RESPIRATORY SUPPORT INCLUDING EMERGENT EXTRACORPOREAL MEMBRANE OXYGENATION AS A BRIDGE TO AIRWAY DILATATION FOLLOWING PERIOPERATIVE BRONCHIAL OCCLUSION

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Abstract

During the perioperative period, various factors may lead to intraoperative and postoperative respiratory failure including upper airway obstruction, bronchospasm, acid aspiration, laryngospasm, and pulmonary hypertension. Regardless of the etiology, prompt recognition with treatment of the inciting event is required to ensure a successful recovery. We report the intraoperative development of respiratory insufficiency and failure in a 17-year-old girl who was status post lung transplant undergoing bronchoscopy. During bronchoscopy, complete left main stem obstruction occurred due to a fibrinous mass near the bronchial anastomosis site. Various modalities were used to support the patient intraoperatively and then postoperatively including low tidal volume/high PEEP ventilation, inhaled nitric oxide (iNO), and high frequency oscillatory ventilation (HFOV). In the CTICU, emergent bedside venovenous extracorporeal membrane oxygenation (ECMO) was used as a bridge to the recovery of respiratory function which was achieved with removal of the occluding fibrinous airway tissue followed by balloon dilatation and stenting of the left main stem bronchus. The potential perioperative causes of respiratory failure are reviewed and support techniques including conventional ventilator strategies, iNO, HFOV and ECMO discussed.

Introduction

Despite continued advancements and improvements in monitoring, pharmacologic agents, and the skill of anesthesia providers, anesthesia entails the potential risk of morbidity and even mortality[1,2]. In the majority of cases, perioperative cardiovascular arrest and death are the result of either cardiac or respiratory failure. The term respiratory failure is generally used to describe inadequate pulmonary gas exchange resulting in hypoxemia and hypercarbia. During the perioperative period, various factors may be responsible for intraoperative and postoperative respiratory failure including upper airway obstruction, laryngospasm, bronchospasm, acid aspiration and pulmonary arterial hypertension. Regardless of the etiology, prompt recognition and

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treatment of the inciting event are required to ensure a successful recovery. However, despite aggressive therapy, progressive respiratory dysfunction may require increased support to provide adequate oxygenation and ventilation. Various strategies may be employed to provide support while the inciting event is treated and recovery ensues. We report the use of emergent bedside venovenous extracorporeal membrane oxygenation (ECMO) in a patient who developed perioperative respiratory failure due to acute occlusion of the left main bronchus following flexible fiberoptic bronchoscopy and biopsy. Prior to the initiation of ECMO, other modalities of support to treat the patient's life-threatening hypoxemia in both the operating room and the cardiothoracic intensive care unit (CTICU) included low tidal volume/high positive end expiratory pressure (PEEP) ventilation, inhaled nitric oxide (iNO) and high frequency oscillatory ventilation (HFOV). ECMO effectively reversed the hypoxemia and provided the bridge to recovery of respiratory function, which was achieved with removal of the occluding fibrinous airway tissue followed by balloon dilatation and stenting of the left bronchus. The potential perioperative causes of respiratory failure are reviewed and support techniques, including conventional ventilator strategies, iNO, HFOV and ECMO are discussed.

