

itself could occur. Of question also is whether early post-mortem changes of cricopharyngeal and esophageal musculature constriction and/or localized edema of the area may have in some way contributed to superior conditions of occlusion in this experiment. A final question is whether sagittal localization of the nasogastric tube can preclude closure of the esophagus during cricoid compression, allowing the parasagittal lumen of the esophagus to remain open.

Prevention of aspiration cannot be guaranteed. Continual attention to and protection of the airway is mandatory in this setting, even with an inflated endotracheal tube cuff in the proper position.

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In reply:—Our investigation, performed in fresh cadavers, confirmed that firmly applied cricoid pressure is effective in sealing the esophagus around an esophageal tube against an intraesophageal pressure up to 100 cmH₂O.¹ Dr. Kempen raises important questions regarding extrapolating our data to the clinical setting. First, the force applied during cricoid pressure might have been greater than those forces used in clinical situations and, thus, might have compromised the glottic lumen necessary for successful endotracheal intubation or might have distorted and/or displaced the glottis laterally. Second, laryngoscopy may interfere with the efficacy of cricoid compression in occluding the esophagus around the nasogastric tube during endotracheal intubation, which was not done in our investigation.

We believe that these problems can be avoided by simply adhering to the original technique as described by Sellick.* The neck must be extended so that the esophagus will be directly posterior to the cricoid cartilage, and thus sealing of the esophagus would be easily accomplished by cricoid compression.* Furthermore, lateral displacement of the glottis can be prevented by using three fingers. The cricoid cartilage is palpated and lightly held between the second finger and the thumb while the index finger is placed on the anterior surface of the cricoid cartilage.* Pressure is increased by the index finger as consciousness is lost, with the cricoid maintained in its central position.* Unfortunately, Sellick's original description is not strictly

followed by many practitioners, and drawings in textbooks do not represent the maneuver as described by Sellick.

Interference with glottic patency (and intubation) can occur if excessive pressure is exerted, especially in pediatric patients.^{2,3} Unfortunately, there is no easy way of assessing the degree of cricoid pressure. In general, firm pressure is used to prevent gastric contents from reaching the pharynx. If cricoid pressure is used for preventing gastric inflation during bag-mask ventilation, especially in pediatric patients, gentle, rather than firm, pressure would be desirable.²

Laryngoscopy may interfere with the efficacy of cricoid pressure only if improperly performed and if excessive traction is applied by the blade.⁴ Recently, we have been successfully using cephalad displacement of the larynx as a technique to enhance visualization of the larynx in difficult intubations. Perhaps the same technique can be used while applying cricoid pressure in intubating patients with a full stomach. This will facilitate exposure of the glottis without interfering with the efficacy of the maneuver. In our investigation, the pharynx was visualized with a laryngoscope and at no time was the efficacy of cricoid pressure compromised.

The importance of emptying the esophagus in certain situations has previously been emphasized.⁵ However, a functioning nasogastric tube will help maintain a low intragastric pressure until the time of anesthetic induction, but should not be relied on as a guarantee of emptying the stomach. Therefore, if a rise in intragastric pressure occurs during induction, a functioning nasogastric tube will act as a "blow-off" valve while cricoid pressure will prevent gastric or esophageal contents from reaching the

* Sellick BA: The prevention of regurgitation during induction of anaesthesia. First European Congress of Anaesthesiology 89:1-4, 1962.

pharynx. In very rare entities such as megaesophagus and esophageal diverticulum, cricoid pressure may not be effective and may be hazardous. In retropharyngeal abscess, cricoid compression may result in rupturing the abscess and flooding the tracheobronchial tree, especially if excessive cricoid pressure is applied and, therefore, should be avoided.⁵

It is unlikely that early postmortem changes of the cricopharyngeal and esophageal musculature and/or localized edema might have contributed to superior conditions in our experiments. It is also unlikely that sagittal localization of the nasogastric tube hinders the efficacy of cricoid compression. In a previous study in anesthetized pediatric patients,⁶ roentgenographic films of nasogastric tubes previously filled with contrast material and tied at both ends showed displacement of the nasogastric tubes during cricoid compression. Because the maneuver was effective in sealing the esophagus around the nasogastric tubes, we believe that sagittal localization of nasogastric tubes is of no significance.

Finally, our investigation was not intended to compare rapid-sequence induction to awake intubation in the management of patients with a full stomach. We merely evaluated the efficacy of cricoid compression in obliterating the esophageal lumen in the presence of an esophageal tube.

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Isoflurane and Intracranial Pressure

To the Editor:—Grosslight *et al.*¹ have shown that intracranial pressure rose in six of fourteen patients who were paralyzed, breathing 70% nitrous oxide, and given isoflurane, 1% inspired, for several min at the onset of craniotomy. If one is going to ascribe the rise in intracranial pressure to an effect of isoflurane, as the authors tentatively conclude, then at least two sets of conditions must be met—which were not.

First, a control group consisting of patients not given isoflurane is needed. It was not appropriate to use patients given isoflurane but whose intracranial pressure did not rise as a control group. What happens to intracranial pressure in paralyzed patients whose general anesthesia is "maintained with nitrous oxide, 70% in oxygen" during the beginning of a craniotomy? Or, is the answer given in figure 1, which shows a clear increase in both systemic arterial as well as intracranial pressures? And how does one reconcile the *increased* systemic arterial pressure

shown in figure 1 with the *decrease* of 30 mmHg in mean arterial pressure shown in table 1?

Second, steady-state conditions should apply, and they clearly do not in this study. The patients were studied at a time when alveolar and arterial isoflurane concentrations were dynamically changing, rather than in a steady state, not to mention the confounding influence of the surgical stimulation, which commenced at the same time isoflurane was added to the inspired mixture.

Although these unpremedicated patients received thiopental, 3 mg/kg for induction and then 2 mg/kg for tracheal intubation, along with 1.5 mg/kg of lidocaine, considerable time had to elapse prior to skin incision for craniotomy. The airway had to be secured, the head turned to one side and "prepped and draped," a burr hole made, a subarachnoid bolt inserted aseptically, and the head repositioned to the other side and prepped and draped at the very minimum. This does not account for