The Turkish Journal of Pediatrics
1973 / Vol. 15 / No. 1 / pp. 14 – 29

The Management of Acute and Chronic Renal Failure in Children

J. S. Cameron*

The symptomatology of uremia, whether acute or chronic, has much in common. However, the management of the child with previously normal kidneys and (hopefully) return of renal function must necessarily differ from that of the child with chronic renal disease, accompanied by irreversible loss of part or all of functioning renal tissue.

1 ACUTE RENAL FAILURE IN CHILDHOOD

Acute renal failure in children is often secondary to many other events, as Niilo Hallman has indicated, and it should not be forgotten that the managment of the renal failure may form only a small part of the total problem the child presents. This is particularly true of acute renal failure associated with trauma, septicemia, or both.

Many children (30% in our own series[1]) who present with oliguria and a rising blood urea, are not in a state of established acute renal shutdown, but in a potentially reversible *functional renal failure* (as Hallman has called it) or "*prerenal uremia*". Perhaps a better term would be *incipient renal failure,* to emphasize that, if circumstances of prerenal uremia persist, they may in fact lead to established acute renal failure. The central renal event in these patients with acute functional renal failure is *renal hypoperfusion.* The identification of children in this group is of great importance to the general pediatrician since it is much more common than fully established acute renal failure in children, and because prompt diagnosis and treatment will avoid this much more serious state.

---

* Senior Lecturer in Medicine and Physician in Renal Diseases, Guy's Hospital, London, SEL 9RT, UK

A) The identification of incipient, reversible acute renal failure:

Many children in this state are obviously in *shock*[2] that is, at a pathological level there is failure of microcirculation in many tissues, often with disseminated intravascular coagulation[2]; and at a clincal level a cold periphery, rapid pulse, and often cyanosis and a low blood pressure. If volume depletion is a feature (for example in acute gastroenteritis) this may be obvious; but the problems of assessing hydration in infants are well known, and a lack of effective circulating volume may not show itself as a general extracellular deficit.

The identification of a shocked state with acute renal failure of incipient renal failure depends upon the presence of several simply assessed measurements:

(i) the presence of oliguria

(ii) a failure to maintain homeostasis - a rising blood urea and inappropriate Na+ and water excretion

(iii) a low central venous pressure

(iv) a cold periphery with a large central-peripheral temperature gradient

(v) a characteristic urine composition (see below) and finally

(vi) hypotension *may* be present in advanced states of shock.

The importance of identifying shock in terms of a failure of *prefusion*, lack or maldistribution of *volume*, rather than as a low blood pressure, cannot be emphasized too strongly.

The central venous pressure, measured by a catheter passed from the basilic, jugular or subclavicular vein (perhaps the best) into the right atrium is central to the management of severe shock in infancy and childhood. We have also found formal measurement of the central and peripheral temperature gradient by thermistors of great use, since in the shocked, peripherally constricted patient there tends to be central hyperthermia (which in itself may present a problem) as well as peripheral cooling. Under these circumstances, axillary temperatures will grossly mislead. If a measure of core temperature is required, then it must be a rectal one.

It is the urine *composition*, however, that is of greatest use in distinguishing incipient from established acute renal failure (Table I). In any state of renal hypoperfusion in a normal kidney, from renal artery stenosis through shock to the nephrotic syndrome, a characteris-

TABLE I

COMPOSITION OF URINE IN INCIPIENT RENAL FAILURE AND ESTABLISHED ACUTE RENAL FAILURE

	Incipient renal failure	Established acute renal failure
Volume :	< 0.5 ml/kg/min	0.5–1.5 ml/kg/min
Na+ :	< 10 mM/l	20–40 mM/l
Urea :	1500–3500 mg/100 ml	300–600 mg/100 ml
Urea ratio U/P :	5–10: 1	3: 1 or less
Osmolar ratio U/P :	1.3–3: 1	1.1: 1 or less
Urine SG :	1015 or more	1010 on less

tic pattern of urine composition is seen. The urine is concentrated with respect to plasma, the urea concentration is high, and irrespective of intake the Na + concentration is very low. This contrasts with the urine of established renal failure, when the urine is iso-osmolar with, or less concentrated than plasma, shows a low urea concentration and usually has a Na + concentration of 30-40 mEq/l.