Case Report

Institutional Review Board approval for single case reports is not required at Nationwide Children's Hospital (Columbus, Ohio). A 17-year-old adolescent with a history of cystic fibrosis (CF), who was status post lung transplantation 6 months prior to the current admission presented to the operating room for direct laryngoscopy with bronchoscopy and sinus debridement. The patient had been hospitalized for the past 2 weeks and had tolerated flexible fiberoptic bronchoscopy with biopsy without complications, which was performed due to concerns of allograft rejection. The procedure was performed with intravenous sedation using propofol. During the course of her admission, she was also treated for cytomegalovirus (CMV) viremia and pneumonitis. Due to progressive respiratory distress as evidenced by the development of biphasic stridor (inspiratory greater than the expiratory component) and concerns for worsening graft function due to the CMV pneumonitis, the decision was made for a repeat bronchoscopy. The planned procedure was biopsy and an airway evaluation by the otorhinolaryngology service to investigate the etiology for the new onset stridor. Upon arrival to the operating room, the patient was in no acute respiratory distress with no active audible stridor. Preoperative vital signs included blood pressure 117/78 mmHg, heart rate 109 beats per minute, respiratory rate 24 breaths per minute and oxygen saturation of 100% on room air. The patient was held *nil per os* for 6 hours. Routine American Society of Anesthesiologists' monitors were placed. Anesthesia was induced with propofol (50 mg) and the incremental inhalation of increasing concentrations of sevoflurane in 100% oxygen. Spontaneous ventilation was maintained during anesthetic induction. Once an adequate depth of anesthesia was achieved, direct laryngoscopy was performed and the vocal cords were sprayed with 5 mL of 2% lidocaine. Direct laryngoscopy and bronchoscopy revealed no signs of tracheal narrowing and a normal airway. A 6.0 mm endotracheal tube was placed by otorhinolaryngology and the pulmonology service proceeded with the flexible fiberoptic bronchoscopy and biopsy. During bronchoscopy, a fibrinous mass in the left main stem bronchus was noted (Fig. 1). Approximately one hour after the start of the bronchoscopy and biopsy, the patient acutely became increasingly more difficult to ventilate with the oxygen saturation decreasing from 100% to 92-94%. Using a metered dose inhaler, albuterol was administered through the endotracheal tube for presumptive bronchospasm. Although there was no change in the depth of placement of the endotracheal tube positioning, auscultation revealed decreased breath sounds on the left side. Repeat bronchoscopy was performed to investigate the changes. Although the endotracheal tube was correctly positioned in the mid-trachea, there was edema of the left bronchial mucosa and complete occlusion of the left bronchus along the suture lines of the graft. An additional 8 mg of dexamethasone was administered (total for the case of 12 mg) and neuromuscular blockade achieved with the administration of rocuronium (20 mg) in an attempt to facilitate ventilation. Despite hand-ventilation with

100%, the oxygen saturation continued to decrease to 72-74%. A radial arterial cannula was placed. Ventilatory settings included F_iO_2 1.0, respiratory rate 24 breaths per minute, peak inflating pressure 42 cmH_2O which was necessary to generate a tidal volume 5-6 mL/kg, and PEEP 16 cmH_2O. Intraoperative chest radiograph showed complete opacification of the left lung, proper positioning of the ETT and no evidence of pneumothorax (Fig. 2). Arterial blood gas analysis revealed pH 7.211, P_aCO_2 54 mmHg, and P_aO_2 55 mmHg. Nitric oxide was started at 20 ppm, and the ETT was advanced into the right main stem bronchus. The remainder of the procedure was aborted and the patient was transported to the pediatric cardiothoracic intensive care unit (CTICU). During transportation, the oxygen saturation varied from 85-89% utilizing a F_iO_2 of 1.0. Shortly after arrival to the CTICU, there was an abrupt decrease of the oxygen saturation to 40-50%. Repeat laryngoscopy was performed and the endotracheal tube was changed to a 7.0 and placed into the right bronchus using fiberoptic guidance. Inhaled NO was increased to 40 ppm; however, the oxygen saturation remained at 50-60%. Given the ongoing hypoxemia, HFOV was initiated with settings of mean airway pressure (MAP 24) cmH_2O, amplitude 50, and Hz 3 without therapeutic effect. Thirty minutes later, a 20 French Avalon Elite™ catheter (Avalon Laboratories LLC, Rancho Dominguez, CA) was placed by the cardiothoracic surgery service into the patient's right internal jugular vein for venovenous ECMO (VV-ECMO). Following the initiation of VV-ECMO, flexible fiberoptic bronchoscopy was performed by the pulmonology service and demonstrated edema and near 100% occlusion of the left main stem bronchus with tissue and airway debris. Vigorous lavage was performed and small lumens were created into both subsegments of the left bronchus using pediatric size biopsy forceps followed by the instillation of 10 mg of dexamethasone. The next morning, the interventional pulmonology service performed bedside flexible fiberoptic bronchoscopy with balloon dilatation of the left bronchus and debridement of the fibrinous tissue while the patient was still on VV-ECMO. Following this procedure, there was gradual expansion of the left lung over the next 8-12 hours (Fig. 3). The patient was decannulated from ECMO and transitioned from HFOV to conventional ventilation on the 2nd postoperative day and her trachea was extubated on the 3rd postoperative day. She was supported with non-invasive ventilation for 48 hours and subsequently weaned to room air. There was no change in her baseline neurological status. The remainder of her postoperative course was unremarkable and she has returned to her baseline status.

