

A DESCRIPTION OF THE GROWTH OF YOUNG CHILDREN  
WITH CHRONIC RENAL FAILURE

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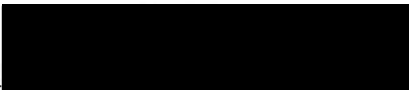
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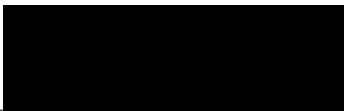
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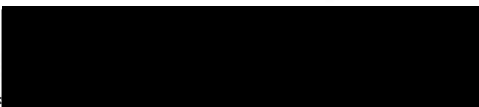
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## CHAPTER I

### INTRODUCTION

This is a retrospective study concerned with describing and documenting growth in children under four years of age who have chronic renal failure. Comparing these chronically ill children with essentially normal children is a difficult task. Attention must be given not only to the ill child's pattern of growth, but also to his change in proportions. (Smith, 1977). These children often fall below the average expected weight and height ranges for their age. (Fomon, 1974). However, for the purpose of this study, growth will be defined only as the rate of change in length.

Chronic renal failure in infants has a prognosis that is difficult to predict. Ideally, it can be said that conservative therapy plus perhaps dialysis or kidney transplantation should lengthen a child's life span. Unfortunately, current treatments involve complications themselves and often contribute additional problems for the child. Among such complications are drug reactions, infection and hypertension.

The nurse, as a member of the team of specialists required to manage children in renal failure, can provide regular and consistent services to the patient and his family. Such contact will provide many opportunities for influencing a family's understanding of the disease process and their willingness to adapt to recommended treatments such as the therapeutic diet and medications. The emphasis of the health team is to promote optimal kidney function and growth while considering the family compromises that will be necessary.

Growth retardation is a consistent finding associated with children who have renal disease. However, the magnitude of the retardation has not been documented. Parents frequently are concerned about the children's size, but tend to reluctantly accept it without question. The children, particularly as they grow older, also become concerned and may become distressed by peer's remarks about their comparatively small size. Documentation of the growth changes in these children would assist the nurse in helping the family and child to understand and cope with the problem.

These issues were faced by this investigator in six years of work with young children in chronic renal failure and served as the impetus for this study. In addition, in an on-going review of the literature, it was noted that there was a limited amount of information related to growth problems in this younger population.

#### Review of the Literature

There are a number of considerations relevant to growth retardation that were reviewed in the literature. These considerations include physiological events, nutritional factors, genetic issues, endocrine factors, and environmental factors. These considerations will be discussed in relation to normal growth patterns and how they are affected during renal failure. Some of the significant complications of renal failure, which include metabolic acidosis, anemia and renal osteodystrophy, also affect growth. The effect of these complications on growth will also be discussed.



### Physiological Events

Normal physiological events alone may influence growth in children. At birth, eighty percent of a baby's birth weight is water. There are obligatory losses through the skin, lungs and urine that are particularly increased with illness. The thirst mechanism insures replacement when a child is beyond infancy. (James, 1977). But for young children, unable to make their needs known, dehydration may be a factor since complications of ingestion and excretion frequently occur with renal failure. With illness they may be unable or unwilling to take fluids. Most children with renal failure are observed to have frequent periods of anorexia and have difficulty or disinterest in eating. They may be nauseated and regurgitate with a resultant loss of water due to limited compensatory mechanisms from decreased kidney mass, or fewer functioning nephrons due to chronic renal failure. There may be sodium depletion and hypovolemia leading to further impairment of existing renal function. (Chantler and Holliday, 1973). Infants and small children with renal failure need to be observed carefully and frequently for their state of hydration because a further decrease in glomerular filtration would compromise an already insulted kidney. (Lewy and Hurley, 1976). Poor renal function in infancy when the growth velocity is the greatest may be a most important factor in growth retardation. (Stickler and Bergen, 1973).

### Nutritional Factors

This leads to a discussion of children's metabolic processes. These are directed toward energy and growth. According to Timiras (1970), dietary factors regulate growth at all stages of development. To satisfy

these growth needs, nutritional requirements undergo constant change. A guide for adequate nutrition, the Recommended Daily Allowance (RDA) has been established by nutritionists. These allowances are recognized as being above a minimum level, but this margin may be lower than that needed in pathological states. (Fomon, Thomas, Filer, Anderson and Bergmann, 1973). Basically, most of these diets recommend high quality protein such as is found in milk, eggs and meat, plus fruits, vegetables and cereals. (Nelson and Vaughn, 1969). Minimum protein allowances have not been established for children although the World Health Organization in 1965 suggested 1.8 gm/kg of body weight per day for infants during the first three months of life. Children in renal failure have frequently been restricted in protein to 0.5 gm/kg of body weight per day in an effort to decrease the urea and solute load for failing kidneys to manage. This decreased amount of protein and thus, also calories, may lead to malnutrition and severe negative nitrogen balance if the diet is extended over several months. (Villiee, 1975).

#### Genetic Factors

Changes after birth may be determined by genetic background as reflected by mid-parental size. A full term infant will double his birth weight by five months of age and triple it by one year of age. By six months of age, boys will grow 17 cm, girls 16 cm, and by one year they will each increase an additional 8 cm. (Frasier, 1979). Some children may be noted to move from one centile to another as serial heights are reported over the first several months. Normal babies who are large at birth, but have smaller parental size as their genetic background, may

decelerate to a lower percentile over the next eight to nineteen months. Small babies may accelerate to a higher percentile in accordance with their own genetic influence. This change may be termed "catch-up" growth as a child moves to a higher centile. (Smith, 1977). Though linear growth is one of the most heritable traits, this cannot be predicted in children with chronic renal failure since growth outcomes are so influenced by the severity of the disease and the child's age at its onset. There is also a wide variation in osseous maturation rates because of genetic make-up and parental history may be important in interpreting the child's ultimate growth. (Smith, 1977).

#### Endocrine Factors

Many hormones are thought to influence growth. Some are important in the transport of chemicals needed by certain cells. Energy is provided by others as the body forms proteins and other nutrients necessary for its maintenance and growth. Testosterone may increase numbers of muscle and bone cells, and estrogens speed up osseous maturation, but neither truly affects the ultimate height of an individual. (Smith, 1977). Thyroid hormone is necessary for energy metabolism but excess amounts do not necessarily cause excess growth. Thyroid, glucocorticoids and growth hormone have been found to be normal in children with renal failure. The effect of insulin on growth activities is not clear. Theories that insulin may regulate preliminary products that lead to production of the somatomedins (formerly referred to as sulfation factors) have been proposed. (Chuimello and Laron, 1977). The contribution of this type of hormonal activity to growth needs further study. Somato-

medins are thought to speed up growth of cartilage without increasing bony maturation. The kidney along with the liver is said to be one of the main producers of these somatomedins. Levels of somatomedins were found to be reduced in children with chronic renal failure and found to be returned to normal following kidney transplantation. (Saenger, 1973). Somatomedins were measured in thirteen children, ages five to seventeen, whose chronic renal failure was documented by creatinine clearances between 5 and 31 ml/min/1.73 m<sup>2</sup>. The levels of somatomedins were below the mean for bone ages and decreased proportionately with decreased clearances. (Schwalbe, Betts, Rayner, Rudd, 1977). The authors also reported that somatomedins are normally at lower levels in early childhood. No studies were found that included children below four years of age. Two studies showed that normal concentrations of growth hormone were found present in an unknown number of children with uremia (Orskow and Christensen, 1971) and in nine children with chronic renal failure. (Piel and Roof, 1975). Frasier (1979) has suggested that there may be defective receptor mechanisms or abnormal synthesis of these products causing the growth failure.

