indeed possible.
It is concluded, therefore, that sickle cell trait should be considered in the differential diagnosis of all Negro patients who present with suspected vasocclusive episode or sudden death, and that sickle cell trait may be potentially lethal under certain circumstances.

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Management of Bronchopleural Fistula Complicating Therapy with Positive End Expiratory Pressure (PEEP)*


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Positive pressure ventilation is possible in the presence of a large air leak if a volume-cycled ventilator and adequate pleural suction are used. The ventilator, the lungs, and the pleural suction apparatus are an interdependent system and cause a number of difficult problems in respiratory management. Although seemingly contraindicated, the addition of PEEP improved oxygenation in our patient when his course was complicated by the adult respiratory distress syndrome. Guiding principles based on an analysis of a complex situation were developed which can be useful in the future care of patients with this difficult problem.

The addition of positive end expiratory pressure (PEEP) during the course of ventilator therapy has become increasingly popular as a means of increasing arterial oxygenation and avoiding an excessively high fraction of inspired oxygen.12 This technique is not without complications, among which pneumothorax is one of the most hazardous.

This report relates our experience in managing a patient with Pseudomonas pneumonia and lung abscess who developed tension pneumothorax while being supported with PEEP. The presence of a bronchopleural fistula with large air leak and continued need for ventilator support and later development of the adult respiratory distress syndrome posed a challenging therapeutic dilemma and a number of unique problems during ventilator support and the weaning process.

CASE REPORT

A 35-year-old white man was admitted because of acute upper gastrointestinal bleeding. Treatment included a vagotomy and pyloroplasty and later a subtotal gastrectomy and Billroth II gastroenterostomy. Following the latter procedure he eviscerated and during induction of anesthesia there was massive aspiration of gastric contents. He developed respiratory insufficiency with temperature of 40°C and rales throughout both lung fields.

He was treated with dexamethasone, penicillin, kanamycin sulfate, and controlled ventilation with a volume-cycled ventilator. The alveolar-arterial oxygen gradient (AaDO2) rose to 612 mm Hg on 100 percent O2. When 15 cm H2O PEEP was added the AaDO2 on 100 percent O2 narrowed to 372 mm Hg and PaO2 was 102 mm Hg on 40 percent O2. The WBC rose to 25,900/cu mm, fever persisted, and copious amounts of bloody, purulent secretions were aspirated from the trachea. An attempt at weaning from ventilator support was unsuccessful two days after aspiration and he required assisted ventilation with tidal volume 1000 ml, 10 cm H2O PEEP, and 50 percent O2 to maintain a PaO2 of 60 mm Hg.

Twelve hours after attempted weaning he developed right tension pneumothorax. A right tube thoracostomy yielded 450 ml of bloody, purulent fluid and there was evidence of a continuous air leak. Cultures of sputum and empyema fluid grew Pseudomonas aeruginosa.

Antibiotic therapy was changed to gentamicin sulfate and disodium carbencillin. Despite increased pleural suction, the right lung failed to fully expand. On controlled ventilation with a delivered tidal volume of 1000 ml the expired tidal volume varied from 435 to 850 ml, minute ventilation from

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but pneumothorax persisted. PEEP was discontinued resulting in decreased air leak and increased expired tidal volume, but the right lung remained incompletely expanded. There was a diffuse pneumonic infiltrate throughout both lungs, more severe on the right, with an abscess cavity in the right upper lobe (Fig 1).

Over the following three days, temperature and leukocytosis decreased, but the chest x-ray film showed a ground-glass appearing radiodensity superimposed on the pneumonic process (Fig 2). The AaDO₂ on 100 percent O₂ rose to 539 mm Hg and 70 percent O₂ was required to maintain a PaO₂ between 60 and 75 mm Hg. After increased pleural suction, dehydration, and albumin infusion failed to improve oxygenation, PEEP was reinstated. The air leak and right pneumothorax increased. With a delivered tidal volume of 1200 ml the expired tidal volume varied from 300 to 800 ml minute ventilation from 4.5 L to 11 L, and PaCO₂ from 32 to 48 mm Hg. PEEP was set at 15 cm H₂O, but actual end expiratory pressure varied between 6 and 12 cm H₂O. Following reinfusion of PEEP the AaDO₂ on 100 percent O₂ fell to 369 mm Hg and adequate oxygenation was maintained on 35 percent to 40 percent O₂ for the next three days.

PEEP was then removed and oxygenation remained satisfactory on assisted ventilation with 35 percent O₂. A third chest tube was inserted, pleural suction was increased, and the lung became more fully expanded. Tidal volume was increased to 1300 ml and expired tidal volume varied from 300 to 550 ml and minute ventilation from 6 L to 12 L. Three-hundred-fifty ml mechanical deadspace was needed to maintain the PaCO₂ between 30 and 36 mm Hg. The ventilator indicated rapid assisted respiration without apparent patient effort and the suction applied to the chest tubes was found to be causing the ventilator to cycle.

Three days after PEEP was removed he was again evaluated for weaning from ventilator support. The AaDO₂ on 100 percent O₂ was 302 mm Hg. Inspiratory force measurement indicated a continuous vacuum. Vital capacity varied from 275 ml (4.8 ml/kg) to 400 ml (7 ml/kg) depending on the level of pleural suction. Chest tube suction was decreased and he was placed on 70 percent and later 40 percent O₂ at high flow rates with a 250 ml reservoir. During the following 72 hours he was gradually weaned from ventilator support. The right lung became fully expanded, but the air leak persisted.

