Original article

Growth of children following the initiation of dialysis: a comparison of three dialysis modalities

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Abstract. Maintenance dialysis usually serves as an interim treatment for children with end-stage renal disease (ESRD) until transplantation can take place. Some children, however, may require dialytic support for an extended period of time. Although dialysis improves some of the problems associated with growth failure in ESRD (acidosis, uremia, calcium, and phosphorus imbalance), many children continue to grow poorly. Therefore, three different dialysis modalities, continuous ambulatory peritoneal dialysis (CAPD), cycler/intermittent peritoneal dialysis (CPD), and hemodialysis (HD), were evaluated with regard to their effects on the growth of children initiating dialysis and remaining on that modality for 6-12 months. Growth was best for children undergoing CAPD when compared with the other two modalities with regard to the following growth parameters: incremental height standard deviation score for chronological age $[-0.55 \pm 2.06 \text{ vs.} -1.69 \pm 1.22]$ for CPD (P < 0.05) and -1.80 ± 1.13 for HD (P < 0.05)]; incremental height standard deviation score for bone age $[-1.68 \pm 1.71 \text{ vs. } -2.45 \pm 1.43 \text{ for CPD } (P = \text{NS}) \text{ and}$ -2.03 ± 1.28 for HD (P = NS)]; change in height standard deviation score during the dialysis period $[0.00\pm0.67 \text{ vs.}]$ $-0.15 \pm .29$ for CPD (P = NS) and $-0.23 \pm .23$ for HD (P = NS)]. The reasons why growth appears to be best in children receiving CAPD may be related to its metabolic benefits: lower levels of uremia, as reflected by the blood urea nitrogen $[50 \pm 12 \text{ vs. } 69 \pm 16 \text{ mg/dl} \text{ for CPD } (P < 0.5)]$ and 89 ± 17 for HD (P < 0.05)], improved metabolic acidosis, as indicated by a higher serum bicarbonate concentration $[24\pm2 \text{ mEq/l vs. } 22\pm2 \text{ for CPD } (P < 0.05) \text{ and}$ 21 ± 2 for HD (P < 0.05)]. In addition, children undergoing CAPD receive significant supplemental calories from the glucose absorbed during dialysis. CAPD, and possibly, other types of prolonged-dwell daily peritoneal dialysis

Correspondence to: B. A. Kaiser, St. Christopher's Hospital for Children, Erie Avenue at Front Street, Philadelphia, PA 19134, USA appear to be most beneficial for growth, which may be of particular importance for the smaller child undergoing dialysis while awaiting transplantation.

Pediatric

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Key words: Growth – Hemodialysis – Continuous ambulatory peritoneal dialysis – Cycler/intermittent peritoneal dialysis

Introduction

In the 3 decades since West and Smith [1] first attempted to elucidate the multiple factors responsible for growth impairment in children with renal disease [2-4], much progress has been made in both dialysis and transplantation; growth failure, however, remains a persistent problem [5, 6]. It is clear that the etiology of the growth failure associated with end-stage renal disease (ESRD) is multifactorial and includes problems such as: caloric and protein malnutrition [7-11], metabolic acidosis, and other electrolyte disturbances [12, 13], the accumulation of uremic toxins [14-16], renal osteodystrophy [17-19], hormonal disturbances [20-22], psychosocial problems [23], age at onset, and the natural history of the primary disease [24]. Although dialysis may control or lessen the impact of some of these factors, in general it has not greatly benefited growth, which has prompted pediatric nephrologists to more aggressively pursue transplantation [25]. There have been many reports detailing the success, or lack thereof, of dialysis on growth in children [26-34]. In an attempt to document whether any of the various dialysis modalities available to children affect growth differently, we compared growth, nutrition, and various biochemical values of children initiating either hemodialysis (HD), short-cycle cycler/ intermittent peritoneal dialysis (CPD), or continuous ambulatory peritoneal dialysis (CAPD). To our knowledge this is the first single-center study comparing these three modalities in a population of children from the time of initiation of maintenance dialysis.

