Ultrafiltration in the management of refractory congestive heart failure

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SUMMARY Ultrafiltration was performed in nine patients with congestive cardiac failure that was refractory to conventional medical treatment. A mean of 12.7 litres of fluid was removed, and there was a sustained symptomatic improvement in all patients. Weight loss continued after ultrafiltration and a sustained increase in serum sodium concentration was also noted. A transient fall in right atrial pressure was seen only at four hours after ultrafiltration. No adverse haemodynamic effects were seen four and eighteen hours after fluid removal. Intracardiac dimensions measured by echocardiography remained unchanged. Ultrafiltration can be used to relieve symptoms in patients with refractory congestive heart failure and gross oedema.

Some patients with severe heart failure and gross peripheral oedema are resistant to conventional forms of medical treatment including vasodilators. In addition some patients become unresponsive to treatment because of the development of pharmacological tolerance or because of a further deterioration in cardiac function. Management of patients with refractory congestive heart failure is difficult because they have a poor prognosis and distressing symptoms of dyspnoea and gross peripheral oedema. The removal of large volumes of extracellular fluid by venesection, haemodialysis, and peritoneal dialysis is likely to provide such patients with symptomatic relief but is limited by the tendency for adverse haemodynamic effects, such as hypotension and tachycardia, to develop.

Ultrafiltration, however, is a rapid method of selectively removing body water in which controlled fluid removal can prevent sustained haemodynamic upset. In addition ultrafiltration can be performed by a venous technique and is less likely than haemodialysis to cause adverse haemodynamic effects. Though symptomatic improvement has been demonstrated after ultrafiltration in individual patients with refractory congestive heart failure, there are no data on its haemodynamic effects. The aim of this study was to assess the symptomatic and haemodynamic response to ultrafiltration of patients who had severe congestive heart failure that was resistant to conventional forms of medical treatment.

Patients and methods

All patients admitted with chronic congestive heart failure over a three year period were considered for ultrafiltration. In order to maintain an haemodynamically stable study population patients with valvar heart disease were excluded. Only nine patients remained in congestive heart failure with gross peripheral oedema after they had been treated with diuretics, vasodilators, and inotropic agents. These were the patients in whom we tried ultrafiltration (Table). Their ages ranged from 47 years to 65 years (mean 53 years). Fluid intake was restricted to 1 litre per day. The mean dose of frusemide was 600 mg/day (range 160–1000 mg/day) either orally or by intravenous infusion. Vasodilator treatment was continued in six patients. All patients had New York Heart Association class IV symptoms and all had gross peripheral oedema. Informed consent was obtained from all patients.

The following were measured daily—weight (kg); fluid balance; heart rate; systemic blood pressure (standard cuff sphygmomanometry); and serum...
Urea, creatinine, and electrolytes. In six patients invasive haemodynamic monitoring and simultaneous M mode echocardiography were performed before and four hours and eighteen hours after the completion of each ultrafiltration procedure.

**HAEMODYNAMICS**

A Swan-Ganz catheter was inserted by internal jugular venous cannulation and manipulated into the right main pulmonary artery. The right atrial, pulmonary artery, and pulmonary capillary wedge pressures were measured and cardiac output was evaluated by the thermodilution technique by means of an IL 720 cardiac output computer. All measurements were made in triplicate and the mean of these results was taken.

**ECHOCARDIOGRAPHY**

M mode echocardiograms were obtained with an SKI Ekoline echocardiograph and left atrial, right ventricular, left ventricular end diastolic, and left ventricular end systolic dimensions were measured.

**ULTRAFILTRATION**

Ultrafiltration was performed by venous cannulation through a double lumen dialysis cannula in two patients, and by the femoral approach in eight patients, and by the internal jugular approach in one. In seven patients a Gambro AK10 machine was used with a Cordis-Dow 1.5 m² cellulosic based membrane. In two patients a Fresenius A2008C machine was used with a new highly permeable polysulphone membrane (F60). The latter has the advantage of providing improved permeability and biocompatibility.9,10 Ultrafiltration was continued until the development of either distressing muscle cramps or sustained hypotension. If peripheral oedema persisted the procedure was repeated on subsequent days depending on patient tolerance.

