

Propofol Dependency in a Lay Person

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PROPOFOL is a short-acting, intravenous agent used extensively in anesthesia and intensive care medicine to provide dose-dependent sedation and hypnosis. It is characterized by a short onset, a short duration of action, low toxicity, ability to control sedation, and ease of administration. Thus, it quickly found general acceptance after its introduction into the market in the mid 1980s. The abuse potential of propofol has not completely been defined, but there are anecdotal case reports in the literature about abuse of and dependency on propofol.¹⁻⁴ We report a case of a 25-yr-old man with attention deficit hyperactive disorder (ADHD) who experienced propofol dependency and, under remarkable circumstances, succeeded in providing himself with this drug on a regular basis.

Case Report

An otherwise healthy, 25-yr-old man presented to the neurology and psychiatry clinic because he was thought to have ADHD. Urged by his wife, he also reported regular usage of a white milky substance called propofol. At the age of 8 yr, he was given methylphenhydrate (ritalin) for 1 yr because of his hyperactive behavior and attention deficits at school. After his parents stopped his ritalin administration for fear of long-term side effects, his symptoms of hyperactivity worsened, but he managed to graduate from high school, and studied to be an accountant in his father's company. At the age of 21 yr, he started to experience tension headaches, which were treated with propofol injections by an anesthesiologist. He had several appointments with this anesthesiologist for propofol treatment. From this time on, he started to inject himself with this drug. Before, he had occasionally self-administered benzodiazepines and morphine, and sometimes, he consumed marijuana. He obtained prescriptions for propofol from various veterinarians whom he told that he was a tropical fish enthusiast and he needed propofol to anesthetize his fish. He had three or four "propofol sessions" per week, lasting 1-2 h. He would inject 5 ml propofol, 1%, in an antecubital vein, fall into a deep, relaxing sleep for approximately 5-10 min, wake up, and inject another 5 ml of the drug, using up to 60-100 ml propofol, 1%, which he had drawn up in 20-ml syringes, per "session." After he accidentally injected an overdose of this drug, he was found by his wife, unconscious and cyanotic. After a stay of several days in a hospital for clinical observation, he was convinced to attend an in-hospital drug rehabilitation program. There, he did not show

any withdrawal symptoms, but after 7 days, he refused to participate in therapy anymore and left the hospital.

Discussion

Because of its pharmacokinetic and pharmacodynamic features (ultrashort action, potent respiratory depressant), propofol seems not to be the substance of choice for drug abuse. However, reports about sexual disinhibition, reports about pleasant and euphoric feelings during recovery from propofol anesthesia and sedation since the beginning of its use in clinical practice, and several case reports of propofol abuse, the first one in 1992,¹ shed some light on the abuse potential of this drug. Three cases were reported of young men with some connection to a hospital, such as being employed as theater technicians, who were stopped by the police for dangerous driving and were found to be injecting themselves with propofol while driving.² An anesthesiologist experienced an uncontrollable compulsion toward this drug such that he could not abstain from injecting propofol despite the fact that he already had revealed his drug problem to his psychiatrist and the head of his department.³ He reported his previous benzodiazepine and opioid (fentanyl) use rather than admitting his regular propofol injections. In 1997, a case of propofol abuse was published about a general practitioner who injected the substance repeatedly up to more than 100 times a day because the drug effect lasted not more than 5-10 min.⁴ After injections, mild euphoria and a feeling of relaxation were followed by heavy sedation and loss of consciousness. An intense craving made it impossible to stop taking the drug. Like the anesthesiologist in the earlier case report, he relapsed twice despite psychiatric help. Thus, both individuals fulfilled the criteria of substance dependence, which are as follows: compulsion or craving; loss of control over the amount of the frequency of the drug used; and continued use of the substance despite adverse physical, psychological, social, or occupational consequences.⁵ Remarkably, none of these individuals experienced signs of withdrawal.

