Nutrition in the Newborn with Renal Disease

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Core Messages

- The neonatal period through the first year of life is a critical growth period during which nutritional intake is the prime growth stimulant.
- During infancy, chronic kidney disease is mainly due to renal structural abnormalities. These infants demonstrate significant renal tubular defects including sodium and bicarbonate losses as well as the inability to concentrate urine. Treatment in these infants must be adjusted to repair their excessive electrolyte and water losses.
- Neonatal acute kidney injury is often present as part of a more general picture of neonatal sepsis, congenital heart disease or hypoxic ischemic encephalopathy. Weighing the nutritional requirements for growth against the need to restrict fluid and electrolyte intake requires the collaborative efforts of multiple subspecialists.

Case Vignette

At 28 weeks gestation, a fetal ultrasound reveals that there is a male singleton fetus present but that there is bilateral hydronephrosis and an enlarge bladder. The ultrasound report notes that there is sufficient amniotic fluid present, and lung development is reported to be adequate. The parents ask to speak to you regarding antenatal counseling. You note that the presence of adequate amniotic fluid is a good sign but believe that it is highly likely that their son will be born with posterior urethral valves and will require surgical relief of his urinary obstruction shortly after birth. You stress the importance of delivery at a neonatal center with both pediatric urologic and nephrologic care providers and counsel the parents that their son will likely have some degree of chronic kidney disease but that it is too early to determine the severity of the problem. The infant is born at a regional

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center and is enrolled in a comprehensive chronic kidney disease program. The child goes on to require three hospitalizations within the first 18 months of life for dehydration secondary to infectious diarrhea/emesis but is thereafter stable. At 7 years of age, chronic kidney disease has progressed to **Stage 4** with an estimated creatinine clearance of 22 mL/min/1.73 m^2, and he is referred for renal transplantation. His care to date has required multiple medications and the use of nutritional supplements, but at the time of transplantation, his height and weight are both within −1 standard deviation score of the mean, his head circumference is at the mean for age, and he is developmentally normal.

6.1 Introduction

The role of nutrition is to provide the essential elements necessary for maintenance and growth. This is accomplished through a combination of metabolic adaptations as well as the kidney's exceptional ability to maintain the body's internal milieu. Children born with abnormal renal function are at a significant disadvantage at either conserving or excreting a variety of nutrients and as a result, often suffer nutritional debility during their earliest, most vital growth period. This chapter will attempt to outline both the challenges faced and some of the solutions currently available to achieve optimal growth during the first months of life.

Karlberg [1] proposed a hypothesis that divides growth into three distinct but overlapping phases. The first begins in utero and continues until sometime during the first year. The primary driver in this phase is nutritional not hormonal. The second phase begins later in the first year when nutrition remains important, but the primary stimulant is now growth hormone. The third phase begins at puberty when sex hormone secretion provides an additional stimulant to ongoing growth hormone secretion.

Linear growth rates are highest during the fetal period, averaging approximately 12 cm/month in the third trimester [2]. While growth rates decrease in the neonatal period, they are still higher than those at later periods of life. A normal infant gains about 25 cm in length during the first year of life, which is an increase of almost 50 % over birth length. By the end of the second year of life, a normal infant has completed approximately 30 % of its total growth and has reached 50 % of its final height [3]. At 1 year of age, the standard deviation (SD) for length is approximately 2.5 cm. This means that a growth rate as high as 80 % of normal (20 cm) would still leave them at a height of −2 SD below normal, putting it below the third percentile on the height chart.

During fetal life, it is the placenta that serves as the metabolic filter with the fetal kidney primarily being responsible for the production of amniotic fluid. While the kidney's role is limited, it is vital since infants born with oligohydramnios often die due to pulmonary hypoplasia. For infants born with structural renal disease and decreased renal function, growth failure often begins during the fetal period with newborn length measurements averaging approximately 1 standard deviation score (SDS) below the mean. Growth rates are diminished throughout the first several months of life, and infants with chronic kidney disease (CKD) may lose up to another SDS within the first few months of life [4]. This can account for up to 2/3 of the overall reduction in height SDS occurring over childhood. Data from the Growth Failure in Children with Renal Diseases Study and the North American Pediatric Renal Transplant Collaborative Studies group (NAPRTCS) indicate that the younger a child is at the onset of renal disease, the larger height deficits she/he has [5, 6]. Both height and weight are affected, which explains the classic literature description of infants with CKD as one of small, often cachectic individuals. The importance of this growth failure is magnified by the fact that both in renal disease and in studies in otherwise normal children, growth lost during the first year of life is not fully regained [7, 8].

