

## CASE REPORT

# First experiences in the Netherlands with a new single catheter-based veno-venous extracorporeal carbon dioxide removal system

AHM Knook<sup>1</sup>, R Baak<sup>2</sup>, J Epker<sup>3</sup>, J Bakker<sup>3</sup>

<sup>1</sup> Department of Intensive Care, De Gelderse Vallei Hospital, Ede, The Netherlands

<sup>2</sup> Department of Intensive Care, Haga Hospital, the Hague, The Netherlands

<sup>3</sup> Department of Intensive Care, Erasmus Medical Center Rotterdam, Rotterdam, The Netherlands

**Abstract** - A 34-year-old woman suffering from severe systemic lupus erythematosus was admitted to the ICU with respiratory failure due to a pneumocystis jirovecii infection resulting in severe hypercapnia which could not be controlled with conventional strategies. A new single catheter-based CO<sub>2</sub> removal system was initiated in order to control severe hypercapnia. The case and relevant literature are discussed.

**Keywords** - Artificial respiration, Pulmonary fibrosis, Extracorporeal membrane oxygenation/instrumentation, heparin

## Introduction

Extracorporeal carbon dioxide (CO<sub>2</sub>) removal has been used in the treatment of patients with acute respiratory distress syndrome (ARDS). As a consequence, tidal volumes and plateau pressure could be reduced in these patients [1,2].

Previous reports have demonstrated the safety and feasibility of a pumpless arteriovenous CO<sub>2</sub> removal system [2,3]. However, this system requires cannulation of the femoral artery and vein with large bore catheters and may induce a considerable left-to-right shunt [4-7].

Recently, a new technique has been launched, allowing a pump-driven system with a veno-venous approach using a standard 13 French single double lumen CVVH-catheter. Results of an experimental study in adult sheep were promising [8].

We describe a case in which veno-venous CO<sub>2</sub> removal was used to control PaCO<sub>2</sub> in a patient suffering from severe systemic lupus erythematosus (SLE) and a pneumocystis jirovecii infection resulting in severe hypercapnia which could not be controlled with conventional strategies.

## Case Report

A 34 year old woman was admitted to the Internal Medicine department of Erasmus Medical Center Rotterdam with fever. Her past medical history revealed SLE, diagnosed at the age of 26, leprosy, genital herpes, hypertension and steroid myopathy. Cerebral manifestations had been present since Autumn 2008, for which she had been treated with cyclofosamide. Because of a high suspicion of a pneumocystis infection, she was started on trimethoprim/sulfamethoxazole 1920 mg tid iv and prednisolone 40 mg bid iv therapy. Two days after admission, she developed

respiratory failure requiring intubation and mechanical ventilation. Broncho-alveolar lavage (BAL) showed pneumocystis jirovecii and computerized tomography of the chest revealed extensive bilateral infiltrates with hardly any aerated areas left. High positive end-expiratory pressures were required to maintain adequate oxygenation. Peak inspiratory pressure rose to 52 cmH<sub>2</sub>O.

In the first 3 weeks, her clinical condition improved with a PaO<sub>2</sub>/FiO<sub>2</sub> ratio increasing from 80 up to 230, and declining peak end-expiratory pressures (from 19 cm H<sub>2</sub>O to 8 cm H<sub>2</sub>O). A percutaneous tracheostomy was performed to facilitate weaning from mechanical ventilation.

On day 37, her condition suddenly deteriorated due to a sepsis with positive blood cultures for enterococcus. Despite treatment with vancomycin, her respiratory condition also deteriorated with increasing requirements for oxygen. The chest X-ray showed increasing bilateral infiltrates, suggestive of an intercurrent pneumonia. Furthermore, possible signs of pulmonary fibrosis were present.

At this time it was felt that despite the severe underlying disease, the clinical condition might still be partially reversible.

High inspiratory peak pressures (up to 52 cmH<sub>2</sub>O) were needed to control hypercapnia. Permissive hypercapnia was used, however, despite all measures taken, pCO<sub>2</sub> levels rose to high levels (11 kPa) and oxygenation and haemodynamic problems were encountered when trying to decrease the pCO<sub>2</sub> level. Full extracorporeal membrane oxygenation was not an option due to marginal arterial access in this patient.