Fig. 1
Intraoperative photograph taken during bronchoscopy showing edema of the left bronchial mucosa and total occlusion of the left bronchus just above the suture site for the graft

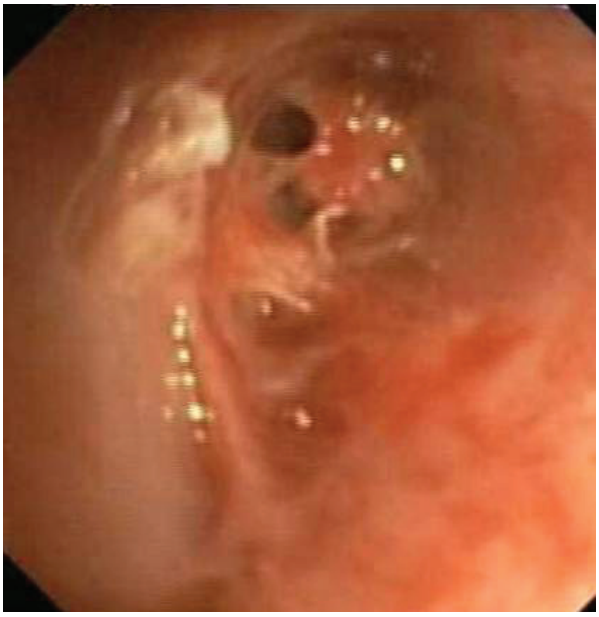


Fig. 2
Intraoperative chest radiograph showing complete opacification of the left lung, proper positioning of the endotracheal tube, and no evidence of pneumothorax

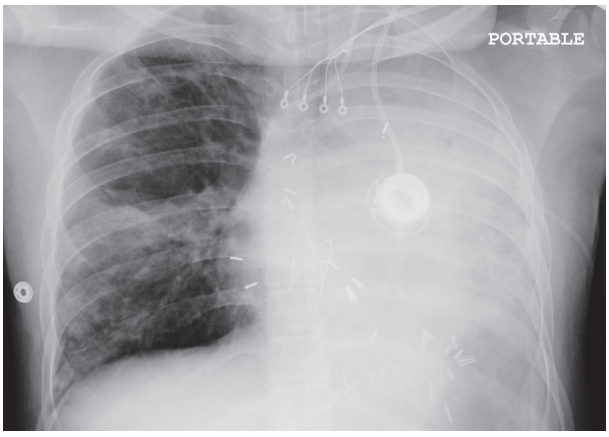
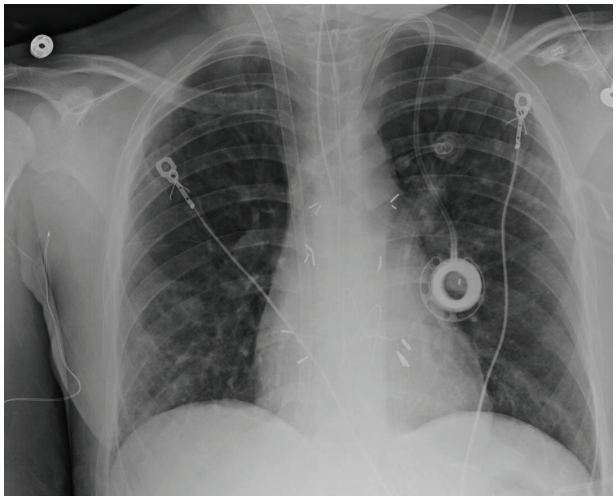


