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Induction of Thiopental Anesthesia without Tracheal Intubation in a Patient with Hiatal Hernia: Use of Esophageal *p*H Monitoring

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Presence of hiatal hernia can be a vexing problem in patients in whom gradual induction of anesthesia without tracheal intubation is preferred.[1] At our institution the protocol for evaluation of patients with intractable epilepsy for surgical removal of epileptic foci includes a thiopental test with depth electrodes placed into suspect areas of the brain (*e.g.*,hippocampus). This test entails administration, to an otherwise unmedicated patient, of thiopental 25 mg every 30 s until a dose sufficient to abolish the corneal reflex or 1 g (whichever is less) is administered.[2] This protocol precludes the use of additional drugs needed for insertion of an endotracheal tube. We recently cared for a patient with a history of symptomatic hiatal hernia and intractable epilepsy who required a thiopental test.

CASE REPORT

A 27-yr-old woman was admitted for evaluation of medically intractable epilepsy. She had complex partial seizures dating back to infancy following vaccination for diptheria, pertussis, and tetanus. Seizures occurred as often as six to 12 times per day. Other medical problems included obesity, hiatal hernia, and peptic ulcer disease. The diagnosis of hiatal hernia was based on the patient's description of symptomatic reflux and an endoscopic diagnosis made 11 yr earlier. She complained of epigastric discomfort while in the supine position that improved when the head of her bed was elevated 45 degrees. Medications included carbamazepine, levothyroxine, and Mylanta®. She underwent insertion of depth electrodes on the second hospital day. For this procedure rapid sequence induction of anesthesia was employed using 450 mg of thiopental, 140 mg of succinylcholine, preceeded by 3 mg d-tubocurare and cricoid pressure.[3] Subsequently, anesthesia was uneventfully maintained with isoflurane, nitrous oxide, and fentanyl.

Because of concerns regarding the severity of her gastric reflux, she underwent ambulatory esophageal *p*H monitoring for 24 h. This test disclosed mild esophageal reflux that was most pronounced at night while supine and postprandially. She thus seemed to be at risk of gastric reflux and acid aspiration with induction of thiopental anesthesia.

The night before and the morning of her thiopental test which was scheduled for 10 A.M., she received ranitidine 150 mg orally. On the morning of her test she also received metoclopramide 10 mg iv and had a Salem sump nasogastric tube inserted that was placed to low intermittent suction.[4] This nasogastric tube was one modified by Sensatronix Medical Systems Incorporated (Plainfield, NJ) which had an antimony *p*H probe on the tip that was electrically connected to a monitor that continuously displayed *p*H at the tip of the nasogastric tube.

Prior to induction of anesthesia the nasogastric tube was suctioned and the tip withdrawn to approximately midesophageal position. The thiopental test was performed with monitoring of the depth electrode electroencephalogram in the Epilepsy Center Monitoring Suite. Additional monitoring consisted of EKG, automatic indirect blood pressure, pulse oximetry, and precordial stethoscope auscultation in the suprasternal notch. The test was performed with the head of the bed elevated 30 degrees. While breathing spontaneously she received 25 mg of thiopental every 30 s, receiving a total of 1 g. Airway management was not problematic as she breathed spontaneously throughout the test and did not develop airway obstruction. Oxygen was administered *via* face mask at 10 l/min and cricoid pressure[5] was applied continuously throughout the 35-min procedure. Esophageal *p*H varied sub-

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stantially, remaining >4 for most of the test. There were several occasions where *p*H did decrease to < 2 but each such episode was short lived (less than 30 s) with a return of *p*H to <4. There was no evidence on continuous auscultation of the airway of fluid accumulation. In addition to occurring spontaneously, *p*H decreases also occurred with episodes of coughing on emergence, but again were short lived. Because of the short nature of these *p*H decreases and no evidence of fluid accumulating in the airway we elected not to intubate this patient's trachea (and therefore abort the test). With return of responsiveness to verbal stimulation cricoid pressure was released and, when she was fully awake, the esophageal tube was removed. No postanesthetic problems occurred and she returned to the ward without need for supplemental oxygen.

Discussion

Hiatal hernia is a problem for which rapid sequence induction to prevent passive reflux and regurgitation of acidic gastric contents [5–7] is usually employed.[8–10] We were thus faced with conflicting therapeutic priorities. An immediately prethiopental test "awake" intubation with iv sedation and topical anesthesia of the airway was not an option because it is not known how these drugs, once transmucosally absorbed, may affect the test results (EEG). We had two options. The first was to intubate her trachea facilitated by topical anesthesia or a rapid sequence induction. This would then have to be followed by an interval sufficient to permit elimination of anesthetic drugs to allow a valid thiopental test to be performed with the airway adequately protected. During this time she should have to receive no sedation and would have to be in a monitored setting wherein she could be assisted with clearance of secretions and monitored for urgent mechanical problems with the endotracheal tube. The second option was to perform the test with careful monitoring for signs of reflux, at which point the test would be aborted by a standard rapid induction of neuromuscular blockade and tracheal intubation. With the advent of recent technology that makes readily available esophageal *p*H monitoring, the latter approach seemed optimal.

The *p*H probe we used has a virtually instantaneous response time.‡ Thus, any regurgitating acidic gastric contents would have been instantly apparent and manifested by a rapid esophageal *p*H decrease. The safety of the approach we used presupposes that cricoid pressure will effectively prevent aspiration of passively regurgitated material in the time between detection of acidic regurgitation and tracheal intubation. This approach furthermore presupposes that active vomiting, for which cricoid pressure would not be expected to be effective, will not occur. Thus, the risks being weighed were: 1) the morbidity of resecting a seizure focus without a thiopental test *versus* 2) the probability of passive acidic regurgitation occurring with cricoid pressure not preventing aspiration of regurgitated material or of active vomiting occurring.

Evidence of acidic regurgitation did, in fact, occur. However, the observation that the esophageal *p*H rapidly increased thereafter was unexpected. The brevity of these episodes suggested that sustained regurgitation was not occurring. Airway auscultation did not suggest that a significant volume of this material was reaching the hypopharynx. Thus, intubation did not seem to be indicated. If *p*H had remained low for >30 seconds (empirically suggested) or there was evidence of hypopharyngeal fluid accumulation, we would have intubated the trachea using a rapid sequence induction of anesthesia and aborted the test followed then by evaluation for evidence of pulmonary acid aspiration.

This approach may have uses in other situations wherein rapid sequence induction may be relatively contraindicated such as patients with intracranial hemorrhage, intracranial hypertension, coronary artery disease, valvular heart disease, congestive cardiomyopathy, or undefined airway anatomy.

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