EXPERIENCES IN THE TREATMENT OF RENAL FAILURE BY PERITONEAL DIALYSIS IN MEDICAL UNIT II

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The property of the peritoneal membrane as a semi-permeable membrane was demonstrated in the 1890's by physiologists. In 1923, the first attempt at peritoneal dialysis on a human patient was made by Ganter. However, because of technical difficulties and the development of the artificial kidney, further progress was slow. In 1951, Grollman and others described a system of closed intermittent peritoneal dialysis and with minor modifications this is the method widely used now.

In peritoneal dialysis, the peritoneal membrane is used as a dialysing membrane for exchange between body fluids and an artificial fluid introduced into the peritoneal cavity. An adult's peritoneal surface is about 22,000 sq. cms. which is approximately the tubular filtration area.

By this procedure, it is possible to remove certain waste products of the body, excessive water, and some ingested poisons and to correct body electrolyte levels. Thus, it can be used in acute and chronic renal failure, hyperpotassaemia, intractable oedema and poisoning by dialysable poisons such as barbiturates. Recent abdominal surgery is not a contraindication. It is not used in localised intra-abdominal sepsis for fear of spreading the infection, but it has been used in treating generalised peritonitis.

The bladder is emptied first. The abdomen is prepared as for a surgical operation. The site of puncture can be any of the sites used for abdominal paracentesis. We use a point in the midline one-third to half way from umbilicus to pubic crest.

Under local anaesthesia, a small incision is made through the skin, subcutaneous tissue and linea alba. The trocar is pushed through this incision to pierce the peritoneal lining. A "give" is felt on entering the peritoneal cavity. The catheter is introduced via the trocar to point towards the pelvic cavity or paracolic gutter.

The trocar is then removed and a purse-string stitch put in to pull the tissues tightly around the catheter. The catheter is tied to this stitch. The catheter is connected to the rest of the set.

2 litres of dialysing fluid are used in each exchange. An average dialysis done here consists of 30-40 exchanges and lasts 48-72 hours. Usually hourly exchanges are done. We add heparin and tetracycline to each exchange.

The composition of the dialysing fluid introduced into the peritoneal cavity contains all the essential electrolytes (*viz.* sodium, potassium, calcium, magnesium and chloride). Dextrose in the solution is made up in two concentrations—isotonic (1·5%) and hypertonic (7%). Hypertonic solutions are used in the presence of excess fluid in the body. The pH of the dialysing fluids is around 5·0.

In medical Unit II up to June 30th 1965, 49 dialyses have been done on 42 patients. They include chronic and acute renal failures, hypnotic poisonings and excess water. Of the 42, 12 (29%) are still alive. (Fig. 1).

The drop in blood urea depends on the duration of dialysis and the number of exchanges made. (Fig. 2). In hyperpotassaemia, serum potassium drops rapidly when no potassium is added to the dialysing fluids. Normal potassium levels can be obtained in 5 hourly exchanges. (Fig. 3). In cases with low potassium levels, despite 4 mEq/L potassium in the dialysing fluids, the serum potassium rises very slowly. (Fig. 4).

Urea diffuses rapidly through the peritoneum. Usually in 2 hours, there is equilibrium between blood and peritoneal urea. In hourly exchanges, we have obtained from 67-100% equilibrium of peritoneal urea with blood urea. (Fig. 5).

PERITONEAL DIALYSES UP TO JUNE 30th, 1965.

Diagnosis	No. of patients	No. of dialyses	No. Alive	No. Dead
Chronic renal failure	20	24	—	20
Acute renal failure	16	19	7	9
Poisons	4	4	4	—
Excess fluid	2	2	1	1
Total	42	49	12	30

