

## Peritoneal Dialysis

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Peritoneal dialysis was first used for the treatment of renal failure by Ganter (1923). It never became a popular method because of the frequency of complications such as peritonitis, leakage, inadequate peritoneal drainage, fluid and electrolyte disturbances, and because of the development of haemodialysis. However, haemodialysis is a procedure which can be performed only at special centres, since it requires a trained team using rather complicated and costly apparatus. The simplicity of peritoneal dialysis still appealed to some workers, and Maxwell *et al.* (1959), described a technique using a newly designed nylon catheter and commercially prepared solutions. They claimed that the method could be undertaken in any hospital and could largely be managed by nursing staff. They reported their experience in 76 cases and showed the method to be effective and remarkably free from complications.

After 1959 the use of peritoneal dialysis in the United States rapidly increased at the expense of haemodialysis. For example, at the Peter Bent Brigham Hospital in 1959 only three peritoneal dialyses were performed compared with 85 haemodialyses, but in 1961 there were 121 peritoneal dialyses and only 37 haemodialyses (Burns *et al.*, 1962). Despite its popularity in America there have been few reports of the use of this method in this country (Miller, 1962). We would like to report our experience of peritoneal dialysis in the treatment of 30 patients with acute or chronic renal failure and one patient with refractory oedema.

### Method

The method is slightly modified from that described by Maxwell *et al.* (1959). They used the intermittent method whereby irrigation fluid is alternately run into and out of the peritoneal cavity via a single catheter. The catheter (Baxter Laboratories Ltd.), which is made of nylon, has a solid rounded end and an external diameter of 0.345 cm. It is 28 cm. long and there are 80 holes of 0.05 cm. diameter in the distal 7.6 cm. It is introduced via the cannula of a 17F trocar and cannula which has been inserted into the peritoneal cavity under local anaesthesia and full aseptic precautions. The usual site is in the midline 3-10 cm. below the umbilicus, but we have undertaken successful dialysis with catheters inserted in any convenient site in the lower abdomen. The catheter is directed either into the pelvis or towards one paracolic gutter. On occasion the catheter cannot be easily inserted to an adequate depth. In these conditions fluid can often be run into the peritoneal cavity, after which the catheter can usually be adequately positioned. The cannula is withdrawn, a purse-string suture is tied around the catheter to reduce leakage of fluid, and dressings are applied.

We used a dialysis fluid of the composition suggested by Maxwell *et al.* (Table I). It was usually obtained commercially,<sup>1</sup> but on occasion, when supplies were difficult, we were able to prepare solutions in the hospital pharmacy. To each bottle of

fluid are added heparin 1.25 mg./l. and intravenous tetracycline 12.5 mg./l. Maxwell *et al.* suggest that heparin need be added only to the first few exchanges, but we prefer to add it throughout the dialysis to prevent the formation of fibrin clots. Potassium chloride may be added to the fluid whenever indicated, to give a concentration of approximately 3 mEq of potassium per litre. We usually add potassium when the plasma level is below approximately 4 mEq/l. The fluid containing dextrose 1.36 g./100 ml. is approximately isotonic with the plasma of most uraemic patients, and when this fluid is used there is neither a gain nor a loss of irrigation solution. If the patient is overhydrated and removal of fluid is required the hypertonic solution (dextrose 6.36 g./100 ml.) can be used. When both litres in each exchange are hypertonic there is a

TABLE I.—Composition of Peritoneal Irrigation Solution

	Na mEq/l.	Ca mEq/l.	Mg mEq/l.	Cl mEq/l.	Lactate mMol/l.	Dextrose g./100 ml.	Meta- bisulphite %
Isotonic	141	3.6	1.5	100.8	44.6	1.36	0.005
Hypertonic	141	3.6	1.5	100.8	44.6	6.36	0.005

danger of circulatory collapse from excessively rapid removal of fluid from the circulation. We find that excess fluid can be removed quickly and safely, using only 1 litre of hypertonic with 1 litre of isotonic irrigation solution in each or even alternate exchanges.