Patients and methods

Since 1980 we have offered three distinct modalities to children starting maintenance dialysis: (1) HD performed as an in-center procedure, usually 4 h three times weekly. Most of these treatments were performed using Gambro parallel-plate dialyzers with an acetate-based dialysate containing glucose. Blood flow and kidney size were initially chosen to provide urea clearances of 2-3 ml/min per kg body weight and were subsequently increased or decreased on the basis of the results of monthly blood studies and patient tolerance. (2) CAPD used peritoneal dialysate (Dianeal PD-1) volumes of 40 (range 30-50) ml/kg and four or five daily exchanges of 4-8 h duration. The dialysate glucose concentrations used varied from 1.5% to 4.25% depending upon the ultrafiltration needs of each child. (3) CPD was performed only as a home procedure at night, using an automated cycler (American Medical Products) with dialysate volumes of 40 (range 30-50) ml/kg, fill/dwell times of 20-90 min, and drain times of 10-15 min. The total number of exchanges per treatment ranged from 6 to 24, and dialysate glucose concentrations ranged from 1.5% to 3.2%. These were both adjusted, along with the number of treatments per week, based upon the metabolic and ultrafiltration needs of each child. Most children required treatments 4 (range 3-6) nights per week. At the end of each CPD treatment each child's peritoneal cavity was filled with only a quarter to a half of their routine exchange volume.

Families were generally allowed to choose between the three modalities, with more specific recommendations made only when dictated by a given child's unique clinical situation. Peritoneal dialysis was recommended for all children less than 10 kg and for smaller children (<25 kg) whose families lived at a great distance from our center, in whom HD seemed less desirable. Finally, parents selecting peritoneal dialysis were required to complete a 2-week training course and to pass an examination, since peritoneal dialysis was only performed at home for the children included in this report. The children evaluated were starting dialysis for the first time (no history of prior maintenance dialysis or transplantation); during the time of study, all had been receiving one form of dialysis continuously for a minimum of 6 to a maximum of 12 months in order to standardize data comparisons. All dialysis prescriptions were controlled at our center.

Seventeen children receiving CAPD (age range 0.8-16.3 years) and 25 receiving CPD (0.9-16.3 years) and 23 receiving HD (1.6-15.1 years) were evaluated for dialysis periods of 9.4 ± 2.5 , 10.1 ± 2.6 , and 10.1 ± 2.5 months, respectively (P = NS). Only children with growth potential, either pre-pubertal (Tanner I) or early puberty (Tanner II or III) were evaluated. The etiologies of their renal failure were divided for comparison into three categories: nephritis (including hemolytic uremic syndrome) 6 patients (35%) starting CAPD, 12 (48%) starting CPD, 9 (39%) starting HD; congenital/urological (hypoplastic/dysplastic and obstructive) 6 patients (35%) starting CAPD, 10 (40%) starting CPD, and 9 (39%) starting HD; miscellaneous inherited disorders (cystinosis, infantile polycystic kidney disease) 5 patients (29%) starting CAPD, 3 (12%) starting CPD, 5 (17%) starting HD. The initiation of dialysis was based on clinical needs after failure of conservative management, and all children had levels of renal function below 20% of normal. Failure of a patient to complete a full 12-month study period was due to either transplantation [CAPD 6 patients (35%), CPD 11 (44%), HD 8 (35%)] or change in modality [CAPD 1 patient (6%), CPD 2 patients (8%), HD 1 patient (5%)]. Pre-dialysis growth rates were not evaluated because a majority of our dialysis population were either referred near the time of initiation of dialysis (25%) or had sudden onset of chronic renal failure (23%) with incomplete growth data.

Height was measured every 1-2 months using a stadiometer or infant stature board. The growth variables monitored included the following; bone age determined at the initiation of dialysis using estimates from the Greulich and Pyle standards [35]; height age, that is the age at which a child's actual height would correspond to the 50th percentile; standard deviation score (Z score) [36] for height or length, calculated as the difference between the patient's height (X₁) and the mean height (X₂) for chronological age and sex of a reference population, divided by the standard deviation of that mean for the reference population [37]; Z score = (X₁-X₂/SD).

Incremental statural growth was calculated during the period of dialysis. These growth rates were evaluated and compared using three methods; the change in (\triangle) Z score for height based on chronological age over the dialysis interval studied; incremental growth velocity, compared by using a Z score determined from incremental growth tables [38] for both chronological and bone age.

Biochemical variables, including blood urea nitrogen (BUN), serum sodium, potassium, bicarbonate (S_{HCO3}), total calcium (S_{Ca}), phosphorus (S_{phos}), alkaline phosphatase, and albumin, were measured monthly by routine automated laboratory techniques. The values expressed for each variable represent means for the entire dialysis study period. To better evaluate the ability of dialysis to correct metabolic acidosis, we also compared the number of children in each group whose initial requirement for supplemental oral bicarbonate, to maintain the S_{HCO3} above 20 mEq/l, resolved after the initiation of maintenance dialysis.

