Pediatric Anesthesia 2007 **17**: 187–188 doi:10.1111/j.1460-9592.2006.02052.x

Case report

Difficult ventilation from increased abdominal pressure

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Summary

Airway management is one of the most important responsibilities of every anesthesiologist. A problematic situation arises when a patient is intubated correctly but not being ventilated. A case of a 4-year-old child who was involved in a car accident, presented for an exploratory laparotomy. Although she was successfully intubated, she could not be ventilated. The situation only improved after a large volume of fluid (urine) was drained from the abdomen.

Keywords: airway; intubation; intra-abdominal pressure

Introduction

Airway management is one of the most important responsibilities of every anesthesiologist. The best technique to secure a safe airway for maintaining ventilation is tracheal intubation. The disappointing situation is when a patient is intubated correctly but not being ventilated. A case of a 4-year-old female child who was involved in a car accident, presented for an exploratory laparotomy. Although she was successfully intubated, she could not be ventilated.

Case report

A 4-year-old female child was brought to the operating room for an exploratory laparotomy. On arrival, her Glasgow Coma Scale score was 11/15 and vital signs were pulse rate, 130 $b \cdot min^{-1}$; respiratory rate, 36 $b \cdot min^{-1}$ and blood pressure, 85/55. Her abdomen was severely distended and pulse oximetry showed an arterial saturation 80–85%, while she was spontaneously breathing oxygen via facemask.

Anesthesia was induced using thiopental, 5 $mg \cdot kg^{-1}$ and succinylcholine, 1.5 $mg \cdot kg^{-1}$. She was intubated with a cuffed tracheal tube size of 5 mm. Cricoid pressure was maintained until inflation of the cuff. After intubation, positive pressure ventilation started but the patient could not be ventilated. There was a continuous decline in arterial saturation and no endtidal CO_2 was detected. With continued Sellick maneuver the patient was extubated and immediately reintubated with another tracheal tube. The state of 'no ventilation' continued and patient became severely cyanosed. With the impression of extreme increase in the intra-abdominal pressure, we requested the surgeon to make a small incision on the patient's lower abdomen, which was performed as an emergency measure without proper prep and drape time. A large amount of fluid shot out, and immediately ventilation was possible with suctioning of the intra-abdominal fluid. Endtidal CO_2 rose to 11.5 kPa (85 mmHg) and arterial saturation returned to normal. The volume of fluid (urine) suctioned from

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the abdomen was 2.3 l. Analgesia and muscle relaxant were given and anesthesia maintained with isoflurane and oxygen until the bladder repair was completed. After reversal of anesthesia and muscle relaxant, the patient's trachea was extubated.

Discussion

The onset of general anesthesia, paralysis, a supine position, and increased intra-abdominal content, such as fluid may cause a progressive cephalad displacement of the diaphragm.

Induction of anesthesia is consistently accompanied by a significant (15–20%) decrease in functional residual capacity (FRC) (1–3), which usually causes a decrease in compliance (4). The maximum decrease in FRC appears to occur within the first minutes of anesthesia (1), but in the absence of any other complicating factors, does not seem to decrease progressively during anesthesia.

A supine position decreases FRC simultaneously because of a 4 cm displacement of the diaphragm by the abdominal viscera (1). The diaphragm separates the two compartments of markedly different hydrostatic gradients. On the thoracic side, pressure increases by approximately 0.25 $cmH_2O \cdot cm^{-1}$ of lung height (5,6) and on the abdominal side, by 1.0 $cmH_2O \cdot cm^{-1}$ of abdominal height (7). This means that in the horizontal position, progressively higher transdiaphragmatic pressures must be generated toward dependent parts of the diaphragm to keep the abdominal contents out of the thorax. In the unparalyzed patient, this tension is developed either by passive stretch and shape changes of the diaphragm (causing an increased contractile force) or by neurally mediated active tension. With acute muscle paralysis, neither of these two mechanisms can operate and a shift of the diaphragm to a more cephalad position occurs. The latter position may express the true balance of forces on the diaphragm, unmodified by any passive or active muscle activity. Increased intra-abdominal pressure, for example, as seen after rupture of the bladder resulting in the accumulation of a massive intra-abdominal volume of urine, will multiply the effects of these three factors, especially when it happens acutely. They cause functional residual capacity to be less than closing volume and decrease the time of safe apnea before hypoxia occurs. Moreover, it is an obvious cause of inability to ventilate the patient, even though correctly intubated.

When we are faced such a situation with massive intra-abdominal fluid, such as urine and blood, especially in pediatric patients, it is mandatory to ask a surgeon to drain or evacuate the abdominal cavity before induction of anesthesia.

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Accepted 30 May 2006

