Anesthesiology 2010; 112:970–8

Bruno Riou, M.D., Ph.D., Editor

Case Scenario: Perioperative Airway Management of a Patient with Tracheal Stenosis

Shiroh Isono, M.D.,* Yuji Kitamura, M.D.,† Takashi Asai, M.D., Ph.D.,‡ Tim M. Cook, F.R.C.A.§

TRACHEAL stenosis is a rare but a life-threatening condition and is caused by congenital problems, postintubation injury, trauma, tracheal tumor, and compression of the trachea by tumor. Although accurate prevalence of this condition is unknown, an incidence of 4.9 cases per million per year is estimated for postintubation tracheal stenosis.[1] A stenosis commonly occurs at the cuff of the tube (intrathoracic trachea) or at the level of the tracheostomy stoma (extrathoracic trachea).

Anesthesia of a patient with tracheal stenosis is challenging for anesthesiologists. Depending on the severity and location of the stenosis and the type of surgical procedure, there may be a variety of choices for perioperative airway management such as a facemask, laryngeal mask airway,[2] an tracheal intubation tube,[3,4] cardiopulmonary bypass,[5] and extracorporeal membrane oxygenation.[6] The American Society of Anesthesiologists practice guidelines for management of the difficult airway primarily focus airway problems caused at the extrathoracic airway and may not be helpful, particularly for managing patients with intrathoracic tracheal stenosis.[7] In this case scenario, we present a patient with severe intrathoracic tracheal stenosis, who required surgery for a lumbar fracture in the prone position. Various airway management strategies and the actual management used are discussed.

Preoperative Information of the Case

A 38-yr-old obese man (height, 172 cm; weight, 95 kg; body mass index, 32 kg/m^2) was scheduled to have a thoracolumbar laminectomy and fixation for a burst fracture of the first lumbar vertebra. Surgery was to be performed in the prone position. The operation duration and blood loss were preoperatively estimated to be 4 h and 500 ml. He had a history of prolonged intubation when he suffered a traumatic brain injury at 8 yr of age. He had epilepsy treated with phenobarbital but had no impairment of neurologic development and was cooperative. Despite undergoing tracheal resection and plasty for severe postintubation tracheal stenosis at 17 yr of age, he had relatively loud inspiratory and expiratory stridor while awake. Spirometry in the sitting position revealed reduced forced expiratory volume in the first second (FEV_1 = 1.95 l, 53%-predicted) and peak expiratory flow rates (PEF = 180 l/min, 30% predicted). Arterial blood gas analysis indicated mild impairment of oxygenation but normal ventilation (F_{IO_2} = 0.2, pH = 7.43, Pa_{O_2} = 71 mmHg, Pa_{CO_2} = 33 mmHg). A flow-volume loop showed a typical upper airway obstruction pattern (fig. 1). Three-dimensional computed tomography (CT) of the trachea revealed severe intrathoracic tracheal stenosis more than 3 cm in length. In cross-section, the stenotic lesion was elliptical with a minor axis of 0.5 cm and a major axis of 1.5 cm (fig. 2). Despite the tracheal stenosis, he had no dyspnea during daily activities and was otherwise healthy. Nurses in the ward witnessed loud snoring and occasional apnea during sleep. Preoperative airway examination revealed Mallampati class 3, normal thyromental distance, and no limitation of neck or mandible movements. The orthopedic surgeons considered that neither conservative therapy nor surgery with regional anesthesia was appropriate because of his neurologic symptoms and the estimated operational duration and invasiveness of the surgery.

* Associate Professor, Department of Anesthesiology, Graduate School of Medicine, Chiba University, Chiba, Japan. † Staff Anesthesiologist, Department of Anesthesiology, Chiba Emergency Medical Center, Chiba, Japan. ‡ Assistant Professor, Department of Anesthesiology, Kansai Medical University Takii Hospital, Osaka, Japan. § Consultant in Anaesthesia and Intensive Care Medicine, Department of Anaesthesia, Royal United Hospital, Bath, United Kingdom.

Received from the Department of Anesthesiology, Graduate School of Medicine, Chiba University, Chiba, Japan; Department of Anesthesiology, Chiba Emergency Medical Center, Chiba, Japan; Department of Anesthesiology, Kansai Medical University Takii Hospital, Osaka, Japan; and Department of Anaesthesia, Royal United Hospital, Bath, United Kingdom. Submitted for publication November 22, 2009. Accepted for publication December 18, 2009. Support was provided solely from institutional and/or departmental sources.

Address correspondence to Dr. Isono: Department of Anesthesiology, Chiba University, Graduate School of Medicine, 1-8-1 Inohana-cho, Chuo-ku, Chiba 260-8670, Japan. isonos-chiba@umin.ac.jp; shirohisono@yahoo.co.jp.

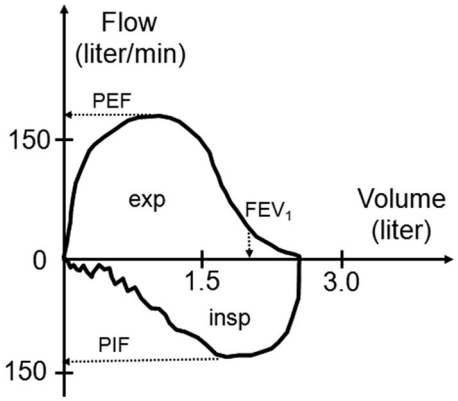

Fig. 1. A flow-volume loop during forced expiration and inspiration in this case. Note marked reduction in both PEF and PIF. insp = inspiration; exp = expiration; PEF = peak expiratory flow rate; PIF = peak inspiratory flow rate; FEV_1 = forced expiratory volume in the first second.

Airway Management Plans by Airway Experts

Only the preoperative information with the figures described above was initially sent to both Drs. Asai and Cook. They were selected because of their previous reports of similar cases.[3,4] The following are their airway management plans for this patient.