#### The Environment.

A discussion of growth in children with chronic illness must necessarily include their environment. Environment can refer to the house, town and people immediately within a child's scope. This means that the family is the child's major environmental support group. Some families will experience severe difficulties in coping with a chronic illness. Others will adapt and still others will grow with the experience.

(Battle, 1975). The parent's perception of the disease is dependent on their comprehension, personality and sometimes their socioeconomic status. Growing children thrive in an atmosphere of loving care, understanding, patience, protection and comradeship. (Getchell and Howard, 1979). Even with the best of intentions and care, a sick child may function far below that of a normal child in both physical and cognitive areas. Failure to thrive may result from both organic and psychosocial factors, and the psychosocial factors may aggravate the organic ones. (Ross Laboratories). Continued family turmoil and emotional deprivation is known to cause poor growth. (Ambuel, 1963). Studies have shown that the attitudes of the parents, particularly the mother, are crucial in promoting optimal growth and development. (Pless and Pinkerton, 1975). However, there are few researchers who have studied the impact of chronic illness on entire families, so other data is not available.

### Economics

Though it is not a primary consideration, the economic status of a family does affect a child's growth and wellbeing. Family finances may determine a family's access to health services. Cost may be a concern in selecting the kinds of food a family buys and this may be important in implementing dietary therapy. There may be frequent trips to the hospital or clinic for on-going therapeutic measures. Both parents may work and a third person may need to be involved as caretaker. Social Security amendments were passed in 1972 to help meet the high cost of chronic renal failure. However, this measure covers only two treatment modalities; hemodialysis after the first three months, and kidney

transplantation for the first year. Medications are not covered. The triad of economic-social-emotional deprivation may be very difficult to evaluate in terms of its effect on growth. (Ambuel, 1969).

### Complications of Renal Failure

Thus far, essentially normal factors influencing growth have been discussed, with some reference being made to the deviations occurring with chronic renal failure. Some of the metabolic complications of renal insufficiency are also of interest since they will influence growth. These complications include: osteodystrophy, metabolic acidosis and anemia.

### Osteodystrophy

The combination of disturbed calcium, phosphorous and biochemical mechanisms may be an important cause of growth failure in children. (Stickler, 1975). Osteodystrophy is seen in about 45% of children with severe renal failure. (Tsang, Noguchi and Steichen, 1979). Several processes seem to contribute to this condition. With the increased retention of phosphorous due to decreased renal function and mass, there is hyperphosphatemia. This state increases the secretion of the parathyroid gland, thus influencing calcium reabsorption. This will result in demineralization of the bone and possible rickets and fractures. (Tsang, et al., 1979). Vitamin D and calcium carbonate have been given in large amounts in an attempt to reverse this disorder. (Kaplan and Drummond, 1975). This is a successful maneuver until kidney mass is further depleted. The experimental use of different forms of Vitamin D continues to be investigated. (Personal communication, Talwalkar, 1978).

### Metabolic Acidosis

Metabolic acidosis has been noted to cause growth retardation. (West and Smith, 1956). It may result from an inability of the child in chronic renal failure to excrete the acids produced from the metabolism of dietary protein and other body substances. (Lewy and Hurley, 1976). In children with renal tubular acidosis and chronic loss of bicarbonate, correction of the acidosis with bicarbonate may result in an increase in growth. (Nash, Torrado, Grierfer, Spitzer and Aldeman, 1972). However, in chronic renal failure, even with correction of the acidosis with bicarbonate, normal growth did not occur. Thus, perhaps acidosis may be of limited importance in the growth retardation seen in chronic renal failure. (Stickler and Bergen, 1973). This may be because many children with uremia tolerate lower levels of serum bicarbonate, between 15-20 mEq/liter, without obvious symptoms.

### Anemia

The effects of uremic toxins on the hematological system are well documented. (Aronson, 1975; James, 1976). Anemia is almost always present in chronic renal disease due to an apparent suppression of the secretion of erythropoietin, the hormone produced by the kidney to stimulate red blood cell production. In addition, there may be platelet dysfunction due to uremia. There is also a decreased rate of red cell production and shortened red cell life-span. (Lewy and Hurley, 1976). The anemia present is most often normochromic and normocytic. There may also be iron and folic acid deficiencies with the diet restrictions. The extra-renal erythropoietin produced does not seem sufficient to correct

the anemia present. Blood transfusions give only transient relief and are kept at a minimum with patients who may be transplant recipients to avoid sensitization with leukocytes or antigens that might destroy a kidney graft. (Lewy and Hurley, 1976). A study in 1969 concerning growth in 28 children with Thalassemia Major whose hemoglobins ranged from 5.6 to 6.2 g./100 ml (with normal being 14.5 g./100 ml), showed no improvement in growth even with an aggressive depression of hemoglobin. (Brook, Thompson, Marshall and Whitehouse, 1969). They had previously received transfusions when hemoglobins were at high levels, 8 to 9 g./100 ml and marked depression of velocity of growth was shown.

#### Effects of Treatment on Growth

Regardless of its etiology, chronic renal failure leads to gradual deterioration of the ability of the kidneys to adapt and maintain homeostasis. When creatinine clearances fall below 30 ml/min/1.73 m<sup>2</sup>, three treatment modalities are available to deal with the irreversible kidney failure. These modalities are kidney transplantation, dialysis and conservative medical management.

Transplantation. There are only a few contraindications for transplantation in children. The chief ones would be histo-incompatibility, severe mental retardation, or the possibility of recurrence of the original disease in the allograft (transplanted kidney). However, because of the physiologic demands of the kidney (frequently donated by an adult parent), altered renal blood flow and cardiac output, long-term success is more likely in children of at least 10 kg in weight. Even if the transplant is successful, optimum growth is seldom achieved. The reasons for this



fact are not known. (James, 1976; Fine, 1975). Children with a bone age of less than twelve years have a greater potential for growth, but rarely "catch-up" to peers. The corticosteroid therapy necessary to prevent rejection may be at fault, but the greatest growth velocity is usually achieved during the first year following transplantation when dosages of steroids are at their highest. (Talwalkar, Harner, Musgrave, Lawson and Campbell, 1975).

Thirty-one children received kidney transplants through 1975 at the University of Oregon Health Sciences Center. (Talwalkar, et al., 1975). Fifteen children were less than fourteen years of age at the time of transplantation. Five were less than four years of age. Of these five, one has a functioning allograft following two unsuccessful cadaver transplants. He continues to do well at age nine. Two children, one eight months and one eleven months of age were transplanted but rejected the kidneys, and are included in this study. One child succumbed to her original disease, bilateral Wilm's tumor, and one child of less than a year died from the complications of renal failure and surgery. The overall survival rate of patients was 84% with 72% having functioning grafts one year post transplant. This is similar to other centers. However, linear height velocity improvement has rarely been achieved in these transplant recipients.

The University of Minnesota reported twenty-one children transplanted between 1970 and 1976. Transplantation of a kidney from a living related donor was fairly successful between ages one and four. Transplants in infants of less than one year of age were not successful. (Hodson, Narjarian, Kjellstrand, Simmons and Mauer, 1978). Growth to normal

centiles for age was shown in only one child who was four years of age at the time of his first transplant.

Thirty-five children involved in kidney transplantation were matched by age and compared to other children either chronically ill with cystic fibrosis or reportedly normal. (Korsch, Fine, Grushkin and Negret, 1973). With the kidney transplantation group, who were one to five years post transplantation, there was a high frequency of maladjustment, anxiety, and low ego as measured by the California Test of Personality, Sarason's General Anxiety Scale, and the Piers Harris Self Esteem Scale, plus the Draw a Person tests. There was noted a good potential for rehabilitation following successful transplantation, but many children had psychological problems. These studies mentioned the need for much supportive counseling if the family was to survive the ordeal of chronic renal disease.

