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Intravenous Atropine Rapidly Reduces Lower Esophageal Sphincter Pressure in Infants and Children

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The lower esophageal sphincter (LES) is the most significant component of the physiological barrier preventing reflux of the stomach contents into the esophagus. Hence, the competence of this mechanism is the most important factor in the prevention of regurgitation and aspiration during anesthesia. The effect on the LES of drugs which may be used before or during anesthesia is also, therefore, of great importance.

The effect of atropine on the LES in adult patients has been measured by several investigators,[1–4] with conflicting results. The effect of atropine on the LES of infants and children has not been previously described.

MATERIAL AND METHODS

Sixteen infants and children, eight boys and eight girls, ranging in age from 8 months to 17 yr (mean age 7 yr) were studied during diagnostic esophageal motility studies (table 1). All the studies were performed during general anesthesia using nitrous oxide and halothane with spontaneous ventilation. Atropine was not given until completion of the initial studies, and no preanesthetic medication was used. No muscle relaxants were administered; the patients were intubated under halothane anesthesia.

Pressure tracings were obtained using a quadruple lumen, polyvinyl, microlumen catheter with 0.6-mm diameter side holes at 1, 3, 5, and 8 cm from the distal tip. During recording, three lumens were infused with debubbled sterile water at 0.6 ml/min using a high fidelity, pneumohydraulic capillary infusion system (Arndorfer Medical Specialities, Greendale, WI). The intraluminal pressures were measured using Statham P231D transducers and recorded.

The quadruple catheter assembly was inserted through the nasopharynx into the esophagus and stomach. During slow withdrawal of the catheter, the LES is identified as the zone between the initial rise above resting gastric pressure and the first below base-line negative inspiratory pressure swing measured in the esophagus. Repeated withdrawal pressure tracings were obtained to determine the mean initial LES pressure. This was measured at mid-respiration as the difference above resting intragastric pressure. The catheter was then positioned to continuously record LES pressure, and atropine 0.02 mg/kg was administered intravenously. The record was continued over 2–3 min after a tachycardia was established. Two repeat withdrawal tracings were then obtained to confirm the change from initial LES pressure. In the total group of 16 patients, 64 pairs of measurements were made; before and 30–120 s after iv atropine administration.

Statistical analysis was performed using Student's *t* test for paired samples.

RESULTS

Before atropine, mean LES pressure was 21.3 ± 9.5 mmHg; after atropine, this was reduced to 14.4 ± 7.7 mmHg ($t = 4.85$, $P = 0.0006$). A significant decrease in LES pressure was also seen in two subgroups of these patients: those with a normal initial LES pressure (group 1), and those with abnormally low initial LES pressure (group 2) (table 2). The reduction in the LES pressure occurred over less than 1 min, and was simultaneous with the onset of tachycardia (fig. 1).

DISCUSSION

Previous reports of the effect of atropine on LES pressure in adults have been conflicting. Clark and Riddoch[1] reported that atropine increased the resistance to reflux, which led Salem *et al.*[5] to conclude that atropine decreases the risk of regurgitation and aspiration of gastric contents in children. However, Hill[2] reported that atropine had little effect on LES pressure measured intraoperatively, and Cotton and Smith[3] demonstrated a significant decrease in LES barrier pressure 5 min after atropine administration which persisted for 1 h.

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TABLE 1. Patients Studied

Symptoms or Diseases Present	Number of Patients*
Gastroesophageal reflux	13
Cystic fibrosis	5
Abdominal pain	3
Failure to thrive	3
Hematemesis	1
Metabolic disease	1

* Some patients had more than one symptom or disease.

TABLE 2. Effect of Atropine on Lower Esophageal Sphincter Pressure (Mean ± SD)

	Group 1	Group 2	All Patients
Initial LES pressure	26.5 ± 8.2	12.7 ± 2.7	21.3 ± 9.5
After iv atropine	18.4 ± 6.9	7.7 ± 2.9	14.4 ± 7.7
Paired *t* value:	3.329	3.66	4.30
P	.0088	.0146	.0006

Group 1 = normal initial LES (>16 mmHg); group 2 = low initial LES (≤16 mmHg).

