The loss of protective airway reflexes during the induction of general anesthesia can result in pulmonary aspiration of the gastric contents. Pulmonary aspiration remains an infrequent but serious cause of anesthesia-related morbidity and mortality, with little change in incidence over the past 20 years. In a study of 133 cases of aspiration reported to the Australian Anesthetic Incident Monitoring Study database [1], 56% of the cases of pulmonary aspiration occurred during the induction of anesthesia, and obese patients were over-represented in the group of patients who aspirated. Obesity is one of a number of factors that contributes to the risk of aspiration during anesthesia, including emergency surgery, trauma, airway difficulties, inadequate depth of anesthesia, and gastrointestinal disorders such as intestinal obstruction, hiatal hernia, and gastroesophageal reflux.

The technique of rapid sequence induction includes four separate actions designed to reduce the risk of pulmonary aspiration: the use of pharmacologic agents with or without gastric suctioning to reduce gastric acid volume and acidity; the administration of a rapidly acting hypnotic agent and muscle relaxant to limit the apneic time and reduce the at-risk period of an unprotected airway; the application of cricoid pressure; and the absence of mask ventilation after a period of preoxygenation. Although these actions seem simple and logical and are based on basic and clinical science, not one randomized controlled trial has confirmed the benefits of rapid sequence induction. Yet, rapid sequence induction
is considered the standard of care in US anesthesia practice for all patients with an increased risk of pulmonary aspiration.

The use of rapid sequence induction is common practice in patients with obesity and sleep apnea syndrome. However, aspects of the rapid sequence induction technique may be deleterious in some patients with these disorders. A review of the current medical literature elicits as many questions as answers regarding the usefulness of this technique. This article addresses a number of these questions: Are patients with obesity and sleep apnea really more at risk than the general population for pulmonary gastric aspiration? Does rapid sequence induction reduce the risk of pulmonary acid aspiration? Are there situations in which the rapid sequence induction technique is potentially dangerous in patients with morbid obesity or obstructive sleep apnea? Is cricoid pressure a beneficial technique, and when is it deleterious? Which muscle relaxant and sedative combination is the most useful for rapid sequence induction?

**Rapid sequence induction and the difficult airway**

The specific alterations in airway anatomy and physiology leading to difficult mask ventilation and tracheal intubation in patients with morbid obesity and sleep apnea syndrome are addressed elsewhere in this issue. A number of important differences in cardiopulmonary physiology that affect the safety of anesthetic induction in these patients also are addressed elsewhere. These anatomic and physiologic differences must be taken into account when considering the risks and benefits of the rapid sequence induction technique in these patients.

Although there are several studies that have concluded that no increased risk of difficult intubation is posed in the morbidly obese population [2,3], numerous other studies demonstrate a clearly defined increased risk of difficult intubation in both the morbidly obese [4–6] and sleep apnea syndrome populations [7–9]. Hiremath et al [10] found that eight of 15 individuals with Cormack and Lehane grade 4 laryngoscopic views had apnea-hypopnea indices consistent with previously undiagnosed sleep apnea syndrome, whereas only two matched controls without a difficult laryngoscopic view had similar scores. Although these data do not establish that patients with sleep apnea syndrome have difficult airways, the authors propose that the two conditions appear to be related and to share anatomic features that act to reduce the skeletal confines of the tongue. Additionally, the incidence of diabetes mellitus is increased in the obese population, and long-standing diabetes mellitus has been associated with an increased risk of difficult intubation caused by the glycosylation of collagen and its deposition in the joints, resulting in limited joint mobility syndrome [11].

The presence of a potentially difficult airway during the induction of anesthesia in obese and sleep apnea syndrome patients is a risk to several aspects of patient safety. The ability to assure oxygenation and ventilation during management of the difficult airway is the paramount concern. A secondary concern is that both difficult airways and the development of inadequate anesthetic depth
during difficult airway management have been found to predispose to regurgitation, vomiting, and aspiration, as reported in the Australian Anesthetic Incident Monitoring Study database [1] and in nearly one-third of the patients who aspirated during induction of anesthesia in a study of pulmonary aspiration by Warner et al [12].