Renal transplantation is widely accepted for use in the management of renal failure in children. It appears that the age at the time of transplantation is important to growth potential. The problem becomes one of maintaining these small children in optimum condition for transplantation. (Crawford, 1971).

Hemodialysis. The second form of treatment to be discussed is dialysis. Children make up less than 10% of patients who are involved in a chronic dialysis program. Indications for dialysis may be uncontrolled acidosis, pericarditis, hyperkalemia, hypertension, edema, or other uncontrolled biochemical problems. (Lewy and Hurley, 1976). Hemodialysis in children under three years of age is reported but not commonly performed except in extreme cases. It is possible to dialyze infants under

10 kg by using small dialyzers with shortened intravenous blood lines. Access to the vascular system is always a problem because of the small size of the peripheral vessels. Children also have more vascular spasms and clotting problems than adults. (Firlit, 1974). Hemodialysis was used in treating 150 children in Minnesota using both external cannulae and arteriovenous fistulas as the access routes. (Mauer and Lynch, 1976). Methods were described with children of greater and lesser than 20 kg weights. Complications described included those similar to adults; e.g. small vessel size, coagulation difficulties, infections and disequilibrium syndrome. Physical reactions, length of time on dialysis and growth velocities were not tabulated. The survival rate of young children for either dialysis or kidney transplantation is improving. However, complications of arteriosclerosis may ultimately affect this population as well as the adult one. (Scribner, 1974). Some authorities recommend that a long-term plan be made for transplantation before dialysis is begun. (Mauer, et al., 1973).

Chan and De Luca (1977) report one eleven year old patient who was dialyzed over a three year period with improvement of growth following treatment with a Hydroxivitamine D3. Reports from Broyer (1974) have reported growth changes with eleven children dialyzed between 18 and 24 months. Growth was normal in two, fair in three, poor in four and plateaued in two. Specific ages were not correlated with these growth patterns. A review of 861 children in Europe over 10 years of age showed that they appeared to have better survival rates on dialysis than younger age groups. (Scharer, et al., 1974). Rehabilitation of children on home dialysis was better (91% survival) than with those in

hospital situations (70%). In these children, the growth velocity was below the third percentile and noted to fall even lower after the first year of dialysis. (Scharer, et al, 1974).

Peritoneal dialysis. Peritoneal dialysis is another method of treatment for acute and chronic renal failure in infants, children and adults. The peritoneal surface area per unit of body weight in infants is much larger than in adults and this makes a very efficient semi-permeable membrane. (Baliah, 1976). Permanent indwelling catheters are utilized in individuals needing chronic peritoneal dialysis, daily or weekly, to preserve homeostasis. One child (CB) age five, has been dialyzed at the University of Oregon Health Sciences Center for 18 months. Growth, however, has been minimal, which is consistent with data reported in the literature.

Chronic ambulatory peritoneal dialysis (CAPD) has been utilized in some adult centers with success. (Popovitch, Moncreif, Nolph, Ghods, Twardowski and Pyle, 1978). One child at the University of Oregon Health Sciences Center, age nine months, not included in this study, recently began a CAPD program. No data is available on growth as yet, as related to peritoneal dialysis; but because of his renal failure he has had no documented linear growth since July, 1978.

Conservative therapy. The third treatment to be discussed is conservative therapy. In renal failure, this is aimed at lowering serum nitrogen levels by using a restricted protein and increased caloric intake. (Chan, 1973). The children are also given the least amount of acid and solute load possible. This diet will decrease the workload of the failing kidneys in handling phosphates, organic acids, sulfates and salts. There are fewer waste products from the metabolism of this diet for the

kidneys to excrete. However, with this restriction of protein, malnutrition can result. (Kopple and Swendseid, 1977). There can be a loss of lean body mass, with the breakdown of endogenous protein, as the body demands protein for continued survival. (Chan, 1973).

There are very few studies documented in the literature concerning the nutritional needs of very young children during the stressful times of chronic renal failure, kidney transplantation, and dialysis. Providing a greater amount of calories was suggested by Simmons in 1971 as a method of promoting positive nitrogen balance. He pointed out that even with severe protein restriction, children can grow if they have sufficient calories. He defined sufficient calories as being at least 67% of the RDS for that age group. Carbohydrates taken concurrently with protein may prevent the catabolism of protein and amino acids. Lewy in 1975 described the process of restricting dietary products in renal failure as contributing to "undernutrition" with the suggestion that the individual uses the ingested calories for energy before he uses them for growth.

Exact requirements of amino acids for growth and maintenance are not known. Essential amino acids must be supplied by diet whereas non-essential amino acids may be synthesized by the organism. L-forms are hydrolysis products of protein. Studies by Holt and Snyderman in 1961 listed the probable basic requirements of essential and semi-essential amino acids, based on what was found in mother's breast milk. These requirements have been utilized in baby formulas in dietary management since that time. (Holt and Snyderman, 1961). Fomon suggested in 1964 that levels of methionine might be lowered. (Fomon, Owen and Thomas, 1964). He advocated a requirement of amino acids that would be much

greater for younger than for older subjects. He felt the reason for this was that the protein requirement was greater for the synthesis of new tissue (that is, for growth) than for maintenance (that is, for repair and replacement). He also mentioned that requirements of amino acids may be different when given as a mixture of amino acids than as a whole protein (that is, as meat, milk or cheese).

The possibility of reutilization of a patient's own blood urea nitrogen (BUN), which is greatly elevated in chronic renal failure, has been suggested by several nutritionists. In studies by Richards (1972), two-thirds of the subjects studied who had uremia and were given keto acids, improved their nitrogen balance.

The goal of inducing the synthesis of some essential and non-essential amino acids could be accomplished by formulating a diet with practically no dietary protein. With supplementary amino acids, the uremic patient might reutilize his own BUN in protein synthesis. (Richards, Metcalfe-Gibson, Ward, Wrong and Houghton, 1977). Fomon was able to achieve nitrogen balance in a patient who received a restricted protein diet and essential amino acids. (Fomon, et al., 1973). Bergstrom treated forty patients with essential amino acids, both intravenously and orally. (Bergstrom, Furst, Josephson and Noree, 1972; Noree and Bergstrom, 1975; Bergstrom, Furst and Noree, 1975). These patients had poor renal function as indicated by creatinine clearance (Ccr) of less than 5 ml/min/1.73 m<sup>2</sup>. They were given protein poor diets for twelve weeks. They then received this same diet plus intravenous amino acids for two weeks. This was followed by oral amino acids for two weeks. The patients had an improved sense of well-being and had stable laboratory values. Their need for dialysis was postponed for many months.

Nitrogen balance was measured by Giordano in both healthy and uremic men in order to show the use of urea nitrogen if dietary nitrogen protein was restricted. It was shown that a nitrogen-free diet would allow reutilization of the patient's own retained urea nitrogen if amino acid compounds were given in appropriate amounts. (Giordano, 1963; Giordano, Phillips, DePascale and DeSanto, 1972; Giordano, Plurio and Esposito, 1975). Reduction of BUN was shown by Schloerb who followed six uremic patients. These were given a reduced protein diet with adequate calories and oral amino acids. (Schloerb, 1966). Thirteen subjects in renal failure were described by Walser (June, 1975). These subjects were given keto acid (amino acids) therapy with a resultant positive nitrogen balance.

