Is overnight tube feeding associated with hypoxia in stroke?

SIR—Dysphagia is found in about 45% of patients who have had a stroke [1], and nasogastric (NG) or percutaneous endoscopic gastrostomy (PEG) tubes are often used for enteral nutrition. The insertion of an NG or PEG tube in stroke patients has been associated with a modest drop in arterial oxygen saturation (SaO₂) [2]. In a study of NG tube-fed patients with chronic obstructive airways disease, there was a small drop in SaO₂ associated with feeding [3]. Reduced SaO₂ during swallowing and oral feeding has also been demonstrated in stroke [4–6]. However, it is not known whether tube feeding in stroke patients leads to significant episodes of arterial desaturation and whether there is a case for routine pulse oximetry or supplemental oxygen during tube feeding. A study that compared SaO₂ during a 30 min period of tube feeding with orally fed stroke controls found a very small but statistically significant change in SaO₂ in tube-fed patients [7].

The detection of hypoxia following a stroke is of great importance because a reduced SaO₂ is thought to be associated with worse outcomes after a stroke [8, 9]. Stroke patients have been shown to have lower baseline oxygen saturations compared to controls and are at risk of further hypoxia [6, 8, 10]. Several factors such as weakness of respiratory muscles [11], altered central regulation of respiration [12], sleep apnoea [13], aspiration [4, 6], chest infections [10, 14] and pulmonary emboli [10, 14] contribute to post-stroke hypoxia, which can be intermittent and often worse at night. Overnight pulse oximetry during sleep in stroke patients shows an SaO₂ about 1% less than that of controls [15]. Body posture can also influence SaO₂, and a 1% difference in SaO₂ between the supine and upright positions has been demonstrated [16]. Other studies, however, have had more inconsistent results, and no difference has been shown between the supine position and lying on the right or left side [8] or in oximetry readings between the hemiparetic and non-hemiparetic side [17]. Non-stroke factors such as cardiovascular or respiratory problems and body mass index can also influence SaO₂. This study was designed to determine whether overnight tube feeding is associated with hypoxia in stroke patients who are not hypoxic at baseline and not on supplemental oxygen.

Methods

This was a prospective, observational study of consecutive dysphagia acute stroke patients fed by NG or PEG tubes in a stroke unit. Patients acted as their own controls, and repeated oximetry readings were taken under the same conditions with and without tube feeding. Brain imaging was performed, and both ischaemic and haemorrhagic strokes were eligible. The stroke was classified according to the Oxfordshire Community Stroke Project classification system as a total anterior circulation stroke (TACS), partial anterior circulation stroke (PACS), lacunar stroke (LACS) or posterior circulation stroke (POCS) [18]. The modified Rankin scale was used as a measure of disability [19, 20]. Demographic data and previous medical problems were recorded. All patients were assessed by speech and language therapists, and patients who were unsafe for oral feeding had NG tubes initially and some went on to have PEG tubes. Patients who were terminally ill or already on supplemental oxygen were excluded. Patients who were confused or restless and who tended to displace their feeding tubes were also excluded. The study had the approval of the local ethics committee, and consent was written, witnessed oral or with permission from next of kin in a few cases.

Recordings were taken twice for each patient on two consecutive nights with a finger probe. The hand on which the sensor was placed was usually the paretic side [17] or randomly chosen in those without hemiparesis. The probe was repositioned once during the recording to reduce the likelihood of probe burns. The readings were taken in the same, semi-recumbent position on both occasions. On one night, the tube was kept in situ during the recording but not used for feeding. Monitoring was commenced at midnight and continued till 8 am. On the other night, tube feeding was commenced at midnight at a rate of 75–100 ml/h with continuous monitoring. To reduce bias, the night on which the patient received tube feeding was randomly selected by a randomisation method comprising sealed envelopes.