These findings emphasize the need for early and aggressive intervention to maximize growth. By intervening early, growth velocity can be

improved with some degree of catch-up growth obtained. Current studies suggest, however, that for infants with CKD, average height SDS still hover below the mean as opposed to being normally distributed around zero [6, 9].

6.2 Etiology of CKD in Infancy

Approximately 20 % of children diagnosed with CKD each year are under 2 years of age (3/4 of whom are diagnosed in the first year of life). Of these, 70–75 % have a structural abnormality, such as obstructive uropathy, renal aplasia/hypoplasia/dysplasia, or reflux nephropathy, currently grouped together under the acronym CAKUT, which stands for congenital abnormalities of the kidneys or urinary tract. While present, autosomal recessive polycystic kidney disease forms only a small percentage of this group, and the incidence of inflammatory/immunologic disease is quite small [6]. The prevalence of CAKUT is associated with a high incidence of tubular dysfunction, due to both a combination of tubular adaption to hyperfiltration and to the tubular dysfunction present in these infants.

6.3 Conditions with Decreased GFR

6.3.1 Nutritional Intake and Growth

Given that multiple dietary alterations will be required for infants with CKD both as a function of their growth and their changing renal capacity, it is appropriate to consider as a base diet, a low-electrolyte, low-osmolar formula to which substances can be added as necessary. The most efficient infant formula known is human milk, and its use is to be encouraged for nutritional as well as psychological reasons. However, since the infant's volitional intake cannot be counted upon and a variety of additives are likely to be necessary, it is unlikely that the mother will be able to feed her infant at the breast. We have attempted to have mothers express their own milk and feed it to their infants, adding supplements as necessary. Unfortunately, this has rarely been a successful long-term solution, and the majority of mothers switch to one of the low-osmolar, human-milk substitutes. Table 6.1 notes some of the commercial formulas that may be utilized during the first year of life and thereafter, while Table 6.2 lists additional modules utilized to increase energy or protein intakes. The role of the renal dietician is crucial in assisting the mother in the choice of a suitable formula, in monitoring nutritional parameters, and in helping the family with the multiple formula changes required.

Progression to dialysis will affect also the nutritional requirements. Prior to dialysis, remnant kidney function and the requirements associated with solute clearance produce copious amounts of dilute urine. At end stage, urine output decreases and fluid overload becomes a concern. Attention to dietary intake will require marked alteration of fluid and nutrient intake and, where appropriate, will be noted in the sections below.

Infants with CKD do not require higher energy intakes than normal infants. Estimates of total energy requirements for normal infants have been calculated based on average values for breast-fed infants [10]. Since there are variations present within the normal population, it is understandable that infants with CKD would show at least the same variability. The Fluid and Nutrition Board has determined estimated energy requirements (EER) for healthy infants based on estimates of total energy expenditure plus the energy required for growth and tissue deposition. This data can be presented several ways but is most easily seen in Table 6.3 [11]. It should be noted that these calculations assume normal body size/age and were not designed for use in infants who were growth retarded and in whom catch-up growth is desired. It has been suggested that for these children, the appropriate choice for energy intake would be that of an average child of the same height [11]. As can be seen, moving higher on the table automatically increases the energy intake/kilogram body weight. Experimental evidence suggests that intakes of 100–120 % of normal are appropriate and that energy intakes >130 % of normal are unlikely to promote linear growth but may lead to obesity [5, 12, 13].

