Bilateral tension pneumothorax during jet ventilation: A case report

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Jet ventilation (JV) involves high-pressure ventilation for upper laryngeal laser procedures. Anesthetic management for the patient undergoing JV can be challenging, as complications of JV can include subcutaneous emphysema and tension pneumothorax.

A 52-year-old woman with a diagnosis of vocal cord polyps presented for direct microlaryngoscopy and laser laryngoplasty with JV. Intraoperatively, the patient developed lack of bilateral chest movement and an audible change in jet-ventilatory sounds. The patient was reintubated with a standard endotracheal tube. Subsequent attempts to ventilate the patient failed. A diagnosis of bilateral tension pneumothorax was made. Immediate pleural decompression resulted in improved ventilatory and hemodynamic status. The purpose of this case report is to discuss the pathophysiology related to tension pneumothorax and anesthetic implications for management of cases involving JV.

Key words: Bilateral tension pneumothorax, jet ventilation, laser surgery.

Introduction
Jet ventilation (JV) was first described in 1967 by Sanders. Since then, JV has been instrumental in upper laryngeal surgical procedures. The jet ventilator is an open system in which gases are projected through an elongated tube. A 12 French copper stylet, 8 to 16 inches long, is commonly used. The narrow diameter of the stylet accelerates the velocity of gases through the tube. This acceleration is an example of the Bernoulli effect, which states that gases flowing through a tube possess a constant kinetic energy. When the diameter of the tube is narrowed and the kinetic energy is held constant, the velocity of the gases will increase. Insufflation pressures are delivered at less than 50 psi at a rate of 10 to 12 breaths per minute, with each insufflation lasting 1 to 2 seconds.

The primary goal of anesthetic management is maintenance of adequate ventilation and oxygenation. Preoperative assessment should include a thorough airway evaluation for obstructive conditions involving the upper airway as well as chronic pulmonary and cardiac disease. Intraoperative complications related to tension pneumothorax can develop rapidly with ensuing hemodynamic collapse.

Case summary
A 52-year-old woman, 165 cm tall and weighing
100 kg, underwent direct microlaryngoscopy and laser laryngoplasty for vocal cord polyps. Her medical-surgical history included being a 2-pack-per-day smoker for 30 years, bronchitis, and cervical fusion. Preoperative treatment included 10 mg metoclopramide and 50 mg ranitidine intravenously.

After induction with 300 mg propofol and 100 mg succinylcholine, an 8-inch copper stylet was inserted under direct laryngoscopy and attached to the jet ventilator. The recorded insufflation pressures were in the range of 28 to 30 psi. Approximately 15 to 20 minutes after induction, the pulse oximetry waveform dampened. Readjustment of the probe established an oxygen saturation of 99%. The pulse oximetry waveform dampened again. Ventilatory sounds changed, and proper tube placement was questioned. Chest expansion was no longer observed. The copper tube was withdrawn and a 7.0-mm endotracheal tube (ETT) was inserted with continued attempts to ventilate the patient. Removal of the drapes revealed a cyanotic patient with significant subcutaneous emphysema of the face and neck. Two subsequent attempts to intubate the patient produced identical results: lack of chest movement with ventilation, absence of end-tidal CO₂, and lack of improvement in oxygen saturation. Pulse oximetry at this time showed an oxygen saturation of 30%. No arterial blood gases were available. A diagnosis of tension pneumothorax was made, and a 14-gauge angiocatheter was placed in the right second intercostal space. This resulted in a release of air followed by a slight improvement in the ability to ventilate via ETT. Bilateral chest tubes were then placed. The patient’s vital signs and saturation showed gradual improvement following vigorous ventilation.

The patient was transferred to the intensive care unit with vital signs as follows: blood pressure, 157/79 mm Hg; heart rate, 106 beats per minute; and 100% oxygen saturation with ventilatory settings of intermittent mandatory ventilation 12, tidal volume 800, and FiO₂ 1.0. Intraoperative chest x-ray results revealed subcutaneous air, lungs fully expanded, absence of pleural effusions, and bilateral chest tubes intact.

