Spontaneous tension pneumothorax
and mediastinal emphysema associated with
anesthesia for cesarean section

CHARLES H. HUBBERT, MD, FACP
WILLARD T. ROBERSON, CRNA
JOE A. SOLOMON, CRNA, BS
Memphis, Tennessee

Spontaneous tension pneumothorax is a catastrophic event which may occur during anesthesia and surgery, causing dramatic changes in the pulmonary and cardiac systems. The authors present two case reports of spontaneous tension pneumothorax occurring during anesthesia for cesarean section. They discuss the prompt treatment which is necessary to decompress the high intrapleural tension, thereby preventing organ system injury.

Spontaneous tension pneumothorax, occurring during anesthesia and surgery, is a serious complication that requires prompt recognition and treatment. The first report presented here concerns a case of bilateral pneumothorax occurring during anesthesia for cesarean section. It includes a review of the pathophysiology, diagnosis and management of spontaneous pneumothorax. The second report included in this article illustrates a case of mediastinal emphysema.

Case 1
A healthy 17-year-old Caucasian woman underwent an emergency cesarean section for a prolapsed umbilical cord. Preoxygenation with 100% oxygen was given for two minutes. A crash induction with cricoid pressure and thiopental 250 mg and succinylcholine 80 mg IV preceded an atraumatic intubation. A healthy infant was delivered in five minutes.

Shortly thereafter, during suturing of the uterus, massive swelling of the upper thorax, neck and head developed together with cyanosis and increased pressure to ventilate. It was thought that there had been extubation or obstruction of the endotracheal tube, therefore, the tube was withdrawn. Because of severe soft tissue swelling and distortion of the neck and pharynx, re-intubation could not be accomplished. Regurgitation of stomach contents occurred, and an emergency tracheostomy was performed. Air under pressure was noted when the soft tissue of the neck and the trachea were entered.

A chest x-ray showed an almost complete collapse of the left lung and a 70% collapse of the right lung. Bilateral chest tubes were inserted. Initial arterial blood gases showed a severe hypoxemia ($pO_2$ 45 on 100% oxygen) without acidemia. With therapy, the hypoxemia rapidly improved. Cardiac arrest did not occur though fluctuating bradycardia and tachycardia were noted. Anesthetic management thereafter consisted of controlled ventilation with 100% oxygen, fentanyl 4 ml, diazepam 10 mg, and intermittent succinylcholine.

Postoperatively, treatment was begun for aspiration pneumonitis. Eclampsia manifested by hypertension and seizures developed on the fourth postoperative day. Improvement occurred gradually, and the tracheostomy and chest tubes were removed on the sixth postoperative day. The
patient was discharged in good health on the tenth day.

Case 2

A healthy 18-year-old Caucasian woman in labor underwent cesarean section for cephalopelvic disproportion. The anesthetic induction was similar to that in Case I. Shortly after atraumatic intubation, prominent swelling with crepitation of the upper thorax, neck and head developed, associated with varying arrhythmias and transiently increased pressure to ventilate. Delivery of a healthy infant was accomplished in less than five minutes.

Anesthesia was maintained with 50% nitrous oxide and 50% oxygen, diazepam 10 mg, and intermittent succinylcholine. There was no evidence of pneumothorax by examination or on the x-ray, and arterial blood gases were within the normal range. However, mediastinal and soft tissue air was quite evident on the x-ray. Assisted ventilation was given postoperatively, and the patient was extubated six hours after surgery. The extensive subcutaneous air resolved completely over a 24-hour period, and the patient was discharged in good health five days after surgery.

Pathophysiology

Spontaneous pneumothorax, as opposed to traumatic pneumothorax associated with defined trauma to the chest wall or lung, can occur in apparently healthy individuals or in patients with pulmonary disease. In the healthy patient, it is assumed to be caused by a rupture of an otherwise insignificant emphysematous bleb or bulla or an association with previous inflammatory lung disease. Spontaneous pneumothorax understandably might be more likely to occur in patients with known emphysematous or cystic lung disease and pulmonary infections, especially tuberculosis. Occasionally, it occurs in asthmatic patients.

