Severe Bronchospasm and Desaturation in a Child Associated with Rapacuronium

Steven S. Kron, M.D.*

The use of rapacuronium has been associated with occasional episodes of self-limited increased airway pressure with or without mild oxygen (O\textsubscript{2}) desaturation and wheezing. To our knowledge, this is the first report of severe bronchospasm associated with a transient inability to ventilate and marked O\textsubscript{2} desaturation after its administration.

Case Report

The patient was a 10-yr-old girl who was brought to the operating room to undergo appendectomy. Except for 1 week of abdominal pain and mild nausea but no vomiting, her history was negative. There were no previous anesthetics, environmental or drug allergies, recent upper respiratory infection, or reactive airway disease. No one in her household smoked. She weighed 62 kg and was 138 cm tall. Except for findings related to her surgical problem, her physical examination was unremarkable, and her chest was clear.

While the patient was preoxygenated for 3 min using a 6-l/min flow of O\textsubscript{2}, the usual monitors were applied. Then, 1 mg midazolam and 50 μg fentanyl were administered intravenously. After an additional 2 min of oxygen administration, a rapid sequence induction was performed with 150 mg propofol, immediately followed by 100 mg intravenous rapacuronium (approximately 1.6 mg/kg). After 30 s, O\textsubscript{2} saturation was noted to decrease from 100% to approximately 95%. We elected to administer ventilation by mask for an additional 45 s with 100% O\textsubscript{2} while applying cricoid pressure. Her chest rose, but O\textsubscript{2} saturation was noted to decrease from 100% to approximately 95%. We elected to extubate the patient deeply, so after 5 min of 4% sevoflurane, 2 l/min O\textsubscript{2}, and an equal train-of-four, nitrous oxide was discontinued. Surgery proceeded uneventfully with the removal of an inflamed retrocecal appendix. Muscle relaxation was reversed with 1 mg neostigmine and 0.2 mg glycopyrrolate. With the patient spontaneously breathing, volumes of 250–400 ml at a respiratory rate of 20 breaths/min, an equal train-of-four, nitrous oxide was discontinued. We elected to exubate the patient deeply, so after 5 min of 4% sevoflurane in O\textsubscript{2}, the stomach and oropharynx were thoroughly suctioned, and the endotracheal tube was removed. She continued to breathe 100% O\textsubscript{2} spontaneously, awoke, and was transferred to the postanesthesia care unit, where her O\textsubscript{2} saturation with room air was 99–100% and her chest was clear. Chest radiography results were negative. She was transferred to the pediatric unit and was discharged on the second postoperative day.

Discussion

This patient had no factors predisposing her to a reactive airway. Of the anesthetics used for induction, midazolam, fentanyl, and propofol usually are not associated with bronchospasm. Increased airway pressure with or without wheezing and O\textsubscript{2} desaturation has been reported in clinical studies of rapacuronium. The manufacturer’s package insert reports an incidence of 3.2%, with no data about the severity. Kahwaji et al.\textsuperscript{1} reported bronchospasm and erythema that developed in an American Society of Anesthesiologists class I patient 30 s after 2 mg/kg rapacuronium and gradually subsided after salbutamol. Mild wheezing developed in a second patient after a dose of 1 mg/kg. These authors suggest that histamine release is responsible. Fleming et al.\textsuperscript{2} studied 336 patients; half were intubated with 1 mg/kg succinylcholine, and half were intubated with 1.5 mg/kg rapacuronium. Bronchospasm, defined as wheezing, occurred in five of the rapacuronium patients, as opposed to only two of the succinylcholine patients. All but one of these patients had factors predisposing to broncho-
How Serious Is the Bronchospasm Induced by Rapacuronium?

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RAPACURONIUM (16-N-allyl, 17-β-propionate analog of vecuronium) is a new rapid-onset, short-acting, nondepolarizing steroidal neuromuscular blocking drug. Concern has been raised with respect to possible respiratory effects of rapacuronium. The following case report describes a patient in whom severe bronchospasm developed after rapacuronium administration.

