Among the many factors contributing to mortality and morbidity in dialysis patients, nutrition is one of the most important. The ADEMEX (Adequacy of Peritoneal Dialysis in Mexico) study suggests that increasing the amount of daily dialysis to compensate for loss of residual renal function (RRF) does not change mortality or morbidity in peritoneal dialysis (PD) patients. Our purpose in the present study was to determine whether a gradual increase in daily dialysis volume to replace lost RRF interferes with the nutrition of patients. We studied the correlation between normalized protein catabolic rate (nPCR) and daily dialysis volume in 150 PD patients. The Student t-test was used to discover if the correlation was statistically significant. We found that, as the daily dialysis volume increases to replace lost RRF, nPCR declines significantly. This reverse relationship was statistically significant at a p value of 0.007. Replacement of lost RRF by an increase in daily dialysis volume in PD patients contributes significantly to their state of protein malnutrition. The large quantity of carbohydrate acquired through dialysis interferes with the patients' intake of protein. The resulting condition of malnutrition probably plays a significant role in mortality and morbidity in those patients.

Key words
Nutrition, residual renal function, daily dialysis volume

Introduction
The survival of a peritoneal dialysis (PD) patient depends on many factors: dialysis adequacy (1), nutrition (2–4), phosphate or calcium ÷ phosphate product (5), hypertension (6,7), type of peritoneal membrane (8), and diabetic state (1,9), among others. The same factors also affect comorbidity in those patients.

The researchers who carried out the CANUSA study thought that adequacy of dialysis measured by weekly Kt/V or creatinine clearance was one of the major factors predicting morbidity and mortality (1). The higher the solute clearance, the better the survival. Gradually, nephrologists started to doubt the conclusions from the CANUSA trial. On reanalysis of the CANUSA study, Bargman et al. (10) showed that the survival was related to residual renal function (RRF) and not to adequacy of dialysis.

In the CANUSA trial, only 2% of patients were on a cycler. That statistic suggests that, as patients lost RRF, few prescriptions were changed. The patients with the best survival were those who maintained RRF the longest.

The ADEMEX (Adequacy of Peritoneal Dialysis in Mexico) trial is now the major study showing that increasing solute clearance above a certain level does not reduce morbidity or improve patient survival (9). And, just as the ADEMEX trial showed a lack of improvement for PD patients with increased solute clearance, the HEMO trial showed the same result for hemodialysis patients (11).

Because malnutrition is an important factor in PD comorbidity and mortality, we asked ourselves this question: Is nutrition affected by loss of RRF in PD patients? We also sought to find out whether, by increasing solute clearance to try to reach the suggested Dialysis Outcomes Quality Initiative (DOQI) guidelines, we are creating protein malnutrition in PD patients to whom we give large quantities of sugar calories in dialysis fluid.

Patients and methods
We studied the relationship between normalized protein catabolic rate (nPCR) and RRF in 150 chronic PD patients. Using the Pearson correlation coefficient, we looked at the correlation between nPCR and the daily dialysis volumes used in those patients. We also studied the relationship between nPCR and daily dialysis volumes in two specific groups: patients using more than 19 L of PD fluid daily and patients using 10 L or less daily. Our purpose was to find out if the relationship between nPCR and daily dialysis volumes in those two groups would be more explicit. We also...
studied the specific relationship by multiple linear regression analysis, using nPCR as the dependent variable and using membrane type, volume, age, and serum albumin as independent predictors.

Results
The statistical evidence is definite: as shown by a decrease in nPCR, the protein intake of a PD patient decreases significantly as RRF is gradually lost. A positive correlation exists between the decrease in nPCR and the decrease in RRF \( p = 0.035 \).

As the patient’s daily dialysis volume is increased to compensate for loss of RRF, the nPCR decreases significantly. The negative relationship between nPCR and total daily dialysis volume is much more significant \( p = 0.007 \) than the negative relationship between decreased nPCR and decreased RRF \( p = 0.035 \). The relationship is still more evident in a comparison of our two specific groups [patients using 19 L or more of dialysis fluid daily, and patients using 10 L or less of dialysis fluid daily \( p = 0.001 \), Figure 1].

Multiple linear regression analysis showed that only daily dialysis volume and albumin are independent predictors of nPCR. We were not able to find any relationship between age or type of membrane and nPCR (Table I).