Table 6.1 Common neonatal and infant formula

Formula name	kcal/oz	kcal/100 mL	Prot (g)[a]	CHO (g)[a]	FAT (g)[a]	Na mEq[a]	K mEq[a]	Ca mg [a]	Ca mEq[a]	P mg[a]	P mmol[a]	Mg mg[a]	Mg mEq[a]	mOsm/kg	RSL mOsm/L[b]
Human milk	21	73	1.1	7.2	4.6	0.8	1.4	33	1.7	15	0.5	3.2	0.3	290	91
Similac® PM 60/40	20	68	1.5	6.9	3.8	0.7	1.4	37.9	1.9	19	0.6	4.1	0.3	280	124
Similac® Advance	20	68	1.4	7.2	3.8	0.7	1.8	53	2.6	28	0.9	4.1	0.3	310	127
Good Start® Gentle Plus	20	67	1.5	7.8	3.4	0.8	1.8	45	2.3	25	0.8	4.7	0.4	250	130
Enfamil® Lipil	20	68	1.4	7.6	3.6	0.8	1.9	53	2.7	29	0.9	5.4	0.4	300	130
Isomil®	20	68	1.7	7.0	3.7	1.3	1.9	71	3.6	51	1.6	51	4.2	200	155
Prosobee®	20	68	1.7	7.2	3.6	1.1	2.1	71	3.6	47	1.5	5.4	0.4	170	156
Pregestimil®	20	68	1.9	6.9	3.8	1.4	1.9	64	3.2	35	1.1	5.4	0.4	320	168
PediaSure®	30	100	2.9	13.9	3.8	1.7	3.4	106	5.3	84	2.7	17	1.4	352	278
Nutren Junior Fibre®	30	100	3.0	11.0	5.0	2.0	3.4	120	6.0	84	2.7	20.0	1.6	350	256

[a]Values per 100 mL of standard dilution formula
[b]Renal solute load

Table 6.2 Modular additions to formulas and diets

Module	Measure	kcal	Prot (g)	CHO (g)	Fat (g)	Na (mEq)	K (mEq)	Ca (mg)	Ca (mEq)	P (mg)	P (mmol)
Similac® Human Milk Fortifier	1 packet (.09 g)	3.5	0.3	0.5	0.09	0.16	0.40	29.3	1.5	16.8	0.5
Enfamil® Human Milk Fortifier	1 vial	7.5	0.55	<0.3	0.58	0.29	0.29	29	1.5	15.8	0.5
Duocal®	1 scoop (5 g)	25	0	3.6	1.12				0.0		0.0
Polycose®	1 teaspoon (2 g)	8		1.9		0.11		0.6	0.0	0.3	0.0
Polycose® Liquid	1 mL	2		0.5		0.03	0.00				
Microlipid®	1 mL	4.5			0.5						
MCT Oil® (medium-chain triglycerides)	1 Tablespoon (15 mL)	116			14						
Beneprotein®	1 scoop (7 g)	25	6			0.7	0.9	<20			

Table 6.3 Average energy requirements during infancy

Age (months)	Average length[a]	Average Wt	EER[b]	EER/kg
1	54.2	4.4	472	107
2	57.8	5.3	567	107
3	60.6	6	572	95
4	63.0	6.7	548	82
5	65.0	7.3	596	82
6	66.7	7.9	645	82
7	68.3	8.4	668	80
8	69.7	8.9	710	80
9	71.1	9.3	746	80
10	72.4	9.7	793	82
11	73.7	10	817	82
12	74.9	10.3	844	82
15	78.3	11.1	908	82
18	81.5	11.7	961	82
21	84.4	12.2	1,006	82
24	87.0	12.7	1,050	83

Adapted from [52]
Total energy expenditure for normal infants of both sexes = 0.89 × body weight (kg) − 100
[a]Average length of male and female infants
[b]EER = estimated energy requirements as the sum of total energy expenditure (TEE) and energy deposition

The institution of hemodialysis does not appear to alter caloric requirements. However, for infants on peritoneal dialysis, it is important to take into account the energy derived from dialysate glucose, which can reach 8–12 kcal/kg/day and make a significant contribution to the total energy intake [14, 15].

6.3.2 Protein

The body's hierarchy places maintenance energy requirements above growth so that when energy requirements are not being met, amino acids will be broken down into their carbon backbones plus nitrogen, with the carbon being converted to energy and the nitrogen to urea. Thus, the first step to ensuring the anabolism necessary for growth is the provision of adequate energy. Again, in the absence of intercurrent illness or surgery, there is no evidence to suggest that infants with CKD have increased protein requirements. On the other hand, provision of excess protein will not lead to improvement in nitrogen balance but rather the formation of excess urea nitrogen and subsequent azotemia. Attempts to delay the progression of renal failure by the institution of low-protein diets to infants and children with CKD have been unsuccessful and may be associated with diminished growth [16, 17]. As renal function declines, however, protein restriction should be enforced and intake limited to currently published standards (Table 6.4).

