

## Children Referred for Acute Dialysis

S. R. MEADOW,\* J. S. CAMERON, C. S. OGG, and H. M. SAXTON

From Guy's Hospital, London

**Meadow, S. R., Cameron, J. S., Ogg, C. S., and Saxton, H. M. (1971).** *Archives of Disease in Childhood*, 46, 221. **Children referred for acute dialysis.** Acute unexplained renal failure of infancy and childhood is a rewarding condition to treat. Of 27 children referred for acute dialysis, only 3 died. Half were managed without dialysis, and of those dialysed, 6 recovered full or adequate renal function while 6 had end-stage renal disease requiring transplantation or regular dialysis.

Complications of the nephrotic syndrome causing hypovolaemic uraemia were a major cause of referral. Septicaemia was commonly associated with renal failure regardless of its aetiology.

The child in acute renal failure requires considerable resources in addition to dialysis. Therefore, peritoneal dialysis should not be started in paediatric units which do not have the skills and services required for full investigation and care.

Children with unexplained acute renal failure present one of the most challenging problems in paediatrics, and their investigation and management involve many specialists besides the paediatrician. The problem is sufficiently uncommon for there to be a shortage of units with experience of acute renal failure in childhood.

Reports describing dialysis of children make depressing reading, for while they stress the technical simplicity of dialysis, the end result is that 40 to 80% of the children die (Lee and Sharpstone, 1966; Lloyd-Still and Atwell, 1966). These series give a misleading view of the the problem of acute renal failure mainly because they do not report the many children who were restored to health without dialysis.

We present details of those children who were referred from other hospitals to us for dialysis in the past 18 months. The origins of the renal failure should suggest ways of preventing it in other children, and the management and outcome should be a guide to paediatricians who encounter children with acute renal failure.

### Cases

All children under the age of 14 referred during the past 18 months as an emergency for dialysis are included. Children with chronic renal failure referred for elective dialysis or transplantation are not included, nor are

those children already in Guy's Hospital who developed acute renal failure (for instance after burns or major surgery). Essentially the 27 children were referred from elsewhere acutely ill with recent unexpected, and usually unexplained, renal failure.

### Clinical Details

Table I summarizes the information.

**Age.** Ages ranged from 7 days to 13½ years. 10 of the 27 children were under the age of 3 years.

**Reason for referral.** The reason for the referring paediatrician considering that dialysis might be needed can be seen from the 'main clinical feature' and the urea level. An additional motivating factor was that many of the children had prolonged oliguria.

**Cause of renal failure.** 11 children had intrinsic renal failure, 3 had postrenal (obstructive) failure, and 9 had primarily 'prerenal' uraemia. 4 other children had renal failure from a mixture of factors. 9 children had septicaemia on admission, in 5 of whom it was associated with obstruction of the urinary outflow tract. 6 had the nephrotic syndrome with hypovolaemia and prerenal uraemia which was severe enough in Case 9 to progress to intrinsic renal failure.

**Dialysis.** 11 patients had peritoneal dialysis, and 7 haemodialysis; 5 patients were treated by both techniques, so that 13 patients out of 27 referred for dialysis actually received it. 2 patients in whom peritoneal dialysis was begun required haemodialysis because peritoneal dialysis failed; one of these (Case 9) had had a laparotomy for peritonitis two days previously, and in the other (Case 15) peritoneal dialysis fluid leaked into

\*Present address: Department of Paediatrics and Child Health, Leeds.