Tension pneumothorax may result from excessive airway pressure, usually caused by malfunctioning anesthesia systems or exhaust valves. Martin and Patrick have reviewed pneumothorax and its significance to the anesthetist in other types of surgery. When pneumothorax under tension occurs, dramatic changes in pulmonary and cardiac physiology may appear.

With a small defect or closed pleural lesion, inspiration causes the mediastinum to move to the ipsilateral side and expiration to the contralateral side. A large or open defect allows a much higher intrapleural tension to develop so that inspiration does not allow for the shift of the mediastinum to the side of the pneumothorax.

A check valve mechanism allows air to enter the pleural cavity during inspiration, but the air is prevented from leaving during expiration. A relatively lower subatmospheric pressure on the unaffected side allows the mediastinal structures to be shifted away from the side of the pneumothorax. Eventually airflow to the good lung is compromised, effective lung volume is decreased and hypoxemia develops.

Greatly increased intrapleural pressure and a shift of the mediastinum impede the filling of the great veins in the chest, resulting in a decrease in cardiac output. Vagal reflexes probably play a role in decreased cardiac function as well as causing bronchospasm. A combination of acute severe hypoxemia and associated hemoglobin oxygen desaturation, hypotension, and decreased cardiac output may produce multiple organ dysfunction or damage. The pathophysiology produced by tension pneumothorax explains the abnormal signs.

Symptoms and signs of rupture of the tracheobronchial tree in the awake patient include pain, dyspnea, and tachypnea. When the patient is under anesthesia, however, the anesthetist has to rely on the following signs: (1) increased pressure to ventilate and/or bronchospasm; (2) cyanosis; (3) cardiac arrhythmias (ectopic activity, bradycardia or tachycardia); (4) blood pressure changes, most significantly hypotension; and in many cases, (5) subcutaneous air (emphysema) of the head, neck and upper thorax.

Other physical signs, dependent on the size and acuteness of the air leak and the intrathoracic compromise it produces, include decreased breath sounds and hyper-resonance over the involved lung, displacement of the heart and trachea to the contralateral side, and subcutaneous emphysema with crepitition.

Diagnosis should be readily made from the above signs since delay in decompression of a tension pneumothorax (i.e., while waiting for a chest x-ray or arterial blood gas determinations) may be disastrous. A chest x-ray could show lung collapse, shift of the mediastinum (heart, great vessels, and trachea) to the opposite side, depression of the diaphragm on the affected side, and/or air in the tissues. Bilateral tension pneumothoraces might not show a shift of the midline structures.

Anesthetic management

Tension pneumothorax should be suspected in any catastrophic development after induction of anesthesia, during maintenance of anesthesia, or in
the immediate postoperative period. A high index of suspicion will allow for diagnosis and treatment before central nervous system or other organ system injury occurs.

The highest inspired oxygen concentration available should be used. Minimal use, if any at all, of nitrous oxide is in order to prevent further rapid increase in intrapleural pressure.6-7 The patient may be already intubated when the pneumothorax occurs; this should be continued as is necessary for better and controlled ventilation.

The need for ventilation of the lungs given a high airway resistance poses a dilemma. The high pressure required to overcome the resistance may make the tension pneumothorax worse. The lowest possible pressure should be used until definitive therapy has been accomplished. The frequent, very high pressure necessary to ventilate (greater than 40 cm H2O and often greater than 60 cm H2O) should not be confused with malplacement or malfunctioning of the endotracheal tube unless this is indeed the case.

Replacement of the endotracheal tube may be difficult or impossible because of airway obstruction and distortion caused by air under pressure in the soft tissues of the neck, pharynx, and larynx. An obstructed airway predisposes the patient with a full stomach toward vomiting and aspiration.

Definitive therapy includes prompt reduction of intrapleural tension through thoracostomy tube placement by the surgeon. Placement of a large-bore needle through an intercostal space may be life-saving until thoracostomy drainage can be effected.