Table 6.4 Macronutrient intakes for normal infants[a]

Infants	Protein g/kg/day	Sodium g/day	Phosphorous mg/day	Calcium mg/day	Potassium g/day
0–6 month	1.5	0.12	100	210	0.4
7–12 month	1.5[b]	0.37	275	270	0.7

Adapted from [26, 52, 53]

[a]Values are noted as adequate intake (AI), the recommended average daily intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate—used when an DRI cannot be determined

[b]Value listed as the DRI—adequate for 97.5 % of the population [52]

6.3.3 Sodium

Sodium is required for growth, and during the first 6 months, infants must retain approximately 2 mEq/day to account for the increase in total body sodium associated with increased ECF [18]. Breast-fed infants consume about 1 mEq/kg/day and are able to achieve a positive sodium balance due to the ability of normal renal tubules to avidly conserve sodium. In children with CAKUT, tubular damage leads to an increase in the fractional sodium excretion and a negative sodium balance [19–21]. These infants are particularly at risk at times of intercurrent illness when decreased oral intake, emesis, and diarrhea may combine to further increase losses and decrease renal function. The development of dehydration can lead to the rapid development of acidosis, shock, and hyperkalemia, the combination of which can be acutely life threatening. Parents need to be counseled about the importance of maintaining hydration and contacting their physicians at an early stage in their child's illness.

Sodium depletion affects extracellular volume, as well as growth and nitrogen retention [22, 23]. Maintenance of a normal extracellular volume has been shown to be vital for muscle development and bone mineralization.

Standards for normal infant sodium intakes are noted in Table 6.4, but as a rule, infants with neonatal CKD will require additional sodium chloride to be added to their feeds [24, 25]. We begin supplementation with 2–3 mEq sodium/kg/day and aim to keep serum sodium concentrations at or above 140 mEq/L, decreasing sodium intake in the presence of either hypertension or edema formation. Preterm infants may require even larger amounts due to the decreased sodium reabsorptive ability and rapid growth rates. When volume status is still uncertain, serum renin determinations may also be helpful. Early on, the presence of high urinary sodium losses is generally protective against the development of hypertension [26]. As kidney failure progresses and GFR further declines, the amount of sodium intake will have to be adjusted to prevent volume overload.

6.3.4 Potassium

For infants receiving one of the humanized milk formulas, potassium intakes are often acceptable, and normal serum potassium concentrations are maintained through a combination of limited potassium intake and augmented excretion secondary to high urine flow rates, increased urine sodium loss, and hyperaldosteronism. Occasionally, infants with CAKUT develop a form of pseudohypoaldosteronism and may require the small doses of fludrocortisone. More commonly, hyperkalemia presents in the face of intercurrent volume depletion and metabolic acidosis.

When serum potassium concentrations remain elevated in the absence of volume depletion, the use of a sodium-potassium polystyrene resin (Kayexalate®) can be added during preparation of the infant formula. The formula is then decanted and the supernatant fed to the infant [27]. This is effective in lowering potassium intake, but the exchange of potassium for sodium can lead to chronic sodium overload as each gram of resin contains 4 mEq of sodium.

6.3.5 Water

Under normal circumstances, the kidney has enormous capacity to excrete water with up to 10 % of total renal water reabsorption controlled in the collecting duct under the action of antidiuretic hormone. Renal concentrating ability is lost early in infants with CAKUT, while the ability to excrete dilute urine is maintained. Since the amount of urinary osmols to be excreted is related to dietary intake and growth [28], the inability to concentrate urine requires that sufficient free water be presented during feeding. These infants often require as much is 180–200 mL H_2O/kg/day. This can be achieved by diluting infant formula from the standard 20 kcal/oz to approximately 17–18 kcal/oz. Since infants eat to caloric satiety, an average intake of 100 kcal/kg/day will provide sufficient water intake. The ability to dilute urine is preserved until late in the course of CKD so that it is unlikely that fluid intake in this range will lead to volume overload. One area often overlooked is the contribution of formula intake to the production of urinary osmols. This is designated as renal solute load (RSL). A major determinant of RSL is protein content so that appropriate formulas for this group of patients are those with low RSL (see Table 6.1).

6.3.6 Acidosis

Infants with CKD develop acidosis due to both decreased proximal tubular bicarbonate reabsorption and decreased distal hydrogen ion excretion. It has become increasingly clear that acidosis exerts a variety of adverse effects including increased protein degradation, decreased bone mineralization, and possibly even accelerating the rate of GFR decline [29–34].