The presence and severity of renal osteodystrophy was evaluated by determining the changes in radiographic findings in each patient over the dialysis interval studied. Hand and knee radiographs were performed at the initiation of dialysis and then every 6-12 months, and changes in bone findings graded as stable, improved, or worse. All children received vitamin D supplementation (calcitriol or dihydrotachysterol) while receiving dialysis and doses were adjusted to maintain the S_{Ca} concentration between 9.5 and 11.0 mg/dl. Vitamin D was in use pre dialysis in 10 patients (66%) starting CAPD, 12 (48%) starting CPD, and 13 (52%) starting HD (P = NS). All children required phosphate binder therapy while undergoing dialysis, regardless of the modality. Phosphate binders, both calcium carbonate and aluminum hydroxide, were administered in doses which were adjusted to maintain the Sphos concentration below 7 mg/dl for patients 5 years of age and less and below 6 mg/dl for older children. Aluminium binders were gradually discontinued and not in use by 1985, unfortunately aluminum levels are not available in these children, but none had clinical evidence of aluminum-related bone disease. Because of the extended time period over which patients were evaluated, the changes in parathyroid hormone methodology did not allow for group comparisons.

Nutritional status was evaluated at the initiation of dialysis and then every 3–6 months using a 3-day diet recall. From this, the percentage of recommended daily allowance (RDA) for energy (calorie) and protein, as established by the Food and Nutrition Board of the National Academy of Science [39] and based on height age, was estimated for each patient. Nutritional supplements [glucose polymers (Polycose), corn syrup, calcium caseinate (Casec)] were prescribed for all children whose intake persistently fell below 80% of the RDA. Nasogastric tube feedings were also used for supplementation in 5 children (2 CAPD, 2 CPD, 1 HD). All children and families received individualized dietary recommendations in an attempt to modify their diets not only for calories, but also for protein, phosphorus, and potassium intake, as needed. No children in this study were receiving erythropoietin or human growth hormone.

Statistical comparisons between dialysis modalities were performed by single analysis of variance and chi-squared analysis using SPSS (V.4.1) format IBM VM/CMS. Data are expressed as mean plus or minus standard deviation.

Results

The patient population data for the three dialysis modalities are presented in Table 1. There were no statistically significant differences between the groups for any of the variables listed. However, bone age and height age were slightly lower, while the degree of initial growth retardation, as indicated by the degree of negativity of the Z scores for initial height, was higher in the CAPD group. This is attributable to the higher proportion of children in this group who developed renal insufficiency at an early age (prior to 2 years), which would be expected to cause a greater degree of

Table 1. Study variables at the initiation of dialysis^a

<u> </u>	Chronolo- gical age (years)	Bone age (years)	Height age (years)	Initial height (cm)	Initial SDS ^b	Sex (M/F)	Puberty ^c I/II – III	Onset of disease ^d (E:L)	Chronicity of disease ^e (A:C)	Residual renal function ^f (Yes:No)
$\overline{\text{CAPD}}_{(n = 17)}$	8.3 ±6.0	5.8 ±5.1	5.6 ±5.3	107.0 ±35.0	-2.60 ± 2.70	11:6	13:4	11:6	4:13	4:13
CPD (<i>n</i> = 25)	8.2 ±4.4	6.9 ±4.3	6.7 ±3.9	117.0 ± 26.6	-1.41 ±1.43	14:11	21:4	10:15	7:18	8:17
HD (<i>n</i> = 23)	8.9 ±4.2	7.2 ±3.6	6.9 ±3.6	119.5 ±22.9	$^{-1.48}_{\pm1.28}$	14:9	20:3	12:11	5:18	6:17

CAPD, Continuous ambulatory peritoneal dialysis; CPD, cycler peritoneal dialysis; HD, hemodialysis

^a No statistically significant differences were noted between modalities

^b Initial SDS, standard deviation score of height for chronological age at the initiation of dialysis

 $^\circ\,$ Puberty, by Tanner stage either pre-pubertal (I) or early puberty (II or III)

^d Onset of disease, age when renal insufficiency had significant effect: $E = early (\leq 2 years); L = late (> 2 years)$

• Chronicity of disease: length of time over which renal insufficiency had significant effect prior to dialysis A = actue (<6 months); C = chronic (>6 months)

f Residual renal function; based on urine output ≥ 1 ml/kg per hour

Table 2. Biochemical status expressed as means of monthly values during time on dialysis

