

Short reports

Treatment of diuretic resistant cor pulmonale by continuous arteriovenous haemofiltration

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ABSTRACT A woman with diuretic resistant cor pulmonale had 24 litres of fluid removed over four days by arteriovenous haemofiltration. She was discharged one week later and has remained ambulant and independent for one year.

Diuretics usually improve the disabling symptoms of fluid retention in cor pulmonale. A few patients are refractory to treatment and may need protracted hospital care. We used continuous arteriovenous haemofiltration to remove more than 24 litres of excess body water from a patient who was bedfast with oedema. Ultrafiltration led to early discharge from hospital, and the patient has remained well for a further 12 months.

Case report

A woman of 64 years who smoked 30 cigarettes daily presented in February 1983 with headaches, breathlessness, cyanosis, and ankle swelling. Her body weight was 67.6 kg. Her haemoglobin concentration was 19.5 g/dl and a chest radiograph showed increased heart size (cardiothoracic ratio 0.64) and enlarged pulmonary arteries. Arterial oxygen tension (PaO₂) was 6.6 kPa and arterial carbon dioxide tension (PaCO₂) 7.27 kPa. Secondary polycythaemia was confirmed by an increased red cell mass (47.5 (normal range 20-30) ml/kg) and normal plasma volume (37 (normal range 36-50) ml/kg). After she had had venesection to remove 8 units of blood over two months and had stopped smoking she reported an improvement in effort tolerance that enabled her to walk 50-100 m, and her peak expiratory flow rate increased from 80 to 130 l/min.

In 1987 her body weight rose progressively to 93 kg despite increases in maintenance diuretics to 160 mg frusemide, 100 mg spironolactone, and 5 mg metolazone daily. In March 1987 she was admitted with massive truncal and dependent oedema and was unable to get out of bed. The plasma urea concentration was raised (14 mmol/l), but creatinine was normal (120 μmol/l). FEV₁ was 0.4 (predicted 1.64) l, FVC 1.2 (predicted 2.26) l, and transfer coefficient (K_{co}) 0.87 (predicted 1.6) mmol/min⁻¹ kPa⁻¹ l⁻¹. After three weeks of fluid restriction, 24% oxygen, and intensive diuretic treatment (frusemide 250 mg intravenously twice daily and

metolazone 10 mg daily) there was no improvement, and her weight had increased further to 95 kg. As she was resistant to drug treatment fluid was removed by ultrafiltration.

HAEMODYNAMIC CHANGES

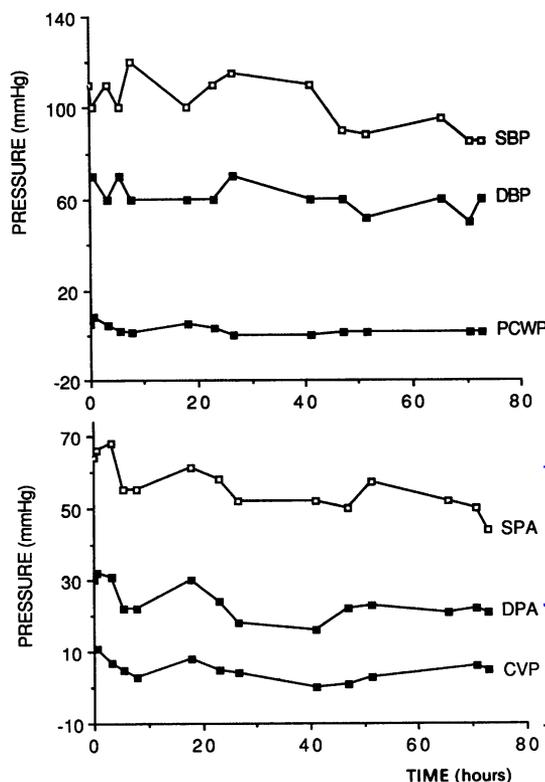


Fig 1 Changes in pulmonary and systemic pressure during arteriovenous haemofiltration. There are small but significant falls in systolic blood pressure ($SBP = 110.8 - 0.31 \times \text{time}$; $r = 0.73$, $F = 14.04$, $p < 0.005$), diastolic blood pressure ($DBP = 66.4 - 0.16 \times \text{time}$; $r = 0.66$, $F = 8.47$, $p < 0.025$), pulmonary artery systolic ($SPA = 62.2 - 0.20 \times \text{time}$; $r = 0.80$, $F = 20.70$, $p < 0.001$) and diastolic ($DPA = 27.2 - 0.11 \times \text{time}$; $r = 0.59$, $F = 6.35$, $p < 0.05$) pressures, and pulmonary capillary wedge pressure ($PCWP = 4.09 - 0.06 \times \text{time}$; $r = 0.62$, $F = 7.00$, $p < 0.025$). The trend in central venous pressure (CVP) was not significant.

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Femoral artery and vein catheters (8 FG) were connected to a Gambro FH55 haemofilter (a polyamide membrane of 0.6 m²). During ultrafiltration fluid lost was not replaced and heparin was infused to maintain clotting times between two and three times the normal. A Swan-Ganz catheter was inserted to record cardiac output (determined in triplicate by thermodilution), central venous pressure, pulmonary arterial pressure, and pulmonary capillary wedge pressure. Systemic blood pressure and blood gas tensions were measured repeatedly.