Several of the crucial components of the rapid sequence induction technique have even been shown to be deleterious in patients who have potentially difficult airways. A key element in the rapid sequence technique is the nonhypoxemic apneic period, designed to avoid gastric insufflation that would reduce barrier pressure and promote regurgitation. Because the ability to mask ventilate is not tested before the administration of the muscle relaxant, a distinct risk is the development of a cannot-intubate and cannot-ventilate situation. Furthermore, because the functional residual capacity of obese patients is reduced, they develop oxygen desaturation faster than the nonobese, and the safe apneic period is reduced from more than 5 minutes to less than 2 to 3 minutes in the preoxy- genated state [13–15]. The absence of ventilation in the rapid sequence technique promotes atelectasis that additionally reduces the apneic duration of adequate oxygenation. A recent study [16] in morbidly obese individuals (limited to 160 kg by the weight limit of the CT scanner) has demonstrated that the administration of constant positive airway pressure (CPAP) during the preoxygenation period and gentle ventilation with positive end-expiratory pressure (PEEP) during anesthetic induction significantly reduce atelectasis, as documented by chest CT scans. An associated benefit to the reduced atelectasis was a significantly increased average PaO₂ (457 ± 130 pascals [Pa] versus 315 ± 100 Pa, \(P = 0.035\)) in those subjects who received CPAP-PEEP versus the control subjects. Theoretically, that increase in PaO₂ would increase the apneic time to oxygen desaturation if an airway event were to occur.

A second component used in the rapid sequence induction that may be deleterious in patients with obesity and sleep apnea syndrome is the use and misuse of cricoid pressure. As discussed later, the correct application of cricoid pressure is difficult for the untrained practitioner and difficult to maintain even for the trained practitioner, and the pressure can affect both the ability to mask ventilate and the direct laryngoscopic view. Allman [17] has demonstrated that the application of cricoid pressure by an experienced anesthetist causes both a significant reduction in mean exhaled tidal volume (\(P \leq 0.001\)) and an increase in peak inspiratory pressures (\(P \leq 0.001\), with complete airway occlusion occurring between 6% and 11% of the time when cricoid pressure is applied. Saghaei and Masoodifar [18] prospectively bimanually applied 4.5 kg (40–50 Newtons [N]) of cricoid force to 80 American Society of Anesthesiologists grade-1 adult patients who were randomly divided into those who received cricoid pressure and controls (simple placement of hands without exerting pressure). The effects on mask ventilation were identical to those of Allman [17], with a significant increase in peak inspiratory pressure and a significant decrease in tidal volume after the application of cricoid pressure (\(P \leq 0.001\)). Hartsilver and Vanner [19] studied 52 female patients undergoing elective surgery, and facemask ventilation
was assessed under five different conditions: no cricoid pressure; backward cricoid pressure applied with a force of 30 N; cricoid pressure applied in an upward and backward direction with a force of 30 N; backward cricoid pressure with a force of 44 N; and cricoid pressure applied with a tracheal tube inserted. The authors found that cricoid pressure applied with a force of 44 N can cause airway obstruction, but if cricoid force is reduced to 30 N, airway obstruction occurs less frequently \((P=0.0001)\), unless the force is applied in an upward and backward direction.

Cricoid pressure can also adversely affect direct laryngoscopy. Haslam et al [20] recorded the laryngoscopic view using a rigid, zero-degree endoscope to define the effect of cricoid pressure on laryngoscopy. In 40 patients undergoing elective surgery, cricoid pressure was increased by 10 N increments, and the change in laryngoscopic view with increasing cricoid pressure was evaluated (Fig. 1). In five subjects in whom the initial laryngoscopic view was good, a marked deterioration in view occurred as cricoid pressure increased. In three of these subjects, the view decreased to completely obscure the larynx at a force of 30, 40, and 60 N, respectively. The laryngeal view was obscured by a down-folding of the epiglottis, adduction of the vestibular folds, and encroachment of the pharyngeal soft tissues into the line of sight, or rotation of the larynx also behind the right aryepiglottic fold during cricoid pressure application.