Richards also points out (Richards, 1972; Richards, 1975; Richards, Brown, Houghton and Wrong, 1975; Richards, et al., 1977) that inorganic nitrogen compounds have been used to supplement cattle diets for fifty years and should be considered in man. The author mentions that the therapeutic potential resulting from the reduction of BUN alone is of little benefit, but the reduction of the toxic products of uremia and the achievement of nitrogen balance may be of considerable advantage.

Most of the studies reviewed have been performed with adults or animals. Aronson (1975) treated a five year old child who was in chronic renal failure for twenty-two months with intravenous essential amino acids. She gained weight and height and survived without dialysis for many months. Subsequently, however, she died from the complications of chronic renal failure.

Kopple and Swendseid discussed amino acid supplements with low protein diets for therapy in renal failure as an alternative to dialysis and transplantation. Though the advantages are not as yet completely clear and the requirements not clearly established, they were able to cite several animal studies with growth shown following the ingestion of amino acids. (Kopple and Swendseid, 1977).

Thus far, only the dietary approach to treatment for chronic renal failure has been discussed. Medications may also be necessary to achieve homeostasis. Acidosis, if not corrected with sodium bicarbonate, could possibly be a reason for growth failure. (James, 1975). Calcium carbonate or aluminum hydroxide may be given along with Vitamin D when there is clinical evidence of osteodystrophy which will also cause poor linear growth. (Chan, 1973). The anemia of chronic renal failure is not generally treated directly, that is by transfusion, since most children must be considered as candidates for a kidney transplantation. However, because of the nutritional deficits, most children are given vitamins with folic acid to try to correct possible inadequacies of diet. (Lewy, et al. 1976). The symptoms of uremia are treated as they appear. Hypertension is not a common feature with children, but is seen in polycystic disease and then appropriate antihypertensives are used. There may also be pruritis and hyperkalemia. Peripheral neuropathies, however, are not common in these small children.

Non-compliance is a well-known problem with medications. The mother's view of the severity of the illness may determine whether or not she gives the child's medicine on a regular basis. Medication errors tend to occur more frequently with very young and very old



patients. (Blackwell, 1973).

#### Summary

In children with chronic renal failure, growth and development may be compromised by protein and calorie reduction because of the therapy recommended to decrease the workload of the failing kidneys. Growth is also complicated by illness and renal insufficiency with its accompanying acidosis, anemia, impaired formation of metabolites of Vitamin D, disturbances of calcium and phosphorous, osteodystrophy and resistance to growth hormone. (Rubin and Barratt, 1975).

#### Statement of the Problem

Studies to date (Barratt, 1971; Betts and White, 1976), and the clinical records at the University of Oregon Health Sciences Center indicate that children with renal insufficiency are unable to manifest normal growth patterns. However, the linear growth of children zero to four years in chronic renal failure has not been documented. The problem then was to determine the linear growth of children ranging in age from birth to four years who were in chronic renal failure.

#### Purpose

The purpose of this study was to measure and describe the linear growth of children ranging in age from birth to four years of age who were in chronic renal failure.

## CHAPTER II

### METHODOLOGY

The sample in this study consisted of thirteen children under the age of four years who were in chronic renal failure as indicated by a creatinine clearance of less than 30 ml/min/1.73 m<sup>2</sup>. A retrospective chart review at the University of Oregon Health Sciences Center indicated that this number included all known patients between the years of 1974 and 1978. Excluded from the study were two families who elected to take their children home following diagnosis rather than participate in the opportunity for conservative management by hospital staff.

Creatinine clearance as a measure of chronic renal failure was established by use of the formula developed by Schwartz (1976) for estimating the glomerular filtration rate. This formula was based on data from 186 children, ages 1 to 12 years. The formula specifies that body length in centimeters be multiplied by a factor of 0.55 and divided by the patient's plasma creatinine obtained in mgm%. The inability to collect a standard 24 hour urine specimen from small children led to the development and use of this formula. The formula was used in this study to give uniformity to the data to be presented.

Initially, laboratory data were collected on all the subjects to define the magnitude of their renal failure. Conservative monitoring included following serum electrolytes, BUN, creatinine, bicarbonate, calcium and phosphorous levels. This information was useful in understanding the disease process, but was not correlated with the growth measurements obtained from the children. Though it will be included on

individual growth charts.

A further important aspect of the conservative management of this study population was dietary therapy individualized according to the needs of each child. Caloric requirements were based on the revised Recommended Daily Allowances (RDA) for children by weight and age as published by the National Research Council in 1974. These requirements for well children are aimed at maintaining an average of 100-120 cal/kg for the youngest children. For the children in this study who had chronic renal failure, the intent was to maintain calories at a rate of 150 cal/kg. Special dietary adjustments included obtaining 70% of the protein allowance from low protein formula (SMA-20) and 30% from other foods. Supplements were provided to correct caloric intake to the desired 150 cal/kg/body weight per day. Dietary monitoring represented an essential aspect of management of these children, but was not correlated with growth measurements.

This brief discussion of management is included to advise the reader of the therapeutic regimen available to the thirteen children during the period of this study. The formula for protein requirements by age is included in Appendix A.

Growth measurements were obtained by following a recommended procedure for obtaining linear measurements in young children. (Smith, 1977). This consisted of placing the child in a recumbent position with the soles of the feet flexed upright and the head resting firmly against a flat, firm object such as a book or wall. The measurement was recorded in centimeters on an individual growth chart (Ross, 1976) permitting comparison with an age standardized pediatric population. Such charts

also permitted collection of serial information on the same child and served as a graphic illustration of growth patterns. No attempt was made to correlate the data with the bone age of the child. In addition to this measurement, growth velocities were plotted with comparative tables and charts. Growth charts appear in Appendix B, page 54. Changes in linear measurements (height) are shown as velocity of growth. The weight for height index (WHI) as proposed by West and Smith, 1956, was included to illustrate the growth patterns in relation to nutritional intake. (Stickler, 1976). This measurement is obtained by dividing the actual weight by ideal weight for a 50th percentile of height by age.

## CHAPTER III

### RESULTS AND DISCUSSION

#### Introduction

This investigation was made to document and describe the linear growth of thirteen children, ages three days to four years, in chronic renal failure. Measurements and other observations were made by this investigator during hospital stays and out-patient visits that the children made to the University of Oregon Health Sciences Center during the period of January, 1974 to June, 1978. Numerically, this sample size was small, but represented all of the complicated pediatric nephrology patients identified through a state-wide referral system and sent to this institution. These patients came from all over the state of Oregon and southwest Washington. The only known patients not included in this study were two children whose families declined medical management.

#### Characteristics of the Sample

The sample consisted of nine boys and four girls. Information describing their characteristics on entry to this study are summarized in Table I. The different entry ages for each child should be noted. Again, because of the small number of patients involved, it seemed important to include all children as they were identified by the referral system so that the amount of data on linear growth would be as inclusive as possible.