The need for alkali therapy may be masked by volume contraction. With the use of additional dietary chloride (as the sodium salt) and the use of dilute formulas, intravascular volume is likely to be restored to normal. In this situation, serum bicarbonate concentrations will decrease and the addition of base, either as bicarbonate or citrate, is required.

Acid is produced both from dietary intake and from the calcification of osteoid to bone where the uptake of calcium leads to the release of hydrogen ion. While acid production in adults is generally 1–2 mmol/kg/day, during periods of rapid growth, this may increase to as much as 3–5 mEq/kg/day [26, 35]. There is, therefore, no absolute value for bicarbonate supplementation, but sufficient alkali should be provided to maintain serum bicarbonate concentrations at or above 22 mmol/L. Development of significant metabolic alkalosis in the face of moderate doses of alkali suggests chloride depletion alkalosis. The addition of sodium without chloride generally does not lead to volume overload.

6.3.7 Calcium/Phosphorus

Both calcium and phosphorus are necessary for cellular and bone growth. Human milk provides limited amounts of both of these substances (compared with cow's milk), and adequate renal mass is required to maintain an appropriate balance for both elements. While adequate calcium absorption requires the presence of the activated vitamin D metabolite (1,25-dihydroxcholecalciferol), gastrointestinal phosphate can easily be absorbed in the absence of vitamin D stimulation. Most infant formulas contain excessive quantities of both calcium and phosphate and are inappropriate for infants with CKD. Formulas appropriate for infants with CKD more closely resemble the calcium/phosphate content of human milk with approximately a 2:1 ratio of calcium to phosphorus. It is also true that the development of rickets has been reported in severely premature infants both without evident renal disease. In these infants, however, the etiology is often due to absolute dietary calcium and/or phosphate deficiency in the face of rapid growth rates [36]. While all individuals with CKD will at some point in their progression require the administration of 1,25-dihydroxcholecalciferol, it now appears that there is a role for the maintenance of serum 25-hydroxyvitamin D concentrations as well. While there is still debate as to the minimum

requirement, these infants should, at a minimum, receive current recommended daily doses of calciferol or ergocalciferol [37].

6.3.8 Supplemental Feeding

Approximately 40 years ago, Holliday [38] first noted that CKD was associated with decreased caloric intake and that growth could be improved, but not normalized, by caloric supplementation. This has been abundantly confirmed by others [6, 9, 39]. Early attempts to improve intake led to the use of highly concentrated formulas with a high-osmolar load; this approach had the paradoxical effect of increasing renal solute load in the face of decreased tubular concentrating ability. The net effect was the worsening of azotemia, hyperkalemia, and, often, the premature institution of dialysis.

Poor intake may be secondary to poor appetite and/or frequent vomiting, both of which may be clinical manifestations of acidosis or uremia [40]. Symptoms of vomiting, irritability, and discomfort may also be suggestive of gastroesophageal reflux in infants with CKD [41].

When oral intake does not suffice, tube feeding should be initiated to provide adequate nutrition for both predialysis and dialysis-dependent individuals [40]. The use of enteral feeds has allowed physicians to tailor their nutritional therapy and insure more consistent intakes. It also relieves parents of concerns regarding their inability to provide adequate nutrition when their infant will not drink adequate daily volumes. In a large study of infants with CKD who presented at less than 6 months of age, 80 % were tube fed and had a mean height SDS within normal range at 1 year of age [42]. In another study, 12 infants were started on supplemental enteral feeding in association with PD and showed significant improvements in height, weight, and head circumference SDS at 1 year [43].

Nissen fundoplication may be required in severe cases of intractable vomiting and is often performed at the time of gastrostomy tube placement. More recently, the use of gastrojejunal tubes has been suggested as an alternative to fundoplication, but there is little published data on the use of this approach in children with CKD.

Infants who require tube feeds often develop oral feeding dysfunction [44] and require referral to feeding clinics. In our experience, this remains a problem throughout the pretransplant and early transplant period, resolving only after good renal function is established.

For infants and children with advanced renal disease, several formulas have been designed to provide adequate energy and protein intakes with limited fluid and electrolyte content. The use of these formulas implies a significant degree of renal insufficiency and the importance of nephrologic as well as nutritional involvement (Table 6.5).

6.3.9 Growth Hormone

The administration of growth hormone to infants is controversial. If the growth schema noted by Karlberg [1] is correct, then growth hormone is not a major growth stimulant until the later part of the first year. The first 6–9 months of life should therefore be devoted to aggressive attempts to institute and maintain appropriate oral/enteral nutrition and prevent the previously described decline in growth parameters often seen in these children. For those infants in whom growth rates are still not acceptable after adequate nutritional intake is assured and metabolic bone disease has been addressed, the use of recombinant human growth hormone may provide additional improvement in statural growth [45].

