TWO YEARS EXPERIENCE IN INTERMITTENT HAEMODIALYSIS

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During the past two years we have treated 13 patients with maintenance haemodialysis; our methods and the unit have been described elsewhere (Evans, De Wardener, Curtis, Storey and Jennings, 1965; De Wardener, Evans and Curtis, 1965). Our total patient experience to the end of April 1966 is 10 patient years. We report here our results and make a few special points from our experience.

Results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age at start</th>
<th>Diagnosis</th>
<th>Plasma creatinine in mg/100 ml</th>
<th>Ccr in ml/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.T.</td>
<td>M</td>
<td>29</td>
<td>Malignant hypertension</td>
<td>29.1</td>
<td>1.5-1.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chronic glomerulonephritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.C.</td>
<td>F</td>
<td>23</td>
<td>Chronic pyelonephritis*</td>
<td>22.8</td>
<td>2.8-3.3</td>
</tr>
<tr>
<td>P.W.</td>
<td>M</td>
<td>42</td>
<td>Polycystic kidneys</td>
<td>17.4-20.0</td>
<td>4.0-4.5</td>
</tr>
<tr>
<td>M.D.</td>
<td>M</td>
<td>38</td>
<td>Malignant hypertension</td>
<td>23.6</td>
<td>1.0</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>Chronic glomerulonephritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P.F.</td>
<td>M</td>
<td>33</td>
<td>Malignant hypertension</td>
<td>18.4</td>
<td>0.8-3.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chronic glomerulonephritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B.N.</td>
<td>M</td>
<td>45</td>
<td>Chronic glomerulonephritis</td>
<td>18.0</td>
<td>2.6-3.2</td>
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<td>C.C.</td>
<td>M</td>
<td>50</td>
<td>Malignant hypertension</td>
<td>19.2-20.6</td>
<td>2.0-3.0</td>
</tr>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>D.J.</td>
<td>M</td>
<td>31</td>
<td>Malignant hypertension</td>
<td>20.0</td>
<td>1.4-4.2</td>
</tr>
<tr>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>A.L.</td>
<td>M</td>
<td>44</td>
<td>Chronic pyelonephritis</td>
<td>18.4</td>
<td>—</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Primary gout</td>
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<td></td>
</tr>
<tr>
<td>B.W.</td>
<td>F</td>
<td>22</td>
<td>Chronic pyelonephritis*</td>
<td>17.2-18.8</td>
<td>0.9-3.5</td>
</tr>
<tr>
<td>S.B.</td>
<td>F</td>
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<td>Chronic pyelonephritis*</td>
<td>22.8-25.2</td>
<td>1.2-1.6</td>
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<tr>
<td>A.J.</td>
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<td>Familial renal disease</td>
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<td></td>
<td></td>
<td>Hyperprolinaemia</td>
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<td></td>
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<tr>
<td>W.F.</td>
<td>F</td>
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<td>Malignant hypertension</td>
<td>16.5</td>
<td>0.1-0.4</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Chronic glomerulonephritis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = Bilateral nephrectomy

The table summarises the diagnoses and ages of our group of patients at the time of their being put on the programme. We have treated 8 men and 5 women. Seven had a presumptive diagnosis of chronic glomerulonephritis and six of these presented with malignant hypertension. One has polycystic kidneys. One has familial renal disease associated with hyperprolinaemia. In the other four the aetiology was chronic pyelonephritis, three were young
women and two of these had an abnormality of the lower urinary tract and the fourth was a
man of 44 with a history of primary gout. Bilateral nephrectomy has been carried out in
each of the three young women with chronic pyelonephritis. This has been done not to
control blood pressure but because in each case the kidneys were the site of infection which
could not be cured by antibiotics. One had recurrent septicaemia, one loin pain and repeatedly
infected urine. The third had a chronically infected ileal bladder. The ileal bladder had been
fashioned because of neurological bladder disease secondary to spina bifida.

The patients' renal function at the time of going on the programme is also tabulated. The
plasma creatinines ranged from 14.6-29.1 mg/100 ml and were mostly about 20 mg/100 ml.
In all the patients the endogenous creatinine clearance has been less than 5 ml/min and in
most less than 3 ml/min. Urine volumes have fallen since dialysis, but the patient with
polycystic kidneys continues to produce in excess of 1 litre per day, although he has now
been dialysed for 17 months.

Fig. 1 shows diagrammatically the length of time for which each of our patients has been
dialysed. Our first patient (top of figure) has been dialysed for 25 months, our most recent
(bottom of figure) for 2 months. We are currently treating ten patients. Two of the patients
are now being treated in other centres. Failure to mix the dialysis fluid in the preparation
tanks has precipitated death due to cerebellar haemorrhage in one patient who had been
maintained on dialysis for 1 year. This patient was dialysed with hypertonic dialysis fluid
resulting in hypernatraemia. We now routinely measure the electrical conductivity of the
dialysis fluid before starting dialysis.

The stippled area represents the total time spent in hospital by each of the patients since
they were launched on the programme. Most of this time was for recannulation. We keep a
patient in for two weeks for a fresh leg cannula. One patient had to be readmitted for
gastrectomy and one for bilateral nephrectomy. The sum total of this incidental hospitalization
amounts to 10% of the total outpatient dialysis life that we have given to the group. This

\[
\begin{array}{c}
\includegraphics[width=0.5\textwidth]{chart.png} \\
\text{Fig. 1. Duration of dialysis.}
\end{array}
\]

Each block represents the span of time for which each individual patient has been dialysed at Fulham.
The left hand end of each block is set opposite the date of entry onto the programme.