Dr T. Asai

I follow an algorithm for anesthetic management of patients with tracheal stenosis based on its pathophysiology (fig. 3). Although the patient had relatively loud inspiratory and expiratory stridor and apnea during sleep, he had no dyspnea during daily activities. Therefore, I consider that spontaneous breathing or mechanical ventilation is likely to be possible through the stenosis with general anesthesia. Nevertheless, severe airway obstruction may occur during induction of general anesthesia, and thus the appropriate backup method will be required to prevent disaster.

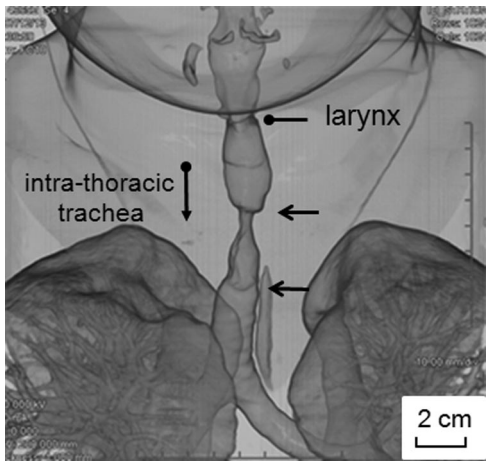

Fig. 2. Tracheal three-dimensional computed tomography of this case revealed severe intrathoracic tracheal stenosis over 3 cm in length (*arrows*) and elliptical airway shape with a minor axis of 0.5 cm and a major axis of 1.5 cm.

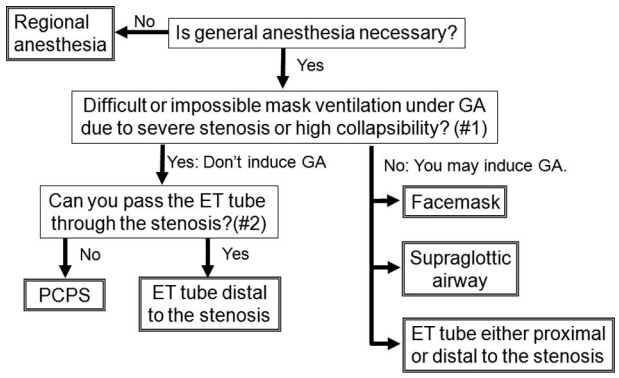

Fig. 3. An algorithm for anesthetic management of patients with tracheal stenosis. #1: Airway imaging and pulmonary function test are helpful for this decision making. #2: Collapsibility or expandability of the trachea needs to be assessed for this decision making. GA = general anesthesia; ET = endotracheal tube; PCPS = percutaneous cardiopulmonary support.

There are three possible methods for airway management of this case: (1) the use of a supraglottic airway alone, (2) the use of a supraglottic airway and a tube-exchange catheter, and (3) the use of a supraglottic airway, an endotracheal tube, and a tube-exchange catheter such as Cook Airway Exchange Catheter (Cook Medical, Bloomington, IN; 2.7 mm internal diameter [ID]). The choice of method would depend on both the risks of airway obstruction or dislodgement of the selected airway device and accessibility of the airway for reinsertion of the device.

In this case, the airway might be managed with a supraglottic airway alone, but there are two major potential problems with this method: airway obstruction after induction of anesthesia and dislodgement of the supraglottic airway (particularly when the patient is turned to prone position from the supine position). One possible solution is to place the patient in the prone position and insert a supraglottic airway while the patient is still awake, and then induce anesthesia with increasing concentrations of sevoflurane while maintaining spontaneous breathing. Although the presence of the supraglottic airway would prevent airway obstruction above the vocal cords, worsening of the tracheal stenosis and hence severe airway obstruction may develop during inhalational induction with sevoflurane. In such a case, administration of sevoflurane should be terminated and the patient should be woken up. If inadvertent dislodgement of the airway device in the prone position is a risk, safety would be increased by prior insertion of a tube-exchange catheter, because this would enable both the maintenance of oxygenation until reinsertion of the supraglottic airway and the tracheal intubation through it.[4,8]

Alternatively, a more conservative but a safer approach, which I consider the most appropriate in this case, is tracheal intubation with the two backup methods of the use of both a supraglottic airway and a tube-exchange catheter. In this case, the narrowest caliber of the trachea is 5 mm, and thus the largest size of an endotracheal tube, which can be passed through the stenosis, would be 4.0 mm ID, and ventilation

may not be sufficient. Therefore, it would be necessary to insert a larger endotracheal tube with its tip proximal to the stenosis. Three-dimensional CT indicates that the stenosis is in the mid to lower trachea, and thus it would only be possible to insert the distal 3–4 cm of the endotracheal tube into the trachea, necessitating backup plans, in case of tube dislodgement. In such an event, either the supraglottic airway or the exchange catheter could then be used for maintaining oxygenation and reinserting the endotracheal tube. I would prepare for jet ventilation through the exchange catheter.

After preoxygenation of the patient in the supine position, I would allow the patient to breathe increasing concentrations of sevoflurane in oxygen, and then assist ventilation manually *via* a facemask. After injection of a neuromuscular blocking agent, I would insert a Cook airway exchange catheter into the trachea under direct laryngoscopy, and then insert either the *ProSeal Laryngeal Mask Airway*™ (*PLMA*™; Laryngeal Mask Company, Henley-on-Thames, United Kingdom) #5 or i-gel (Intersugical Ltd., Wokingham, Berkshire, United Kingdom), another supraglottic airway, while the exchange catheter is placed outside the supraglottic airway. With the aid of a fiberoptic bronchoscope, I would pass a reinforced endotracheal tube through the supraglottic airway into the trachea so that the tip of the endotracheal tube is approximately 1–2 cm proximal to the stenosis. I would not inflate the endotracheal tube cuff, because it would be positioned at the glottis. Wrapping adhesive tape around the endotracheal tube at the connecter of the supraglottic airway would prevent both dislodgement of the endotracheal tube and gas leakage through the supraglottic airway. I would then adjust the position of the exchange catheter so that its tip is beyond the tracheal stenosis. After the patient is turned to the prone position, I would confirm (using a fiberscope) the appropriate positions of both endotracheal tube and exchange catheter. I would maintain the tidal volume as low as possible allowing hypercapnia to prevent excessive peak airway pressure. When possible, spontaneous breathing would be resumed. After surgery, I would remove the supraglottic airway once the patient has recovered from general anesthesia and is responsive to verbal commands, but would leave the tube exchange catheter in place, until it becomes certain that the patient can maintain a clear airway.

