Letters to the Editor

Oxygen Desaturation on swallowing in Patients with Stroke: What Does it Mean?

Sir—With great interest, we read the article by Zaidi and coworkers concerning ‘Oxygen desaturation on swallowing as a potential marker of aspiration in acute stroke’ [1]. The authors demonstrated that a fall in SaO₂ on swallowing fluid was common in patients with acute stroke, and the presence or absence of desaturation was consistent with the assessment of aspiration by a speech and language therapist. Because aspiration pneumonia frequently occurs in elderly patients, the simple water-swallowing test introduced by the authors may be a unique and important approach in the geriatric ward. However, we do not agree with their hypothesis that ‘aspiration of fluid into the airway will lead to hypoxia by reflex bronchoconstriction and consequent ventilation-perfusion imbalance’. Water-induced bronchoconstriction is one of the mechanisms of hypoxia in asthmatic patients, but does not apply to most elderly subjects. Furthermore, micro-aspiration commonly occurs in the elderly, although massive aspiration (as in Mendelson syndrome) is very rare. As even 50 ml of water injected into one lobe of a lung for bronchoalveolar lavage does not always cause significant desaturation, the hypoxia in the current study may not be due to 10 ml of water. We believe that the hypoxia might result from inco-ordination between swallowing and breathing rather than aspirated water.

In our experience, more than 20–30 s of breath-holding (cessation of breathing) results in a significant drop in SaO₂ (more than 4%) [2]. Also, 24-h monitoring of oxygen saturation with a pulse oximeter revealed that most episodes of desaturation in daily activities are associated with breath-holding and/or poor breathing, for instance, typical and severe desaturation often occurs during evacuation. In the current study, the swallowing procedure was performed over a 2 min period. Because swallowing is closely related to the cycles and patterns of breathing under the control of the suprahyoid nerve [3, 4], abnormal swallowing can lead to poor breathing over a period of time, resulting in ventilation-perfusion mismatching owing to diminished inspiratory volume [5]. This may be a major factor of oxygen desaturation on the water-swallowing test.

We also would like to point out that measurement of SaO₂ assesses the arterial oxyhaemoglobin saturation, but not swallowing itself. In this regard, the conclusion of the authors that ‘SaO₂ measures may aid bedside assessment of swallowing’ may be confusing. Because swallowing can be divided into three phases of movements of food intake, many abnormalities of muscles, neural functions, and salivary gland functions may be involved in swallowing disorders. The SaO₂ measurement may be useful for detecting a swallowing-related abnormality of gas exchange, but not a swallowing disorder. Another problem is that oxygen saturation is monitored by a pulse oximeter. Although the convenient, non-invasive and accurate appraisal of oxyhaemoglobin saturation in arterial blood by oximeters is well established, the measurement is, however, affected not only by abnormal gas exchange in the lung, but also by abnormal blood flow at the monitored site. It is, therefore, possible that desaturation may, at least in part, be caused by circulatory abnormalities due to stroke-associated alterations in coagulation and/or circulation in the fingers of the non-paralysed hand.

Taken together, a fall in SaO₂ on swallowing fluid in patients with stroke could be explained by other mechanisms than that hypothesized by the authors.

Shinji Teramoto
Yoshinosuke Fukuchi
Yasuoshi Ouchi

Department of Geriatrics, Faculty of Medicine, University of Tokyo, 7-3-1 Hongo Bunkyo-ku, Tokyo, 113, Japan


Sir—The letter of Teramoto and colleagues raises some interesting points. We are grateful to them for pointing out mechanisms other than aspiration which may lead to desaturation when swallowing in stroke patients. However, we believe that our original paper was quite careful in not claiming that desaturation on swallowing was the result of aspiration and indeed we particularly stated in the last paragraph of our paper that we had not proved this.

We accept that the introduction of larger volumes of fluid directly into the lung during broncho-alveolar lavage does not always cause significant desaturation. However, as Teramoto and colleagues point out in their letter, it is possible that pre-existing ventilation-perfusion mismatches in stroke patients may have already produced a small degree of resting hypoxia and that (because of the sigmoid shape of the oxygen dissociation curve) a further minor mismatch on aspiration is more likely to result in oxyhaemoglobin desaturation.

All the above is of course to a degree conjectural and the only true way of evaluating the relationship between desaturation and swallowing requires a blinded study of assessment of oxygen saturation during videofluoroscopy in stroke patients (as stated in the last paragraph of our original