable,^{35–38} broadening the indications for this intervention along the lines suggested could change this assessment.

However, there are some important objections to this approach. Endotracheal intubation itself is not free of risk and may lead to injury to the teeth, the vocal cords, and the trachea. Additionally, the sedation and analgesia required for endotracheal intubation may cause a substantial fall in arterial blood pressure, thereby endangering the penumbral tissue in acute stroke patients. Furthermore, the protection against aspiration afforded by a cuffed endotracheal tube may not be complete. Although Treolar and Stechmiller had found no evidence of aspiration in their study,³⁹ others reported to the contrary.^{40–41} Finally, endotracheal intubation and sedation seriously interfere with early rehabilitation, which has been shown to be important for the outcome of acute stroke patients.⁴²

There are two methodological limitations of our study that may decrease the impact of the results presented. First, the assessment of pneumonia was not made blind to the

patients' baseline characteristics. This may have introduced an expectation bias. Second, more than 10% of the patients were lost to follow up at three months. Although the proportion of patients lost to follow up was similar for the pneumonia and non-pneumonia groups (11% *v* 14%), a significant effect on our results cannot be ruled out.

Conclusions

Overall, feeding tubes offer only limited protection against aspiration pneumonia in patients with dysphagia caused by acute stroke. Pneumonia occurs mainly in the first days of the illness, and patients with a decreased level of consciousness and a severe facial palsy are especially endangered. Risks and benefits of more protective strategies such as early endotracheal intubation have to be established in randomised controlled trials.

Authors' affiliations

R Dzewas, M Ritter, M Schilling, C Konrad, S Oelenberg, D G Nabavi, F Stögbauer, E B Ringelstein, P Lüdemann, Department of Neurology, University Hospital of Münster, Münster, Germany

Competing interests: none declared

REFERENCES

- Daniels SK, Brailey K, Foundas AL. Lingual discoordination and dysphagia following acute stroke: analyses of lesion localization. *Dysphagia* 1999;14:85–92.
- Daniels SK, McAdam CP, Brailey K, *et al.* Clinical assessment of swallowing and prediction of dysphagia severity. *Am J Speech Lang Pathol* 1997;6:17–24.
- Smithard DG, O'Neill PA, Park C, *et al.* Can bedside assessment reliably exclude aspiration following acute stroke? *Age Ageing* 1998;27:99–106.
- Mann G, Dip PG, Hankey GJ, *et al.* Swallowing function after stroke. *Stroke* 1999;30:744–8.
- Robbins J, Levine RL, Maser A, *et al.* Swallowing after unilateral stroke of the cerebral hemisphere. *Arch Phys Med Rehabil* 1993;74:1295–300.
- Smithard DG, O'Neill PA, Park C, *et al.* Complications and outcome after acute stroke – does dysphagia matter? *Stroke* 1996;27:1200–4.
- Horner J, Massey EW, Riski JE, *et al.* Aspiration following a stroke: clinical correlates and outcome. *Neurology* 1988;38:1359–62.
- Gordon C, Hewer RL, Wade DT. Dysphagia in acute stroke. *BMJ* 1987;295:411–14.
- Barer DH. The natural history and functional consequences of dysphagia after hemispheric stroke. *J Neurol Neurosurg Psychiatry* 1989;52:236–41.
- Finestone HM. Safe feeding methods in stroke patients. *Lancet* 2000;355:1662–3.
- Smithard DG. Assessment of swallowing following acute stroke. *Stroke Rev* 2002;6:7–10.
- Smithard DG. Gag reflex has no role in ability to swallow. *BMJ* 1996;312:972.