The urea and osmolarity measurements are of particular value if they can be compared with plasma levels or concentrations. The urine/plasma (U/P) ratio of urea will usually be 5:1 (infants) and often 10: 1 (children) or more in incipient renal failure;[3] and the U/P osmolarity ratio is over 1.1, and often 2-3/1. The *osmolarity* is particularly valuable. Small, relatively cheap portable osmometers are readily available, only 0.1 ml. of plasma or urine is needed, and the result is available immediately. U/P osmolarity ratios of 1.1-1.3 indicate that the patient is verging on established acute renal failure, but this state is still potentially reversible.

If an osometer is not available, the extremely simple, quick and cheap *refractometer*, which again requires only a drop of urine (or plasma), will decide whether the urine is concentrated or dilute. This measurement, although less accurately reflecting total solute, is still a useful guide.

B) The management of reversible incipient acute renal failure

The early management of reversible incipient acute renal failure is the same as that for established acute renal failure, and centers around three measures:

(1) the restoration of circulating volume and/or cardiac output

(2) the detection and treatment of infection, especially septicemia

(3) the maintenance or establishment of urine flow.

Let us consider these in order:

(1) *The restoration of circulating volume* can be carried out quickly and safely provided the measurements made initially to establish the state of the circulation are repeated at frequent intervals. There are central venous pressure, central-peripheral temperature gradient, blood pressure, urine volume and urine composition. Note should also be taken of pulse rate and arterial oxygen tension in severely ill infants or children. The agent to use for volume replacement is a matter of some controversy. There is little doubt to my mind that colloid is much preferable to (for example) saline alone in these circumstances, but whether whole blood, or plasma or dextrose should be given largely depends upon personal preference. Unless, of course, the patient is notably anemic, when whole blood will be preferable. After the initial phase, one may transfer to crystalloid solutions according to the urine output.

The detailed discussion of children in cardiogenic shock, or renal failure following cardiovascular surgery[5] is beyond the scopt of this article. One point, however, is worth making: children are often treated with digitalis on the assumption that their enlarged hearts, rapid respiration, rapid pulse and raised CVP indicates cardiac failure. More often in this situtation it represents simply inappropriate fluid or salt administration to an oliguric child incapable, either because of functional or established renal failure, of excreting the load.

(2) Septicemia is extremely common in both acute renal failure and in states of shock, either as a precipitating factor or as a complication - sometimes as both. 60 % of our series of patients[6] had proven or clinically suspected septicemias in established acute renal failure. In children, these organisms come with especial frequency from the *urinary tract*, above all where there is obstruction. A blood and urine culture are best taken immediately, with urine microscopy and a broad spectrum, preferably bactericidal antibiotic begun. In these very ill children the choice probably rests between an aminoglycoside such as gentamycin or kanamycin, cephalothin, or chloramphenical. Carbenicillin may also be used if sensitivites indicate. Reservations with regard to dosage should be borne in mind (see below). Meanwhile, a high dose IVP (about 2 ml contrast medium/kg) should be arranged as soon as circumstances permit, preferably with tomograms, to evaluate

the urinary tract: this gives invaluable information[1]. Occasionally, dialysis may be needed before this can be done (see below) and one should remember the circulatory load all contrast media impose, as well as the intense acidosis some media may induce, particularly sodium diatirazoate (Hypaque). For the reason iothalamate contrast media are preferable (e.g. Conray).

(3) *Maintenance of urine volume* centres round the volume replacement discussed above, and the slow injection of a powerful diuretic in large dosage. Frusemide 5-10 mg/kg. may be given over 10-20 minutes, usually into the line of the I.V. infussions. If a copious diuresis *is* obtained, then the fluid regime must be altered to allow for this.

To summarize the early stages of diagnositic management in a very ill oliguric patient:

(1) Take a clinical history, directed towards:
  (a) information on previous renal function
  (b) precipitating circumstances

(2) Do a physical examination: assess cardiovascular and renal systems with particular care, and attempt to assess hydration. Look for possible sources of infection.

(3) Pass a urinary catheter and measure urine output hourly for initial period.

(4) Send blood and urine for:
  (a) electrolytes and urea
  (b) culture

(5) Do urine and plasma osmolarity (or SG if not available.)

(6) Microscope urine for pus and organisms

(7) Pass CVP catheter into great veins of right atrium

(8) Take x-ray plain films of chest and abdomen

(9) Inject 5-10 mg/kg. frusemide intravenously over 10-30 minutes

(10) Start antibiotic cover with a single loading dose of a broad spectrum bactericidal antibiotic active against gram negative organism and staphylococci (e.g. gentamycin 1 mg/kg I.V.)

