SOME STUDIES IN COOLING AND PERFUSION OF KIDNEYS

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The method described by Farrant (1965) for preservation of smooth muscle by freezing seems in principle to be the most promising approach to the freezing of solid organs such as the kidney for the purpose of long-term preservation for transplantation. Inevitably the use of this method for freezing a kidney would demand long periods of perfusion at low temperatures in order to expose the organ to graded concentrations of protective agent.

At the present time the protective agent most likely to be of value for kidney freezing is dimethylsulphoxide (DMSO). We have shown previously that rat kidneys tolerate direct exposure to 20% DMSO and supercooling to —10°C (Keeler et al., 1966). Direct exposure of rat kidneys to 20% glycerol caused complete necrosis and loss of function (unpublished findings).

We have designed an apparatus in which dog kidneys can be perfused for long periods with accurate control of temperature, flow rate, and perfusion pressure. A series of dog kidneys was perfused with graded concentrations of DMSO and cooled to various temperatures below 0°C without freezing. When re-implanted in the neck as autografts, they retained an intact blood supply but they all suffered gross structural damage and failed to regain any function.

We have now shown that kidneys do not even tolerate prolonged cold perfusion with conventional physiological fluids, although it is well known that after brief initial cooling by perfusion kidneys can be stored in a viable state for up to 20 hours at 4°C.

It seemed possible that the damage caused by prolonged cold perfusion might be due to loss by passive diffusion of essential intracellular cations. The sodium pump mechanism is known to be inactive at low temperatures. If such losses occurred it seemed possible that they would be increased by perfusion with DMSO which might be expected to increase cell membrane permeability (Mussett et al., 1965).

To investigate the nature and extent of electrolyte losses at low temperatures, rat kidneys were perfused at 0°C at a pressure of 120 mm Hg for periods up to 3 hours with 0.9% NaCl solution.

A further series of kidneys was similarly perfused with 0.9% NaCl containing 20% DMSO. At the end of perfusion the kidneys were analysed for their content of water, sodium, potassium, magnesium, and phosphate. These figures were compared with the normal content of fresh, unperfused kidneys. The results obtained are shown in Table I. The column headed 'normal kidney' shows the absolute amounts per gram of dried fat-free solids. The other columns show percentage gains or losses.

It will be seen from these results that during perfusion with saline at 0°C potassium loss occurs rapidly—approximately a 50% deficit after half an hour. Magnesium loss occurs much
TABLE 1

<table>
<thead>
<tr>
<th></th>
<th>Normal kidney</th>
<th>1/4 Hour</th>
<th>1 Hour</th>
<th>3 Hours</th>
<th>3 Hours 20% DMSO</th>
</tr>
</thead>
<tbody>
<tr>
<td>H₂O</td>
<td>3.79 ml/g</td>
<td>+ 81</td>
<td>+ 67</td>
<td>+ 73</td>
<td>+ 40</td>
</tr>
<tr>
<td>Na⁺</td>
<td>277 μEq/g</td>
<td>+174</td>
<td>+150</td>
<td>+172</td>
<td>+136</td>
</tr>
<tr>
<td>K⁺</td>
<td>328 μEq/g</td>
<td>- 49</td>
<td>- 56</td>
<td>- 66</td>
<td>- 74</td>
</tr>
<tr>
<td>Mg²⁺</td>
<td>88.2 μEq/g</td>
<td>nil</td>
<td>nil</td>
<td>- 16</td>
<td>- 52</td>
</tr>
<tr>
<td>PO₄⁻</td>
<td>1230 μEq/g</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>- 24</td>
</tr>
</tbody>
</table>

more slowly, there is no significant loss after one hour but a 16% loss after 3 hours. There is no loss of phosphate. Perfusion with DMSO greatly increases the loss of potassium and magnesium, and after 3 hours there is a 24% loss of phosphate.