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to 28 cm H₂O, and SpO₂ increased to 96%. The content of one albuterol nebulizer was administered during this event. Each actuation administers 90 μg albuterol. After this event, anesthesia was maintained with 60% nitrous oxide and desflurane (end-tidal concentration, 2.6–3.0%) in oxygen. Neuromuscular block was maintained with 0.2 mg/kg rocuronium. At the end of surgery, 0.03 mg/kg neostigmine and 0.006 mg/kg glycopyrrolate were administered to the patient for antagonism of residual neuromuscular block. The patient had an uneventful recovery. No further bronchospasm was noted during her postoperative course.

Discussion

Although the overall reported incidence of bronchospasm (in controlled trials) after rapacuronium and succinylcholine were 3.2 and 2.1%, respectively (data from Organon Inc., West Orange, NJ), others noted 18 incidents of such events (10.7%) after rapacuronium compared with 7 cases (4.1%) after succinylcholine. In the latter study, only one patient in the rapacuronium group, who had a history of obstructive airway disease, had severe bronchospasm after intubation. Our patient had severe bronchospasm after rapacuronium despite the facts that she was not asthmatic and that she received propofol for induction. Kahwaji et al. reported two serious adverse effects (tachycardia and bronchospasm) that occurred in a 29-yr-old, 100-kg man with American Society of Anesthesiologists physical status I within 30 s of administration of 2.0 mg/kg rapacuronium.

Rapacuronium may release histamine and produce slight changes in blood pressure and heart rate after administration. However, it seems that the bronchospasm noted with rapacuronium is mediated via mechanisms that do not seem to be related to histamine release. The affinity of neuromuscular blockers for the muscarinic receptor seems to have some influence on neural control of airway caliber. Pancuronium and atracurium (but not vecuronium) were found to enhance the increases in pulmonary resistance induced by vagus nerve stimulation, probably by blocking prejunctional muscarinic receptors (M2) that physiologically inhibit vagally mediated increases in pulmonary resistance. Blockage of M3 muscarinic receptors on airway smooth muscle inhibits vagally induced bronchoconstriction. Pancuronium and gallamine had affinities for the M2 muscarinic receptor within the clinical dose range. The affinity of rapacuronium for muscarinic receptors has not been studied. Therefore, there is insufficient evidence to make a definite statement on how rapacuronium induces bronchospasm. It seems prudent, therefore, to avoid using rapacuronium in patients with asthma or airway hyperreactivity.

References

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IN December 2000, subscribers to the Paediatric Anaesthesia Conference discussion group <PAC@anaes.sickkids.on.ca> were invited to post their experiences with rapacuronium (Raplon; Organon Inc., West Orange, NJ) in children in the USA. Specific side effects or responses to rapacuronium were neither requested nor encouraged. The postings yielded 19 cases of bronchospasm, of which 12 were described as severe, from six respondents. These reports caused such concern that the respondents felt obliged to bring this to the attention of their colleagues. Three of the cases are summarized below.

Case Reports

Case 1

The patient was a 12-yr-old, 64-kg boy with a diagnosis of acute appendicitis. He had a history of mild reactive airways disease that was characterized by occasional use of an albuterol inhaler but no previous hospital stays. His last occurrence of wheezing was 1–2 months before this admission. Chest examination results were unremarkable. In the operating room, a rapid sequence induction consisting of 1 mg intravenous midazolam, 75 μg fentanyl, 200 mg propofol, and 100 mg rapacuronium was performed. Cricoid pressure was applied, and the trachea was intubated by the resident. When squeezing the reservoir bag, the resident reported that the tube was not “in the trachea” and removed it. After reintubation, he remained unable to ventilate the lungs. No expired carbon dioxide was noted on the capnograph. The tube passed through the vocal cords, the lungs were ventilated vigorously, but there was no capnogram trace after two breaths. The tube was removed and the trachea was reintubated. Again, there was no capnogram trace. After confirming by direct laryngoscopy that the tube passed through the vocal cords, the lungs were ventilated vigorously for 60–90 s with 100% oxygen. During that time, air entry was auscultated, but there was no capnogram trace. After two breaths, the tube was removed and the trachea was reintubated. Again, there was no capnogram trace. After confirming by direct laryngoscopy that the tube passed through the vocal cords, the lungs were ventilated vigorously for 60–90 s with 100% oxygen. During that time, air entry was auscultated bilaterally. After treating these, the child was stabilized and recovered.