Once this hectic early phase is over, perhaps two or four hours have elapsed and the situation should become clearer. Hopefully, the child begins to pass urine, the periphery warms up, perfusion improves, the pulse rate and respiration rate falls, and the core temperature settles. At this point all the aspects of management need review, especially

antibiotic policy and further dosage, the fluid regime and the timing of discontinuing the intravenous infusion, plus treatment of the primary events (if known). The urinary catheter should be removed no later than at 24 hours. Further investigations will be dictated by what is found: for example the management of a severe meningitis or a burnt child will differ from investigation of a disordered urinary tract.

(c) Management of established acute renal failure

However, if the urine is dilute with a moderate sodium concentration, the CVP is normal or is rendered normal without increase in urine flow, and frusemide also fails to produce a diuresis, then *established acute renal failure* may be considered present. This type of patient is probably best managed in a center with experience of the condition, since these very sick children require prolonged and complex management which may include dialysis.

At this point, the child is on the *oliguric phase* of the condition, and the main immediate dangers (apart from the ever present one of infection) are:

(i) *fluid overload* with possible consequences of cardiac failure, hypertension and convulsions. If the overload is produced by intravenous solutions containing little sodium, then the plasma sodium may fall considerably.

(ii) *hyerkalemia* which may give rise to cardiac arrest, especially if also present is:

(iii) *acidosis*. This is worse in the presence of shock and hypoperfusion and further worsens the clinical state by dropping the cardiac output.

The shortest way out of these situations, should they be present already, is to *dialyse* the patient, but the oliguric phase of acute renal failure in infants is often very short, and this may not be necessary. Whether dialysis is used or not, it is best to aim for a fluid intake (orally or intravenously according to circumstances) which results in a constant or slowly falling weight; remembering the relatively high insensible loss in infants, especially if febrile. Twice daily weighing is the best guide to fluid balance in the short term.

Hematological complications may be a prominent feature, especially if a primary hemolytic-uremic syndrome is the cause of the renal failure. However, thrombocytopaneia, prolonged prothrombin, thrombin and cephalin-kaolin times, and depleted fibrinogen ,with bleeding may

also be seen as a complication of the disseminated intravacular coagulation in septicaemic patients or in accelerated hypertension. Red cell fragmentation may also be present. Heparinization of all these groups of patients is theoretically attractive, but its use in the hemolytic-uremic syndrome, whilse improving the peripheral blood picture, has never been shown to influence the relatively good prognosis of the renal lesions. Locally infused streptokinase or urokinase directly into the renal artery are also unproven techniques as yet, and carry some technical dangers. In patients with septicemia, treatment is best directed to the organism.

Hypertension may also be a problem in the hemolytic-uremic syndrome, even with good fluid control. In emergency, hydrallazine 2.5 or 5 mg I.V. repeated as necessary will nearly always control the blood pressure while Methyl Dopa or other hypotensive agent is started for longer term use.

Dialysis may be begun for a variety of reasons: clinical deterioration, convulsions, hyperkalemia, extreme catabolism, severe acidosis, cardiovascular overload, or more commonly a combination of several or all of these. It is better to go ahead than delay if doubt is felt whether dialysis is necessary.

The choice between *peritoneal dialysis* and *hemodialysis* is beyond the scope of this discussion, but in general the only *absolute* indication for hemodialysis is an abdomen which for reasons of sepsis, surgery or technical accident is not suitable for peritoneal dialysis. However, in a much wider range of circumstance hemodialysis may be preferable. Particularly in children over 2 years of age, severe sepsis, a prolonged oliguria (more than one week) hypercatabolism or a conscious patient who should be mobilized are all relative indications for hemodialysis.

There are technical problems of both peritoneal and hemodialysis in infants and young children of less than 15 kg in body weight. With *peritoneal dialysis*, no suitable small access cannula is commercially available, and the peritoneal dialysis sets and solutions are also designed for adults. Peritoneal dialysis machines are also geared to adult needs and require modification for the exchange volumes required in infancy. Hypothermia may be a problem if the heating of the fluid is not efficient and lactate in the dialysis fluid may not be metabolized by sick infants.