	BUN (mg/dl)	S _{Na} (mEq/l)	S _K (mEq/l)	S _{Cl} (mEq/l)	Shco3 (mEq/l)	Bicarbonate therapy pre ^a (Yes:No)	Bicarbonate therapy during ^b (Yes:No)
$\begin{array}{l} \hline CAPD \\ (n = 17) \end{array}$	50*,** ±12	138* ±3	4.5** ±0.5	100 ± 4	$24^{*,**}$ ± 2	12:5	3:14**
CPD (<i>n</i> = 25)	69*,*** ±16	$^{141^{*,***}}_{\pm 2}$	$4.8^{***} \pm 0.4$	103*** ±3	22* ±2	13:12	11:14***
HD (<i>n</i> = 23)	89**,*** 土17	138*** ±3	5.1**.*** ±0.7	99*** 土4	21** 土2	19:4	21:2**,***

BUN, Blood urea nitrogen; S_{Na} , serum sodium; S_K , serum potassium; S_{Cl} , serum chloride; S_{HCO3} , serum bicarbonate

^a Number of children requiring supplemental bicarbonate therapy prior to dialysis and ^b after being stable on dialysis

Table 3. Factors evaluating renal osteodystrophy of children undergoing dialysis $^{\rm a}$

	Calcium ^b (mg/dl)	Phos- phorus ^b (mg/dl)	Alkaline phosphatase ^b (U/l)	Aluminum binders ^c (Yes:No)	Bone Δ^d (S:W)
$\overline{\begin{array}{c} \text{CAPD} \\ (n = 17) \end{array}}$	9.6 ±0.7	5.1 ±0.7	270 ±176	9:8	8:7
CPD (<i>n</i> = 25)	9.8 ±0.8	5.6 ±1.2	238 ±149	11:14	18:7
HD (<i>n</i> = 23)	9.8 土0.7	$\begin{array}{c} 5.3 \\ \pm 0.8 \end{array}$	335 ±189	11:12	10:12

^a No statistically significant differences were noted between modalities

^b Means for monthly values during the study period

 Patients receiving aluminum hydroxide phosphate binders while undergoing dialysis

^d Degree of change in severity of renal osteodystrophy as determined from radiographs of the hands and knees obtained during the study period: S = stable or improved; W = worse

growth retardation than that resulting from the later onset of renal disease. However, the differences regarding these variables did not reach statistical significance and their effect on growth if any is uncertain.

The biochemical differences between groups are summarized in Table 2. The overall degree of biochemical *
 P < 0.05 CAPD vs. CPD; ** P < 0.05 CAPD vs. HD; ***
 P < 0.05 CPD vs. HD

control appeared to be much better in the peritoneal dialysis groups. It is important to note that these differences were due at least in part to the time of blood sampling: during CAPD cycles, and usually post CPD treatments, but pre treatment for those children undergoing HD. Nonetheless, children receiving CAPD had normal mean values for electrolytes and a mean BUN of only 50 ± 12 mg/dl, values nearly 30% and 45% lower than those for the CPD and HD patients, respectively. In addition, these represent a steadystate of biochemical and metabolic balance for the CAPD patient. Although the BUN provides only a rough estimate of the degree of uremia, it would appear that children undergoing CAPD are less uremic than those children undergoing CPD or HD. Also important was the superior degree of correction of metabolic acidosis by CAPD: not only did children undergoing CAPD have higher SHCO3 concentrations, but almost all were able to discontinue their oral sodium bicarbonate supplements as well (Table 2).

Factors associated with the control of renal osteodystrophy are reviewed in Table 3. All dialysis modalities resulted in nearly identical S_{Ca} and S_{phos} concentrations, similar levels of alkaline phosphate, and aluminum binder use and they were equally inept at preventing the progression of renal osteodystrophy. Of the 65 children evaluated by initial and follow-up bone films after up to a year of dialysis, 26 showed a worsening of renal osteodystrophy

 Table 4. Evaluation of nutritional status of children undergoing dialysis

	% RDA ^a	% RDA ^a	Albumin ^b
	calories	protein	(g/dl)
$\overline{\text{CAPD}}_{(n = 17)}$	71	119	3.5*
	±15	±25	±0.3
CPD	71	123	3.6
(<i>n</i> = 25)	±22	±30	±0.5
HD	67	123	3.9*
(<i>n</i> = 23)	±14	±25	±0.3

RDA, Recommended daily allowance

*P < 0.05 CAPD vs. HD

^a Percentage of RDA for height age from all dietary sources, reported as a mean for all dietary recalls done over the dialysis period