Two litres of fluid, warmed to approximately 37° C., is run into the abdomen via a special pre-sterilized plastic Y-piece giving-set.<sup>2</sup> This normally takes 10 to 20 minutes. If the fluid does not run freely and there is no air-lock or kink in the tubing, the catheter is repositioned. The fluid is left *in situ* for 30 to 45 minutes and then drained by siphoning. Maxwell *et al.* suggest that the fluid is siphoned back into the two empty giving-bottles, which are now placed on the floor, but, because more than 2 litres is often recovered, we prefer to drain the fluid via a length of sterile tubing attached to the side arm of the Baxter giving-set. This has a Luer fitting and will accept any standard transfusion tubing. The fluid is collected into a sterile graduated Winchester bottle containing an antiseptic. Time for drainage is 15 to 30 minutes; a slower outflow time than this suggests obstruction either in the external tubing or in the catheter. The external tubing can become kinked under bedclothes or dressings, or blocked by air-bubbles. If the fault appears to be in the catheter, it may be repositioned, using an aseptic technique, and sterile heparinized saline may be injected down the catheter. If this is unsuccessful a new catheter can usually be easily inserted through the existing track.

The dialysis is better performed in a side-room or isolation ward, though on occasion we have dialysed patients in the open ward. Once the dialysis is seen to be proceeding satisfactorily, the management is left to the nursing staff. This usually requires one specially trained nurse. We have found it helpful to construct a chart on which the times and volume of each fluid exchange are recorded. A final column gives the dialysis

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<sup>1</sup> Peritoneal dialysis fluid can be obtained from Baxter Laboratories Ltd. or from Allen and Hanburys Ltd.

<sup>2</sup> Plexitron Y type "diancal" solution administration set (Baxter Laboratories Ltd.).

fluid balance so that dangerous overhydration or underhydration of the patient should not occur. The blood-pressure, pulse, respiration, and normal fluid balance of the patient are regularly recorded. The total volume dialysed will vary with the requirements of the patient, but it will be of the order of 40 to 100 litres over a period of 36 to 72 hours.

*Indications for Dialysis.*—Maxwell *et al.* gave as indications for peritoneal dialysis acute and chronic renal failure, intractable oedema, hypercalcaemia, and barbiturate poisoning. We have been mainly interested in the treatment of acute and chronic renal failure. The indications for dialysis in these cases have been a rising blood urea with deterioration in the clinical state, a high or rising plasma potassium, or a low total CO<sub>2</sub>.

We have usually performed peritoneal dialysis when plasma potassium reached 7 mEq/l. despite standard treatment.

*Contraindications to Dialysis.*—There are probably no absolute contraindications to peritoneal dialysis provided the peritoneum is not obliterated. Recent abdominal surgery or injury in which there is extensive damage to the peritoneum is a relative contraindication, but successful dialysis has been undertaken within three days of laparotomy by Burns *et al.* (1962) and we have successfully dialysed a patient 10 days after exploration of the abdominal aorta. Peritonitis was stated by Maxwell *et al.* to be an absolute contraindication to peritoneal dialysis, but Burns *et al.* have successfully dialysed a patient with this condition, and our experience is similar.