Because of failure to control the progressive hypercapnia while limiting inspiratory pressures, the patient was placed on a extracorporeal veno-venous carbon dioxide removal system.

This veno-venous carbon dioxide removal system (Decap®, Hemodec srl, Salerno, Italy) is a low-flow system using a percutaneous single venous access with a double lumen catheter. The machine has two circuits. In the first circuit, blood flows to the decapneizator, containing a CO<sub>2</sub> filter. In this circuit, oxygen

## Correspondence

R Baak

E-mail: remonbaak@hotmail.com

is used as a sweep gas at a flow rate of 5 litres per minute. This device allows a maximum oxygen flow rate of 10 l/min resulting in 8-10% oxygen increase in the patient's blood. The second circuit is a pre-dilution circuit removing  $\text{CO}_2$  from the plasmatic water, improving the decapneizator efficiency (Figure 1). The maximum blood flow is 350-400 ml/min allowing maximal  $\text{CO}_2$  extraction of approximately 30%. In the present case, the blood flow averaged 300 ml/min. Heparin was needed as a coagulant in order to prevent thrombus formation. In order to reach adequate anticoagulation, an activated partial thromboplastin time (APTT) between 60-80 seconds is required. Figure 2 shows the  $\text{PaCO}_2$ , pH and peak pressures after starting the veno-venous removal system. The inspiratory peak pressures could be decreased to from 52  $\text{cmH}_2\text{O}$  to 37  $\text{cmH}_2\text{O}$ .

At day 41, an accidental bolus of intravenous heparin was administered, leading to heparin overshoot with an APTT over 240 seconds. Within a couple of hours, the patient suffered an uncontrolled bleeding from the groin at the insertion site of the double-lumen catheter. After multiple transfusions of erythrocyte concentration and fresh frozen plasma, the decision was made to remove the catheter in order to control the bleeding more optimally with a pressure-device. After a few hours of fluid resuscitation, the patient's condition stabilized. Considering the marginal vascular access in this patient, we decided not to restart the  $\text{CO}_2$  removal therapy at this point.

A few days after termination of the  $\text{CO}_2$  removal therapy, again a progressive hypercapnia developed. The diagnosis of progressive pulmonary fibrosis was made. In agreement with her husband and her family, treatment was stopped.

## Discussion

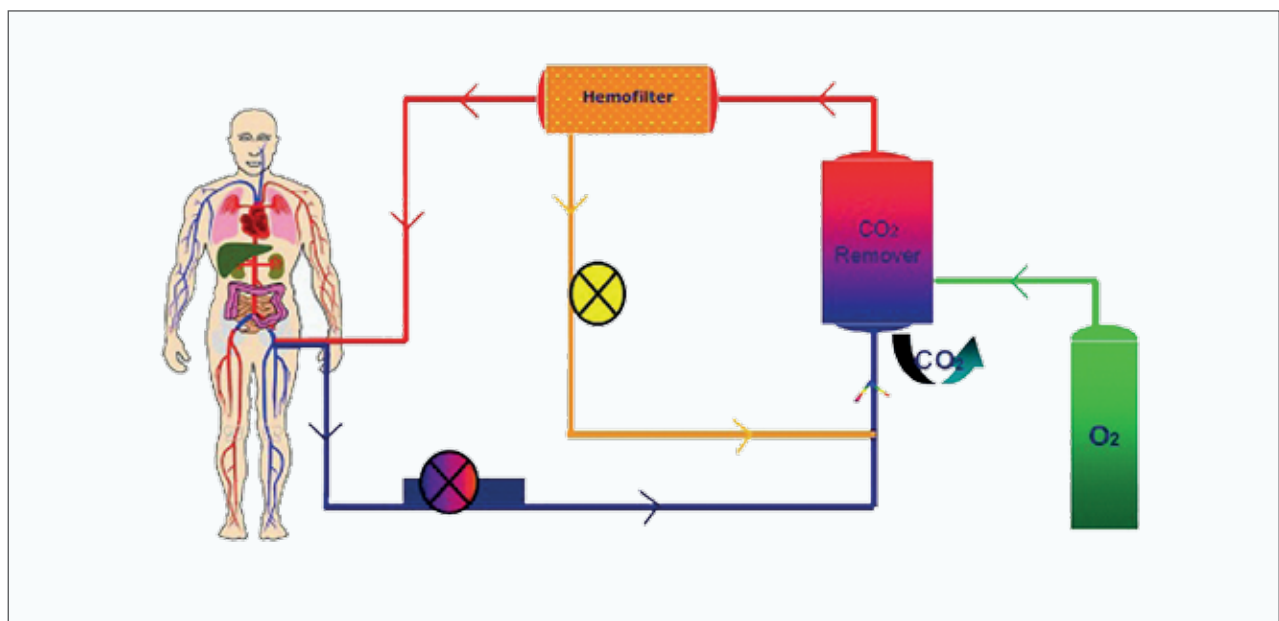
Our case describes a young female suffering from pneumocystis infection leading to severe pulmonary fibrosis. Conventional therapeutic strategies were not effective, resulting in very high arterial  $\text{PaCO}_2$  with corresponding respiratory acidosis and low, but not yet life-threatening  $\text{PaO}_2$  levels. When  $\text{PaCO}_2$  rose to levels as high as 11 kPa (82,5 mmHg), and oxygenation deteriorated further, we decided to apply an extracorporeal gas exchange device.

