J Anesth (2012) 26:796–797
DOI 10.1007/s00540-012-1417-3

LETTER TO THE EDITOR

Altered anesthetic requirements and carbon dioxide setpoint in chronic airway obstruction

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Received: 18 April 2012 / Accepted: 7 May 2012 / Published online: 29 May 2012

Abstract Accidental ingestion of a foreign body into either tracheobronchial tree or esophagus is not an uncommon occurrence. However, there is limited literature available on sequelae of post foreign body ingestion—carbon dioxide set point and apneic threshold due to chronic respiratory acidosis. We report a case of chronic airway obstruction in a 14-month-old boy with prior history of battery ingestion and share our experience in the management.

Keywords Prolonged apnea · Stridor · Apneic threshold

To the Editor:

Accidental ingestion of a foreign body into either the tracheobronchial tree or esophagus is not an uncommon occurrence [1, 2]. However, there is limited literature available on carbon dioxide setpoint and anesthetic requirements in chronic respiratory acidosis. We report such a case with stridor and share our experience in its management.

A 14-month-old (9 kg) boy in respiratory distress was shifted to the operating room (OR) for emergency tracheostomy. He had ingested a battery 3 months earlier, resulting in corrosive injury to his esophagus. Respiratory difficulty present since this incident 3 months earlier had worsened in the past 24 h. Examination revealed stridor, tracheal tug, intercostal retraction, and inspiratory wheeze. Neck roentgenogram revealed subglottic tracheal narrowing (Fig. 1). His heart rate, noninvasive blood pressure, and SpO_2 were 136/min, 83/41 mmHg, and 92 %, respectively. Intermittent positive pressure ventilation (IPPV) with incremental sevoflurane in 100 % oxygen was administered subsequently, inducing anesthesia; this resulted in apnea at end-tidal CO_2 ($EtCO_2$) levels of 45 mm Hg. The trachea was intubated with an endotracheal tube (ETT) of 4 mm ID. Sevoflurane concentration was reduced to 2 %, following which an intravenous access was secured. Spontaneous breathing failed to return at $EtCO_2$ levels of 50 mm Hg, even with a further reduction of sevoflurane to MAC of 0.1. An arterial blood gas analysis revealed a pH of 7.35, pCO_2 level of 59 mmHg, bicarbonate of 32.6 mmol, and pO_2 of 302 mm Hg, suggesting compensated chronic respiratory acidosis. Return of spontaneous efforts occurred finally at a higher level of $EtCO_2$. The remainder of the procedure and his recovery were uneventful.

In upper airway obstruction, increased inspiratory resistance results in inadequate ventilation and CO_2 retention. CO_2 has anesthetic properties; MAC is estimated at 245 mmHg in dogs [3]. Hence, hypercapnia could itself increase the depth of anesthesia. Spontaneous respiration in a child with airway narrowing is maintained because of better airflow physics. Although no neuromuscular blocker was administered, apnea continued post induction; also it is difficult to adjudge if the tube has completely bypassed the stenotic segment. Even if ETT had bypassed the friable stenotic segment, the need of positive pressure could lead to pneumomediastinum with a possibility of mucosal damage.

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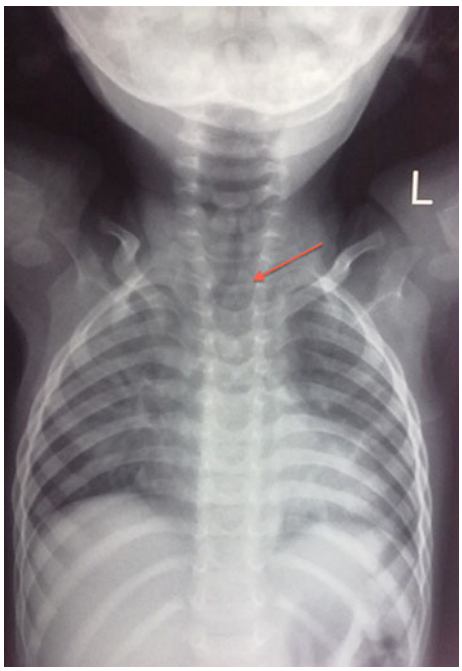

Fig. 1 Neck and chest neck roentgenogram show subglottic narrowing and dilation below constriction

Volatile anesthetics depress the ventilatory response to hypercapnea in a dose-dependent fashion [4]. A subanesthetic dose of sevoflurane has been found to not affect response to acute or sustained hypercapnia. Respiratory acidosis in chronic obstruction caused by raised CO_2 is compensated by an increase in bicarbonate. In such cases, apneic threshold shifts to a higher setpoint of CO_2 as was observed in our case. Hence, return of spontaneous breathing occurred only when high $EtCO_2$ was achieved.

Children compensate for their narrower upper airway by increasing upper airway neuromotor tone, via an increased central ventilatory drive [5]. We speculate that children with chronic airway obstruction lack these compensatory upper airway neuromotor responses. This case suggests that in pediatric chronic airway obstruction the apneic threshold under anesthesia is raised not only because of high $EtCO_2$ setpoint but also in response to the possible additive anesthetic effect of CO_2.

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