TABLE II

Case No.	Age in Years	Diagnosis	Volume of Dialysis Fluid (Litres)	Blood Urea mg./100 ml.		Plasma K mEq/l.		TCO <sub>2</sub> mMol/l.		Complications	Remarks
				Pre	Post	Pre	Post	Pre	Post		
1	28	Acute glomerulonephritis .. ..	64	462	240	6.2	4.6	19	26	—	Alive and well
2	30	Acute tubular necrosis .. ..	76	595	210	9.4	2.7	8	28	—	Alive and well
3	60	Anuria after abdominal aortic surgery	78	380	140	5.5	2.7	19	27	—	Haemodialysis 4 days post-op. Peritoneal dialysis 10 days post-op. Alive and well
4	72	Pyelonephritis .. ..	72	360	123	4.0	4.3	3	25.5	—	Alive and well
5	22	Bacterial endocarditis. Polyarteritis nodosa	96	392	240	5.8	4.3	15	24	Peritonitis	Died 2 days after dialysis. Peritoneum normal at necropsy
6	42	Polyarteritis nodosa .. ..	144	324	228	3.3	4.2	22	26.5	Peritonitis. Scrotal oedema	Died 2 days after dialysis. Peritoneum normal at necropsy
7	68	Resected abdominal aortic aneurysm. Renal infarction	45	420	330	5.6	4.2	16	19	Fluid retention	Technically unsatisfactory. Died (see text)
8	76	Myocardial infarct. Tubular necrosis	76	410	210	3.8	3.6	18	28	—	Died 24 hours after dialysis. Necropsy: large cardiac infarct; normal peritoneum
9	70	Acute and chronic pyelonephritis ..	68	402	96	5.8	4.2	18	26	—	Returned to work. Died 10 months later
10	74	Glomerulonephritis .. ..	48	308	107	6.6	4.7	12.5	23	—	Alive and well
11	36	Polycystic kidneys .. ..	40	360	168	3.8	3.6	18	28	—	Alive and well
12	50	Polycystic kidneys .. ..	50	306	102	5.1	3.9	21	28	—	Returned home
13	62	Prostatic obstruction ..	92	540	125	6.4	3.8	8	26	—	Transfused 4 pints (2.3 l.) blood during dialysis.
14	74	Hydronephrosis. Cerebral thrombosis	16	560	490	6.8	6.0	4	9	—	Alive and well
15	26	Glomerulonephritis .. ..	58	576	120	6.5	4.9	25	37	—	Admitted in extremis. Immediate dialysis. Died 10 hours later. Peritoneum normal at necropsy
16	44	Malignant hypertension ..	68	300	108	8.2	4.8	25	36	—	Pulmonary oedema disappeared during dialysis (see text)
17	76	Malignant hypertension .. ..	80	476	236	8.8	4.3	6.3	26	—	Died in uraemia 10 days later. Peritoneum normal at necropsy
18	53	Henoch-Schönlein nephritis	38	1,012	940	4.2	—	25	—	—	Melaena (from uraemic enteritis) before and during dialysis. Bleeding stopped on last day. Pleural and pericardial rub disappeared
19	50	Glomerulonephritis .. ..	40	395	195	5	4.5	13	25	—	Melaena before dialysis. Bled to death during dialysis. Peritoneum normal at necropsy
20	42	Malignant hypertension .. ..	58	430	190	6.5	4.2	18	26	—	Died of pulmonary embolus 8 days after dialysis. Peritoneum normal at necropsy
21	54	Chronic nephritis .. ..	62	320	150	4.8	3.9	20	29	—	Pericardial rub disappeared during dialysis on each occasion. Died in uraemia 3 weeks later. No necropsy
22	31	Chronic nephritis .. ..	72	402	204	6	3.8	16	25	Scrotal oedema	Died 6 weeks after dialysis. No necropsy
23	17	Renal cysts. Hypoplastic kidney	22	324	264	6.8	5.3	18	26	—	Died 2 weeks after dialysis. Peritoneum normal at necropsy
24	14	Nephrocalcinosis .. ..	66	306	180	5.8	4.2	22	28	—	Died 4 weeks later in heart failure. No necropsy
25	27	Chronic pyelonephritis .. ..	44	285	198	5.8	4.1	21	26	—	Died 4 weeks later in heart failure. No necropsy
26	15	Chronic glomerulonephritis ..	60	462	186	5.3	3.3	16	25	—	Renal transplant
27	46	Hypertensive nephrosclerosis	100	444	135	5.1	4.0	19	31	Peritonitis	9.5 l. excess fluid removed on each of last 2 dialyses. Peritoneum normal at necropsy
28	44	Chronic glomerulonephritis	62	456	126	5.6	3.0	21	25	—	Awaiting renal transplant (see text)
29	38	Chronic glomerulonephritis ..	60	426	130	5.2	3.3	20	28	—	—
30	68	Carcinoma of prostate. Broncho-pneumonia	64	290	92	6.6	5.1	21	29	—	—
31	80	Resistant cardiac failure .. ..	45	561	168	6.2	4.7	18	27	—	—
			84	504	120	5.0	3.8	18	32	—	Died in heart failure 5 days later. Peritoneum normal at necropsy
			108	480	124	5.4	4.6	12	26	—	Platelet count 22,000/c.mm. No bleeding. Renal transplant. Died 3 months later. Peritoneum normal at necropsy
			64	348	138	4.0	3.5	23	21	—	Died of pneumonia 24 hours after dialysis. Peritoneum normal at necropsy
			70	315	60	5.0	4.0	22	32	Peritonitis	Died in uraemia 10 days later. (See text). Areas of chronic peritoneal inflammation at necropsy
			68	380	75	5.5	4.6	21	32	—	Renal transplant. Died after homograft rejection. Peritoneum normal at necropsy
			78	384	96	5.1	3.2	20	31	—	Renal transplant. Died after homograft rejection. Peritoneum normal at necropsy
			72	424	114	5.6	3.7	14	31	—	Renal transplant. Died after homograft rejection. Peritoneum normal at necropsy
			90	420	114	6.1	4.0	16	33	—	Renal transplant. Died after homograft rejection. Peritoneum normal at necropsy
			70	370	120	5.0	4.0	21	29	—	Died 2 days after dialysis. Peritoneum normal at necropsy
			70	408	112	4.4	4.1	27	31	—	Deliberate 4.9 l. negative fluid balance (see text)
			72	400	114	5.2	3.7	20	27	—	—
			90	620	180	3.6	2.6	13	22	—	—