^b Mean of monthly values during dialysis

Table 5. Growth data of children during the dialysis study period

	Final height	Final height	Height ∆SDS ^b	Incremental height ^c Z score for	
	(cm)	2D2ª		CA	BA
CAPD (n = 17)	112.3 ±33.1	-2.55 ±2.41	-0.004 ± 0.67	$-0.55^{*,**}$ ± 2.06	$^{-1.68}_{\pm 1.71}$
CPD (<i>n</i> = 25)	$\begin{array}{c} 120.0 \\ \pm 25.3 \end{array}$	-1.65 ±1.43	-0.15 ± 0.29	-1.69* ±1.22	-2.45 ± 1.43
HD (<i>n</i> = 23)	122.3 ± 23.0	$^{-1.80}_{\pm 1.51}$	0.23 ±0.23	$^{-1.80**}_{\pm 1.13}$	-2.03 ± 1.28

CA, Chronological age; BA, bone age

*P < 0.05 CAPD vs. CD; **P < 0.05 CAPD vs. HD

^a Final height SDS: SDS for height, based on CA at the end of the study period

^b Change (Δ) in the height SDS for CA for the initial 6-month period of dialysis

^c SDS (Z score) for the incremental growth in height over a 6-month period during dialysis based both on CA and BA

while 36 were stable or slightly improved, and 3 did not have follow-up films. This deterioration occurred despite the use of active vitamin D metabolites, calcium supplementation, and phosphate binders, prescribed in an effort to maintain near normal serum calcium and phosphorus concentrations.

Nutritional status, as evaluated by serum albumin concentrations, and the average of all 3-day diet recalls for calorie and protein intake during the study period are reviewed in Table 4. The results are similar, with the CAPD patients having lower serum protein concentrations, probably due to greater peritoneal losses. Protein intake was similar and high in all groups, representing attempts to improve dietary compliance. Caloric intake was disappointingly low, but similar in all groups despite our attempts to liberalize diets. CAPD patients had an advantage, however, in that a significant amount of glucose can be absorbed from the dialysate during this procedure. In an attempt to estimate the degree of peritoneal glucose absorption, 9 of the children receiving peritoneal dialysis underwent an evaluation to determine the percentage of glucose absorbed over 8 h from a 40-ml/kg exchange with 2.5% glucosecontaining dialysate solution [40]. Mean percentage glucose absorption was as follows: 30% at 1 h (0.72 cal/kg), 39% at 2 h (1.4 cal/kg), 54% at 4 h (1.9 cal/kg), and 80% at 8 h (2.9 cal/kg). Thus, for a typical child undergoing CAPD and receiving four daily 4-h exchanges and one for 8 h overnight using all 2.5% solution, the estimated calorie delivery for a 24-h period would be 10.5 cal/kg from dialysate glucose, representing a substantial amount of non-protein calorie supplementation. CPD patients would also receive some caloric boost with glucose absorption, but only about a quarter the amount and not on a continuous basis.

Growth evaluations are summarized in Table 5. In children undergoing CAPD the change in mean Z score for height was essentially zero during the period of dialysis, meaning they did not fall further from normal, while children receiving CPD and HD had negative changes indicating a further decrease during the dialysis period, although these differences did not reach statistical significance. However, the Z score for incremental height velocity based on chronological (but not bone) age was significantly higher for CAPD patients when compared with the corresponding group values for children receiving HD and CPD. Normal growth can be defined as being present in children with interval height increments within two standard deviations of the normal mean [38]. This was highest in children receiving CAPD, 13 of 17 (76%) compared with 13 of 25 (57%) and 14 of 23 (61%) children undergoing CPD and HD, respectively. These differences, however, did not reach statistical significance.

Discussion

From the earliest reports on chronic renal disease in childhood [1], it became apparent that any one of a number of associated metabolic problems [7-22] could result in short stature. Moreover, poor growth appeared to persist from the period of conservative management of renal insufficiency [3, 4, 14] to those of dialysis [26–34], and transplantation [6, 14, 25, 29, 31, 41, 42]; thus, at the present time, growth failure and its attendant psychological consequences remain unresolved problems [5, 6, 43–45]. As a result, trials of growth hormone therapy have been initiated in selected patients [44, 46]. Thus, it is clear that dialysis alone cannot be expected to reverse all of the effects of chronic renal failure necessary to restore normal growth.

Early growth data came from European reports and focused on HD; as reviewed by Kleinknecht et al. [26], children receiving HD seem to fall into three groups: a third grow normally, a third exhibit moderately and a third severely reduced growth. Catch-up growth was never seen, and children tended to continue to grow at their pre-dialysis rates. Early reports comparing CAPD with HD found improved growth [27, 28]. Fennell et al. [29] reported significantly improved linear growth in patients receiving CAPD, with the heights of 50% of the children studied increasing at a normal rate, that is, at greater than 80% of expected height velocity. In studies in which prolonged dwell continuous daily cycler dialysis (4–6 cycle exchanges overnight and a 12- to 16-h daytime exchange) was evaluated, it was found to be as beneficial as CAPD [30–32, 34], with children noted to have grown exceptionally well, at 88% of expected height velocity [30]. Thus, the literature to date suggests that the continuous forms of peritoneal dialysis are associated with better growth then HD.