Discussion
Many studies have shown that nutrition is an important predictor of mortality and morbidity in PD patients. The two largest studies—CANUSA (1) and ADEMEX (9)—showed that nutrition is a significant factor predicting survival and morbidity. In the ADEMEX trial, patients with a normalized protein equivalent of total nitrogen appearance (nPNA) value greater than 0.8 g/day had a significantly better survival as compared with patients whose nPNA value was less than 0.8 g/day. As patients gradually lose RRF, their intake of protein seems to decrease significantly, and they become malnourished.

Nutrition is related to RRF for two major reasons. Peritoneal dialysis probably cannot adequately replace RRF: the kidney, for the same amount of clearance, does much better than PD does at removing the uremic toxins that contribute to a patient’s decreased appetite. Perhaps a more important factor may be the way in which RRF is replaced by PD. We increase PD by using larger amounts of solution containing very high glucose. As we give more dialysis fluid to compensate for loss of RRF, we enormously increase glucose absorption by the peritoneum. That very high glucose intake cuts the appetite of PD patients—mainly their protein intake. The effect is worse in patients with poor ultrafiltration, who require large amounts of PD fluid high in glucose to remove enough fluid to prevent fluid overload.

Tables II and III show the large of glucose intake in various PD modalities. We used the PD Adequest program (Baxter Healthcare, Deerfield, IL, U.S.A.) to calculate the calorie intake in various peritoneal modalities for patients with a low-average membrane. The glucose absorption is noticeably high in most of the modalities. The calories shown in the tables increase by 20% – 40% if the calculation is done for patients with a membrane classed as high-average or

![Table I: Correlation between the studied parameters](image)

**Table I** Correlation between the studied parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pearson correlation coefficients</th>
<th>Multiple linear regression analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>nPCR vs. daily dialysis volume</td>
<td>( p = 0.007 )</td>
<td>Predictors</td>
</tr>
<tr>
<td>nPCR vs. residual renal function</td>
<td>( p = 0.035 )</td>
<td>nPCR</td>
</tr>
<tr>
<td></td>
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<td>nPCR</td>
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</table>

Only dialysis volume and albumin were independent predictors of nPCR.

nPCR = normalized protein catabolic rate.

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**FIGURE 1** Relationship between normalized protein catabolic rate (nPCR) and daily dialysis volume in two different groups: patients using > 19 L of dialysis fluid daily and those using ≤ 10 L daily.
high. For many patients, that large intake of glucose is like having an extra meal daily—but only of glucose. As Tables II and III show, a partial solution to the problem is to replace some of the exchanges with solutions containing icodextrin and amino acids. The tables show that the quantity of glucose absorbed daily can be significantly reduced, probably permitting a better protein intake.

**Conclusions**

The ADEMEX trial confirmed that mortality is not improved by increasing solute clearance above a certain limit (9). Residual renal function (10) and nutrition (2–4) play a more significant role in patient survival. A definite relationship exists between RRF and nutrition. As RRF is gradually lost, protein intake decreases, causing a state of malnutrition. Peritoneal dialysis cannot fully replace RRF. Avoiding any drug that could cause deterioration of RRF in PD patients is extremely important. Loss of RRF means deterioration of nutrition, which can lead to higher mortality and morbidity.

Attempting to reach the DOQI solute clearance guideline by giving huge amounts of glucose dialysis fluid daily can also lead to protein malnutrition. The high glucose content of the PD fluid reduces the patients’ appetite. The problem has two solutions: In patients who are feeling well, practitioners must be less compulsive about the DOQI guidelines. In patients who need larger quantities of PD fluid to obtain adequate dialysis, some of the daily fluid should be replaced with sugar-sparing fluids such as those containing icodextrin and amino acids.

**References**

5. Block GA, Hulbert–Shearon TE, Levin NW, Port FK.

**TABLE II** The daily calories (glucose absorption) in continuous ambulatory peritoneal dialysis (CAPD) calculated for a patient with a low-average membrane

<table>
<thead>
<tr>
<th>Calories/day</th>
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<tbody>
<tr>
<td><strong>CAPD (4 exchanges)</strong></td>
</tr>
<tr>
<td>2 $\times$ 2.25% glucose + 2 $\times$ 1.5% glucose</td>
</tr>
<tr>
<td>1 $\times$ icodextrin + 1 $\times$ amino acids + 2 $\times$ 1.5% glucose</td>
</tr>
<tr>
<td>1 $\times$ icodextrin + 1 $\times$ amino acids + 1 $\times$ 2.25% glucose + 1 $\times$ 1.5% glucose</td>
</tr>
<tr>
<td><strong>Quantum (5 exchanges)</strong></td>
</tr>
<tr>
<td>2 $\times$ 2.25% glucose + 3 $\times$ 1.5% glucose</td>
</tr>
<tr>
<td>1 $\times$ amino acids + 1 $\times$ icodextrin + 2 $\times$ 1.5% glucose + 1 $\times$ 2.25% glucose</td>
</tr>
</tbody>
</table>

