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The Efficacy of Cricoid Pressure in Preventing Regurgitation of Gastric Contents

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One of the most challenging situations confronting the anesthesiologist is the management of the patient with a full stomach. Among the more popular methods of inducing general anesthesia in such patients is the "crash induction," utilizing rapid-acting intravenous induction agents and muscle relaxants combined with cricoid pressure to prevent regurgitation and allow rapid endotracheal intubation. The use of cricoid pressure to prevent regurgitation in the unconscious, paralyzed patient was first reported by Sellick[1] in 1961; since that report few or no data have been offered to indicate exactly how much pressure from within the esophagus is necessary to overcome cricoid compression and result in regurgitation.

The present study, performed in canine and human postmortem specimens, was undertaken to determine quantitatively the efficacy of cricoid pressure in preventing regurgitation of gastric contents.

Methods

Two canine and five human cadavers were studied in similar fashion: the stomach was exposed, and a plastic catheter (i.d. 5 mm) was inserted into the distal esophagus through a small incision near the gastroesophageal junction. A tape was placed around the distal esophagus to secure the catheter and form a water-tight seal. The catheter was connected to a four-way stopcock system to allow instillation of water into the esophagus or measurement of esophageal pressure with a water manometer (fig. 1). Water was instilled into the esophagus in 50-ml increments (10 ml in one child) while firm pressure was applied to the cricoid cartilage, displacing it posteriorly against the body of the sixth cervical vertebra. Following each instillation of water esophageal pressure was measured. When water was observed to leak into the pharynx despite firm cricoid pressure, instillation was stopped and the pressure recorded.

Results

The first canine specimen accommodated 200 ml of water and developed 70 cm H_2O pressure before leakage occurred. In the second, 80 cm H_2O pressure developed following instillation of 250 ml of water; then "regurgitation" began to occur. Both of these animals had been sacrificed at the termination of unrelated experiments and were studied prior to the onset of rigor mortis.

In the five human cadavers pressures necessary to overcome cricoid compression varied from 50 to 94 cm H_2O (mean, 74 cm H_2O), and regurgitation did not occur in every case even at high pressures. Volumes varied from 30 to 150 ml, but the 30-ml volume produced a pressure of 94 cm H_2O, the subject having been a 5-year-old child. The lowest pressure (50 cm H_2O), recorded in an obese 68-year-old man, represented leakage at the point of entrance of the catheter, rather than into the pharynx. This was the only case in which it was not possible to achieve a water-tight seal around the catheter. All human specimens had died several hours prior to study.

Discussion

The findings of this study indicate that in cadaveric specimens cricoid compression can provide protection from intraesophageal pres-

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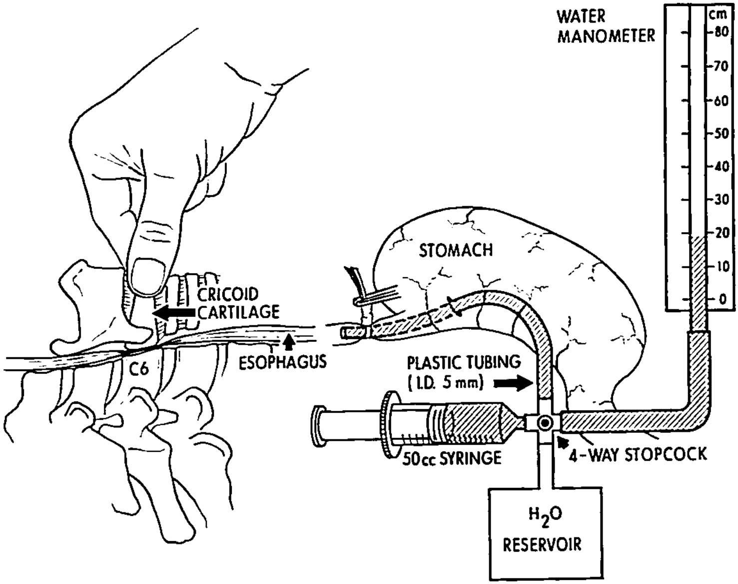

FIG. 1. Schematic representation of the method used to measure esophageal pressure during cricoid compression.

sures of at least 50 cm H_2O, and often considerably more. Volumes accommodated by the human specimens were somewhat less than those in the dogs which may in part reflect the fact that the canine specimens were fresher and hence, more pliable. Conceivably, therefore, in patients the esophagus will accommodate a greater volume than in cadavers. It is doubtful that any great difference in the pressures necessary to overcome cricoid compression will be seen in patients compared with cadavers, as evidenced by the comparable pressures achieved in the fresh canine specimens in spite of the greater volumes required.