It was felt that extracorporeal membrane oxygenation or NovaLung™ in this patient, requiring optimal arterial access was not an option due to the very poor arterial status. For this reason, a new veno-venous  $\text{CO}_2$  removal system was the only available option. This new technique is attractive because of its simplicity permitting a single venous access. The rationale for using this device was primarily to reduce  $\text{PaCO}_2$  levels and thus reduce inspiratory peak pressures. At the time of starting the treatment with Decap, the patient had already required high peak pressures for more than a week and  $\text{PaCO}_2$  was progressively rising.

Several studies have shown the safety and feasibility of using pumpless  $\text{CO}_2$  removal systems in patients with acute lung injury exhibiting hypercapnia and respiratory acidosis [3,4,9-12]. Systems used in these studies were either arterio-venous or veno-venous systems. An experimental study in sheep has demonstrated that a significant reduction in  $\text{PaCO}_2$  using the Decap system can be achieved [8].

In our case, suboptimal anticoagulation led to clotting of the system. Due to an accidental bolus of heparin, a severe bleeding of the groin at the insertion site occurred requiring multiple

**Figure 1.** Schematic overview of the DECAP system



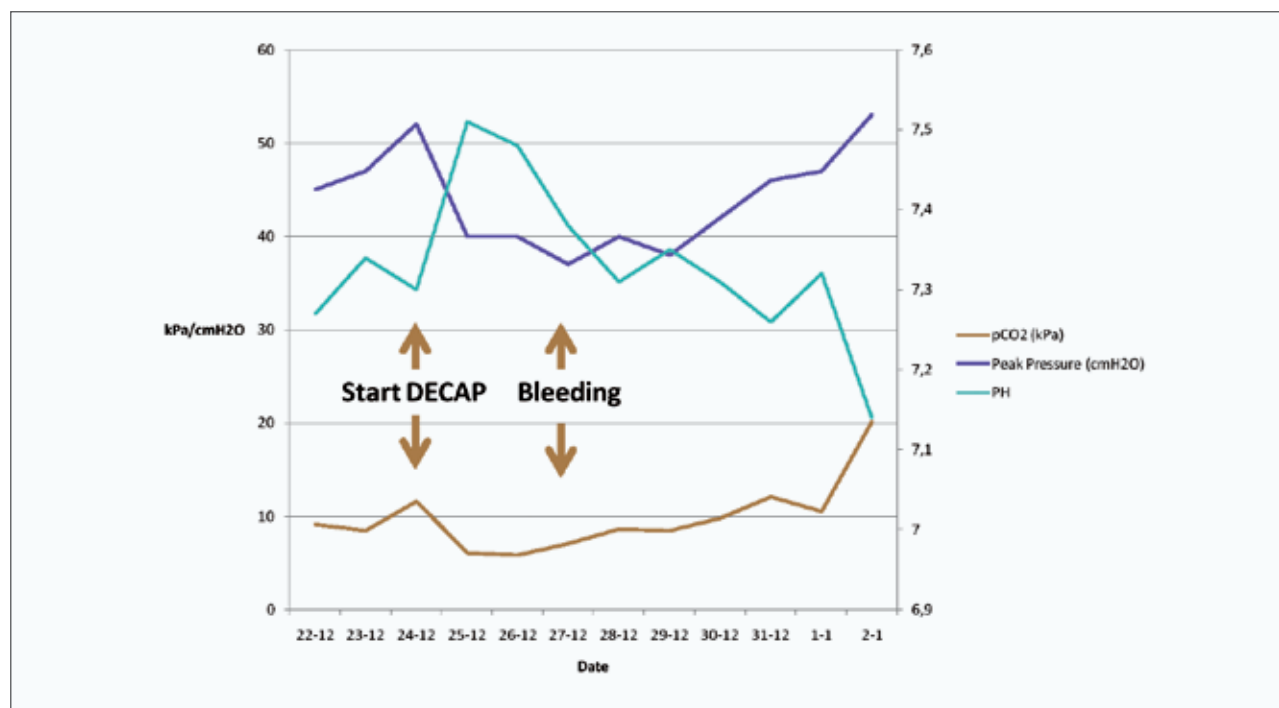
transfusions. It was necessary to terminate the CO<sub>2</sub> removal and remove the venous catheter in order to control this bleeding.

