Online Case Report

A Technique of Awake Bronchoscopic Endotracheal Intubation for Respiratory Failure in Patients With Right Heart Failure and Pulmonary Hypertension

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Objective: Patients with pulmonary hypertension and right heart failure have a high risk of clinical deterioration and death during or soon after endotracheal intubation. The effects of sedation, hypoxia, hypoventilation, and changes in intrathoracic pressure can lead to severe hemodynamic instability. In search for safer approach to endotracheal intubation in this cohort of patients, we evaluate the safety and feasibility of an alternative intubation technique.

Data Sources: Retrospective data analysis.

Study Selection: Two medical ICUs in large university hospitals in the United States.

Data Extraction: We report a case series of nine nonconsecutive patients with compromised right heart function, pulmonary hypertension, and severe acute hypoxemic respiratory failure who underwent endotracheal intubation with a novel technique combining awake bronchoscopic intubation supported with nasally delivered noninvasive positive pressure ventilation or high-flow nasal cannula.

Data Synthesis: All patients were intubated in the first attempt without major complications and eight patients (88%) were alive 24 hours after intubation. Systemic hypotension was the most frequent complication following the procedure.

Conclusions: Awake bronchoscopic intubation supported with a noninvasive positive pressure delivery systems may be feasible alternative to standard direct laryngoscopy approach. Further studies are needed to better assess its safety and applicability. (*Crit Care Med* 2017; 45:e980–e984)

Key Words: awake bronchoscopic intubation; difficult airway; high-flow nasal cannula; noninvasive ventilation; right heart failure

Pulmonary hypertension (PH) and right heart (RH) failure may confer an increased risk of hemodynamic collapse during and immediately after endotracheal intubation (ETI) (1–6). Traditionally, clinicians use deep sedation and supine positioning to facilitate direct laryngoscopy for intubation. However, this technique may cause atelectasis, hypoxemia, and hypercapnea, all of which may increase pulmonary vascular resistance (PVR). The induction of deep anesthesia causes systemic vasodilation, which can decrease the pressure gradient for coronary perfusion. Increased myocardial work and decreased perfusion can cause right ventricular (RV) ischemia and unleash a vicious self-perpetuating cycle of deterioration (6–9). These hemodynamic insults may result in hemodynamic collapse during intubation of patients with RH failure (3, 6). Accordingly, experts recommend avoiding ETI in this population when possible. If intubation is unavoidable, experts recommend using a sedation strategy that limits systemic hypotension (10, 11). However, there is little data examining different methods of emergency airway management in this uniquely fragile population.

Previously, we described the use of an intubation technique for high-risk patients with severe acute hypoxemic respiratory failure (AHRF). The procedure uses bronchoscopic intubation in spontaneously breathing awake patients while being supported by high-flow nasal cannula (HFNC) oxygen or noninvasive ventilation (NIV) (12–14). This intubation technique avoids deep sedation and therefore may help maintain ventilation and systemic tone (**Table 1**). Furthermore, the technique uses spontaneous breathing, positive end-expiratory pressure (PEEP), and upright positioning. Theoretically, these features might prevent an increase in PVR associated with atelectasis (15–17). NIV or HFNC systems may provide low levels of PEEP, which could

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TABLE 1. Theoretical Physiologic Benefits of Awake Bronchoscopic Intubation During Noninvasive Support of Spontaneous Ventilation

Topical Anesthetics, Anxiolysis, and Reduced Need for Systemic Anesthesia	Upright Positioning	Spontaneous Breathing	Noninvasive Positive End-Expiratory Pressure Support
Maintain systemic vascular tone	Avoid supine position–induced increase in right atrial pressure	Prevent hyperinflation	Gradual reduction in venous return and avoidance of sudden onset of positive pressure impact caused by mechanical ventilation by early noninvasive start on positive pressure ventilation
Enable awake upper airway manipulation	Maintain alveolar recruitment and prevent atelectasis	Avoid hypoventilation	Pneumatic splint to the upper airways
Avoid hypoventilation, maintain oxygenation and ventilation	Optimize airway patency	Maintain oxygenation	Maintain alveolar recruitment
Maintain alveolar recruitment and prevent atelectasis	Prevent aspiration	Maintain delivery of inhaled pulmonary vasodilator therapy	
	Reduce hypoxemic effect of shift in pulmonary perfusion distribution		