It now seemed important to know whether acute losses of intracellular cation were recoverable, particularly potassium, which had been shown to leave the cells so rapidly. Therefore in the next group of experiments pairs of rat kidneys were perfused in situ with cold NaCl solution for periods ranging from 10-30 minutes, after which one of the pair was removed for analysis of potassium content while the blood supply was re-established in the other. This second kidney was analysed for potassium content after variable periods of return of blood flow. The results showed that potassium deficits of 30-50% were completely replenished within two hours. These kidneys could be assumed to have been still viable; and some other factor must therefore be invoked to explain the irreversible damage caused by more prolonged cold perfusion. The irreversible damage might be explained by more profound potassium depletion or by an associated magnesium depletion, or there may be several factors concerned.

Finally we wished to know whether damage during prolonged cold perfusion could be prevented by using a solution having a high potassium and magnesium content, since this would presumably prevent cellular depletion.

To answer this question dog kidneys were perfused for three hour periods in the cold perfusion chamber at 0°C before being re-implanted in the neck as autografts. The kidneys were assessed after transplantation by direct collection of urine from the skin ureterostomy, and biopsy with a Menghini needle. In the case of dogs whose cervical kidneys recovered good function after grafting, contralateral nephrectomy was performed five days later. Renal function was then assessed by serial estimation of blood urea and creatinine clearance.

Experiments and results

1 dog. Kidney perfused with Tyrode solution.

There was no secretion of urine at any stage. The biopsy taken 24 hours after grafting showed advanced cellular disintegration, and when the kidney was removed 5 days after implantation it was found to be completely necrotic.

2 dogs. Kidneys perfused with a solution resembling Tyrode solution except that the predominant cation was potassium (154 mEq/litre) instead of sodium.

These kidneys initially produced some urine after transplantation but never in measurable quantities. Histologically they showed better preservation than the Tyrode perfused kidney but they were still grossly abnormal, the main feature being thinned and dilated tubules containing amorphous casts.
SOME STUDIES IN COOLING AND PERFUSION OF KIDNEYS

1 dog. Kidney perfused with a solution containing:

K⁺  150 mEq/litre
Mg ++  40 mEq/litre
Na⁺  10 mEq/litre
Ca ++  2 mEq/litre

This kidney developed good function after grafting and the biopsy showed relatively normal histology at 5 days. Contralateral nephrectomy was followed by a rise in blood urea to 190 mg% but this later settled to between 60 and 100 mg% with a creatinine clearance of 35 ml/min.

1 dog. Kidney perfused with a solution containing:

K⁺  150 mEq/litre
Mg ++  50 mEq/litre
Na⁺  10 mEq/litre
Ca ++  2 mEq/litre

The concentration of potassium and magnesium in this solution must approximate closely to the mean concentration in renal intracellular fluid.

This kidney had a normal output of urine on the first postoperative day, and histology was normal on the fifth day. There was a transient rise in blood urea to 73 mg% following contralateral nephrectomy but this later settled to a normal level. The creatinine clearance was 45 ml/min.

2 dogs. Kidneys perfused with a solution containing:

Na⁺  154 mEq/litre
Mg ++  50 mEq/litre
Ca ++  2 mEq/litre

There was an immediate but impaired return of function in both kidneys. The first animal had to be killed because of intestinal obstruction but the biopsy on the fifth day was normal apart from interstitial oedema.

Two biopsies taken from the second dog’s kidney on the 6th day showed patchy necrosis with fairly normal intervening tissue. Renal function was slow in improving and contralateral nephrectomy had to be delayed for 14 days, after which function was not adequate to support life.

Finally we have tried using a high potassium high magnesium solution (K⁺ 150 mEq/litre, Mg ++ 50 mEq/litre) as a vehicle for DMSO in an experiment in which the concentration of DMSO was gradually increased to 20% v/v and the kidney temperature was lowered to —6°C. Re-warming was an exact reversal of the cooling process. The total period of DMSO perfusion was only 80 minutes, but there was no secretion of urine after grafting. Histological examination of the kidney when it was removed on the 6th day revealed grossly thinned and dilated tubules.

Discussion

Our results show that the damage inflicted on kidneys by prolonged cold perfusion with predominantly sodium-containing physiological fluids is related to potassium and magnesium loss. Good structure and function can be preserved by perfusing with a solution having a potassium and magnesium content approximating to that of intracellular fluid. Our experience and that of others suggest that moderate acute potassium loss such as occurs with brief intravascular cold perfusion is well tolerated. Profound potassium loss or any significant degree of magnesium loss may each cause damage even in the absence of any deficiency of the other cation. It seems that both these cations are necessary for protection during prolonged perfusion, though magnesium deficiency is more destructive than potassium deficiency.