**STATISTICAL METHODS**

We used the paired Wilcoxon test for statistical analysis.

**Results**

The overall fluid loss ranged from 3.7 litres to 23 litres (mean 12.7). Ultrafiltration was performed 2–5 times per patient (mean 2.9) and lasted 45 minutes to 12 hours (mean 3.8 hours). The amount of fluid removed at each procedure was 1.7–6.0 litres (mean 3.0 litres) with the standard cellulosic membrane and 20 and 23 litres on the two occasions on which the polysulphone membrane was used. Each individual treatment was stopped because of muscle cramps or sustained hypotension. Transient

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**Table Data on nine patients with congestive heart failure who were treated by ultrafiltration**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Aetiology</th>
<th>Daily drug treatment</th>
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<td>Coronary artery disease</td>
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<td></td>
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<tr>
<td>9</td>
<td>F</td>
<td>47</td>
<td>Congestive cardiomyopathy</td>
<td>Frusemide 1000 mg, spironolactone 200 mg</td>
</tr>
</tbody>
</table>

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![Fig. 1 Weights (kg) of individual patients and mean (SEM) measured one week before ultrafiltration, after the completion of ultrafiltration, and at one week after fluid removal.](http://heart.bmj.com/)

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**Note:**

Images and figures are not visible in the text. The provided text is a continuation of the article, discussing the results and methodology of the study on ultrafiltration in the management of refractory congestive heart failure.
hypotension developed in all patients during the procedure; this responded rapidly to temporary dis-
continuation of fluid removal or, in four patients,
to intravenous infusion of 20 g of albumin. Ultrafiltration was discontinued when it was consid-
ered clinically that the maximum benefit had been
obtained.

Figure 1 shows patients' weights over the study period. No significant change in weight was seen
during the week before ultrafiltration, indicating both a stable clinical state and a lack of response to
drug treatment. Significant weight reduction occurred after ultrafiltration and continued in the
week after the completion of ultrafiltration, indicating an improved response to diuretic treatment.
Figure 2 shows the serum sodium concentrations. There was a significant increase in the serum sodium
concentration after ultrafiltration and this increase was sustained over the subsequent week. No
significant change occurred in either the serum urea or creatinine concentrations over the study period.
Mean (SD) albumin concentration was unchanged
(33 (4) g/l vs 35 (5) g/l) after ultrafiltration, despite the infusion of intravenous albumin during the pro-
cedure in four patients.

HAEMODYNAMICS
Right atrial pressure was significantly reduced four
hours after the completion of ultrafiltration, though
by 18 hours it had returned towards the value before
ultrafiltration. No change was seen in the heart rate,
mean systemic blood pressure, or mean pulmonary
artery pressure. Pulmonary capillary wedge pressure
(mean (SD)) which was 26 (6) mm Hg before ultrafiltration was unchanged four hours (27 (6) mm
Hg) and 18 hours (31 (9) mm Hg) after completion
of each ultrafiltration period. Similarly cardiac
output (mean (SD)) which was 3.2 (0.7) l/min before
ultrafiltration remained unchanged four hours (3.3
(0.6) l/min) and 18 hours (3.3 (0.4) l/min) after each
procedure. Echocardiographic measurement of intracardiac dimensions remained unchanged
throughout.

PROGRESS AFTER ULTRAFILTRATION
Symptoms improved and all patients became con-
siderably less distressed immediately after ultrafiltration. Two patients who were non-insulin
dependent diabetics developed systemic infection
after ultrafiltration; the one with cellulitis had been
treated for staphylococcal septicemia before
ultrafiltration. The source of infection in both cases
was related to the indwelling Swan-Ganz catheter
used for haemodynamic monitoring and not to the
ultrafiltration procedure itself. In both cases staphy-
lococci were isolated from blood culture within one
week of ultrafiltration. Antibiotic treatment was suc-
cessful in one, but the other patient died of ventricu-
lar fibrillation five days after the start of antibiotic
treatment. Two patients were readmitted within
three months of ultrafiltration with recurrence of
peripheral oedema. Both these patients responded
quickly to increased diuretic treatment and were
able to be discharged home free of oedema after a
short hospital admission. The remaining patients
were free of oedema during subsequent follow up
and one patient improved to New York Heart Assos-
ciation class III.