Since 1980 we have provided three major dialysis modalities for our patients: HD, with three 4-h weekly treatments performed in our pediatric center; CAPD performed with four or five daily exchanges; and a form of cycler peritoneal dialysis, beginning with four nightly 8- to 12-h treatments per week. We selected this type of cycler dialysis instead of continuous daily treatments to allow children and their families time away from the procedure in the hope of minimizing burnout, a major problem during our early CAPD program. The children we studied were all starting chronic dialysis for the first time and had not yet undergone renal transplantation. They were selected in this manner in the hope of developing a clearer picture of the impact of each dialysis modality on growth. During the study period, physician and nutritionist input was consistent as was the medical therapy prescribed for the children, with the exception of two gradual changes: a shift from aluminum to calcium-containing phosphate binders and the initiation of nasogastric tube feedings in a few children. In this reported population, nasogastric feedings were used only as a supplement after oral intake alone failed to reach adequate intake, and not as a form of total nutritional replacement therapy.

The retrospective nature of this study and the relatively small number of children evaluated for each dialysis modality did not allow us to compare growth prior to with that following initiation of dialysis in individual patients, nor control for the potential motivational bias that can influence a family's decision to select home versus in-center dialysis. However, our results again confirm that children undergoing CAPD experience the best growth of the three modalities studied, as indicated from standard deviation scores for incremental height growth velocity which were significantly better than for CPD or HD based on chronological age. In addition, comparing growth rates during dialysis using the change in standard deviation for height during dialysis, CAPD was associated with the least negative results of all three modalities. Nonetheless, overall standard deviation scores remained negative in all groups, indicating that growth remained suboptimal for many children undergoing dialysis.

We believe that the relative improvement in growth seen in children undergoing CAPD was secondary to some of the metabolic advantages this modality confers over the others. Metabolic acidosis is better controlled even without medication and in a much more constant manner which should also function to benefit growth. However, the current change to bicarbonate-based dialysate for children may make this benefit less dramatic. Although, dietary protein and calorie intakes were similar in all groups, children undergoing CAPD also receive a significant caloric supplement from absorbed dialysate glucose, which may represent 5%-10% of their RDA for calories. Similar benefit never occurs with HD and in a much smaller quantity with CPD. In addition, this constant infusion of glucose during CAPD should increase insulin release, which may result in better protein utilization [47]. Finally, since our study lacks the information needed to calculate urea kinetics and thus to compare the adequacy of dialysis, among the three groups [48], it is difficult to evaluate differences with regards to the true degree of uremia between them. However, children undergoing CAPD did have significantly lower BUN concentrations than those receiving CPD or HD, and although this resulted, in part, from sampling methods and the continuous nature of CAPD, based on the results of the National Cooperative Dialysis Study [49], these observations may predict a lower incidence of uremia-related morbidity among CAPD patients. In as much as growth failure may be the most significant manifestation of uremia-related morbidity in children, the fact that our CAPD patients exhibited standard deviation scores for growth velocity which were closer to normal than those of either the other groups would suggest that this modality is in fact associated with the greatest degree of adequacy of dialysis.

While we continue to believe that children should receive maintenance dialysis only until successful transplantation can be achieved, selecting the most beneficial forms will remain an important goal. Until a more extensive study of growth and dialysis is completed (one that will hopefully be prospective and will control for important variables such as nutrition and pre-dialysis growth, growth potential, and measures of dialysis adequacy), the available data indicate that either CAPD and, possibly, other forms of prolongeddwell daily peritoneal dialysis appear to be the most beneficial for growth or our standard HD modality may not be sufficient enough. These observations are of particular importance to the young child awaiting transplantation, when growth is most critical. Finally, although growth hormone has been shown to benefit dialysis patients [46], its cost may limit its use and it will be of interest to see whether children receiving different dialysis modalities respond differently or whether the presently recognized benefits of CAPD persist.