TABLE I  
*Clinical and Pathological Details of 27 Children Referred for Acute Dialysis*

Case No.	Age	Blood Urea (mg/100 ml)	Main Clinical Feature	Pathology	Special Investigations	Dialysis	Outcome
1	7 dy	168	Anuria	Multicystic kidneys, ureteric obstruction	IVU tomogram; renal cyst pucture	0	Died aged 10 days
2	10 dy	175	Acidosis	Hypoplastic kidneys	IVU tomogram; <sup>131</sup> I scan	0	Well, on diet
3	3 wk	150	Septicaemia, oliguria	Ureteroceles; bladder neck obstruction	IVU tomogram; cystoscopy; cystogram	0	Well, after surgery
4	1 mth	176	Septicaemia	Megacystis—megaureter	IVU tomogram, cystogram; cystoscopy	Peritoneal	Well, after surgery
5	1 mth	500	Septicaemia	Acute pyelonephritis	IVU tomogram; cystogram	Peritoneal	Well
6	6 mth	350	Oliguria; BP 190/140	Atrophic L. kidney; dystrophic R. kidney pelviureteric obstruction	IVU tomogram; retrograde pyelogram, renal biopsy	Peritoneal	Well, continued medical management
7	10 mth	160	Septicaemia	Hydronephrosis	IVU tomogram; cystogram; cystoscopy	0	Well, after surgery
8	1½ yr	150	Septicaemia; cardiac failure	Congenital heart lesions; staphylococcal endocarditis	IVU tomogram	0	Died of cardiac failure
9	2½ yr	220	Nephrotic; laparotomy; hypovolaemia	'Minimal change' lesion; pneumococcal peritonitis	IVU tomogram; renal biopsy	Peritoneal and haemodialysis	Well
10	2½ yr	144	Septicaemia	Hydronephrosis; calculus	IVU tomogram; cystogram; cystoscopy	0	Well, after surgery
11	3½ yr	310	Nephrotic hypovolaemia; peritonitis	Focal glomerular hyalinization (segmental hyalinosis)		Peritoneal (before transfer)	Died in ambulance before admission
12	4 yr	110	Septicaemia	Hydronephrosis; uretric reflux	IVU tomogram; cystogram; cystoscopy	0	Well, after surgery
13	4½ yr	114	Acute nephritis; BP 180/140	Systemic lupus; renal artery thrombosis	IVU tomogram; arteriogram; renal biopsy	0	Continued medical management
14	5 yr	190	Nephrotic hypovolaemia; septicaemia	'Minimal change' lesion	IVU tomogram	0	Recovered from renal failure
15	5½ yr	440	Anuria	Pelviureteric obstruction of single kidney; ureteric reflux	Renogram; IVU tomogram; percutaneous nephrostomy; cystogram; cystoscopy	Peritoneal and haemodialysis	Well, after surgery
16	6 yr	280	Acute nephritis; BP 195/160	End-stage kidney; ureteric reflux	IVU tomogram; cystogram	Peritoneal and haemodialysis	Home haemodialysis
17	6½ yr	240	Oliguria; BP 250/110	Bilateral pelviureteric obstruction; hydronephrosis	IVU tomogram; renogram; percutaneous nephrostomy	Peritoneal	Well, after surgery
18	6½ yr	46	BP 300/230	Fibromuscular hyperplasia of segmental arteries in both kidneys	IVU tomogram; arteriogram	0	Continued medical management
19	6½ yr	130	Nephrotic; oliguria	Membranoproliferative glomerulonephritis	IVU tomogram; renal biopsy	0	Well
20	7½ yr	256	Acute nephritis	Membranoproliferative glomerulonephritis	IVU tomogram; renal biopsy	0	Continued medical management
21	8 yr	152	Nephrotic hypovolaemia	'Minimal change' lesion	IVU tomogram	0	Well
22	8 yr	120	Nephrotic hypovolaemia		IVU tomogram	0	Well

