■ CLINICAL INVESTIGATIONS

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Cricoid Cartilage Pressure Decreases Lower Esophageal Sphincter Tone

Jean-Pierre Tournadre, M.D., Dominique Chassard, M.D.,* Khalid R. Berrada, M.D.,* Paul Boulétreau, M.D.†*

Background: **Cricoid cartilage pressure induced to prevent pulmonary aspiration from regurgitation of gastric contents has been recommended, and its efficacy requires a force greater than 40 Newtons. For regurgitation to occur, both an increase in gastric pressure and relaxation of the lower esophageal sphincter (LES) are necessary. However, the effect of cricoid cartilage pressure on the LES is unknown. This study evaluated the effects of cricoid cartilage pressure on LES in human volunteers.**

Methods: **Lower esophageal sphincter and esophageal barrier pressures (which equals LES pressure − gastric pressure) were measured using a manometric method in eight unanesthetized volunteers (4 men, 4 women) classified as American Society of Anesthesiologists physical status 1. The force applied to the cricoid cartilage was measured continuously, and LES pressure was recorded at a cricoid force of 20 and 40 Newtons.**

Results: **Cricoid pressure decreased LES pressure from 24 ± 3 mmHg to 15 ± 4 mmHg at a force of 20 Newtons ($P < 0.05$) and to 12 ± 4 mmHg with a force of 40 Newtons ($P < 0.01$).**

Conclusions: **These findings may explain the occurrence of pulmonary aspiration before tracheal intubation despite application of cricoid cartilage pressure. (Key words: Anesthetic techniques: cricoid pressure. Complications: aspiration, regurgitation. Gastrointestinal tract: lower esophageal sphincter.)**

CRICOID cartilage pressure during induction of anesthesia, first described in 1961 by Sellick,[1] is applied to occlude the esophagus and to prevent pulmonary aspiration of gastric contents. Complete occlusion occurs only at a force of at least 40 Newtons.[2] Other publications refer to this correct cricoid pressure as "firm"[1] or as the "pressure which would cause pain if applied to the bridge of the nose."[3] It is generally accepted that a decrease of the lower esophageal sphincter (LES) pressure combined with an increase of gastric pressure is the primary mechanism for regurgitation.[4] Thus any drugs or mechanisms that decrease sphincter tone may increase the risk for regurgitation during anesthesia.[5,6] We previously showed that cricoid cartilage pressure decreased LES pressure (LESP) in anesthetized swine.[7]

The aim of the present study was to investigate the effects of Sellick's maneuver on LESP in humans volunteers.

Materials and Methods

Study Design

After we received approval of the institutional human investigation committee and written and informed patient consent, we enrolled eight healthy volunteers (4 men and 4 women; ages 28 ± 4 y; weight, 62 ± 10 kg) with no diseases involving cardiac, respiratory, or the digestive systems. None of the participants was taking any long-term medications and all were studied without sedation or topical pharyngeal anesthesia. All volunteers had fasted for 12-16 h.

Each volunteer was studied at midday while laying supine with the head and neck extended. Heart rate was continuously monitored throughout the study and recorded before and during application of cricoid cartilage pressure.

Cricoid cartilage pressure was applied through a water-filled PVC balloon (25 ml), maintained with the thumb and index. The balloon was connected to a pressure transducer (Bentley Trantec, Irvine, CA) that displayed the applied force in Newtons (N). This system, which was adapted to the cricoid cartilage surface, allowed us to properly apply the range of force chosen. The system was zeroed and then calibrated (0 to 40 N) before each use. Lower esophageal sphincter, esophageal, and gastric pressure were recorded using perfused polyethylene catheters (Multilumen probe, C71 A, Marquat, Boissy, France). The method for LESP measurements was previously published.[8,9] We used polyethyl-

* Staff Anesthesiologist.

† Professor of Anesthesiology.

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Address reprint requests to Dr. Tournadre: Service d'anesthésie-Réanimation, Hôpital de l'Hôtel-Dieu, 69002, Lyon, FRANCE.

Table 1. Manometric Data during Cricoid Pressure 20 N and 40 N

	Before Cricoid Pressure	Cricoid Pressure 20 N	Cricoid Pressure 40 N	After Release
LESP (mmHg)	24 ± 3	15 ± 4*	12 ± 4†	24 ± 6
GP (mmHg)	5 ± 2	5 ± 1	5 ± 2	5 ± 0
BrP (mmHg)	18 ± 2	10 ± 3*	7 ± 3†	19 ± 4

Data are mean ± SD.

LESP = lower esophageal sphincter pressure; GP = gastric pressure; BrP = esophageal barrier pressure (LESP − GP).

