On Cricoid Pressure: "May the Force Be with You"

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Cricoid pressure has become the quintessential cornerstone of the rapid sequence induction of anesthesia in patients at risk for passive regurgitation of gastric fluids. Despite an almost 5-decade history, the level of evidence to support its effectiveness in preventing passive regurgitation is a paltry 4 or 5, resulting in a Grade D recommendation for its use.[1,2] However, guidelines in anesthesia and other medical specialties endorse its use, and the legal community exploits its omission from the anesthetic record as evidence of a practice below the "accepted" standards. In this month's issue of *Anesthesia & Analgesia*, Rice et al.[3] explore the cricoid-hypopharyngeal unit to determine whether cricoid pressure occludes the esophageal lumen when the latter is displaced lateral to the cervical spine. The time has come to critically review what we know and what we do not know about cricoid pressure and to set the record straight once and for all.

Cricoid pressure was first reported by Sellick[4] in 1961 for use at induction of anesthesia. His seminal report in which he established the effectiveness of cricoid pressure in 26 patients who required emergency surgery transformed the practice of anesthesia. However, several details in his report are not well known and merit our consideration. First, each patient in his report was positioned "head down slightly with the head turned" for induction of anesthesia. Upon release of the cricoid pressure, he noted that 3 of the patients (12%) regurgitated. Some readers may be surprised that such a large proportion of patients regurgitated when cricoid pressure was released (it contrasts sharply with our clinical experience), whereas others might attribute the high incidence of regurgitation simply to the patients' head-down position. The pervasive fear of regurgitation and aspiration during induction of anesthesia with the early ether anesthetics led to this positioning to direct regurgitant fluids away from the larynx. Currently, the head-down position is never used at induction of general anesthesia; moreover, Sellick's study has never been repeated with patients in the supine position.

Second, Sellick[4] suggested that "firm" pressure be applied to the cricoid ring after positioning the neck in the "tonsil" position (in which the neck is fully extended). Sellick never determined how much force was required to occlude the lumen of the esophagus. Currently, a minority of anesthesiologists and assistants are aware of the magnitude of the force required to occlude the esophageal lumen.[5–9] Many more apply an insufficient force (although some apply excessive force) to occlude the lumen of the esophagus and prevent passive fluid regurgitation (a force of 30–44 N is required with loss of consciousness, where 10 N is the force of gravity on an object with a mass of approximately 1 kg).[6–8,10] Although Sellick recommended that the tonsil position be used when performing this maneuver, the majority of patients are positioned with the head in the "sniffing" or "neutral," but not tonsil, position.

Third, Sellick claimed that a nurse or assistant could be instructed in this maneuver in "a few seconds."[4] Currently, anesthesiologists infrequently instruct others in the correct technique for applying cricoid pressure. If the principles espoused by Sellick were followed, then anesthesiologists need to revisit the details of how we perform this maneuver: we must agree on a uniform position for the patient's head and neck and ensure that all

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health care personnel are aware of how much force to apply,[6–9] when to release it, and when to avoid applying it altogether.[8,9] Until a consensus is reached on the proper technique for cricoid pressure and substantive evidence is forthcoming, simply ticking off a box on the anesthetic record or adding a notation on the record that cricoid pressure was performed in some arbitrary manner is nothing short of disingenuous.

"Light" cricoid pressure (i.e., a force of 20 N) is recommended for awake patients because it is generally well tolerated. As anesthesia is induced and muscle relaxants are administered, the upper esophageal sphincter pressure decreases from approximately 40 to <10 mm Hg,[11] virtually eliminating this barrier to regurgitation. In addition, light cricoid pressure along with the sedative/anesthetics and insertion of a laryngeal mask airway decrease lower esophageal sphincter tone,[12] reducing the gastroesophageal barrier to regurgitation. In the supine position, gastric pressure may reach 25 torr in patients without full stomachs[13,14] and 35 torr with fluid distention.[15] To prevent regurgitation with a gastric pressure of 35 torr as anesthesia is induced, a force of ≥30 N, not 20 N, is required at the cricoid ring.[16] However, clinicians have neither the metrics by which to gauge the degree of relaxation of the anatomical sphincters nor the tools to measure the force applied to the cricoid ring. With this combination of shortcomings, passive regurgitation is bound to occur in some patients, despite the application of cricoid pressure.

