

# Clinical Reports

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## The Relationship between Intragastric and Lower Esophageal Sphincter Pressures during General Anesthesia

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"Silent" regurgitation of gastric contents has been reported to occur in 25-70 per cent of patients having general anesthesia,<sup>1,2,3,14</sup> with as many as 76 per cent having tracheal aspiration.<sup>4</sup> Mortality rates following massive aspiration may be as high as 70 per cent.<sup>5</sup> A strong direct relationship between anesthetic agent, patient position, and operative site has been shown.<sup>6</sup>

As a result of recent advances in manometric methods, considerable information concerning lower esophageal sphincter function and dysfunction in conscious subjects is now available.<sup>7,8</sup> To date, however, there has been no such study of gastroesophagopharyngeal pressure gradients and responses of lower esophageal sphincters to increased intragastric pressure in the anesthetized state. It was for this purpose that this investigation was undertaken.

### METHOD

Fifteen patients undergoing lower abdominal operations, such as abdominal hysterectomy and lower-urinary-tract surgery, known to be free from symptoms of reflux, regurgitation, and hiatus hernia, were chosen for this study. Patients were informed about the study and consent obtained. The age range of the patients was 22-56 years (average 37 years); six were male and nine, female. Mean anesthesia time was  $98 \pm 36$  minutes.

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### Anesthesia

Premedication consisted of morphine, 0.15 mg/kg, im, and atropine, 0.008 mg/kg, 90 minutes before anesthesia. Thiamylal, 3.5 mg/kg, and succinylcholine, 1.0 mg/kg, iv, facilitated induction of anesthesia and endotracheal intubation. Anesthesia was maintained with halothane, 0.75-1.0 per cent in nitrous oxide (3 l) and oxygen (2 l) in a semiclosed circle absorption system. Dimethyltubocurarine, 0.1-0.2 mg/kg, was administered iv 5 minutes before incision of the skin. Its action was reversed with neostigmine, 0.03 mg/kg, and atropine, 0.017 mg/kg, after skin closure. Adequacy of relaxation and reversal was monitored with a peripheral-nerve stimulator (Block-Aid Monitor).

Respiration was controlled with Emerson volume respirator using tidal volumes of 10-12 ml/kg.

### Manometric Measurement

Pressure was measured with a thin polyethylene balloon with a diameter of 5 mm and a length of 7 mm, located at the end of a hard polyethylene tube with a diameter of 2 mm. The tube was filled with water and connected to a pressure transducer (Statham Model P23 DC). Three pressures were continuously displayed and recorded on a Sanborn Model No. 956A.

Intragastric, lower esophageal and pharyngeal pressures were measured continuously at the end of expiration during the whole course of the anesthesia, and analyzed in the following three periods: 1) after intubation, before the operation was started; 2) after the abdomen was opened; 3) after the abdomen was closed, following reversal of the muscle relaxant.

Three of these polyethylene tubes were inserted separately. The distal balloon measured gastric pressure; the middle balloon, lower esophageal sphincter pressure; the proximal balloon, pharyngeal pressure.

The probe was inserted through the mouth and the lower esophageal high-pressure zone was detected with the middle balloon with a pull-through technique during expiration, as described by Winans.<sup>9</sup>

Since the patients in this study were under general anesthesia, the balloon technique, rather than the technique of constant infusion of water, was used for safety purposes.

## RESULTS

The mean resting intragastric pressure before incision of the skin was  $9.8 \pm 2.0$  mm Hg, which is close to the mean resting intragastric pressure in conscious subjects.<sup>18</sup> This decreased to  $6.2 \pm 1.0$  mm Hg on opening the abdomen and increased to  $17.1 \pm 1.9$  mm Hg after the abdomen was closed following administration of neostigmine and atropine.

Before and after the abdomen was opened, the mean resting lower esophageal sphincter pressures were  $20.2 \pm 6.0$  and  $16.2 \pm 4.8$  mm Hg, respectively. This pressure increased to  $26.2 \pm 5.8$  mm Hg after relaxant reversal.

It is interesting that when the abdomen was opened, lower esophageal sphincter pressures in two patients were 7 mm Hg, and intragastric pressures, 5 mm Hg. Sudden manual gastric compression produced regurgitation in one of these two cases, in which intragastric pressure was 53 mm Hg. No regurgitation occurred in the remaining 14 cases, even though intragastric pressures in seven were 108 mm Hg or higher. Regurgitation was proven by methylene blue dye, which had been introduced into the stomach prior to manipulation.