- 13 **Alberts MJ**, Horner J. Dysphagia and aspiration syndromes. In: Bogousslavsky J, Caplan L, eds. *Stroke syndromes*. Cambridge: Cambridge University Press, 1995:213–22.
- 14 **Nakajoh K**, Nakagawa T, Sekizawa K, et al. Relation between the incidence of pneumonia and protective reflexes in post-stroke patients with oral or tube feeding. *J Intern Med* 2000;**247**:39–42.
- 15 **Teramoto S**, Matsuse T, Fukuchi Y. Simple two-step swallowing provocation test for elderly patients with aspiration pneumonia. *Lancet* 1999;**353**:1243.
- 16 **Teramoto S**, Fukuchi Y. Detection of aspiration and swallowing disorder in older stroke patients: simple swallowing provocation test versus water swallowing test. *Arch Phys Med Rehabil* 2000;**81**:1517–19.
- 17 **DePippo K**, Holas MA, Reding MJ. Validation of the 3-oz water swallow test for aspiration following acute stroke. *Arch Neurol* 1992;**49**:1259–61.
- 18 **Wade DT**. Rankin scale. In: Wade DT, ed. *Measurement in neurological rehabilitation*. Oxford: Oxford University Press, 1992:238–9.
- 19 **Hilker R**, Poetter C, Findeisen N, et al. Nosocomial pneumonia after acute stroke—implications for neurological intensive care medicine. *Stroke* 2003;**34**:975–81.
- 20 **Finegold SM**. Aspiration pneumonia. *Rev Infect Dis* 1991;**13**(suppl 9):737–42.
- 21 **Huxley EJ**, Viroslav J, Gray WR, et al. Pharyngeal aspiration in normal adults and patients with depressed consciousness. *Am J Med* 1977;**64**:564–8.
- 22 **Joshi N**, Localio R, Hamory B. A predictive risk index for nosocomial pneumonia in the intensive care unit. *Am J Med* 1992;**93**:135–42.
- 23 **Nishino T**, Takizawa K, Yokokawa N, et al. Depression of the swallowing reflex during sedation and/or relative analgesia produced by inhalation of 50% nitrous oxide in oxygen. *Anesthesiology* 1987;**67**:995–8.
- 24 **Nishino T**, Hiraga K. Coordination of swallowing and respiration in unconscious subjects. *J Appl Physiol* 1991;**70**:988–93.
- 25 **Moulton C**, Pennycook AG. Relation between Glasgow coma score and cough reflex. *Lancet* 1994;**343**:1261–2.
- 26 **Wang Y**, Lim LLY, Levi C, et al. A prognostic index for 30-day mortality after stroke. *J Clin Epidemiol* 2001;**54**:766–73.
- 27 **Reith J**, Jorgensen HS, Pedersen PM, et al. Body temperature in acute stroke: relation to stroke severity, infarct size, mortality, and outcome. *Lancet* 1996;**347**:422–5.
- 28 **Marik PE**. Aspiration pneumonitis and aspiration pneumonia. *N Engl J Med* 2001;**344**:665–71.
- 29 **Cole MJ**, Smith JT, Molnar C, et al. Aspiration after percutaneous gastrostomy: assessment by Tc-99m labeling of the enteral feed. *J Clin Gastroenterol* 1987;**9**:90–5.
- 30 **Balan KK**, Vinjamuri S, Malby P, et al. Gastroesophageal reflux in patients fed by percutaneous endoscopic gastrostomy (PEG): Detection by a simple scintigraphic method. *Am J Gastroenterol* 1998;**93**:946–9.
- 31 **Norton P**, Homer-Ward M, Long RG, et al. A randomised prospective comparison of percutaneous endoscopic gastrostomy and nasogastric feeding after acute dysphagic stroke. *BMJ* 1996;**312**:13–16.
- 32 **Hasset JM**, Sunby C, Flint LM. No elimination of aspiration pneumonia in neurologically disabled patients with feeding gastrostomy. *Surg Gynecol Obstet* 1988;**167**:383–8.
- 33 **Baeten C**, Hoefnagels J. Feeding via nasogastric tube or percutaneous endoscopic gastrostomy. A comparison. *Scand J Gastroenterol Suppl* 1992;**194**:95–8.
- 34 **Park RH**, Allison MC, Lang J, et al. Randomised comparison of percutaneous endoscopic gastrostomy and nasogastric tube feeding in patients with persisting neurological dysphagia. *BMJ* 1992;**304**:1406–9.
- 35 **Mayer SA**, Copeland D, Bernardini GL, et al. Cost and Outcome of mechanical ventilation for life-threatening stroke. *Stroke* 2000;**31**:2346–53.
- 36 **Berrouschof J**, Rössler A, Köster J, et al. Mechanical ventilation in patients with hemispheric ischemic stroke. *Crit Care Med* 2000;**28**:2956–61.
- 37 **Bushnell CD**, Phillips-Bute BG, Laskowitz DT, et al. Survival and outcome after endotracheal intubation for acute stroke. *Neurology* 1999;**52**:1374–81.
- 38 **Santoli F**, De Jonghe B, Hayon J, et al. Mechanical ventilation in patients with acute ischemic stroke: survival and outcome at one year. *Intensive Care Med* 2001;**27**:1141–6.
- 39 **Treolar DM**, Stechmiller J. Pulmonary aspiration in tube-fed patients with artificial airways. *Heart Lung* 1984;**13**:667–71.
- 40 **Pavin EG**, Van Nimwegen D, Hornbein TF. Failure of a high compliance, low pressure cuff to prevent aspiration. *Anesthesiology* 1975;**42**:216–19.
- 41 **Seegobin RD**, von Hassel GL. Aspiration beyond endotracheal cuffs. *Can Anaesth Soc J* 1986;**33**:273–9.
- 42 **Indredavik B**, Bakke F, Slordahl SA, et al. Treatment in a combined acute and rehabilitation stroke unit: which aspects are most important? *Stroke* 1999;**30**:917–23.