The damage produced by perfusion with DMSO, even in the absence of potassium or
magnesium deficiency, may be related to loss of phosphate which we have shown to occur under these conditions; but there could also be loss of other essential cellular components which have not yet been studied. Clearly the mechanism of damage during perfusion with DMSO requires further investigation.

Summary and conclusions

1. Kidneys are destroyed by prolonged periods of cold perfusion with conventional physiological fluids.
2. We have shown that under these conditions cellular losses of potassium and magnesium occur.
3. These losses are increased by DMSO which also promotes a loss of phosphate.
4. In the absence of DMSO, good structure and function can be preserved during prolonged cold perfusion by use of a solution having a potassium and magnesium content approximating to that of intracellular fluid.
5. Further information is required about the mechanisms of damage during perfusion with DMSO.

REFERENCES


DISCUSSION

The CHAIRMAN: Thank you very much. Je voudrais faire une courte remarque préliminaire pour vous dire combien sont importants ces travaux destinés à trouver de meilleurs moyens de préservation des reins.

En effet, si l'on veut appliquer ce que nous avons appris ce matin au sujet de l'importance du groupage leucocytaire à la transplantation de reins de cadavres, il est évident que, dans beaucoup de cas, il faudra disposer d'un certain temps entre la mort du sujet et la réimplantation du rein, et ce sont des méthodes du genre de celles qui viennent de vous être présentées qui pourront peut-être à l'avenir nous permettre de disposer de ce temps nécessaire.

These two papers are now open for discussion.

KULATILAKE (London): Longer perfusion was carried out in 1964 by Humphries at 4°C, using balanced solutions and about 5% blood. He did nephrectomies immediately after the transplant and the dogs survived, five out of seven.

As regards the second paper, by Dr. Anderson, I would like to know his results after his renal transplantations.

LEGRAIN (Paris): Depuis quatre ans, nous avons travaillé avec plusieurs collaborateurs sur le problème du refroidissement et de la congélation du rein de rat et de chien. À Newcastle, en septembre 1965, nos échecs de congélation utilisant la perfusion d'un gaz froid par la voie vasculaire avaient été présentés. Nous avions par contre laissé apparaître un certain espoir à partir de nos premiers travaux sur le sur-refroidissement (super-cooling).

Je dois dire que, depuis, nous avons continué ce travail sur le sur-refroidissement, et que notre impression actuelle est moins favorable que ne le laissaient penser nos premiers résultats.

En effet nous avons pu obtenir des chiens vivants uniquement avec un rein sur-refroidi à —12°C, et l'urographie intra-veineuse est satisfaite. Ces résultats étaient très encourageants.

En fait, quand on multiplie les expériences, et nous avons fait actuellement une assez longue série, on s'aperçoit qu'au bout de quelques semaines, les reins sur-refroidis sont l'objet d'une pyélo-néphrite chronique atrophique qui semble mettre en discussion l'utilité réelle de la méthode de conservation par sur-refroidissement.

Je tiens à dire que les reins de chien sont sur-refroidis à —12°C et en aucune façon à —79°C. Sinon la technique est très voisine de celle qui a été proposée par le premier orateur.

The CHAIRMAN: Y a-t-il d'autres questions?

ALEXANDRE (Heverle): I would like to ask Dr. Uldall how much time did he preserve his kidneys after perfusion, and what was the concentration of potassium. I did not get that in the paper.

The CHAIRMAN: Pas d’autres questions?

ULDALL (Newcastle): If I could answer Dr. Kulatilake’s question first about the work done in America, I think there is an essential difference between aiming to preserve kidneys with a blood containing solution at 4°C and perfusing entirely ischaemic kidneys with a pure electrolyte solution at 0°C. Although there does not appear to be much difference in temper-
nature, I think one has to assume that the blood perfused kidneys were able to maintain some metabolic activity which helped them to preserve their magnesium content. This is how I would interpret the American work. Does that answer your question?