The patient described herein came in contact with propofol when receiving treatment for his tension headaches. Propofol in subanesthetic doses was novel, but was used increasingly to treat migraine and other headaches.⁶

Recently, a clinical study in healthy volunteers assessed the rewarding effects and thus abuse potential of this drug.⁷ The authors of this study concluded that use of propofol may be rewarding (reinforcing) in some people without history of drug abuse and asked for further abuse liability testing.

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Why more of these millions of patients who were exposed to propofol do not develop dependency remains a matter of speculation. First, it could be hypothesized that the majority of patients anesthetized with this drug are unaware of which drug was used for this purpose. Thus, a major prerequisite for addiction is missing: knowing what to look for. Second, this case report, as well as the others, suggests that some psychiatric pathology or previous drug abuse was involved. The first reported propofol abuser had alcoholism¹; the second was a stressed anesthesiologist³ who had experimented before with other drugs, such as marijuana, cocaine, and other chemicals; the third was a general practitioner² who had depression; and the current patient met the criteria for ADHD and had some other drug abuse experience as well.

Patients with ADHD are said to have an increased risk for development of psychoactive substance use disorders.⁸ However, even assuming that this patient had ADHD, he is not necessarily prone to dependency on such an "exotic" drug as propofol.

In conjunction with the aforementioned case reports, this case is highly troublesome for two reasons¹: for more than 15 yr, this drug has been widely used in clinical practice, and because of its unique pharmacologic qualities and its excellent safety record, it was administered to millions of patients. Now, there is evidence emerging that this drug might have addictive properties that we did not consider.² The complete lack of withdrawal phenomena might prevent a potential victim from recognizing this addiction, and might prevent the careworkers involved from understanding and

interpreting the problem as addiction, because with other substances the presence of withdrawal symptoms serves as a warning signal and increases readiness to intervene.

In summary, this is the first reported case of a layperson who became highly dependent on propofol. Because of its sole use in anesthesia and intensive care, access to this drug is restricted, and knowledge of what this agent is used for, in any sense, is limited to anesthesiologists, intensivists, and, unfortunately, to persons who detect its unforeseen effects by chance. The potential danger is highlighted, and we believe it is time for thorough abuse liability testing.

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Air Embolism during Radical Cystectomy with Ileal Conduit Urinary Diversion

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VENOUS air embolism (VAE) has been reported in a wide variety of procedures. In most cases, signs of clinically significant VAE are present soon after the entry of air into the circulation. We present a case of urologic surgery in which no signs of VAE were recognized during surgery, but circulatory collapse occurred when the patient was transferred from the operating room bed to

the transport stretcher. The presumptive diagnosis of VAE as the cause of circulatory collapse was made on aspiration of frothy blood from the central venous pressure (CVP) catheter and was confirmed with the immediate placement of a transesophageal echocardiography (TEE) probe.

Case Report

A 72-yr-old man was diagnosed with high-grade transitional cell carcinoma of the bladder after a radical prostatectomy in 1993 for prostate carcinoma. He was scheduled to undergo radical cystectomy and formation of ileal conduits. The patient had a history of hypertension but was not taking medication. He had no history of coronary artery disease. Although he had a 22-pack/year history of smoking, he had ceased smoking 28 yr previously. He had undergone an uneventful right inguinal herniorrhaphy 9 months previously.

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Intravenous, arterial, and lumbar epidural catheters were placed preoperatively. Standard monitoring was used, including continuous end-tidal carbon dioxide monitoring and an esophageal stethoscope. A CVP catheter was inserted into the right internal jugular vein after induction of general anesthesia. The patient was positioned supine in the low lithotomy position with a slight Trendelenburg tilt.