Because of the potential for airway deformation with cricoid pressure, using the minimum force necessary to prevent gastric regurgitation will optimize the maintenance of a patent airway and an adequate laryngeal view. In patients in whom the laryngoscopic view of the glottis is poor, posteriorly applied cricoid pressure may cause the view to further deteriorate, and although backward and upward laryngeal displacement can improve laryngeal visualization, this displacement unfortunately worsens the ability to mask ventilate. Currently, far more

Fig. 1. The number of subjects in whom the laryngoscopic view improved, remained unchanged, or worsened with each 10 N of increase in cricoid force. (Data from Haslam N, Parker L, Duggan JE. Effect of cricoid pressure on the view at laryngoscopy. Anaesthesia 2005;60(1):41–7.)
morbidity occurs owing to hypoxemia during difficult or failed intubation than from aspiration. If the need to mask ventilate is crucial, the cricoid pressure should be directed directly posterior and may need to be gradually released.

Failed intubation in rapid sequence induction is an uncommon event, even in patients with morbid obesity. The incidence of failed intubation in the general population is approximately 1:2300 in the general population and 1:230 in the obstetric population [21,22]. In a 1999 survey of academic anesthetists in the United Kingdom, 94 of 209 respondents (45%) had experienced failed intubation during rapid sequence induction at least once in their career [23]. The incidence of failed intubation in the morbidly obese population is ill defined. In the study of difficult intubation in morbid obesity by Juvin et al [6], there were no cases of failed intubation, but as many as eight attempts at direct laryngoscopy by as many as four persons were required. Brodsky et al [3] had one failed rapid sequence induction in 100 subjects with a body mass index (BMI) greater than 40 kg/m². In a study of 117 morbidly obese parturients whose weight exceeded 136.4 kg (300 pounds) at the time of delivery, general anesthesia was administered to 17 patients. Difficult tracheal intubation occurred in six of the 17 morbidly obese parturients, compared with zero of eight nonobese control subjects ($P=0.06$). There were no failed intubations, but two of the morbidly obese patients underwent an awake laryngoscopy or intubation [24].

Unfortunately, several of the techniques commonly used in the failed intubation algorithm also may be adversely affected by cricoid pressure. Cricoid pressure has been shown to make laryngeal mask airway (LMA) insertion more difficult [25,26] and makes the use of the intubating LMA less likely to be successful [27]. Because of these potentially adverse affects of cricoid pressure on ventilation, laryngoscopy, and laryngeal mask insertion, most failed intubation guidelines now recommend placing the patient in Trendelenburg position, with suction available and graded release of cricoid pressure in the cannot-intubate, cannot-ventilate situation.

Risk of gastroesophageal reflux and pulmonary aspiration

In common practice, obese patients are considered a group that is at increased risk for gastric aspiration, but what are the supporting data that all obese patients are at a higher risk of gastric acid aspiration? Because the use of rapid sequence induction has not been proven to prevent gastric aspiration and may be hazardous in some circumstances, is it reasonable to apply the technique to all obese individuals? Factors that increase the risk for pulmonary gastric aspiration syndrome include low pH of the gastric contents; an increased volume of gastric fluid, which depends on both the production of gastric fluid and on gastric emptying; the presence of gastroesophageal reflux, which depends on the barrier pressure between the stomach and lower esophageal sphincter; and the time fasted. A number of authors have evaluated the risk of gastroesophageal reflux in the morbidly obese patient. Several authors have demonstrated a rate of gastro-
esophageal reflux similar to the general population. Warner et al [12] found no increase in the incidence of aspiration in those with a body mass index greater than 35 kg/m². Zacchi et al [28] demonstrated a resistance gradient between the stomach and gastroesophageal junction comparable to nonobese individuals. Kadar et al [29] reported 650 mask general anesthetics for electroconvulsive therapy in 50 obese subjects with no episodes of clinical pulmonary aspiration, although asymptomatic reflux or aspiration may have been present. Coussa et al [16] found no evidence of gastric aspiration on the chest CT scans of 18 morbidly obese subjects after anesthetic induction without cricoid pressure, including nine patients who received gentle mechanical ventilation with PEEP by mask.