## Results

The diagnosis, results of dialysis, and fate of the patients are summarized in Table II.

## Technique

Of the 48 dialyses, 47 were technically satisfactory. The only unsatisfactory dialysis occurred in a patient (Case 7) who became anuric after resection of a leaking abdominal aneurysm. Five days after operation the blood urea was 420 mg./100 ml. Haemodialysis was advised, but no compatible blood was available and peritoneal dialysis was therefore attempted. There was some difficulty in inserting the catheter owing to extra-peritoneal haematoma. Once it was inserted dialysis was satisfactory, except that despite the use of hypertonic irrigation solution there was retention of fluid and dialysis was abandoned 24 hours later, when 4 of the 46 l. of fluid run in had been retained. At necropsy both kidneys showed multiple recent infarcts, the posterior peritoneum was deficient, and there was extensive retroperitoneal haematoma.

## Plasma Chemistry and Clinical State

In all cases there was not only a fall in blood urea but also a return to normal of plasma potassium and bicarbonate. The plasma sodium or chloride either remained or became normal. In nearly all cases there was a coincident improvement in the patient's clinical state, and where a pericardial rub was present before dialysis this usually disappeared.

Two cases illustrating the use of peritoneal dialysis in the treatment of acute and acute on chronic renal failure are described.

*Case 2.*—A 30-year-old man had a febrile illness three weeks before admission. For several days he had persistent hiccups and nasal and tooth-socket bleeding. On admission he was semistuporous with pulmonary oedema. His blood urea was 594 mg./100 ml. and the serum potassium 9.4 mEq/l. The E.C.G. showed gross hyperkalaemic changes. Peritoneal dialysis was started immediately. Six hours later the serum potassium was 7.6 mEq/l. and a further six hours later it was 6.4 mEq/l. with marked improvement in the E.C.G. He continued to improve after dialysis. A diagnosis of acute tubular necrosis was made by renal biopsy during recovery. He was alive and well 10 months later.

*Case 4.*—A 72-year-old man who had a carcinoma of the bladder treated by partial cystectomy gave a history of recurrent urinary infections. He was admitted semicomatose, in heart failure, and with a gross urinary infection. His blood urea was 360 mg./100 ml. and  $\text{TCO}_2$  3mMol/l. Peritoneal dialysis was performed and antibiotics were given. His blood urea fell to 123 mg. and the heart failure disappeared. Subsequent conservative management left him with a blood urea of 70-100 mg., and a year later he was back at work.

## Fluid Removal

We have found the ability to remove excess water from the patient by the use of hypertonic dialysing solutions of great value. Three cases are described to illustrate this:

*Case 31.*—An 80-year-old woman was admitted in congestive cardiac failure with ascites due to ischaemic heart disease. For two weeks she was treated with digitalis, mersalyl, ammonium chloride, chlorothiazide, and spironolactone without effect. Her ascites was drained (2.4 l.) and over the next three days there was still no response to diuretics. Peritoneal dialysis was therefore started with hypertonic irrigation solution. At the time of insertion of the catheter no ascitic fluid could be drained out. Owing to her severe dyspnoea only 1 l. of fluid was used for each exchange: 9 l. was run in and 13.9 l. recovered. During the dialysis all signs of cardiac failure disappeared except for slight swelling of the ankles which subsequently regressed. Since dialysis she has been maintained free of heart failure on digitalis and chlorothiazide.

