Facemask Ventilation during Induction of Anesthesia
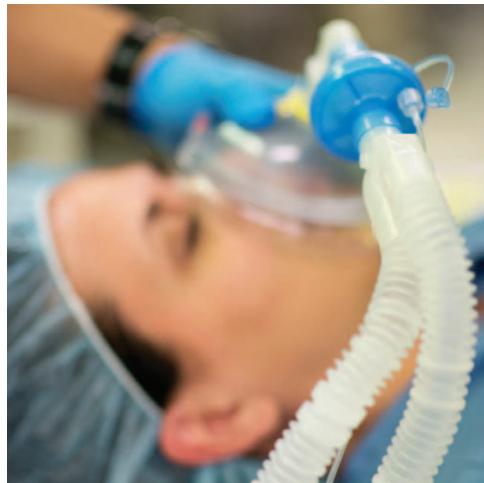
How "Gentle" Is "Gentle" Enough?

Shiroh Isono, M.D., Ph.D., Matthias Eikermann, M.D., Ph.D., Takeo Odaka, M.D., Ph.D.

DURING anesthesia induction, even skilled anesthesiologists may sometimes have the impression that they just have insufflated gas in the patient's stomach during manual facemask ventilation. To avoid the consequences of such a gastric inflation in patients at risk of aspiration, most of us were taught not to conduct facemask ventilation before intubation—even though this strategy may need to be revisited when severe hypoxemia develops or is expected. "Gentle" facemask ventilation has been recommended[1,2]—but what does that mean? In this issue of ANESTHESIOLOGY, Bouvet *et al.* give us new important information to answer that question. The authors have developed and validated a real-time ultrasonography technique for visualized entry of the gas into the antrum of the stomach.[3] By using this sensitive method to detect gastric insufflations, they determined a threshold inspiratory pressure for preventing the gastric gas insufflation during pressure-controlled ventilation with a facemask to be approximately 15 cm H_2O which is lower than the recommended target peak airway pressure of 20 cm H_2O previously determined by the traditional epigastric auscultation.[4,5] The results of this study seem to provide a straightforward answer that helps prevent gastric inflation during anesthesia induction, but need to be discussed from various perspectives.

"'Gentle' facemask ventilation has been recommended—but what does that mean? ... Bouvet et al. give us new important information to answer that question."

What Do We Know about the Critical Gastric Gas Volume That Predicts an Increased Aspiration Risk?

Gastric inflation may increase the risk of life-threatening regurgitation and pulmonary aspiration of the gastric contents in patients at risk, for example, those with full stomach and an impaired ability to protect their airway as a consequence of anesthesia and/or muscles weakness.[6] Critical volume of gastric contents for pulmonary acid aspiration was estimated to be 0.8 ml/kg in humans based on animal studies.[7] Bouvet *et al.*,[8] the same research group, previously reported by using ultrasonography that a high-risk (full) stomach occurs in 60 of 76 (78.9%) of emergency patients but only in 3 of 104 (2.9%) presenting for elective surgery. However, based on existing data it is challenging to define the safe gastric volume that does not translate to an increased aspiration risk in patients at risk for aspiration. It is important to mention that the association between gastric volume and aspiration risk also depends on other factors that promote regurgitation of the gastric contents during anesthesia induction such as the composition of gastric content and upstream and downstream intraluminal esophageal and intestinal pressures.[9]

How Can the Physiological Barriers to Aspiration Be Broken by Gastric Insufflation?

The esophagus is not a simple conduit connecting the pharynx and the stomach but actively prevents similar to a unidirectional valve the entry of gases and fluids into the stomach (upper esophageal sphincter [UES]) as well as the regurgitation of the gastric contents to the pharynx at the lower esophageal sphincter (LES).[10] The unidirectional valve can be deactivated by esophageal distention which induces a reflexive peristalsis to remove the esophageal content to the stomach or the pharynx. Barrier pressures can be estimated by the UES pressure minus airway pressure for the gastric inflation and the LES pressure minus gastric pressure for

---

Image: J. P. Rathmell.

Corresponding article on page 326.

the regurgitation. Reduction of the LES barrier pressure and subsequent regurgitation can occur *via* three mechanisms: (1) transient increase in the gastric pressure, (2) lower resting LES pressure, and (3) transient complete relaxation of the LES.[11] In particular, the transient LES relaxation usually producing belching which is now considered to be a major cause of the gastroesophageal reflux disease.[12]

Why Does the Gastric Inflation Possibly Lead to Regurgitation?