Recently, several studies have demonstrated an increased risk of reflux in the morbidly obese [30,31]. Fisher et al [30] reported 30 subjects with morbid obesity presenting for bariatric surgery evaluation. Sixteen of the 30 patients had symptomatic reflux, and 11 of the 30 had abnormal esophageal acid exposure (pH ≤ 4 for more than 5% of observed time) as determined by pH probe and esophageal manometry. Individuals with abnormal acid exposure had a body mass index that was larger than were those without abnormal acid exposure (BMI of 56.5 versus 48.3 kg/m², P ≤ 0.05), even though lower esophageal sphincter pressure was not correlated with body mass index. In addition to the baseline risk of gastroesophageal reflux, obese individuals also are more likely to have diabetes mellitus and be prone to gastroparesis.

There are conflicting data regarding the risk of gastroesophageal reflux specifically in patients with obstructive sleep apnea. Because the transdiaphragmatic pressure increases in parallel with the increased intrathoracic pressure generated during obstructive apnea episodes, the attendant increases in gastric volume and pressure can lead to passive regurgitation whenever there is lower esophageal sphincter insufficiency [32]. On the other hand, there are data showing that sleep apnea syndrome and gastroesophageal reflux are common entities that share similar risk factors but appear not to be linked causally [33].

**Cricoid pressure**

Cricoid pressure provides a second line of defense when the lower esophageal sphincter fails to prevent passive regurgitation. The practice of cricoid pressure is attributed to Sellick [34] in 1961, but its use to obstruct the esophagus is over 200 years old. Monro [35] in the 1770s, Hunter in 1776 [35], and Curry in 1796 [36] described the use of esophageal compression at the throat to assist ventilation and reduce gastric insufflation during resuscitation.

The original study by Sellick postdated by 10 years the description by Morton and Wylie [37] of protecting the airway with the patient in the sitting position using barbiturate and relaxant, followed by rapid intubation. When evaluating the data reported by Sellick [34], it is essential to remember that his technique was different from cricoid pressure as practiced today. In that study, cricoid pressure prevented fluid passing from esophagus under the cricoid ring and at lower
pressures controlled the flow of fluid. The patient’s head was extended as if for a tonsillectomy rather than in the “sniffing” position to increase the anterior convexity of the cervical spine, thus stretching the esophagus to prevent lateral displacement. Unfortunately this position also makes laryngoscopy more difficult, and most patients undergoing anesthetic induction are in the sniffing rather than extended neck position. Sellick also used a light Trendelenburg position to help clear any leakage away from airway. He then evaluated 26 high-risk patients using cricoid pressure. None of the patients demonstrated reflux during the application of the cricoid pressure, and three had reflux subsequently, after cricoid pressure was released, supporting the effectiveness of the technique.

Despite considerable dogma regarding the benefits of cricoid pressure in the past 40 years, the use of cricoid pressure is being revisited as recent information supports a reevaluation of the use of this seemingly simple and safe technique. Unfortunately, a well-designed controlled, randomized prospective trial of cricoid pressure has not been performed. Brimacombe and Berry [38], Ng and Smith [39], and Landsman [40] have examined the topic of cricoid pressure and provided comprehensive reviews describing the advantages and risks associated with the technique. Some authors have described the technique as cricoid force rather than pressure because the downward force is measured in Newtons rather than in millimeters of mercury (30 N of cricoid pressure is equivalent to 40 mm Hg of upper esophageal sphincter pressure).