TABLE 1—continued

Case No.	Age	Blood Urea (mg/100 ml)	Main Clinical Feature	Pathology	Special Investigations	Dialysis	Outcome
23	8½ yr	194	BP 180/140	Chronic diffuse glomerulonephritis	IVU tomogram; renal biopsy	Haemodialysis	Home haemodialysis
24	9½ yr	468	Acute nephritis BP 140/100	Proliferative glomerulonephritis	IVU tomogram; renal biopsy	Peritoneal and haemodialysis	Transplant
25	10 yr		BP 200/160	'Pylonephritis', ureteric reflux	IVU tomogram renal biopsy; cystogram	Haemodialysis	Transplant
26	10 yr	460	Acute nephritis	Microangiopathic glomerulonephritis	IVU tomogram; renal biopsy	Peritoneal and haemodialysis	Home haemodialysis
27	13½ yr	420	Acute nephritis	Proliferative glomerulonephritis	IVU tomogram renal biopsy	Peritoneal	Continued medical management

the abdominal wall. In 2 patients (Cases 23 and 25) end-stage renal failure was found and haemodialysis begun as a prelude to regular haemodialysis treatment.

**Outcome.** 13 children recovered satisfactory renal function without dialysis. Of the 13 who required dialysis, 5 recovered fully, 1 partially, and 6 had end-stage renal disease and subsequently required either regular haemodialysis treatment or renal transplantation. One died (dialysis had been begun elsewhere but he died in the ambulance on the way to us).

Case 1 a neonate with gross congenital renal abnormalities died of renal failure, no dialysis being attempted.

Care

The children were admitted to the paediatric ward. They were under the joint care of the paediatricians and the renal physicians who acted through the paediatric junior staff.

Referring hospitals sent their notes and x-rays, which were helpful, but though there were usually careful records of the blood chemistry, it was unusual to find any record of the urine chemistry or osmolality. Therefore, one of the first investigations was measurement of

urine electrolytes, urea, and osmolality. These gave a good differentiation between intrinsic renal failure and prerenal renal failure.

The other urgent investigation with which referring hospitals had often had difficulty, sometimes because of lack of facilities, was an intravenous urogram (IVU) with nephrotomography. Only a few children arrived with x-rays on which the kidneys could be satisfactorily visualized; for the rest, provided that the child in renal failure was given a large enough dose of contrast medium and adequate tomograms taken, it was possible to obtain good pictures of the kidneys except in Case 2. From the appearance it was often possible to distinguish the small scarred kidneys of children whose acute renal failure was superimposed on chronic renal failure, from those with enlarged or obstructed kidneys who were likely to have recoverable conditions.

Many departments of the hospital became involved in the care of these very ill children (Fig.). The list below merely shows the frequency with which a few of the specialist services or investigations were required in the care of the 27 children. *Paediatric and renal for 27 children including dialysis (13), renal biopsy (11); radiology. IVU with nephrotomogram (27), micturating*

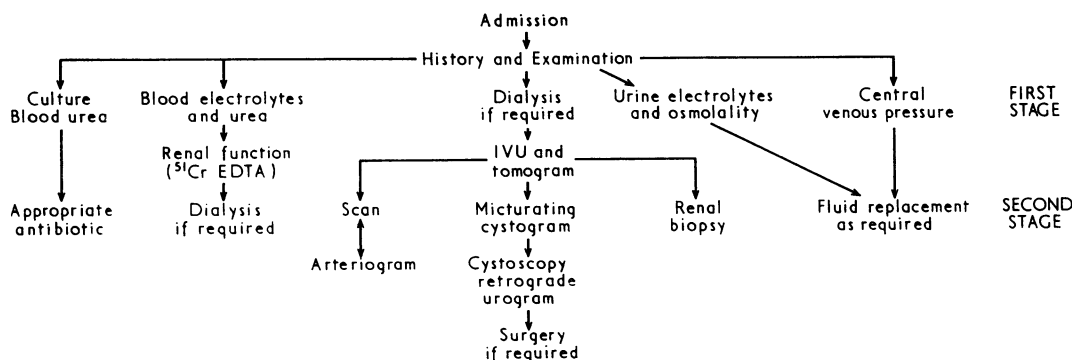


FIG.—Outline plan of management for children with acute uraemia. (Only the most important aspects are mentioned; see text for details.)

cystogram (10), arteriogram (2), percutaneous needle nephrostomy (2), diagnostic cyst puncture (1); *genito-urinary*, cystoscopy (9) major surgery (7); *radioisotopes*  $^{51}\text{Cr}$  EDTA clearance (21), renogram (2),  $^{131}\text{I}$  hippuran scan (1); *child psychiatry* (3); *clinical chemistry* (27); *microbiology* (27). The haematology department supervised control of anticoagulant therapy for 2 children, and provided the necessary data for safe and effective cytotoxic drug therapy for 6 children. Dietitians supervised the special feeds required by most of the children.