Case 2

The patient was a 41/2-yr-old girl who presented to the emergency department with the sudden onset of a prolonged seizure. The seizure had been treated with rectal diazepam. An anesthesiologist was consulted to intubate the trachea electively because of increasing respiratory distress. Medical history included a mild upper respiratory tract infection without fever or anorexia. Chest radiography was unremarkable. Rhinitis was noted in the history. Immediately after a single dose of both propofol and rapacuronium, the anesthesiologist noted “severe bronchospasm, dropping oxygen saturations, very difficult to ventilate.” The child then experienced asystole for 30 s, with a return of the pulse after a single dose of epinephrine. Chest radiography after stabilizing the child revealed bilateral pneumothoraces. After treating these, the child was stabilized and recovered.

Case 3

The patient was a 3-week-old, 3.4-kg healthy infant who presented for pyloromyotomy. After the stomach was suctioned, a rapid sequence induction was performed using 10 mg propofol, 7 mg rapacuronium, and 0.050 mg intravenous atropine. The trachea was intubated, but there was no capnogram trace after two breaths. The tube was removed and the trachea was reintubated. Again, there was no capnogram trace. After confirming by direct laryngoscopy that the tube passed through the vocal cords, the lungs were ventilated vigorously for 60–90 s with 100% oxygen. During that time, air entry was poor and bronchospasm was auscultated bilaterally. At this time, the chest appeared to move somewhat with inflation. The pulse oximeter did not register an oxygen saturation during this period, and heart rate slowed. As bradycardia developed, a capnogram trace began to appear, and, soon thereafter, the pulse oximeter registered a saturation of 99%. There was no evidence of a rash. With the return of the capnogram trace and an oxygen saturation reading, surgery and anesthesia proceeded uneventfully.

Discussion

A total of 19 cases of bronchospasm after rapacuronium were reported to the discussion group. All of the events occurred in children, whose ages ranged from infancy to adolescence. Four of the children had a confirmed history of reactive airways disease, whereas one did not. In two cases, rapacuronium was used to facili-
High-frequency Jet Ventilation in Life-threatening Bilateral Pulmonary Contusion

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TRIUMA is the leading cause of death among young people in developed countries. 1 Because up to 80% of trauma deaths occur during the first 24 h after trauma, 1 early resuscitation and rapid assessment of trauma lesions are of paramount importance to improving the prognosis. Among traumatic lesions, pulmonary contusion is frequent but has not been recognized as an independent prognosis factor. 2-4 In very few cases, pulmonary contusion may lead to severe hypoxia and hypercarbia, which cannot be adequately controlled using conventional mechanical ventilation. Hypoxia and hypercarbia may have deleterious effects, such as enhancement of brain injury and development of circulatory shock. 5 In the most severe cases, aggressive therapeutic methods, such as extracorporeal membrane oxygenation (ECMO), have been reported. 6 At our institution, high-frequency jet ventilation (HFJV) has been used routinely for many years for the treatment of severe acute respiratory distress syndrome. 7,8 We report a series of severe trauma patients with life-threatening pulmonary contusion successfully treated with HFJV when the conventional mechanical ventilation approach failed to provide appropriate gas exchange. The current data suggest that HFJV can be a life-saving technique in severely hypoxic patients with bilateral pulmonary contusion.
Table 1. Evolution of the Main Hemodynamic, Ventilatory, and Arterial Blood Gas Parameters in Nine Patients with Life-threatening Pulmonary Contusion during Conventional Mechanical Ventilation and High-frequency Jet Ventilation (HFJV)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Admission</th>
<th>Conventional Ventilation</th>
<th>HFJV</th>
<th>24 h after Admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial pH</td>
<td>7.21 ± 0.13</td>
<td>7.19 ± 0.14</td>
<td>7.31 ± 0.14</td>
<td>7.39 ± 0.08*</td>
</tr>
<tr>
<td>PacO₂ (mmHg)</td>
<td>56 ± 21</td>
<td>60 ± 18</td>
<td>51 ± 18</td>
<td>40 ± 6†</td>
</tr>
<tr>
<td>PaO₂ (mmHg)</td>
<td>91 ± 71</td>
<td>73 ± 24</td>
<td>237 ± 26†</td>
<td>185 ± 52†</td>
</tr>
<tr>
<td>FiO₂ (%)</td>
<td>100 ± 0</td>
<td>100 ± 0</td>
<td>96 ± 13</td>
<td>58 ± 17†</td>
</tr>
<tr>
<td>PaO₂/FiO₂</td>
<td>91 ± 71</td>
<td>73 ± 24</td>
<td>254 ± 101†</td>
<td>322 ± 49†</td>
</tr>
<tr>
<td>Tidal volume (ml)</td>
<td>563 ± 95</td>
<td>669 ± 151</td>
<td>136 ± 59†</td>
<td>143 ± 45†</td>
</tr>
<tr>
<td>Ventilatory rate (breaths/min)</td>
<td>21 ± 9</td>
<td>16 ± 2</td>
<td>250 ± 51†</td>
<td>233 ± 83†</td>
</tr>
<tr>
<td>EEP (cm H₂O)</td>
<td>4 ± 5</td>
<td>10 ± 3†</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Pₐap (cm H₂O)</td>
<td>—</td>
<td>—</td>
<td>11 ± 2</td>
<td>10 ± 2</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>70 ± 23</td>
<td>86 ± 16</td>
<td>85 ± 16</td>
<td>90 ± 13</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>100 ± 18</td>
<td>114 ± 7</td>
<td>112 ± 19</td>
<td>109 ± 16</td>
</tr>
</tbody>
</table>