Anesthesia was maintained with sevoflurane and nitrous oxide in 42% oxygen, and continuous infusion of 0.0625% bupivacaine with 40 µg/ml morphine into the epidural catheter. Vecuronium was used for muscle relaxation, and the lungs were mechanically ventilated. Surgical dissection was tedious and difficult because of dense adhesions from previous extensive surgery. The total estimated blood loss was 3.2 l, with a brief period of brisk blood loss associated with inadvertent division of the right external iliac vein at 2.5–3 h after the start of surgery. During this time, there were no notable changes in end tidal carbon dioxide, CVP, cardiac rhythm, or heart sounds, and a decrease in blood pressure was minimized by administration of blood and crystalloid. Peak airway pressures were constant throughout the procedure at 25 cm H₂O. Total fluid replacement was 6.7 l, which included 4 units of erythrocytes, resulting in a hematocrit of 37% and a CVP of 9 mmHg at the end of surgery. The duration of surgery was 7 h.

At the conclusion of surgery, the patient was responsive to commands and breathing spontaneously through the endotracheal tube. Blood pressure was stable at 100/60 (mean 75) mmHg, and heart rate was 62 beats/min. The monitors were temporarily disconnected to move the patient to a stretcher for transfer to the postanesthesia care unit. After the patient was moved, he was noted to be unresponsive and pulseless. The monitors were reconnected. Blood pressure was not recordable, and electrocardiogram rhythm was documented as showing ventricular fibrillation.

Cardiopulmonary resuscitation was commenced immediately with 100% oxygen and manual ventilation *via* the endotracheal tube. Circulatory support was achieved with chest compressions and epinephrine. Although sinus rhythm was restored within 4 min, ongoing treatment with epinephrine, bicarbonate, and calcium chloride was required.

The differential diagnosis included VAE, pulmonary embolism, a primary cardiac event, tension pneumothorax, respiratory insufficiency, or intraabdominal hemorrhage. Breath sounds were audible bilaterally. The electrocardiogram monitor showed sinus tachycardia and did not suggest acute myocardial infarction. Aspiration of the CVP catheter within the first few minutes of resuscitation yielded 10–20 ml frothy blood. A working diagnosis of VAE was made. A TEE probe inserted 15 min after resuscitation revealed a stream of bubbles in all four chambers of the heart. Repeated attempts to aspirate additional air from the CVP catheter were unsuccessful.

Heart rate, rhythm, and blood pressure normalized 25 min after resuscitation began, and epinephrine was discontinued. Continued TEE monitoring showed gradual resolution of the bubble stream over the same interval. Chest radiograph and fiberoptic bronchoscopy performed during the stabilization period were unremarkable. The patient was transferred to the intensive care unit 45 min after onset of cardiovascular collapse.

The patient required interim ventilatory and inotropic support for adult respiratory distress syndrome. A primary cardiac event was ruled out with further evaluation. A computed tomograph of the head on the second postoperative day revealed no abnormality, and there was no clinical evidence of neurologic deficit after sedation was discontinued. The patient improved and was discharged home 10 days after the event.

Discussion

Venous air embolism has been documented in association with several urologic procedures. These include

transvesical,¹ transurethral,^{2,3} radical retropubic,⁴ and radical perineal⁵ prostatectomies. Venous air embolism has also occurred during retrograde pyelography,⁶ the use of lasers for urethral surgery,⁷ and percutaneous ultrasonic lithotripsy.⁸ We report a patient undergoing radical cystectomy with ileal conduit urinary diversion urologic surgery in which no signs of VAE were recognized during surgery, but circulatory collapse occurred when the patient was transferred from the operating room bed to the transport stretcher.

Venous air embolism occurs when air enters the right side of the heart by entrainment from negative intrathoracic venous pressure or by being pushed into an open venous channel by positive pressure. The patient was positioned supine in the low lithotomy position with slight Trendelenburg tilt. Extensive previous surgery caused dense adhesions and difficulty for the surgeon in identifying normal tissue planes and in performing the required dissection for the current procedure. The total blood loss for the procedure was approximately 3 l. Most of this occurred during the first 4 h of the procedure. This correlates temporally with the dissection. The patient's position, volume status, and open pelvic veins were favorable conditions for air to enter the venous system, which most likely occurred at 2.5–3 h after the start of surgery. The use of nitrous oxide may have contributed to the size of the bubbles that accumulated in the pelvic veins.