There are unquestionably data to support the practice of cricoid pressure. In the study by Sellick [34], the reflux of gastric contents occurred in three of 26 patients when cricoid pressure was released. A 30-N cricoid force provided protection against gastric reflux even with 40 mm Hg of upper esophageal sphincter pressure in cadavers with the head extended [41]. A 40-N cricoid force increased the upper esophageal sphincter pressure to more than 38 mm Hg in 24 patients, with or without a pillow under their head [42]. Also, cricoid pressure has been shown to prevent gastric insufflation in both adults [43] and children [44,45] during mask ventilation.

Data are also evolving against the routine use of cricoid pressure. As discussed above, cricoid pressure can impede airway patency and make direct laryngoscopy more difficult, clearly vital issues during the induction of anesthesia in those with obesity and sleep apnea syndrome. It is important to remember that airway difficulty itself has been demonstrated to be an independent factor in pulmonary aspiration [12].

Second, cricoid pressure has been demonstrated to reduce lower esophageal pressure by a yet uncertain mechanism. The lower esophageal pressure, which is normally 25 to 35 mmHg, is the first line of defense against the passive regurgitation of gastric contents. Lower esophageal sphincter pressure falls with most anesthetic induction and nondepolarizing neuromuscular blocking agents, reducing the barrier pressure (lower esophageal pressure minus gastric pressure). Intragastric pressure is normally less than 7 mm Hg at rest, but succinylcholine increases intragastric pressure to 40 mm Hg, and during vomiting, intragastric pressure rises to 45 mm Hg. Tournadre et al [46] studied eight healthy, fasted
awake volunteers who underwent intragastric, esophageal, and lower esophageal sphincter (LES) pressure monitoring. Cricoid pressures of 20 N followed by 40 N were each applied for 15 seconds. The application of cricoid pressure reduced LES pressure from $24 \pm 3$ to $15 \pm 4$ mm Hg at a force of 20 N ($P \leq 0.05$) and to $12 \pm 4$ mm Hg with a force of 40 N ($P \leq 0.01$). Garrard et al [47] reported a similar reduction in lower esophageal sphincter pressure during the application of cricoid pressure in anaesthetized patients. The gastric pressure was less than the esophageal pressure, and the barrier to reflux remained intact in all cases.

If cricoid pressure were universally beneficial, there would be few cases of pulmonary aspiration when cricoid pressure was used and, alternatively, many cases when cricoid pressure and rapid sequence induction were not used. In the Australian Anesthetic Incident Monitoring Study [1], 11 patients (8% of those with pulmonary aspiration) experienced aspiration despite the application of cricoid pressure. Warner et al [12] retrospectively reviewed the perioperative courses of 172,334 consecutive patients 18 years of age or older who underwent 215,488 general anesthetic procedures from July 1985 to June 1991 and found pulmonary aspiration had occurred in 67 patients. Of the 67 patients who aspirated, 33 did so during induction, and 14 patients had pulmonary aspiration despite rapid sequence induction. In a survey exploring anesthetists’ practice in rapid sequence induction in the United Kingdom, 59 of 209 respondents (28%) had seen regurgitation with rapid sequence induction, and three respondents reported the death of a patient as a result of regurgitation with rapid sequence induction [23]. In a prospective study from France, at a time when cricoid pressure was rarely used, there was a lower rate of aspiration during anesthesia than in comparable studies in countries where cricoid pressure was used [48].

Another problem with the use of cricoid pressure is that cricoid pressure is often incorrectly or inconsistently applied in the clinical setting [49]. Untrained individuals do not apply the correct amount of force, and it is often not sustained for the entire duration that the airway is unprotected [50]. It has been suggested that training in the application of cricoid pressure or the use of a mechanical device be used to assure correct and consistent application of the cricoid pressure.