Failed dialyses: 4

Figure 1

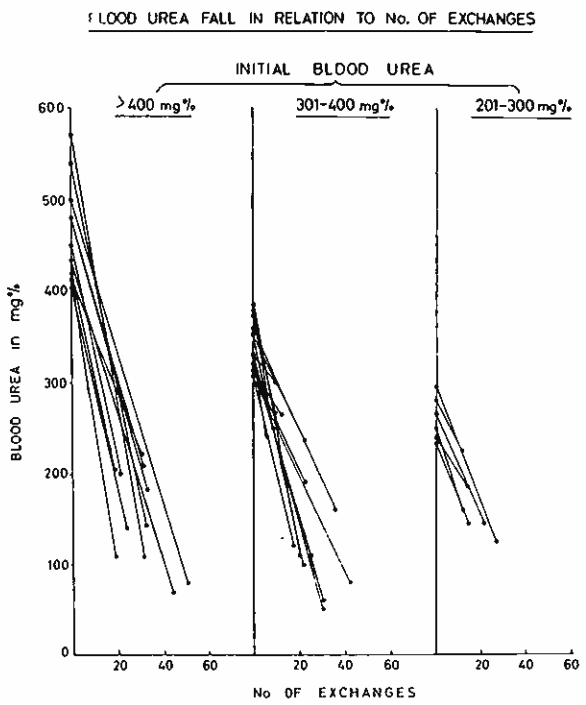

Figure 2

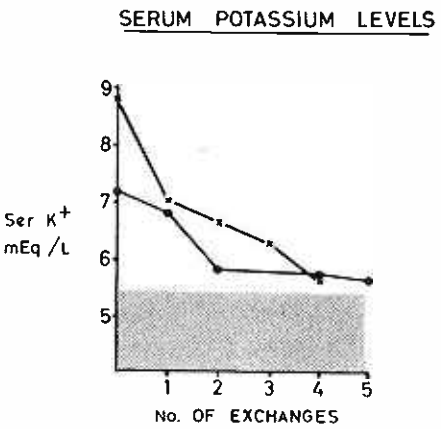

Figure 3

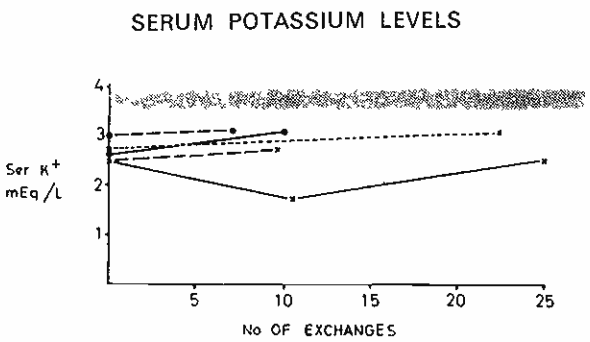

Figure 4

Total urea removed varies with the number of exchanges and the total duration of dialysis. We have removed from 55 G. to almost 200 Gms. urea. (Fig. 6).

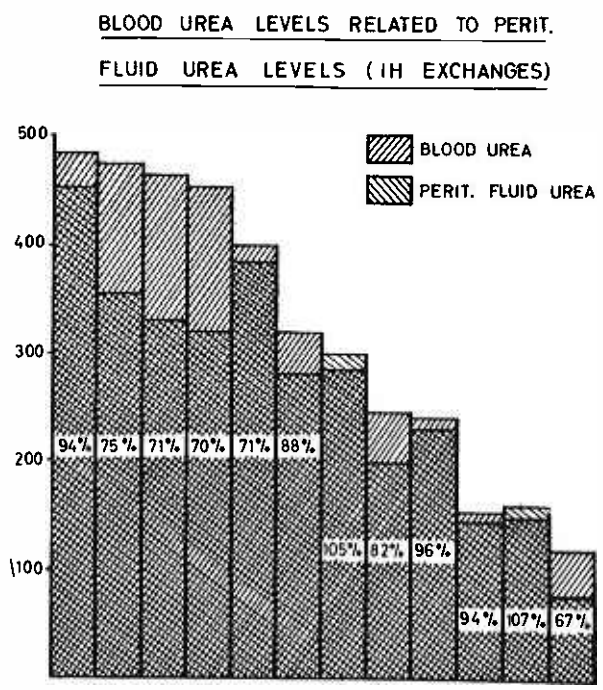

Figure 5

Total Urea Removed	Init. urea mgs.%	Hrs. Dialysis / No change	End urea mgs.%
193 Gms.	500	75H / 51E	82
178	364	47H / 36E	163
149	572	48H / 31E	110
133	450	33H / 21E	202
115	312	62H / 43E	50
99	315	54H / 34E	68
87	320	29H / 25E	110
82	237	29H / 14E	184
72	340	52H / 23E	96
62	330	42H / 31E	48
64	420	27H / 19E	110
62	235	22H / 14E	143
58	313	19H / 11E	258
55	250	24H / 12E	163

Figure 6

CHRONIC RENAL FAILURE

24 dialyses were done on 20 patients. There were 12 chronic glomerulonephritis (5 confirmed at necropsy), 7 chronic pyelonephritis (6 confirmed at necropsy), and 1 polycystic kidneys diagnosed at laparotomy. (Fig. 7).

They were all very ill and clinically were estimated to be unable to survive more than 2-3 days without correction of gross biochemical abnormalities. All but 3 had blood urea levels of 300-500 mg.%. Serum potassium were mostly normal (11), or low (11). One was 6·3 mEq/L and one was not known. All were acidotic and had low alkali reserves; some were corrected with intravenous sodium bicarbonate drip before start of dialysis. All were anaemic; all but two had Hb. levels of 6-7·5 G.%. In none was there a definite history of an acute factor precipitating them into severe renal failure.

