Airway Collapse with an Anterior Mediastinal Mass Despite Spontaneous Ventilation in an Adult

Jeffrey C. Gardner, MD, and Roger L. Royster, MD

Patients with anterior mediastinal masses are at increased risk for perioperative complications. Our case demonstrates that airway collapse and inability to ventilate may occur in the asymptomatic adult despite spontaneous ventilation with inhaled anesthesia and an endotracheal tube. Given the sudden and profound presentation of cardiopulmonary collapse, rigid bronchoscopy should be immediately available to facilitate life-saving ventilation. Though repositioning the pediatric patient lateral or prone has been reported to reestablish airway patency, this maneuver may be of limited benefit in the adult population because of a more ossified and developed chest wall. Lastly, if a high-risk patient requires a general anesthetic, strong consideration should be given to preinduction placement of femoral cardiopulmonary bypass cannulae and the availability to immediately initiate cardiopulmonary bypass. (Anesth Analg 2011;113:239–42)

CASE REPORT

A 19-year-old, Caucasian male with a 6-week history of fever, lassitude, unintentional weight loss, cough, and a 13-cm anterior mediastinal mass presented for left thoracoscopy and diagnostic biopsy. Review of the preoperative imaging, including computed tomography scan obtained 2 days before presentation, demonstrated a heterogeneous anterior/middle mediastinal mass encasing the arch vessels, superior vena cava, and trachea with a 60% compression of the distal trachea and right mainstem bronchus (Figs. 1, 2, and 3). Despite the impressive appearance of his imaging, he denied dyspnea, orthopnea, or cough exacerbated by supine positioning. Physical examination was remarkable for a thin male who appeared to be his stated age, supra and infracavicular fullness, and decreased breath sounds over the right apical lung field, absence of facial or upper extremity plethora and who was breathing comfortably in the supine position. Preoperative vital signs are detailed in Table 1.

Our plan was to perform anesthesia for the thoracoscopy and biopsy with spontaneous ventilation during the entire case because of his extensive disease. The patient was lightly premedicated with titrated midazolam (2 mg total) in the holding area, after which he remained alert and spontaneously interactive, and he maintained his baseline room air saturation of 98%. He was escorted to the operating suite, where standard ASA monitors were placed. An inhaled induction was accomplished with 8% sevoflurane with 100% oxygen with concomitant placement of a left radial arterial catheter. As spontaneous ventilation continued, tidal volumes were noted to decrease with increasing anesthetic depth. The patient’s spontaneous mask ventilation was not difficult. After achieving a stable plane of anesthesia, a 7.5 French Univent® (Fuji Systems Corporation, Tokyo, Japan) tube was placed via uncomplicated direct laryngoscopy for eventual lung isolation in anticipation of planned fiberoptic bronchoscopy by the surgical team. There were bilateral breath sounds and bilateral chest expansion. Pulse oximetry showed an arterial saturation of 100%, and end-tidal CO2 remained in the normal range. The patient remained stable, ventilating spontaneously while waiting for the surgeons to perform bronchoscopy.

Bronchoscopy revealed total collapse of the right main-stem bronchus; this was verified as the endotracheal tube (ETT) was retracted to near the glottic opening so that the surgeon might better ascertain the anatomy. At this time it was noted that movement of the right hemithorax and breath sounds of the right lung fields were notably absent. During bronchoscopy the arterial saturation began decreasing despite manual synchronous airway pressure supplied during spontaneous ventilation. Bronchoscopy was discontinued; the arterial saturation continued to decrease to dangerous levels, and the end-tidal carbon dioxide was virtually undetectable despite having discontinued the volatile anesthetic.

The patient continued to make respiratory efforts, but bradycardia and ST segment elevations were noted. Aggressive measures were used to reestablish adequate oxygenation and ventilation. These included attempted...
manual ventilation, prolonged sigh breaths, repositioning the patient in a left-lateral semiprone position, removal of the ETT, and attempted bag-mask ventilation. A rigid bronchoscope was requested. After repositioning the patient supine, the surgical team began preparation for femoral access for emergent cardiopulmonary bypass (CPB).

We continued maneuvers to achieve adequate ventilatory pressure and reestablish air movement. Suddenly, the patient’s right hemithorax began to move as he began stronger respiratory efforts as he awoke from his anesthetic. Pulse oximetry showed a rapid increase in his arterial saturation. His heart rate increased to the 130’s with a mean arterial blood pressure of 120 mm Hg, and his ST segments normalized. Throughout this process an adequate perfusion pressure was maintained without pharmacologic intervention and adequate spontaneous ventilatory efforts continued. An arterial blood gas obtained during his respiratory and hemodynamic recovery demonstrated a profound respiratory acidosis with a pH of 7.012, $P_{CO_2}$ of 132.3 mm Hg, $P_{O_2}$ of 96.2 mm Hg, and a base deficit of 1.5 mEq/L. The patient was allowed to emerge from his anesthetic and was taken to the postanesthesia care unit where he was conversant and had normal vital signs and electrocardiogram. The following day a cervical node biopsy was obtained under local anesthesia that revealed a diagnosis of Hodgkin’s lymphoma.

**DISCUSSION**

Airway and vascular collapse is a well-recognized complication when caring for the individual with an anterior mediastinal mass. Generally accepted tenets of care include avoidance of general anesthesia when possible in symptomatic patients with large masses, in children with orthopnea and tracheal compression with $<50\%$ residual cross-sectional area, in the presence of superior vena cava syndrome, and in those with large pericardial effusions.