The age on entry ranged from three days to 27 months. Seven of the boys presented with some type of uropathy which needed urgent attention to relieve obstructive pressures. These subjects (1, 2, 4, 5, 6, 7, 9 - Table I) entered the study shortly after birth. The predominance of

TABLE I. Pediatric Chronic Renal Failure, University of Oregon Health Sciences Center  
January, 1974 to June, 1978

Boys	Diagnosis, Anomalies	Age on Entry	BUN mgm/%	Laboratory Values		Weight Height Index	Place in Family	Occupations	Height Ft. In.
				Cr mgm/%	Ccr/min/1.73m <sup>2</sup>				
1	Renal dysplasia, post urethral valves	8 months	32	2.3	15 ml	0.77	2nd child (Sister 3)	Fa: Mill worker Mo: Housewife	5 10 5 4
2	Renal dysplasia, obstructive uropathy, cutaneous ureterostomies, anoxia at birth	14 days	35	6.4	4.9 ml	0.74	1st born	Fa: Student, chem tech Mo: Medical Tech.	6 0 5 6
3	Polycystic kidneys, hypertension	30 days	16	1.1	30 ml	1.07	1st born (3rd child for father)	Fa: Ambulance driver Mo: Mail carrier	5 10 5 10
4	Renal dysplasia, obstructive uropathy, colostomy	8 months	16	2.5	12 ml	0.76	1st born	Fa: Laborer Mo: Housewife	5 7 5 0
5	Renal dysplasia, imperforate anus, colostomy, skeletal anomalies	4 months	53	1.7	13 ml	0.84	1st born	Fa: Student, Laborer Mo: Housewife	5 4 5 0
6	Renal dysplasia, obstructive uropathy, cutaneous ureterostomies	7 days	61	4.7	7 ml	0.88	1st born	Fa: Salesman Mo: Housewife	5 10 5 2
7	Prune belly syndrome, hydro-nephrosis, urachus, skeletal anomalies	3 days	21	1.4	14 ml	0.96	1st born	Fa: Teacher Mo: Teacher (grad stud)	5 11 5 9
8	Renal dysplasia	27 months	38	2.1	12 ml	0.70	3rd child	Fa: Laborer Mo: Housewife	5 3
9	Renal dysplasia, obstructive uropathy, post urethral valves	14 days	101	6.1	10 ml	0.76	1st born	Fa: Teacher Mo: Housewife	5 8 5 1
Girls									
10	Renal dysplasia	7 months	34	1.6	13 ml	0.67	1st born	Fa: Ill Alcoholic Mo: Housewife	5 8 5 0
11	Renal dysplasia, imperforate anus, partial nephrectomy, asymmetry of facies	21 days	30	1.7	15 ml	0.92	1st born	Fa: Not known Mo: Housewife	5 8 5 6
12	Wilm's bilateral, bilateral partial nephrectomies	20 months	33	1.0	20 ml	0.77	2nd girl	Fa: Mill worker Mo: Housewife	5 0
13	Renal dysplasia	9 months	63	3.1	10 ml	0.68	1st born	Fa: High school student Mo: High school student - Mill worker	5 10 5 2

obstructive pathology in boys was consistent with the data reported in the literature. (Stansfeld, 1966; Royer, et al., 1974). Only one girl (subject 11) had obstructive uropathy. Her diagnosis also included imperforate anus, a multicystic kidney, and other genito-urinary anomalies. It is interesting to note that she was the only child who had asymmetry of the facies, other than one child with a familial asymmetry (subject 2). She also had ears that were slightly enlarged, with poorly formed helices. Though these may be non-specific anomalies, they have come to be associated with renal disease. (James, 1976; Mead Johnson, 1976). Conditions present in the other children included one boy with polycystic kidneys and hypertension, a girl with Wilm's tumor and partial nephrectomies, and three children (subjects 8, 10, 13) whose conditions were termed simply renal dysplasia.

Ten of the children were the first-born in their family. This order of birth has been identified as a stress in itself and might have a potential influence on growth. (Scipien, et al., 1979). Smith (1977) notes that the first born is frequently smaller in the neonatal period than subsequent offspring but, conversely, tends to have a more rapid growth rate in the first few months. Of the other three subjects, one boy had an older sister, one boy was the third child, and one girl was the second child in the family. One child, though first born to the mother, was the product of the second marriage for the father, whose first two children also lived with this family.

Seven of the 13 children were the only children born to the family during the period of this investigation. Subsequently, parents of subjects 2 and 6 did plan to enlarge their families and the mother of

subject 2 delivered two normal boys. The circumstances of separation and divorce with the other families influenced family size. Subjects 4, 5 and 13 were divorced by the third year of the study, but were separated during the time of the study. It is not known whether the catastrophic illness of the subject was an additional factor in limiting family size.

The degree of renal failure in these children is demonstrated by the serum and urine laboratory values in Table I. The blood urea nitrogen (BUN) values ranged from 16 mgm% to 101 mgm%. Serum creatinine values ranged from 1.1 mgm% to 6.4 mgm%. The normal laboratory values in this institution are BUN 6-23 mgm% and creatinine 0.7-1.4 mgm% for adults. According to Nelson (1969) BUN ranges 5-15 mg/dl for infants and 10-20 mg/dl for children and serum creatinine ranges 0.3-1.1 mg/dl. Creatinine clearances of urine ranged from 7 ml/min/1.73 m<sup>2</sup> to 30 ml/min/1.73 m<sup>2</sup>. Normal urine creatinine clearances for children change with age, but generally increase to about 70 ml/min/1.73 m<sup>2</sup> by two months of age and range to 127 ml/min/1.73 m<sup>2</sup> by two years of age. (Ross Laboratories, 1978). Ten of the children had clearances of 15 ml/min/1.73 m<sup>2</sup> or less. The significance of these values can be noted by comparing them with a study of linear growth in 23 children with renal failure. (Betts, et al., 1974). In Betts' study, clearances of 25 ml/min/1.73 m<sup>2</sup> have been associated with linear growth failure. Betts further noted that 52% of the children in his sample whose renal failure dated from infancy were on or below the 3rd percentile in height. It should also be mentioned that only nine of the 23 children in his sample had clearances of less than 30 ml/min/1.73 m<sup>2</sup>.



In this investigation, seven of the 10 children with clearances below 15 ml/min/1.73 m<sup>2</sup> were boys. All of these boys were on or below the 3rd percentile of linear growth on entry to the study. An eighth boy, subject 3, presented an entirely different entry picture. His clearance was 27 ml/min/1.73 m<sup>2</sup>. His birth weight placed him in the 95th percentile, with a height percentile which remained between the 25th and 50th percentile until his second year. (Appendix B, Figure 3). The ninth boy, subject 8, entered this investigation at 27 months of age and there is limited data to report. (Appendix B, Figure 8). Creatinine clearance on admission to this study was 12 ml/min/1.73 m<sup>2</sup>. Initial growth measurements placed him below the 3rd percentile in weight and at the 10th percentile in length. A second measurement plotted at a nine month interval illustrated plateauing in length, but minimal increments in weight gain following his own growth curve in that dimension.

Of the four girls (Appendix B, Figures 10-13), two were far below the 3rd percentile in both height and weight when they first entered the study. Creatinine clearances were 10 and 13 ml/min/1.73 m<sup>2</sup>. A third girl, (Figure 11) had a creatinine clearance of 15 ml/min/1.73 m<sup>2</sup>, and was at the 3rd percentile on initial observation. The fourth girl, (Figure 12), had a clearance of 20 ml/min/1.73 m<sup>2</sup> and was well within the 50th percentile on entry to the study.

One of the reasons for poor linear growth in children in chronic renal failure is said to be malnutrition. (Barratt, 1971). The Weight for Height Index (WHI) as proposed by West and Smith (1956) is included to illustrate the malnourishment of the children. The Index, which

demonstrates poor caloric intake, has been previously described in the Chapter on methodology, and is considered to be very low if the factor is less than 0.95. Only two of the 13 children in this sample were within normal limits. One boy, subject 3, had a WHI of 1.07, and another boy, subject 7, had a WHI of 0.96. The other eleven children had WHI's between 0.67 and 0.92. Growth charts for all 13 children (Appendix B, Figure 1-13) shows weights far below the 3rd percentile in seven of the boys and in all four of the girls in the study.

More descriptive information about the 13 subjects in this study has been presented in Table I. A discussion of linear growth in children must also include a reference to family composition since inadequate parenting may lead to severe retardation of growth. (Nelson, 1969).