The instrument used was a Ohmeda 3700 pulse oximeter (Datex-Ohmeda, Hertfordshire, UK) with a downloadable software package that allowed automatic recording of SaO₂ levels (with mean, median and standard deviations), pulse rate, a graphical representation of SaO₂ levels and percentage of readings with an SaO₂ level of <93%. Artefacts were identified by concurrent anomalies in the graphical trace and pulse recording and were excluded from the recording. Hypoxia was defined as an SaO₂ level of <90%. Because the mean duration of actual recordings was ~6 h, results for T90 (the time the subjects spent with an SaO₂ <90%) [15] were corrected to a standard 6 h period with the formula: T90c (in minutes) = (T90 in minutes/actual recording time in minutes) × 360 [15]. The minimum sample size needed to detect a change in the SaO₂ of 1% or more at a significance level of 5% and power of 90% was calculated to be 16 based on data from an earlier study [7]. Data were assessed for normality and the paired sample t-test used in all instances using the statistical package SPSS.

Results

Of 165 patients admitted to the stroke unit during January to September 2005, ∼32 were fed by tube for >7 days. There were several exclusions because of pre-existing hypoxia, terminal prognosis and frequent NG tube displacement. Sixteen tube-fed patients consented to enter the study during this period. Baseline characteristics are summarised in Table 1. The mean time to assessment from stroke onset was 25.19 days.

Results of pulse oximetry are summarised in Table 2. The mean duration of recording was 6.4 h (range 5–8 h). The mean SaO₂ for patients not fed was 94.9% (SD 1.11)
Results of pulse oximetry

<table>
<thead>
<tr>
<th>Readings with</th>
<th>Readings with</th>
<th>( P ) values</th>
</tr>
</thead>
</table>
| mean \( \text{SaO}_2 \) % (SD)
| no feed \( (n = 16) \) | feed \( (n = 16) \) |
| Mean \( \text{SaO}_2 \) % (SD) | 94.9 (1.11) | 94.3 (1.03) | 0.09* |
| Percentage of readings with \( \text{SaO}_2 \) < 93% (SD) | 14.2 (11.66) | 19.9 (15.56) | 0.17* |
| Mean T90c in minutes (SD)* | 8.1 (9.35) | 18.8 (21.26) | 0.08* |

*The number of minutes spent with \( \text{SaO}_2 \) < 90% corrected to a standard period of 6 h.
*Paired sample \( t \)-test.

Table 1. Baseline characteristics of study patients

| Subjects \( (n = 16) \) | \( \text{Mean age in years (SD)} \) | \( \text{Male sex (\%)} \) | \( \text{Ischaemic stroke (\%)} \) | \( \text{Intracerebral haemorrhage (\%)} \) | \( \text{Feeding tube} \) | \( \text{POCS} \) | \( \text{LACS} \) | \( \text{PACS} \) | \( \text{PACS, partial anterior circulation stroke} \) | \( \text{LACS, lacunar stroke} \) | \( \text{OCSP, Oxfordshire Community Stroke Project} \) | \( \text{NG, nasogastric} \) | \( \text{PEG, percutaneous} \) |
|---------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|
| Mean age in years (SD) | 80.19 (5.56) | 37.5 | 13 (81.3) | 3 (18.8) | NG tube | 12 | PEG tube | 4 | OCSP classification (\%) | TACS 6 (37.5) | PACS 7 (43.8) | LACS 2 (12.5) | POCS 1 (6.3) | Modified Rankin score (\%) | 1 3 (18.8) | 2 4 (25.0) | 5 9 (56.3) | Co-morbidities (\%) | Hypertension 12 (75) | Atrial fibrillation 4 (25.0) | Congestive cardiac failure 1 (6.3) | Chronic obstructive airways disease 3 (18.8) | Ischaemic heart disease 2 (12.5) | Diabetes 4 (25.0) | Previous stroke 5 (31.3) | POCS, posterior circulation stroke; TACS, total anterior circulation stroke. |

Discussion

There are several factors that can potentially contribute to hypoxia after a stroke, and there are reasons to suspect that tube feeding might be one of them. Reduced \( \text{SaO}_2 \) can adversely affect stroke outcomes though we do not know what constitutes a clinically significant degree of hypoxia very early after a stroke. It is normal practice to monitor oxygen saturations after a stroke and prescribe oxygen in patients with hypoxia. The aim of this study was simply to make an observation of oxygen saturations in overnight tube-fed patients to look for an association of feeding with hypoxia. Patients acted as their own controls, and the only variable that was different in the circumstances of the two paired readings was the infusion of the feed through the feeding tube. We believe that this method had the greatest chance of eliminating the large number of confounding factors that can influence \( \text{SaO}_2 \) after a stroke.