With hemodialysis, the problems relate principally to vascular access on the one hand and dialyser priming columes and potency on the other. Vascular access, even down to 3 kg infants, can usually be

obtained using proximal stem arteries such as the brachial and femoral and their corresponding veins, for external arterovenous teflonsilastic shunts. Veno-venous major vein cannulae have also been used in the newborn. Dialysers suitable for infants, with small priming volumes and low potency, are now available and the subject has been reviewed[7]. Above 15-20 kg adult flat-plate dialysers with shortened lines can be used. The danger of over-large priming volumes which may equal or exceed the infant's own blood volume is obvious. The more subtle danger of excess water removal, excess fluid shifts due to rapid fall in extracerebral Na + and urea, leading to cerebral edema and convulsions, are now well recognized. Hemodialysis should be as efficient and safe produre as in the adult, but to achieve this experience and appropriate apparatus are necessary. Hence the need for referral of cases to an experienced centre.

Finally, *nutrition*. During the phase of severe illness it is best to aim for a high calorie, high protein intake in an attemp to reverse catabolism and to dialyze intensively or frequently. Feeding may often need to be intravenous, using protein hydrolysates and lipid solutions, even if the gut is working. Some points of importance in the feeding of uremic infants[8] in both incipient and established renal failure, are brought out in the next section on chronic renal failure.

Hopefully, the child in acute renal failure will next enter the *diuretic phase* of his or her illness, when the dangers of overload are replaced by the dangers of electrolyte and fluid loss, with the continuing dangers of sepsis. Balance needs careful attention, and it is a sad fact that most deaths occur at this last stage with modern management of acute renal failure, sepsis being the main problem.

Children, however, do relatively well in surviving this severe illness. In our own series[6], only 27 % of children under 10 years of age with established acute renal failure died, compared with an overall mortality at all ages of 57 %, and a mortality of 80-90 % over 60 years of age. Usually, death is due to the severity of precipitating cause rather than complications. Sepsis is still a major factor in causing death. Hopefully, all children with acute reversible states will survive.

II CHRONIC RENAL FAILURE IN CHILDHOOD

The cardinal feature of chronic renal failure is an inability to regulate the urine volume and composition appropriately to alterations brought about by variations in intake or extra-renal output. This becomes progressively more severe with declining renal function, and urine composition is virtually fixed when the glomerular filtration rate app-

roaches the lowest levels compatible with continued life (about 2% normal function).

General Management

Table II summarizes the main points which the pediatrician should watch in a child with chronic renal failure. Unless hypertension is present, little need be done with chronic renal failure, until the blood urea reaches 100 mg/100 ml or more (plasma creatinine 3-4 mg/100 ml) at which point the glomerular filtration rate will be about 20 ml/min/m^2. The exception to this is *hypertension.* A diastolic BP repeatedly recorded at 15 mm/Hg or more in excess of that specified for age is an indication for treatment, whatever the renal function.

Statural growth is perhaps the best indicator of good general management, and maintenance of growth is all the more important, since relative height loss is very rarely retreivable following either regular dialysis treatment or successful transplantation; the child may thus be permanently dwarfed.

In Table II, particularly, attention must be paid to the careful control of *hypertension,* and the search for and treatment of *urinary tract infection* since both may lead to sharp, sometimes irreversible decline in renal function ("acute on chronic renal failure"). Balancing-salt and water intake to output, which is usually fixed, depends on measurement of urine volume and body weight. Some patients (particularly those with vescicoureteric reflux of urine and pyelonephritis) may lose very large amounts of Na+ in their urine and be vulnerable to

TABLE II

PRINCIPLES OF MANAGEMENT IN CHRONIC RENAL FALURE

Watch:	Growth:
Control:	Diet: protein caloric intake salt and water Ca (and Vit. D)
Detect and control:	Hypertension Urinary (and other) infection Salt depletion Bone disease (acidosis)

salt depletion, postural hypotension and a worsening of their uremia. This may of course occur in association with infection and is another cause of "acute on chronic" renal failure. Others, (particularly those with glomerular disease and hypertension), retain salt avidly and need salt restriction)rather than supplementation. The individual patient may change his salt requirements with time, and regular assessment is necessary. If supplementation *is* necessary, NaHCO3 is useful since it helps correct the acidosis to some extent, and may aid in the prevention of bone disease (see below).

When using any drug in a child with renal functional impairment, thought must be given as to what medication might be necessary in dosage because of the renal failure. Good reviews of the use of antibiotics[9] and drugs in general[10] are available for patients with renal impairment. Tetracyclines should be avoided altogether, with the exception of doxycycline.

