

Investigating the Failure to Aspirate Subglottic Secretions with the Evac Endotracheal Tube

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BACKGROUND: Aspiration of subglottic secretions is a widely used intervention for prevention of ventilator-associated pneumonia. However, using the Hi-Lo[®] Evac endotracheal tube (Hi-Lo Evac; Mallinckrodt; Athlone, Ireland) (Evac ETT), dysfunction of the suction lumen and subsequent failure to aspirate the subglottic secretions are common. Our objective in this study was to determine the causes of suction lumen dysfunction experienced with the Evac ETT.

METHODS: We studied 40 adult patients intubated with the Evac ETT. In all cases for which dysfunction of the suction lumen was observed, the subglottic suction port was examined visually using a flexible bronchoscope.

RESULTS: Dysfunction of the suction lumen occurred in 19 of 40 patients (48%). In 17 of these (43%), it was attributed to blockage of the subglottic suction port by suctioned tracheal mucosa.

CONCLUSION: Evacuation of subglottic secretions using the Evac ETT is often ineffective due to prolapse of tracheal mucosa into the subglottic suction port.

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Aspiration of the secretions pooled immediately above the endotracheal tube cuff (subglottic space) is considered an important mechanism in the pathogenesis of ventilator-associated pneumonia (VAP). The Hi-Lo[®] Evac endotracheal tube (Hi-Lo Evac; Mallinckrodt; Athlone, Ireland) (Evac ETT), incorporating a separate dorsal suction lumen, is designed for evacuation of subglottic secretions. The Evac ETT suction lumen has two ports: a subglottic port located 15 mm above the cuff, with an elliptical shape (major axis 6 mm, minor axis 3 mm), and an external port for connection to suction. Clinical trials have shown that the evacuation of subglottic secretions using the Evac ETT (with continuous or intermittent suction) contributes to reduction in VAP rates (1–5), whereas several reviews and guidelines favor its use as a VAP preventive measure (6–9). However, Rello et

al. reported failure to aspirate subglottic secretions using the Evac ETT with an incidence of 34% (28 of 83 patients), and considered the above mechanism as a risk factor for VAP development (5). Nevertheless, causes of Evac ETT aspiration failure have not been clearly identified. Therefore, we conducted a prospective observational study to investigate the incidence and possible causes of Evac ETT aspiration malfunction in 40 critically ill patients.

METHODS

Forty adult patients were enrolled in our study after approval by our Institutional Scientific Board. After orotracheal intubation with the Evac ETT (internal diameter 7–7.5 mm for women and 8–8.5 mm for men), confirmation of proper tube position with capnography, palpation of the cuff at the sternal notch, and lung auscultation, the tube was secured at right angle of the orifice of the mouth. The depth of insertion of the Evac ETT was 23 cm in men and 21 cm in women. The cuff was inflated to a pressure between 20 and 25 mm Hg.

After initial stabilization of the patients, we evaluated the Evac ETT suction lumen for dysfunction as follows: Sedated and paralyzed patients were placed in a 30 degrees semirecumbent position, with the head in a neutral position, and given ventilatory support. The suction lumen was connected to wall suction through a continuous suction regulator set at –15 mm Hg negative pressure via a 20-mL sputum trap, and 20 mL of water for injection was administered in the oral cavity. The pressures of the wall suction regulator and the sputum trap

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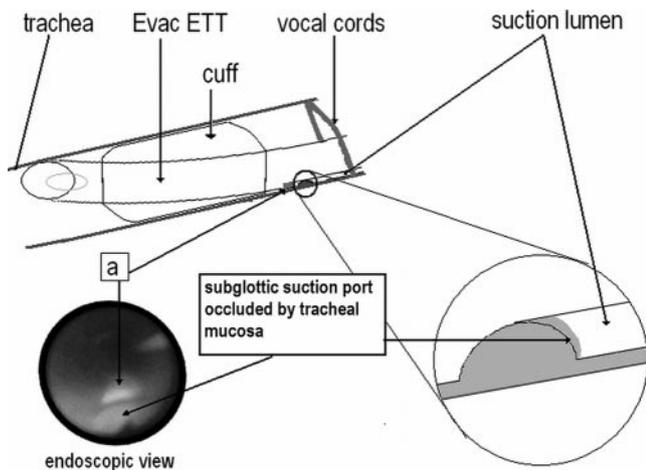