The mechanism of air entrainment into the right side of the heart manifested by circulatory collapse of this patient occurred at 4.5 h subsequent to the presumed time of entry of air into the venous system. We propose that accumulated air in the pelvic venous system may have been entrained into the right side of the heart during spontaneous ventilation when the patient was taken out of the lithotomy position for transfer to the stretcher. The possible formation of an air lock in the right side of the heart may have resulted in obstruction to cardiac output. A similarly delayed, but fatal presentation of air embolism has been described during transurethral resection of the prostate under spinal anesthetic.³ In that case, although the mechanism for the presence of intravenous air was different, the patient's cardiac arrest occurred only after his legs were lowered from the lithotomy position at the completion of the surgical procedure. A report of fatal air embolism during radical retropubic prostatectomy⁴ also describes the onset of hemodynamic instability consistent with VAE after the pelvic dissection had been completed and during the relatively quiescent period of bladder neck reconstruction.

The presence of air in all four chambers of the heart as seen with TEE in this patient is consistent with transpulmonary passage of air emboli. The differential diagnosis includes patent foramen ovale, which clearly cannot be ruled out during resuscitation. The continuing presence of air emboli in the left-sided cardiac chambers from

transpulmonary passage has been demonstrated to persist for 15 min after the cessation of venous air entrainment and clearing of the right atrium and ventricle of any air in the absence of intracardiac shunt.^{9,10} TEE is the most sensitive method for detection of intracardiac air and can detect bubbles of 5-10 μm in diameter. However, it is not possible to quantify the amount of air seen, and the clinical significance of such small bubbles is not known.¹⁰

In the current case, a computed tomograph of the head obtained on the second postoperative day did not reveal any intracranial air. Had air been present, treatment with hyperbaric oxygen could have been considered. However, it is unlikely that such treatment begun on the second postoperative day would improve the quality of neurologic outcome.

It is a concern that the risk of VAE is present in urologic surgery when a gravitational gradient is introduced with lithotomy positions and a degree of Trendelenburg tilt. Procedures requiring these positions include transvesical, transurethral, radical retropubic, and radical perineal prostatectomies and radical cystectomy. All these procedures are also associated with blood loss from vascular surgical fields, resulting in an increased risk for VAE. Anesthesiologists should be aware of the

range in temporal association of surgical events and the manifestation of signs of VAE and should monitor patients accordingly. In particular, monitoring should continue during repositioning of the patient at the end of surgery and should only be briefly interrupted, if at all, during transfer of the patient from the operating table to the transport stretcher.

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Brain Tumor Presenting with Fatal Herniation following Delivery under Epidural Anesthesia

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LUMBAR puncture is usually contraindicated in patients with increased intracranial pressure or space-occupying lesions because lumbar cerebrospinal fluid (CSF) drainage can increase the pressure gradient between the supratentorial and infratentorial compartments and thus result in rapid herniation.^{1,2} Some authors have also suggested that epidural anesthesia is dangerous in patients with space-occupying lesions not only because of the risks associated with accidental dural puncture, but also because epidural drug and fluid injection can increase intracranial pressure.^{3,4}

We present a case of a pregnant woman with an occult brain tumor who received an epidural anesthetic for relief of labor pain, which was complicated by an inadvertent lumbar puncture. Several hours after delivery, she had fatal transtentorial brain herniation.

Case Report

A healthy, 35-yr-old woman (primigravida) at 38 weeks' gestation was admitted to the labor ward of a local obstetric hospital in spontaneous labor. The course of pregnancy had been uneventful. At the time of admission, she was afebrile, her pulse rate was 86 beats/min, and her blood pressure was 136/104 mmHg. Laboratory studies were normal.

Epidural analgesia was initiated during the first stage of labor. An initial attempt was made with an 18-gauge Tuohy needle at the L3-L4 interspace, with the patient sitting. However, an accidental dural puncture occurred; 3 ml air being used for "loss-of-resistance" testing was injected into the intrathecal space. She reported no symptoms. A second attempt was performed at the L2-L3 interspace, and an epidural catheter was successfully introduced into the epidural space. After an uneventful test dose, 12 ml bupivacaine, 0.25%, was injected over 20-30 s. This injection was accompanied by a transient headache. Analgesia was satisfactory. Two further injections with same volume and concentration of bupivacaine were administered during the first stage of labor.

