TECHNIQUE AND MANAGEMENT OF CONTINUOUS HAEMODIALYSIS

G. Mitchell* and D.J. Blackmore**

The necessity for frequent dialysis of hypercatabolic uraemic patients is well recognised. Prophylactic daily dialysis and continuous haemodialysis have been employed. 1, 2, 3 Experiences with the minicoil artificial kidney have been described. 4 In discussing the results of 43 dialyses in 21 patients the authors comment that:-

1 Patients with the high catabolic rates associated with severe trauma or surgical mishap and sepsis are generally believed to have a better prognosis if the serum urea level is kept below 300 mg%: Unless a system of continuous dialysis on the minicoil is attempted, this can be achieved only by using machines with a larger dialysing surface area. 1 We attempted to use minicoils for continuous dialysis a year ago and encountered a variety of problems. Some of these were overcome by modifications to the apparatus and are described elsewhere 5. The dialysing coil is unchanged but the arterial and venous lines, air-trap and filter-chamber have been redesigned as demonstrated 6. Blood pumping facilities should be available for continuous dialysis and in our early trials with the apparatus sigmamotor pumps were used when necessary. However it was felt that an integrated pumping unit should be produced for use with the modified minicoil under any conditions of blood or bath pumping, continuous heparin infusion or regional heparinisation. Such a unit based on four roller pumps has been demonstrated 6 (Figure 1).

The difficulties of prolonged intermittent haemodialysis are well known. The purpose of this communication is to present some of the problems we have encountered during prolonged continuous pumped haemodialysis.

MATERIAL

Eight patients have been dialysed on 12 occasions for a total of 28 days. Four patients were oliguric following severe accidental trauma; two patients were in the terminal phase of chronic renal failure and the remaining two patients had acute renal failure complicating infective hepatitis and diabetic coma respectively.

TECHNIQUES

Dialysing Bath: 1. Chemical Composition
(a) Calcium: The system uses a single pass dialysate of 300 l./day. Bath composition was standard 7 and when stored at room temperature or at 37°C for prolonged periods calcium precipitation occurred. This was attributed to an increase in pH of the dialysing fluid as the carbon dioxide

* Renal Unit, Princess Mary's Hospital, Halton, Bucks.
**Research Department, R.A.F. Institute of Pathology and Tropical Medicine, Halton, Bucks.
Current address: Department of Pathology, R.A.F. Hospital Steamer Point, Aden.
content diminished. This was overcome by storing the bath in 50 l poly-
thene aspirators at room temperature and slowly aerating with 5% CO₂ in
oxygen. Maintenance of an atmosphere of carbon dioxide over the surface
of the dialysing fluid was effected by loosely plugging the container with
wet cotton wool.
(b) Sodium: The sodium concentration of the bath was made up initially
to 150 mEq/l by the addition of sodium lactate. When the patient's plasma
sodium reached 140 mEq/l the bath sodium concentration was reduced to
140 mEq/l(8).

Dialysing Bath: 2. Temperature

The dialysing fluid was heated to 42°C before being passed through
the coil envelope. At this temperature blood emerged from the coil at 36 -
37°C.

Dialysing Bath: 3. Bacteriology

Sterility of bath fluid was maintained by adding 500 mg. of Tetracy-
ccline to each 50 l. Tetracycline has been shown to dialyse(9) and to be
catabolic(10). However if antibiotics were not added to bath fluid a positive
bacterial culture could be obtained from the dialysing fluid envelope. Other
methods of bath sterilisation are being investigated avoiding the use of
antibiotics which, if dialysed into the blood, might adversely affect the
patient.

Blood Coagulation

Heparinisation may be intermittent, continuous or regional. In
patients with a tendency to bleed, regional heparinisation is desirable.
Coagulation of blood from the patient and the coil can be measured by a
whole blood clotting time. This may give rise to problems when coagula-
tion is affected by factors other than heparin. In addition, during the
period of determining a single clotting time a significant alteration in blood
coagulability may have occurred. We have found that a plasma recalci-
fication time is a useful supplement to whole blood clotting time estimates.
(Table I).

