days postoperatively (fig. 1) was interpreted as showing part of the stethoscope in the duodenum, later review of the film, together with the location of the stethoscope at gastroscopy, indicated it never left the stomach. Thus, we would recommend elective removal of an errant esophageal stethoscope with an endoscope upon recognition of the problem, rather than nonintervention in the hope of spontaneous distal passage.

In summary, we report a case of inadvertent passage of an esophageal stethoscope into the stomach of an anesthetized patient. Diagnosis was delayed until the sixth postoperative day and was aided by the characteristic "booster rocket" silhouette of the proximal end of the esophageal stethoscope (fig. 1). Removal was accomplished without difficulty 5½ weeks after insertion of the stethoscope. Elective removal, rather than expectant hope for passage of an esophageal stethoscope located in the stomach, is recommended.

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Atracurium: Hypotension, Tachycardia and Bronchospasm

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Histamine release following iv administration of atracurium can cause hypotension and tachycardia.[1] We describe such a case where significant tachycardia, hypotension, and bronchospasm occurred following an iv bolus of atracurium.

Report of a Case

A 34-year-old, 48-kg, ASA physical status I woman was scheduled for laparoscopy, lysis of adhesions, and possible laparotomy for infertility. She gave a negative history for drug allergies. Following premedication with hydrocortisone 100 mg, morphine sulfate 8 mg and glycopyrrolate 0.4 mg im, she received fentanyl 100 μg and diazepam 5 mg iv prior to induction of anesthesia with thiopental 425 mg iv following which BP was 110/80 mmHg, heart rate 100 bpm with controlled respirations via face mask. Atracurium 30 mg iv was administered prior to tracheal intubation. Less than 30 s later, heart rate increased from 100 to 150 bpm, systolic BP decreased from 110 to 55 mmHg, and ventilation via face mask became difficult. The ECG monitor showed a supraventricular tachycardia. Skin flushing was not observed. Carotid massage, edrophonium 10 mg, and methoxamine 6 mg in divided doses iv were without effect. Her trachea was intubated easily and auscultation of her lungs revealed inspiratory and expiratory wheezing bilaterally. The systolic BP was now 60 mmHg and heart rate 150 bpm. One hundred per cent oxygen was given and the rate of iv fluids increased. An iv neosynephrine drip 40 μg/ml to a total of 400 μg was given, resulting eventually in a BP of 110/50 mmHg and heart rate of 110 bpm. At this time, pH_a was 7.31, Pa_{O_2} 485 mmHg, Pa_{CO_2} 47 mmHg, and HCO_3^- 23.2 mEq/l. The return of her cardiovascular variables toward preatracurium values occurred 20 minutes following iv atracurium. At this time, the bronchospasm resolved spontaneously and the neosynephrine was discontinued. It was decided to proceed with the planned surgical procedure.

During the 2-h operation, anesthesia was maintained with enflurane, N_2O, O_2, and intermittent iv injections of pancuronium totaling 3 mg. At the end of surgery, muscular blockade was reversed without incident, following the administration of neostigmine 4 mg and glycopyrrolate 0.4 mg iv while monitoring neuromuscular activity with a nerve stimulator. Her recovery room and postoperative courses were unremarkable.

Discussion

This patient's initial increase in heart rate was interpreted as a spontaneous supraventricular tachycardia (SVT). It was treated with carotid massage and iv edrophonium. When the systolic BP of 55 mmHg was noted, methoxamine and neosynephrine were administered iv. The appearance of bronchospasm suggested

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an etiology other than SVT, and the possibility of histamine release was entertained even though cutaneous flushing was not observed. The BP started to increase, and further drug therapy did not appear to be indicated. Bronchospasm resolved spontaneously.

Significant cardiovascular changes following the iv atracurium injection at doses of 0.5–0.6 mg/kg have been reported. These consisted of decreases in arterial blood pressure to 86.7% and 79.5% of control and heart rate increases to 105.5% and 108.3% of control.[1] The changes in arterial BP and heart rate disappeared within 5 min and usually were associated with flushing of the face, suggesting the possibility of histamine release. In a study with contrasting results, Payne and Hughes[2] found no significant cardiovascular changes at doses up to 0.6 mg/kg. The speed of injection may be important in explaining these disimilar results.[3] In our patient, a dose of 0.6 mg/kg was administered over a period of approximately 5 s. The patient's body weight preoperatively was estimated to be higher than her actual weight as recorded on her hospital chart. No attempt was made to modify the speed of the atracurium injection.

The atracurium package insert recommends 0.5 mg/kg to be the maximum dose for tracheal intubation. With a higher dose as used in this patient, the release of histamine would not have been unexpected, retrospectively. However, the return of her cardiovascular variables toward normal should have occurred within a 5-min time period.

Histamine is a naturally occurring vasoactive amine with varying distribution from tissue to tissue within the human body. Among its pharmacologic actions are an increase in myocardial contractility and automaticity. Peripherally, histamine acts as a potent vasodilator. Thus, hypotension occurs, as does tachycardia, either as a reflex effect related to the hypotension or a direct effect, depending on the plasma histamine level. Dilatation of cutaneous blood vessels with increased microvascular permeability results in flushing of the skin and increases in skin temperature. Stimulation of bronchial musculature results in bronchoconstriction. All of these effects are dependent on plasma histamine levels.[4]

Following the administration of clinical doses of some neuromuscular blocking drugs, histamine may be released in quantities sufficient to increase circulating plasma histamine.[5,6] These drugs include *d*-tubocurarine, succinyl-choline, gallamine, decamethonium, alcuronium, pancuronium, and atracurium.[5–7]

In our patient the tachycardia, hypotension, and bronchospasm may be explained by the actions of histamine. Of concern is the length of time that elapsed before the return to induction levels of BP, heart rate, and the resolution of wheezing. The use of edrophonium and methoxamine to treat the initially perceived supraventricular tachycardia with hypotension were without effective cardiovascular response. However, neither of these agents are specific H_1 or H_2 histamine receptor antagonists. Simultaneous blocking of H_1 and H_2 histamine receptors can counteract the vasodilator response to histamine in forearm blood flow in humans.[8] In subsequent animal studies, H_1–receptor antagonists block the immediate response and H_2–receptor antagonists block the sustained response to histamine.[9]

Was the response of this patient to atracurium an anaphylactic or anaphylactoid reaction? An anaphylactic reaction is associated with antibody formation or activation of the complement system by an alternate pathway. An anaphylactoid reaction does not involve antibodies. However, we did not obtain plasma histamine levels or subsequent IgE or IgG antibody levels. There was no previous history of exposure to atracurium. If there is an associated reagin that can occur or was triggered to form an antigen by atracurium, it is not presently known.

In summary, this case demonstrates significant tachycardia, hypotension, and bronchospasm following a large intubation dose of atracurium. The presently proposed mechanisms for this response include histamine release and/or a possible allergic reaction (anaphylactoid *vs.* anaphylaxis).

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