

Two cases of progressive vocal cord closure during desflurane–remifentanil anesthesia relieved after administration of propofol

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To the Editor:

We report two adult cases of vocal cord closure during desflurane and remifentanil anesthesia that were relieved by propofol administration. A 56-year-old woman and a 63-year-old woman who underwent orthopedic hand surgery showed similar clinical courses.

General anesthesia was induced with thiopental at 3 mg/kg and remifentanil at 0.5 µg/kg/min in both patients. Then an i-gel supraglottic airway was inserted after giving 5 mg of rocuronium. Adequate mechanical ventilation was attained with no audible leak. After insertion of the i-gel, the vocal cords were fully opened (Fig. 1a) using a 5-mm fiberoptic. After that, an ultrasound-guided brachial plexus block was added. Anesthesia was maintained with 3–5 % desflurane and remifentanil at 0.15–0.2 µg/kg/min to keep the bispectral index (BIS) at 40–60. A muscle relaxant was not added after induction.

Before the start of surgery, the peak airway pressure gradually increased, while blood pressure, heart rate,

oxygen saturation, and BIS values did not change. The patient did not move, and spontaneous breathing was not observed at that time. The fiberoptic view through the i-gel showed progressive vocal cord closure (Fig. 1b, c). There were no findings indicating systemic muscle rigidity. Next, target-controlled infusion of propofol at 3 µg/ml was started, and desflurane was stopped. After that, the effect-site concentration of propofol was maintained at 1.8–2 µg/ml to keep BIS values at 40–60. Peak airway pressure was gradually decreased with the use of propofol, and the fiberoptic view showed opening of the vocal cords (Fig. 1d). The operation was finished with no airway problems, and the postoperative course was uneventful.

In the present cases, neither patient showed signs of inadequate anesthesia when the vocal cords were closed. Additionally, an ultrasound-guided brachial plexus block was appropriately performed in both cases. Therefore, the depth of anesthesia in both cases was considered to be appropriate.

Opioid administration can cause vocal cord closure [1], which commonly occurs during the induction of anesthesia by a rapid increase in concentration of the opioid. In contrast, we observed vocal cord closure during the maintenance phase of anesthesia with remifentanil at 0.15–0.2 µg/kg/min. The vocal cords did not reclose in the present cases, which may indicate that the closure seen in our cases was not caused by remifentanil.

Volatile anesthetics, especially desflurane and isoflurane, stimulate the airway by activating transient receptor potential A1 (TRPA1) in a dose-dependent manner [2, 3]. Isoflurane activates TRPA1 with an EC₅₀ of 0.18 mM [0.57 minimum alveolar concentration (MAC)]. Thus, less than 1 MAC of desflurane can induce airway stimulation. In contrast, propofol has been reported to decrease the occurrence of laryngospasm after laryngeal stimulation compared with sevoflurane [4].

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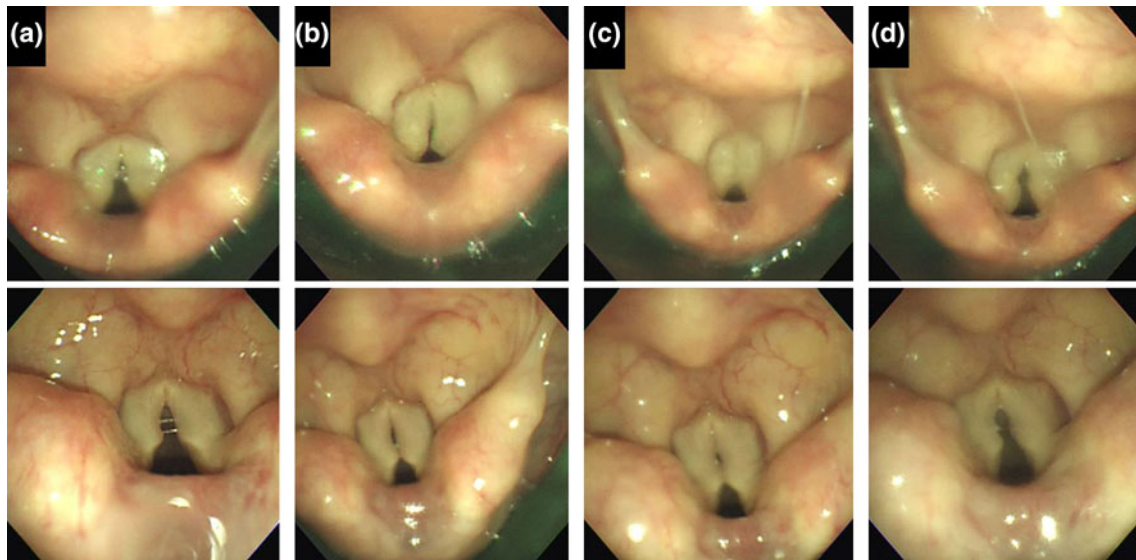


Fig. 1 The development of anatomical alterations of the glottis in each patient. **a** After insertion of the i-gel. **b** Twenty minutes after the administration of desflurane. **c** Thirty-five minutes after the

administration of desflurane. **d** After the discontinuation of desflurane and administration of propofol

Although it is unclear whether discontinuation of desflurane and/or administration of propofol opened the vocal cords in our cases, our findings indicate that care should be taken to check for vocal cord closure in desflurane anesthesia when the airway is secured by a supraglottic airway device without the use of neuromuscular blockade.

Conflict of interest The authors reported no conflicts of interest.

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