Does Feeding Alter Arterial Oxygen Saturation in Patients With Acute Stroke?

Anne M. Rowat, RGN, BSc Joanna M. Wardlaw, BSc, MB ChB, FRCP, FRCR, MD
Martin S. Dennis, BS, MB ChB, FRCP, MD Charles P. Warlow, MB ChB, FRCP, MD

Background and Purpose—We measured arterial oxygen saturation (SaO₂) during eating in acute stroke patients to establish the frequency of any meal-related hypoxemia, which could further damage already vulnerable brain tissue.

Methods—Stroke patients (≥12 days from stroke onset) classified as “safe to feed orally” were compared with elderly hospitalized (for nonneurological causes) and young healthy controls. SaO₂ was measured noninvasively at the bedside by pulse oximetry continuously for 10 minutes before the patient ate a meal, during the meal, and for 10 minutes after completion of the meal.

Results—The median baseline SaO₂ was significantly lower in stroke patients (n = 106, 95.7%) than elderly (n = 50, 96.7%) or young control subjects (n = 20, 97.9%; P < 0.001). There was a small decrease in the median SaO₂ during eating in stroke and elderly patients (95.6%, P = 0.08, and 96.3%, P = 0.004, respectively) but not in young controls. Only stroke patients had a significantly lower median SaO₂ after completion of the meal (95.4%, P < 0.001). SaO₂ of ≤90% during and after eating occurred in 24% of stroke and 16% of elderly patients but not in young controls, and it was significantly more common in those who had SaO₂ of ≤90% during the baseline recordings (P = 0.003).

Conclusions—Eating a meal was associated with a small fall in median SaO₂ among stroke and elderly patients, but only in stroke patients did this persist for at least 10 minutes after eating. A quarter of stroke patients had episodes in which the SaO₂ fell to ≤90% saturation (ie, hypoxemia) during or after eating, although this rarely coincided exactly with swallowing and was more common in patients who also experienced desaturation during the baseline recordings. Further studies are required to establish whether these changes are clinically important. (Stroke. 2000;31:2134-2140.)

Key Words: feeding behavior ■ oximetry ■ oxygen ■ stroke, acute

It is generally agreed that hypoxemia should be avoided in the acute phase of stroke.¹ A fall in oxygenation could adversely affect the vulnerable ischemic brain lesion² and therefore reduce the chance of a good clinical outcome after stroke. Hypoxemia may increase during eating,³–⁵ and acute stroke patients may be specifically affected, because approximately one half have swallowing problems in the first weeks after the event and in many this may not be recognized.⁶,⁷ Previous investigations of patients with stroke have explored changes in arterial oxygen saturation (SaO₂), but only during a swallowing assessment performed by a speech and language therapist⁸ or during videofluoroscopy,⁹–¹¹ and then just as a method to detect clinically silent aspiration. However, these tests are somewhat artificial because the patients are tested with only small amounts of food of different consistencies under controlled conditions. Therefore, we aimed to determine whether the SaO₂ of acute stroke patients is influenced during swallowing for more natural purposes, such as eating a meal after being classified as “safe to feed orally.”

Subjects and Methods

Patients and Controls

During the study period (October 1, 1996, to August 30, 1998), we assessed all consecutive hospital admissions with first or recurrent ischemic or hemorrhagic stroke classified as “safe to feed orally” within 12 days of the onset to establish their eligibility for the study. We excluded patients if they had a subarachnoid hemorrhage; their disease was considered terminal and no feeding was given; further investigation later showed that the patient did not have a stroke; they were uncooperative during the observation period, making it difficult to obtain reliable physiological data; or they were unable to take food orally. Permission to participate was sought from all patients or their caregivers, and the study had the approval of the ethics committee.

A stroke physician examined all the patients and entered demographic and clinical data into the local registry, the Lothian Stroke Register (LSR). Stroke was defined according to the World Health Organization criteria.¹² The stroke was then classified, according to the Oxfordshire Community Stroke Project clinical classification system, as a total anterior circulation stroke (TACS); partial anterior circulation stroke (PACS); lacunar stroke (LACS), or posterior circulation stroke (POCS).¹³ The severity of the motor deficit affecting the arm and/or leg was described as either being mild (Medical Research Council [MRC] motor grade 4), moderate (MRC motor grade 3), or severe (MRC motor grade 0 to 2).