Having demonstrated that cricoid compression can prevent regurgitation from esophageal pressures of 50 cm H_2O or more, it is necessary to consider how much intragastric pressure can reasonably be anticipated in the patient with a full stomach. In patients who have been fasted and submitted to routine anesthesia and surgery, intragastric pressures most often are below 10 cm H_2O,[2,3,4] although figures of 15–16 cm H_2O are not infrequent.[4] In patients with distended abdomens (gravid uterus, supine position) O'Mullane[2] measured pressures of 5 to 18 cm H_2O. Few data are available to indicate how high intragastric pressure may be in the traumatized patient who has recently ingested a full meal. However, since trauma, pain, and anxiety are associated with delayed gastric emptying time and reduced motility,[5] it is reasonable to assume that in the absence of mechanical overdistention (presence of large quantities of air, blood, etc.) gastric pressures are very likely to be much less than 50 cm H_2O in these patients.

Intragastric pressure may be greatly elevated under certain conditions. Belching has been demonstrated to produce gastric pressures well over 20 cm H_2O,[3] and vomiting is associated with pressures well in excess of 60 cm H_2O.[3] Fasciculations caused by depolarizing muscle relaxants can raise gastric pressure to more than 20 cm H_2O and, in some cases, more than 40 cm H_2O.[4,6,7] However, total paralysis provided by adequate doses of relaxants can prevent the rise in gastric pressure caused by vomiting, and there is now evidence that small doses of nondepolarizing muscle relaxants given three to six minutes prior to depolarizing agents will prevent the increased gastric pressure observed during fasciculations.[7]

The proper selection of patients is very important when considering the use of crash induction with cricoid pressure to prevent regurgitation. The patient must be evaluated in terms of his ability to tolerate a large dose of intravenous inducing agent, since it is of paramount importance that anesthesia be induced rapidly to prevent struggling and excitement. The anesthesiologist must also make a judgment as to the ease with which a given patient can be intubated. Should any of the commonly-recognized barriers to easy intubation exist (very prominent incisors, receding mandible, facial fractures, etc.), alternative methods of inducing general anesthesia should be considered. Since facility at intubation is so frequently related directly to the skill of the anesthesiologist, this technique appears to be too risky for neophytes except under the closest possible supervision.

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References

1. Sellick, B. A.: Cricoid pressure to control regurgitation of stomach contents during induction of anaesthesia, Lancet 2: 404, 1961.
2. O'Mullane, E. J.: Vomiting and regurgitation during anesthesia, Lancet 1: 1209, 1954.
3. Marchand, P.: A study of the forces productive of gastro-oesophageal regurgitation and herniation through the diaphragmatic hiatus, Thorax 12: 189, 1957.
4. Roe, R. B.: The effect of suxamethonium on intragastric pressure, Anaesthesia 17: 179, 1962.
5. Bannister, W. K., and Sattilaro, A. J.: Vomiting and aspiration during anesthesia, Anesthesiology 23: 251, 1962.
6. Andersen, N.: Changes in intragastric pressure following the administration of suxamethonium, Brit. J. Anaesth. 34: 363, 1962.
7. Miller, R. D.: Inhibition of succinylcholine-induced increased intragastric pressure by non-depolarizing muscle relaxants. *In* Abstracts of Scientific Papers, 1969 annual meeting, The American Society of Anesthesiologists.

The Search for Better Anesthetic Agents: Clinical Investigation of Ēthrane

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There are currently available at least four potent inhalation anesthetic agents whose human and animal pharmacology is well known. Each of these drugs, however, has limitations, and therefore none is an ideal anesthetic agent. Cyclopropane and ether, once the mainstays of anesthesia, have been replaced largely by either halothane, usually given with nitrous oxide, or, to a smaller extent, methoxyflurane with nitrous oxitde. A prime consideration in the widespread acceptance of halothane is its nonflammability; but the lower incidences of postoperative nausea, vomiting and hangover are important factors. The absence of sympathetic stimulation, now regarded favorably, was once considered a drawback by those ac-

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