which Ile, in, control to tion. Ventilator increasing sensitivity" mines inspiratory threshold. We believe this is the first report of autocycling due to a cuff leak. (Chest 1991; 100:1172-73)

Ventilator autocycling is a recognized hazard of mechanical ventilation resulting in hyperventilation of the patient. Initiation of a mechanical breath during assist control ventilation occurs when an inspiratory effort causes the proximal airway pressure to drop below a specified level, i.e., the inspiratory threshold. The ventilator operator determines the inspiratory threshold by manipulating the ΔP which is the pressure drop the patient must generate to decrease proximal airway pressure from the preset PEEP to the desired inspiratory threshold pressure (Fig 1). The inspiratory threshold pressure varies inversely with the ΔP, i.e., to lower the inspiratory threshold pressure, one must increase the ΔP. The ΔP is commonly referred to as "the sensitivity" of the ventilator, but this is a misnomer since increasing "the sensitivity" actually makes the ventilator less sensitive to patient effort. Autocycling is defined as repetitive triggering of a mechanical breath because of reductions in proximal airway pressure due to causes other than patient effort. An example of this has been described in patients with large bronchopleural fistulas needing chest tube suction. In these cases negative pressure from the pleural space is transmitted to the proximal airway and triggers a mechanical breath.1,4 We report a case of ventilator autocycling from an endotracheal cuff leak that was misinterpreted as spontaneous assisted ventilation in a paralyzed patient. This misinterpretation prompted the administration of unnecessarily large doses of pancuronium bromide to suppress an erroneously perceived patient effort.

**CASE REPORT**

A 21-year-old woman was admitted to the hospital complaining of fever, nonproductive cough and dyspnea over three days. She had been diagnosed with stage IIb Hodgkin's disease one year earlier and had completed eight cycles of chemotherapy (BCNU, cytoxan, indomethacin and L-asparaginase). The patient was intubated on day 3 of admission for respiratory failure. On day 5 of hospitalization, the patient developed cyanosis and had an increased respiratory rate. The patient was found to have a fever of 39°C and a white blood cell count of 17,000. A chest X-ray showed bilateral infiltrates. A diagnosis of Legionnaires' disease was made based on the patient's clinical presentation and a positive urine antigen test for Legionella pneumophila. The patient was treated with intravenous cefotaxime, and her fever and respiratory rate improved over the next few days. However, on day 10 of hospitalization, the patient developed hypotension and required intubation and mechanical ventilation. A chest X-ray showed diffuse infiltrates, and a bronchoscopy was performed to rule out pulmonary complications of Legionnaires' disease. The patient's condition improved after initiation of ventilator support, and she was discharged from the hospital on day 20 of admission.

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**Figure 1.** A: During assist control ventilation, when a patient's inspiratory effort causes the proximal airway pressure to drop below a specified level, i.e., the ITP, a ventilator breath is triggered. The ventilator operator determines the ITP by manipulating the pressure drop (ΔP) below PEEP which is required to trigger a breath. Thus, ITP = PEEP - ΔP. B: When the patient is intubated, the presence of a cuff leak, without assist from the patient, end-expiratory pressure drifts below the pre-set PEEP until it drops below the ITP triggering a ventilator breath. This increases the respiratory rate independent of patient effort. C: By increasing ΔP, ITP is lowered, and autocycling is averted. Respiratory rate is no longer determined by the cuff leak but becomes dependent upon the patient's assisted efforts or, as shown here for a paralyzed patient, upon the ventilator's backup respiratory rate.
vinblastine, procarbazine and prednisone) and received 3,000 rads of mental and mediastinal radiation prior to admission. On admission, she was treated with broad-spectrum antibiotics, including trimethoprim-sulfamethoxazole, for a left lower lobe pneumonia. Two days later because of deteriorating gas exchange and progressive infiltrates on the chest x-ray film, the patient was admitted to the medical intensive care unit where bronchoscopic lavage was diagnostic for *Pneumocystis carinii*. In spite of continued therapy with trimethoprim-sulfamethoxazole, the patient's respiratory status worsened, and she was intubated. Mechanical ventilation was begun with a Puritan Bennett 7300a and initial settings included the following: assist control mode, 100 percent inspired oxygen concentration, a respiratory rate of 20 breaths per minute and a tidal volume of 550 ml. An arterial blood gas analysis revealed a pH value of 7.47, a PaO\(_2\) of 32 mm Hg, a PaO\(_2\) of 53 mm Hg, and an SaO\(_2\) of 90 percent (actual respiratory rate, 35 to 40 breaths per minute). Measured static respiratory system compliance was 8 ml/cm H\(_2\)O. Positive end-expiratory pressure was added to improve oxygenation. Subsequently, the patient developed a significant cuff leak from her endotracheal tube, resulting in a loss of 150 ml from each tidal volume. Increasing the cuff pressure from 20 to 40 cm H\(_2\)O did not improve the leak, and the chest x-ray film revealed satisfactory endotracheal tube position. The patient's endotracheal tube, a number 6, was replaced with a number 8 tube to reduce the leak; unfortunately, the leak persisted. Thus, the patient was paralyzed with pancuronium bromide in an effort to stabilize the position of her endotracheal tube, minimize cuff leak and maximize tidal volumes. Soon after starting pancuronium bromide the respiratory rate was noted to be regular at 30 to 35 breaths per minute instead of at the pre-set rate of 20 breaths per minute. Because machine inspirations were being properly delivered after decreases in the patient's proximal airway pressure, it was assumed that the patient was assisting the ventilator. This prompted the further administration of pancuronium bromide in order to suppress apparent patient effort, but no decrease in respiratory rate occurred. Upon examination of the patient it was confirmed that she was appropriately paralyzed and incapable of assisting the ventilator. The cuff leak was unchanged. When the airway pressure required to trigger the ventilator, i.e., the inspiratory threshold pressure, was decreased, the respiratory rate returned to the pre-set rate of 20 breaths per minute. Thereafter, no further episodes of ventilatory autocycling occurred.