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From the Department of Clinical Neurosciences, University of Edinburgh, Western General Hospital, Crewe Road South, Edinburgh, UK.
Correspondence to Anne Rowat, Department of Clinical Neurosciences, University of Edinburgh, Western General Hospital, Crewe Road South, Edinburgh, EH4 2XU UK. E-mail annerowat@hotmail.com

Stroke is available at http://www.strokeaha.org
We also recruited a group of elderly hospitalized patients and young healthy subjects to investigate whether the magnitude of any change in \( \text{SaO}_2 \) during or after eating a meal was simply related to aging rather than stroke. The elderly hospitalized patients were under the care of physicians for investigation of various acute nonneurological medical problems. Elderly patients were excluded if they had previous history of stroke, transient ischemic attacks (TIAs), or swallowing problems. The young healthy subjects, who were recruited from staff working in the Department of Clinical Neurosciences, had no known medical problems at the time of the study or any relevant past history.

We estimated our sample size on the standard deviation of the fall in absolute \( \text{SaO}_2 \) of 2.69% after swallowing in stroke patients who were definitely aspirating in a previous study.\(^8\) This study also found that 3% was the minimum desaturation detectable after swallowing which could not simply be attributed to machine variability and which also could be clinically relevant.\(^8\) Therefore, we estimated that approximately 20 stroke patients would be needed to detect a within-group change in mean \( \text{SaO}_2 \) of 3% with a power of 90% at the 5% significance level.\(^14\)

### Equipment

Oxygen saturation was measured noninvasively by pulse oximetry with a Kontron 7250 (Kontron Instruments Ltd).\(^15\) The Kontron has been independently validated to have bias and precision estimates of \( \leq 3\% \pm 2\% \) between 70% to 100% saturation in healthy subjects.\(^16\) Measurements were taken at bedside, which was warm and dimly lit to minimize data collection errors.\(^15\) Young controls were studied under similar conditions in a quiet room in the hospital. Measurements were taken with a finger probe and secured with Velcro brand hook-and-loop fastening to shield it from ambient light.\(^15\) The hand on which the sensor was placed was randomized either between the paretic and nonparetic sides (in those with hemiparesis) or between the right and the left sides (in those without hemiparesis). The sensor was then attached to the tip of the patient’s middle finger after the skin was cleaned and warmed. The patient was instructed to keep the hand as still as possible and level with the heart.\(^17\) Random allocation of the sensor was not always possible, particularly if patients could feed themselves only with the nonparetic hand. In such cases, the finger sensor was placed on the paretic or nondominant hand (if the patient had no motor deficits), because this provided the greatest chance of reliable recordings (ie, least movement artifact). Motion artifact could also be recognized by false or erratic heart rate displays or by distorted photoplethysmographic waveforms.\(^15,17\) \( \text{SaO}_2 \) and heart rate measurements were displayed continually, updated every 8 seconds, on the Kontron monitor. Data from the Kontron were collected online with a microcomputer running software designed in-house.\(^18\) This system also allowed us to add text comments to the data at any time, so that the effect of significant events and artifact could be identified and edited during later analysis.

### Procedure

Recordings of \( \text{SaO}_2 \) were made during breakfast, lunch, or the evening meal, served at the patients’ bedside on the stroke ward. We made no attempt to standardize the meal for nutritional composition or caloric content, nor did we require the patients to complete the meal within a certain time period. Recordings were measured continually at baseline (before) for at least 10 minutes, during (until the meal was completed), and 10 minutes after eating the meal. Patients were observed throughout the recording period so that reasons for any discrepancies in the collected data could be identified (eg, patient restlessness, electrical faults, or detachment of the pulse oximeter probe) and edited before analysis.