That cricoid pressure and in some instances, "sloppily" applied cricoid pressure, is without risk to patients is sophistry. A force of 20 N applied to the cricoid ring in awake patients causes patient discomfort, breathing difficulties, promotes vomiting, aspiration, and even rupture of the esophagus.[2,17] With induction of anesthesia, a force greater than 20 N is required to occlude the esophageal lumen. Wraight et al.[10] recommend ≥44 N force to occlude the esophageal lumen, although they noted that 44 N was effective in only 50% of those in whom very high intragastric pressures were present. Cricoid pressure directly affects the airway by deforming the ring in 90% of patients and occluding the lumen in up to 50% of patients.[18] Difficulty in ventilating the lungs occurs in up to 50% of patients. The relationship between cricoid pressure and the view of the larynx during laryngoscopy seems to be complex, with moderate to severe distortion of the view occurring at forces of 30 and 40 N in 12.5% and 40% of patients, respectively.[19] Nonetheless, the incidence of failed intubation is similar in patients with and without cricoid pressure.[20] Hartsilver and Vanner[21] determined that when the cricoid ring was directed backward and upward, airway obstruction occurred 28 times more frequently than when simple backward pressure was applied. Hence, only backward pressure is recommended. One reason that large forces are required to occlude the esophageal lumen is that the force is distributed primarily over the apex of the crescent-shaped esophagus that overlies the vertebral body. To occlude the lateral aspects of the lumen, a large force is required. With the head in a "neutral" position rather than the "tonsil" position, the esophagus is not tethered over the cervical spine and may be displaced entirely or, in part, laterally. As Rice et al.[3] discuss in this month's journal issue, cricoid pressure may or may not occlude the lumen when it is paravertebral (see below). Although some authors have suggested that cricoid pressure should be maintained while ventilating the lungs during a failed intubation, studies in health care personnel have demonstrated that the applied force wanes after 2–4 min,[22] reducing the barrier to regurgitation. In an obstetric unit in the United Kingdom, cricoid pressure was maintained after failed intubations.[23] The authors reported that the lungs could be ventilated easily by facemask in 60% of patients, with difficulty in 30% and impossibly in 9%. Evidence suggests that cricoid pressure also prevents proper placement of the Classic and ProSeal laryngeal mask airways, possibly precluding their role in a failed intubation scenario.[24] In neonates and infants, the adult hand that applies cricoid pressure often restricts mouth opening and interferes with proper positioning of the laryngoscope handle. Furthermore, the force required to occlude the esophagus in infants and children and that deforms the cricoid ring in infants and children is unknown, and the effect of cricoid pressure on our ability to visualize the larynx in infants and children is debatable, leading many to abandon cricoid pressure in infants and children altogether. In fact, several reports have documented that only 50% of pediatric anesthesiologists use cricoid pressure during induction of anesthesia in children who require emergency surgery.[25–27]

Since Sellick's report, there has been a dearth of studies attesting to the efficacy of cricoid pressure in preventing passive regurgitation. Indeed, 2 systematic reviews concluded that there was no evidence for or against the application of cricoid pressure.[1,2] This being the case, how should we begin to determine the validity of this maneuver? We could begin by designing a randomized study based on Sellick's data to determine the incidence of passive regurgitation with and without cricoid pressure. A sample size of approximately 50 subjects per group would be required to prove that cricoid pressure is tenfold more effective than no pressure at all in reducing the incidence of passive regurgitation from 12% to 1.2% (α_1 0.05, β 0.2).[4] On the other hand, we could test whether cricoid pressure reduces the incidence of pulmonary aspiration, which would be a much more formidable undertaking and require a much larger, and in my view unjustifiable, sample size. With a frequency of aspiration of approximately 0.15% in adults, a randomized trial to reduce that incidence of aspiration by 50% would require a sample size of approximately 25,000 patients in each group. I strongly urge investigators to

take-up the challenge and determine the effectiveness of cricoid pressure to prevent passive regurgitation in the current anesthetic milieu.