Dr. T. M. Cook

This is a truly difficult patient. I would first reiterate to the surgeons that perioperative airway complications are a potential risk to the patient's life. The options of conservative treatment or transfer to a center with facility for combined tracheal reconstructive and trauma surgery must be explicitly considered.

Assuming neither is possible I would premedicate the patient with a proton pump inhibitor 12 h before anesthesia. Two experienced anesthetists and an experienced anesthetic assistant would be required and briefed. I would start by placing a narrow gauge cricothyroid cannula specifically a 13-gauge Ravussin cannula (VBM Medizintechnik, Sulz, Germany) with local anesthesia and confirm its position by feeling expired gas, seeing gas exit through a bubble of saline and with capnography. If there was concern about the position of the Ravussin cannula, I would perform awake fiberoptic inspection to confirm its position before proceeding. Next, I would place a *PLMA*™. If the patient was cooperative, I would do this during topical anesthesia. If he was not I would place it during general anesthesia. I would preoxygenate the patient fully with continuous positive airway pressure performed with at least 25 degrees head up position to increase lung volumes and maximize the apnea period before hypoxemia develops. I would administer a modest dose of opioid (*e.g.*, fentanyl 100 μg titrated in > 2–3 min) and propofol by target-controlled infusion. I would start with a low propofol effect site target (1–1.5 μg/ml) and increase this in steps of 0.5 μg/ml for every 1–3 min while maintaining spontaneous ventilation. At the point of eye closure, but before full anesthesia, I would assess ease of assisted ventilation. If ventilation was difficult or impossible, I would abandon this attempt and allow the patient to wake up. After confirmation of adequate mask ventilation, I would then paralyze with rocuronium, increase the depth of anesthesia, and insert a *PLMA*™, using a bougie-guided technique.[9,10] After placement of the *PLMA*™, I would then intubate the trachea through it. If the *PLMA*™ was placed awake, I would induce anesthesia after *PLMA*™ placement. For intubation, I would use a 4.2-mm fiberscope on which an Aintree Intubating Catheter (AIC; Cook Medical, Bloomington, IN) was mounted. After passage of the AIC, I would railroad a 6.5-mm ID intubating *Laryngeal Mask Airway*™ (*LMA*™) endotracheal tube over it. If the AIC passed easily, I anticipate that the intubating *LMA*™ endotracheal tube would also pass. If the AIC was tight/snug, I would pass a Cook airway exchange catheter through the AIC and railroad a 5.0-mm ID microlaryngoscopy tube over the airway exchange catheter. I would then confirm position of the endotracheal tube beyond the stenosis. If the AIC could not pass without undue force, I would ventilate until paralysis was reversed (sugammadex may be useful here due to its ability to produce rapid and complete reversal of rocuronium paralysis) and then wake up the patient. During surgery, I would administer 8.0 mg of intravenous dexamethasone to minimize edema of the stenotic region. At the end of surgery, I would exchange the endotracheal tube for a Cook airway exchange catheter and a *PLMA*™. I would then assess ease of ventilation (and spirometry) with the patient still anesthetized. I would then allow the patient to wake and remove the *PLMA*™, but not the exchange catheter. If there was any suggestion of trauma during the intubation or concern about edema at the time of extubation, I would admit the patient to intensive care unit for 24–48 h of sedation, ventilation, and steroid to allow airway edema to settle.

Actual Airway Management in This Patient

The history of tracheal surgery and persistent stridor suggested a rigid tracheal wall at the stenotic region allowing insertion of an endotracheal tube with 5–7 mm outer diameter, that is, only

4.0–5.5 mm ID without injuring the tracheal wall. We considered that positive pressure ventilation with such a small diameter tube might be difficult in this obese patient during surgery in prone position. Furthermore, traumatic insertion and prolonged placement of a larger diameter tube were considered to be disadvantageous because of the potential for development of mucosal edema and further narrowing of the trachea after tracheal extubation. Therefore, we decided not to intubate the trachea, but to use the *PLMA*™ for positive pressure ventilation. The patient agreed with this strategy after we explained its potential benefits and risks to him.

Because of clinical symptoms and body habitus suggesting potential obstructive sleep apnea, we performed nocturnal oximetry preoperatively. We calculated 4% oxygen desaturation index (*i.e.*, the average number of oxygen desaturations by 4% or more below the baseline level per hour). Although an oxygen desaturation index greater than 5 h^{-1} is suggestive of sleep-disordered breathing, the index was 3 h^{-1} in this patient.[11] Despite the negative result of the sleep study, nasal continuous positive airway pressure was prescribed because this could help maintain tracheal patency both for treatment of his snoring and in case of mucosal edema at the tracheal stenosis developed after surgery. The patient tolerated this treatment well.

General anesthesia was induced with intravenous administration of remifentanil, propofol, and vecuronium, and a *PLMA*™ (#5) was inserted, guided by a gum elastic bougie. Anesthesia was maintained with inhaled sevoflurane and an infusion of intravenous remifentanil. With pressure-controlled ventilation during surgery (peak inspiratory pressure 22 cm H_2O, positive end-expiratory pressure 7 cm H_2O, respiratory rate 8 breaths/min, inspiratory expiratory ratio 1:3) through the *PLMA*™, we saw no signs of high airway resistance or airflow limitation such as low tidal volume or lack of formation of an alveolar plateau on capnography (tidal volume 730 ml, end-tidal CO_2 31 mmHg). The surgery was uneventfully accomplished. The *PLMA*™ was removed when the patient was fully aroused. Optimal postoperative analgesia was achieved by intravenous injection of nonsteroidal antiinflammatory drug and a continuous intravenous infusion of fentanyl. After the patient arrived on the ward, nasal continuous positive airway pressure with oxygen was applied immediately and was continued for three postoperative nights. This was effective in eliminating both snoring and stridor during sleep.[12] He did not complain of dyspnea after surgery and was discharged fully mobile.