### Statistical Analysis

All data analyses were performed with SPSS for Windows (version 7.5.1, SPSS Inc). The mean \( \text{SaO}_2 \) at baseline, during the meal, and after the meal was calculated for each patient. The between-patient data were not normally distributed and could not be easily transformed; therefore, we analyzed data with nonparametric tests for paired and unpaired data. Data calculated across patients are presented as the median and the interquartile range (IQR) of the mean \( \text{SaO}_2 \) (hereafter referred to as the median \( \text{SaO}_2 \)) for each period of observation. As in previous studies, significant desaturation was defined as a fall in \( \text{SaO}_2 \) of \( \geq 3\%\) and hypoxemia was defined as \( \text{SaO}_2 \) of \( \leq 90\% \) saturation.\(^3\) Appropriate statistical tests (specified in the text) were used for dichotomized variables. Statistical significance was taken at the 5% level.

### Results

During the study period, a total of 134 patients were seen (other stroke patients were admitted, but either had very mild strokes and so were discharged quickly, or the monitoring equipment was already in use). Of these, 108 (81%) were categorized as “safe to feed orally” either by a speech and language therapist (57, 53%) or by the admitting nurse or doctor (51, 47%). The median time to the study of oxygenation during eating was 3 days (range 1 to 12 days) from stroke onset. Sixty-two patients (57%) were studied within 3 days of their stroke, 34 (32%) between 4 to 6 days, and 12 (11%) between 7 to 12 days after stroke. Patients who were studied later were those in whom recovery of swallowing took considerably longer (ie, was not recovered until nearly 12 days). Two patients refused to have physiological variables measured while eating. Therefore, 106 (98%) of those patients classified as “safe to feed orally” had physiological measurements taken at baseline and during and after a meal on the ward. Of these, 18 (17%) had a TACS, 51 (48%) had a PACS, 20 (19%) had an LACS, 14 (13%) had a POCS, and 3 (3%) remained unclassified. Twenty-seven patients (25%) had severe motor deficits, 56 (53%) had mild or moderate motor deficits, and in 23 (22%) the stroke did not affect arm or leg function.

We also recruited 50 elderly hospitalized patients without a history of stroke and 20 young healthy subjects. Elderly hospitalized patients were admitted with the following medical conditions: heart disease (n=9), pulmonary disease (n=7), gastrointestinal disease (n=10), deep venous thrombosis (n=3), poor mobility (n=15), or general malaise (n=6). These patients were observed with a median delay of 8 days after hospitalization.

Details of demographic comparisons between the 106 stroke patients and the control groups are displayed in Table 1. The elderly hospitalized patients were approximately 7 years older than the stroke patients. There were significantly fewer male subjects in the 2 control groups. Seventeen of the stroke and elderly control patients were taking beta-blockers, and 27 had an irregular pulse (which might affect the accuracy of pulse oximetry recordings) at the time of the study.\(^15\) Fourteen stroke patients (13%) and 10 elderly hospitalized patients (20%) had breathing problems (chest infection, bronchitis, chronic airway obstructive disease); 2 of these required oxygen therapy throughout the period of observation.

None of the elderly and young healthy control subjects had any identified swallowing problems, and all were able to eat a normal diet without help from a nurse. Some of the stroke patients had their diet modified and were fed by a nurse (Table 1). Most measurements were recorded during lunch; however, in order to see >1 patient per day, 6 stroke patients...
and 12 elderly control patients (24%) had recordings taken during the evening meal and 6 stroke patients (6%) had recordings taken during breakfast. As expected, young healthy subjects took significantly less time to complete their meal (median of 12 minutes) than either the stroke or elderly control patients (median of 15 and 16 minutes, respectively; Table 1). Twenty stroke (19%) and 2 elderly control (4%) patients remained in bed in the propped-up position while eating the meal, whereas the rest of the stroke patients and controls sat in a chair (Table 1). It is important to note that too few patients had recordings taken at breakfast or evening meals or when propped-up in bed to determine the influence of the time and type of meal or patient position on the SaO₂ recordings.