**TABLE III** The daily calories (glucose absorption) in continuous cycling peritoneal dialysis (CCPD) calculated for a patient with a low-average membrane

<table>
<thead>
<tr>
<th>Calories/day</th>
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<tbody>
<tr>
<td><strong>CCPD with 5 nightly exchanges</strong></td>
</tr>
<tr>
<td><strong>CCPD with 5 nightly exchanges + “wet” day (2.25% glucose)</strong></td>
</tr>
<tr>
<td><strong>CCPD with 5 nightly exchanges + “wet” day (2.25% glucose)</strong></td>
</tr>
<tr>
<td><strong>CCPD with 5 nightly exchanges + 2 daily exchanges (1.5% glucose + 2.25% glucose)</strong></td>
</tr>
<tr>
<td><strong>CCPD with 5 nightly exchanges + 2 daily exchanges (icodextrin + 1 $\times$ 1.5% glucose)</strong></td>
</tr>
<tr>
<td><strong>CCPD with 5 nightly exchanges + 2 daily exchanges (icodextrin + 1 $\times$ amino acids)</strong></td>
</tr>
</tbody>
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Corresponding author:
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Patients with severe congestive heart failure (CHF), mainly class IV on the New York Heart Association (NYHA) scale, became refractory to standard medical therapy. The factor that favored that evolution was renal insufficiency with inadequate renal perfusion. Our objectives in the present study were to make a preliminary assessment of the usefulness of automated peritoneal dialysis (APD) for the treatment of volume overload in those patients.

Our study was carried out in the peritoneal dialysis unit of the clinical hospital of Pontificia Universidad Católica de Chile. We studied 3 non uremic patients with refractory CHF secondary to ischemic cardiomyopathy, severe secondary lung hypertension (>70 mmHg), and associated moderate renal insufficiency. The patients (2 women and 1 man) ranged in age from 55 years to 68 years. A Tenckhoff double-cuff peritoneal catheter was placed in each patient, and peritoneal dialysis was carried out using the nightly intermittent peritoneal dialysis (NIPD) modality. The treatment used was appropriate to obtain a gradual removal of fluids: dialysate dextrose at 1.5% and 2.5%, 4 – 5 cycles, and total volume of 10 – 12 L per night.

All three patients were able to remain at home, with no signs of hypervolemia. The frequency and length of hospitalizations decreased on average from 59 days pre-APD to 37 days post-APD. No hospitalizations for cardiac problems occurred, and the mean survival was 11 months (range: 6 – 22 months). This preliminary observation suggests that APD could be offered as an effective treatment for helping to remove fluids in patients with refractory CHF, reducing the number and length of hospitalizations, and improving quality of life.

Key words
Congestive heart failure, automated peritoneal dialysis

Introduction
Over the last decade, advances in the pharmacologic handling of congestive heart failure (CHF) have been remarkable. Early administration of angiotensin-converting enzyme inhibitors and vasodilators—together with loop diuretics, digitalis, and spironolactone—constitutes the base of current therapy (1). Nevertheless, certain patients [mainly class IV on the New York Heart Association (NYHA) scale] become refractory to the maximal well-tolerated drug therapy. Such patients are susceptible to the complications of reduced effective blood volume, orthostatic hypotension, and pre-renal azotemia. Decompensated CHF stimulates the renin–angiotensin, aldosterone, and vasopressin systems, which predisposes CHF patients to hypototremia, hypokalemia, arrhythmias, and metabolic alkalosis (2). Together, those problems imply a poor quality of life and high morbidity and mortality.

The use of intermittent peritoneal dialysis to treat refractory CHF was proposed by Schneierson in 1949 (3), but the utility of the treatment was limited by the necessity for hospitalization and by recurrent episodes of peritonitis. Later on, the introduction of a permanent catheter for access to the peritoneal cavity facilitated the development of continuous ambulatory peritoneal dialysis and, eventually, automation of that technique, so that it could be offered as an effective form of assistive treatment in medical therapy (4–9).

Here, we report the cases of 3 non uremic patients with CHF who failed to respond to traditional medical therapies, for whom fatal outcomes were expected within months, and whom we treated with peritoneal dialysis.