Fig. 3
Repeat chest radiograph following interventional bronchoscopy and balloon dilatation of the left bronchus. The single lumen catheter for venous-venous extracorporeal oxygenation can be seen in the right internal jugular vein



Discussion

The care of the patient with postoperative respiratory failure begins with the identification of its etiology. In general, there are 5 primary causes of hypoxemia including 1) a low inspired concentration of oxygen, 2) hypoventilation, 3) ventilation-perfusion inequalities, 4) shunt, and 5) diffusion abnormalities. Although a low inspired oxygen concentration is frequently not considered in the differential diagnosis of hypoxemia, it must be considered in the operating room setting and its possibility ruled out by ensuring that the inspiratory oxygen concentration is 100%. In our patient, prompt evaluation with auscultation of the chest during manual ventilation with 100% revealed an inequality of breath sounds that prompted investigation via bronchoscopy. Although movement of the ETT is the most likely scenario resulting in inequality of breath sounds, we noted that the placement of the ETT (depth of insertion) had not changed. Rather, the acute onset of edema and plugging with debris of the left main stem bronchus resulted in total obstruction and significant ventilation-perfusion mismatch resulting in hypoxemic respiratory failure.

In the setting of hypoxemia, an evaluation of the severity of the lung disease may be based on the difference between the PaO_2 and the partial pressure of oxygen in the alveoli. This is commonly referred to the A-a (alveolar-arterial) oxygen gradient. In our patient, the severity of the respiratory failure was demonstrated by an A-a gradient of approximately 600 with a PaO_2 of 55 mmHg and an F_iO_2 of 1.0. Another measure of severity of lung injury that is used is if PaO_2:FiO_2 ratio. When the ratio is less than 300 mmHg, acute lung injury (ALI) is considered to be present while adult respiratory distress syndrome (ARDS) is present when the ratio is less than 200 mmHg. In our patient, the PaO_2:FiO_2 ratio was 55.

During conventional positive pressure ventilation, oxygenation is regulated by the F_iO_2 and the mean airway pressure (MAP). MAP is determined by the PIP, PEEP and the inspiratory time[3]. Increasing the MAP by manipulation of any of these variables recruits alveoli, improves ventilation-perfusion matching and decreases intrapulmonary shunting. In addition to reducing ventilation-perfusion inequalities and increasing functional residual capacity (FRC), increasing MAP may also result in a significant improvement in respiratory compliance thereby allowing for more effective spontaneous ventilation and decreasing the PIP required to provide adequate tidal ventilation. This may limit the potential for barotrauma during mechanical ventilation[4,5]. Intraoperatively, a relatively simple maneuver to increase MAP is to lengthen the inspiratory time, a technique that is frequently accomplished by setting an inspiratory to expiratory ratio of 1:1. A longer inspiratory time such that the ratio exceeds 1:1 is not recommended as expiratory time is compromised which may result in insufficient time to exhale with the development of auto-PEEP and an increased risk of barotrauma.

Mechanical ventilation also provides the minute ventilation for CO_2 removal, calculated as the respiratory rate (RR) times the tidal volume. $PaCO_2$ is directly related to the body's production of CO_2 during the catabolism of fats and carbohydrates and is inversely related to alveolar ventilation. In most clinical circumstances, the control of $PaCO_2$ is via alterations in the minute ventilation; however, some control of the body's endogenous CO_2 production is possible through the use of fats versus carbohydrates for nutrition or by control of body temperature. Prevention of hyperthermia and even induction of mild

hypothermia (35°C) can be used clinically to control hypercarbia as well as hypoxemia and limit mechanical ventilatory requirements.