Control subjects had the hand on which the pulse oximeter sensor was placed randomized equally between the right and left sides. However, more stroke patients had the pulse oximeter sensor situated on their left (n=63, 59%) than on their right (n=43, 41%) middle finger. Of the 77 patients who had a hemiparesis, 50 had the sensor situated on their weak side. All of these patients were able to feed themselves with their unaffected hand; therefore, locating the sensor on the weak side reduced movement artifact and improved the accuracy of the SaO₂ readings.

**Baseline SaO₂**

The 106 stroke patients had a significantly lower baseline median SaO₂ compared with both elderly hospitalized patients and young healthy subjects (Table 2). Elderly control patients also had a significantly lower baseline median SaO₂ than young healthy control subjects (Table 2). In both the stroke patients who were able to take normal diet (n=61) and those who were on a modified diet (n=43), median baseline SaO₂ was 95.7% (IQR=3%).

**SaO₂ at Baseline Versus During the Meal**

In stroke and elderly control patients, but not in young healthy control subjects, median SaO₂ was lower during eating than at baseline (Table 2). This difference was statistically significant in elderly control patients (P=0.004, Wil-
coxon test; Table 3). There was a small group of stroke (6/106, 6%) and elderly (3/50, 6%) patients whose median SaO\textsubscript{2} levels fell by \(\geq 3\%\) from baseline to during the meal; falls of up to 5.6% saturation were observed. There was no association between falls in median SaO\textsubscript{2} during meals and preexisting breathing problems in either the stroke or the elderly hospitalized patient groups. There was also no association between falls in median SaO\textsubscript{2} and the types of meals (normal or modified) eaten by the stroke patients. One of the recording period.

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**Sao\textsubscript{2} at Baseline Versus After the Meal**

Compared with baseline, stroke patients had a small but statistically significant decrease in median SaO\textsubscript{2} after the meal (Table 3). This difference was similar regardless of whether the stroke patients were on a normal or modified diet. In the elderly and young controls, median SaO\textsubscript{2} recorded after the meal was either slightly above or equal to baseline values (Table 2). The change in median SaO\textsubscript{2} from baseline to after the meal between the 3 study groups was not quite statistically significant (\(P=0.07\), Kruskal-Wallis test; Table 3).

**TABLE 2. Median of the Mean SaO\textsubscript{2} (IQR) and Range Recorded in Stroke Patients, Elderly Hospitalized Control Patients, and Young Healthy Subjects at Baseline (Before), During, and After Eating a Meal**

<table>
<thead>
<tr>
<th></th>
<th>Baseline to Eating</th>
<th>Baseline to After Eating</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stoke patients</strong></td>
<td>95.7 (2.9), 88.0–99.6*</td>
<td>95.6 (2.8), 86.2–99.7*</td>
</tr>
<tr>
<td>(n=106)</td>
<td>95.4 (3.2), 86.0–99.3*</td>
<td></td>
</tr>
<tr>
<td><strong>Elderly hospitalized</strong></td>
<td>96.7 (2.9), 87.2–99.2†</td>
<td>96.3 (2.9), 87.2–99.2</td>
</tr>
<tr>
<td>patients (n=50)</td>
<td>96.5 (2.7), 77.5–99.7</td>
<td></td>
</tr>
<tr>
<td><strong>Young healthy subjects</strong></td>
<td>97.9 (2.0), 93.0–99.6</td>
<td>97.9 (2.0), 93–99.6</td>
</tr>
<tr>
<td>(n=20)</td>
<td>98.1 (2.6), 93.7–100</td>
<td></td>
</tr>
</tbody>
</table>

*Significantly lower than elderly hospitalized and young healthy control subjects (\(P<0.001\), Kruskal-Wallis test).†Significantly lower than both elderly hospitalized and young healthy control subjects (\(P<0.001\), Kruskal-Wallis test).