Patients and methods
Our prospective study enrolled 3 non uremic patients with chronic progressive refractory congestive heart failure, NYHA class IV, secondary to coronary heart disease and ischemic cardiomyopathy. The patients...
had been admitted into cardiology intensive care at the clinical hospital of Pontificia Universidad Católica de Chile between August 2000 and July 2002. All begin treatment with automated peritoneal dialysis (APD), using the nightly intermittent peritoneal dialysis (NIPD) modality through a permanent Tenckhoff (swan-neck) peritoneal catheter. Ultrafiltration was adjusted using 1.5% – 2.5% dextrose dialysis solution (Baxter Healthcare, Deerfield, IL, U.S.A.), with the dwell time adjusted depending on a peritoneal equilibration test. Table I provides details about the patients.

Results

Patient 1
A 68-year-old woman had decompensated CHF owing to ischemic heart disease, cardiac dilation, atrial fibrillation, severe secondary lung hypertension, chronic obstructive pulmonary disease, and hypothyroidism. On physical examination, her mean arterial blood pressure was 60 mmHg, and she showed lung congestion, hepatomegaly, jugular venous dilation, and edema in both legs. Laboratory tests revealed pre-renal azotemia with serum creatinine 2.37 mg/dL, blood urea nitrogen (BUN) 69 mg/dL, and plasma Na 133 mEq/L. An echocardiogram showed a left atrial size of 59 mm and a left ventricle diameter at end of diastole (LVID) of 70 mm, a shortening fraction below 20%, markedly extensive right cavities, severe mitral and tricuspid regurgitation, and a systolic lung artery pressure of 73 mmHg. She was being treated with a steady infusion of vasoactive drugs (dopamine 2 µg/kg/min and dobutamine 4 µg/kg/min). Between February and August of 2000, she had required 8 hospitalizations (63 days) owing to the decompensation of her heart pathology.

We started APD in August 2000, using 1.5% – 2.5% dextrose dialysate solution (10 – 12 L) to achieve ultrafiltration of up to 2 L daily. In the first week of treatment, her weight decreased by 6.3 kg, and she experienced a vast improvement in her symptoms. She was sent home in good condition without a need for vasoactive drugs. Three months into treatment with APD, her laboratory test results showed these values: serum creatinine, 4.6 mg/dL; BUN, 42 mg/dL; and plasma Na, 136 mEq/L. The patient was clearly stabilized at NYHA class II. Over the 6 months following initiation of APD, she was hospitalized twice for a total of 15 days for non heart causes (a gastrointestinal episode and an accidental wound in the left leg). Unfortunately, she died from complications of the leg wound, which were caused by her critical ischemia.

Patient 2
A 55-year-old man had ischemic heart disease, dilated cardiomyopathy, and acutely decompensated chronic CHF, together with coronary artery bypass graft, arterial hypertension, hyperlipidemia, and chronic obstructive pulmonary disease. Laboratory tests revealed serum creatinine 1.48 mg/dL (which increased to 3.16 mg/dL with depletion treatment), BUN 62 mg/dL, and plasma Na 129 mEq/L. On echocardiogram, the patient’s left atrial size measured 50 mm and his LVID 67 mm. The patient had a left ventricle shortening fraction below 20%, dilated right cavities, mitral regurgitation, severe tricuspidal regurgitation, severe secondary lung hypertension (later becoming extreme), and lateral inferior akinesia. With good pharmacologic treatment, he presented a mean blood pressure of 60 mmHg. The severe left ventricle dysfunction kept him hospitalized for 52 days with a steady infusion of vasoactive drugs (dopamine 2.5 µg/kg/min and dobutamine 5 µg/kg/min).

A Tenckhoff catheter was inserted under local anesthesia in May 2001. His APD treatment then began with 1.5% dextrose dialysate solution (10 L daily), achieving ultrafiltration of 800 – 900 mL daily. He showed significant clinical improvement and was sent home in good condition, but with a prescription for a low dose of vasoactive drugs.