* $P < 0.05$ *versus* control.

† $P < 0.01$ *versus* control.

ene catheters connected to transducers (Bentley Trantec) and constantly perfused with water by a low-compliance infusion pump (Type 871012 pump; Braun, Melsungen, Germany) at 1 ml/min (compliance of 200 mmHg/s). This catheter was introduced orally without anesthetic gel. The high-pressure zone, defined as the LES, was identified using a station pull-through technique, and measurements were taken after a 15-min rest. Transducers were zeroed to the midchest position and calibrated using a water column before each measurement. Pressure tracings were recorded using a multiple-channel recording system (Kontron Medical Electronics, England). The force applied to the cricoid was increased until 20 N over 5 s, then to a maximum of 40 N over 5 s and maintained for 15 s. Lower esophageal sphincter pressure and gastric pressure were recorded continuously. Esophageal barrier pressure is defined as LES pressure − gastric pressure.

Statistical Analysis

Values are expressed as mean ± SD. Data management and calculations were performed with commercially available software (Instat, GraphPad Software, San Diego, CA). A Kruskal-Wallis nonparametric analysis of variance test was used to detect any changes in LESP induced by cricoid pressure. A repeated-measures one-way analysis of variance was used to determine the cricoid pressure-dependent effects in LESP followed by a Bonferroni corrected multiple-comparisons test. Statistically significant differences were confirmed with a nonparametric Wilcoxon paired test because our data did not follow a Gaussian (bell-shaped) distribution. Changes were considered statistically significant when the probability value was less than 0.05.

Results

Results are shown in table 1. Lower esophageal, barrier, and gastric pressures were in the normal range for humans at baseline. Cricoid pressure caused a significant decrease in LESP ($P < 0.002$). Lower esophageal sphincter pressure decreased from 24 ± 3 mmHg before cricoid cartilage pressure to 15 ± 4 mmHg at a pressure of 20 N ($P < 0.05$) and to 12 ± 4 mmHg at a pressure of 40 N ($P < 0.01$). The difference in LESP between 20 and 40 N was statistically significant ($P < 0.05$). After release of the cricoid pressure, LESP returned to baseline values.

Gastric pressure (table 1) and heart rate remained unchanged throughout the tests. All volunteers tolerated cricoid pressure and none reported difficulty in breathing.

Discussion

The upper esophageal sphincter usually prevents regurgitation into the pharynx while a person is awake.[10] The reduction in upper esophageal sphincter pressure with anesthetic agents begins before consciousness is lost.[11] Cricoid cartilage pressure is designed to compensate for this reduction, and thus it should be applied before loss of consciousness.[1,11] Our previous study showed that cricoid cartilage pressure decreased LESP in anesthetized pigs with increased intraabdominal pressure.[7] However, application of these findings to clinical practice had several potential limitations because the animals were tracheally intubated and anesthetized before cricoid cartilage pressure was applied.

Cricoid cartilage pressure of at least 40 N is necessary to occlude the esophagus.[2] The current study shows that cricoid cartilage pressure as little as 20 N reduces LESP and LES barrier pressure in conscious humans.

We could argue that this decrease was observed while barrier pressure (barrier pressure = LESP − gastric pressure), which is the pressure gradient across the LES and the major mechanism preventing regurgitation of gastric contents, stayed positive. Although it is well

known that there is a relation between any decrease in LESP and reflux, it is not possible to indicate barrier pressure or LESP values below which reflux would occur.[12,13]

The phenomenon we observed may provide a possible explanation for gastric content aspiration during induction of anesthesia despite the application of cricoid cartilage pressure.[14,15] For example, if only moderate cricoid pressure, as recommended before loss of consciousness (about 20 N), is applied when the upper esophageal sphincter is not completely intact,[11] the efficacy of LES barrier pressure may be reduced, leading to regurgitation.

A pharyngeal reflex in conscious humans and anesthetized animals may explain the decrease in LESP. Our study is comparable to those of Mittal and colleagues[16] and Rabey and associates,[17] who recently used manometric catheters to show that insertion of a laryngeal mask airway reduced LESP as a result of pharyngeal stimulation. These authors concluded that mechanoreceptors in the pharynx mediate the induction of LES relaxation. Pharyngeal stimulation induced by cricoid cartilage pressure may also reduce the LESP. Further work is necessary to establish the precise nature of this reflex and its clinical significance, particularly when anesthesia is used for patients with increased gastric pressure.

Cricoid cartilage pressure in conscious humans induces a decrease in LESP and barrier pressure. This decrease is present at cricoid cartilage pressure less than that necessary to occlude the esophagus.

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