By using high spatial resolution esophageal manometry in anesthetized patients, recent clinical studies illuminate possible mechanisms of regurgitation of the gastric contents. de Leon *et al.*[13] found that the LES barrier *function* is preserved even under general anesthesia and paralysis whereas the LES barrier *pressure* decreases substantially even in non-obese patients. Moreover, individual variability of the LES barrier pressure is wide and some patients under anesthesia have a near-zero LES barrier pressure, which suggests that regurgitation will likely occur even with a slight increase in gastric pressure. Under the condition of an anesthesia-induced impairment of the LES pressure, we speculate that the amount of gastric gas inflation observed in the study presented in this issue of Anesthesiology may further increase the risk of regurgitation. In obese patients, the LES barrier is even more vulnerable compared with nonobese patients, and therefore, it is likely that the risk of regurgitation is greater in obese patients.[13]

Gastric inflation physiologically induces gastric adaptive relaxation and a transient LES relaxation, and the effects of anesthesia on these physiological functions are unknown. The gastric adaptive relaxation may be advantageous for prevention of regurgitation by reducing the gastric pressure. However, also in response to gastric pressure increase, a transient, simultaneous relaxation of the LES and the UES occurs, leading to a subsequent retrograde escape of gas (possibly with gastric contents) from the stomach to the esophagus in association with belching. Accordingly, an increase in pressure from gastric insufflation during face mask ventilation may translate to an increased aspiration risk by reflex relaxation of the UES and LES. Many pieces of information regarding the response of the gastroesophageal conduit to gastric insufflation during anesthesia induction are currently missing and further pharmaco-physiological interaction trials clearly are needed.

Bouvet's study supports the conclusion that it is impossible to exclude gastric insufflations in all patients. In fact, Bouvet *et al.*[3] observed ultrasonographical evidence of gastric insufflation in 33 and 19% of patients ventilated with an inspiratory pressure as low as 15 and 10 cm H_2O, respectively—these low mask pressures were insufficient to ventilate the patients in 12 and 75%. The anesthesia regimen seems to affect the relationship between mask pressure and probability of gastric insufflations. de Leon *et al.* reported

that the UES pressure in patients anesthetized with remifentanil and propofol infusions amounts to 40 cm H_2O on average with values greater than 20 cm H_2O in majority of the subjects. However, the UES is sensitive to muscle relaxation and total neuromuscular blockade decreased the UES pressure below 15 cm H_2O in more than half of the patients[13] indicating high risk of the gastric inflation even when the inspiratory pressure is set at 15 cm H_2O which is recommended in the Bouvet's study. Accordingly, the "optimal" mask pressure reported in clinical trials must always be interpreted in the context of patients' disease entity as well as the anesthesia technique applied.

What Is the Optimal Inspiratory Pressure during Rapid-sequence Induction and Intubation for a Full-stomach Obese Patient?

Then, what are the optimal induction techniques for a full-stomach obese patient while awake intubation may be certainly an option? First, how do we handle a nasogastric tube? Its presence possibly weakens anatomical integrity of the UES and LES, increases the frequency of transient LES relaxations, and impairs the upper airway protective reflexes while it could serve to decrease the gastric pressure.[14] To date, it is unclear whether the nasogastric tube increases the risk of clinically meaningful aspiration. Although some of anesthesiologists may choose no positive-pressure ventilation before intubation, they must prepare for failure of the first intubation attempt and therefore facemask ventilation because oxygenation with facemask ventilation is the first priority in case of desaturation knowing the risk of regurgitation.[2] A higher inspiratory pressure than that recommended by Bouvet *et al.* may be required to achieve adequate ventilation in patients with lower respiratory compliance such as in obese patients or in patients with increased intra-abdominal pressure. Although upper (retropalatal and retroglossal) airway obstruction is considered to be a risk factor for the gastric inflation, it may not cause the gastric inflation because high mask pressure does not translate to an increased pressure at the level of the UES under these conditions. However, in clinical practice, anesthesiologists tend to over-compensate the required increase in the inspiratory pressure during manual facemask ventilation in response to the upper airway obstruction. Similarly, the inspiratory pressure unintentionally exceeds the UES pressure during volume-controlled ventilation mode. Pressure-controlled ventilation is a preferred ventilation mode for this purpose. Two hands airway maintenance maneuvers during pressure-controlled ventilation can improve airway patency, and respiratory compliance may increase with progression of muscle paralysis.[15] These techniques can maximize ventilation efficiency and reduce the optimal inspiratory pressure.