6.4 Conditions with Normal GFR

6.4.1 Congenital Nephrotic Syndrome (NS)

Congenital nephrotic syndrome is by definition a condition appearing either at birth or within the first 3 months of life. Unlike CKD, GFR is generally normal early in life, and the main nutritional concerns appear to relate to the provision of a high-energy, high-protein, and low-sodium

Table 6.5 Specialized renal formulas

Formula name	kcal/oz	kcal/100 mL	Prot (g)[a]	CHO (g)[a]	FAT (g)[a]	Na mEq[a]	K mEq[a]	Ca mg[a]	Ca mEq[a]	P mg[a]	P mmol[a]	Mg mg[a]	Mg mEq[a]	mOsm/kg	RSL mOsm/L[b]
Nepro with Carb Steady®[c] Vanilla	54	180	8.1	16.1	9.6	4.6	2.7	106.0	5.3	72.0	2.3	21.0	1.7	745	555
Novasource Renal® (BrikPak)	60	200	9.1	18.3	10.0	4.1	2.4	84.0	4.2	81.9	2.7	19.7	1.6	800	635
Re/Gen HP/HC®[c] Vanilla	58	195	6.8	26.6	9.6	4.4	0.3	8.5	0.4	38.4	1.2	1.7	0.1	225	770
Renalcal®[d]	60	200	3.4	29.0	8.2	0.3	0.2	6.0	0.3	10.0	0.0	2.0	0.0	600	145
Renastart®[e]	30	100	1.5	12.5	4.8	2.1	0.6	22.6	1.1	18.4	0.6	10.6	0.9	225	125
Suplena with Carb Steady®[f] Vanilla	54	180	4.5	19.6	9.6	3.5	2.9	105.5	5.3	71.7	2.3	21.5	1.8	600	344

[a]Values per 100 mL of standard dilution formula
[b]Renal solute load
[c]Intended from individuals on dialysis
[d]Not a nutritionally complete formula
[e]For birth through 10 years of age
[f]Designed for patients with CKD not on dialysis

intake. While the best described form of congenital nephrotic syndrome is the Finnish type, noted for genetic defects in the Nephrin gene, there are a variety of other genetic causes known. In all cases, it is necessary to aggressively limit sodium intake while providing adequate energy and protein intake. These children have traditionally been treated with high-energy formulas, providing up to 130 kcal/kg/day [46]. Human milk or milk formulas may be used, with added glucose polymers to increase the caloric value. Rapeseed, sunflower, or fish oil are generally added to increase the ratio of monounsaturated and polyunsaturated fatty acids. Protein intakes as high as 3–4 g/kg/day are necessary [46, 47] and may be provided in the form of casein-based protein additives. These children may also require intravenous albumin infusions as an adjunct to their enteral feeds to maintain adequate serum albumin concentrations and prevent gross edema necessary [46].

Due to the massive protein loss in the urine, all of these children develop hypothyroidism and require early thyroxine supplementation. Vitamin D supplementation is required, but correction of 25-hydroxyvitamin D concentrations is difficult due to massive urinary losses. During the first several years of life, there is a progressive decline in renal function. These children all develop CKD and will eventually require renal transplantation. The timing of nephrectomy and transplantation is dependent at least to some degree upon the ability to maintain adequate nutrition in the face of nutritional debility [46–48].

6.4.2 Tubular Lesions

Tubular lesions such as renal tubular acidosis, hypophosphatemic states, diabetes insipidus, neonatal Bartter syndrome, or rare cases of the Fanconi syndrome may present within the neonatal period with evidence of volume depletion and electrolyte abnormalities. While specific diagnoses may be delayed, the rapid institution of volume replacement therapies may be lifesaving. In the absence of a family history, the diagnoses of stone-forming diseases such as cystinuria or hyperoxaluria are rarely made within the neonatal period. A full discussion of renal tubular disorders is beyond the scope of this chapter and the reader is referred to Chap. 4 for more complete information.