**Kulatilake (London):** Humphries originally used artificial solutions, and he started using blood purely because they did not do as well. Once he started to use some blood in his perfusion system, he got much better results.

**Uldall (Newcastle):** I would like to ask how long he perfused kidneys without blood, with a pure electrolyte solution? What was the state of the kidney at the end of it. And at what temperature did he do it?

**Kulatilake (London):** He used the same type of solutions you have used and the same type of damage occurred at the end of it. The results were not as good. But after using blood, the results improved tremendously and in fact he summarized that by saying that it was the red cells in the blood which gave the tremendous advantage he had.

**Uldall (Newcastle):** I think you have answered the question yourself.

The other question, from Louvain I think, was for how long were our kidneys ischaemic and what was the potassium concentration used in the perfusing solution.

The average length of ischaemia of our dog kidneys was four hours from start to finish. The average period of warm ischaemia was only three minutes.

We now recommend in the final high potassium high magnesium solution a potassium content of 150 milli-equivalents per litre, and a magnesium content of 50 milli-equivalents per litre. This has given the best results so far, but possibly further assessment will be required to assess the benefit of marginal differences in these two electrolytes.

**The Chairman:** Thank you very much, Dr. Uldall. Dr. Kemp, will you give any closing remarks?

**Kemp (Leeds):** Until now, we have only made acute experiments. So I cannot really answer the question concerning the function of our kidneys in the thirty cats and in the dogs.

The only thing I can say is that they produced these few millilitres of urine per hour for the first hours after transplantation.

Of course, the next step is to make long-term studies.
FAST AND SLOW HAEMODIALYSIS COMPARED IN PATIENTS ON REGULAR
DIALYSIS

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Slow haemodialysis employing the Kiil kidney with Cuprophane membrane for 24 to 30 hours
per week will consistently maintain anuric patients in reasonable health over a period of years
(Pendras and Erickson, 1965; Comty et al., 1966; Evans et al., 1966; Dalla Rosa et al.,
1966). This cannot be said with equal confidence about any other system of dialysis. However,
slow haemodialysis in hospital is extremely expensive in staff time; in the home it
makes great demands on the family unless it is controlled by expensive monitoring equipment
which is in short supply and requires expert servicing. It is therefore likely to remain available
only to a minority of patients with chronic renal failure.

Faster haemodialysis with larger dialysers has been successful for the few months necessary
to prepare patients for transplantation (Dunea et al., 1965; Hamburger et al., 1965;
Alexandre et al., 1966) but it has been disappointing when used in the longer term for regular
dialysis (Barber et al., 1963; Bunn et al., 1964; Alberts and Drukker, 1965; Maher et al.,
1965), though the method has never been evaluated under ideal conditions. One possible
explanation for the limited success of fast haemodialysis in the past is that large dialysers
have been used for excessively short times chosen arbitrarily from analogy with acute renal
failure (e.g., 6 hours with the Twin Coil) or controlled by blood urea, which is a poor index
since urea is removed preferentially during fast dialysis. We therefore believe that a con-
trolled trial of fast and slow haemodialysis is required and that the control of all accepted
indices of uraemia should be equally good by both methods. We describe here our efforts to
develop a safe fast dialysis system suitable for such a controlled trial.

STUDIES WITH THE 4-LAYER KIIL DIALYSER

The only method of improving dialysance readily available to us was an increase in surface
area. Accordingly we used two standard 2-layer Kiils in parallel to give a ‘double Kiil’ with
2 sq.m. of Cuprophane membrane. This was primed with one unit (approx. 500 ml) of whole
donor blood.

We estimated that with our standard blood flow of about 250 ml/min. (achieved with the
aid of a blood pump) this double Kiil would achieve about the same reduction of plasma
urea, creatinine and urate levels in 9 hours as the 2-layer Kiil does in 12. This proved to be
correct. The two systems were compared on 3 patients who happened to be above average
weight so that end of dialysis levels were less favourable than usual. The weighted averages
on the 3 patients are shown in Table I.

