Cricoid Cartilage Pressure Decreases Lower Esophageal Sphincter Tone

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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.”13 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.56 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 middaw 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.89 We used polyethyl-
ene catheters connected to transducers (Bentley Trans
tec) 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 techni-
que, and measurements were taken after a 15-min
rest. Transducers were zeroed to the midchest posi-
tion and calibrated using a water column before each
measurement. Pressure tracings were recorded using a
multiple-channel recording system (Kontron Medical Ele-
tronics, England). The force applied to the cricoid was
increased over 20 N over 5 s, then to a maximum of
40 N over 5 s and maintained for 15 s. Lower eso-
gagheal 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. Sta-
tistically 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, bar-
rier, and gastric pressures were in the normal range for
humans at baseline. Cricoid pressure caused a signifi-
cant 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 base-
line values.

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

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

Cricoid cartilage pressure of at least 40 N is necessary
to occlude the esophagus. 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 pres-
sure), which is the pressure gradient across the LES
and the major mechanism preventing regurgitation of
gastric contents, stayed positive. Although it is well

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

<table>
<thead>
<tr>
<th></th>
<th>Before Cricoid Pressure</th>
<th>Cricoid Pressure 20 N</th>
<th>Cricoid Pressure 40 N</th>
<th>After Release</th>
</tr>
</thead>
<tbody>
<tr>
<td>LESP (mmHg)</td>
<td>24 ± 3</td>
<td>15 ± 4*</td>
<td>12 ± 4†</td>
<td>24 ± 6</td>
</tr>
<tr>
<td>GP (mmHg)</td>
<td>5 ± 2</td>
<td>5 ± 1</td>
<td>5 ± 2</td>
<td>5 ± 0</td>
</tr>
<tr>
<td>BrP (mmHg)</td>
<td>18 ± 2</td>
<td>10 ± 3*</td>
<td>7 ± 3†</td>
<td>19 ± 4</td>
</tr>
</tbody>
</table>

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.
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 colleagues16 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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References