*Case 22.*—A 31-year-old man had chronic pyelonephritis and renal failure. Renal transplantation was undertaken. While waiting for a donor kidney he was dialysed on two occasions with good results. The second dialysis was concluded three hours before operation. The peritoneum was inspected at operation and appeared normal. After rejection of the transplanted kidney six weeks later dialysis was performed on two further occasions. The indications for dialysis were a rising blood urea and overhydration, as indicated by weight gain and oedema. As he was receiving large doses of steroid hormones in an attempt to overcome transplant rejection we preferred to continue a normal protein-containing diet rather than impose strict protein-and-fluid restriction. During each of these dialyses 9.5 l. of fluid was removed, with coincident disappearance of the oedema. Death occurred six days after the last dialysis, and at necropsy the peritoneum was normal macroscopically and microscopically.

*Case 15.*—A 26-year-old woman with rapidly progressive Ellis type II glomerulonephritis was admitted with a blood urea of 576 mg./100 ml., plasma potassium 6.5 mEq/l., and haemoglobin 7.0 g./100 ml. She had severe pulmonary oedema, which was confirmed radiologically (Fig. 1). Peritoneal dialysis was started immediately, 1 l. of hypertonic dialysis fluid being used in alternate exchanges. During the first two hours of dialysis 550 ml. of fluid was removed and after 16 hours the negative balance was 1,400 ml. This was associated with a marked reduction of her dyspnoea. The total

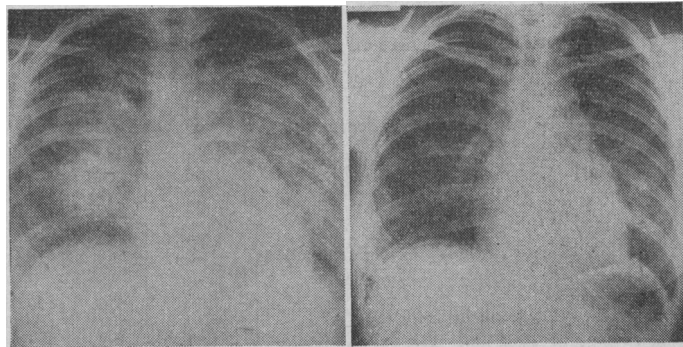


FIG. 1

FIG. 2

FIG. 1.—Case 15. Chest radiograph before peritoneal dialysis. FIG. 2.—Case 15. Chest radiograph after peritoneal dialysis.

amount of fluid removed was 4.9 l. During the dialysis 1 l. of whole blood was given without any cardiac embarrassment occurring, and a chest radiograph immediately after dialysis showed clearing of the pulmonary oedema (Fig. 2). This patient remained severely oliguric and died 10 days after a second dialysis. At necropsy the peritoneum was normal.

## Complications

*Pain.*—Many of the conscious patients complained of pain or discomfort and in about half this was severe enough to require analgesics such as pethidine. In some, 5 ml. of 1% or 2% procaine hydrochloride injected into the ingoing fluid gave relief. No patient refused a second dialysis, and one patient who was conscious throughout both a haemodialysis and a peritoneal dialysis preferred the latter.

*Scrotal Oedema.*—This was seen on two occasions and was probably due to fluid leaking into the anterior abdominal wall via the puncture wound in the peritoneum. Apart from slight discomfort no ill effect was seen.

*Infection.*—Returning irrigation fluid was cultured routinely at least daily and at the end of dialysis. In four cases organisms were grown and in two (cases 23 and 27) clinical evidence of peritonitis developed. In Case 23 *Escherichia coli* was grown from the dialysis fluid during his second dialysis. Chloramphenicol was added to the dialysing fluid and given systemically. Within 18 hours the signs of peritonitis had disappeared and the fluid was sterile. He subsequently had three successful dialyses. Case 27 was dialysed three times. During the second dialysis he developed clinical signs of peritonitis, and the irriga-

tion fluid, which became turbid, grew *Pseudomonas pyocyanea*. He was treated with intramuscular and intraperitoneal colimycin with recovery. A third dialysis eight days later was satisfactory. The patient died a further 10 days later in uraemia, and at necropsy the peritoneum showed some areas of congestion, fibrin deposition, and infiltration with inflammatory cells. In both the other cases (Nos. 5 and 6) in which dialysis fluid culture was positive renal failure was due to polyarteritis nodosa and large doses of steroids were being given. *E. coli* was grown in each case, but there was no clinical evidence of peritonitis, which may have been masked by steroids. Chloramphenicol in one case and colimycin in the other were given both intraperitoneally and intramuscularly. Both patients died within two days of completing the dialysis, three to four days after the infection was detected, and at necropsy the peritoneum was normal both macroscopically and microscopically.