- (interrupted line) = Patient transferred.
- (thick line) = Patient died.

percentage is decreasing as the patients' well-being improves and cannula survival becomes
more prolonged.

The weights of the patients are shown in Fig. 2. Gain in weight above the patients' baseline
weight is shown. By baseline weight we mean the weight of the patient when sufficient fluid
has been removed for there to be no oedema and for the blood pressure to be within the normal range. In all our patients the blood pressure has become normal once a critical weight loss has occurred (Comis, Rottka and Shaldon, 1964). This is achieved by sodium and fluid restriction and by ultrafiltration during dialysis. The baseline weight is usually reached after 1-2 months dialysis. All of the patients have put on flesh weight since reaching this level. One of the most remarkable has been a patient who has gained 13.3 kg since his gastrectomy for gastro-intestinal bleeding 6 months ago; and another, a recent patient, has put on 9.2 kg during the first 4 months of her dialysis.

\[ \text{weight} \]
\[ \begin{array}{c}
\text{baseline} \\
\text{weight gain} \\
70 \\
50 \\
\hline
25 \quad 21 \quad 16 \quad 12 \quad 13 \quad 3 \quad 9 \quad 10 \quad 9 \quad 5 \quad 4 \quad 3 \quad 2
\end{array} \]

\text{months of dialysis}

\text{Fig. 2. Weights of patients}

All our present patients are either working, or in the case of the women, fulfilling a useful role as housewives. The men have all been able to get back to the type of work that they were doing before they entered the phase of terminal renal disease. Most of them have sedentary or office jobs, and put in about 40 hours per week. The housewives claim that they are able to be busy for up to 70 hours per week. One patient failed to return to useful employment although he was no less fit than the others. The well-being of this group of patients is further reflected by the fact that all of the present patients are having sexual intercourse.

TECHNICAL POINTS

1. Heparinization

Since our patients are dialysed in a hospital unit, we are able to use intermittent heparinization (Table I). Thus the difficulties of regional heparinization are avoided. The nurses prefer to give intermittent heparin rather than to have the added technicalities of continuous infusion pumps. We aim to give as little heparin as is necessary. The required dosage is assessed for each individual patient. This is done by clotting time estimations carried out during 2-3 dialyses after the patient has become stabilized on dialysis treatment. It is our impression that the required dosage may fall after 6 months to 1 year's treatment, and also if long term anticoagulants are used. On this routine we are currently using around 17,000 units (170 mg) for a 14 hour dialysis. We have not encountered rebound phenomena and there have been no haemorrhagic complications attributable to heparin.

Regional heparinization is reserved for special circumstances; pericarditis; when gastrointestinal haemorrhage has occurred recently; and following major surgery, but not generally following fresh cannulation.
2. Sterilization of the kidneys

We have recently abandoned acetic acid for sterilization of the prepared kidneys. During the period of time for which we were using acetic acid there was an increase in pyrexial reactions for which no cause was apparent such as blood transfusion (see Fig. 3). At the same time we were detecting an increasing count of an aerobic spore-bearing organism (*Bacillus cereus*) in our dialysate and from the kidneys. This organism is known to be present in our local water supply which is 'surface' water. We then grew the same organism on 15 blood cultures from patients who were experiencing sweating attacks and occasional rigors both during dialysis and also at home. One patient had 8 positive blood cultures. We gave him antibiotics in accordance with the in vitro sensitivity pattern of the Bacillus cereus and the fevers ceased. We wish to make it clear that we have not demonstrated that this organism is pathogenic but we are not the first to suggest that it may be so (Stopler, Camescu and Voiculescu, 1964). Further bacteriological studies are proceeding (Curtis *et al.*, in preparation). However, we have now changed back to formalin. We suggest that where tap water is used to prepare the dialysate, and where this tap water may contain such contamination, it is not safe to use acetic acid for sterilizing the kidneys.

![Fig. 3. Association of pyrexial episodes with positive blood cultures](image)

HCHO = Formalin sterilization
CH₃COOH = Acetic acid sterilization
○ = Positive cultures for a variety of organisms (Coagulase negative staphylococcus, streptococcus faecalis, Bact. coli)
● = Positive cultures for Bacillus cereus.
3. Cannula experience

The average life of arterial cannulae has been 7.6 months, and of the venous cannulae, 5.6 months. These figures do not include any of the cannulae that are functioning at present. 5 cannula infections have occurred in 3 patients. Coagulase positive staphylococci caused 3 of these and Pseudomonas pyocyanea, 2. In 4 infections, including both those due to the Gram-negative organism, the site was lost. In the remaining infection, only the venous cannula had to be moved and with antibiotic therapy the shunt functioned for a further 7 months.

Declotting of the shunts has been required on 49 occasions. Half of our present patients, all patients who were having clotting problems, are now on long term anticoagulation with Warfarin (Coumadin sodium). So far we have had 25.5 patient months experience with continuous anticoagulant therapy. One clotting episode has occurred in this time. This compares very favourably with the 48 clotting episodes that have occurred during 94.5 patient months experience without anticoagulant therapy.

REFERENCES