Comments by the Airway Experts on Perioperative Airway Management

Dr. T. Asai

I believe that preoperative assessment and anesthesia management of the case described are generally reasonable and accord with my assessments and plans.

The preoperative respiratory state during wakefulness and sleep was sufficiently assessed, and the visual assessments of the stenotic region with three-dimensional CT imaging of the trachea in addition to chest radiographs were informative. These meticulous assessments may certainly be useful to plan a safer anesthesia management (providing that the time and cost can be spent). Nevertheless, caution may be required, because the absence of significant airway obstruction during sleep does not guarantee that there will be no airway obstruction during anesthesia. There have been several reports of complete airway obstruction in patients with mediastinal masses, without any preoperative signs of airway obstruction.[13] In this case, the three-dimensional CT and preoperative assessments suggest that complete airway obstruction is unlikely, but it might have been safer to induce anesthesia by inhalation of increasing concentrations of a volatile anesthetic (such as sevoflurane) and then to give a neuromuscular blocking agent after confirming that no airway obstruction has occurred.

There may be two major possible problems with the anesthesia management performed by Drs. Isono and Kitamura. First, air trapping may occur beyond the stenosis, when the ventilation is controlled. This can be reduced by decreasing the inspiratory/expiratory ratio (that is increasing the expiratory time). In this case, because the stenosis had a minimally acceptable caliber, the pressure-controlled ventilation worked well, and sufficient ventilation volume was obtained. Spontaneous breathing might have been a better choice if ventilation had been insufficient during controlled ventilation.

Another possible problem is that the use of the *PLMA*™ alone might have become difficult if (although not likely in this case) the device had been inadvertently dislodged or airway obstruction at the stenotic region had occurred. If there are concerns about access to the patient's face, or about a prolonged operation time, I would use a backup method of leaving a tube-exchange catheter beyond the stenosis and possibly an endotracheal tube with its tip proximal to the stenosis.

Postoperatively, nasal continuous positive airway pressure, known to minimize airway obstruction, was applied for three postoperative nights. Because tracheal intubation seemed not difficult, the choice of applying nasal continuous positive airway pressure postoperatively seems better than my plan of leaving a tube exchanger to the trachea after operation.

Dr. T. M. Cook

For each possible solution that is explored, several potential complications arise. I will limit myself to answering the following specific questions. I do not know whether my plans would work: Drs. Isono and Kitamura have the massive advantage of knowing their plans did! In many respects what matters most in this case is not what plan A is, but how the anesthetist plans to respond if plan A fails immediately or mid-surgery. It is essential for the anesthetist to have a plan B, as a minimum, before induction of anesthesia.

How Do I Assess the Problem? My first consideration is that all aspects of this patient's care potentially put him at

risk. His features offer the possibility of supraglottic and subglottic problems leading to both difficult mask ventilation and difficult intubation.[14] Hypoxemia will be rapid and severe if the airway is lost in this obese patient who will have a limited functional residual capacity. Passage of a tube beyond his tracheal narrowing may be the most problematic.

However, there are two patient features that I find reassuring. First, the fact that the tracheal narrowing (although only 5 mm in its minor diameter) is 15 mm in its major diameter suggests strongly to me that it will admit a larger endotracheal tube than one with an external diameter of 5 mm. The trachea is a dynamic and nonrigid structure, and anyone who has observed tracheal dilation will confirm that it will often accept a larger diameter tube than its resting dimensions. Second, the fact that the patient suffers no limitation to his daily activities indicates that gas flow is considerably better than some of the patient's features suggest.

Why Do I Believe This Patient Needs Intubation? I did consider use of the *PLMA*™ as the primary airway throughout anesthesia. The *PLMA*™ is my "go to" airway and I have extensive experience with it for both routine[15] and difficult airway management.[16] Such is my confidence in it that I have abandoned use of the *LMA-Classic*™ (Laryngeal Mask Company), because I believe that its performance and safety profile is inferior to that of the *PLMA*™. Despite this, I rejected its use for this case because I was concerned that if it failed with the patient in the prone position, rescue would be hazardous and might fail with fatal consequences.

I did not consider the option of use of *PLMA*™ with an airway exchange catheter in place through the vocal cords in case rescue was required. This is ingenious and adds a level of safety. Despite this, if I was required to anesthetize this patient I would still advocate tracheal intubation before commencing a 4-h procedure in the prone position. I have used the *PLMA*™ myself in approximately 10 patients in the prone position and I am aware that it has been used in several series of patients in the prone position.[17,18] However, the patients enrolled in these studies were at low risk and the largest series is 245 patients[17]: airway obstruction occurred in three patients (>1%). In contrast, the patient in this case has an increased risk of problems with both ventilation and oxygenation. Minor degrees of misplacement and airway swelling during surgery in the prone position would likely lead to airway obstruction. If airway obstruction occurred in this patient, hypoxia would likely be rapid and profound. His airway is likely difficult to manage in the supine position in ideal circumstances. Despite my experience, I would be concerned that problems occurring in the prone position, mid-surgery would be so difficult to manage that the patient's life would be at risk. Despite the difficulties posed by intubation (and extubation), I would chose tracheal intubation in this case.