If general anesthesia is necessary, a titrated stepwise induction with maintenance of spontaneous ventilation and avoidance of neuromuscular blockers is strongly advised. Only after a successful trial of controlled ventilation should the use of neuromuscular blocking drugs be considered. If ventilatory difficulties ensue, reported interventions have included repositioning the supine patient laterally or prone, placement of a rigid bronchoscope past the obstruction, or initiation of CPB.

One publication asserts that airway compromise in the adult patient is a rare occurrence intraoperatively and is more likely to manifest in the postoperative period. Despite this observation, our experience underscores the fact that airway compression can and does occur in the asymptomatic adult, and that one should not be lulled into a false sense of security when formulating an anesthetic plan with spontaneous ventilation.

Airway patency in the nonpathologic state exists as a delicate balance between 2 opposing forces: the expansive tendency of the thoracic cage and the contractile nature of lung-elastic recoil. At equilibrium, these 2 opposite vectors generate a small negative intrapleural pressure facilitating parenchymal distention and airway patency. During periods of inspiration (spontaneous ventilation), a more negative intrathoracic pressure and transpulmonary gradient is generated because of chest wall expansion and diaphragmatic excursion, which results in parenchymal expansion with airway dilation. During expiration, with intercostal...
and diaphragmatic muscle relaxation, the transpulmonary gradient decreases and luminal diameters are reduced.

General anesthesia reduces functional residual capacity (FRC) and alters respiratory mechanics in a manner that favors airway collapse. In the supine position, the abdominal contents tend to push the diaphragm cephalad into the chest, which reduces negative intrathoracic pressure and FRC. The distensile nature of the thoracic wall is reduced in the anesthetized state, further decreasing FRC by approximately 20%. Consequently, the chest wall expansion/lung-elastic recoil equilibrium favors smaller lung volumes and closure of susceptible airways especially in dependent areas of the lung.

In the pathologic state, such as in the presence of an intrathoracic mass, both anatomic and physiologic perturbations may occur. Mediastinal tumor invasion may compromise the cartilaginous structural integrity, making the airway more susceptible to direct tumor compression. With tumor invasion, intrathoracic collapse with forced expiration and reduced or lack of airway dilation during inspiration may occur. Similarly, partial upper airway obstruction in the rapidly spontaneously breathing patient may generate sufficiently negative intraluminal pressure to collapse the compromised segment.

Because bilateral chest rise and bilateral breath sounds were noted in our patient in the supine position after intubation, we believe that the compressive effect of the mass in conjunction with the increasing anesthetic depth led to right mainstem collapse after intubation but before bronchoscopy. His small spontaneous tidal volumes probably reduced his negative intrathoracic pressure, which was helping to maintain his airway. Paralytics were not administered after intubation because of airway compression beyond the tip of ETT. A likely key to his resuscitation was discontinuing the sevoflurane, which in turn allowed him to increase his tidal volumes, thus increasing the magnitude of the subatmospheric intrapleural gradient, and thereby allowing expansion of the collapsed bronchial segments. Because no other maneuvers appeared to be working, the decision was made to extubate the trachea, because mask ventilation had not been difficult and it might allow the patient to take deeper breaths.

This case also underscores prior observations regarding the unsuitability of CPB as a rescue modality for acute compressive pathology unless cannulae are placed before induction. Given the sudden and profound onset of symptoms, anoxic brain injury is likely to ensue in the time required for groin cannulation in a potentially pulseless patient. Similarly, the presence of an experienced rigid bronchoscopist and bronchoscope can be lifesaving and should be physically present during induction should a general anesthetic be undertaken.

Lastly, the preponderance of reported intraoperative airway catastrophes has occurred in the pediatric population with intraoperative airway crises being less commonly reported in the adult. Given the many anatomic and physiologic differences between these 2 populations, it is not surprising that decompressive interventions may not be as effective when dealing with larger patients. Particularly when positional changes are considered after airway collapse has occurred, the thicker, more ossified, and less compliant thoracic cage of the adult may not afford displacement of the rigid mass from vital intrathoracic structures as happened in this case.

In summary, we believe that our patient highlights the potential for dynamic airway collapse and the inability to ventilate despite maintenance of spontaneous ventilation under general anesthesia in patients with an anterior mediastinal mass. The decision to use paralytics after induction and intubation may be perilous in the high-risk patient with disease distal to the ETT. Use of these drugs is strongly discouraged if the planned procedure is solely diagnostic in nature and thus devoid of any meaningful tumor debulking. Repositioning adult patients lateral or prone in comparison with children may be less effective owing to a more ossified and developed chest wall. Should airway collapse occur, rigid bronchoscopy should be immediately available to facilitate lifesaving ventilation. Lastly, if a high-risk patient requires a general anesthetic, strong consideration should be given to placement of the femoral cannulae with pump stand-by before induction.

### REFERENCES


### DISCLOSURES

**Name:** Jeffrey C. Gardner, MD.  
**Contribution:** Participated in actual case, reviewed data and analysis, and prepared a manuscript.

**Name:** Roger L. Royster, MD.  
**Contribution:** Participated in actual case, reviewed data and analysis, and prepared a manuscript.

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<th>Table 1. Patient’s Perioperative Vital Signs</th>
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<td>Preoperative holding</td>
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<td>BP (mm Hg)</td>
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PACU = postanesthesia care unit; BP = arterial blood pressure; HR = heart rate; RR = respiratory rate; N/A = not applicable; SPO₂ = saturation of peripheral hemoglobin; FiO₂ = fraction of inspired oxygen.