After removal of the catheter, the PaCO<sub>2</sub> level remained acceptable for three more days, without the need to increase ventilatory pressures to control PaCO<sub>2</sub>. For this, we do not have a satisfactory explanation. Unfortunately, our patient died five days after discontinuing Decap due to progressive respiratory failure caused by severe pulmonary fibrosis.

### Conclusion

We show that this new veno-venous CO<sub>2</sub> removal technique is an effective method in controlling PaCO<sub>2</sub> in a patient with severe respiratory failure due to pneumocystis infection, complicated by pulmonary fibrosis. More experience and clinical trials are needed to evaluate this technique.

**Figure 2.** PaCO<sub>2</sub>, pH and peak pressures at start, during and after cessation of the treatment.



### References

1. Tao, W, et al. Significant reduction in minute ventilation and peak inspiratory pressures with arteriovenous CO<sub>2</sub> removal during severe respiratory failure. *Crit Care Med*, 1997. 25: p. 689-95.
2. Zwischenberger, JB, et al. Percutaneous extracorporeal arteriovenous CO<sub>2</sub> removal for severe respiratory failure. *Ann Thorac Surg*, 1999. 68: p. 181-7.
3. Liebold, A, et al. Pumpless extracorporeal lung assist - experience with the first 20 cases. *Eur J Cardiothorac Surg*, 2000. 17: p. 608-13.
4. Scott, L.K. et al. Extracorporeal carbon dioxide removal to control arterial pH and PaCO<sub>2</sub> in a heart-beating donor with acute lung injury. *Transplantation*, 2003. 76: p. 1630-2.
5. McKinlay, J, et al. Pre-emptive Novalung-assisted carbon dioxide removal in a patient with chest, head and abdominal injury. *Anaesthesia*, 2008. 63: p. 767-70.
6. Fischer, S, et al. Bridge to lung transplantation with the extracorporeal membrane ventilator Novalung in the veno-venous mode: the initial Hannover experience. *Asaio J*, 2007. 53: p. 168-70.
7. Bein, T, et al. [Pumpless extracorporeal lung assist using arterio-venous shunt in severe ARDS. Experience with 30 cases]. *Anaesthesist*, 2004. 53: p. 813-9.
8. Livigni, S, et al. Efficacy and safety of a low-flow veno-venous carbon dioxide removal device: results of an experimental study in adult sheep. *Crit Care*, 2006. 10: p. R151.
9. Ruettimann, U, et al. Management of acute respiratory distress syndrome using pumpless extracorporeal lung assist. *Can J Anaesth*, 2006. 53: p. 101-5.
10. Liebold, A, et al. Pumpless extracorporeal lung assist using an arterio-venous shunt. Applications and limitations. *Minerva Anestesiol*, 2002. 68: p. 387-91.
11. Bein, T, et al. [ARDS and severe brain injury. Therapeutic strategies in conflict]. *Anaesthesist*, 2002. 51: p. 552-6.
12. Reng, M, et al. Pumpless extracorporeal lung assist and adult respiratory distress syndrome. *Lancet*, 2000. 356: p. 219-20.