Diet in chronic renal failure in childhood[11,12]

Dietary control is now of great importance in the management of both adults and children with chronic renal failure. The aim is to provide a controlled but adequate protein intake, which should mostly be of high biological value, together with a high caloric intake. In Table III, suggested minimum intake for adults and children are recommended. At low protein intakes in adults 55 k cal/kg/day are needed to maintain a positive nitrogen balance. Children probably require a greater caloric intake in health to utilize their dietary protein optimally. Although the factors responsible for growth failure in children with chronic renal failure are complex and ill-understood (Table IV) we believe at present that failure of caloric intake from the anorexia of uremia, and from psychological and cultural factors associated with adherence to a diet, is an important factor. To achieve the high calorie and predominant animal protein intake suggested in Table III may require the use of artificial protein free calorie supplements, such

TABLE III

SUGGESTED MİNİMUM PROTEİN AND CALORİE İNTAKE İN ADULTS AND CHİLDREN WİTH ADVANCED RENAL FAİLURE

	Child	Adult
k.Cal/kg/day	90	55
Protein g/kg/day	1.3	0.3
k.Cal/g protein	70	170

TABLE IV

FACTORS INVOLVED IN GROWTH FAILURE IN CHILDREN WITH CHRONIC RENAL FAILURE

. nitrogen intake	. infection
. caloric intake	. dehydrotian
. calcium/vitamin D intake	. acidosis
. renal osteodystrophy	. "uremic toxins"
	. ? anemia

as Contralyte*, Hycal** or Caloreen*** and close liason between parent, dietician and doctor. Results may be gratifying and one of our children[13] grew 1 cm. each month for eight months after control of infection and establishment of a diet of the type described.

An important point in both acute and chronic renal failure in infants is the differing composition of cow's and human milk[8] (Table V). Cow's milk contains, per unit volume of water, three times as much protein, three times as much sodium and nearly four times as much total solute as human breast milk.

TABLE V

COMPARISON OF SOLUTE LOAD IMPOSED BY COW'S MILK AND HUMAN BREAST MILK

	Infant		
	Cow	Breast	Adult
Water ml/kg/day	150	150	30
Protein g/kg/day	6	2	1
Na^+ mM/kg/day	4	1.5	1
Total solute mOs/kg/day	43	14	17
Phosphate mg/kg/day	140	20	15

(after Barratt[8])

In acute incipient renal failure, sodium conservation is obligatory, urine volume low and urinary concentration maximal. In established acute renal failure, sodium and urea excretion may be zero. In chronic

*

** Beecham Laboratories, London, U.K.

*** Scientific Hospital Supplies, Liverpool, U.K.

renal failure, the urine composition is relatively fixed with larger volumes of water being than normal for each unit of solute excreted, and there is a relative inability either to increase the concentration, or to decrease the volume of the urine. In all three states a tendancy towards catabolism is present so that exogenous protein is more readily converted to urea than to tissue protein.

In all these three situations, solute load greatly in excess of requirements can be expected with cow's milk feeds. The plasma sodium tends to rise, as does the blood urea, und in chronic renal failure this excess solute (Na+ and urea) will carry out excess water, increasing the urine output above the fluid intake. The resulting dehydration further worsens the uremia and hypernatremia, and reduces renal perfusion. A vicious circle is established as more feed is given. The use of a humanized milk formula (such as S.M.A.*) in all uremic infants is essential.

Bone disease

A major problem in children with severe chronic renal failure is renal osteodystrophy, and this is increasing in importance with better management of other complications. The bone disease associated with uremia shows a confusing mixture of rickets, hyperparathyroidism and osteosclerosis, and contributes to the dwarfing of chronic renal failure.

Recent advances have led to a much better understanding of the pathogenesis of this mixed picture, and also have practical consequences for management.

Calcium absorption has been known for some time to be defective in chronic renal failure[14] and this is of consequence in constructing diets. Too many uremic children (and adults) are fed too little calcium and an intake of at least 1 g/day should be maintained, by calcium supplements if necessary.