Initial data with HFJV were obtained within 1 h after the onset of HFJV. Data are mean ± SD.
* P < 0.05 versus admission. † P < 0.05 versus conventional ventilation.

PacO₂ = arterial partial pressure of carbon dioxide; PaO₂ = arterial partial pressure of oxygen; FiO₂ = inspired fraction of oxygen; EEP = end-expiratory pressure; Pₐap = mean airway pressure; MAP = mean arterial pressure.

Patients and Methods

During a 6-yr period (1990–1996), 1,241 severe trauma patients were admitted to our Level 1 Trauma Centre. All trauma patients who received HFJV during the first 24 h after admission were identified and included in the study. One of these patients has been reported previously.7,8 The decision to perform HFJV was made by the senior anesthesiologist in charge of the emergency room because of the severity of pulmonary contusion. All patients fulfilled the following criteria: (1) partial pressure of oxygen (PacO₂) less than 100 mmHg with an inspired fraction of oxygen (FiO₂) of 100%; (2) progressive decrease in PaO₂ during the last hours without any trend to stabilization or improvement; (3) failure of further increases in positive end-expiratory pressure (PEEP) to improve PaO₂ or impossibility to increase PEEP because of hemodynamic consequences; (4) bilateral pulmonary contusion. An anesthesiologist and a nurse from the surgical intensive care unit initiated and adjusted HFJV in the emergency room. The decision to implement HFJV during the early period after trauma admission always was related to severe hypoxemia or circulatory shock. We did not include patients in whom HFJV was initiated because of systemic gas embolism related to pulmonary contusion.10

The following data were collected: age; sex; trauma lesions; administration of colloids, crystalloids, blood, and catecholamines; duration of stay in intensive care unit; and mortality. The following ventilatory parameters were recorded during conventional mechanical ventilation: ventilatory rate, tidal volume, and PEEP. The following parameters were measured at admission, during conventional mechanical ventilation, just before HFJV, 15–30 min after HFJV, and 24 h after admission: arterial pH, PaO₂ and partial pressure of carbon dioxide (PacO₂), PaO₂/FiO₂ ratio, mean arterial pressure measured using an indwelling radial or femoral artery catheter, and heart rate. The following indices were calculated: injury severity score (ISS), probability of survival according to the Trauma and Injury Severity Score (TRISS) methodology,11 and the Lung Injury Severity Score. (LIS)12

High-frequency jet ventilation was performed using an AMS 1000 ventilator (Acutronic Medical Systems AG, Hirzel, Switzerland). Rewarming and humidification of gases were provided by a HH-812 Jet humidifier (Acutronic Medical Systems). Additional conventional ventilation (low rate, 4–6 breaths/min; low tidal volume, 75–100 ml) was obtained using a CPU 1 ventilator (Ohmeda, Maurepas, France). Mean airway pressure was monitored continuously with a catheter located in the trachea 10 cm distal to the tip of the injector cannula, as previously reported.7,8

Data are mean ± SD. Comparison of several means was performed using repeated-measures analysis of variance. A P value less than 0.05 was considered significant.