Why Do I Induce with Incremental Target-controlled Infusion Propofol? Most anesthesiologists, performing spontaneous breathing induction of anesthesia, reach for a volatile agent. With the experience of both, I have a strong preference for use of a slowly increasing, incremental dose of target-controlled propofol, while maintaining spontaneous ventilation. There are only limited descriptions of this technique.[19,20] The main advantage of incremental target-controlled infusion of propofol over rapid intravenous induction is that spontaneous ventilation is maintained. The technique also has advantages over gaseous induction. First, low-dose propofol provides excellent anxiolysis that notably assists the progress of anesthesia. Second, increasing depth of anesthesia is independent of the patient's ventilation. This allows rate of increase of the depth of anesthesia to be titrated carefully, by the anesthetist (rather than dictated by the patient). It also means that if difficulty is encountered stopping the infusion immediately enables anesthesia to lighten, without requiring the patient to "blow off" anesthesia *via* an airway that was partly obstructed and has now worsened. Finally, airway reflexes are rapidly obtunded, so coughing increased secretions and the complications these lead to during gaseous induction are rare. In many cases, patients will tolerate gentle manual ventilation even when still responsive to verbal stimulus. This enables confirmation of ability to ventilate, or of increasing difficulty, and airway adjuncts (*e.g.*, Guedel airway) are tolerated considerably earlier and better than during gaseous induction. Importantly, the technique does demand scrupulous attention to technique to detect problems with the airway early.

Why Introduce the *PLMA*™ over a Bougie? The *PLMA*™ is a device with a pedigree in management of the difficult airway.[16,21] If there is one weakness in *PLMA*™ performance, it is that insertion can be more difficult than for other supraglottic airway devices: conventional insertion techniques enable a first-time insertion rate with the *PLMA*™ of 87%, (~5% lower than that for the *LMA-Classic*™).[21] There is ample evidence that the use of a bougie aids first pass success with the *PLMA*™, increasing success close to 100% without any increase in morbidity.[9,10] Therefore, in circumstances where I consider first time success to be strongly desirable, I insert the *PLMA*™ over a reusable Smith Portex gum elastic bougie: the type of bougie is important to minimize the risk of esophageal trauma.[15,21]

Why the Aintree Intubation Catheter? The AIC is perhaps the ideal tube to use to (a) intubate *via* a supraglottic airway and (b) intubate narrow airways.[22] It has the smallest external diameter (a little less than 7.0 mm) of any endotracheal tube that will fit over a standard size fiberscope and its ID (4.6 mm) means that it does not "rattle around" during use, making impingement on the glottis unusual. Its use *via* the LMA-Classic™ and *PLMA*™ in difficult airway management is reported in two series.[3,23] Once placed it enables oxygenation and then, depending on circumstances, placement of a larger endotracheal tube (railroaded over it), or a smaller one (by passing an airway exchange catheter through it, followed by the smaller tube).

What Are My Rescue Plans and Plans for Management of Problems at Extubation? The small minor diameter of the trachea raises the significant possibility of difficulties with

ventilation or intubation. If ventilation becomes impossible at any stage after induction or intubation with an AIC is not possible, my plan is "graceful withdrawal" before considering another plan. Both are designed before any "bridges have been burnt." Certainly I would avoid aggressive attempts at passing an endotracheal tube, risking tracheal edema.

In the event of loss of the airway, presumably with hypoxia, my emergency rescue technique will be high-pressure source ventilation using a Manujet (VBM Medizintechnik, Sulz, Germany): unlike other "injectors" the Manujet incorporates a pressure-reducing mechanism so that the anesthesiologist may deliver only the minimum driving pressure to ventilate the lungs (so reducing the risk, and extent, of any barotraumas).[24] Ventilation would be *via* the Ravussin cannula, placed before induction of anesthesia. Profound hypoxia and a peri-arrest situation is not an ideal time to place a cricothyroid cannula. Peterson examined almost 200 cases of difficult airway management which led to medicolegal claims[25]: 42% of cases ended with "Cannot Intubate Cannot Ventilate" situation and in two-thirds of these cases a surgical airway was obtained but was placed too late to avoid poor outcome. Needle cricothyroidotomy and high-pressure source ventilation performed in these circumstances were each associated with a high incidence of barotrauma. Although these cases are undoubtedly affected by outcome bias, the message is clear: waiting for Cannot Intubate Cannot Ventilate situation and a peri-arrest patient before intervention is a poor plan. The technique of insertion of a prophylactic cricothyroid cannula for difficult airway management has been previously described[26] and is a frequent component of my management of patients with airway obstruction. It is easily performed in the awake patient, is almost painless, and is well tolerated. Ideally, the catheter would be placed under fiberscopic control as this improves position and reduces complications[27] but in this case that may not be practical. It is remarkable how much more confident one feels when the rescue route is established before undertaking other difficult techniques that may fail. Should I need to use the cricothyroid cannula I would not anticipate great difficulty with inspiration but great care would be needed to ensure that full exhalation had occurred (perhaps over 5–10 s or longer) before commencing the next breath: placing a hand on the patient's chest and palpating complete chest fall is a useful technique. Failure to confirm complete expiration would rapidly lead to severe barotraumas.[28]

Knowledge Gap

For some questions in clinical anesthesia, there may be no correct answer or alternatively several appropriate answers. The airway experts chose different airway management strategies for this patient. Importantly, this does not support thoughtless airway management: through this case scenario, one of the notable findings is that we all fundamentally agreed that ventilation would be possible during anesthesia induction. Despite this, why did we each choose different airway management strategies and include in these plans backups in case of failure? Probably, this is either because preoperative assessments of airway size and collapsibility may be imperfect in predicting ease of ventilation during general anesthesia or because the airway management techniques and devices currently available for managing tracheal stenosis is also imperfect.

