Extracorporeal carbon dioxide removal to “protect” the lung

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ABSTRACT

The case histories are presented of three adults who had severe hypercapnic acidosis despite mechanical ventilation with what were considered to be injurious tidal volumes and airway pressures. The use of a percutaneously inserted arteriovenous extracorporeal carbon dioxide removal (AV-ECCO₂R) device facilitated a dramatic reduction in the amount of ventilatory support required, achieving a “lung-protective” level. Two patients survived to hospital discharge. One patient died after it became apparent that her late-stage interstitial lung disease was unresponsive to immunosuppression. AV-ECCO₂R may be a useful strategy in facilitating lung-protective ventilation.

Ventilator-associated lung injury caused by the administration of excessive volume and/or inspiratory pressure is associated with increased mortality.^{1,2} Ventilatory support sufficient to achieve acceptable rather than normal blood gas tensions may reduce mortality. The use of extracorporeal gas exchange (ECGE) as an adjunct to mechanical ventilation may facilitate this. Hence, the results of the completed randomised trial of (conventional) extracorporeal membrane oxygenation (ECMO) in severe respiratory failure (CESAR, ISRCTN47279827) are eagerly awaited.

ECMO requires complex and potentially hazardous technology. The large-bore vascular access, anticoagulation, high blood flow and technical support required are significant. Sudden device failure can be catastrophic. Recently, an arteriovenous system (iLA; Novalung, Hechingen, Germany) has been developed to remove primarily carbon dioxide (AV-ECCO₂R). Blood flows passively through the device at 0.8–1.0 l/min, dependent on the pressure gradient between the femoral artery and the vein to which vascular access is achieved percutaneously. Blood passes across a network of heparin-bonded hollow fibres across which gas exchange occurs with a counterflow of oxygen. The device has not been subject to investigation in a randomised clinical trial. We describe its application in three patients with potentially reversible type II respiratory failure in whom we considered that required mechanical ventilation was prohibitively injurious.

CASE REPORTS

Patient A was a 26-year-old woman presenting with established and rapidly progressive interstitial lung disease proven by surgical open lung biopsy and possibly caused by a collagen vascular disease or extrinsic allergic alveolitis. Postoperatively, despite ventilation with high inflation pressures

and a rapid respiratory rate, a significant hypercapnic acidosis persisted. AV-ECCO₂R was instituted, permitting reduced ventilatory support (table 1). Despite appropriate immunosuppression, the patient failed to improve clinically. As her lung disease was considered to be irreversible and she was unsuitable for lung transplantation, support was withdrawn. She died 37 days after admission.

Patient B was a 70-year-old man who required mechanical ventilation for acute severe dyspnoea 8 days after right upper and middle lobectomy for non-small cell lung cancer. CT revealed pulmonary thromboemboli, and airspace shadowing typical of acute lung injury in the non-operative lung. Severe airways disease and high minute volume requirements resulted in AV-ECCO₂R being initiated (table 1). The patient improved over the following 7 days and AV-ECCO₂R was withdrawn. Successful weaning from mechanical ventilation occurred 8 days later.

Patient C was 33 years old and presented with methicillin-sensitive *Staphylococcus aureus* (Panton–Valentine–Leucocidin toxin secreting) necrotising pneumonia. At referral she had been ventilated for 24 days. AV-ECCO₂R was instituted to facilitate reductions in airway pressure and tidal volume and to control her hypercapnic acidaemia (table 1). Clinical progress permitted the removal of the device after 15 days, and mechanical ventilation after 33 days.

DISCUSSION

AV-ECCO₂R has been described as salvage therapy, bridging patients to transplantation or even recovery.^{3,4} Our small series quantifies the extent to which mechanical ventilation can be reduced in a heterogeneous group of patients. In all three cases AV-ECCO₂R allowed dramatic and clinically significant reductions in tidal and minute volumes and peak inspiratory pressure, whilst also reducing arterial CO₂ tensions. The impact upon outcome is unclear, but all three patients were at high risk of death judged by illness severity scores.