References

- West CD, Smith WC (1956) An attempt to elucidate the cause of growth retardation in renal disease. Am J Dis Child 91: 460–476
- Potter DE, Greifer I (1978) Statural growth of children with renal disease. Kidney Int 14: 334–339
- Broyer M (1982) Growth in children with renal insufficiency. Pediatr Clin North Am 29: 991–1001
- Rizzoni G, Broyer M, Guest G, Fine R, Holliday MA (1986) Growth retardation in children with chronic renal disease. Scope of the problem. Am J Kidney Dis 7: 256–261
- Gilli G, Scharer K, Mehls O (1984) Adult height in pediatric patients with chronic renal failure. Proc Eur Dial Transplant Assoc 21: 830–836
- Henning P, Tomlinson L, Rigden SPA, Haycock GB, Chantler C (1988) Long term outcome of treatment of end stage renal failure. Arch Dis Child 63: 35–40
- Simmons JM, Wilson CJ, Potter DE, Holliday MA (1971) Relation of calorie deficiency to growth failure in children on hemodialysis and the growth response to calorie supplementation. N Engl J Med 285: 653-656
- Betts PR, Magrath G, White RHR (1977) Role of dietary energy supplementation in growth of children with chronic renal insufficiency. BMJ 1: 416–418
- 9. Chantler C, Bishti M, Counahan R (1980) Nutritional therapy in children with chronic renal failure. Am J Clin Nutr 33: 1682–1689

- Holliday MA (1986) Nutrition therapy in renal disease. Kidney Int 30: S3-S6
- Wassner SJ, Abitbol C, Alexander S, Conley S, Grupe WE, Holliday MA, Rigden S, Salusky IB (1986) Nutritional requirements for infants with renal failure. Am J Kidney Dis 7: 300-305
- Cooke RE, Boyden DG, Haller E (1960) The relationship of acidosis and growth retardation. J Pediatr 57: 326–337
- Tsuru N, Chan JCM (1987) Growth failure in children with metabolic alkalosis and with metabolic acidosis. Nephron 45: 182-185
- Scharer K, Gilli G (1984) Growth in children with chronic renal insufficiency. In: Fine R, Gruskin A (eds) End stage renal disease in children. Saunders, Philadelphia, pp 271–290
- Kleinknecht C, Salusky I, Broyer M, Gubler M-C (1979) Effect of various protein diets on growth, renal function, and survival of uremic rats. Kidney Int 15: 534–541
- Broyer M, Guillot M, Niaudet P, Kleinknecht C, Dartois AM, Jean G (1983) Comparison of three low-nitrogen diets containing essential amino acids and their alpha analogues for severely uremic children. Kidney Int 24 [Suppl 16]: S290–S294
- Mehls O, Ritz E (1983) Skeletal growth in experimental uremia. Kidney Int 24 [Suppl 15]: S53-S62
- Hodson EM, Shaw PF, Evans RA, Dunstan CR, Hills EE, Wong SYP, Rosenberg AR, Roy RL (1983) Growth retardation and renal osteodystrophy in children with chronic renal failure. J Pediatr 103: 735-740
- Chesney RW, Mehls O, Anast CA, Brown E, Hammerman MR, Portale A, Fallon MD, Mahan J, Alfrey AC (1986) Renal osteodystrophy in children: the role of vitamin D, phosphorus, and parathyroid hormone. Am J Kidney Dis 7: 275–284
- Holliday MA, Kulin HE, Lockwood DH, Rosenfeld RG (1986) The endocrine control of growth in children with chronic renal failure. Am J Kidney Dis 7: 262–267
- Mehls O, Ritz E, Gilli G, Heinrich U (1986) Role of hormonal disturbances in uremic growth failure. Contrib Nephrol 50: 119-129
- 22. Chesney RW (1987) Growth retardation in childhood renal disease: a hormonal or nutritional problem? Am J Nephrol 7: 253-256
- Korsch BM, Negrete VF (1984) Psychosocial adaptation of children with ESRD: factors affecting rehabilitation. In: Fine R, Gruskin A (eds). End stage renal disease in children. Saunders, Philadelphia, pp 553–559
- Fine RN (1990) Growth in children with renal insufficiency. In: Nissenson AR, Fine RN, Gentile DE (eds) Clinical dialysis, 2nd edn. Appleton and Lange, Norwalk, pp 667–676
- Fine RN (1985) Renal transplantation for children the only realistic choice. Kidney Int 28 [Suppl 17]: S15–S17
- 26. Kleinknecht C, Broyer M, Gagnadoux MF, Martihenneberg C, Dartois AM, Kermanach C, Pouliquen M, Degoulet P, Usberti M, Roy MP (1980) Growth in children treated with long-term dialysis. A study of 76 patients. Adv Nephrol 9: 133–164
- Baum M, Powell D, Calvin S, McDaid T, McHenry K, Mar H, Potter D (1982) Continuous ambulatory peritoneal dialysis in children. Comparison with hemodialysis. N Engl J Med 307: 1537-1542
- Stefanidis CJ, Hewitt IK, Balfe JW (1983) Growth in children receiving continuous ambulatory peritoneal dialysis. J Pediatr 102: 681-685
- 29. Fennell RS, Orak JK, Hudson T, Garin EH, Iravani A, Van Deusen WJ, Howard R, Pfaff WW, Walker D, Richard GA (1984) Growth in children with various therapies for end-stage renal disease. Am J Dis Child 138: 28–31