### Discussion

Though acute renal failure in children is uncommon, our experience is that it is a most rewarding condition to treat; only 3 of 27 patients died. Most of the children go home well; a minority are found to have end-stage renal disease requiring long-term dialysis or transplantation. In our series it was the older children who tended to have irrecoverable lesions: however, a hospital with a larger neonatal surgical service might receive more infants with grossly disorganized kidneys and urinary tract than we did.

The children came from a large catchment area, for which we were not the only children's renal unit; therefore no conclusions can be drawn about the epidemiology of acute renal failure, nor the number of units that are required to deal with it.

Similarly the origins of uraemia as listed under 'Pathology' in Table I are not a comprehensive list of the possible causes. The haemolytic-uraemic syndrome is a notable absentee, for though we have treated several cases in the past, no child with the syndrome was referred during the review period. Nor was any child referred for dialysis because of drug overdose: this is not so surprising since serious childhood poisoning is very rare—of the last 300 admissions to the Leeds Children's Poisons Unit, none has required dialysis (R. W. S. Smithells, 1970, personal communication).

The two aspects which dominate discussion are prevention of acute renal failure and its management, with particular reference to the role of the general paediatrician.

**Prevention.** By strict criteria, several of the children in Table I did not have acute renal failure. However, their blood urea was rising or their urine output falling at such a rate that the referring paediatrician feared that dialysis might be required. Quite correctly they usually referred the child before the procedure was needed. A third of the children had renal failure from prerenal causes, glomerular perfusion being insufficient to permit adequate function.

Prerenal uraemia is particularly common in the

nephrotic syndrome of childhood, and we are impressed by the management problems that the acute nephrotic syndrome may present. Despite increasing knowledge about its pathology and treatment, and despite the overall favourable prognosis for young children with the nephrotic syndrome, many still develop lethal complications.

Five children had the nephrotic syndrome associated with minimal histological abnormality. During severe relapse they had developed hypovolaemia and renal failure. Children with the nephrotic syndrome have a 20 to 30% reduction in plasma volume and if there is a sudden extracellular fluid loss for instance after paracentesis or laparotomy (Case 9), they cannot maintain a plasma volume that is sufficient for adequate renal plasma flow (Squire, 1955). Diuretics may reduce the plasma volume similarly (Garnett and Webber, 1967).

Septicaemia from either Gram-positive or Gram-negative organisms further impairs renal perfusion. Other causes of severe oliguria in the nephrotic syndrome, such as progressive glomerular disease, renal vein thrombosis, or blockage of the tubules with protein casts are relatively rare.

The importance of recognizing hypovolaemic renal failure has been stressed by Chamberlain, Pringle, and Wrong (1966). Its recognition is all the more important in childhood nephrotic syndrome because most children have 'minimal changes' histologically and a prospect of complete recovery however ill they may be (Conolly, Wrong, and Jones, 1968). Once recognized the hypovolaemia can be corrected with intravenous albumin or dextran. In moribund children we have used central venous pressure measurements as an index of adequate replacement. At the same time diuretics are given, together with the usual steroid therapy for nephrotic syndrome. In all but one child these measures led to a diuresis. The exception, Case 9, developed renal failure after a laparotomy for pneumococcal peritonitis and required three weeks' haemodialysis before making a complete recovery.

Prerenal uraemia may occur in cardiac failure, septicaemia, gastroenteritis, and after burns or other trauma, and may progress to intrinsic renal failure. Therefore its recognition is important so that prompt treatment can be given. The value of urine electrolyte and urea estimation cannot be overstressed, particularly as it had rarely been done before referral. A useful working differentiation between prerenal and established renal failure can be made (Table II).