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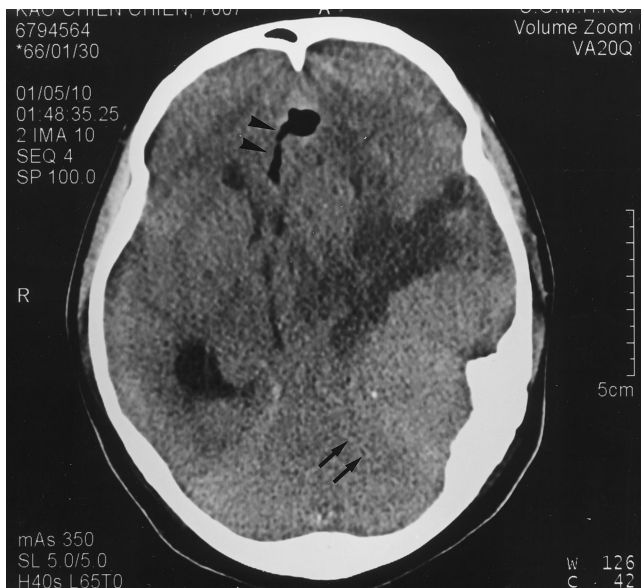


Fig. 1. Computed tomography revealed a slightly hyperdense tumor (arrows) with marked perifocal edema in left hemisphere and pneumocephalus (arrowheads). Midline shift to right side and tentorial herniation were also noted.

During labor, mean arterial pressure varied from 140 to 160 mmHg. The process of labor was uneventful, except that the patient reported occasional severe headache during labor, particularly during the second stage. Estimated blood loss was approximately 150 ml. Overall, the first stage of labor lasted approximately 8 h, and the second stage lasted approximately 20 min. Delivery was uneventful.

After delivery, she was sent to the ward for routine observation. However, 2 h after delivery, a marked decrease in her level of consciousness was noted. Her pupils were equal and reacted normally to light, and there were no focal neurologic deficits. Blood pressure was 156/112 mmHg; heart rate was 102 beats/min. No evidence of postpartum hemorrhage was noted. Laboratory studies were normal. However, 1 h later, left pupillary dilatation was noted. Her trachea was emergently intubated, and she was transferred to our hospital, arriving in the emergency room approximately 4 h after delivery.

At the time of arrival, blood pressure was 74/35 mmHg, and heart rate was 108 beats/min. Neurologic examination revealed deep coma (Glasgow Coma Score = 3). Her pupils were fixed and dilated bilaterally. Brain stem reflexes were absent. Hemodynamic resuscitation was accomplished with intravenous saline and a dopamine infusion. Computed tomography revealed a slightly hyperdense tumor in the left hemisphere with marked perifocal edema (fig. 1). There was a left-to-right midline shift of forebrain structures; tentorial herniation was also noted. A small amount of intracranial air was noted.

Because of her poor neurologic and hemodynamic status, her family declined emergent craniotomy. She was moved to the neurosurgical intensive care unit for further care and died 10 days later. An autopsy was not performed.

Discussion

The young woman described in this report had a tragic, fatal complication of her pregnancy. Although it is possible that her death may have been due solely to the combination of labor, delivery, and her tumor, we believe that her anesthetic may have contributed to this outcome. First, she had a large but unknown brain tu-

mor. The only possible symptom attributable to this lesion was headache at the time of her initial epidural injection, and recurrent headaches during labor. Her hypertension may have been in response to increased intracranial pressure or, even if unrelated, may have led to worsened intracranial hypertension and cerebral edema. Episodic increases in venous pressure, due to Valsalva maneuvers, could have been another contributing factor. The CSF leak induced by the inadvertent lumbar puncture then resulted in an increased transtentorial pressure gradient, which resulted in herniation.