![Fig. 1. Evolution of the partial pressure of oxygen/inspired fraction of oxygen (PaO₂/FiO₂) ratio during the first 24 h in nine patients with life-threatening pulmonary contusion. Initial data with high-frequency jet ventilation (HFJV) were obtained within 1 h after the onset of HFJV. CV = conventional mechanical ventilation; 24 hrs: 24 h after admission, during HFJV.](image-url)
Results

Over a 6-yr period, HJV was used during the first 24 h after admission in nine patients (six male, three female) because of life-threatening hypoxemia related to pulmonary contusion. The incidence of life-threatening pulmonary contusion defined by this criteria was 0.73% (95% confidence interval, 0.26–1.20%). The mean age was 29 ± 15 yr (range, 8–58 yr), the mean ISS was 50 ± 17 (range, 20–75), the mean TRISS was 0.63 ± 0.36 (range, 0.08–0.96), and the mean LIS was 3.4 ± 0.3 (range, 3–4). These scores signify severe multisystem injuries. Associated head trauma was present in seven patients, abdominal trauma was present in four patients, spine trauma was present in four patients, and pelvic trauma in was present in two patients. All patients had severe thoracic trauma with bilateral pulmonary contusion. Chest radiography showed bilateral extensive and diffuse alveolar hyperdensities in all cases. Other traumatic thoracic lesions included hemothorax in three patients, pneumothorax in two patients, and aortic rupture in one patient. All patients underwent transesophageal echocardiography: mean left ventricular ejection fraction was 45 ± 29% (range, 10–71%), myocardial contusion was observed in five patients, and three patients had severe decrease in left ventricular ejection fraction (<30%). A search for a patent foramen ovale was performed in five patients; all results were negative. During the first 24-h period, fluid resuscitation consisted of 1.8 ± 1.3 l crystalloids, 3.7 ± 1.8 l colloids, 7 ± 8 units packed erythrocytes, 3 ± 3 units fresh frozen plasma, and 4 ± 5 platelet units. Catecholamines were administered in eight patients (epinephrine in three patients, norepinephrine in four patients, dopamine in five patients, and dobutamine in one patient). After HJV, the dose of catecholamines was decreased in six patients and increased in two patients.

Tracheal intubation was performed in all patients during the early resuscitation phase (delay, 51 ± 68 min after trauma). HJV was initiated 7 ± 6 h after trauma. Table 1 shows the evolution of the main hemodynamic, ventilatory, and blood gas parameters. Figure 1 depicts the evolution of the PaO2/FIO2 ratio during the first 24 h.

The mean stay in the intensive care unit was 40 ± 38 days. Death occurred in four patients and was always related to severe brain injury. In the five surviving patients, HJV was maintained for 7 ± 5 days (range, 3–15 days).

Discussion

We report that HJV dramatically increased PaO2 in a group of trauma patients with life-threatening hypoxemia related to bilateral pulmonary contusion. Despite severe pulmonary contusion, death always occurred because of brain injury and not because of pulmonary contusion, and weaning of HJV was obtained successfully in all the remaining patients. These results confirm previous reports that suggest that HJV can be effective as a rescue therapy for refractory acute lung dysfunction.

The current study cannot definitely assess the mechanisms involved in the beneficial effect of HJV. Because of the emergency and critical conditions, a pulmonary computed tomography scan could not be obtained in most of these patients at the early phase. Nevertheless, thoracic radiography highly suggested that diffuse pulmonary contusion occurred, and thus that alveolar recruitment induced by HJV was likely the main mechanism responsible for the marked increase in PaO2 observed in our patients. HJV is known to induce an increase in functional residual capacity by trapping intrapulmonary gases because of incomplete exhalation during the short expiratory time (auto-PEEP effect). Two other mechanisms should be considered. First, closure of a patent foramen ovale may have reduced hypoxemia, as previously reported. However, it should be noted that an increase in pulmonary artery pressure (potentially leading to right-to-left intracardiac shunt) usually is not observed at the very early stage of pulmonary contusion and that we failed to find evidence of any patent foramen ovale using transesophageal echocardiography in five of these patients. Second, an improvement in hemodynamic conditions may have contributed to the HJV-induced increase in PaO2. This last effect can be complex because an increase in cardiac output can decrease PaO2 through capillary recruitment but also can increase PaO2 through an increase in mixed venous oxygen saturation. In patients with septic shock, Fusiardi et al. have shown that mean arterial pressure and cardiac output are higher during HJV than during conventional mechanical ventilation when compared at the same airway pressure and PACO2. However, this hemodynamic improvement was associated with a small deterioration in arterial oxygenation.