**TABLE 3. Median Falls in Mean SaO\textsubscript{2} Between Baseline and During and After Eating in Stroke Patients, Elderly Hospitalized Control Patients, and Young Healthy Control Subjects**

<table>
<thead>
<tr>
<th></th>
<th>Baseline to Eating</th>
<th>Baseline to After Eating</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stoke patients</strong></td>
<td>0.1 (1.8), −2.4–4.3</td>
<td>0.4 (1.4), −2.6–6.0*</td>
</tr>
<tr>
<td>(n=106)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Elderly hospitalized</strong></td>
<td>0.2 (1.1), −2.2–5.6*</td>
<td>0.1 (1.5), −3.7–6.0</td>
</tr>
<tr>
<td>patients (n=50)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Young healthy subjects</strong></td>
<td>−0.1 (1.0), −1.1–4.3</td>
<td>−0.4 (1.9), −1.8–3.6</td>
</tr>
<tr>
<td>(n=20)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Negative values indicate an increase in SaO\textsubscript{2} from baseline to during or after completion of the meal.

\(\text{**Duration of hypoxemia (≤90% Saturation) at Baseline and During and/or After Completion of the Meal**}

Twenty-five stroke (24%) and 8 elderly control (16%) patients, but none of the young healthy control subjects (without cold hands), had episodes in which the SaO\textsubscript{2} fell to \(\leq 90\%\) saturation (ie, hypoxemia) during and after eating. This difference in frequency of hypoxemia (24% versus 16%) between stroke patients and elderly controls was not statistically significant. One stroke patient and 2 elderly control patients spent virtually all of the recording period below this level. Two of these patients (1 stroke and 1 elderly control) had chest infections, and the other elderly control subject smoked 20 cigarettes a day. In the remaining patients there were large variations in the pattern of desaturation. Of the remaining 24 stroke patients who had episodes of hypoxemia, 18 had SaO\textsubscript{2} of \(<90\%\) during eating that lasted <3 minutes. Three of the 18 also had these episodes at baseline, 3 others also had episodes after eating, and 4 had episodes in all 3 periods of observation. In the remaining 6 stroke patients, SaO\textsubscript{2} fell to \(\leq 90\%\) only after eating. Of the 6 elderly control patients who experienced brief episodes of hypoxemia, all experienced episodes during eating, 1 also had falls at baseline, and 2 had falls after eating. Only 2 stroke patients (and none of the elderly controls) had episodes in which SaO\textsubscript{2} was \(<90\%\) saturation on swallowing but recovered within 3 minutes.

Hypoxemia recorded at baseline (whether it recovered within 3 minutes or not) was more common in the stroke patients who also had hypoxemia during and/or after eating (8/25, 32%) than those who did not (3/81, 4%) (\(P<0.001\), Fisher exact test; Table 4). The proportions for elderly control patients were very similar (\(P<0.003\), Fisher exact test; Table 4). There were no significant associations between hypoxemia and any other patient features (ie, preexisting breathing problems, severe strokes, or cardiac arrhythmias).

**Discussion**

Several authors have reported changes in oxygenation during assessment of swallowing in stroke patients.\(^8\text{–}^{11}\) but this is the first study (that we know of) to examine changes in saturation while stroke patients classified as “safe to feed orally” ate a meal. In the present large study, stroke patients had a lower
TABLE 4. Association Between Hypoxemia at Baseline and Subsequent Hypoxemia During and After Eating in Stroke Patients (n=106) and Elderly Hospitalized Control Patients (n=50)

<table>
<thead>
<tr>
<th></th>
<th>No Hypoxemia at Baseline</th>
<th>SaO2 Fell to ≤90% Saturation During and After Eating</th>
<th>SaO2 Remained at &gt;90% Saturation During and/or After Eating</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes Stroke patients</td>
<td>8*</td>
<td>3</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>No Stroke patients</td>
<td>17</td>
<td>78</td>
<td>95</td>
<td></td>
</tr>
<tr>
<td>Total Stroke patients</td>
<td>25</td>
<td>81</td>
<td>106</td>
<td></td>
</tr>
<tr>
<td>Yes Elderly controls</td>
<td>5</td>
<td>42</td>
<td>47</td>
<td></td>
</tr>
<tr>
<td>No Elderly controls</td>
<td>5</td>
<td>42</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

*Hypoxemia (whether recovery within 3 minutes or not) during and/or after eating occurred more commonly in patients with hypoxemia at baseline (P<0.001 and P=0.003, respectively, Fisher exact test).

baseline SaO2 than controls. Both stroke and elderly patients, but not young healthy controls, experienced small falls in median SaO2 while eating their meal. However, only in stroke patients did the median SaO2 remain lower after completion of the meal.