There were two hospital deaths after ultrafiltration. One patient with congestive cardio-
myopathy and staphylococcal septicemia died of
documented ventricular fibrillation and the other, a
patient with restrictive cardiomyopathy, of pro-
gressive deterioration in heart failure. A further
five patients died during subsequent follow up
(mean time to death 3.4 months, range 1–9 months).
Sudden cardiac death was the presumed cause of all
deaths and all the patients had remained free of
oedema since ultrafiltration. The two surviving
patients were oedema free throughout the sub-
sequent year of follow up.

Discussion
This study has demonstrated that ultrafiltration is a
safe, effective, and relatively simple method for pro-
ducing symptomatic improvement in patients with
refractory congestive heart failure.

The ultrafiltration procedure used in this study
has several advantages in the management of these
patients. First, the rate at which fluid is removed can
be accurately controlled by altering the trans-
membrane hydrostatic pressure. This fine control of
fluid removal is of considerable importance in pre-
venting sustained haemodynamic upset in patients
who are already haemodynamically compromised. Towards the end of the study a new highly permeable, biocompatible filter became available and was used in two patients. In both of them over 20 litres of fluid was removed during a single ultrafiltration procedure. Use of this type of membrane may make it possible to render patients oedema free without the need for repeated intervention. The other major advantage is that vascular access is simple and necessitates only percutaneous central venous cannulation.

In our patients there was a significant improvement in serum sodium concentration, which was of similar magnitude to that found after ultrafiltration in fluid overloaded patients with renal failure. All patients experienced transient hypotension when fluid was removed too rapidly during ultrafiltration. This was confirmed by pulmonary capillary wedge pressure measurement made in one patient during the procedure. When fluid was removed too rapidly from the intravascular compartment the wedge pressure fell from 30 mm Hg to 12 mm Hg with consequent fall in cardiac output and blood pressure. When ultrafiltration was tran-

siently discontinued and intravenous albumin was infused, there was a rapid return to a stable haemodynamic state as the wedge pressure increased to 28 mm Hg. We therefore advise that the ultrafiltrate should be removed at a rate of less than 400 ml per hour and that intravenous albumin should be infused in hypoalbuminaemic patients in order to redistribute fluid from the interstitium into the intravascular compartment and promote removal by ultrafiltration.

As expected the reduction in weight found im-
mediately after ultrafiltration reflected the amount of fluid removed during the procedure. Weight reduction continued over the week after ultrafiltration, however, and since fluid intake was restricted to one litre daily before and after ultrafiltration this indicates an improved response to diuretic treatment. This finding has been reported in fluid overloaded patients without pump failure but its mechanism remains unclear. Though improved absorption of diuretic due to improvement of intestinal oedema may play a part in some patients, the continued weight reduction was also seen in those patients on intravenous therapy. In the absence of improved haemodynamics, an effect on the renin angiotensin system or a resetting or baroreceptors must be postulated as a possible underlying mechanism.

Though sustained symptomatic relief from gross oedema was achieved in these patients there was no improvement in haemodynamic variables, except for a transient fall in right atrial pressure. This finding is not surprising in patients in whom the underlying pathophysiological mechanism is primary myocardial failure, since ultrafiltration is dealing only with the secondary effects. Nor can it be expected to improve outcome in a group of patients with such a poor prognosis.

References


10 Martens H, Budde C, Goppinger S, et al. Bio-

compatibility of 4 different membranes in acetate and bicarbonate dialysis [Abstract]. Proceedings of the Euro-


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*Br Heart J* 1986 55: 344-347
doi: 10.1136/hrt.55.4.344

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