AV-ECCO₂R has a substantial effect on CO₂ clearance. The total CO₂ content of hypercapnic blood is around 50–70 ml/dl. Even clearance of 25% equates to 150 ml/min, relative to the adult CO₂ production of approximately 200 ml/min. In contrast, AV-ECCO₂R had little effect on arterial oxygenation. The shape of the oxygen dissociation curve (which plateaus) and the solubility of oxygen in blood (relatively low) means the capacity of arterial blood to carry ever greater amounts of oxygen is limited. Moreover, any device outflow that subsequently passes through a functioning alveolar unit will gain no incremental oxygen since

Table 1 Ventilatory and oxygenation parameters for patients before and after AV-ECCO₂R

	Patient A		Patient B		Patient C	
	Pre	Post	Pre	Post	Pre	Post
Oxygenation parameters						
Inspired oxygen fraction	0.3	0.50	0.55	0.50	0.90	0.55*
PEEP, cm H ₂ O	1	6	8	10	10	10
Arterial pO ₂ , kPa	10.7	18.1	10.3	9.7	13.1	8.6*
Alveolar–arterial difference, kPa	1.7	17.8	28.6	24.8	55.4	30.8*
Ventilatory parameters						
Plateau inspiratory pressure, cm H ₂ O	39	29	27	22	36	28
PEEP, cm H ₂ O	1	6	8	10	10	10
Tidal volume, ml	179	111	474	316	362	260
Tidal volume, ml/kg	4.0	2.6	6.2	4.2	6.0	4.3
Respiratory rate, per min	23	12	18	17	16	14
Minute volume, litres	4.2	3.0	8.8	6.6	5.6	3.4
Arterial pCO ₂ , kPa	9.4	6.8	8.4	7.9	13.1	8.0
pH	7.27	7.43	7.22	7.23	7.18	7.37

*Inhaled nitric oxide was commenced simultaneously for right ventricular dysfunction and concern about the ability of the patient to cope with a litre arteriovenous shunt following institution of arteriovenous extracorporeal carbon dioxide removal (AV-ECCO₂R).

Values are those recorded by the nursing staff before (Pre) and after (Post) implantation of the AV-ECCO₂R.

Tidal volumes are also expressed relative to actual body weight. The alveolar–arterial difference was calculated from the alveolar gas equation presuming a respiratory quotient of 0.8.

PEEP, positive end-expiratory pressure.

pulmonary venular blood is usually fully saturated irrespective of pulmonary arterial saturation. Typically, two-thirds of the cardiac output must pass through a device to increase prepulmonary saturations sufficiently to improve systemic arterial oxygenation; the situation that pertains in conventional ECMO.

A 10% incidence of distal ischaemic complications has been described.³ We detected a pseudoaneurysm at the arterial cannulation site in one patient. This was successfully obliterated with a fibrin injection at its neck. Venovenous pumped ECGE obviates the need for arterial access.

What is the potential role for ECCO₂R in clinical practice? As yet an unproven therapy, it may permit the reduction of the potentially negative aspects of mechanical ventilation. This may safely bridge patients with ever more extreme but reversible disturbances in gas exchange to recovery. Moreover, it is not known whether minimisation of ventilator-induced lung injury can also influence longer term functional outcome for patients, although this is possible. Finally, and probably most intriguing for respiratory physicians, is the technical possibility to employ short-term ECCO₂R in severely hypercarbic patients with reversible exacerbations of very advanced lung disease and in whom non-invasive ventilation is ineffective and invasive mechanical ventilation is deemed undesirable. Whether this impacts upon long-term quality of life is unknown. There is a clear need for randomised evaluations of the efficacy of AV-ECCO₂R.

Competing interests: None.

Patient consent: Obtained.

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