Critical Stenosis for Breathing and Mechanical Ventilation during General Anesthesia

Even in patients with severe tracheal stenosis, normal gas exchange is maintained by respiratory compensatory mechanisms. Therefore, the presence of hypercapnia in preoperative patients without other respiratory diseases strongly indicates the potential for failure of both spontaneous and mechanical ventilation during general anesthesia. In contrast, the ability to compensate breathing through a narrow airway is well marked during general anesthesia and sometimes better than that during wakefulness, because behavioral influences such as panic and anxiety are eliminated and oxygen consumption is reduced.[29] Nunn and Ezi-Ashi[29] found breathing through a tubular resistor of 4.5 mm ID and 2.5 cm in length or 3.0 mm ID and 2.5 cm in length reduced the mean minute ventilation by 7 and 21%, respectively, during general anesthesia, although responses were variable and unpredictable. The subsequent studies by Moote *et al.* (inspiratory resistive loads), Kochi and Nishino (inspiratory resistive loads), and Isono *et al.* (expiratory resistive loads) confirm that the critical fixed narrowing through which anesthetized patients can spontaneously breathe without an increase in $Paco_2$ is 4.0–4.5 mm ID.[30–32] Respiratory compensation for breathing through a tubular resistor is achieved by decreasing the respiratory rate, with prolongation of both inspiratory and expiratory times and the increase in respiratory drive. Although the patient's respiratory drive decreases or ceases during assisted and controlled ventilation, tolerable size of the stenosis for breathing may not be greatly affected by the ventilatory modes.[2] However, I found that no study has systematically examined the critical stenosis for mechanical ventilation in anesthetized and paralyzed patients and the speculation needs to be tested in the future.

Imaging Techniques for Assessment of Severity of Tracheal Stenosis

How can we assess structural and functional severity of tracheal stenosis? Patients with mild to moderate tracheal stenosis rarely have clinical symptoms such as dyspnea. Even patients with severe tracheal stenosis may present without stridor and dyspnea during quiet breathing and, therefore, clinical symptoms may not be good indices for severity of tracheal stenosis.[33] Various imaging techniques possibly assess the structural severity of the stenosis. Chest radiographs have limited clinical utility for determining the presence and the severity of an airway stenosis. Saggital CT images along the airway provide significant information regarding the severity, location, and shape of the airway narrowing and the

structures surrounding the airway.[34] Recently developed three-dimensional imaging techniques such as CT and magnetic resonance imaging can more accurately delineate complicated airway shapes and determine the minimal airway size.[35] Using a fiberoptic bronchoscope for measuring the cross-sectional area and the length, the mean cross-sectional area and the length of stenotic region of the trachea in patients who underwent trachea reconstruction surgery were reported to be 48.3 ± 31.9 mm^2 (8 mm in diameter) and 9.3 ± 3.3 mm, respectively, suggesting large structural variability among them.[36] Because of the structural variability and complexity of the stenotic airway determined by the imaging techniques, functional impact of the tracheal stenosis on breathing during general anesthesia is often difficult to predict even by the sophisticated imaging techniques.

Potential Usefulness of Spirometry for Predicting the Obstruction Site and Airway Collapsibility

Airway size is determined by the transluminal pressure across the airway wall and its stiffness. A collapsible extrathoracic airway narrows during inspiration and dilates during expiration because the transluminal pressure decreases during inspiration and increases during expiration. In contrast, the relationship between airway caliber and respiratory phase is opposite in the collapsible intrathoracic airway. Therefore, airflow limitation predominantly (but not exclusively) occurs in the extrathoracic airway during inspirations and in the intrathoracic airway during expiration.[37,38] This dynamic airway behavior is exaggerated during forced expiratory and inspiratory maneuvers particularly when collapsibility of the airway caliber is increased. In this context, old-fashioned pulmonary function tests may be clinically useful for predicting the site of airway occlusion and airway collapsibility.[39,40] Harrison[39] found that the mean PEF/PIF values during a cycle of forced expiratory and inspiratory maneuvers were greater in 12 patients with extrathoracic airway obstructions than those of normal subjects (2.26 ± 0.84 *vs.* 1.32 ± 0.26). Two patients with collapsible intrathoracic tracheas had PEF/PIF values less than 0.7, which increased to more than 1.0 after removal of the mediastinal tumor and insertion of tracheal stent. In two patients with posttracheostomy tracheal stenosis, both PEF and PIF were significantly decreased but PEF/PIF value did not differ from normal subjects. According to the flow-volume loop in figure 1, our patient had significant reduction in both PEF and PIF and a normal PEF/PIF ratio (1.33), suggesting significant central airway stenosis and arguably adding evidence for rigidity of the airway wall. Nevertheless, caution is required for interpretation of the results, because PIF can be influenced greatly by patient effort.

Shamberger *et al.*[40] demonstrated a direct association between the tracheal cross-sectional area measured by the CT scan and PEF values in children with anterior mediastinal tumors. Interestingly, they found significant but variable PEF reduction (2–42% reduction) in the supine position compared with the sitting position and significant PEF improvement in all children after therapeutic mass reduction. Azizkhan *et al.*[41] reported complete airway obstruction after induction of anesthesia in children with greater than 50% tracheal narrowing. As these authors suggested, the patient with a highly collapsible trachea (suggested by lower PEF and narrower tracheal cross-sectional area) should not have general anesthesia induced before securing the airway. In summary, spirometric variables may be useful for functionally