Even when cricoid pressure is correctly applied, there is no guarantee that the esophagus will be actually occluded between the posterior cricoid ring and the vertebral body. In a recent study [51], 22 awake healthy volunteers underwent sagittal and axial magnetic resonance imaging of their necks, with their heads in a neutral position at baseline and with 20 to 30 N of applied cricoid force. In 53% of the patients at baseline, the esophagus was viewed lateral to the cricoid ring, and in 90% of the subjects, the esophagus was found to be displaced laterally after cricoid pressure was applied (Fig. 2). Lateral laryngeal displacement was noted in 67% of the subjects with cricoid pressure, and airway compression of at least 1 mm was demonstrated in 81% of the subjects as a result of cricoid pressure. The authors, based on their results, challenged the assumption that the cricoid, esophagus, and vertebral body are juxtaposed along the axial plane.

In addition to airway compromise, there are other complications of cricoid pressure, both common and unusual. The timing of the application of cricoid
pressure is critical. If it is applied before the loss of consciousness or with too much force, it is painful and may lead to vomiting or retching, which could promote pulmonary aspiration. Before the loss of consciousness, the currently used force of 30 to 40 N is too uncomfortable for volunteers to tolerate. Awake volunteers tolerated 20 N of cricoid force, whereas 40 N caused difficulty in breathing in 50% of volunteers [52]. Cricoid pressure also can have a negative hemodynamic consequence. Saghaei and Masoodifar [18] found a mean 20-mm Hg increase in systolic arterial blood pressure ($P < 0.001$) and a mean 10-beat per minute increase in heart rate ($P < 0.001$) occurred as a result of the application of 45 N of cricoid pressure in healthy adult patients after induction of anesthesia with sodium thiopental, 5 mg/kg, fentanyl, 1 μg/kg, and 1% halothane by mask. Rare complications of cricoid pressure have been described, including esophageal rupture, which can occur during active vomiting. Cricoid pressure should be released during active vomiting. Data suggest that a nasogastric tube does not impair the efficacy of cricoid pressure. Studies support suctioning the nasogastric

Fig. 2. The esophagus is seen at midline before cricoid pressure (A) and is deviated laterally with cricoid pressure (B). Note that the esophagus is only partially occluded. C, cricoid; E, esophagus; VB, vertebral body. (From Smith KJ, Dobranowski J, Yip G, et al. Cricoid pressure displaces the esophagus: an observational study using magnetic resonance imaging. Anesthesiology 2003;99:60–4. with permission.)
tube before induction and leaving the nasogastric tube open to allow the intra-
gastric pressure to be released [53].

The optimal cricoid force that will provide a barrier to reflux yet minimize
airway and respiratory compromise has been recently reappraised as well. The
force believed initially to be required to provide a barrier to reflux was 40 N and
was subsequently reduced to 30 N. Recent data suggest that 20 N may be suffi-
cient to provide the second layer of protection at the upper esophageal sphincter
yet minimize the distortion of the airway. Haslam et al [54] studied the gastric
pressure in a large group of heterogeneous surgical patients during rapid se-
quence induction with sodium thiopental and succinylcholine. The highest gastric
pressure observed was 14 mm Hg, and most values were considerably less than
this value. This demonstrates a gastric barrier pressure of less than 15 mm Hg,
even with wide-open gastroesophageal reflux under these conditions. The authors
have concluded that cricoid pressure applied at 20 N of force is more than
adequate to protect most anesthetized patients from regurgitation. Until more data
confirm the low gastric pressure, the current recommendations are to initiate
cricoid pressure with a force of 20 N as the induction agents are administered and
to increase the force to 30 N as loss of consciousness occurs.

Does induction agent pharmacology make a difference?