Bouvet's study reminds us to aim for "gentle" pressure-controlled ventilation without putting our patients at risk of

hypoventilation. It also emphasizes anesthesiologist's common wisdom that one size (mask pressure) does not fit all patients' needs during induction of anesthesia. We should apply the lowest possible mask pressure that allows for adequate ventilation. We should use devices and strategies such as the two hands airway maintenance maneuvers to keep the mask pressure lower than the UES barrier pressure that varies in a wide range across patients.

Competing Interests

The authors are not supported by, nor maintain any financial interest in, any commercial activity that may be associated with the topic of this article.

Correspondence

Address correspondence to Dr. Isono: shirohisono@yahoo.co.jp

References

1. Brown JP, Werrett G: Bag-mask ventilation in rapid sequence induction. Anaesthesia 2009; 64:784–5
2. Clements P, Washington SJ, McCluskey A: Should patients be manually ventilated during rapid sequence induction of anaesthesia? Br J Hosp Med (Lond) 2009; 70:424
3. Bouvet L, Albert M-L, Augris C, Boselli E, Ecochard R, Rabilloud M, Chassard D, Allaouchiche B: Real-time detection of gastric insufflation related to facemask pressure-controlled ventilation using ultrasonography of the antrum and epigastric auscultation in nonparalyzed patients: A prospective, randomized, double-blind study. ANESTHESIOLOGY 2014; 120:326–34
4. Ruben H, Knudsen EJ, Carugati G: Gastric inflation in relation to airway pressure. Acta Anaesthesiol Scand 1961; 5:107–14
5. Lawes EG, Campbell I, Mercer D: Inflation pressure, gastric insufflation and rapid sequence induction. Br J Anaesth 1987; 59:315–8
6. Mirzakhani H, Williams JN, Mello J, Joseph S, Meyer MJ, Waak K, Schmidt U, Kelly E, Eikermann M: Muscle weakness predicts pharyngeal dysfunction and symptomatic aspiration in long-term ventilated patients. ANESTHESIOLOGY 2013; 119:389–97
7. Raidoo DM, Rocke DA, Brock-Utne JG, Marszalek A, Engelbrecht HE: Critical volume for pulmonary acid aspiration: Reappraisal in a primate model. Br J Anaesth 1990; 65:248–50
8. Bouvet L, Mazoit JX, Chassard D, Allaouchiche B, Boselli E, Benhamou D: Clinical assessment of the ultrasonographic measurement of antral area for estimating preoperative gastric content and volume. ANESTHESIOLOGY 2011; 114:1086–92
9. Sarma J, Eikermann M: Images in anesthesiology: A large gastric Bezoar: Preanesthetic considerations. ANESTHESIOLOGY 2011; 115:175
10. Shaker R: Airway protective mechanisms: Current concepts. Dysphagia 1995; 10:216–27
11. Dodds WJ, Dent J, Hogan WJ, Helm JF, Hauser R, Patel GK, Egide MS: Mechanisms of gastroesophageal reflux in patients with reflux esophagitis. N Engl J Med 1982; 307:1547–52
12. Holloway RH, Kocyan P, Dent J: Provocation of transient lower esophageal sphincter relaxations by meals in patients with symptomatic gastroesophageal reflux. Dig Dis Sci 1991; 36:1034–9
13. de Leon A, Thörn SE, Wattwil M: High-resolution solid-state manometry of the upper and lower esophageal sphincters during anesthesia induction: A comparison between obese and non-obese patients. Anesth Analg 2010; 111:149–53
14. Gomes GF, Pisani JC, Macedo ED, Campos AC: The nasogastric feeding tube as a risk factor for aspiration and aspiration pneumonia. Curr Opin Clin Nutr Metab Care 2003; 6:327–33
15. Isono S: One hand, two hands, or no hands for maximizing airway maneuvers? ANESTHESIOLOGY 2008; 109:576–7