Aspiration and the laryngeal mask airway

Editor—I very much enjoyed reading the article by Keller and colleagues1 and the accompanying editorial2 referred to in your editorial, but I’m not sure how much of this is based on fact or how much on instinct. Does it make a difference to you that patients at risk of pulmonary aspiration (full stomach, hiatus hernia, etc.) the satisfactory use of an LMA entails a number of factors coming together all at the same time: the position of the LMA in the pharynx, pharyngeal muscle tone and the presence or otherwise of pharyngeal reflexes, respiratory muscle movement and the synchrony of the vocal cords. With the patient breathing spontaneously, all these factors do tend to come together under deep inhalational anaesthesia; it is when the patient is not as deep as you think they are that everything start to fall apart as they did with the LMA removed.

Apart from avoiding patients at increased risk of pulmonary aspiration (full stomach, hiatus hernia, etc.) the satisfactory use of an LMA entails a number of factors coming together all at the same time: the position of the LMA in the pharynx, pharyngeal muscle tone and the presence or otherwise of pharyngeal reflexes, respiratory muscle movement and the synchrony of the vocal cords. With the patient breathing spontaneously, all these factors do tend to come together under deep inhalational anaesthesia; it is when the patient is not as deep as you think they are that everything start to fall apart as they did with the LMA removed.

I would like to suggest that anaesthesia using an LMA is more predictable, more controllable and safer when the patient is fully paralysed than when they are allowed to breathe on their own. The device is easier to insert, ventilation is more reliably assessed, and does not have to ‘compete’ with the patient’s own unpredictable efforts (thereby lessening the risk of inflating the stomach), and depth of anaesthesia is more easily controlled. For short cases, mivacurium is the ideal neuromuscular blocking agent and spontaneous ventilation can be allowed to recover towards the end of surgery. The limiting factors of course are size, position (lithotomy) and the pressure required to ventilate the lungs. Most of us have a ‘cut-off’ of 20–25 cm H₂O because of the risk of stomach inflation referred to in your editorial, but I’m not sure how much of this is based on fact or how much on instinct. Does it make a difference whether the patient is fully paralysed, or partly paralysed or is breathing spontaneously and so still has muscle function and the ability to vomit with the mask in situ? Your editorial gives a reference for duration of anaesthesia affecting potential aspiration but quotes

1LMA® is the property of Intavent limited.
Editor—We read with interest the report of three cases of aspiration with the use of LMA\(^1\) and accompanied editorial.\(^2\) Early this year we conducted an audit of ‘Clinical practice of prevention of the aspiration pneumonitis’ at Belfast City Hospital, in part of which we asked our colleagues about their use (Yes, No or occasional) for LMA in patients with obesity, hiatus hernia or for laparoscopy (Fig. 1).

None used routinely the LMA in obese patients and 80% would not use an LMA in patients undergoing laparoscopic surgery. This may reflect our local practice where many patients are undergoing staging laparoscopy for cancer. For obesity the use of LMA needs more careful evaluation and consideration of other risk factors. Apart from the possibility of aspiration, effective oxygenation and ventilation may become difficult, particularly in the lithotomy position.

We agree with Asai that pulmonary aspiration with the use of LMA is under-reported. If we want to find out the true incidence of aspiration with LMA, a confidential database should be created.

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Editor—We would like to thank Drs Vaughan, Campbell, Patel and Turner for their interest in our case series and review, which seems to have provoked more reaction than Dr Vaughan’s excellent editorial on tracheal extubation.\(^3\) We consider that the lack of response probably reflects the readership concuring with his views rather than complacency or conflicting opinion.