The role of the epidural drug injection in her outcome is less clear. Epidural fluid injection can increase intracranial pressure, probably because of compression of the lumbar thecal sac with translocation of CSF into the intracranial space.^{3,4} However, in this case, such changes might have actually delayed the development of herniation. The increased epidural pressure produced by the injection may have slowed the loss of CSF. By increasing infratentorial pressure, the pressure gradient between the supratentorial and infratentorial space may also have been reduced. The 11-h interval between her accidental lumbar puncture and deterioration may have been the result of these temporarily “beneficial” changes. No additional epidural injections were performed after delivery. As fluid in the epidural space was absorbed, her CSF leak may have increased, leading to a worsened transtentorial pressure gradient and subsequent herniation.

How can we prevent this devastating complication? Certainly, epidural anesthesia would not be considered in patients with known mass lesions because of the risk of unintentional dural puncture. The difficulty is identifying patients with occult lesions. It is possible that a more carefully obtained history and physical examination might have been helpful. If there is an unexplained history of worsening headache, intractable vomiting, seizure, focal neurologic signs, altered consciousness, or papilledema, computed tomography should be performed. In this case, no symptoms were evident (other than headache). The delayed recognition of her changing neurologic status—and hence a delay in the institution of aggressive treatment (intubation, hyperventilation, osmotic diuretics, and others)—almost certainly contributed to the poor outcome. Even if preanesthetic diagnosis is not possible, closer postdelivery observation may be lifesaving.

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Use of a Modified Single-lumen Endobronchial Tube in Severe Tracheobronchial Compression

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A MEDIASTINAL mass causing compression of the tracheobronchial tree poses a difficult challenge for the anesthesiologist. Obstruction of the airway extending into the bronchi may require bronchial intubation or stenting. We describe the modification of a single-lumen endobronchial tube for airway management in a patient with bilateral bronchial compression due to Hodgkin's lymphoma.

Case Report

A 19-yr-old man presented to another hospital with a 3-week history of flu-like illness, nonproductive cough, increasing dyspnea, and pleuritic left-sided chest pain. There was no history of hemoptysis, night sweats, or fevers, and his medical history was otherwise unremarkable. Physical examination showed that he was cyanotic, tachypneic, and dyspneic at rest with prolonged expiration and accessory muscle use. Auscultation showed that chest breath sounds were absent on the left but normal on the right.

Blood chemistry and hematology were both within normal limits except for hypoxemia and mild respiratory alkalosis (partial pressure of oxygen $[P_{O_2}] = 53$ mmHg, arterial oxygen saturation $[Sa_{O_2}] = 86\%$, partial pressure of carbon dioxide $[P_{CO_2}] = 30$ mmHg, and pH = 7.48; fraction of inspired oxygen $[F_{I_{O_2}}] = 1$). A chest radiograph showed a large left pleural effusion with a large mass in the anterior and middle mediastinum abutting the heart and hilar structures. Subsequent computed tomographic examination showed a mass in the anterior mediastinum measuring $12.3 \times 5.6 \times 11$ cm, compressing the tracheobronchial tree posteriorly, with near complete occlusion of the left mainstem bronchus (fig. 1). In addition, there was severe compression of the right pulmonary artery.

Drainage of 1.8 l pleural fluid from the left pleural cavity provided little symptomatic relief. The patient was transferred to our institution for further treatment. At the time of arrival, the patient was immediately brought to the operating room for emergent intubation for management of severe respiratory failure and anterior mediastinoscopy for histologic diagnosis.

Anesthesia was induced with use of sevoflurane inhalation, with the patient sitting at a 45° angle. Assisted respiration was provided, and tracheal intubation was achieved with an 8.5-mm endotracheal tube positioned above the lesion, without use of neuromuscular blockade.