Because the mean PEEP value was not high in our study, one can argue that a marked increase in PEEP might have induced an effect similar to that observed with HJV. In our patients, such an increase in PEEP could not be applied without marked alteration in hemodynamic conditions. Associated right ventricle contusion is likely to explain that our patients poorly tolerated any further increase in intrathoracic pressure. At an identical level of mean airway pressure, HJV is better hemodynamically tolerated than PEEP in shocked patients. It should be pointed out that high PEEP is associated with high peak inspiratory pressure that can be harmful in patients with pulmonary contusion because it increases pulmonary edema, causes barotrauma through alveolar rupture, and facilitates pulmonary venous gas embolism. It has been demonstrated recently that reducing tidal volume during mechanical ventilation in ARDS decreases mortality. High PEEP and low tidal volume induce hypercapnia that is delete-
rious in patients with head trauma. As shown in table 1, HFJV enabled control of PaCO₂ and PaO₂ in patients with severe life-threatening pulmonary contusion. Moreover, the fluid loading required to overcome the hemodynamic effects of PEEP on venous return also may increase extravascular lung water.

In conclusion, in rare cases of severe bilateral pulmonary contusion refractory to conventional mechanical ventilation, HFJV may be a life-saving procedure. Because of the rarity of these cases, there is a low possibility that a randomized trial could ever be conducted. Therefore, traumatologists, intensivists, and anesthesiologists should be aware of this therapeutic possibility and should try HFJV before irreversible consequences of hypoxemia or hypercarbia occur in these severe trauma patients. Moreover, HFJV is probably a more simple procedure than ECMO, which sometimes has been used in such patients but usually is contraindicated in severe head trauma.

References


Jet Ventilation for Fiberoptic Bronchoscopy

ACUTE pulmonary or lobar collapse is a troublesome complication after thoracic surgery and may mandate aggressive intervention. Therapeutic fiberoptic bronchoscopy is a standard intervention for the treatment of atelectasis in the intensive care setting.¹


Jet Ventilation for Fiberoptic Bronchoscopy

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After tracheal resection, airway management for fiberoptic bronchoscopy in patients with general anesthesia is frequently difficult, particularly in cases in which the resected tracheal segment is long (e.g., 4-5 cm) and the degree of postoperative neck flexion is necessarily extreme. We report a case of intermittent low-frequency jet ventilation, applied via the instrument channel of a flexible fiberoptic bronchoscope (FFB), for the emergency treatment of postoperative atelectasis after tracheal reconstruction.

Case Report

An 18-year-old male underwent cardiopulmonary resuscitation after arrhythmia complicating acute myocarditis. After 3 days of ventilatory support, he was extubated successfully and was discharged subsequently. Six weeks later, he was referred to our university hospital. At the time of presentation, he exhibited respiratory distress with inspiratory and expiratory stridor. Rigid bronchoscopy revealed severe tra-
CASE REPORTS

The optimum technique for airway management was the subject of much deliberation in this case. Extreme forward tilting of the head prevented insertion of a laryngeal mask airway or an endotracheal tube via a traditional intubation technique. In addition, the possibility of direct tracheal suture injury, even with careful insertion of the endotracheal tube, was of great concern. Finally, we opted for the combination of intravenous anesthesia and manual ventilation via face mask as described.