Even though the sample studied was small, it was adequate to detect a difference of 1% or greater in \( \text{SaO}_2 \) in the two sets of readings. No patients were hypoxic at baseline because patients with pre-existing hypoxia who required supplemental oxygen were excluded. Although there seemed to be a trend towards a lower \( \text{SaO}_2 \) in the tube-fed readings and a greater period of time spent with \( \text{SaO}_2 \) readings < 90%, this difference was small, not statistically significant and possibly because of chance alone. The night-time reduction in \( \text{SaO}_2 \) detected in our patients is in keeping with the demonstration of nocturnal hypoxia in an earlier study of acute stroke patients [15]. In this study, patients who were normoxaemic at baseline dropped their oxygen saturations by a mean of 1% as compared with non-stroke controls and about 25% had periods of hypoxia (\( \text{SaO}_2 \) < 90%) lasting half an hour or more [15]. In this small study, we have shown that tube feeding does not cause large (>1%) falls in oxygen saturation. Feeding \( \text{per se} \) is not an indication for routine pulse oximetry or supplemental oxygen. There was, however, a trend towards lower \( \text{SaO}_2 \) in patients fed over night. A larger study would be needed to conclusively determine whether tube feeding contributes to the hypoxic burden in stroke.

Key points

- Hypoxia during swallowing, oral feeding and feeding tube placement has been demonstrated in stroke patients.
- It is not known whether overnight tube feeding in stroke patients causes episodes of hypoxia.
- In this small study, we have shown that tube feeding does not cause large (>1%) falls in oxygen saturation.

Conflicts of interest

None.

Funding

No external funding.

Dipankar Dutta*, Tracy Wood, Rhys Thomas, Muhammad Asrar ul Haq
Gloucestershire Royal Hospital, Great Western Road, Gloucester GL1 3NN, UK
*To whom correspondence should be addressed
Tel: (+44) 08454 226321; Fax: (+44) 08454 226979;
Email: dipankar.dutta@glos.nhs.uk
Telephone assessment of cognitive function in adulthood: the Brief Test of Adult Cognition by Telephone

SIR—Increased understanding of cognitive function in normal ageing is of major importance for both theoretical advancement and practical public health reasons. Cognitive function plays a critical role in an individual’s ability to function independently across the lifespan, with substantial documented links to quality of life, morbidity [1], mortality [2] and dementia [3]. The Brief Test of Adult Cognition by Telephone (BTACT) addresses the need for a brief test that is sensitive to cognitive differences in normal ageing, including episodic verbal memory, working memory span and executive function [4], reasoning and speed of processing [5]. It includes tasks based on laboratory research as well as modified versions of well-documented psychometric tests that have been adapted for telephone administration with non-demented adults.

Although in-person testing is often the preferred mode, it is not always feasible; telephone testing offers advantages such as convenience, low expense and the opportunity to test a greater number of individuals, including those who cannot be tested in person. This method can reach a wide range of respondents who vary in physical mobility, health status and educational level.

Previous telephone testing has focused primarily on screening for dementia using instruments that are not sensitive to cognitive performance in normal healthy adults [6–8]. Nevertheless, the validity of telephone testing in normal ageing has been supported by other studies [9, 10], such as the HRS/AHEAD study [11], which reported no significant difference in performance between telephone and in-person tests [12, 13].

The BTACT extends the range of these previous studies in two important ways. First, the range of cognitive domains tested is extended beyond orientation and memory to include key abilities that are paramount in current theories of cognitive ageing. Second, the BTACT is appropriate for testing a wider population including well-functioning younger and middle-aged adults as well as older adults. This allows for sensitivity to individual differences in cognition that may be associated with a large array of biological, social, health and psychological factors [14].

Methods

The Basic BTACT battery requires <20 min to administer in person or by telephone, using paper-and-pencil scoring methods. The only equipment recommended for the Basic BTACT is a recording device (e.g. a tape recorder or computer) with a phone jack to record the interview. We also have developed an alternative form of the BTACT for repeated testing, available on our website (http://www.brandeis.edu/projects/lifespan). In addition, an optional task-switching test can assess attention and task-switching ability; this requires a computer for recording a digitised sound file.

The interviewer first screens to ensure that the participant can hear the materials clearly. To minimise cheating, we specifically ask participants not to write down anything during the test and to close their eyes to facilitate concentration.