In patients with severe lung disease, ventilation to normocarbia is not necessary and may in fact be harmful. Current practice in the ICU during the care of patients with ALI and ARDS includes permissive hypercarbia or allowing the $PaCO_2$ to increase, provided that the pH is above 7.25. This strategy has been shown to improve outcome in patients with adult respiratory distress syndrome (ARDS)[6]. When dealing with a critically ill patient in the operating room, although new models of anesthesia machines provide newer modalities of ventilatory support, older versions may be incapable of supporting the critically ill patient. In such cases, the early switch to a standard ICU ventilator is suggested. These ICU ventilators not only provide higher working pressures with the delivery of effective PIP and PEEP, but also allow for newer modalities of ventilation which may be helpful in patients with ALI such as pressure-regulated, volume controlled ventilation (PRVC).

Low tidal volume ventilation: The 2000 ARDS Network publication in the *New England Journal of Medicine* is viewed as a milestone in the practice of critical care medicine. In a prospective randomized trial of adults with ARDS, ventilation was provided with a tidal volume of either 6 mL/kg or 12 mL/kg[7]. Survival was greater in the low tidal volume group (31% versus 39.8%). In addition, ventilator-free days and number of days without non-pulmonary organ system failure were significantly increased in the low tidal volume treated group. Despite the compelling results set forth from the adult trials, lingering controversy remains whether the lung protective strategy of low tidal volume should be universally applied to all patients with ARDS/ALI and specifically the pediatric population. Detractors to the universal approach have instead advocated an approach more tailored to the individual patient, based on the underlying pulmonary process[8,9]. In the operating room, during the short term support of patients, such strategies cannot be universally advocated, especially when dangerously low oxygen saturations are present. In such scenarios, the short term consequences of the increased PIP relate primarily to its effects on preload and cardiovascular performance as well as the risk of barotrauma resulting in pneumothorax.

Extrapolation of low tidal volume ventilation to pediatric patients has been challenging given the scarcity of date to support its use in this younger patient population and the controversy that exists even among the adult patient population. Critics of a universal application of low tidal volume to pediatric patients have cited specific concerns. Neonates and infants may not be as susceptible to the barotrauma seen with high tidal volume ventilation as demonstrated by animal models[10,11]. Additionally, low tidal volumes may inadequately ventilate infants and neonates who at baseline have higher respiratory compliance versus their adult counterparts making them more prone to atelectasis.

Despite the arguments against low tidal volume ventilation in younger patients, some studies have suggested undeniable benefits in patients with ARDS/ALI. In a retrospective study examining pediatric patients ranging in age from 1 week to 17 years with ALI who were mechanically ventilated from 1988-1992 versus patients from 2000-2004, Albuali et al[12] reported that the group cared for during the 1988-1992 time frame were ventilated with significantly higher mean tidal volumes (10.2 ± 1.7 versus 8.1 ± 1.4 ml/kg), lower levels of PEEP (6.1 ± 2.7 versus 7.1 ± 2.4 cm H_2O) and higher PIP (31.5 ± 7.3 versus 27.8 ± 4.2 cmH_2O). Most importantly, those cared for during the 2000-2004 period had a lower mortality (21% versus 34%) and greater number of ventilator free days. In our patient, we chose low tidal volume/high PEEP ventilation as an appropriate ventilatory strategy because of her age, the history of a pre-existing CMV pneumonitis, and the requirements of a high PEEP to main adequate oxygenation. As such, the tidal volume was decreased to limit the PIP during use of a high PEEP technique. Although the decision to use low tidal volume ventilation may be more straightforward in older pediatric patients whose pulmonary mechanics are similar to their adult counterparts, the decision still remains unclear in younger patients. It has been suggested that until this clinical dilemma is resolved by more definitive randomized controlled trials examining the use of low tidal volume ventilation in younger pediatric patients, application of adult data to pediatric patients still remains a valid endeavor[13,14].