From May 2001 to May 2002, this man was hospitalized 5 times for a total of 15 days. He was hospitalized for placement of the central catheter, for

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Basal characteristics of the patients</th>
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<tbody>
<tr>
<td>Patient 1</td>
<td>Patient 2</td>
</tr>
<tr>
<td>Age (years)</td>
<td>68</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>2.37</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>69</td>
</tr>
<tr>
<td>Plasma Na (mEq/L)</td>
<td>133</td>
</tr>
<tr>
<td>Urine volume (mL/24 h)</td>
<td>450</td>
</tr>
<tr>
<td>Shortening fraction (%)</td>
<td>11</td>
</tr>
<tr>
<td>Left atrial size (mm)</td>
<td>59</td>
</tr>
<tr>
<td>Left ventricle diameter a (mm)</td>
<td>70</td>
</tr>
<tr>
<td>Mean blood pressure (mmHg)</td>
<td>60</td>
</tr>
</tbody>
</table>

a At end of diastole.  
BUN = blood urea nitrogen.
placement of a feeding tube, and for gastritis. No hospitalizations were related to decompensation of the heart disease. Laboratory control tests revealed serum creatinine 1.92 mg/dL, BUN 38 mg/dL, and plasma Na 134 mg/dL. He died from myocardial infarction 14 months after the start of APD treatment.

**Patient 3**

A 71-year-old woman had ischemic heart disease, coronary artery bypass graft, atrial fibrillation, double-chamber pacing, cirrhosis of the liver with portal hypertension, and ascites. Her laboratory tests showed creatinine 2.7 mg/dL, BUN 49 mg/dL, and plasma Na 121 mEq/L. An echocardiogram showed a left atrial size of 48 mm and an LVID of 72 mm. Her shortening fraction was below 20%, and she had dilated right cavities, aortic regurgitation, severe mitral and tricuspidal regurgitation, and a pulmonary artery pressure of 60 mmHg.

Between August 2000 and April 2001, this woman was hospitalized for a period of 62 days for decompensated CHF refractory to medical therapy. She required support with 8 extracorporeal ultrafiltration methods for removing excess fluid, and she was maintained on a steady infusion of vasoactive drugs (dopamine 4 µg/kg/min and dobutamine 5 µg/kg/min).

The woman began APD in April 2001 with 2.5% dextrose dialysate solution (12 L daily). Clinical improvement included regression of the fluid overload, a weight decrease of approximately 8.2 kg, and clear improvement in the CHF (to NYHA class II from class IV). She was sent home in good condition without the need for vasoactive drugs. Laboratory control tests after 2 months of treatment showed serum creatinine 2.4 mg/dL, BUN 28 mg/dL, plasma Na 134 mEq/L.

Between March 2001 and April 2002, this woman required 4 hospitalizations for a total of 36 days. She was hospitalized for an abdominal hernia, pancreatitis, diarrhea, and peritonitis. No hospitalization was related to her heart decompensation. At the time of writing, she was continuing her monthly medical check-ups at the peritoneal dialysis unit.

**Summary of cases**

Table II shows the characteristics of the three patients post-APD. The patients were able to remain at home, with no signs of hyponatremia. The frequency and average length of hospitalizations decreased to 37 days post-APD from 59 days pre-APD. No hospitalizations for heart pathology problems occurred, and the average survival to the date of writing was 15 months (range: 6 – 22 months).

**Discussion**

The satisfactory results obtained with the patients reported here demonstrate that peritoneal dialysis can be considered an effective long-term therapy for patients with CHF—especially for those who have comorbidities such as lung hypertension or pre-renal azotemia. Stegmayr et al. (10) reported similar findings in 16 patients (9 of them non-uremic) with severe refractory CHF (NYHA class III or IV). Those authors observed a reduction in the size of the heart (something we did not find). Continuous ambulatory peritoneal dialysis can also serve as a bridge for definitive surgery or future transplant (11).

Our 3 patients showed improvement in their heart conditions to NYHA class III or II from class IV, and they also showed an increased quality of life. They were able to stay at home, without signs of hypervolemia. Their hyponatremia was either partially or totally improved, and their dry weight stabilized and was maintained. Hospitalization decreased to an average of 37 days (a 60% reduction).

Previous reports indicated good results in controlling water overload in patients with CHF by using CAPD treatment (12). We used APD, which we consider to be a simple, well-tolerated technique. Removal of fluid is gradual and continuous, and hemodynamic stability is assured. The quantity of ultrafiltration was adjusted with dextrose dialysate solution and a dwell time determined by a peritoneal equilibration test.

Previous studies of CAPD treatment demonstrated a diminution of ventricular volume and, thus, an increase in the systolic function of the left ventricle (2,10), restoring the response to diuretics (3–5). Clinical improvement was achieved (13), although a correlated improvement in ejection fraction did not occur (14).

**Conclusion**

We suggest that APD can be offered as an effective treatment in patients with CHF to assist in removing fluid without endangering hemodynamic stability. Treatment with APD reduced hospitalization days (and the associated costs) and improved the quality of life in our patients.
References


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