Measuring the central venous pressure is an additional aid. It will record a low cardiac filling

TABLE II

Typical urine composition in 'prerenal' and 'renal' uraemia

	Prerenal	Renal
Urine osmolality (mOsm/l)	400	250-320
Urine sodium (mEq/l)	10	40
Urine urea (g/100 ml)	1.5	0.5

pressure in hypovolaemia, and allows accurate and safe replacement therapy. A further differentiating feature between hypovolaemia and established renal failure is the inability of a dose of diuretic such as frusemide or an infusion of mannitol to produce a diuresis when there is established renal failure.

Speedy analysis of urine from a child who becomes either oliguric or uraemic followed by energetic implementation of the appropriate treatment undoubtedly will reduce the number of children requiring dialysis.

Detection and treatment of children with urinary tract infection may prevent some cases of renal failure in later life. It will certainly prevent a few neonates from developing septicaemia and renal failure. The work of O'Doherty (1968) and Littlewood, Kite, and Kite (1969) suggests that up to 2% of boys have neonatal urine infections—usually not associated with detectable congenital abnormalities of the urinary tract. Case 5 was such an infant who nearly died from the resulting renal failure. After peritoneal dialysis he made a complete recovery and is now a healthy toddler.

Other major advances in the prevention of acute renal failure in childhood must await more knowledge about acute nephritis and especially the development of effective therapy.

**Management.** The chief aim of the paediatrician investigating a child with acute renal failure is to find the cause and to plan treatment. The Fig. divides the procedures into first and second stages of management, but the precise order of investigation is dictated by the initial findings.

Blood urea and electrolyte levels are always measured, as are Hb and white cell values. If there is any likelihood of haemolytic-uraemic syndrome, it is worth asking the haematologist to look specifically for signs of red cell fragmentation and consumption of coagulation factors (low fibrinogen titre and low platelet count). Blood culture should be done in all cases. Of the 27 children referred to us, 10 had septicaemia; in those with urinary tract infection or obstruction it was expected, but in others it was not. The role of septicaemia in

exacerbating renal failure has already been mentioned. When septicaemia is suspected or diagnosed there is sometimes understandable confusion about what dose of antibiotic to give to children who are passing little or no urine. The ideal is to adjust the dose according to serum antibiotic levels, but few paediatricians have such a service. Kunin (1967) and Linton and Lawson (1970) give useful advice about antibiotic doses in renal failure.

Urine osmolality is of great value, but many hospitals do not have an osmometer. For these units urine electrolyte and urea estimations are a good substitute; they will reveal whether or not the kidneys are likely to work satisfactorily or not in the short-term future. Urine specific gravity is useful particularly as no laboratory is needed for the estimation. All paediatricians are only too fully aware of the difficulties of measuring the specific gravity of small volumes of urine with floating urinometers. The cheap and reliable refractometer\* now available, which requires only one or two drops of urine, is a great asset in any children's unit.

Urine culture and routine examination is essential. It is a sad triumph for those who have spoken strongly about the absolute need for urine colony counts in diagnosing urine infection, that we have received several children for dialysis whose only record of urine examination was a cryptic 'urine sent for colony count'. The value of colony counts does not reduce the great value of routine urinalysis—dipstix tests and microscopy; and colony counts will never compete with the instant availability of routine urinalysis.

If no urine has been passed catheterization is indicated, firstly to obtain some for testing, and secondly to exclude lower urinary tract obstruction. Once the catheter is in the bladder it may be useful to inject 30-50 ml contrast, for even though full micturition cystography may not be possible at the time, a film of the bladder may be helpful for example in revealing ureteroceles.