Nitric oxide: First described in 1987 as endothelial-derived growth factor[15], iNO was introduced into clinical practice during the early part of the 1990's[16]. Currently, the only FDA-approved indication for iNO is the treatment of hypoxic respiratory failure for term or near-term infants due to conditions such as primary pulmonary arterial hypertension, diaphragmatic hernia, aspiration, sepsis, or infectious pulmonary processes. The pulmonary vasodilatory effects of iNO are mediated through activation of guanylate cyclase with increased production of intracellular cyclic guanine monophosphate (cGMP) leading to decreased intracellular calcium concentration, smooth muscle relaxation and decreased pulmonary vascular resistance. Alterations in the pulmonary vascular tone result in the optimization of ventilation-perfusion matching with improvement of oxygenation via preferential dilatation of vessels in the better ventilated alveoli[17,18]. Rapid inactivation of iNO occurs through hemoglobin binding and formation of methemoglobin thereby limiting its systemic effects. Methemoglobin is metabolized to hemoglobin by methemoglobin reductase within the erythrocytes. The incidence of methemoglobinemia is low with inhaled NO concentration ≤40 ppm[19-21].

The use of iNO in both adult and pediatric patients for acute hypoxemic respiratory failure has been reported in multiple studies. In neonates ≥34 weeks gestation who had hypoxic respiratory failure, the Neonatal Research Network reported that iNO reduced the use of ECMO but had no effect on mortality[22]. Van Meurs et al reported similar findings in a cohort of premature infants with severe respiratory failure[23]. More recently, meta-analyses looking at the use of iNO in both pediatric and adult patients with ALI concluded that iNO could not be recommended in patients with acute hypoxemic respiratory failure. Despite the transient improvement in oxygenation that occurred for the first 24 hours, no reduction in mortality was noted[24,25].

Although the use of iNO as a definitive treatment for ALI is controversial, its use as a temporizing measure to improve oxygenation is promising in specific clinical scenarios. Dietrich and Tobias reported the successful intraoperative administration of iNO in a pediatric patient who developed hypoxemia following institution of one lung ventilation during thoracoscopic lung resection[26]. In our patient, iNO was administered to optimize oxygenation and facilitate an uneventful transport to the CTICU. Inhaled NO at a concentration of 20 ppm was administered to the manual resuscitation bag for use during transport. Following the patient's acute decompensation with profound hypoxemia after arrival to the CTICU, iNO proved ineffective even when increased to 40 ppm. When iNO is administered intraoperatively using the anesthesia machine ventilator, specific modifications in the intraoperative ventilator technique are needed to prevent rebreathing and disruption of the normal function of the delivery system[27].

High frequency oscillatory ventilation: High frequency oscillatory ventilation (HFOV) is a subtype of high frequency ventilation, which uses a piston to generate small tidal volumes with very high rates. It was been introduced into ICU care to provide ventilation and oxygenation in patients with respiratory failure while limiting the risk of barotrauma. Barotrauma is limited by avoiding high PIP's. High frequency ventilator techniques rely on respiratory rates greater than 120 breaths per minute (2 Hz) with tidal volumes less than dead space. Gas exchange is thought to occur via convection rather than bulk flow[28]. HFOV uses a piston-driven, diaphragm oscillator to provide a constant mean or distending airway pressure. Lung volumes are maintained above FRC thereby providing a constant distending pressure for alveoli while avoiding PIP's. Superimposed around the MAP are the oscillations provided by the inward and outward movement of the diaphragm. The movement of the diaphragm provides not only active inspiration, but also active expiration thereby decreasing gas trapping and inadvertent PEEP[29]. Unlike conventional mechanical ventilation, HFOV is unique in that oxygenation and ventilation are separated. Oxygenation is determined by the independent adjustments of MAP and FiO_2. Ventilation is manipulated by adjustments of the amplitude (delta P), frequency (Hz), and inspiratory time. In general, when caring for patients with ALI, the MAP is set at 2-4 cmH_2O above the MAP on conventional ventilation and adjusted up as needed to allow for a decrease of the FiO_2 to the desired level. The efficacy of the MAP and its effect on alveolar recruitment is assessed by a chest