reduce the impact of sudden transition from negative to positive pressure ventilation caused by initiation of invasive mechanical ventilation. In addition, this technique allows patients to continue to receive inhaled pulmonary vasodilators without interruption during intubation. We hypothesized that these theoretical benefits of the awake bronchoscopic intubation technique might be particularly useful in patients with PH and RHF failure who require intubation for respiratory failure. In this article, we describe our early experience with awake bronchoscopic intubation during continuous support either via HFNC or NIV, specifically in patients with RH dysfunction and AHRF.

MATERIALS AND METHODS

Study Population

We identified nonconsecutive patients with PH and RH dysfunction who required emergency ETI for severe AHRF. Patients had PH due to acute pulmonary embolism or World Health Organization Groups 1, 3, and 4 PH. Patients were critically ill and were treated at either of the two tertiary ICUs.

Inclusion criteria required estimated RV systolic pressure greater than 40 mm Hg by echocardiography or a mean pulmonary artery pressure greater than or equal to 25 mm Hg in patients who had undergone RH catheterization. Patients had echocardiographic evidence of RV enlargement and dysfunction. AHRF was defined a new requirement for NIV or HFNC. Patients were excluded if they had evidence of reduced left ventricular systolic function (ejection fraction < 50%) by echocardiogram or a pulmonary artery occlusion pressure of greater than 15 mm Hg on RH catheterization.

All treatment decisions including the timing and technique of ETI or decision to use HFNC or NIV were made by the treating physicians. The clinicians performing the procedure were attending and fellow physicians trained in pulmonary and critical care medicine with extensive experience in flexible bronchoscopy. Institutional review board approval was obtained for retrospective data analysis.

Peri-Intubation Oxygenation With HFNC or Nasal NIV

HFNC was delivered via Optiflow (Fisher & Paykel Healthcare, Irvine, CA). NIV was delivered with Respironics BiPAP Vision or V60 ventilators (Respironics, Murrysville, PA). The Respironics Contour Deluxe (Respironics) nasal mask served as the interface for the nasal NIV system. Patients had been treated with nasal NIV or HFNC for at least 15 minutes prior to and then throughout the intubation procedure.

Orotracheal Intubation

Patients underwent bronchoscopic intubation in semirecumbent position, awake, and breathing spontaneously with support of HFNC or NIV (**Fig. 1**). Oropharyngeal topical anesthesia was achieved atomizing 5 mL of 2–4% lidocaine. Additionally, 5% lidocaine ointment was applied onto the surface of a 9- or 10-cm intubating "Williams" airway, which was slowly advanced into the oropharynx. After achieving topical anesthesia, the treating physician prescribed systemic sedation, targeted to anxiolysis and analgesia but also to maintain consciousness and avoid hypotension and respiratory depression.

A flexible video bronchoscope was advanced through a lubricated size 7–8 mm endotracheal tube (ETT) previously loaded into the intubation channel of Williams airway. With the ETT resting on the distal end of the bronchoscope, the bronchoscope was inserted into the oropharynx via the Williams airway, and 1% atomized lidocaine was sprayed on to the supraglottic area and the vocal cords. The bronchoscope was then inserted through the vocal cords and into the trachea.