We agree with Dr Vaughan that pharyngeal suction and the lateral head-down position represent current best practice for emergence after tracheal intubation in the at risk patient—we teach this to our trainees and as such could also be considered ‘old dinosaurs’. We also agree that this practice has stood ‘the test of time’. However, we do not share his confidence in ‘the test of time’ as an adequate tool for judging best practice, as many factors, for example the threat of litigation, impede the ‘natural selection’ of the best technique. Another unproven technique that represents current best practice for the prevention of aspiration is the application of cricoid pressure.\(^4\)

We do not agree that pharyngeal suction and the lateral head-down position represent current best practice with the LMA. Passing a suction catheter down the airway tube to remove infra-cuff secretions, or into the mouth to remove supra-cuff secretions can stimulate the patient and provoke aspiration rather than prevent it.\(^5\) Furthermore, supra-cuff secretions pose little risk to the respiratory tract as the LMA forms an effective throat pack. Although the lateral head-down position might protect against aspiration, the process of moving the patient might increase the risk of displacement and patient stimulation. We postulate that the body position for emergence with the LMA is less important than avoiding the movement required to achieve it.

Finally, we hope that the established techniques for preventing aspiration in the at-risk patient will be the subject of ongoing scientific scrutiny despite the large number of patients required to provide meaningful data.

We would like to thank Dr Campbell for presenting two new cases of aspiration with the LMA. These cases not only illustrate the dangers of moving the patient during the emergence phase but also how many aspiration events go unreported, assuming these happened some time ago.

We agree that one of the keys to safe use of the LMA is to ensure that the patient is adequately anaesthetized, and that the easiest way of avoiding the problems of inadequate anaesthesia is to paralyse the patient. However, this exposes patients to the risks of these drugs and is not applicable during the emergence phase where full muscle power is essential to patient safety. There is a moderate body of literature supporting the use of neuromuscular blocking agents with the LMA. For example, there is evidence that insertion success is higher in semi-\(^6,7\) and fully paralysed patients and there are 44 studies where non-depolarizing neuromuscular blocking agents were used during the maintenance phase and no major problems were reported.\(^7\) There is one study showing that ventilation is easier with neuromuscular blocking agents.\(^8\) At first glance, these data suggest that neuromuscular blocking agents are safe and effective but it is difficult to attribute outcome to the use of neuromuscular blocking agents or the use of positive pressure ventilation. We prefer to ventilate but not paralyse patients unless there is an indication for administering neuromuscular blocking agents, such as intraocular surgery, persistent vocal cord closure, or the surgeon retrospectively requests neuromuscular block. It is surprising how few patients require neuromuscular blocking agents for intra-abdominal surgery.

We consider peak airway pressures of 20 cm H\(_2\)O to be a safer cut-off to prevent gastric insufflation than 20–25 cm H\(_2\)O.\(^9\) However, higher pressures are acceptable if the cuff is correctly positioned, as the efficacy of seal with the hypopharynx is >40 cm H\(_2\)O.\(^9\) Gastric insufflation is readily detected by epigastic auscultation, which should be performed on all patients being ventilated with the LMA.\(^10\) The recommended figures are based on fact rather than instinct with at least 14 studies measuring hypopharyngeal leak pressure.\(^5\) These studies show that hypopharyngeal leaks are rare if airway pressures are <20 cm H\(_2\)O and common if >30 cm H\(_2\)O. The incidence of clinically detectable gastric insufflation is 0–0.3%. We are unaware of any data supporting an increased risk of aspiration with increasing duration of anaesthesia, although a study conducted in 1970 suggested that gastric volume increased in tracheally intubated patients after 2 h.\(^10\)

Finally, the obvious solution to these and other issues with the classic LMA is to use the ProSeal\(^11,12\) LMA, which not only allows greater ventilatory capability but also prevents gastric insufflation and pulmonary aspiration, provides information about distal cuff portion and exerts pressures against the surrounding mucosa.\(^1\) The main disadvantage of the ProSeal LMA over the classic LMA is a lower first attempt insertion success rate, but by using a guided insertion technique, such as use of a gum elastic bougie, first attempt success rates are probably higher than the classic LMA and with accurate hypopharyngeal placement.\(^5,11\)

![Fig 1 Preference for the use of LMA in patients with ‘at risk’ conditions for pulmonary aspiration.](image-url)
We thank Drs Patel and Turner for providing new audit data about the prevention of aspiration. Frankly, we were astonished to see that nobody in the Belfast City Hospital admitted to preferring the LMA in obese patients, as there is a substantial body of knowledge supporting its use in this situation. Perhaps more than anything, however, these new data illustrate the wide variation in clinical practice among institutes. In Cairns Private Hospital, for instance, where one of us works, more than 90% of patients undergoing laparoscopic surgery, or who are obese, or who have had reflux, are managed with the ProSeal LMA.

