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Extracorporeal lung assist for patient with hypercapnia due to status asthmaticus

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Abstract. A 62-year-old female was brought to our emergency room in status asthmatics. She needed mechanical ventilation because of hypercapnia. However, effective mechanical ventilation was difficult because of severe airflow obstruction. Instead of conventional mechanical ventilation, we used extracorporeal lung assist (ECLA) to prevent barotrauma and lung tissue damage, and to reduce the doses of sedatives and muscle relaxants needed.

Key words: Extracorporeal lung assist (ECLA) – Status asthmatics – Barotrauma

ECLA and low-frequency positive pressure ventilation with extracorporeal CO_2 removal (LFPPV-ECCO$_2$R) have been given considerable attention as new methods of treatment for adult respiratory distress syndrome (ARDS) and exacerbations of chronic respiratory disease. Morioka et al. [1] and Gattinoni et al. [3] have suggested that ECLA and LFPPV-ECCO$_2$R are better than conventional respiratory support with mechanical ventilation to prevent futher lung deterioration, the so called "respirator lung", and the development of barotrauma.

We have used ECLA for a patient with status asthmaticus to reduce the risk of complications from mechanical ventilation.

Case report

A 62-year-old female was brought to our emergency room in status asthmaticus. She was unconscious with hypercapnia. Despite initial agressive treatment with intravenous aminophylline (1 mg/kg/h), nebulised salbutamol and hydrocortisone (1000 mg), she required mechanical ventilation for acute hypercapnia (pH = 7.168, $PaCO_2$ = 63.8 mmHg, PaO_2 = 395 mmHg, FiO_2 of 1.0; immediately after mechanical ventilation with a Servo 900 C) was commenced. Her bronchospasm did not improve after two days of aggressive treatment with bronchodilators and hydrocortisone, and her airway pressure remained about 65 cmH_2O on a ventilator (Tidal volume of 12 ml/kg, respiratory rate of 12/min, I/E ratio of 1:2). She needed large doses of diazepam (10 mg/h) and pancuronium bromide (2 mg/h) to prevent her breathing spontaneously. ECLA was begun in order to prevent barotrauma and further lung damage. The axillary vein and sapheno-femoral junction were surgically prepared, and cannulated with 16 Fr. silicone catheters and connected to the ECLA circuit. The silicone membrane artificial lung with a surface area of 0.8 m^2 (Senko Ika, Tokyo) was incorporated into the ECLA circuit. Heparin was given to maintain activated coagulation time at about 200 s. About 1 h after the start of ECLA (with a bypass blood flow of 800 ml/min and 40% oxygen flow of 4 l/min), she was settled on the ventilator without any sedation. Tidal volume, rate and mode setting of the ventilator were successfully changed from 650 ml and 12/min of control mode to 400 ml and 2/min IMV mode respectively. Her spontaneous breathing during IMV was 8–10 breath/min. Her $PaCO_2$ fell from 59.8 mmHg to 46.8 mmHg 2 h after the start of ECLA and pH value returned to normal. At the same time, wheezing disappeared and she could breathe easily (vital capacity almost 1000 ml). Figure 1 demonstrates the temporal changes in PaO_2, $PaCO_2$ and airway pressure, and also shows the disappearing of wheezing. Twenty hours after starting ECLA, the ventilator was disconnected, but the patient remained intubated to facilitate tracheal suctioning. Weaning from ECLA after 24 h occurred without complication. One day later the patient was discharged from the ICU.