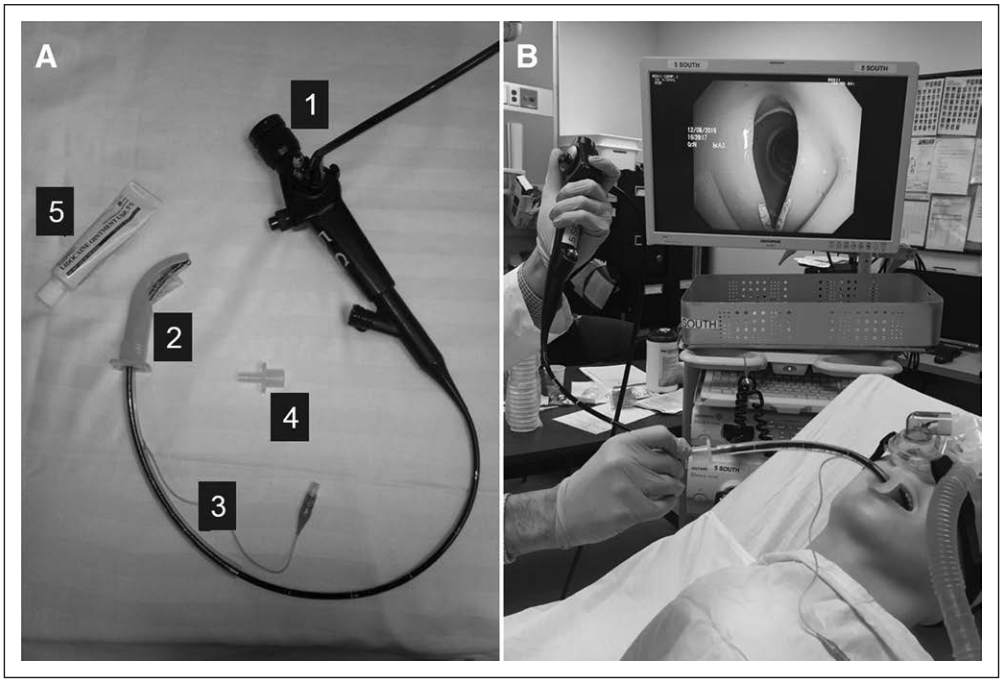

Figure 1. A, Equipment required for awake bronchoscopic intubation: 1) Video bronchoscope, 2) Williams intubating airway, 3) endotracheal tube (ETT), 4) ETT connector transiently removed to facilitate removal of Williams airway after intubation, 5) and 5% lidocaine ointment to be applied to the Williams airway to facilitate oropharyngeal topical anesthesia and insertion of the Williams airway. **B**, Demonstration of bronchoscopic intubation while the patient is being supported with, in this case, nasal noninvasive ventilation, sitting in a semi-recumbent position. With the ETT loaded onto the bronchoscope, the bronchoscope is advanced through the Williams airway. The vocal cords should be well-visualized and surrounding soft tissue is not manipulated. After systemic and local anesthesia is achieved, the bronchoscope is advanced through the vocal cords into the trachea and the ETT is subsequently advanced into the trachea. Placement of the ETT is confirmed with the bronchoscope.

Next, the ETT was advanced over the bronchoscope into the trachea. The bronchoscope was used to confirm appropriate placement of the ETT, the invasive ventilator circuit was connected, and HFNC or nasal NIV was removed.

Statistical Analysis

The values were presented as median or mean, and variability was expressed by interquartile range and SD. Chi-square analysis was performed to evaluate categorical baseline characteristics and complications arising following ETT placement. Paired *t* test and Wilcoxon signed rank tests were performed to compare continuous variables before and after intubation. A value of *p* less than 0.05 was considered statistically significant. The analyses are descriptive and no a priori hypothesis was tested in the case series.

RESULTS

Patient Characteristics

Patient characteristics are shown in **Supplemental Table 1** (Supplemental Digital Content 1, http://links.lww.com/CCM/C738). All patients had echocardiographic evidence of PH. The median RV systolic pressure was 65 mm Hg (55–165 mm Hg). All patients had echocardiographic evidence of RV enlargement and dysfunction. Four patients were on chronic pulmonary vasodilator therapy. Six patients, including one started on IV treprostinil, were treated with pulmonary vasodilators for decompensated RH failure before the decision to intubate was made. Seven patients had elevated brain natriuretic peptide (> 150 pg/mL) (value not available for two patients). Four patients had elevated troponin (> 0.04 ng/mL) (value not available for three patients). Two patients required hemodialysis prior to intubation. The median Sequential Organ Failure Assessment score was 12 (range, 7–14).