We did not consider a fall in median SaO2 of ≤1% during eating to be clinically, even if statistically, significant. However, 6% of stroke and elderly patients had a fall in median SaO2 of ≥3% while eating. It is worth noting that a 3% change from a baseline of 99% is unlikely to be clinically important, but a change of 3% from 93% may be; thus, it was important to find out the number of individuals in whom SaO2 fell to ≤90% saturation. We found that slightly more stroke (24%) than elderly (16%) patients experienced episodes of hypoxemia (ie, SaO2 of ≤90%) during and after eating. In most cases, these episodes of hypoxemia (and thus desaturation) did not coincide precisely with swallowing, and SaO2 recovered back to levels >90% saturation within 1 to 3 minutes. Also, these episodes were significantly more common in patients who also had hypoxemia at baseline. Thus, the majority of patients in whom we observed desaturation were likely to experience these changes regardless of whether they were eating or not.

It is important to note that our study was not powered to identify subgroups of patients who might experience meal-related episodes of hypoxemia (SaO2 decrease to levels of ≤90%) more commonly. Certainly, swallowing problems, and therefore possibly meal-related hypoxemia, are more common in patients with severe strokes, whereas patients classified as “safe to feed orally” in our hospital primarily had a PACS and mild to moderate motor deficits.8 Also, large falls in SaO2 during meals are more common in patients with chronic obstructive pulmonary diseases (COPD), whose resting mean SaO2 is on the steep portion of the oxygen dissociation curve.3 However, in our study there were only 14 stroke and 6 elderly control patients who had preexisting breathing problems. Therefore, there were too few patients with chest disease to expect to see such large changes in median SaO2. However, in populations where the frequency of COPD is higher in patients with acute stroke, the frequency of meal-related hypoxemia (a quarter of patients in the present study) might be higher too. It is also likely that many patients with severe strokes and/or chest disease could not have been passed as “safe to feed orally.” Therefore, our results may actually be a reflection of the sensitivity of the speech and language therapists and admitting nurses or doctors in our hospital when classifying whether patients are safe to swallow. In other hospitals however, where swallowing assessment is less systematic or staff are keen to encourage patients to eat more freely, a higher frequency of hypoxic episodes during feeding might be observed. It is also possible that we may have missed some patients who were aspirating in the first 1 to 2 days after stroke onset, because swallowing is known to be recovered in some patients quite rapidly.9 However, some stroke patients experience swallowing problems for the first time much later in their illness.9,10 Nonetheless, it would be important for future studies to include patients earlier than 3 days after onset, when potentially salvageable but vulnerable brain surrounding the stroke lesion is thought to have the greatest chance of survival.20 Finally, the results of this study also suggest that desaturation may be age related, although others8 have found no association between desaturation and swallowing and age. Nevertheless, if the stroke and elderly patients had been better matched for age, the difference in the number of patients with desaturation between the 2 groups may have been greater. Thus, one cannot rule out the possibility that in other hospitals with a greater proportion of elderly patients with severe strokes observed earlier than 3 days after stroke onset, or with a less-stringent swallowing assessment, there might be a higher proportion of patients who experience more serious hypoxic events during eating.

It has been suggested that meal-related episodes of desaturation occur as a result of aspiration of fluid/food into the airway, which causes a mismatch of pulmonary ventilation-perfusion and, ultimately, hypoxemia.4,5,8 Stroke patients have been found to inhale on swallowing, rather than exhale like normal individuals, which may increase their risk of aspiration during meals.21,22 However, as yet no study has been able to confirm whether there is a definite relationship between aspiration and altered SaO2.8–11 Also, as in previous studies,8–11 we found that these episodes often resolved within 1 to 3 minutes, whereas one would anticipate desaturation to persist if the patient had aspirated.