However, end of dialysis levels are not the best guide to the efficacy of haemodialysis; a
post-dialysis rebound occurs due to redistribution between plasma and intracellular fluid
which is negligible after slow dialysis but considerable after very fast dialysis as used in acute
renal failure. The rebound after 9-hour dialyses with the double Kiil was estimated by taking
a 3-hour post-dialysis sample and comparing the rate of rise in the first 3 hours with sub-
Fast and Slow Haemodialysis Compared in Patients on Regular Dialysis

Table I
Post-dialysis plasma levels as percentage of pre-dialysis with single and double Kiil (compared on the same 3 patients).

<table>
<thead>
<tr>
<th></th>
<th>Single Kiil (12 hour)</th>
<th>Double Kiil (9 hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dialyses</td>
<td>Post D. level (%) Pre D.</td>
</tr>
<tr>
<td>Urea</td>
<td>21</td>
<td>39</td>
</tr>
<tr>
<td>Creatinine</td>
<td>21</td>
<td>48</td>
</tr>
<tr>
<td>Urate</td>
<td>22</td>
<td>43</td>
</tr>
</tbody>
</table>

Fig. 1. Post-dialysis rebound following 9-hour dialysis with double Kiil.

sequent rate of rise to the next pre-dialysis sample. Previous studies had shown that although the rebound is not complete (particularly for creatinine) by 3 hours, the majority of it occurs in that period. It is clear from Figure 1 that the rebound was sufficiently slight that it did not seriously affect the comparison of this system with slow haemodialysis.

The double Kiil was used in 65 dialyses to assess whether the greater speed of haemodialysis introduced a prohibitive increase in side effects. Our standard dialysate glucose concentration of 0.5% was used and there was a consistent fall in plasma osmolality during dialysis which averaged 23 milliosmols per kg. We were therefore particularly concerned about a possible increase in symptoms of dysesthesia. Consequently continuous EEG monitoring was carried out during about one third of the dialyses by both systems.

EEG method

S.L.E. chlorided silver cup electrodes were left in place throughout dialysis and were connected to an 8-channel Offner type T EEG machine. A B.N.I. type twin-channel analyser was tuned to frequencies 2-14, 16, 18, 20, 22, 24, 27, 30, 33 c/s with bandwidth adjusted to be 50% down at mid-frequency. It was calibrated with a Solatron L.F. oscillator.
Initially a full 10-20 electrode placement was used but various simplifications of this system were employed in most cases. The results shown in Table II were based on analysis of one lead only in each case—the right occipital, left occipital or mid-occipital channel derived in a unipolar manner with respect to a contralateral under-ear reference electrode. All recordings were taken at a standard sensitivity of 100 μV/cm. The sensitivity of the analyser was adjusted until a moderate amplitude write-out was obtained, whenever possible maintaining the maximum deflection within the range 4-7 cm to minimize errors due to non-linearity.

Recordings were assessed on the basis of a single 10 second epoch of analysis from an occipital channel beginning 10 seconds after eye closure. When the patient’s EEG appeared fairly constant in characteristics little variation was noted during subsequent epochs of analysis; when this was not the case the epoch showing most alpha or quasi-alpha rhythm was selected. This was usually coincident with minimal evidence of drowsiness.

The epoch in question was examined for the dominant frequency (F₀) and the amplitudes (A₀; A₁; A₂; A₀; A₁; A₂) of the write-outs for it and the two frequencies on either side were measured. The following parameters were calculated:

Mean amplitude: \[ \text{Mean amplitude} = \frac{A_{-2} + A_{-1} + A_0 + A_1 + A_2}{5} \]

Mean frequency: \[ \text{Mean frequency} = \frac{F_{-2} \cdot A_{-2} + F_{-1} \cdot A_{-1} + F_0 \cdot A_0 + F_1 \cdot A_1 + F_2 \cdot A_2}{A_{-2} + A_{-1} + A_0 + A_1 + A_2} \]

These parameters were plotted on a time scale during dialysis. A subjective assessment was also made of the abnormality of each tracing on a five point scale: normal; slightly abnormal; moderately abnormal; very abnormal; grossly abnormal. This subjective assessment took account of focal and sporadic abnormalities which may be missed on the frequency analysis.