The environment is an important aspect of growth in children. (Pless, 1975). There has been no attempt to correlate environmental data, but some brief observations have been included.

All of the children came from Caucasian families. When classified by occupation, the families could be termed middle to lower class socio-economically. Jobs held by either or both parents included laborers, mill workers, salesmen, an ambulance driver, a mail carrier, teachers, and undergraduate and graduate college students. The age of the parents was not documented.

Eight of the families were intact with the parents giving each other strong support. Four children were cared for by single parents (mothers), following separation or divorce which happened shortly after the birth of the affected child. The thirteenth family was on public assistance because of the father's reported illness and inability to

maintain a job.

The heights of the parents were included since stature has been termed as one of the most heritable traits. (Smith, 1977). Smith correlates size at birth to the size of the mother and reports that height at two years can be correlated to mid-parental height. The parental heights of these subjects are listed in Table I.

The characteristics of this sample of children in chronic renal failure have been described in relation to the following: diagnosis, age on entry, degree of renal failure, malnutrition (WHI), and family characteristics.

Most reports concerning children in renal failure have discussed the growth failure from the aspect of the older child with records beginning at age five years or older. No data has been found that documents growth from birth of a group of children in chronic renal failure. Therefore, this data which has been collected over a period of time, will provide valuable information to help answer the questions concerning growth retardation in children in chronic renal failure.

Many factors may have influenced the patterns of linear growth in this group. These include the disease etiologies and related complications of chronic renal failure. Some of the subjects were discussed individually and by sex in relation to these areas. The implications of malnutrition, acidosis, osteodystrophy and anemia were discussed for all subjects. Parenting, as an illustration of a significant environmental support for the children was emphasized throughout the study.

## Results

The main causes of chronic renal failure in this group of young children were renal dysplasia and obstructive uropathy. Regardless of the etiology of the disease, there was severe retardation of growth in all of the subjects. This is shown in the growth charts of the children which compare their growth to that of normal children. (Appendix B, Figures 1-13). Growth retardation in this study can be defined as failure of the child to follow his own growth curve in a smooth upward line according to normal percentiles.

### Boys:

Five of the boys with a common diagnosis of obstructive uropathy and renal failure had remarkably similar patterns of growth as demonstrated by a report of mean growth and a composite of individual growth patterns. (Appendix B, Figure 14). All boys had creatinine clearances below 15 ml/min/1.73 m<sup>2</sup>. These boys were followed for twelve months and some for more than thirty-six months. It can be seen that the first twelve months of life were marked by plateaus and small rises, but there is a continuing progression in linear growth following a 3rd percentile curve. No attempts were made to formally correlate the laboratory data, but its influence on growth may be important. Therefore, it was included on each individual growth chart. BUN, creatinine, and creatinine clearance identified the degree of renal failure at intervals. The effect of these abnormal values on the symptoms of chronic renal failure, i.e., azotemia and anorexia, has not been documented. (Barratt, 1971; James, 1976).

Within this group of five subjects, the boys with elevated BUN and creatinine had about the same pattern of growth as those with lower laboratory values. The subjects included are numbers 1, 2, 4, 6 and 9, and on Table I it can be seen that their BUN's ranged from 16 to 101 mgm% and their serum creatinines from 2.3 to 6.4 mgm%. The average BUN for the subjects at one year of age was 41 mgm% and the average creatinine was 2.8 mgm%. Their weight dropped significantly at about nine months of age and continued far below the 3rd percentile.

Of the other four boys, subject 3, with a diagnosis of polycystic kidneys, grew well until his second year when his linear growth started to decelerate. His weight, however, continued at the 75th percentile. It is of interest to note that his hypertension remained difficult to control and ranged between 140-200 systolic and 80-90 diastolic, which represents values above 95th percentile in blood pressure for his age. (James, 1976). Concurrent symptoms reported by the mother included only irritability and occasional malaise. Medications used in combinations for control of hypertensive symptoms included Hydralazine, Propranolol, Methyldopa and Guanethidine. His creatinine clearance of 27 ml/min/1.73 m<sup>2</sup> at completion of the study was similar to figures recorded for him at one month of age.

Subject 5 (Figure 5), with a diagnosis of multiple genito-urinary anomalies, showed an improvement in linear growth. His overall renal function also improved as noted by referring to laboratory values. The growth chart also illustrated a marked increase in linear growth following surgical repair of his imperforate anus.

The eighth boy (Figure 8), entered the study at the age of 27 months and it can be noted that his linear growth remained static. His weight was below the 3rd percentile but no other data on linear growth was available. The mother commented on his small stature, but reported that he was active and did not appear ill to her. However, the investigator was concerned with the linear growth and noted significant laboratory findings. The subject's BUN was 38 mgm%, creatinine was 2.1 mgm% and his creatinine clearance was 12 ml/min/1.73 m<sup>2</sup>.

The last boy in the study, (Figure 3), with a diagnosis of prune belly syndrome and hydronephrosis, had a clearance of 14 ml/min/1.73 m<sup>2</sup> shortly after birth and this had improved to about 42 ml/min/1.73 m<sup>2</sup> at the age of seven months. His linear growth had advanced to the 50th percentile at that age but further data is unavailable due to inability to follow this subject.

Growth velocity of the boys with obstructive uropathy was included (Figure 16) as another means of illustrating linear growth. Because of the variety of disease conditions, averaging the linear growth of all nine boys could not be considered representative of the true velocity of the group. However, the growth mean of the five boys with the common diagnosis was presented. (Appendix B, Figure 14).

#### Girls:

The smaller number of girls in this sample (four), in relationship to boys (nine) again supports the findings in the literature that more boys are affected with renal disease than girls. This small number does reduce the impact of any attempts at generalizing or averaging. However, since three of the girls were followed for an extended period of time, up to

three years, their linear growth has been compared and the average presented. (Appendix B, Figure 15). Individual growth charts for all four of the girls are also presented. (Appendix B, Figures 10-13). Growth velocity of two girls with similar diagnoses are shown in Figure 15.

Subject 12 was a girl with Wilm's tumor and partial nephrectomies who was identified in Tumor Clinic and included in the study. Only brief contacts were made with the girl and her family. Her linear growth dropped from the 50th percentile during the time of treatment for the Wilm's tumor which included regular periods of chemotherapy. Her weight dropped even further to the 5th percentile. At the end of the study, she had returned to her earlier 50th percentile growth curve. Her clearance at age three was 31 ml/min/1.73 m<sup>2</sup>; BUN was 23 mgm% and creatinine 0.8 mgm%. It is expected that she will probably continue to grow satisfactorily for a few more years when she may face kidney transplantation. (Appendix B, Figure 12).

Subject 11 had a diagnosis of imperforate anus and multiple anomalies, including a multicystic kidney which was removed shortly after birth. Since this surgery she has grown quite well. She followed the 3rd percentile with one period of slowing at about seven months, and then dropped below the 3rd percentile at age two, but has continued a smooth upward curve. (Figure 11). Her weight has continued satisfactorily from birth. Her creatinine clearance has improved from 17 ml/min/1.73 m<sup>2</sup> at three months of age to 37 ml/min/1.73 m<sup>2</sup> at three years of age. Because of the complicated requirements needed to correct the genito-urinary anomalies, no surgery was contemplated at the end of

this study period. She continued to function with a colostomy and urinary diversions. In terms of support offered by the family for this child's special needs, it is of interest to note that the parents separated shortly after birth and remained apart for about one year. They have since reunited.

As an illustration of the difficulties experienced in coping with the complications and special needs of chronic renal failure, the other two girls and their families will be discussed. Both of them had renal dysplasia and failure to thrive. They both entered the system at about nine months of age and have shown consistently severe growth failure in both height and weight as illustrated in their growth charts. (Appendix B, Figures 10 and 13). They were both also small at birth.