Figure 1. The cause of suction lumen dysfunction. Schematic illustration of the prolapse of tracheal mucosa into the subglottic suction port. An endoscopic photograph of the subglottic suction port seen through the tracheal lumen in a case with suction lumen dysfunction is presented. (a) The blue radiopaque marker of the Evac endotracheal tube (ETT).

were observed for 60 min. Suction lumen dysfunction was defined as negative pressure in the manometer of the suction regulator below -20 mm Hg for 15 min with no evidence of secretions being suctioned. In cases where dysfunction was noted, the negative pressure of the wall suction was recorded, and the subglottic port of the suction lumen was evaluated visually via the ETT by two experienced intensivists at the same time, using a fiberoptic bronchoscope (Olympus ENF/P3, Tokyo, Japan). The blue radiopaque marker, placed between the subglottic suction port and the cuff, was used to identify the subglottic suction port (Fig. 1). For patients in whom the suction lumen dysfunctioned, the negative pressure to the suction port was discontinued definitively.

Ninety-five percent confidence intervals (95% CI) were calculated for the incidence and causes of suction lumen dysfunction.

RESULTS

The 40 patients studied (26 men/14 women) had a median (interquartile range; IQR) Acute Physiology and Chronic Health Evaluation II score of 15 (12–17), a median (IQR) age of 62.5 (47.5–73.7) years, a median (IQR) height of 1.69 (1.63–1.74) m, and a median (IQR) weight of 77.5 (66.7–80.7) kg.

The incidence of suction lumen dysfunction was 48% (19 of 40 patients), with a 95% CI: 32%–63%. Endoscopic examination attributed the ETT dysfunction to an obstruction of the subglottic suction port by suctioned tracheal mucosa in 17 of 40 patients (43%) (95% CI: 27%–58%) (Fig. 1). In one case, the subglottic suction port was occluded by thick secretions, and in another, the cause of the dysfunction was not identified because of a poor endoscopic image. In cases with suction lumen

dysfunction the decrease of negative pressure started at a median time of 6 min (IQR: 4–16 min).

DISCUSSION

The observed incidence of Evac ETT suction lumen dysfunction in our study was high, 48% (95% CI: 32%–63%). Moreover, it appears that the dominant cause of suction lumen dysfunction was occlusion of the subglottic suction port by suctioned tracheal mucosa (Fig. 1). This finding raises significant questions concerning the safety of evacuation of subglottic secretions with subglottic suction using the Evac ETT. Negative pressure of less than -20 mm Hg favors prolapse of tracheal mucosa into the subglottic suction port. The herniation of tracheal mucosa within the suction port, caused by the negative pressure, impedes local tissue perfusion, producing local tracheal mucosa ischemia.

Our findings explain those of Bera et al., who found in an animal study that aspiration of subglottic secretions can cause severe tracheal injury in an area immediately adjacent to the subglottic suction port (10). Moreover, Girou et al. (11) considered the very high incidence (40%) of laryngeal edema in patients receiving subglottic suctioning as an adverse effect of continuous subglottic suctioning. The lower rate of aspiration failure reported by Rello et al., 34% (28 of 83 patients) (95% CI: 24%–45%), could be attributed to the lower sensitivity of their method for detection of subglottic suction port obstruction (5).

Orotracheal tubes are curved to facilitate intubation. This shape increases the possibility that the posterior surface of the tube, where Evac's distal suction port lies, is close to or touches the trachea. A modification of the Evac ETT to keep the suction port away from the tracheal surface would probably eliminate suction lumen dysfunction.

In conclusion, evacuation of subglottic secretions with continuous low negative pressure subglottic aspiration using the Evac ETT is often ineffective due to the prolapse of tracheal mucosa into the subglottic suction port, and probably exposes the patient to a high risk of tracheal injury. Consequently, we suggest that aspiration of subglottic secretions with the Evac ETT for prevention of VAP be performed in conjunction with closed monitoring for dysfunction of the suction lumen and with discontinuance of suction when dysfunction occurs.

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