His subsequent course was complicated by massive bleeding from the right chest. At thoracotomy there was a thick, purulent, fibrinous peel encasing the lung. Multiple air leaking areas were oversewn and bleeding vessels at the site of lung abscess ligated. After surgery he developed breakdown of the gastrojejunostomy anastomosis, upper gastrointestinal bleeding, and staphylococcal pulmonary and wound super-infection. He died of progressive respiratory insufficiency 40 days after admission. Post mortem findings included severe bilateral necrotizing pneumonia with multiple abscesses, atelectasis of the right lung and right empyema. There were multiple abdominal, as well as metastatic abscesses.

**DISCUSSION**

The incidence of pneumothorax and bronchopleural fistula during ventilator therapy with or without PEEP is not known. Patients with pulmonary infarction, asthma, emphysema, and necrotizing pneumonia are all predisposed to pneumothorax⁴-⁶ and not infrequently require ventilator support.

When faced with pneumothorax during ventilator
therapy, the most desirable course of action is to avoid further application of positive pressure to the airway. This minimizes parenchymal air leakage and allows pleural suction to achieve full lung expansion and sealing of visceral and parietal pleural surfaces. When ventilator support cannot be withdrawn, full expansion may still be possible if the air leak is small. When large air leaks are present, full expansion during ventilator support may not be possible. Our patient could not be weaned from the ventilator and positive pressure ventilation was necessary to support life. The application of suction through two chest tubes and removal of PEEP allowed adequate ventilation, although there was incomplete lung expansion. This experience and that of anesthesiologists during the surgical repair of bronchopleural fistula, demonstrates that positive pressure breathing can be accomplished with adequate pleural drainage and careful tracheal aspiration.6

The subsequent progressive widening of the AaDO2 and bilateral patchy alveolar infiltrate suggested development of the adult respiratory distress syndrome.1 Many of the factors which have been felt to be etiologic in this syndrome were encountered in our patient. The pathophysiologic basis of this syndrome is probably alveolar collapse leading to reduction in functional residual capacity and lung compliance, low ventilation-perfusion ratio, and arteriovenous shunting.7,8 It is thought that the addition of PEEP in this setting improves oxygenation by increasing the functional residual capacity and preventing terminal airspace collapse during expiration.7,8 A trial of PEEP was undertaken in our patient when he deteriorated despite the use of other therapeutic modalities and when toxic concentrations of oxygen were needed to maintain adequate oxygenation. We felt that the need to reverse a deteriorating clinical situation and to decrease the FIo2 outweighed the expected increase in air leak, atelectasis, and risk of chronic bronchopleural fistula.

We are unable to explain the dramatic fall in the AaDO2 when PEEP was added. Possible mechanisms which might account for the improved oxygenation include increased functional residual capacity of the left lung, decreased arteriovenous shunting due to a fall in cardiac output, or decreased shunting through the right lung because of its collapse.

The large air leak through the bronchopleural fistula caused a number of unique problems in respiratory management (Table 1). The use of a volume-cycled ventilator was mandatory since overventilation by several hundred ml per tidal volume was necessary to compensate for the air leak. Increasing pleural suction, in an attempt to fully expand the lung, caused respiratory alkalosis despite a decrease in expired tidal volume and unchanged minute ventilation. It is presumed that improved expansion of the lung increased alveolar ventilation and that the measured decrease in expired tidal volume was an artifact caused by the greater air leak. On one occasion pleural suction caused the ventilator to cycle because of the transmitted vacuum.

When the development of the adult respiratory distress syndrome prompted the addition of PEEP, further difficulties were encountered. On several occasions hypercapnia occurred and we presume that alveolar ventilation was decreased because PEEP caused an increase in right pneumothorax and lung collapse thereby decreasing the amount of ventilated lung surface. Changes

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<th>pH</th>
<th>Pao2</th>
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Table 1—Selected Data Demonstrating Problems in Respiratory Management When Bronchopleural Fistula Complicated Ventilator Therapy with and without PEEP.

From: ZIMMERMAN, COLGAN, MILLS

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in the patient's position or amount of pleural suction varied the amount of PEEP applied as well as alveolar ventilation, apparently because of further changes in the air leak and degree of lung collapse.

The expired tidal volume and minute ventilation were not reliable in detecting under- or overventilation. Frequent arterial blood gas determinations were the only means of determining changes in oxygenation and alveolar ventilation. The air leak made vital capacity, inspiratory force, and dead space to tidal volume ratio inaccurate or unobtainable as criteria for weaning. Only the AaDO₂ and clinical observation could be used as guides. Since high levels of pleural suction decreased measured ventilation it was decreased prior to weaning. A high oxygen flow and large reservoir were also needed during spontaneous respiration in order to prevent undue air dilution due to pleural suction.

Under similar circumstances in the future, we would recommend that an attempt be made to achieve full lung expansion. If the patient is being supported with PEEP, it should be removed if possible in an attempt to achieve this goal. Strong pleural suction should probably be avoided because of the disturbances in ventilator control caused by the massive air leak. When faced with progressive widening of the AaDO₂ suggesting the development of the adult respiratory distress syndrome, a trial of PEEP along with multiple pleural tubes and gentle pleural suction can be a life-saving temporizing measure.

References

Acute Myocardial Infarction in Idiopathic Hypertrophic Subaortic Stenosis

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A 61-year-old man with idiopathic hypertrophic subaortic stenosis (IHSS) and an acute anterosetal wall myocardial infarction is described. Hemodynamic study after recovery from the infarction revealed complete loss of the outflow tract obstruction, and a coronary arteriogram demonstrated the left anterior descending artery to be occluded. Acute myocardial infarction, not previously described in IHSS, must now be added to the clinical spectrum of this disorder.

Although angina pectoris, sudden death, and electrocardiographic abnormalities suggestive of coronary heart disease are common clinical manifestations of idiopathic hypertrophic subaortic stenosis (IHSS), an acute myocardial infarction is not recognized as part of

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