The mother of subject 10 has tried to cooperate in the management therapy which was recommended. However, despite the efforts of the mother and hospital personnel to improve caloric intake, little change was noted in the child's growth. No reasons were found for her small size except for small size at birth, and the renal dysplasia which was not identified until she was nine months of age.

The second of the two girls with extremely poor growth, subject 13, was born several weeks prematurely, and weighed less than five pounds at birth. She was maintained in an incubator for two and a half months, and was not expected to live. The mother was a young unmarried woman of seventeen. The mother and father were married when the mother was about four months pregnant, but separated shortly after the child's birth. The mother has been essentially the primary support



for this child since birth.

This child was referred to this institution when she was nine months of age and at that time had a BUN of 63 mgm%, creatinine of 3.1 mgm%, and creatinine clearance of 10 ml/min/1.73 m<sup>2</sup>. She received a kidney transplantation at eleven months of age which was acutely rejected and subsequently removed.

Her overall growth has been very poor, far below the 3rd percentile for both height and weight. Attempts to increase caloric intake with supplementation were not too successful. Her renal function continued to deteriorate until about age three when she was placed on chronic peritoneal dialysis. She experienced frequent episodes of peritonitis as can be expected with this mode of treatment. (Beale, et al., 1976). Growth has continued to be poor though it rose sharply immediately following initiation of the chronic dialysis.

### Acidosis

Acidosis is frequently mentioned in the literature as a possible cause for growth retardation. With all of the 13 children, the first year was marked by frequent short periods of acidosis (serum bicarbonate of less than 17 mEq/l). However, the children did not always need hospitalization. All of the children were given calcium or sodium bicarbonate as buffering agents. No documentation was made of compliance. Anecdotal reports from the mothers note that the children played with more enthusiasm and had better appetites when the serum bicarbonate was normal than when the child was slightly acidotic (serum bicarbonate at 18 mEq/l). No overt symptoms such as malaise or hyperventilation were

reported.

### Malnutrition

Another significant concern in growth failure, malnutrition, was not always obvious in this sample. However, the use of the WHI pointed to poor weight gain in all of the children but two. (Table I). On seven patients followed for more than two years, the WHI continued below normal and ranged from 0.83 to 0.84 (normal WHI is 0.95 and over). All of the 13 children followed a pattern of anorexia and disinterest in food and were termed poor eaters. Efforts were made to maintain calories above RDA for age (to 150 cal/kg) in an attempt to average the amounts necessary for linear growth (100-120 cal/kg). The protein was allowed in increasingly liberal amounts according to the laboratory values. (Appendix A). Observations in the literature concerning difficulties in feeding children with chronic renal failure were supported by the experiences with these 13 subjects. (Stickler, 1976).

### Osteodystrophy

Osteodystrophy is mentioned as another cause of poor growth in children with chronic renal failure. Bone ages were not done consistently on these subjects and the manner of interpretation was variable. However, bone age was reported as being statistically below the mean in all of the children who had a clearance of less than 10 ml/min/1.73 m<sup>2</sup> by the age of twelve months. Serum calcium, urinary calcium, and creatinine excretion were monitored as well as parathormone levels. Vitamin D was given at 500 u/kg when the ratio of calcium and phosphorous excretion was disproportionate. Rickets accompanied by fractures and skeletal imbalances

requiring braces and massive calcium and Vitamin D therapy were seen in two children; a boy, subject 1, and a girl, subject 13. As mentioned previously, this girl had had very poor linear growth. The boy had followed a curve slightly below the 3rd percentile but in a continuing upward line. Both of these children required parathyroidectomy with reimplantation of parathyroid tissue in the arm (University of Oregon Health Sciences Center, 1976). Bone disease was arrested and skeletal abnormalities corrected. However, growth in these children did not change appreciably from what it was prior to surgery.

#### Anemia

Although not believed to be specifically related to linear growth, anemia is a common finding in chronic renal failure and was present in 10 of the patients as indicated by hematocrits between 18% and 25%. This is consistent with findings documented in the literature. (James, 1976). An eleventh child, subject 2, maintained a hematocrit of 31% with clearances of less than 6 ml/min/1.73 m<sup>2</sup>. Supplements of amino acids (Amin Aid) were used from birth to three years with this child. The twelfth child, subject 6, also used this supplementary product. His hematocrit ranged between 26-28%. His clearance was about 15 ml/min/1.73 m<sup>2</sup> during the study period. The thirteenth child, subject 3, the boy with polycystic disease, had maintained a close to normal hematocrit value.

#### Parenting

The attitude of the mother in children with chronic illnesses has been emphasized as supporting growth and development. (Pless, et al., 1975). The mothers of these children were the parent most consistently

seen in the hospitals and clinics. It was the mother who investigated the health problems and determined the need for consultation. The mother also devised ways of managing recommended diets. In general, the mother was an ally in treating the child. Although she was fiercely protective of the child at times, she became adept at assisting in procedures that were painful to the child, once she was convinced of their necessity. This supportive attitude contributed to the promotion of optimal growth.

In this study, the limitations of small numbers and multiple diagnoses make generalizations difficult. Linear growth in this study was in accord with the reports in the literature. Decreased creatinine clearance over time showed a relationship to decreased or static linear growth in children. Reasons for this growth failure cannot be specifically identified. This study does document growth from birth to three years in thirteen children with chronic renal failure as defined by a creatinine clearance of less than  $30 \text{ ml/min/1.73 m}^2$ . This information has not previously appeared in the literature.

## CHAPTER IV

### SUMMARY AND RECOMMENDATIONS

This has been a retrospective study to describe and document growth in children under four years of age with chronic renal failure. The 13 children in the study were followed from 1974 through 1978 at the University of Oregon Health Sciences Center.

Documentation in the literature has indicated that children with renal insufficiency are unable to maintain normal growth patterns. It is of interest to note that such documentation has not included children under the age of 12 months.

With this knowledge as a stimulus, the investigator initiated a comprehensive chart review of all of the children known to be in chronic renal failure at the University of Oregon Health Sciences Center during the study period. All of these children were also known to the investigator because of her nursing involvement in the pediatric nephrology department.

The study was descriptive in design. Factors with relevance to growth were identified in the chart review and described as aspects of the major focus of the study: linear growth.

Growth information obtained from the records was plotted on individual growth grids to permit comparison with normal growth curves of a comparable age group. All of the subjects in the study demonstrated linear growth failure.

Multiple disease entities were found in this sample. However, it was interesting to note that seven of the nine boys were found to have obstructive uropathy as an etiologic factor in their chronic renal

failure. One of the four girls also had obstructive uropathy. The disease entity as identified in this sample did not appear to be relevant to linear growth patterns. However, those subjects with conditions resulting in referral in the first days or weeks of life necessitating early treatment are believed to have benefited in terms of long-term growth.

Laboratory values obtained for each child provided a measure of the severity of renal failure. Of particular relevance was the creatinine clearance which was documented for children in this sample. The range varied between 4 and 56 ml/min/1.73 m<sup>2</sup> in this sample during the study period. Although the clearance values were useful in understanding the extent of the renal failure, no conclusions can be made about its relationship to linear growth.

The Weight-Height Index was used as a measure of caloric intake in recognition of the importance of nutritional factors in overall growth. No significant conclusions can be drawn from this information. However, early case finding and referral with attention to protein and calorie control seemed to result in a more consistent upward growth curve. This was in contrast to children entering into conservative therapy at a later date.