Thus, it is not surprising that mortality was 100%. 10 died during or within 24 hours of dialysis. 5 died within one week and only 5 lived 2 weeks or more; of these 1 lived 23 days and another 25 days.

From this it seems pointless to dialyse chronic renal failures except when they have been leading fairly normal lives and an acute episode aggravates their underlying renal disease and threatens life. An acute episode may be any factor aggravating renal function, for example, dehydration, infection, heart failure, etc.

Where the differentiation between acute and chronic renal failure is uncertain or where it is felt that there may have been an acute temporary insult to kidneys already damaged, it is worthwhile to dialyse and tide the patient over while investigating further to ascertain the renal state.

At the moment, we are unable to cope with regular repeated dialyses for chronic progressive renal failure. Renal transplantation cannot be envisaged for some time and thus it is hardly practical to dialyse chronic renal failures as yet.

ACUTE RENAL FAILURES

19 dialyses were done on 16 patients. They are subdivided according to their aetiology. (Fig. 8).

2 had acute glomerulonephritis. Prognosis here is known to be poor even with dialysis. 1 died soon after insertion of the 1st exchange, and the other during the 18th exchange.

CHRONIC RENAL FAILURE

Diagnosis	No.	(Postmortem)	Time of death in relation to dialysis			
			During Dialysis	Within 24 hours	Within 1 week	2 weeks or more
Chronic glomerulonephritis	12	(5)	4	1	3	4 (23 days)
Chronic pyelonephritis	7	(6)	4	—	— 2	1 (25 days)
Polycystic kidneys	1	(—)	—	1	—	—

Figure 7

ACUTE RENAL FAILURE

Diagnosis	No. of patients	(Postmortem)	No. of dialyses	No. Alive	No. Dead
Acute glomerulonephritis	2	(1)	2	—	2
Hepato-renal (3 leptospirosis)	5	(3)	5	2	3
Obstetrical complications	3	(—)	4	2	1
Traumatic shock	2	(1)	3	—	2
Wasp stings	1	(1)	1	—	1
Others	3	(—)	4	2	1

Figure 8

POISONS

Type of poison	No.	No. of dialyses	No. Alive
Barbiturate	3	3	3 (997 mg. or 15.0 gr. sodium amytal)
Glutethemide (Doriden)	1	1	1

Figure 9

Excess Fluid.

	N 40179	N 40657
Fluid removed in ccs.	12,700	7,500
No. of exchanges	37	34
Hours of dialysis	77	53½
Blood urea in mg. %	175 → 64	315 → 68