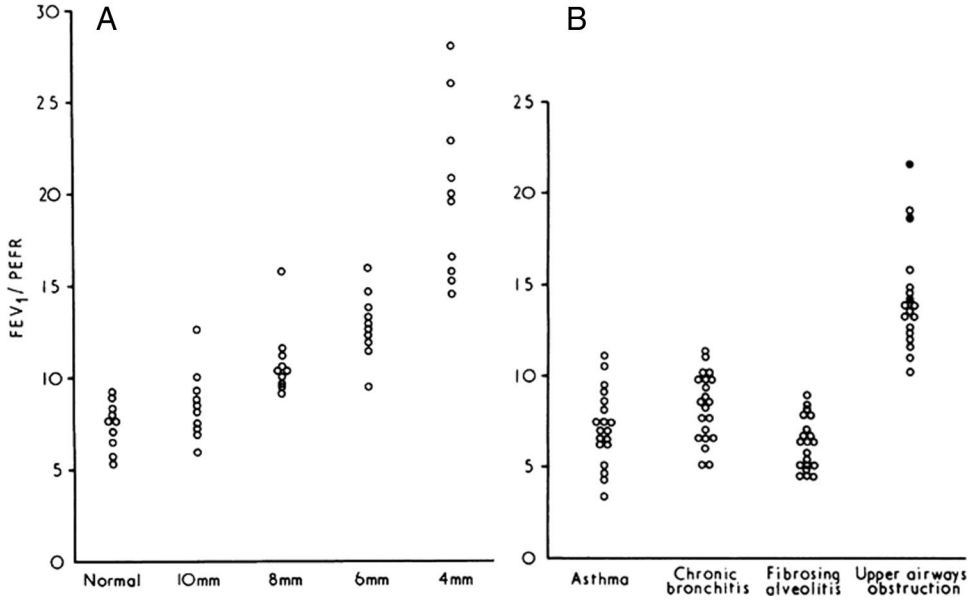

Fig. 4. (*A*) Forced expiratory volume in the first second/peak expiratory flow (FEV_1/PEF) in normal subjects with and without resistances added at mouthpieces. Diameter of orifice is shown in each case. (*B*) FEV_1/PEF in patients with lower airways obstruction, interstitial lung disease, and upper airways obstruction. Black circles indicate cases requiring tracheostomy. PEFR = peak expiratory flow rate. Reproduced and modified from the BMJ, Empey DW, volume 3, pages 503–5, 1972, with permission from BMJ Publishing Group Ltd.[33]

characterizing tracheal collapsibility and for detecting the risk of critical airway collapse after induction of anesthesia, but further investigations are necessary.

Spirometry for Predicting Functional Airway Size for Breathing during General Anesthesia

When the airway is rigid, the severity of airflow limitation is determined by airway size. Among the spirometric variables, Empey[33] clearly demonstrated a significant association between FEV_1/PEF and the cross-sectional area of the central airway stenosis, and therefore FEV_1/PEF might be used as a clinically useful parameter for predicting functional airway size without sophisticated imaging analysis. Because of the complexity of the contribution of stenotic length and airway shape to total airway resistance, FEV_1/PEF may have advantages over imaging information as an index for total upper airway resistance. Empey found that FEV_1/PEF ($[ml \cdot s^{-1}] \cdot [l \cdot min^{-1}]^{-1}$) was greater than 10 in all patients with upper airway obstruction: equivalent to a normal subject breathing through an external resistance of less than 6 mm diameter (fig. 4). Breathing through a 4-mm orifice, possibly resulting in impossible mechanical ventilation during general anesthesia, increased the FEV_1/PEF to more than 15. In our patient with 3 cm tracheal stenosis with minimal cross-sectional area of 59 mm^2, FEV_1/PEF is calculated as $1950/180 = 10.8$. The FEV_1/PEF is much higher than that in normal subjects and corresponds to breathing through a, at least, 6 to 8 mm orifice. Therefore, this assessment suggests possible breathing or mechanical ventilation during general anesthesia, assuming noncollapsible airway characteristics. This functional assessment of the tracheal stenosis can give anesthesiologists significant information for determining perioperative airway management strategies (fig. 1). An FEV_1/PEF of more than 15 (equivalent to 4 mm orifice breathing) may indicate a potential inability of mechanical ventilation after induction of general anesthesia, although this needs to be validated and other approaches should be considered in future studies.

In conclusion, we discussed anesthetic management of a patient with tracheal stenosis and found significantly different airway management strategies among us. Lack of reliable and accurate prediction of airway patency and breathing during general anesthesia in the patient with tracheal stenosis seems to be the fundamental reason for the variable airway management strategies. For patients with difficult airways, our progress in preoperative airway assessment as a predictive tool is rather limited, when compared with the outstanding developments in airway management techniques and devices.

Dr. Cook acknowledges Dominic Hurford, M.B.Ch.B., F.R.C.A., Special Registrar, Royal United Hospital, Combe Park, Bath, United Kingdom, and Andy Giorgiou, B.Sc., M.B.Ch.B., F.R.C.A., Special Registrar, Royal United Hospital, with whom informal discussion of the case assisted in clarification of concepts and details. Dr. Isono thanks Toshihito Sai, M.D., Head of the Department of Anesthesiology, Naruto General Hospital, Sanmu, Japan, for his assistance with preparation of this manuscript.

References

1. Nouraei SA, Ma E, Patel A, Howard DJ, Sandhu GS: Estimating the population incidence of adult post-intubation laryngotracheal stenosis. Clin Otolaryngol 2007; 32:411–2
2. Nouraei SA, Giussani DA, Howard DJ, Sandhu GS, Ferguson C, Patel A: Physiological comparison of spontaneous and positive-pressure ventilation in laryngotracheal stenosis. Br J Anaesth 2008; 101:419–23
3. Cook TM, Seller C, Gupta K, Thornton M, O'Sullivan E: Non-conventional uses of the Aintree Intubating Catheter in management of the difficult airway. Anaesthesia 2007; 62:169–74
4. Asai T, Shingu K: Airway management of a patient with tracheal stenosis for surgery in the prone position. Can J Anaesth 2004; 51:733–6
5. Asai T: Emergency cardiopulmonary bypass in a patient with a mediastinal mass. Anaesthesia 2007; 62:859–60
6. Smith IJ, Sidebotham DA, McGeorge AD, Dorman EB, Wilsher ML, Kolbe J: Use of extracorporeal membrane oxygenation during resection of tracheal papillomatosis. ANESTHESIOLOGY 2009; 110:427–9
7. American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: An updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. ANESTHESIOLOGY 2003; 98:1269–77
8. McCaughey W, Bhanumurthy S: Laryngeal mask placement in the prone position. Anaesthesia 1993; 48:1104–5
9. Howath A, Brimacombe J, Keller C: Gum-elastic bougie-guided insertion of the ProSeal laryngeal mask airway: A new technique. Anaesth Intensive Care 2002; 30:624–7
10. Brimacombe J, Keller C, Judd DV: Gum elastic bougie-guided insertion of the ProSeal laryngeal mask airway is superior to the digital and introducer tool techniques. ANESTHESIOLOGY 2004; 100:25–9
11. Gyulay S, Olson LG, Hensley MJ, King MT, Allen KM, Saunders NA: A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. Am Rev Respir Dis 1993; 147:50–3
12. Isono S: Obstructive sleep apnea of obese adults: Pathophysiology and perioperative airway management. ANESTHESIOLOGY 2009; 110:908–21
13. Viswanathan S, Campbell CE, Cork RC: Asymptomatic undetected mediastinal mass: A death during ambulatory anesthesia. J Clin Anesth 1995; 7:151–5
14. Kheterpal S, Martin L, Shanks AM, Tremper KK: Prediction and outcomes of impossible mask ventilation: A review of 50,000 anesthetics. ANESTHESIOLOGY 2009; 110:891–7
15. Cook TM, Gibbison B: Analysis of 1000 consecutive uses of the *ProSeal Laryngeal Mask Airway*™ by one anaesthetist at a district general hospital. Br J Anaesth 2007; 99:436–9
16. Cook TM, Silsby J, Simpson TP: Airway rescue in acute upper airway obstruction using a *ProSeal™ Laryngeal Mask Airway* and an Aintree Catheter™: A review of the *ProSeal™ Laryngeal Mask Airway* in the management of the difficult airway. Anaesthesia 2005; 60:1129–36
17. Brimacombe JR, Wenzel V, Keller C: The proseal laryngeal mask airway in prone patients: A retrospective audit of 245 patients. Anaesth Intensive Care 2007; 35:222–5
18. García-Aguado R, Tornero F, Otero M, Sanchís R: On inserting the ProSeal laryngeal mask in prone position. Rev Esp Anestesiol Reanim 2008; 55:320–1
19. Ludbrook GL, Hitchcock M, Upton RN: The difficult airway: Propofol infusion as an alternative to gaseous induction. Anaesth Intensive Care 1997; 25:71–3
20. Hamard F, Ferrandiere M, Sauvagnac X, Mangin JC, Fusciardi J, Mercier C, Laffon M: Propofol sedation allows awake intubation of the difficult airway with the Fastrach LMA. Can J Anaesth 2005; 52:421–7
21. Cook TM, Lee G, Nolan JP: The *ProSeal™ Laryngeal Mask Airway*: a review of the literature. Can J Anaesth 2005; 52:739–60

