ventilation has profound hemodynamic consequences. The expiratory airflow obstruction in the ventilator circuit leads to excessive positive pressure during exhalation called the auto-PEEP effect (2). Therefore, the intrinsic PEEP likely greatly exceeded the extrinsic PEEP set on the ventilator. The mechanisms of the hemodynamic consequences of the auto-PEEP effect include reduced cardiac output from diminished venous return and markedly increased pulmonary vascular resistance due to compression of intralveolar vessels. Acute cor pulmonale is a consequence of this last phenomenon. Fluid loading in the setting of diminished left ventricular compliance from acute cor pulmonale can lead to pulmonary edema (3).

Occult auto-PEEP can have significant cardiopulmonary effects. It is an important cause of death among patients treated with mechanical positive pressure ventilation (4).

**Author Disclosure:** The author does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

**References**


**From the Author:**

Berlin proposes an alternative mechanism to explain refractory shock and pulmonary edema about the patient we have reported in the *Journal* (1). The patient was on mechanical ventilation with an obstruction of the expiratory ventilator limb. We agree that hemodynamic consequences of PEEPi with dynamic hyperinflation due to an increase of expiratory impedance can cause acute cor pulmonale. PEEPi increases right ventricular outflow impedance by increasing vascular pulmonary resistance, which can decrease the left ventricular compliance due to leftward shift and thus decrease cardiac output (2, 3). Also, its effect on intra-abdominal pressure may collapse inferior vena cava and impair the venous return to right heart and decrease cardiac output (2).

During the severe complication developed by our patient, airway pressures were high (peak pressure of 60 cm H₂O, and plateau pressure of 55 cm H₂O) (1). In this scenario, it is likely that PEEPi could have been increased and exceeded the extrinsic PEEP set on the ventilator, which was of 16 cm H₂O. Although the mechanism proposed by Berlin and colleagues could have caused acute cor pulmonale, we found that transthoracic echocardiography revealed a mean pulmonary arterial pressure of 12 mm Hg, right atrium and ventricle were of normal size and the ejection fraction was 65%. Furthermore, the intra-abdominal pressure was 8 mm Hg. These findings are not compatible with PEEPi as a cause of shock in this patient.

After changing the ventilator tubing set in our patient and correcting the severe acute hypercapnic acidosis, the patient improved, hemodynamic status stabilized, and the patient recovered from pulmonary failure (1). Therefore, we believe that our case represents an unusual example of acute severe hypercapnia with extreme acidemia, where hypercapnic acidosis is associated with development of severe shock and pulmonary edema. Because of the temporal relationship between hemodynamic parameters and values of CO₂, we believe that severe hypercapnia with extreme acidemia was the cause of shock and respiratory failure in our patient.

**Author Disclosure:** None of the authors has a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

**References**


**Vitamin C and Community-acquired Pneumonia**

*To the Editor:*

Waterer and colleagues considered that nutrition may affect the severity of community-acquired pneumonia, and suggested that further studies are needed to find out which patients with pneumonia are at highest risk of delayed mortality (1). I would like to point out that the possible role of vitamin C on pneumonia should be studied.

Several studies have documented reduced levels of vitamin C in patients with pneumonia (2, 3). Dozens of animal studies found that vitamin C protected against bacterial and viral infections, including pneumonia, indicating that the physiological effects of this vitamin are not limited to preventing overt deficiency (2). Therefore, we performed a systematic review on the effect of vitamin C in the prevention and treatment of pneumonia (4) and found three prophylactic trials and two treatment trials, reporting significant benefit of vitamin C against pneumonia. The randomized double-blind, placebo-controlled treatment trial by Hunt and coworkers (3) is particularly interesting.

Hunt and colleagues studied the effect of 0.2 g/day vitamin C on 66- to 94-year-old patients who were taken into the hospital because of pneumonia or acute exacerbation of chronic bronchitis (3). Vitamin C or placebo was administered in addition to the normal medication. Vitamin C significantly improved the “total respiratory score” in the most severely ill patients, but had no effect on the less ill patients (3). Moreover, there were six deaths during the trial—all among the most severely ill patients.
Five of the deaths occurred in the placebo group, but only one in the vitamin C group.

In a study using a combination of vitamins C and E, Nathens and coworkers (5) found no effect on the incidence of nosocomial pneumonia in critically ill surgical patients, but days of mechanical ventilation (−0.9; −0.6 to −1.2) were significantly reduced in the antioxidant group. Although this study is not specific to vitamin C, it suggests that antioxidants may affect pulmonary morbidity. More research on vitamin C and other antioxidants seems to be warranted.

**Author Disclosure:** The author does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

**References**


**Erratum:** Sleepiness, Quality of Life, and Sleep Maintenance in REM versus NREM Sleep-Disordered Breathing

To the Editor:

In our article published last year in the *Journal* (1), we omitted the following acknowledgments and sources of funding. The authors apologize for the omission.

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**References**