Prior to intubation, all patients had severe AHRF with an average Pao_2 of 70 ± 27 mm Hg on maximal (Fio_2, 1.0) noninvasive supplemental oxygen. Five patients were supported with HFNC, three on NIV, and one on nonrebreather mask that was changed to NIV prior to intubation. Oxygen saturation (Spo_2) prior to intubation ranged from 72% to 99%, with an average Spo2 of 92%. Five patients required vasopressor support prior to intubation.

The median Glasgow Coma Scale was 15 (range, 8–15). All patients were able to tolerate awake bronchoscopic intubation. The most commonly used sedation and analgesia agents for intubation were midazolam and fentanyl in low doses, whereas ketamine was used in two patients. No paralytics were used.

Outcomes and Complications

Postintubation outcomes are described in **Table 2**. All nine patients were intubated in the first attempt. There was no evidence of aspiration during intubation. Systemic hypotension was the most frequent complication following the procedure.

Despite the fact that only one patient (11%) had significant desaturation greater than 10% during the ETI, five patients (56%) experienced oxygen desaturation of greater than 10% within the first hour after intubation, in comparison with preintubation values, with average maximal Spo_2 decrease of 11% ± 8% ($p = 0.002$). Postintubation, all patients were on Fio_2 1.0, with a median PEEP of 5 cm H_2O. With these settings, the mean Pao_2/Fio_2 was 120.4 ± 65, with an average improvement in Pio_2/Fio_2 ratio of 36.1 ± 28.6 ($p = 0.015$). Maximal mean arterial pressure decrease in the first hour after intubation averaged of 11.2 ± 13.4 mm Hg. Vasopressors were initiated or escalated in eight patients (88%). Significant increase in vasopressor

TABLE 2. Induction Agents Used During Intubation and Postintubation Clinical Condition of Patients

Patient	Induction/ Sedation	> 10% Desaturation During ETI	Lowest Spo_2 Within 1 hr Post-ETI, Average Change (% [Δ%Spo_2])	Postintubation Positive End-Expiratory Pressure (cm H_2O)	Postintubation Pao_2 (mm Hg)	pH/ $Paco_2$ (mm Hg)	Lowest MAP Within 1 hr Post-ETI, Average Change (mm Hg [Δmm Hg MAP])	Postintubation Vasopressors, Increased or Newly Initiated	New Renal Failure by Acute Kidney Injury Network Classification	Alive at 24 hr
1	Ketamine 40 mg	No	80 (−17)	5	80	7.23/44	58 (11)	No	No	Yes
2	Midazolam 4 mg, fentanyl 100 μg	Yes	79 (−18)	8	266	7.29/74	79 (36)	Yes	No	Yes
3	Ketamine 50 mg, midazolam 1 mg, fentanyl 75 μg	No	66 (−6)	5	41	7.44/24	56 (1)	Yes	No	Yes
4	Etomidate 10 mg	No	72 (−24)	7	78	7.44/31	62 (13)	Yes	No	Yes
5	Fentanyl 50 μg	No	89 (−10)	5	135	7.22/63	67 (−9)	No	Injury	No
6	Fentanyl 50 μg	No	94 (−3)	5	154	7.27/80	71 (25)	Yes		Yes
7	Fentanyl 50 μg, midazolam 1 mg	No	73 (−14)	5	97	7.41/40	75 (12)	No	No	Yes
8	Fentanyl 100 μg, midazolam 4 mg	No	75 (−7)	14	100	7.57/38	60 (12)	No	No	Yes
9	Fentanyl 50 μg, midazolam 8 mg	No	100 (+1)	5	133	7.09/48	60 (0)	Yes	Failure	Yes
Mean			81 (−11)	6.6	120	7.33/49	65 (11.2)			

ETI = endotracheal intubation, MAP = mean arterial pressure.