Raised levels of circulating parathyroid hormone (PTH) appear early in chronic renal failure, long before detectable changes in plasma phosphate or calcium. Even at glomerular filtration rates of 60-80% normal, the tubular reabsorption of filtered phosphate is lower than normal[15] and this may be mediated by the raised PTH. What the stimulus may be is still in dispute, but minute changes in plasma phosphate may be the first event. Only when there is obvious nitrogen and

* John Wyeth Co.

phosphate retention does the plasma calcium fall. It has been suggested that strict control of plasma phosphate, using alumininium hydroxide or other phosphate-binding agents, may reduce PTH levels, even before obvious uremia is present[15]. In patients with more advanced renal failure, however, this may lead to a *rise* in plasma calcium if there is gross parathyroid hyperplasia.

Occasionally patients with advanced renal failure, instead of showing the reduced plasma calcium characteristic of chronic renal failure, or the relatively "normal" levels indicating parathyroid overactivity, have a plasma calcium of 11-15 mg./100 ml. with severe progressive bone disease. These patients almost invariably have an adenoma in one of their four hyperplastic glands, and may be described as secondary autonomous (or "tertiary") hyperparathyroidism.

Turning to the ricket-like disease, this presents a clinical picture of a vitamin-D resistant rickets, although there are histological features at the epiphysis which make the description of "ricketlike" disease rather more appropriate than rickets itself. It has long been known that a period of time must elapse after the administration of vitamin-D before it exerts an effect. Now it is realized that the first step in the conversion to an active metabolite is conversion of cholecalciferol to 25-OH cholecalciferol in the liver. The active metabolite for bone and gut, however, is 1,25-diOH cholecalciferol,a amd the 1-hydroxylation can only be performed by kidney tissue[16]. The "vitamin-D resistance" of uremic osteodystrophy can thus be explained at least in part by a failure of vitamin-D activation from the relative absence of functioning renal tissue. The administration of 1-25 diOH vitamin D to uraemic subjects[17] will restore calcium absorption to normal, but also mobilizes calcium from bone, and the synthesis of new vitamin D analogues is eagerly awaited.

Even more interestingly, PTH has recently been shown[17] to inhibit 1-25, diOH vitamin D production and favour the synthesis of less active dihydroxy metabolites. The raised PTH levels found even in early uremia could, therefore, not only produce the classical changes of secondary hyperparathyroidism in the bone (including osteosclerosis), but also irritate or aggravate a relative vitamin D deficiency.

What are the practical therapeutic consequences of these observations? It is not usually necessary to rteat on radiological or biochemical evidence of renal osteodystrophy, unless the radiological changes are very severe and advancing rapidly. Only if *symptoms* are present is it usually necessary to treat - either bone pain, fracture or deformity.

Usually, vitamin D in carefully controlled doses will improve both symptoms, biochemical, radiological and histological signs. The dose must be determined for each individual, but is usually some 50,000 units of vitamin D/day or more. Careful watch must be kept on the plasma calcium, because if this rises, metastatic calcification may advance very rapidly.

Parathyroidectomy is rather rarely needed, and the only absolute indication is the existence of secondary autonomous ("tertiary") hyperparathyroidism with adenoma formation. Failure of control with vitamin D is another indication, especially if accompanied by worsening of metastatic calcification. We prefer *total* parathyroidectomy[19] followed by a small maintenance dose of vitamin D.

Control of the plasma phosphate level at normal, or near normal levels, can be achieved with aluminium hydroxide in large doses (e.g. 10-15 ml. of Aludrox four times a day with meals). This may delay or inhibit the appearance of secondary hyperparathyroidism, and reduce the changes of metastatic calcification.

Perhaps too little attention has ben paid to correcting the *acidosis* of children with chronic renal failure and bone disease, since it is known that restoration of the plasma bicarbonate to normal in patients with renal tubular acidosis may heal their ricket-like disease. However, dialysed patients rarely show bicarbonate (even predialysis) below 19-20 mM/l and this does not prevent the appearance or progression of osteodystrophy.

Transplantation and regular dialysis in children

I have reviewed this subject elsewhere in detail[20]. In general, and where possible, children should be taken on to dialysis or transplanted rather earlier than adults, to avoid the severe nutritional, statural and psychological consequences of terminal uremia. This occurs at about a plasma creatinine of 10-15 mg./100 ml. and a GFR of 5-7 ml./min/1.73 m^2. Peritoneal dialysis may be a very traumatic experience for the child.