Figure 10

BEFORE DIALYSIS

AFTER DIALYSIS

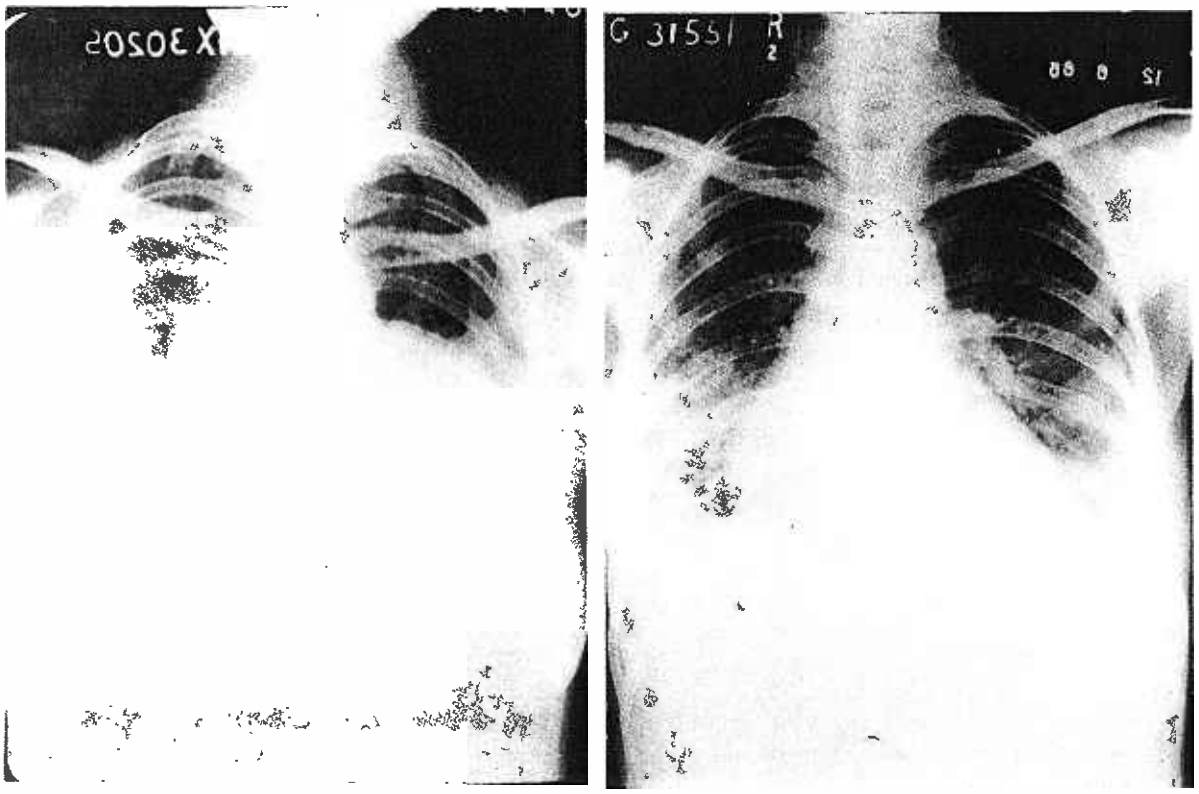

Figure 11

5 were hepato-renal failures. The 2 who survived had positive titres for leptospira. Of the 3 deaths, 2 were during dialysis—at the 9th and 12th exchanges. Both had renal biopsies consistent with leptospirosis, and one had suggestive titres in blood too. Post-mortem examination of the 3rd death showed nephrosclerosis, non-specific liver changes and a psoas abscess.

The obstetrical cases had eclampsia, APH and/or PPH. 2 are alive and well. The 3rd arrived moribund and she died during the 17th exchange.

Both the traumatic cases were those sustained in road accidents. Both were already in the diuretic stage of recovery from renal failure when they died. One died of extensive brain damage and the other died of infection.

1 patient with acute renal failure following wasp stings unexpectedly died about 1 hour after end of the dialysis. Post-mortem examination showed acute allergic angiitis and focal glomerulitis together with acute tubular necrosis in kidneys. The adrenal cortex showed diminished lipoid vacuolation.

Of the 3 where the aetiology is rather obscure, 2 are alive and well. The one death was due to staphylococcal septicaemia in the diuretic phase.

HYPNOTIC POISONINGS (Fig. 9)

4 cases were done of which 3 were due to barbiturate and 1 glutethemide. The most dramatic one was gasping and blue and stopped breathing soon after arrival in the ward. He was intubated and ventilated artificially. Dialysis was started within 2 hours. 22 exchanges were done in 24 hours. 7 hours after starting, spontaneous respirations returned. 997 mg. (approx. 15 gr.) of sodium amytal was removed by the dialysis.

The other 3 cases were deeply unconscious but breathing satisfactorily. The dialysis probably brought them round quicker and so cut down on time of coma nursing care needed and complications of comatosed patients.

EXCESSIVE WATER (Fig. 10)

2 cases of nephrotic syndrome from D.L.E. were done. 7½ L and 12 L were removed. One case improved dramatically from her dyspnoea and distress. Fig. 11 shows her X-ray chest before and after dialysis.

FAILURES

There were 4. 2 were due to bleeding. Both had chronic renal failure and had low platelet counts and prolonged prothrombin times. In one, the inferior epigastric artery had been injured by the trocar.

The other 2 were due to failure of the trocar puncturing the peritoneum and so the catheter lay outside the peritoneum.

PERITONITIS (Fig. 12)

Evidence of Peritonitis

1. Cultures of peritoneal fluid - 15

	Alive	Dead	
		Necropsy	No necropsy
Positive 11	2	4 (5 cultures)	2 (4 cultures)
Negative 3	1	1	1
Contaminated 1	1	-	-

2 Necropsies - 14

No evidence of peritonitis

Figure 12

This is a complication to be avoided.

15 specimens of peritoneal fluid were sent for culture. 3 were negative and of these, 2 died but only 1 had necropsy and the peritoneum was normal. There were 11 positive cultures of which 2 are alive and well. They had no clinical evidence of peritonitis and so were not treated for it. Of 6 deaths, 4 went to necropsy and there was no evidence of peritonitis. 1 culture was reported as contaminated and this patient is alive and well. There were 9 other necropsies and no peritonitis was seen. No cultures were sent from these 9.

CONCLUSION

Peritoneal dialysis is a relatively easy procedure to perform. In Medical Unit II it has been done even by the routine staff on night duty. It is quite feasible to institute this procedure in small district hospitals too.

The results obtained are slower than those in haemodialysis. In Medical Unit II, it is possible to get a peritoneal dialysis started within half to one hour of the decision made to dialyse. Haemodialysis would need a minimum of 2 hours to get started provided blood is already available. For peritoneal dialysis no blood is required to prime the apparatus. It is possible, if necessary, to carry out peritoneal dialysis in a shocked patient.

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