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Fiberoptic bronchoscopy revealed severe ($> 70\%$) compression of the lumen of the lower third of the trachea and right mainstem bronchus with near-complete occlusion of the left mainstem bronchus. Based on the bronchoscopic appearance of left bronchial obstruction and computed tomographic appearance of severe right pulmonary artery compression, a decision was made to intubate the left mainstem bronchus.

A size 8.0 left *single*-lumen endobronchial tube (EBT) with bronchial and tracheal cuffs (model No. 115900; R 252 üsch Inc., Duluth, GA), was modified by cutting a 5×8 -mm oval fenestration in the tube approximately 11 mm below the tracheal cuff (fig. 2A). The modified EBT was inserted over an 8-mm fiberoptic bronchoscope positioned past the obstruction in the left mainstem bronchus until the bronchial cuff lay completely inside the left mainstem bronchus and allowed clear ventilation of the distal, normal-appearing left upper and lower lobes. In this position, the newly cut hole lay at the opening of the right mainstem bronchus (fig. 2B), and the endobronchial portion stented the obstructed proximal left endobronchus. This allowed ventilation of the both lungs and subsequent bronchoscopic examination and toilet of both the right mainstem bronchus and the distal left mainstem bronchus. Uncomplicated left anterior mediastinoscopy and biopsy revealed a high-grade Hodgkin lymphoma.

The patient was kept intubated and was transferred to the intensive care unit. He was sedated, but not paralyzed, and was maintained on pressure-support ventilation. Inadvertent displacement of the modified tube on the first postoperative night during patient positioning resulted in acute desaturation and required urgent repositioning of the tube under bronchoscopic guidance. Combination chemotherapy and radiotherapy was commenced on the first postoperative day. Subsequently, the patient underwent fiberoptic bronchoscopy daily to observe the extent of left mainstem bronchial compression. Tumor shrinkage was noted over the next 3 days, and the patient underwent extubation on the fourth postoperative day.

Discussion

Complete or partial airway obstruction during anesthesia due to a mediastinal mass is a significant cause of morbidity and mortality.¹⁻³ Tracheal intubation is sometimes required to maintain a patent airway in the presence of worsening airway obstruction or during delivery of general anesthesia for surgical biopsy.⁴ If bilateral bronchial obstruction is present, placement of a double-lumen tube or stent may be required to maintain airway patency.⁵

The hardware options for stenting bronchial obstruction include double-lumen tubes, inverted-Y stents,⁶ covered stents,⁷ and single-lumen endobronchial tubes. Use of standard double-lumen tubes is a common practice for maintenance of bronchial patency in this clinical scenario.⁸ However, intubation can be prolonged, and because of the small caliber of double-lumen tubes, regular suctioning and bronchoscopic examination of the airway is difficult; a 39-French double-lumen tube has a narrowest