Before and after the abdominal cavity was opened, the correlation coefficients for intragastric pressure and lower esophageal sphincter pressure were highly significant, 0.75 and 0.62, respectively.

## DISCUSSION

It has been shown that the barrier to reflux of gastric contents at the gastroesophageal junction is dependent upon the intrinsic strength of the physiologic lower esophageal sphincter.<sup>10,11</sup>

Biancani *et al.*<sup>12</sup> recently demonstrated that pressures in competent sphincters were twice as high as pressures in incompetent sphincters, and that an anticholinergic drug decreased the pressure response to half the control value.

Vomiting and regurgitation have often occurred during stormy inductions of anesthesia, and at the end of anesthesia.<sup>4,5,11</sup>

The higher incidence of regurgitation with the  $N_2O$ -narcotic-relaxant technique may be related

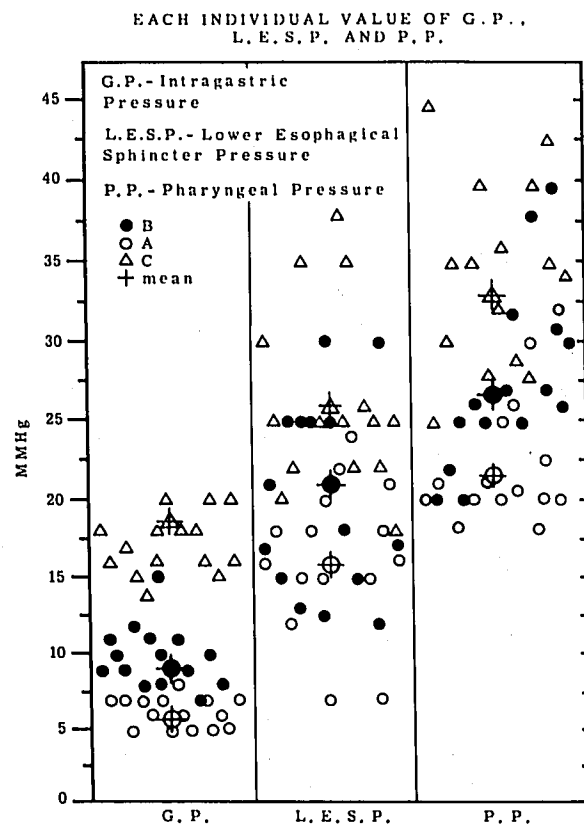


FIG. 1. Correlation coefficients. B.O. = before abdominal cavity was opened; A.O. = after abdominal cavity was opened; A.C. = after abdominal cavity was closed. \* $P < 0.05$ ; \*\* $P < 0.01$ .

to lower esophageal sphincter incompetence resulting from diaphragmatic paralysis, and to increases in intragastric pressure resulting from increases in abdominal muscle tone as analgesia and muscle relaxation intermittently change.<sup>13</sup> High gastric-pharyngeal pressure gradients can occur at these times, as they can during upper-abdominal operations or in the prone position.<sup>6</sup> The lower incidence during nitrous oxide-halothane anesthesia may be related to persistent competence of the lower esophageal and pharyngeal sphincters.<sup>15</sup>

One important characteristic of the lower esophageal sphincter is its ability to increase its pressure in response to increases of intragastric pressure as a protection against gastroesophageal reflux.<sup>16</sup> However, a large increase of intragastric pressure (to 80–100 mm Hg) can produce regurgitation.

In this study, no regurgitation occurred in seven cases in which intragastric pressures were 108 mm Hg or higher, and the control lower esophageal sphincter pressures were all above 10 mm Hg. When lower esophageal sphincter pressures were less than 10 mm Hg, the response of the lower esophageal sphincter to increased intragastric pressure

was much weaker, and thus inadequate to prevent gastroesophageal reflux.<sup>17</sup> The lower esophageal sphincter pressure in the case in which regurgitation occurred with an intragastric pressure of 58 mm Hg was 7 mm Hg.

The highest intragastric, lower esophageal sphincter, and pharyngeal pressure, 20, 38, and 45 mm Hg, respectively, were observed after administration of neostigmine and atropine. Neostigmine could be responsible for these findings.<sup>18</sup> Atropine, however, in doses of 0.015–0.025 mg/kg, reduces lower esophageal sphincter pressure markedly without affecting gastric tone,<sup>17,18</sup> which may explain the observed decrease in the lower esophageal sphincter pressure/intragastric pressure ratio from 2.335 to 1.56.

It can be concluded from this study that the physiologic gastroesophagopharyngeal pressure gradient and lower esophageal and pharyngeal sphincter competence may be well maintained during nitrous oxide–halothane anesthesia.

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