Intravenous urography has proved of considerable assistance in the evaluation of acute renal failure (Saxton, 1968). An adequate dose of contrast is required, and we have used 3 ml/kg of sodium iothalamate (Conray 420) or 4 ml/kg of sodium diatrizoate (Hypaque 45). The injection is given over a period of three minutes to avoid an excessive rise in plasma osmolality (Standen *et al.*, 1965). Tomography is undertaken 3-5 minutes after the injection has been completed; sufficient 'cuts' must be obtained to show the kidneys completely and the radiographs

\*'Uricon' Refractometer. ChemLab Instruments Ltd., 1b Seven Kings Road, Ilford, Essex. Price £12.50.

must be of the highest quality. In most cases excretion is seen on these films and an assessment can be made of renal size and symmetry, parenchymal thickness, and pelvicalyceal configuration. The 'end-stage' contracted kidney shows crowded calyces, while with most types of acute failure of recent onset the kidneys are normal or increased in size, their pelvicalyceal systems often outlining faintly, if at all. Obstruction is recognized in the majority of subjects by the opacification of distended calyces, but in very severe cases the early films may show an 'inverse pyelogram', the non-opacified calyces being less dense than the surrounding parenchyma. In any case where the pelves and ureters are not seen on the early film, further tomograms are obtained after 20 minutes and an hour, and continued examination up to 24 hours may be required before the whole of the obstructed system is outlined. Discussion of the detailed findings is beyond the scope of this paper but a recent review by Fry and Cattell (1970) provides a good introduction to this subject.

Accurate fluid input and output charts are kept by most units, and if combined with twice daily weighing form the basis for planning the fluid regimen.

Much has been written about the techniques of peritoneal dialysis, and its efficacy and safety in children are well established. To stress its convenience or simplicity is rather misleading, for children requiring dialysis are likely to be desperately ill, extremely difficult to manage, and need to be cared for by highly experienced staff with access to a wide variety of different services. There is little point in embarking on peritoneal dialysis unless such resources are available. Table I gives some idea of the requirements: a first-rate pathology service, radiologists skilled at renal investigation, genitourinary surgeons experienced with children, paediatricians and nephrologists expert at renal biopsy and other procedures, and above all junior staff and nurses skilled in the intensive care of children. Moreover, once the acute stage is over many of the children require complicated medical care, for instance cytotoxic and anticoagulant therapy. It is clear that dialysis should not be begun in a paediatric unit without the full range of supporting services unless there is no alternative. Most paediatricians do not undertake dialysis themselves and transfer their patients in anticipation of dialysis. Some are needlessly transferred (one 'anuric' child passed urine 4 times during a 40-mile ambulance journey to us), but all units prefer to have a child transferred early rather than with a blood urea of 440 mg/100 ml at 2 a.m. on a

Saturday. A further reason for restricting childhood peritoneal dialysis to special units is that the number of children requiring dialysis is so small that very few people will have much experience of it. Only with experience can the difficulties and complications be kept to a minimum. The organization of regional services for children with renal failure has been discussed elsewhere (Meadow, Cameron, and Ogg, 1970).

All units that receive many children from other hospitals must be thankful that only a minority of referring hospitals refuse to send the child's notes, x-rays, and charts. While we are all reluctant to part with our carefully kept records, we also know that no summary is an adequate substitute for the complete set of notes and charts. And the one moment in his life when a child needs them with him is when he is transferred, desperately ill, to another hospital where he has never been seen before. If urine is passed shortly before transfer it is best to send it with the child so that the accepting hospital can start its own investigations as soon as possible.

Few of us enjoy transferring a patient to the care of another hospital: there is so often a small feeling of sadness, inadequacy, or defeat. In addition paediatricians are mindful of the fact that they may have to transfer infants and children to specialist 'system' units which have neither children's trained nurses nor junior staff, no facilities for children, and sometimes no attending paediatrician. But in Britain in the past decade there have developed besides the adult renal units—and sometimes linked with them—paediatric units with the facilities and the skills that are required to deal well with the child referred for acute dialysis.

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Correspondence to Dr. Roy Meadow, Department of Paediatrics and Child Health, University of Leeds, Leeds LS1 3ET.

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*The following articles will appear in future issues of this journal:*

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