<table>
<thead>
<tr>
<th></th>
<th>Whole Blood Clotting Time (min.)</th>
<th>Plasma Recalcification Time (min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal blood</td>
<td>&lt; 15</td>
<td>2 - 4</td>
</tr>
<tr>
<td>Blood + excess heparin</td>
<td>&gt; 60</td>
<td>prolonged</td>
</tr>
<tr>
<td>Blood + heparin 10 μg/ml.</td>
<td>&gt; 60</td>
<td>8 - 20</td>
</tr>
<tr>
<td>Blood + excess protamine sulphate</td>
<td>prolonged</td>
<td>2 - 4</td>
</tr>
<tr>
<td>Other coagulation abnormality</td>
<td>prolonged</td>
<td>2 - 4</td>
</tr>
</tbody>
</table>

Method: Fresh citrated 'platelet rich' plasma 5 drops
Patient's blood 1 drop
MIX
3% w/v calcium chloride 1 drop
TIME
When a whole blood clotting time is prolonged a plasma recalcification method allows a rapid estimate of circulating anticoagulant and helps assessment of the nature of the coagulation defect.

Staffing

Supervision of continuous haemodialysis by medical staff is not practicable as it may involve two or three doctors on shift work looking after one patient. Nursing staff can be trained to look after patients and dialysing apparatus, and the task can be made easier by incorporating warning and switch-off devices on the pump unit. Warning systems may be triggered by blood flow (11) or pressure changes (12). In the pump unit demonstrated (6) pressure changes in the air trap actuate audible and visual warning signals followed by pump shut down. Figure 2 shows the relationship of time to pressure change measured at the air trap following complete occlusion of the arterial or venous lines at a blood flow rate of 100 ml./min. From this it will be appreciated that occlusion of arterial or venous flow will produce a rapid change in pressure at the air trap and that the machine pumps will cut out within a few seconds of mishap. Changes in arterial flow produce a fall in pressure at the air trap. For the warning and cut out systems to work dialysis must be carried out with a positive pressure of at least 50 mm. Hg. A float chamber is incorporated in the air trap as a safety device of use when blood pumping is not required.

CONCLUSION

Continuous haemodialysis has been advocated in the management of patients with hypercatabolic renal failure. We consider that the modified minicoil with its advantages of interchangeable components, disposability and cheapness and used in conjunction with a pump unit which allows nursing staff supervision may prove to be a useful apparatus for continuous dialysis.

REFERENCES

7. Travenol leaflet supplied with each coil.
Figure 1. Photograph of Halton minicoil and HAL pumping unit as set up for dialysis.

Figure 2. Graph showing time for pressure changes measured at manometer for complete venous or arterial occlusion at a blood flow rate of 100 ml./min.
C.L.J. VINK (Eindhoven): I should like to ask Dr. Goldsmith whether he does not think that he has rather low clearances?

As it is known, that in the cellophane membrane micro-clots may arise, which will obstruct the pores, my question is: may clotting influence your clearance?

H.J. GOLDSMITH (Liverpool): We have gone into this point, and our conclusion is that in most cases there is no fall-off of the efficiency. We have taken some coils to bits after the end of the dialysis and have not found any clotting in cases where it had not been suspected clinically.

C.L.J. VINK (Eindhoven): We use small kidneys with three parallel coils with a total membrane area of 1.2 sq. m. The half-life of the urea is about 7 to 8 hours. The blood is driven on the arterio-venous pressure difference, that is without pump and therefore with minimal supervision. The clearance will be more than two or three times higher than in your apparatus, and the volume is about 390 ml. of blood.

H.J. GOLDSMITH (Liverpool): Ours is about 250 ml. I have tried to explain that the reason why we like to use the Minicoil is purely a practical one, in that we can proceed with other work with only occasional medical supervision of the patient. We would not have the necessary staff to treat the patients in our present set-up if we always used the Skeggs-Leonards unit which we still tend to use in some cases.

J.HESS THAYSON (Copenhagen): Do you believe that, if you have a traumatized hypercatabolic patient with a rise in serum urea of say 150 mg.% per day in the serum, you can keep his level sufficiently down using only the Minicoil kidney? Could you keep his level at an average of 150 to 200 in the serum?

G. MITCHELL (Aylesbury): I think I can probably best answer that question by showing a chart (Figure 1).

This patient was treated entirely by continuous Minicoil dialysis. He was hypercatabolic, at one time we were getting 120 grammes of urea per day out in the bath. He was injured at the point shown on the chart, and five days later was found to have a blood urea of 250 mg.%; on admission to our unit it was 325 mg.%. He was dialysed continuously for 4 days \(4\frac{1}{2}\) hours, and you can see what happened to his blood urea, and his potassium. He was taken off the Minicoil after 4 days \(4\frac{1}{2}\) hours because of increasing signs of intra-abdominal haemorrhage, and he went to theatre where he had a laparotomy. A delayed rupture of the right kidney was discovered. This did not require suture, but the haematoma was removed. Blood urea at the end of dialysis was 55 mg.% and next morning 220 mg.%.