If there is no evidence that cricoid pressure has improved patient outcome, is there evidence that it has had a neutral or negative effect on patient outcome? In one study, 4% of patients ($n = 12$) showed a new or unexpected infiltrate on chest radiograph after tracheal intubation despite 9 having had cricoid pressure applied during airway instrumentation.[28] In a review of almost 5000 general anesthetics for obstetrics in Malawi, 11 deaths were attributed to regurgitation and 9 had cricoid pressure applied.[29] Additional anecdotal reports included 2 fatal cases of Mendelson's syndrome in women to whom antacids were administered and cricoid pressure was applied, regurgitation in nonobstetric cases despite proper application of cricoid pressure, and fatal regurgitation when cricoid pressure was applied during induction of anesthesia and downfolding of the epiglottis that limited the view at laryngoscopy. These reports do not augur well for the effectiveness of cricoid pressure to prevent poor outcomes.[30,31] Although the numbers of patients in these reports were quite small, the notion that aspiration may occur despite the application of cricoid pressure might be reconciled by one or more of the following: a fixed failure rate may exist even when cricoid pressure is properly applied; cricoid pressure was not applied properly; cricoid pressure was released prematurely; or aspiration occurred at some time other than induction, i.e., at extubation. The absence of data precludes a determination of the contribution of these explanations to the poor outcomes. Nonetheless, a judge in the United Kingdom did rule against an anesthesiologist for failing to apply cricoid pressure in a patient with an irreducible hernia who had regurgitated and aspirated. The judge argued that we cannot assert "that cricoid pressure is not effective until these trials have been performed, especially as it is an integral part of an anesthetic technique that has been associated with a reduced maternal death rate from aspiration since the 1960s."[32] The contributions of preoperative fasting, administration of antacids, administration of oxygen, and avoidance of mask ventilation after induction of anesthesia to the reduction in maternal mortality are unknown. However, it is nothing short of outrageous to suggest that cricoid pressure is the lynchpin in this sequence[17] and that we require evidence to abandon 1 factor in the rapid induction sequence when we required no evidence to include the same factor in the first place!

Those who question the role of cricoid pressure in preventing regurgitation point to recent studies that demonstrate that the lumen of the esophagus cannot be completely occluded by cricoid pressure because it lies or is displaced lateral to the cervical spine.[33,34] In this month's journal issue, Rice et al.[3] provide radiological imaging evidence that the cricoid-hypopharynx moves as a unit (laterally) and that, when force is applied to the cricoid ring when it is paravertebral, the lumen of the esophagus is occluded. I fully endorse their radiological findings, which support an anatomical link between the cricoid ring and the hypopharynx, although I am far less enthusiastic about equating these anatomical measurements to physiological metrics. In Figure 2C of their article, cricoid pressure is seen to compress all of the tissues behind the cricoid ring: esophagus, fascia, prevertebral fat, and other soft tissues. Closer examination of Figure 2C shows that the left longus colli muscle is distorted compared with the right longus colli muscle, suggesting that this muscle can be distorted and compressed when pressure is applied, as is the case with so many other soft tissues. This muscle does not provide the same hard surface against which to compress the lumen of the esophagus as does the cervical vertebra. To equate these anatomical images with physiological metrics, the opening or distending pressure for a laterally displaced upper esophageal sphincter pressure must be measured with and without cricoid. Until those data are forthcoming, these radiological findings remain extremely interesting but of limited physiological import.

I fear we have become far too myopic in focusing more on the documentation that cricoid pressure was applied during induction of anesthesia than on: investigating the validity of the notion that cricoid pressure prevents passive regurgitation; focusing on teaching the proper application of cricoid pressure; focusing on which patients require cricoid pressure; and focusing on the risk of aspiration during maintenance and emergence from anesthesia.[35–37]

Currently, there is insufficient evidence to advocate or abandon the use of cricoid pressure to prevent passive regurgitation in at-risk anesthetized patients.[1,2,37] We need to prove that properly applied cricoid pressure is effective at preventing regurgitation or discard it. It is time to take stock of what we do and do it better. We owe it to our patients.

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