There is very limited information in the literature about the use of the instrument (“suction”) channel of the FFB for jet ventilation. In 1980, Satyanarayana et al.5 evaluated this technique in animals and in eight adult volunteers scheduled to undergo minor surgery. He observed excellent arterial oxygenation and hypocarbia and reported no complications. Dalens et al.4 reported a series of 49 pediatric patients with respiratory distress, aged 3 days to 3 yr, who underwent bronchoscopic examination using a similar technique. As in our report, after every distal exploration, the FFB tip was withdrawn to above the carina to ensure bilateral pulmonary insufflations, which were then given for 15 s at a respiratory rate of 50 to 60 breaths/min. Gas exchange was acceptable, and no complications were observed.

In the latest report, Baraka5 applied a similar technique in a 2-yr-old child who was scheduled for bronchoscopy because of atelectasis and pneumonic infiltrates. Unfortunately, the pediatric bronroscope (3.4 mm OD) could not pass easily through a 4-mm endotracheal tube. He tried to perform the procedure via a size 2 laryngeal mask airway (7.0 mm ID), which was technically feasible, but low lung compliance made mechanical ventilation difficult, and, after a decrease in SpO2 to 85%, intermittent jet ventilation of oxygen via the instrument channel ensured adequate oxygenation.

These latter two reports imply that the technique can be used safely even in patients with ventilatory compromise.4,5 Although they involved children, we believe that the principles involved may be equally applicable in certain situations in the adult.

One of the obvious risks of this approach is the potential for inadvertently causing barotrauma if the tip of the FFB slips beyond the carina and “wedges” in a bronchus. The added advantage of jetting via the instrument channel of the FFB is that the position of the jet source can be directly controlled visually, thus enhancing the safety of the procedure. Excessive hyperinflation must be avoided also, and effective exhalation must be ensured. The FFB OD is 5.8 mm, whereas the narrowest lumen diameter of the normal tracheal segments was measured by computed tomography as 15 mm. Therefore, difficulties with exhalation are more likely to originate at laryngeal or upper airway level and can be minimized by ensuring a clear airway. If gas trapping is still a feature, it is easily

Discussion

This case highlights two particular difficulties that may be faced in the management of pulmonary atelectasis after tracheal reconstruction. First, extreme flexion of the neck presents substantial difficulties for securing a stable airway during an anesthetic procedure. In addition, there is the potential for direct damage to the tracheal anastomoses from any tracheal instrumentation, particularly from blindly placed endotracheal tubes.

The optimum technique for airway management was the subject of much deliberation in this case. Extreme forward tilting of the head prevented insertion of a laryngeal mask airway or an endotracheal tube via a traditional intubation technique. In addition, the possibility of direct tracheal suture injury, even with careful insertion of the endotracheal tube, was of great concern. Finally, we opted for the combination of intravenous anesthesia and manual ventilation via face mask as described.

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These latter two reports imply that the technique can be used safely even in patients with ventilatory compromise.4,5 Although they involved children, we believe that the principles involved may be equally applicable in certain situations in the adult.

One of the obvious risks of this approach is the potential for inadvertently causing barotrauma if the tip of the FFB slips beyond the carina and “wedges” in a bronchus. The added advantage of jetting via the instrument channel of the FFB is that the position of the jet source can be directly controlled visually, thus enhancing the safety of the procedure. Excessive hyperinflation must be avoided also, and effective exhalation must be ensured. The FFB OD is 5.8 mm, whereas the narrowest lumen diameter of the normal tracheal segments was measured by computed tomography as 15 mm. Therefore, difficulties with exhalation are more likely to originate at laryngeal or upper airway level and can be minimized by ensuring a clear airway. If gas trapping is still a feature, it is easily

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recognized clinically and can be managed simply by intermittent interruption of jet ventilation.

Another risk, albeit theoretical, that should be considered is the possibility of direct damage to the FFB. The instrument channel in this type of FFB is a polytetrafluoroethylene tube and is unlikely to be damaged if it remains patent. In addition, the risk that the channel could be blocked so tightly that it could lead to a rupture is negligible.

Although the indications for jet ventilation via the instrument channel of an FFB may be rare, they are more likely to be applicable in a challenging situation and particularly in the presence of a difficult airway management problem. In this case report, the technique greatly facilitated the therapeutic procedure and enhanced its safety. In addition, it decreased the risk of inadvertent tracheal trauma caused by the need for frequent removal and reinsertion of the FFB when applying face mask ventilation.

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