22. Hawkins M, O'Sullivan E, Charters P: Fibreoptic intubation using the cuffed oropharyngeal airway and Aintree intubation catheter. Anaesthesia 1998; 53:891–4
23. Higgs A, Clark E, Premraj K: Low-skill fibreoptic intubation: Use of the Aintree Catheter with the classic LMA. Anaesthesia 2005; 60:915–20
24. Cook TM, Nolan JP, Magee PT, Cranshaw JH: Needle cricothyroidotomy. Anaesthesia 2007; 62:289–90
25. Peterson GN, Domino KB, Caplan RA, Posner KL, Lee LA, Cheney FW: Management of the difficult airway: A closed claims analysis. ANESTHESIOLOGY 2005; 103:33–9
26. Gerig HJ, Schnider T, Heidegger T: Prophylactic percutaneous transtracheal catheterisation in the management of patients with anticipated difficult airways: A case series. Anaesthesia 2005; 60:801–5
27. Gerig HJ, Heidegger T, Ulrich B, Grossenbacher R, Kreienbuehl G: Fiberoptically-guided insertion of transtracheal catheters. Anesth Analg 2001; 93:663–6
28. Meissner K, Iber T, Roesner JP, Mutz C, Wagner HE, Layher C, Bartels U, Gründling M, Usichenko TI, Wendt M, Lehmann C, Pavlovic D: Successful transtracheal lung ventilation using a manual respiration valve: An in vitro and in vivo study. ANESTHESIOLOGY 2008; 109:251–9
29. Nunn JF, Ezi-Ashi TI: The respiratory effects of resistance to breathing in anesthetized man. ANESTHESIOLOGY 1961; 22:174–85
30. Moote CA, Knill RL, Clement J: Ventilatory compensation for continuous inspiratory resistive and elastic loads during halothane anesthesia in humans. ANESTHESIOLOGY 1986; 64:582–9
31. Kochi T, Nishino T: Ventilatory responses to inspiratory resistive loading before and after gastrectomy during isoflurane anesthesia. Anesth Analg 1995; 80:1199–205
32. Isono S, Nishino T, Sugimori K, Mizuguchi T: Respiratory effects of expiratory flow-resistive loading in conscious and anesthetized humans. Anesth Analg 1990; 70:594–9
33. Empey DW: Assessment of upper airways obstruction. BMJ 1972; 3:503–5
34. Pinsonneault C, Fortier J, Donati F: Tracheal resection and reconstruction. Can J Anaesth 1999; 46:439–55
35. Toyota K, Uchida H, Ozasa H, Notooka A, Sakura S, Saito Y: Preoperative airway evaluation using multi-slice three-dimensional computed tomography for a patient with severe tracheal stenosis. Br J Anaesth 2004; 93:865–7
36. Nouraei SA, McPartlin DW, Nouraei SM, Patel A, Ferguson C, Howard DJ, Sandhu GS: Objective sizing of upper airway stenosis: A quantitative endoscopic approach. Laryngoscope 2006; 116:12–7
37. Hyatt RE, Schilder DP, Fry DL: Relationship between maximum expiratory flow and degree of lung inflation. J Appl Physiol 1958; 13:331–6
38. Miller RD, Hyatt RE: Evaluation of obstructing lesions of the trachea and larynx by flow-volume loops. Am Rev Respir Dis 1973; 108:475–81
39. Harrison BD: Upper airway obstruction—A report on sixteen patients. Q J Med 1976; 45:625–45
40. Shamberger RC, Holzman RS, Griscom NT, Tarbell NJ, Weinstein HJ, Wohl ME: Prospective evaluation by computed tomography and pulmonary function tests of children with mediastinal masses. Surgery 1995; 118:468–71
41. Azizkhan RG, Dudgeon DL, Buck JR, Colombani PM, Yaster M, Nichols D, Civin C, Kramer SS, Haller JA Jr: Life-threatening airway obstruction as a complication to the management of mediastinal masses in children. J Pediatr Surg 1985; 20:816–22