**DISCUSSION**

This is the first report, to our knowledge, of ventilator autocycling secondary to an endotracheal tube cuff leak. The ventilator autocyced because it correctly sensed a drop in proximal airway pressure, and when the inspiratory threshold pressure was lowered to a point below the end-expiratory pressure of the proximal airway, the autocyced stopped. Thus, in this type of ventilator autocyced, the ventilator was sensing and responding properly to a drop in pressure caused by the gas leak around the cuff in the endotracheal tube. Although the etiology of the patient's recurrent cuff leak is unclear, our clinical suspicion was that the patient suffered from tracheomalacia which was induced by her previous course of radiation therapy and which prevented a good seal between cuff and tracheal wall.  

Endotracheal cuff leaks are a common problem in the intensive care unit. The incidence of cuff leaks in several large series has ranged from 11 to 24 percent.  

Cuff leaks can lead to significant tidal volume reductions which are exacerbated when respiratory system compliance is poor, as in the present case. Management includes cautiously increasing the cuff pressure not to exceed capillary filling pressure in the tracheal mucosa (20 to 30 mm Hg), changing the endotracheal tube to a larger size, sedation or paralysis if the cuff leak is positional, and increasing the tidal volume to maintain alveolar ventilation. The hazards of overinflating the cuff with subsequent tracheal injury have been well described.  

Ventilator autocyced has also been reported in patients with large bronchopleural fistulas requiring chest tube suction, and the mechanism is not dissimilar to what occurred in the present case.  

The negative intrapleural pressure developed from chest tube suction is transmitted through the fistula to the trachea, dropping proximal airway pressure. The ventilator senses the drop in airway pressure and initiates a mechanical breath unless the inspiratory threshold is set at a more negative pressure by increasing the ΔP from its customary value of 2 cm H\(_2\)O. Autocyced can occur with any ventilator, but three factors appear to promote its occurrence: (1) setting a relatively small ΔP or "sensitivity" on the ventilator, (2) gas leakage from the respiratory system or tubing and (3) application of PEEP or CPAP. In the presence of PEEP or CPAP, any leak in the respiratory system may produce ventilatory autocyced. In the absence of PEEP or CPAP, however, a cuff leak will not alter the end-expiratory pressure, which is already equal to atmospheric pressure, and thus will not be sufficient to cause autocyced. Alternatively, a gas leak secondary to chest tube suction in the presence of an ipsilateral bronchopleural fistula is capable of causing proximal airway pressure to fall below atmospheric pressure and thus may induce autocyced without PEEP or CPAP. If the ΔP is set at too small a value, the likelihood that autocyced will occur is increased. In our patient, the combination of a large cuff leak and PEEP allowed proximal airway pressure to fall below the inspiratory threshold, and this resulted in autocyced. The problem was simply remedied by increasing the ΔP.

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