- Southwest Pediatric Nephrology Study Group (1985) Continuous ambulatory and continuous cycling peritoneal dialysis in children. A report of the Southwest Pediatric Nephrology Study Group. Kidney Int 27: 558-564
- Perfumo F, Verrina E, Degl'innocenti ML, Piaggio G, Gusmano R (1991) Growth in children undergoing dialysis: comparison between hemodialysis and chronic peritoneal dialysis. Acta Med Auxol 23: 45-51
- 32. Lilien T von, Salusky IB, Boechat I, Ettenger RB, Fine RN (1987) Five years' experience with continuous ambulatory or continuous cycling peritoneal dialysis in children. J Pediatr 111: 513-518
- Warady BA, Kriley M, Lovell H, Farrell SE, Hellerstein S (1988) Growth and development of infants with end-stage renal disease receiving long-term peritoneal dialysis. J Pediatr 112: 714–719
- Alexander ST, Lindblad AS, Nolph KD, Novak JW (1989) Pediatric CAPD/CCPD in the United States. In: Steinjh (ed) New concepts and applications. Contemp Issues Nephrol 22: 231-255
- Greulich WW, Pyle SI (1959) Radiographic atlas of skeletal development of the hand and wrist, 2nd edn. Stanford University Press, Stanford
- Potter DE, Broyer M, Chantler C, Gruskin A, Holliday MA, Roche A, Schärer K, Thissen D (1978) Measurement of growth in children with renal disease. Kidney Int 14: 378–382
- Hamill PVV, Drizd TA, Johnson CL, Reed RB, Roche AF, Moore WM (1979) Physical growth: National Center for Health Statistics percentiles. Am J Clin Nutr 32: 607–629
- Baumgartner RN, Roche AF, Himes JH (1986) Incremental growth tables: supplementary to previously published charts. Am J Clin Nutr 43: 711-722
- National Research Council, Food and Nutrition Board (1980) Recommended dietary allowances, 9th edn. National Academy of Sciences, Washington, D.C.
- Morgenstern B, Pyle WK, Gruskin A, Baluarte HJ, Perlman S, Polinsky M, Kaiser B (1984) Transport characteristics of the pediatric peritoneal membrane. Kidney Int 25: 259
- Diemen-Steenvoorde R van, Donckerwolcke RA, Brackel H, Wolff ED, Wong MCJW de (1987) Growth and sexual maturation in children after kidney transplantation. J Pediatr 110: 351–356
- 42. Fine RN (1987) Editor's column. Growth after renal transplantation in children. J Pediatr 110: 414-416
- 43. Ettenger RB, Blifeld C, Prince H, Gradus DBE, Cho S, Sekiya N, Salusky IB, Fine RN (1987) The pediatric nephrologist's dilemma: growth after renal transplantation and its interaction with age as a possible immunologic variable. J Pediatr 111: 1022-1025
- 44. Tejani A, Butt KMH, Rajpoot D, Gonzalez R, Buyan N, Pomrantz A, Sharma R (1989) Strategies for optimizing growth in children with kidney transplants. Transplantation 47: 229–233
- 45. Law CM (1987) The disability of short stature. Arch Dis Child 62: 855-859
- 46. Tonshoff B, Mehls O, Heinrich U, Blum WF, Ranke MB, Schauer A (1990) Growth-stimulating effects of recombinant human growth hormone in children with end-stage renal disease. J Pediatr 116: 561-566
- Cheeck DB, Graystone JE (1978) Insulin and growth hormone: regulators of growth with particular reference to muscle. Kidney Int 14: 317-322
- Nissensan AR, Fine RN, Gentile DE (1990) Clinical dialysis, 2nd edn. Appleton and Lange, Norwalk, pp 118–146, 319–329
- Parker TF, Laird NM, Lowrie EG (1983) Comparison of the study groups in the National Cooperative Dialysis Study and a description of morbidity, mortality, and patient withdrawal. Kidney Int 23 [Suppl 13]: S42-S49