Brock-Utne *et al.*[4] also documented decreases in LES pressure following atropine administration.

The children who were studied in this present investigation were not normal healthy children, but had a history of repeated vomiting, or other symptoms of gastroesophageal reflux. However, ten of the children were found to have a normal initial LES pressure of over 16 mmHg. These children and all but two of the subgroup with an abnormally low initial LES pressure consistently showed a marked decrease in LES pressure which occurred immediately following iv atropine. Thus, the effect of atropine in relaxing the LES is independent of the initial sphincter pressure. This demonstrates that those patients with low initial LES pressure, who might be considered especially at risk for regurgitation, will have a further decrease in LES pressure following iv atropine.

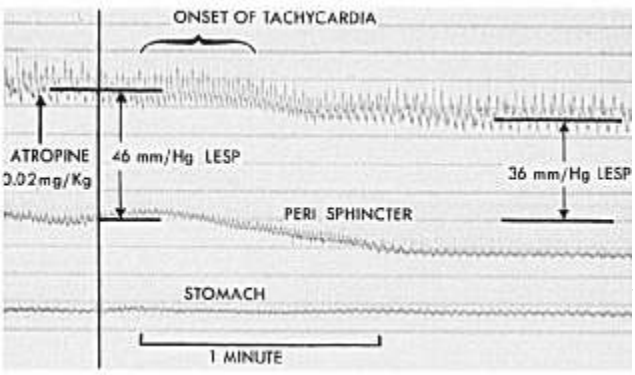

FIG. 1. Tracing of lower esophageal pressure following administration of atropine 0.02 mg/kg intravenously. The change in heart rate can be clearly seen in the upper trace.

The measurements which we made were not performed during the induction of anesthesia; they were made after anesthesia had been established with nitrous oxide and halothane. However, it is not uncommon practice to induce anesthesia for pediatric patients with such a combination of nitrous oxide and halothane, and then to secure an intravenous route and administer atropine iv. We, therefore, consider that the changes in LES pressure which were demonstrated are probably pertinent to this regime for induction of anesthesia. Furthermore, as vagal innervation has been shown to be important in the maintenance of resting LES pressure,[6,7] drugs which block the vagus, such as atropine, must be expected to lower LES pressure if given as a part of any induction sequence.

We conclude that iv atropine rapidly relaxes the LES and may increase the risk of regurgitation of stomach contents during induction of anesthesia. If iv atropine is administered to patients at risk of regurgitation during induction of anesthesia, appropriate measures to prevent pulmonary aspiration (*e.g.*, cricoid pressure) should be instituted as soon as the atropine is injected.

REFERENCES

1. Clark CG, Riddoch ME: Observations on the human cardia at operation. Br J Anaesth 34:875–883, 1962
2. Salem MR, Wong AY, Collins VJ: The pediatric patient with a full stomach. ANESTHESIOLOGY 39:435–440, 1973
3. Hill LD: Intraoperative measurement of LES pressure. J Thorac Cardiovasc Surg 75:378–382, 1978
4. Cotton BR, Smith G: Single and combined effects of atropine and metoclopramide on the lower oesophageal sphincter pressure. Br J Anaesth 53:869–874, 1981
5. Brock-Utne JC, Rubin J, Downing JW, Dimopoulos GE, Moshal MG, Naiker M: The administration of metoclopramide with atropine. Anaesthesia 31:1186–1190, 1976
6. Higgs B, Ellis FH: The effect of bilateral supranodosal vagotomy on canine esophageal function. Surgery 58:828–834, 1965
7. Lind JF, Crispin JG, McIver DK: The effect of atropine on the gastro-esophageal sphincter. Can J Physiol Pharmacol 46:233–238, 1968