The results of the Ninth Human Translant Registry[21] and the European Dialysis and Transplant Registry[22] suggest that children over the age of ten do better than any group of adults when survival is taken as the criterion of success (Table VI). Survival, however, is only the beginning of the problem. Growth and sexual maturation following transplantation, and on long term dialysis, have been very little studied. The impact on the family and the quality of life achieved

TABLE VI

RESULTS OF REGULAR DIALYSIS AND TRANSPLANTATION IN CHILDREN AND ADOLESCENTS 5–20 YEARS OF AGE

		At two years		
		% alive	% functioning graft	(Nos)
Transplantation* (5–20 years)	Donor: Cadaver	45	35	(356)
	Parent	69	60	(466)
	Sibling	83	77	(69)
Dialysis** 5–15 years)	– –	88	–	(296)

* 9th human renal transplant registry ([21])

** 1972 Edta Report ([22])

must be carefully evaluated before we can decide what place, if any, dialysis and transplantation should have in pediatrics. There is virtually no information on transplantation or dialysis in children under age of five years, and many would feel this area should remain experimental for the moment.

REFERENCES

1. Meadow, S. R., Cameron, J. S., Ogg, C. S., et al.: Children referred for acute dialysis, Arch. Dis. Child. 46: 221, 1971.
2. Hardaway, R.: The Clinical Management of Shock, Springfield, Ill., Charles C. Thomas, 1966.
3. Luke, R. G., and Kennedy, A. C.: Prevention and early management of acute renal failure, Postgrad. Med. J. 43: 280, 1967.
4. Eliahou, H. E., and Bata, A.: The diagnosis of acute renal failure. Nephron 2: 287, 1965.
5. Ogg, C. S., and Cameron, J. S.: Cardiovascular surgery and the kidney, Guy Hosp. Rep. 118: 85, 1969.
6. Stott, R. B., Cameron, J. S., Ogg, C. S., et al.: Why the persistently high mortality in acute renal failure, Lancet, 2: 75, 1972.
7. Kjellstrand, C. M., Shideman, J. R., Santiago, R. A., et al.: Technical advances in haemodialysis of vary small pediatric patients, Proc. Dialysis and Transplant Forum 1: 124, 1971.
8. Barratt, T. M.: Renal failure in the first year of life, Br. Med. Bull. 27: 115, 9971.
9. Liton, Al., and Lawson, D. H.: Editorial: Antibiotic therapy in renal failure. Proceedings of the European Dialysis and Transplant Association 7: 371, 1970.

10. Reidenberg, M. M.: Renal Function and Drug action, Philedelphia, W. B. Saunders, 1971.
11. Holliday, M. A.: Calorie deficiency in children with uremia: effect upon growth, Pediatrics 50: 590, 1972.
12. Chantler, C., and Holliday, M. A.: Growth and calorie intake in renal feilure Clinical Nephrology (in press), 1973.
13. Chantler, C.: Conservative management of chronic renal insufficiency in infants and children, Proc. R. Soc. Med. 64: 45, 1971.
14. Ogg, C. S.: Calcium metabolism in chronic renal failure, Brit. J. Urol. Suppl: 97, 1969.
15. Slatopolsky, E., Caglar, S., Gradowski, L., et al.: On the prevention of secondary hyperparathyroidism in experimental chronic renal disease using "proportional reduction" of dietary phosphorous intake, Kidney International 2: 147, 1972.
16. Fraser, D. R., and Kodicek, E. Unique biosynthesis by kidney of a biologically active vitamin D metabolite, Nature 228: 764, 1970.
17. Brickman, A. S., Coburn, J. W., and Norman, A. W. Use of 1.25 dihydroxy cholecalciferol in uremic man, N. Engl. J. Med. 287: 891, 1972.
18. Galante, L., Colston, K., MacAuley, S., et al.: Effect of parathyroid extract on vitamin-D metabolism, Lancet 1: 985, 1972.
19. Ogg, C. S. Total paratpyroidectomy in treatment of secondary (renal) hyperparathyroidism, Brit. Med. J. 4: 331, 1967.
20. Cameron, J. S. Dialysis and transplantation in children, Nephron (in press), 1973.
21. Barnes, B. A. et al. The Ninth Report of the Human Transplant Registry, J. A. M. A. 220: 253, 1972.
22. Scharer, K., Brunner, F. P., Gurland, H. J., et al.: Combined report on regular dialysis and transplantation of children in Europe, 1972. Proceedings of the European Dialysis and Transplant Association 9: 1972. Gurland, H. J. Unpublished observations.