radiograph with the goal of achieving lung expansion of 8-9 ribs. Ventilation is controlled primarily by adjusting the amplitude (delta P) using the power setting on the ventilator. The power knob is adjusted to control the amplitude that is changed in increments of 2-4 cmH_2O to provide for adequate chest movement and CO_2 removal. HFOV has been utilized in full range of ICU patients including adults, children, infants, and neonates. Despite its theoretical advantages over conventional mechanical ventilation, there are limited and conflicting evidence-based data to demonstrate its impact on morbidity and mortality[30-32].

In our patient, HFOV was selected over conventional mechanical ventilation after the hypoxemia failed to improve with ensuring correct placement of the ETT, iNO at 40 ppm, and manual bag ventilation. Given the severity of her lung disease and total left lung obstruction, HFOV did not result in a clinically significant improvement in oxygenation. Following the initiation of ECMO, HFOV was continued to provide a constant distending pressure and optimal recruitment of the left lung. This proved efficacious once the obstructing etiology was removed and treated. Following treatment, the patient tolerated the transition to conventional ventilation without difficulty.

Extracorporeal membrane oxygenation: ECMO was first described in 1972 as a salvage treatment for an adult patient who developed acute respiratory failure following blunt trauma[32]. Since the earliest days of its clinical use, ECMO's role in the treatment of patients with acute respiratory failure has fluctuated. The initial success of ECMO in the adult population was attenuated seven years later following a randomized controlled prospective study evaluating prolonged ECMO therapy for acute respiratory failure[34]. With a 90% mortality rate in both groups, ECMO was soon dismissed as an ineffective treatment for adult patients with acute respiratory failure. However, as the use ECMO waned in the adult population, its use in the neonatal population took over with reports of success in various causes of respiratory failure in the neonate including sepsis, meconium aspiration, congenital diaphragmatic hernia and congenital heart disease. Following success in the neonatal population and with improvements in the technology and techniques used for ECMO, it has become a popular means of rescue for patients of all ages with potentially reversible respiratory or cardiac failure that is unresponsive to conventional therapy. The publication of the CESAR (Conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure) trial in 2009 solidified ECMO's role in the treatment of adult patients with reversible respiratory failure[35]. In evaluating the 180 patients who were randomized to either ECMO or conventional management, 6-month survival without disability was greater in ECMO patients (63% versus 47%) than those patients treated with conventional management. ECMO's role as an invaluable means of support for patients with reversible respiratory failure was further bolstered by the 2009-2010 Influenza A/H1N1 global pandemic in which patients with H1N1-related ARDS treated with ECMO demonstrated a significantly lower mortality rate than those patients who were not referred for ECMO (23.7% versus 52.5%, $p < 0.05$)[36].

Although the majority of ECMO was originally performed using the venoarterial (VA) route, there has been significant progress made in the development of technology to allow for support of oxygenation and ventilation using the VV route in patients with normal cardiovascular function. VA-ECMO requires both an arterial and venous cannula and at times involves ligation of the carotid artery. Currently, this approach is typically reserved for patients requiring cardiovascular support. In VA-ECMO, a cannula is placed in the jugular vein to drain blood from the right atrium. A pump propels the blood through an oxygenator and then into the arterial cannula which returns the blood via direct cannulation of the aorta, carotid, axillary or femoral artery. Given its more invasive nature, there is an increased risk of significant morbidity with VA-ECMO. These issues have led to the increased use of VV-ECMO by most centers when treating isolated respiratory failure. In VV-ECMO, blood is drained from the right atrium and reenters the central venous circulation while preserving normal pulmonary blood flow and without the need for placement of a cannula into an artery. Although initially performed using two cannula (one placed into the internal jugular and the other into the femoral vein), VV-ECMO can now be performed using a single catheter placed into the internal jugular vein[37]. As used in our patient, a