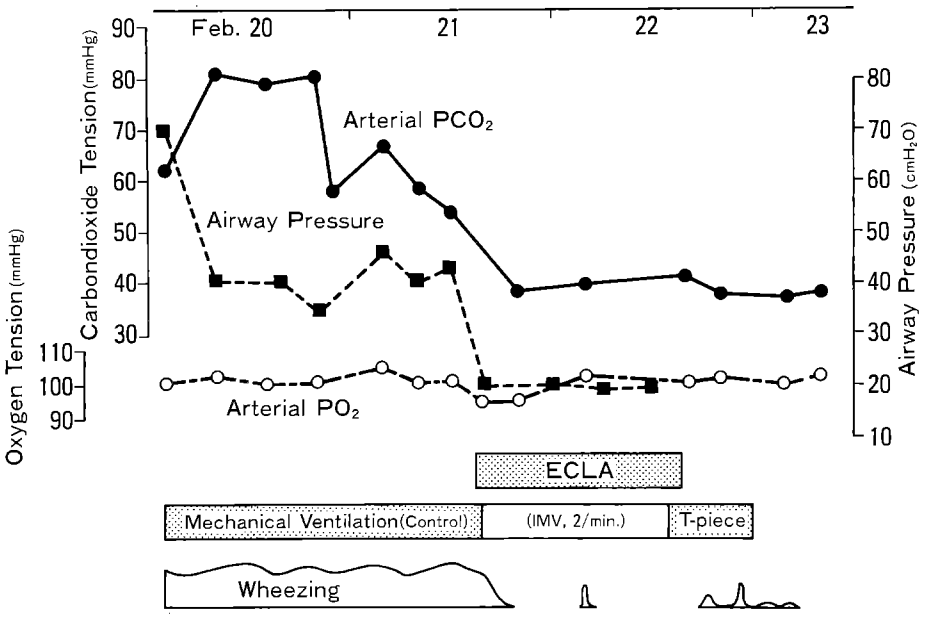

Fig. 1. Arterial Oxygen tension, arterial carbondioxied tension and peak airway pressure during mechanical ventilation and ECLA

Discussion

Although artificial ventilation is a useful and effective tool to correct hypoxaemia and/or hypercapnia, and to reduce work of respiratory muscles in patients with respiratory failure, it is not a direct treatment for the lung tissue itself. It is well known that the long term use of positive pressure ventilation for any type of pulmonary failure damages the lung tissue and may cause a deteriorative pulmonary function [4].

In patients with status asthmatics who have progressive hypercapnia and/or hypoxaemia, intubation and mechanical ventilation should be instituted [5]. The patients in status asthmaticus who requires mechanical ventilation usually is hyperinflated and has an extremely high level of airway pressure [5]. Both hyperinflation and high airway pressure are believed to increase the risk of barotrauma that is a potentially lethal complication [3].

In the patient reported, mechanical ventilation was instituted because progressive hypercapnia could not be reversed by bronchodilators and steroid. During mechanical ventilation, despite generous use of sedatives and muscle relaxant, it was very difficult to find adequate ventilator setting as her expiration was extremly prolonged. Twentyfour hours after the start of mechanical ventilation, her $PaCO_2$ and airway pressure were still high, and we were reluctant to continue mechanical ventilation futher to protect the lung tissue from barotrauma. Our patient did not require any sedative and muscle relaxant after the start of ECLA.

The disappearance of wheezing, immediately after the start of ECLA could not be explained clearly. We did not measure the volume of CO_2 eliminated from ECLA. However, the disappearance of wheezing must be related to the reduction of ventilatory volume.

We conclude that ECLA offers an excellent method of treating hypercapnia due to severe airflow obstruction in status asthmaticus.

References

1. Morioka T (1985) Respiratory care without endotracheal intubation – An aim of ECLA. Jpn J Throrac Dis 23:271
2. Gattinoni L, Agoston A, Pesenti A, Pelizzol A, Rossi GP, Langer M, Vesconi S, Uziel L, Fox U, Longoni F, Kolobow T, Damia G (1980) Treatment of acute respiratory failure with low frequency positive pressure ventilation with extracorporeal removal of CO_2. Lancet II:292
3. Grunfeld C (1978) Status asthmatics, Mechanical ventilation, and pneumothorax. JAMA 239:613
4. Lake KB (1984) Adult respiratory disstres syndrome (High permeability pulmonary edema). In: Burton GG, Hodgkkin JE (eds) Respiratory care. Lippincott, Philadelphia, pp 854–856
5. Westerman D, Benatar S, Potgieter P, Ferguson AD (1979) Identification of the high risk asthmatic patient – experience with patients undergoing ventilation for status asthmaticus. Am J Med 66:565

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