

Direct Laryngoscopy as an Aid to Relieve Airway Obstruction in a Patient with a Mediastinal Mass

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A major concern in the anesthetic management of patients with anterior mediastinal masses is the possibility of severe airway obstruction. A recent report by Neuman *et al.*¹ described the hazards of anesthesia and airway management in these patients. While most of these patients have obstructive respiratory symptoms, there have been reports of patients who were asymptomatic prior to the procedure,² but developed intraoperative airway difficulties. We describe our experience with such a patient.

CASE REPORT

A 13-yr-old girl was admitted to the hospital because of intermittent fevers and a nonproductive cough. A chest radiograph showed a large anterior mediastinal mass, and the patient was scheduled for mediastinoscopy and biopsy of the mass under general anesthesia. History and physical examination were otherwise unremarkable. Although a chest radiograph and computerized tomography scan of the chest showed mild extrinsic compression of the trachea, the patient showed no signs or symptoms of an obstructive airway, in both the erect and the supine position.

Anesthesia was induced with thiopental iv, and endotracheal intubation facilitated with succinylcholine iv. Anesthesia was maintained with N₂O/O₂ and halothane with controlled ventilation.

As soon as the surgeons were ready to make their incision, breath sounds ceased to be heard through the esophageal stethoscope and inflation pressures, which, until that time, had been 20 cm H₂O, rose to 45 cm H₂O. At this point, attempts to ventilate were futile, and a rapid check for endotracheal tube obstruction or anesthesia machine malfunction was done without any relief of the problem. Atropine was administered iv, and 100% oxygen was administered, since the patient showed signs of hypoxia and bradycardia. Laryngoscopy was performed to check the position of the tube, and, suddenly, ventilation was feasible. Relaxation of the blade resulted in recurrence of the airway obstruction, which disappeared with reapplication of laryngoscope traction.

The surgery was postponed, and the patient was allowed to wake up while holding traction with the laryngoscope. After putting her in the lateral position, her trachea was extubated without any problems. On a further date, the procedure was done under local anesthesia without any airway problems.

The diagnosis was Hodgkins lymphoma.

DISCUSSION

Although patients with anterior mediastinal masses may manifest obstructive airway problems, the factors leading to it are unknown. Reduction of the transpleural pressure gradient, relaxation of voluntary muscles and bronchial smooth muscle, and reduction of the functional residual capacity are possibilities.¹

A tissue diagnosis is usually necessary prior to establishing therapy.³ Thus, anesthesia must be given to patients in whom the chances of airway obstruction are high. Some of the ways anesthesiologists have dealt with this problem have been awake tracheal intubation,⁴ inhalation inductions maintaining spontaneous ventilation, splinting the airway by passing a tube beyond the site of obstruction,²⁵ and rigid bronchoscopy. Turning the patient lateral to allow the tumor mass to fall away from the airway may be effective.

The probable mechanism of the obstruction in this case is compression of the trachea or bronchus by the tumor mass. This compression created a critical airway diameter, which had been maintained by intact chest and lung mechanics. Once muscular relaxation was achieved, the airway collapsed. During spontaneous inspiration, the pleural pressure is negative to the atmospheric pressure. Inspiratory forces, therefore, exert a potential widening of the airways greater than that resulting from the increased elastic recoil during inspiration.⁷

During expiration, the caliber of the airways decreases as lung volume decreases, but dynamic compression plays a role in further decreasing the caliber of the large airways, especially during forced expiration. In the bronchial tree of a healthy person, the trachea and first few generations of bronchi are the major site of airway resistance. As the airway muscles contract, they narrow the bronchioles but stiffen the larger bronchi, thereby increasing the expiratory flow rate. If these muscles are relaxed, the maximum expiratory flow rate decreases because of the increased compressibility of the large airways. This may lead to airway obstruction.⁷ If, then, spontaneous ventilation would have been maintained in our patient, it is conceivable that the airway would have remained patent due to these mechanics of respiration.

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Keon⁸ described a patient with tumor enveloping the heart and infiltrating the pericardium who arrested during induction of anesthesia and was unable to be resuscitated. He recommends preoperative cardiac assessment in patients who are symptomatic. Our patient clearly does not fit this description.

Our fortuitous finding that laryngoscopy relieved the obstruction has not been reported previously, and should be added to the list of maneuvers to relieve these airway obstructions. The mechanism of action is not clear, but a possibility is proposed. Extrinsic compression of the airway may be accentuated by voluntary muscle relaxation and bronchial smooth muscle relaxation. Laryngoscopy might conceivably apply tension to the tracheal wall tending to straighten it out somewhat, much as pulling two ends of a rope will tend to straighten it and make it taut. This mechanism presupposes that the airway obstruction is primarily a passive one, in which bronchial smooth muscle relaxation allows the weight of the mass to compress the airway.

In conclusion, we want to reemphasize that patients with anterior mediastinal masses are at risk of developing airway obstruction. If this occurs under anesthesia,

direct laryngoscopy may be carried out as an aid to relieve the obstruction.

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Treatment of Acute Respiratory Failure by Extracorporeal Carbon Dioxide Elimination Performed with a Surface Heparinized Artificial Lung

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Progressive states of acute respiratory failure (ARF) require mechanical ventilation, positive end-expiratory pressure (PEEP), and high inspiratory fractions of oxygen. Despite this treatment, however, hypoxemia continues to be a major problem. The carbon dioxide tension, on the other hand, can be maintained at near-nor-

mal levels, but it is at the expense of excessive hyperventilation, which, in its turn, entails high inspiratory pressures. Accordingly, high inspiratory pressures cause alveolar barotrauma,¹ and the ensuing regional pulmonary hypoxemia² and hypoxemia³ most certainly determine the final outcome of ARF.⁴

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