significant innovation that has enhanced the capability of extracorporeal support was the development of a bicaval dual lumen catheter (Avalon Laboratories, Rancho Dominguez, CA) that allows respiratory support through a single catheter for application of ECMO[38]. This specially designed catheter drains venous blood from the superior and inferior vena cava and then returns oxygenated blood from the ECMO circuit to the right atrium ideally directed at the inlet of the tricuspid valve. This designs limits venous admixture and improves oxygenation during VV-ECMO. Additional modalities of extracorporeal respiratory support are being developed with may offer additional benefits over conventional VV or VA-ECMO techniques. The reader is referred to reference 39 for full descriptions of these modalities[39].

As was noted in our patient, in addition to its role as a therapeutic modality for patients with ARDS and ALI, ECMO can be used as a bridge to definitive treatment in patients with acute upper and lower airway obstruction. Wilms et al reported the use VA-ECMO in a 39-year-old man who experienced total obstruction of a distal tracheal stent by a tumor mass[40]. The occluding tracheal mass was removed with the use of forceps, suctioning and lavage while on ECMO. The authors reviewed 12 other reports in patients ranging from the neonatal period to 73 years of age with obstructing lesions of the upper and lower airway, which were managed with either VV or VA-ECMO before definitive treatment[41-52]. No mortality was reported in any of these cases.

In summary, we present the use of various modalities of respiratory support in an adolescent who was status post lung transplant and developed intraoperative respiratory failure. In addition to identifying the etiology of the respiratory failure, keys in the successful management of such patients includes the ability to move rapidly along a treatment algorithm to treat hypoxemic respiratory failure (Table 1). Key in the management of such patients includes consideration for the use of manipulations of ventilator techniques to augment MAP and oxygenation including low tidal volume/high PEEP techniques, permissive hypercarbia, use of an ICU ventilator in the OR, the addition iNO, as well as none conventional modes of ventilation such as HFOV. When these techniques fail, extracorporeal support may be life-saving especially for acute upper and lower airway obstruction.

Table 1

Algorithm for treatment of perioperative respiratory failure

1. Identify etiology (hand ventilate with 100% oxygen).
2. Treat etiology if feasible.
3. Correct or treat simple problems (auscultation for breath sounds, obtain chest radiograph
    a. Malpositioned endotracheal tube
    b. Secretions and mucus plugging (suction endotracheal tube)
    c. Bronchospasm
4. Adjust ventilator to increase mean airway pressure (use ICU ventilator as needed)
    a. Lengthen inspiratory time
    b. Increase PEEP
    c. Increase PIP (tidal volume)
5. Whenever feasible, use low tidal volume strategy with plateau pressure of less than 32 cmH_2O. Permissive hypercarbia (pH $\geq$7.2) and permissive hypoxemia (oxygen saturations 80%).
6. Maintain normothermia and treat hyperthermia aggressively to limit oxygen consumption and CO_2 production.
7. Limit the use of anesthetic agents that impair hypoxic pulmonary vasoconstriction.
8. Consider use of an ICU ventilator in the operating room with switch to total intravenous anesthesia. Alternative modes of ventilation including pressure-regulated volume controlled (PRVC) may be helpful.
9. Consider prone positioning if technically feasible.
10. Add inhaled nitric oxide starting at 20 ppm.
11. Consider switching to non-conventional mode of ventilation (high frequency).
12. Extracorporeal life support technology.

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