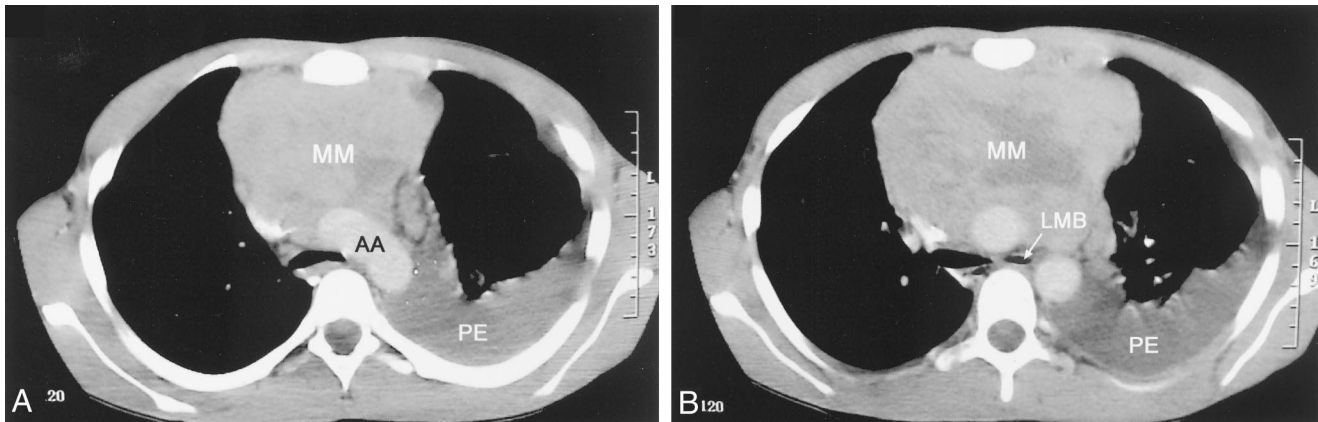


Fig. 1. (A) Chest computed tomography scan showing a large anterior mediastinal mass (MM) causing posterior displacement of the aortic arch (AA) and trachea. The trachea is severely compressed between the mass and the vertebral body. A pleural effusion (PE) is also visible. (B) Severe compression of the left mainstem bronchus (LMB) against the vertebral body is visible.

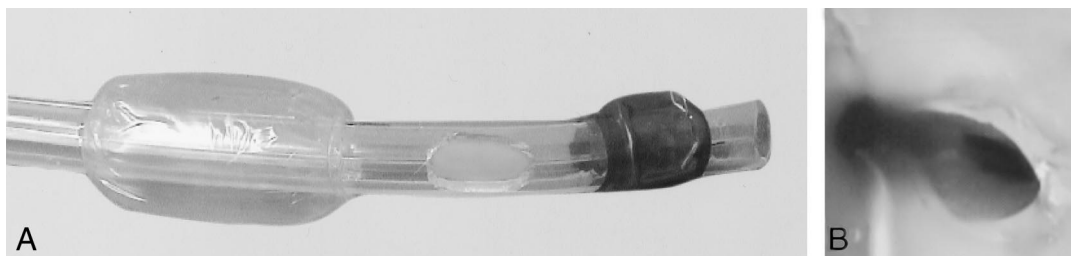


Fig. 2. (A) The modified single-lumen endobronchial tube in which an oval fenestration has been cut into the convex surface. (B) An internal view of the modified single-lumen endobronchial tube showing the partially compressed right mainstem bronchus visible through the newly cut fenestration in the convex surface. The radiographic line is visible posteriorly.

internal dimension of 5 mm. Stents are often unsuitable because inverted-Y stents have a large thickness-to-lumen ratio and covered stents are difficult to remove, particularly when temporary stenting only is required. Rarely, bilateral bronchial obstruction is treated by endobronchial placement of a conventional single-lumen tube, thereby necessarily sacrificing ventilation to the other lung with resultant shunt.

Despite high concentrations of inspired oxygen and adequate ventilation of his right lung, the patient had significant hypoxemia due to severe ventilation/perfusion mismatch. Computed tomographic examination showed severe compression of the right pulmonary artery. Therefore, we considered ventilation of both lungs to be necessary because continued isolated ventilation of the right lung may have proven fatal.

We chose to modify a newly available Rusch left-sided, single-lumen EBT to maximize internal diameter (8 mm) and provide left endobronchial intubation, while ventilating the right lung. These tubes differ from normal single-lumen tubes in that they are longer, have a curved end to facilitate endobronchial intubation, and have both a bronchial and a tracheal cuff. A fenestration was cut in the EBT, adjacent to the orifice of the right mainstem bronchus, thus allowing ventilation of the right lung. In doing this, the tube connecting the pilot balloon to the bronchial cuff was cut, rendering it unable to be inflated.

This did not change our management because this cuff was not used (cutting a similar hole in a right-sided EBT does not result in bronchial cuff deflation).

Currently, single-lumen EBTs are rarely used, occasionally providing lung isolation during one-lung anesthesia, such as for carinal pneumonectomy or bronchopleural fistula. Furthermore, EBTs are usually constructed from single-lumen endotracheal tubes by individual anesthesiologists. The Rusch tube and modification provides a new tool in our armamentarium for management of tracheobronchial airway obstruction.

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