### Deaths

It will be seen from Table II that 21 of the 31 patients died at varying times after dialysis. The majority of those who died had severe chronic renal disease and had not shown improvement of renal function despite dialysis and conservative measures. The eight patients (Cases 5, 6, 7, 8, 14, 16, 26, and 30) who died within 48 hours of dialysis were all extremely ill before dialysis with renal failure secondary to a potentially fatal disease (polyarteritis nodosa, myocardial infarction) or complicated by gastro-intestinal haemorrhage or severe infection. All these cases came to necropsy and in none was there any evidence that dialysis had contributed to death. In all cases dialysis had produced an improvement in the plasma urea and electrolytes.

### Discussion

Our results clearly indicate that the method of peritoneal dialysis as described by Maxwell *et al.* is a simple and effective means of treating severe renal failure. The failure of only one out of 48 dialyses also indicates that it is a reliable method. As Maxwell *et al.* and Boen (1961) point out, peritoneal dialysis is not as efficient as haemodialysis. The biochemical changes produced in 36 hours by peritoneal dialysis can be obtained in six hours by haemodialysis, but we consider that this is only an advantage in cases where there is a high catabolic rate and consequently a very rapidly rising blood urea. For most cases peritoneal dialysis has the advantage that it can be performed in any hospital by one doctor and a nurse, that the technique is simple to learn, and that no blood or blood donors are required. The method is especially useful in the treatment of elderly patients with an acute exacerbation of chronic renal failure where there would be hesitancy about transferring them to a haemodialysis centre. It is also useful for the patient who is admitted *in extremis* with renal failure due to an unknown

cause. Dialysis can be begun as soon as the renal failure is recognized and the patient's condition improved sufficiently to allow a diagnosis to be made. Cases 4 and 10 are good examples of this.

A further point in favour of peritoneal dialysis is the fact that glucose is absorbed from the irrigation solution, thus providing extra calories for these ill patients. As indicated by Maxwell *et al.* some loss of plasma protein into the irrigation solution occurs, but this is of importance only in patients with chronic renal failure who might have repeated peritoneal dialysis over a long period. We found no difficulty in obtaining adequate fluid drainage in all except one patient and overhydration did not occur. In fact, as we have indicated, this method is ideal for removing excess fluid from overhydrated patients. However, the other well-known risk of peritoneal dialysis—namely, infection—still remains. We experienced peritoneal infection in 4 out of 48 dialyses, but in each case the infection appeared to respond rapidly to antibiotic therapy, and Burns *et al.* (1962) suggest that peritoneal dialysis might be a good treatment for peritonitis.

The cost of peritoneal dialysis depends mainly on the cost of the dialysis fluid. The catheter and special giving-set cost approximately 15s. each, whereas 75 l. of commercial peritoneal irrigation solution (Allen & Hanburys) cost approximately £28. If the solution is made in the hospital pharmacy the procedure will be correspondingly cheaper.

### Summary

Experience of 48 peritoneal dialyses in 30 patients with renal failure and one patient with refractory oedema is reported.

Dialysis was technically satisfactory on 47 occasions, and in all cases there was an improvement in the biochemical and clinical state of the patient.

No deaths were attributable to dialysis, but peritoneal infection occurred on four occasions.

It is concluded that peritoneal dialysis is an effective, simple, and reliable method for the treatment of renal failure.

We wish to thank Dr. R. D. Green, of Paddington General Hospital, and the medical staff of St. Mary's Hospital for allowing us to treat their patients; the chemical pathology departments of both hospitals for technical assistance; and the pharmaceutical and nursing staff of both hospitals, without whom this treatment would not have been possible.

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