**Results**

All patients showed some degree of deterioration in the EEG during dialysis by both systems. The commonest finding was slowing of dominant posterior rhythm which became evident after the first hour. Later changes comprised the further slowing of this rhythm and the emergence of independent slow activity. Two subjects showed occasional epileptiform abnormalities. Striking K complexes were sometimes seen. All except the most severe abnormalities could be reduced by eye-opening or by any stimulus that aroused the patient’s interest. During sleep the EEG became less abnormal.

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**TABLE II**

**EEG abnormalities produced during dialysis**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Single Kil</th>
<th>Double Kil</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.K.</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>J.K.</td>
<td>+++</td>
<td>+++++</td>
</tr>
<tr>
<td>W.T.</td>
<td>+</td>
<td>+++++</td>
</tr>
<tr>
<td>J.W.</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>M.W.</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>T.Y.</td>
<td>++</td>
<td>+++++</td>
</tr>
</tbody>
</table>

+++ Slight abnormality
+++ Moderate abnormality
++++ Marked abnormality
+++++ Severe abnormality

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The degree of deterioration during dialysis was generally 1-2 points on the scale with a single Kiil and 2-4 points on a double Kiil. The results in 6 patients compared on the two methods are shown in Table II. The lowering of mean frequency was also greater on the double Kiil than the single Kiil (Table III; Figure 2).

However, it is uncertain whether more rapid dialysis alone was responsible for these more obvious EEG changes. Broad correlations were found between EEG abnormality and loss of weight and rise in body temperature—both of which were commoner with the double Kiil. Particularly striking changes occurred in one patient (J.K.) who had febrile reactions to transfused blood. The onset of abnormality in the EEG at the time of a rigor in the fourth hour of dialysis, and the reversion towards normal when the temperature fell in the last 3 hours of a 12-hour dialysis on the double Kiil are shown in Figure 3. The fact that blood priming was necessary with the double Kiil, but not with the single Kiil, may therefore be responsible for much of the difference in EEG recording between the two methods.
In one or two patients the EEG deterioration during dialysis with the double Kiil was diminished by increasing the dialysate glucose concentration to 1.5%.

The clinical syndrome of dys-equilibrium did not accompany these EEG changes even in the worst affected individuals. However, two other complications were encountered which proved of much greater importance in limiting the use of the double Kiil.

1. Symptomatic hypotensive episodes occurred in more than half of the dialyses even when hourly bed weighing and saline replacement were carried out. Hypotension was always corrected by further infusion of saline and it is clear that these episodes are related to the extremely high rate of ultrafiltration, which averaged more than 500 ml/hour and sometimes rose to 900 ml/hour. These very high rates are no doubt due in part to our use of a blood pump and a constant negative pressure of about 70 mm Hg on the dialysate side. However, in a few runs without the blood pump the ultrafiltration rate remained very high and unpredictable. To prevent unpleasant hypotensive attacks it would therefore be necessary to monitor weight or blood pressure more often than once per hour and we feel that this is impracticable, particularly overnight or in the home. Presumably this problem will be encountered with any dialyser that utilizes 2 sq.m. of Cuprophan.

2. Transfusion requirements doubled during the period that the double Kiil was used. This probably reflects the need for blood priming and the difficulty in washing out the double Kiil at the end of dialysis. However, the effect of a large membrane area on transfusion requirements deserves further study.

STUDIES WITH THE CUPROPHANE CHRONACOIL

Having accepted the need for a ‘fast dialyser’ with less than 2 sq.m. Cuprophan membrane area and no blood priming, we turned our attention to the Chronacoil. With its viscose membrane this coil gives a slightly better urea dialysance than the Kiil but not enough to justify its use for much less than 12 hours twice a week.
Summary

The 2 sq.m. Kiil dialyser reduces plasma urea, creatinine and urate as far in 9 hours as the 1 sq.m. Kiil does in 12 hours under comparable conditions. Since the rebound after 9-hour dialysis is slight the two systems could be used for a comparison of slow and fast haemodialysis. However, the incidence of side effects—particularly hypotension—has led us to abandon the 2 sq.m. Kiil.

A limited experience with the Cuprophan Chronocoil suggests that this will be equally efficient for 9-hour dialysis without causing unpredictable hypotension and without the need for blood priming.

REFERENCES