The patient was awake and alert later that day. The patient extubated herself 4 days later and was placed on supplemental oxygen via aerosol mask. Within the hour, the patient developed coarse rhonchi with left lower lobe crackles and expiratory wheezes. Throughout the next 24 hours, several episodes of desaturation required the patient to be placed on 100% oxygen via nonrebreathing mask. The chest x-ray revealed left lower-lobe atelectasis, and prophylactic vancomycin was administered for suspected pneumonia. The patient was transferred out of the intensive care unit on the seventh postoperative day and was discharged home 4 days later.

Discussion

Jet ventilation. Jet ventilation for intraoperative laser surgery has several advantages when compared with standard endotracheal intubation. There is increased surgical exposure to the posterior one third of the larynx and arytenoid cartilages, in addition to the lack of vocal cord distortion. Another advantage of JV is the removal of smoke and debris from the surgical field during lasering procedures. When the stylet is placed distal to the vocal cords, the passive exhalation of gases following insufflation flushes the smoke outward and prevents aspiration of tissue and debris. Perhaps the most significant benefit of JV is the reduction in the incidence of airway fires during laser procedures that involve the vocal cords. The 3 primary sources of airway fires related to laser procedures include the ETT, the emission of gases, and eschar (built-up carbonized tissues).

The standard ETT usually is made of a flammable polyvinyl chloride material. The use of a nonflammable metal stylet eliminates the ETT as a source of fire. The ignition of gases flowing from the tip of the stylet is another source of fire. However, the risk of fire can be reduced by blending air with oxygen, thereby reducing the percentage of oxygen emitted. The ignition of eschar from lasering is another source of airway fire. The periodic removal of eschar by the surgeon reduces the incidence of ignition.

Relative contraindications to JV include any condition where there is an established risk of aspiration, because the airway is unsecured. Patients with a full stomach, history of active hiatal hernia, or obesity are at risk for aspiration. Obesity is associated with delayed gastric emptying, decreased gastric pH, and decreased extrapulmonary compliance. Decreased compliance requires increased insufflating pressures and can therefore contribute to an increased incidence of barotrauma. Severe lung and cardiac disease also are considered relative contraindications. Absolute contraindications to JV include any conditions that would result in obstruction of the upper airway, such as laryngeal papillomas, arytenoid scarring, or adducted vocal cords. An obstructed
upper airway would not allow for passive exhalation of gases, and with continued JV insufflation would result in ruptured alveoli.

Several potential complications are associated with JV. Tracheal dissection can occur almost instantaneously with a misdirected laser. Tracheal perforation is another complication that could be caused either by blunt trauma from the rigid stylet, or by a heated stylet that was improperly exposed to the laser. Tracheal dissection also may occur if insufflation pressures are at or near 50 psi, and the stylet is in direct contact with the tracheal wall. The most common complication associated with JV is subcutaneous emphysema. Subcutaneous emphysema results when air is forced into the tissue through an incision or laceration. Normally, treatment is not required, and the condition will resolve spontaneously. However, if the air is forced into tissue planes, the air can dissect cephalad, resulting in subcutaneous emphysema of the neck and face; or caudad, producing a pneumothorax.

**Pneumothorax.** Pneumothorax is the presence of air within the pleural space that results in a loss of negative intrapleural pressure. A pneumothorax can differ in severity and size. A pneumothorax that occupies less than 15% of the pleural space is considered small and may not require chest tube placement. A moderate pneumothorax consists of involvement of 15% to 60% of the pleural space. Greater than 60% involvement is considered a large pneumothorax and will inevitably require chest tube placement.

Three types of pneumothorax can be defined. Type I involves an intrapulmonary alveolar rupture that produces retrograde dissection of air along perivascular lines, resulting in mediastinal emphysema. Causes include factors that increase airway pressures, such as expiratory valve dysfunction or coughing, as well as a weakened alveolar septum resulting from infection or chronic lung disease. A type II pneumothorax involves injury to the visceral pleura, which results in the escape of air into the pleural space. Some common causes include esophageal trauma, a ruptured subpleural bleb, rib fracture, stellate ganglion blocks, and central line placement. Type III pneumothorax consists of an interruption of the parietal pleura with air entry into adjoining structures and cavities. Causes include laparoscopy, open chest tubes, tracheostomies, thyroidectomies, as well as hypopharyngeal trauma during laryngoscopy.