We agree that aspiration with the LMA is under-reported and that a confidential database should be created; however, this database should cover all airway devices, as the majority of aspiration events is associated with non-LMA devices, and it is only by comparing the frequency of aspiration among different airway devices in specific clinical situations that we can begin to determine best clinical practice.

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Anaphylaxis and anaesthesia

Editor—We read with interest the recent Editorial outlining the current state of anaphylaxis and anaesthesia. What remains unclear is the pathophysiology behind the variability in features and severity of anaphylaxis under anaesthesia, and why, despite widespread use of the Association of Anaesthetists of Great Britain and Ireland (AAGBI) guidelines, 10% of reactions reported to the UK Medicines Control Agency are still fatal. It is unfortunate that the authors of the AAGBI guidelines made no mention of the use of pure alpha agonists in their anaphylaxis drill for the treatment of severe anaphylactic reactions unresponsive to epinephrine. This was first described by Higgins and Gayatri in 1999, in a series of case reports by ourselves and others in 2001, and in two further case reports from Australia. The possible mechanism for the dramatic response to pure alpha agonists in this setting has been discussed.

We have recently successfully treated another case of severe anaphylaxis with the pure alpha agonist phenylephrine during open heart surgery while the filling status and contractility of the heart remained visible throughout the management of the reaction.

A 76-year-old gentleman, with a history of coronary artery disease and insulin-dependent diabetes mellitus, underwent elective coronary artery bypass grafting. Following successful weaning from cardiopulmonary bypass, he had an anaphylactic reaction to protamine resulting in sudden cardiovascular collapse with an arterial pressure of 55/35 mm Hg, central venous pressure 4 mm Hg and pulmonary artery diastolic pressure 8 mm Hg. There was no change in arterial oxygen saturation or airway pressure. Both the right and left ventricles appeared to be contracting vigorously and were visibly under-filled. Initial management consisted of stopping the administration of further protamine, ensuring adequate mechanical ventilation with oxygen 100% and rapid infusion of i.v. fluid. A total of phenylephrine 500 µg was given in bolus doses with a modest effect in restoring haemodynamic stability. Epinephrine 100 µg was then given with no significant effect. In response to direct observation of the visibly under filled and vigorously contracting ventricles, treatment continued with alpha agonists, i.v. fluids and secondary therapy. Phenylephrine was administered to a total dose of 10 mg over a period of 5 min followed by noradrenaline and dopamine infusions, initially at 1 µg kg⁻¹ min⁻¹ and 10 µg kg⁻¹ min⁻¹, respectively. Over the next 2 h haemodynamic stability was restored and surgery was completed. The patient made an uneventful recovery.

Immunological investigations revealed a significantly elevated serum mast cell tryptase at 1 and 6 h post-event consistent with an anaphylactic reaction. Further assessment revealed that the patient had been receiving a protamine-containing insulin preparation for many years.

It is logical to administer epinephrine as the first line drug of resuscitation during anaphylaxis on the basis of its action on the immunological and cardiovascular systems. However, case reports by ourselves and others make it clear that continued epinephrine administration may not produce return of spontaneous circulation. From direct observation of the empty, vigorously contacting heart in this case of anaphylaxis, it was obvious that restoration of systemic vascular tone was of paramount importance. This was achieved with large doses of phenylephrine while avoiding the potentially harmful ß2 inotropic and chronotropic and ß2 vasodilatory effects of large doses of epinephrine.

The AAGBI guidelines are not alone in failing to advocate pure alpha agonists in severe anaphylactic reactions unresponsive to epinephrine. Recent reviews in both the US and UK literature continue to make no mention of the recent case reports outlining this life saving therapy. The Executive Summary in the AAGBI guidelines states 'there are no clinical trial data and no evidence base is available or likely to become available (for the management of anaphylaxis). Recommendations follow analysis of case reports and summaries of experience.' On the basis of case reports such as this, we believe that guidelines on anaphylaxis should include the administration of a significant bolus of a pure alpha agonist before