He was put on the Minicoil again for 4 days \(6\frac{1}{2}\) hours, but this time we were not lowering the blood urea below 100 mg.%. We were getting 30 mg. to 40 mg. per cent of urea in the bath at a flow rate of 200 ml./min., and we felt that the necrosis of his right leg was the major factor in his hypercatabolic state. He went back to theatre, had a below-knee amputation, blood urea went up to 220 mg.% again and he was put back on the machine again for 3 days 11 hours. When we took him off the blood urea rose, but not quite so rapidly. The urine volume came up, and then we gave him a
Figure 1. Plasma potassium, plasma urea and urine output in a patient with post-traumatic renal failure, treated with the minicoil.

(Reproduced by permission of the Editor, British Medical Journal)
20-hour session at the beginning of the diuretic phase so that he would have a better starting point for diuresis. That was entirely Minicoil dialysis in a hypercatabolic post-traumatic renal failure.

A.C. KENNEDY (Glasgow): One might say that this patient monopolized this particular equipment for something like three weeks, judging by the chart we have seen. What would happen if, during this time, one had an admission rate of two or three such patients per week coming into the unit?

G. MITCHELL (Aylesbury): This patient was dialyzed in the ward in his own bed, and we have two Kolff twin-coils in our dialyzing room. We were, in fact, dialyzing other patients whilst he was being treated. At any one time we might have one Kolff twin-coil dialysis going, a peritoneal dialysis and a Minicoil dialysis in operation.

E. KULATILAKE (London): You have dialyzed patients for over 4 days with a roller pump. How much damage is there to the red cells? Do you have to give them packed cells?

G. MITCHELL (Aylesbury): This particular patient was bleeding in the early phase into his retroperitoneal space. Following that there were circulating toxins from necrosing muscle, so that falls in haemoglobin, a rise in serum bilirubin and so on, reflected a collection of things that were going on simultaneously. In that particular patient we were not able to assess what damage was caused to his red cells by the roller pumps. We have not found it significant in 24, 36 or 48-hourly continuous dialysis. Whether or not it would have made any difference in that particular patient if he had not gone to theatre and we had not dialyzed him continuously for a fortnight, I cannot tell you. We have not treated such a patient yet.

THE CHAIRMAN: I just thought you might be interested to hear the result of the investigation we have conducted here. This is a distillation of the European work; I have excluded Australian results and I have also excluded Dr. Boen's results - I am sure he will not mind. I just thought we would get the results from the members of the Association whose work has been in Europe. There are 20 centres involved, and we represent 79 patients, who are alive today, 49 because of intermittent dialysis, 3 because of peritoneal dialysis and 27 because of transplantation.

I should now like to ask Professor Formijne to close the meeting.

P. FORMIJNE (Amsterdam): Our Conference is almost at its end; I want to thank you all again for your presence here, and especially those who have made an active contribution by reading a paper, giving a demonstration or taking part in the discussion. (Repeated in French).

Now I have the great honour and pleasure to ask Mr. Swinney to take over the Presidency of this Association.

J. SWINNEY (Newcastle-on-Tyne): Ladies and Gentlemen, the organization of this Conference has been so good that this is the first period in which I have received no detailed instructions as to what I should do next!

I am sure, however, that my first duty as President should be to thank on your behalf Professor Formijne, Dr. Drukker and their colleagues for the excellence of the Conference which we have enjoyed during the last two days. (Applause).
I would also like to express a special word of thanks on your behalf to Dr. Drukker's band of charming young ladies who welcomed us at the registration desk, who have handed us the microphones and who have taken part in recording the proceedings of this Conference. (Applause).

In these days Amsterdam seems to be a common starting point for journeys all over the world by sea or air, and it is appropriate I think that we should have had the First Congress of this new Association in this beautiful city. It has certainly been well and truly launched and will, I am sure, be very successful in the future.

The significance of this meeting to me, as a surgeon, has been that we have had enthusiastic surgeons, physicians and scientists gathered together in one place to discuss the problems of renal disease and try to solve them. I am sure that the future attack on these problems depends on team work of the members of associations like this, and that this is the way we should tackle them.

It remains for me to say how deeply conscious I am of the honour you have done me in electing me your President for the ensuing year, and I take it also as a compliment to the team of colleagues with whom I am associated in Newcastle. We look forward to seeing you all in Newcastle on 17th and 18th September next year, and we will do our best to emulate the success and the arrangements of this Conference. (Applause).

THE CHAIRMAN: That closes the meeting.