Historically, succinylcholine is the neuromuscular blocking agent of choice in
rapid sequence induction, for numerous reasons. It has the most rapid onset and
shortest duration of action of currently available neuromuscular blocking drugs.
The rapid onset is essential to reduce the apneic period and shorten the at-risk
period with an unprotected airway. The short duration of action is critical if the
airway is difficult and it is required for the patient to resume spontaneous
ventilation. Several authors have suggested recently that reducing the dose of
succinylcholine to 0.6 mg/kg still allows adequate intubation conditions, and the
lower dose would be attractive as far as reducing the time to return to spon-
taneous ventilation [55,56]. However, it should be remembered that inadequate
anesthetic depth and incomplete muscle relaxation are independent variables that
lead to pulmonary aspiration [1,12].

Unfortunately, succinylcholine has a number of well-described side effects. One
side effect that affects the risk of aspiration itself during rapid sequence
induction is fasciculations that cause a rise in gastric pressure [57]. Succinylcho-
line has been demonstrated to increase the gastric pressure to 40 mm Hg, al-
though recent reports do not demonstrate this degree of gastric pressure increase
[54]. The practice of defasciculation will restrict the intragastric pressure rise but
will require that a larger dose of succinylcholine be administered. Brodsky and
Foster [58] have reported data from 14 morbidly obese patients who received
succinylcholine without pretreatment with a nondepolarizing blocking agent.
Only three of the 14 patients had gross fasciculations, and only two of the
14 individuals developed myalgia, suggesting that the morbidly obese do not
develop fasciculations to the same degree as the nonobese. Because succinylcholine also concomitantly increases the lower esophageal sphincter pressure, the decrease in barrier pressure might be less than anticipated. In a model using pigs with a full stomach and a rapid sequence induction with either propofol or sodium thiopental and succinylcholine, lower esophageal sphincter pressure increased during the fasciculations from $19 \pm 4$ to $28 \pm 5$ mm Hg in the propofol-succinylcholine group and from $23 \pm 6$ to $36 \pm 7$ mm Hg in the thiopental-succinylcholine group. The lower esophageal sphincter pressure remained elevated after the fasciculations. Similarly, gastric barrier pressure was increased. This increase in barrier pressure begins before fasciculations and remains elevated for the period when intubation would occur, thus protecting against reflux of gastric contents during the typical induction period [59].

Rocuronium is the only nondepolarizing neuromuscular blocking agent with an onset similar to succinylcholine. The side-effect profile is much less problematic than succinylcholine. The duration of action is much longer than succinylcholine, and in patients with an increased risk of difficult airway, rocuronium is not as attractive as succinylcholine during airway difficulties. A recent systematic review by Perry et al [60] compared the intubating conditions of rocuronium with succinylcholine. The authors’ conclusions were that succinylcholine creates excellent intubation conditions more reliably than rocuronium and should still be used as a first line muscle relaxant for rapid sequence induction. If an alternative agent is required, rocuronium, when used with propofol, will reliably create excellent intubation conditions and should be used as the second line treatment of choice.

Summary

Patients with obesity and those with sleep apnea syndrome are prone to gastroesophageal reflux. Likewise, both groups demonstrate an increased risk of difficult intubation. Rapid sequence induction remains important in obese and sleep apnea syndrome patients with symptomatic gastroesophageal reflux or other predisposing condition such as diabetes mellitus, pregnancy, emergency surgery, and gastrointestinal conditions. In the case of elective surgery in a fasted patient with no risk factors other than obesity or sleep apnea syndrome, the requirement for rapid sequence induction is debatable. Cricoid pressure is probably efficacious but has not been proven in a randomized, controlled trial to prevent gastric aspiration. The clinician should be aware of the possibility that cricoid pressure will worsen mask ventilation and laryngoscopy and be prepared to loosen or release the pressure if mask ventilation or intubation is compromised. Regular training for the administration of cricoid pressure is indicated. If rapid sequence induction is required, succinylcholine remains the neuromuscular blocking agent of choice if there are no contraindications. Adequate anesthesia and muscle relaxation during induction are vital because coughing or straining during induction is a major risk for pulmonary aspiration.
References

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