Others have suggested that hypoxemia may result from incoordination of breathing.1,23 Certainly, studies have found that stroke patients with interruption (or cessation) of breathing during sleep often have brief but substantial falls in SaO2 detected by oximetry.1 Indeed, Teramoto and colleagues23 reported that 30 seconds of breath-holding was enough to result in a significant fall (≥4%) in SaO2. Perhaps some of the patients included in our study had irregular breathing, regardless of whether they were eating or not. This would certainly explain why some patients experienced brief episodes of hypoxemia at baseline and after eating rather than only during
the meal. It is important to be aware that irregular breathing is often exacerbated by swallowing in acute stroke patients, and this may explain why desaturation occurred in slightly more patients during the meal. However, this frequency of hypoxemia was similar to that observed in the elderly patients, therefore one cannot rule out the possibility that there is some age-related decline in the ability to coordinate rapidly the sequence of throat muscle and breathing actions required for successful swallowing.

Interestingly, we found that young healthy controls, but not stroke or elderly patients, had a nonsignificant increase in median \( \text{SaO}_2 \) after the meal. Increases in arterial oxygen tension \( \text{(PaO}_2 \) have been previously reported to occur after healthy subjects and patients with COPD had eaten a large carbohydrate meal, probably as a result of increased metabolic activity. Certainly, in our study the young healthy subjects were more likely to consume high-carbohydrate meals consisting of sandwiches, whereas the hospital inpatients were more likely to have low-carbohydrate hot meals of meat and vegetables. Schols et al also found that patients who had sandwiches experienced fewer episodes of breathlessness and were therefore less exhausted. However, we were unable to confirm these hypotheses, because none of the meals were standardized for nutritional composition or calorific content and we did not measure whether a rapid eater might experience greater desaturation than one who took time to rest between swallows. Our study was also not set up to detect whether the time or type of meal (breakfast, lunch, or evening meal) influenced the results.

Intuitively, brief exposure to mild levels of hypoxemia, meal related or not, may be particularly harmful to patients with acute stroke who have potentially salvageable or vulnerable areas of brain around the stroke lesion, whereas patients without acute brain damage probably tolerate longer periods of more severe hypoxemia. In the present study we did not include patients thought to be most at risk of meal-related episodes of hypoxia, such as those with severe strokes or chest disease. Also, further studies might specifically examine factors that might be associated with hypoxemia, eg, the effect of nutritional content of the meal (ie, high-carbohydrate content versus small amounts of carbohydrate), the manner in which it is eaten (rapid gulps versus slow, smaller mouthfuls), and poor sitting posture (sitting in a chair versus a propped-up position).

Perhaps deliberate feeding of carbohydrate-rich food to stroke patients deemed “safe to feed orally” would improve oxygenation and hence brain recovery. Such a simple, inexpensive strategy, if effective, could have substantial public health impact as stroke (both ischemic and hemorrhagic) is so common and it could be applied to any patient classified “safe to feed orally.” Physiological monitoring during eating might provide an early warning that could enable earlier intervention, which in turn would prevent or reduce serious complications (eg, brain injury) and improve outcome after stroke, but is not warranted on the basis of the present study results. It is important to note however, that accurate non-invasive monitoring during swallowing assessment would take up much of the nurses’ time and without additional staff, might reduce their ability to do some other part of their job. It would also be important to reduce circumstances in which pulse oximeters produce false readings, ie, cold hands, as observed in one of the young healthy control subjects in our study. Moreover, before instituting routine noninvasive continuous monitoring, even for selected patients, it will be important to establish its efficacy in relation to outcome. Even if mild hypoxemia is indeed related to poor outcome in acute stroke patients, it does not necessarily imply that measures which reduce or prevent these episodes would improve outcome. These questions could be answered definitively only within the context of a randomized controlled trial of adequate size. Further studies of physiological changes that may occur during routine care of stroke patients should be encouraged, because even minor but persistent hypoxemia could contribute adversely to overall outcome.

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References


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