All 3 types of pneumothorax can evolve into a tension pneumothorax. A tension pneumothorax is a life-threatening condition that exists when the communication between the bronchoalveolar and pleural spaces acts as a one-way valve. The effect of this one-way valve allows air to escape into the pleural space during inhalation, but closes during exhalation, leading to progressive accumulation of intrapleural air. The accumulating air increases pressure within the pleural cavity, which results in a shifting of the mediastinum away from the affected side. This mediastinal shift causes compression of the opposite lung and great vessels, decreasing venous return to the heart and consequentially resulting in a decreased cardiac output. Physical signs associated with a tension pneumothorax include the following: decreased ventilatory compliance with increased peak inspiratory pressures, decreased or absent end-tidal CO₂, hypotension, tachycardia, diminished or absent breath sounds, tracheal deviation, venous neck distension, and subcutaneous emphysema.

**Anesthetic implications.** The diagnosis of tension pneumothorax is a difficult task for the anesthesi provider, considering that common JV setups do not allow for monitoring of peak inspiratory pressures or end-tidal CO₂. Additional signs of a developing tension pneumothorax, such as tracheal deviation and subcutaneous emphysema, are not easily identifiable when the patient is fully draped. Asymmetrical chest excursion with diminished or absent breath sounds on the affected side also may prove difficult with a patient who is obese or who has chronic pulmonary disease, due to an increased chest wall thickness and decreased extrapulmonary compliance. Normally a precordial stethoscope is placed on the patient’s left chest, but during JV, the stethoscope may be more beneficial when placed in the suprasternal notch. This placement provides a clearer quality and assessment of JV insufflations and can pick up changes in tone should the tip of the stylet come in contact with the mucosal wall. Hypotension and tachycardia are late signs of a tension pneumothorax, as the increasing intrathoracic pressure retards venous return to the heart, decreasing cardiac output and producing reflex tachycardia.

Once a tension pneumothorax is diagnosed, immediate needle decompression with a large-bore catheter into the second or third intercostal space, midclavicular line, on the affected side is the appropriate intervention. A needle also can be placed in the fourth or fifth interspace laterally on the affected side. Continued attempts to ventilate...
the patient prior to needle decompression will only accelerate the patient’s hemodynamic decompensation. Chest tube placement should follow immediately, along with an intraoperative chest x-ray.

**Causal factors.** A variety of factors can contribute to the incidence of tension pneumothorax during JV. The use of excessive ventilatory pressures can result in barotrauma, as previously discussed. Additionally, the intense mucosal drying that occurs within 10 minutes of initiating JV significantly increases the incidence of barotrauma with resultant alveolar rupture. The patient’s underlying pathology also can play a role in the development of alveolar rupture with resulting pneumothorax. For example, patients with chronic obstructive pulmonary disease require longer expiratory times because elastic recoil is diminished. Inadequate times for passive exhalation can result in distal air trapping and rising end-expiratory volume and pressure, with consequential rupture of blebs and bullae. Direct contact with the tracheal wall when delivering pressures at 50 psi can result in tracheal transection. The most common factor associated with the development of tension pneumothorax is the combination of JV with a lacerated mucosal lining. Mucosal lacerations can occur as a result of laryngoscopy, use of a tube exchanger, or during surgical instrumentation. Usually the lacerations are so small that identification after an incident is difficult if not impossible.

**Considerations for safer JV practice.** Considerations for future JV use include the implementation of a modified surgeon’s laryngoscope, whereby the stylet’s proper placement is secured throughout the entire surgical procedure. Another advantageous device that could aid in proper stylet maintenance is the implementation of a video camera attached to surgical instrumentation that allows viewing of the surgical field and stylet by both surgeon and anesthesia provider. A modified end-tidal CO₂ adaptor for use with the jet ventilator also could prove useful in the early recognition of a tension pneumothorax.

**Summary**

The anesthetic management of a patient undergoing JV presents a challenging task. Focus upon the early recognition of a tension pneumothorax is essential, because its development during high-pressure ventilation can be catastrophic. Considerations for future management techniques present us with many options, but only through vigilance and detection of early warning signs can consequential interventions take place.

**REFERENCES**


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