During 86 hours of continuous arteriovenous haemofiltration the median ultrafiltrate production rate was 290 (range 10–510) ml/h. Body weight fell by 24.5 kg to 70.5 kg, and measured total fluid loss was 24.2 litres. The results of haemodynamic and pulmonary function monitoring are shown in figures 1 and 2.

The haemofiltration procedure was well tolerated, and the only complication was a short period of oliguria and renal insufficiency resulting from intravascular volume depletion, which responded promptly to infusion of 2 units of plasma protein fraction. There was a striking improvement in

oedema and dyspnoea and she became independent of nursing care within three days. When she was discharged from hospital one week later (taking frusemide 120 mg daily) creatinine was 134 μ mol/l and urea 11.4 mmol/l. Spirometric values had improved (FEV₁ 0.75 l, FVC 2.0 l) and Kco had increased to 1.14 mmol min⁻¹ kPa⁻¹ l⁻¹. She was able to walk 50–100 m unaided. The fall in PaCO₂ from 10.34 to 8.93 kPa ($p < 0.025$) during haemofiltration was maintained (fig 2); six months later PaO₂ was 5.3 kPa, PaCO₂ 8.9 kPa, FEV₁ 0.5 l, FVC 1.4 l and Kco 0.87 mmol min⁻¹ kPa⁻¹ l⁻¹. Over the last 12 months she has been independent and has had minimal oedema while taking frusemide 250 mg and metolazone 5 mg a day. Two short readmissions were needed, one to readjust the diuretic regimen and one because of lobar pneumonia. Her weight has remained around 73 kg.

Discussion

Removal of excess body fluid by physical means is not new, but since the development of potent diuretics such methods are rarely required and their usefulness may be overlooked. This case shows that in certain circumstances removal of large quantities of body water and salts may be accomplished by physical means when standard treatment has failed.

Peritoneal dialysis has been used to remove fluid in heart failure^{1,2} and after myocardial infarction³ but is inefficient, carries a risk of peritoneal infection, and causes diaphragmatic splinting, thus impairing ventilation. Refractory congestive cardiac failure has been treated with machine driven venovenous haemofiltration in patients suffering from ischaemic heart disease or cardiomyopathy,⁴⁻⁷ with short term success but limited long term follow up. In all studies the maintenance dose of diuretic was reduced after ultrafiltration, but this phenomenon remains unexplained.

Of the available physical methods, arteriovenous haemofiltration is the simplest and cheapest, with a total equipment cost of around £80–£120. The technique needs no specialised machinery, but requires arterial and venous access. The haemodynamic changes were monitored closely but high rates of fluid removal were achieved with only small changes in blood pressure, gas exchange, and cardiac output (figs 1 and 2). Invasive monitoring is not normally required during haemofiltration and with more experience we suggest that this would not be necessary. Excessive fluid depletion can lead to temporary prerenal insufficiency, and ultrafiltration should be supervised carefully and stopped before all the oedema has resolved.

The prognosis for cor pulmonale remains poor, but our patient benefited greatly from ultrafiltration. She has lived at home for one year with a much improved quality of life and has enjoyed her first holiday for seven years. We suggest that fluid removal by continuous arteriovenous haemofiltration should be considered in cases of diuretic resistant cor pulmonale. It may reduce the need for protracted inpatient management, and should not be reserved purely for palliation before cardiac surgery or transplantation.⁸

We thank Sister J Digiogia and the staff of the renal unit for assistance with the haemofiltration procedure.

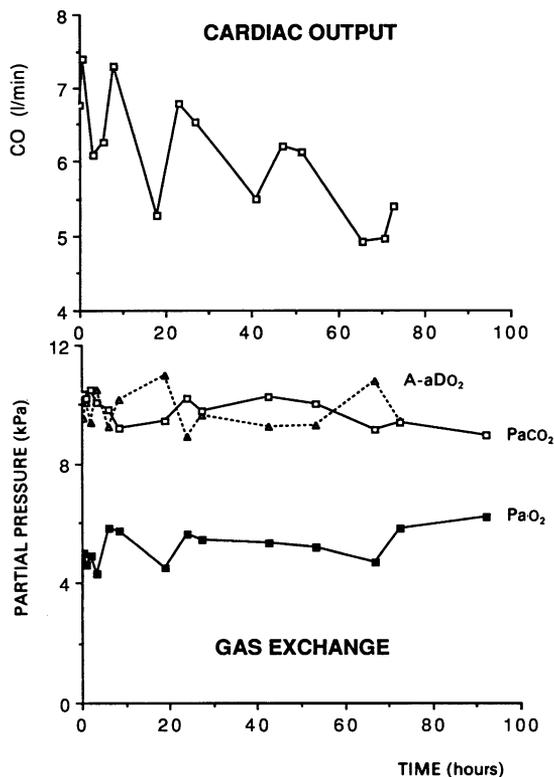


Fig 2 Changes in cardiac output and gas exchange during arteriovenous haemofiltration. Cardiac output (CO) fell throughout the procedure ($CO = 6.79 - 0.22 \times \text{time}$; $r = 0.73$, $F = 14.03$, $p < 0.005$), as does the partial pressure of carbon dioxide ($PaCO_2 = 10.11 - 0.01 \times \text{time}$; $r = 0.62$, $F = 7.36$, $p < 0.025$). The trends in the partial pressure of oxygen (PaO_2) and the alveolar-arterial oxygen difference ($A-aDO_2$) are not significant.

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