6.5 Neonatal Acute Kidney Injury (AKI)

6.5.1 Conservative Care

The nutritional care of neonates and infants with AKI is complex, requires the care of multiple services, and should be done only in medical centers with a full complement of pediatric subspecialty care. Children with AKI often present as part of a more general picture of sepsis, congenital heart disease, or hypoxic ischemic encephalopathy. After the initial period of weight/fluid loss, the neonatal period is characterized by a propensity to anabolism, necessary to fuel cellular growth. It is quite difficult to achieve anabolism in any patient with AKI and perhaps even more so in neonates. Often the best that can be achieved is a limitation of tissue catabolism through the provision of a high concentration of energy delivered either enterally or, more often, parenterally. In this instance, the infant's anabolic drive is helpful, and with careful control of energy, protein, and electrolyte intakes, it is possible to avoid dialysis in a significant percentage of infants. Where possible, enteral intake is preferred and formulas can be adapted in a stepwise fashion to provide increasing amounts of energy and protein intake, generally by starting with human milk or a humanized milk formula with the addition of energy in the form of complex carbohydrates or fat, while protein intake can be adjusted upward through the use of protein concentrates (see Table 6.2).

When AKI persists, it is often necessary to institute dialysis in order to achieve adequate nutritional intake. Again, there is no data to suggest that infants with AKI require larger energy or protein intakes than individuals receiving parenteral nutrition for other reasons, and attempts should be made to provide energy and

Table 6.6 Energy and protein intakes for infants requiring dialysis

	Predialysis		Hemodialysis		Peritoneal dialysis	
	Energy[a]	Protein[b]	Energy[a]	Protein[b,c]	Energy[a,d]	Protein[b,e]
0–6 months	100–110	2.2	100–110	2.6	100–110	3
6–12 months	95–105	1.5	95–105	2	95–105	2.4
1–3 years	90	1.1	90	1.6	90	2.0

[a]kcal/kg/day
[b]g/kg/day
[c]Protein intakes increased by approximately 0.4 g/kg/day to account for hemodialysis losses
[d]*Note*: up to 10 % of the total caloric intake (10 kcal/kg/day) can be absorbed as dextrose via the dialysate. Obesity may become a concern for some children and adolescents on peritoneal dialysis
[e]Protein requirements on peritoneal dialysis reflect the significant loss of proteins through the dialysis fluid

protein intakes based on gestational age and size. If possible, the use of indirect calorimetry can be helpful in determining true energy requirements [49, 50].

Current low-solute infant formulas often contain "humanized" concentrations of electrolytes. It is often necessary to modify these formulas to better control calcium, phosphate, alkali, and potassium intake. The addition of calcium will help to decrease phosphate uptake as well as improve serum calcium concentrations. Base can be adjusted by the addition of sodium bicarbonate or citrate. Potassium can also be adjusted, most often decreased by the addition of a sodium-potassium polystyrene resin (Kayexalate®).

6.5.2 Dialysis

Infants with CKD rarely require dialysis in the neonatal period as severe intrauterine renal failure and oliguria will lead to pulmonary hypoplasia and early neonatal demise. The nutritional care of infants requiring dialysis is complex, and adaptations will reflect the type of dialysis therapy used. Given the small size and limited vascular access, peritoneal dialysis is most often utilized for infants with CKD, but the use of hemodialysis and continuous renal replacement therapy (CRRT) all have been reported for this age group. As noted previously, infants on peritoneal dialysis receive a daily transfer of 8–12 kcal/kg via glucose absorbed. Infants on CRRT are often dialyzed using solutions containing glucose. The glucose lost during hemodialysis is not significant. Protein losses can be significant for infants on dialysis, and it has been suggested that protein intakes be increased during both hemo- and peritoneal dialysis (Table 6.6). There is little data on the loss of amino acids for infants on CRRT, but what data is present suggests that children undergo substantial amino acid and protein losses during this procedure [51]. For infants undergoing peritoneal dialysis, it is important to consider the interaction of increased intra-abdominal volume/pressure and rate of nighttime enteral intake, the combination of which may lead to increased filling pressure, worsening gastroesophageal reflux, and consequent decreased food intake.

Conclusion

Normal kidney function gives humans the ability to ingest a wide range of nutritional intakes allowing us to both excrete excess and conserve as conditions demand. During the first year of life, growth rates are high, and infants are primed to conserve electrolytes while excreting the large fluid volumes present in human milk. When renal disease is present during infancy, it becomes more difficult to achieve these goals. Appropriate nutritional care requires the cooperative efforts of physicians and nutritionists working with the family to achieve the optimal growth possible for these infants.

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