Adjuncts to successful therapy might include muscle relaxants to improve the compliance of the chest wall and bronchodilators to improve associated bronchospasm. Controlled ventilation and appropriate sedation constitute the remainder of the emergency management of tension pneumothorax.

Mediastinal emphysema, as seen in Case 2, can result from the traumatic perforation of the trachea or esophagus, from the rupture of pulmonary alveoli in interstitial emphysema of the lung, or from the spread of air along the fascial planes of the neck or pharynx.9 Traumatic rupture of the bronchial tree may occur in association with pneumothorax from puncture wounds of the chest or in association with endotracheal anesthesia, as during intubation. If there is no associated rupture of the esophagus (and complicating mediastinitis) or bronchial tree (with pneumothorax) the usual course is uncomplicated, with resorption of the air from the tissues in several hours to a few days.

Spontaneous pneumothorax is relatively unusual in the pregnant patient. While this condition occurs most frequently in young adult males, approximately only 15 cases have been reported or reviewed in the pregnant patient who is in the third trimester and peri-partum period.10,11 Two of these patients had more than one episode of pneumothorax during the course of their hospital stay. The decision as to placement of chest tubes seemed to depend on the patient's clinical appearance since many were managed conservatively without difficulty.

Only one patient had tension pneumothorax.11 A summary of this case reveals that the patient had chronic lung disease with emphysematous blebs. She was admitted near term with a small right pneumothorax manifested by cyanosis, tachycardia, tachypnea, and bronchospasm. A pelvic examination under halothane and oxygen anesthesia by mask and spontaneous ventilation was uneventful.

After the induction of labor and while an epidural catheter was being injected, however, the patient appeared to be in extremis (near death). Diagnosis of tension pneumothorax was suspected after the trachea was noted to have shifted from the midline. The pneumothorax was immediately decompressed, first with a trocar, then with a thoracostomy tube, without injury to patient or infant.

The authors have anecdotal, unpublished information about a young woman who expired because of central nervous system injury following unilateral tension pneumothorax on induction of anesthesia for general surgery. It may be that unilateral tension pneumothorax with shift of the mediastinum is more devastating because of circulatory impairment. Bilateral tension pneumothoraces, while capable of producing severe hypoxemia, may be protective of the mediastinal structures and thereby lessen the decrease in cardiac output.

The relationship of pregnancy to the development of pneumothorax is not clear, but hormones may play a role. Estrogens and progesterones are at their highest level at term. Catamenial pneumothorax, reportedly caused by endometriosis in the lung or pleural cavity, occurs during menses when hormone levels also are high.12

**Conclusion**

Spontaneous tension pneumothorax may occur during anesthesia and surgery. Any catastrophic development such as severe hypoxemia, hypotension, cardiac arrhythmias, increased pressure to ventilate, bronchospasm, or cardiac arrest should arouse one's suspicion of tension pneumothorax.

Definitive therapy consists of rapid decom...
pression of the tension by means of a large-bore needle and tube thoracostomy. Prompt therapy, along with high concentrations of oxygen, will help reduce the possibility of permanent organ injury caused by hypoxia, hypotension, and hypoperfusion.

REFERENCES


AUTHORS

Willard T. Roberson, CRNA, AD, received his Associate Degree in Nursing from Columbia State Community College in Columbia, Tennessee. He is a 1979 graduate of Duke University School of Anesthesia.

Charles H. Hubbert, MD, FACP, received his medical degree from the University of Mississippi and took residences in internal medicine and anesthesiology at the University of Tennessee and Baptist Memorial Hospital, Memphis.

Joe A. Solomon, CRNA, BS, received his Bachelor's Degree in both respiratory therapy and nursing from the University of Central Arkansas. He is a 1979 graduate of Duke University School of Anesthesia.

The authors are members of the Department of Anesthesia at Baptist Memorial Hospital East in Memphis, Tennessee.