Acute Respiratory Failure induced by Mechanical Pulmonary Ventilation at a Peak Inspiratory Pressure of 40 cmH$_2$O

Kyoji TSUNO, Yuji SAKANASHI, Yasushi KISHI, Kenji URATA, Tadashi TANOU, Kanemitsu HIGASHI, Toshiyuki YANO, Hidenori TERASAKI and Tohru MORIOKA

The effects of high pressure mechanical pulmonary ventilation at a peak inspiratory pressure of 40 cmH$_2$O were studied on the lungs of healthy newborn pigs (14–21 days after birth). Forty percent oxygen in nitrogen was used for ventilation to prevent oxygen intoxication. The control group (6 pigs) was ventilated for 48 hours at a peak inspiratory pressure less than 18 cmH$_2$O and a PEEP of 3–5 cmH$_2$O with a normal tidal volume, and a respiratory rate of 20 times/min. The control group showed few deleterious changes in the lungs for 48 hours. Eleven newborn pigs were ventilated at a peak inspiratory pressure of 40 cmH$_2$O with a PEEP of 3–5 cmH$_2$O and a respiratory rate of 20 times/min. To avoid respiratory alkalosis, a dead space was placed in the respiratory circuit, and normocarbia was maintained by adjusting dead space volume. In all cases in the latter group, severe pulmonary impairments, such as abnormal chest roentgenograms, hypoxemia, decreased total static lung compliance, high incidence of pneumothorax, congestive atelectasis, and increased lung weight were found within 48 hours of ventilation. When the pulmonary impairments became manifest, 6 of the 11 newborn pigs were switched to the conventional medical and ventilatory therapies for 3–6 days. However, all of them became ventilator dependent, and severe lung pathology was found at autopsy. These pulmonary insults by high pressure mechanical pulmonary ventilation could be occurring not infrequently in the respiratory management of patients with respiratory failure.

(Key words: mechanical pulmonary ventilation, high peak inspiratory pressure, acute respiratory failure, barotrauma)

(Tsuno K, Sakanashi Y, Kishi Y et al.: Acute respiratory failure induced by mechanical pulmonary ventilation at a peak inspiratory pressure of 40 cmH$_2$O. J Anesth 2: 176–183, 1988)

Soon after mechanical pulmonary ventilation (MV) first became popular in the clinical management of patients with respiratory failure caused by poliomyelitis or other neuromuscular disorders, then by pulmonary diseases, the side effects of MV began to

Department of Anesthesiology, Kumamoto University Medical School, Kumamoto, Japan
Address reprint requests to Dr. Tsuno: Department of Anesthesiology, Kumamoto University Medical School, 1-1-1 Honjiyo, Kumamoto, 860 Japan


attention. There were debates concerning the expression “Respirator Lung”, which implied that MV caused pulmonary damage. Nash et al.\textsuperscript{1} performed MV on healthy goats at a peak inspiratory pressure (PIP) of 13 cmH$_2$O for 3 to 4 days, and concluded “The Respirator Lung was a misnomer”. Indeed, MV usually does not cause severe pulmonary complications on patients with healthy compliant lungs, as was the case in poliomyelitis and patients undergoing general anesthesia
for minor surgery. In these cases, a low PIP, as Nash used in his experiment, is enough to get adequate pulmonary gas exchange.

However, a high PIP is often necessary during MV for patients with respiratory distress syndrome (RDS) with low compliant lungs. The pulmonary effects of long-term MV with a high PIP have not been studied in detail. Therefore, we have studied the effects of high pressure MV at a PIP of 40 cmH₂O for 48 hours on the healthy lungs of newborn pigs.

**Methods**

Seventeen newborn pigs, 14 to 21 days after birth, weighing 3.72 ± 0.30 kg, were intubated orotracheally under sodium pentobarbital anesthesia and placed on a mechanical ventilator. The anesthesia was switched to 1–2% halothane in nitrous oxide and oxygen for the following operation. One small catheter was inserted into the left external jugular vein for IV infusion, and another into the right external jugular vein for continuous heparin infusion. The right carotid artery was also cannulated for blood pressure monitoring and blood sampling. Two T-shaped silicone chest tubes were installed into the bilateral chest cavities, and they were continuously drained with a negative pressure of cmH₂O to prevent sudden death from tension pneumothorax. A urinary catheter was placed into the bladder transabdominally. Tracheostomy was done, and the orotracheal tube was replaced with a spiral tracheostomy tube.

After all was finished, the pigs were put in a prone position. Anesthesia and paralization were maintained throughout the experiments with sodium pentobarbital and pancuronium bromide. The pigs were mechanically ventilated by a Newport 100E ventilator (NMI, USA) with a tidal volume (VT) of 13 ml/kg, a respiratory rate (RR) of 20 times/min, and a PIP less than 18 cmH₂O with a positive end-expiratory pressure (PEEP) of 3–5 cmH₂O. A humidified gas mixture of 40% oxygen in nitrogen, warmed to 38°C, was used throughout the experiments. After 2 hours of MV with the above mode, arterial blood gases, total static lung compliance (TSLC), and a chest roentgenogram were taken as control values.

After the control values were taken, the pigs were assigned to either control group A or experimental group B. Group A (n = 6) was ventilated for 48 hours as in the control period described above. Group B (n = 11) was ventilated 20 times/min with a PIP elevated to 40 cmH₂O by increasing VT. A PEEP of 3–5 cmH₂O was the same as in Group A. An adjustable dead space tube was placed in the respiratory circuit to avoid respiratory alkalosis due to hyperventilation. High pressure mechanical ventilation (HPMV) was performed for 48 hours or until the PaO₂ fell to less than 60 mmHg at an FiO₂ 0.4. Then, 5 pigs were sacrificed for autopsy (Group B-1). In the remaining 6 pigs (Group B-2), when they reached the terminal criteria as in Group B-1, conventional MV for the treatment of acute respiratory failure (ARF) was started and continued for 3–6 days. During the management of conventional MV, the respiratory dead space was removed, and adequate VT, PEEP, FiO₂, and other medical efforts were taken for life-saving.

Arterial blood gases were analyzed every hour and TSLC was measured every four hours. TSLC was measured by stepwise inflation of the lungs with air, with an increment of 25 ml by using a large syringe, until the maximal intratracheal pressure reached 20 cmH₂O. The intratracheal pressure was measured with a pressure transducer (Gould P23 ID, USA) at the proximal end of the endotracheal tube. When the base excess fell under −5 mEq/L, NaHCO₃ was given to correct the arterial pH. Half saline (2.5% dextrose and 0.45% NaCl) was continuously given intravenously at the rate of 5 ml/kg/h. KCl was also given intermittently to maintain the serum K⁺ within 3.5–4.5 mEq/L. Massive thrombosis was frequently found in the superior and inferior caval veins of pigs mechanically ventilated with a high PIP in a preliminary experiment, but this problem was avoided by the intravenous administration of heparin. Therefore, heparin