ΔSpo_2–the difference between preintubation Spo_2 and the lowest documented Spo_2 after intubation.

support (defined as increase in norepinephrine dose > 0.1 μg/kg/min or equivalent) was seen in five patients (55.6%).

No new patients required hemodyalysis within 24 hours of intubation, one patient had an acute kidney injury 24 hours after intubation, and one patient had acute kidney failure as defined by Risk, Injury, Failure, Loss of kidney function and End-stage kidney disease classification (18). All nine patients were alive 1 hour after the intubation, and eight patients (88%) were alive 24 hours postintubation.

One episode of significant desaturation occurred in a patient who was pretreated with an inhaled pulmonary vasodilator prior to ETI. However, although there was no correlation between desaturation and duration of PH therapy, patients who were on a background of stable PH therapy prior to intubation were less likely to need increased vasopressor support following intubation ($p = 0.016$). There was no relationship between HFNC support during intubation versus NIV support in terms of the need for increased vasopressor therapy ($p = 0.294$). The severity of RV dysfunction (moderate/severe vs mild), measured tricuspid annular plane systolic excursion, or estimated right ventricular systolic pressure did not correlate with more significant hypotension post-ETI by logistic regression ($p > 0.05$ for all analyses). There was a trend toward increased risk of acute renal failure in patients with worse RV dysfunction (moderate or severe vs mild; $p = 0.151$).

DISCUSSION

The study demonstrates the successful use of awake bronchoscopic intubation during spontaneous ventilation supported by NIV or HFNC in patients with RH dysfunction and AHRF. All nine patients were intubated on the first attempt and none had a major complication from intubation. We cannot evaluate if this procedure is more effective than other approaches since there are no controls in this case series. Although the actual data on complications of transition to mechanical ventilation in severely hypoxemic patients in RV failure are limited, it has been shown that approximately one-third of critically ill patients are not intubated on the first attempt (19). ETI carries a risk for immediate mortality up to 15% in settings of severe hypotension without RV failure (20), and severe hypoxemia itself increases relative risk for cardiac arrest four times (21, 22). Furthermore, the failure of NIV management and the need to intubate hypoxemic patients is associated with mortality and complication rates (23–25).

Importantly, hypotension and hypoxemia often worsened within 1 hour after intubation. The timing suggests that despite uncomplicated intubation, patients with RH failure and AHRF often deteriorate while on mechanical ventilation, highlighting their fragility. We speculate that deep sedation administered after intubation may contribute to derecruitment and hypoxia. Attempts to perform lung recruitment are difficult in patients

with PH and RV failure because positive pressure ventilation may raise PVR through hyperinflation (8).

We combined NIV together with HFNC because they have much in common. Both allow high flow rates and higher F_{IO_2} and provide PEEP. Both maintain end-expiratory lung volumes and generate better oxygenation and ventilation and decrease work of breathing (26). These modalities provide more predictable oxygenation and ventilation support compared with traditional intubation strategies.

There are substantial limitations of our study. First, it is a small retrospective case series, and the patients were not prospectively randomized. The cases represent a spectrum of severity of RH dysfunction; some had frank decompensated RH failure and circulatory shock, whereas others had only morphometric evidence of RV dysfunction and enlargement on echocardiogram. RH catheterization was not performed on all the patients, and the actual extent of PH may be under- or overestimated. The patients in this study were highly selected. This intubation method may not be feasible for all patients. In particular, patients who cannot tolerate bronchoscopy, have inadequate spontaneous respiratory effort, or who are uncooperative are likely poor candidates for this procedure. Blood, secretions, or emesis may obscure the bronchoscopic view. Importantly, operators who perform this procedure need to be skilled in flexible bronchoscopy and topical anesthesia of the airway. Although we did not record time to successful intubation, we presume that this approach may be slower than laryngoscopic intubation because of the time required to set up equipment and provide topical anesthesia.

CONCLUSION

Severely hypoxemic patients with RH dysfunction and PH are at high risk of deterioration during intubation. Our report presents a novel approach using awake bronchoscopic intubation with HFNC or NIV support.

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