Transplantation in this age group has been unsuccessful. Subject one and subject 13 received transplants which were rejected and removed within a few weeks. Peritoneal dialysis has been used for both acute and chronic renal failure in subjects 10 and 13. Linear growth has not been increased although subject 13 had a sharp rise in her growth curve following the first few weeks of dialysis.

Complications common to chronic renal failure and thus contributing to linear growth failure were discussed. It was possible to document both complications and treatment regimens for this group but no clear cut patterns emerged.

Height as a heritable factor was noted by documenting information about parental stature. The small size of the sample precludes generalizations or specific conclusions.

In recognition of the importance of social and environmental factors including parenting and financial assets to overall growth and development, a description of this information was included as available. Although an empirical judgment can be made and supported by the literature, that children's growth patterns are responsive to support and stresses in the environment, this sample provided illustrations, but again, the size of the sample did not permit a conclusive statement.

This latter area, as well as those described earlier speak to the need for intensive nursing involvement in identifying and meeting the needs of children with chronic renal failure as well as those of the family. A parent-professional partnership is essential in providing long-term support to enhance all aspects of growth.

#### Recommendations for Further Study

As a result of this investigation, the following recommendations are made:

1. Continuation of data collection with this subject group to school age and/or kidney transplantation.

2. A specific study documenting calorie and protein ingestion in relation to growth. Dietary supplements need to be documented as an essential part of such a study.

3. Cooperation with other centers to obtain a larger population of young children for documentation of growth.

4. Specific studies identifying and describing measures used by families to cope with the stress of chronic illness.

5. Investigation of 1 (25)-dihydroxycholecalciferol (continuation of beginning work at UOHSC) in treatment of bone disease of chronic renal failure in children.

6. A specific study correlating clinical data to growth data to determine significant patterns.



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APPENDICES

APPENDIX A

Recommended Daily Dietary Allowances

Protein Requirements by Age and Creatinine Level

FOOD AND NUTRITION BOARD, NATIONAL ACADEMY OF SCIENCES-NATIONAL RESEARCH COUNCIL  
RECOMMENDED DAILY DIETARY ALLOWANCES,<sup>1</sup> Revised 1973

Designed for the maintenance of good nutrition of practically all healthy people in the U.S.A.

Age (years)	Weight (kg)	Height (cm)	Energy (kcal) <sup>2</sup>	Protein (g)	Fat-Soluble Vitamins			Water-Soluble Vitamins							Minerals						
					Vitamin A Activity (RE) <sup>3</sup>	Vitamin D Activity (IU)	Vitamin E Activity (IU)	Ascorbic Acid (mg)	Folic Acid (μg)	Niacin (B <sub>3</sub> ) (mg)	Riboflavin (B <sub>2</sub> ) (mg)	Thiamin (mg)	Vitamin B <sub>6</sub> (mg)	Vitamin B <sub>12</sub> (μg)	Calcium (mg)	Phosphorus (mg)	Iodine (μg)	Iron (mg)	Magnesium (mg)	Zinc (mg)	
Infants	0.0-0.5	6	14	60	24	420*	1,400	4	35	50	5	0.4	0.3	0.3	0.3	360	240	35	10	60	3
	0.5-1.0	9	20	71	28	400	2,000	5	35	50	8	0.6	0.5	0.4	0.3	540	400	45	15	70	5
Children	1-3	13	28	86	34	400	2,000	7	40	100	9	0.8	0.7	0.6	1.0	800	800	60	15	150	10
	4-6	20	44	110	44	500	2,500	9	40	200	12	1.1	0.9	0.9	1.5	800	800	80	10	200	10
	7-10	30	66	135	54	700	3,300	10	40	300	16	1.2	1.2	1.2	2.0	800	800	110	10	250	10
Males	11-14	44	97	158	63	1,000	5,000	12	45	400	18	1.5	1.4	1.6	3.0	1200	1200	130	18	350	15
	15-18	61	134	172	69	1,000	5,000	15	45	400	20	1.8	1.5	1.8	3.0	1200	1200	150	18	400	15
	19-22	67	147	172	69	1,000	5,000	15	45	400	20	1.8	1.5	2.0	3.0	800	800	140	10	350	15
	23-50	70	154	172	69	1,000	5,000	15	45	400	18	1.6	1.4	2.0	3.0	800	800	130	10	350	15
	51+	70	154	172	69	1,000	5,000	15	45	400	16	1.5	1.2	2.0	3.0	800	800	110	10	350	15
Females	11-14	44	97	155	62	800	4,000	10	45	400	16	1.3	1.2	1.6	3.0	1200	1200	115	18	300	15
	15-18	54	119	162	65	800	4,000	11	45	400	14	1.4	1.1	2.0	3.0	1200	1200	115	18	300	15
	19-22	58	128	162	65	800	4,000	12	45	400	14	1.4	1.1	2.0	3.0	800	800	100	18	300	15
	23-50	58	128	162	65	800	4,000	12	45	400	13	1.2	1.0	2.0	3.0	800	800	100	18	300	15
	51+	58	128	162	65	800	4,000	12	45	400	12	1.1	1.0	2.0	3.0	800	800	80	10	300	15
Pregnant			+300	+30	1,000	5,000	400	15	60	800	+2	+0.3	+0.3	2.5	4.0	1200	1200	125	18*	450	20
Lactating			+500	+20	1,200	6,000	400	15	60	600	+4	+0.5	+0.3	2.5	4.0	1200	1200	150	18	450	25

<sup>1</sup> The allowances are intended to provide for individual variations among most normal persons as they live in the United States under usual environmental stresses. Diets should be based on a variety of common foods in order to provide other nutrients for which human requirements have been less well defined. See text for more-detailed discussion of allowances and of nutrients not tabulated.

<sup>2</sup> Kilojoules (KJ) = 4.2 x kcal

<sup>3</sup> Retinol equivalents

<sup>4</sup> Assumed to be all as retinol in milk during the first six months of life. All subsequent intakes are assumed to be one-half as retinol and one-half as β-carotene when calculated from international units. As retinol equivalents, three-fourths are as retinol and one-fourth as β-carotene.

<sup>5</sup> Total vitamin E activity, estimated to be 80 percent as α-tocopherol and 20 percent other tocopherols. See text for variation in allowances.

<sup>6</sup> The folic acid allowances refer to dietary sources as determined by *Lactobacillus casei* assay. Pure forms of folic acid may be effective in doses less than one-fourth of the RDA.

<sup>7</sup> Although allowances are expressed as niacin, it is recognized that on the average 1 mg of niacin is derived from each 60 mg of dietary tryptophan.

<sup>8</sup> This increased requirement cannot be met by ordinary diets; therefore, the use of supplemental iron is recommended.

1974 REVISIONS: Vitamin E Activity Females 11-14 12 IU Ascorbic Acid Lactating 80 mg  
Females 15-18 12 IU Vitamin B-6 Males 15-18 2.0 ug

## APPENDIX A

## Protein Requirements by Age and Creatinine Level

Age		Creatinine Clearance/1.73 m <sup>2</sup>		
		20-30 ml	10-20 ml	less than 10 ml
0-2 months	Protein	2.2 gm/kg	2.0 gm/kg	1.5 gm/kg
2-6 months	Protein	2.0 gm/kg	1.6 gm/kg	1.2 gm/kg
6 months-2 years	Protein	1.8 gm/kg	1.2 gm/kg	1.0 gm/kg
2-3 years	Protein	1.5 gm/kg	1.0 gm/kg	0.5 gm/kg

## APPENDIX B

### Data Collection Instruments

Individual Growth Grids - Figures 1-13

Mean Growth and Composite Grid of Five  
Boys Followed 12 Months or Longer -  
Figure 14

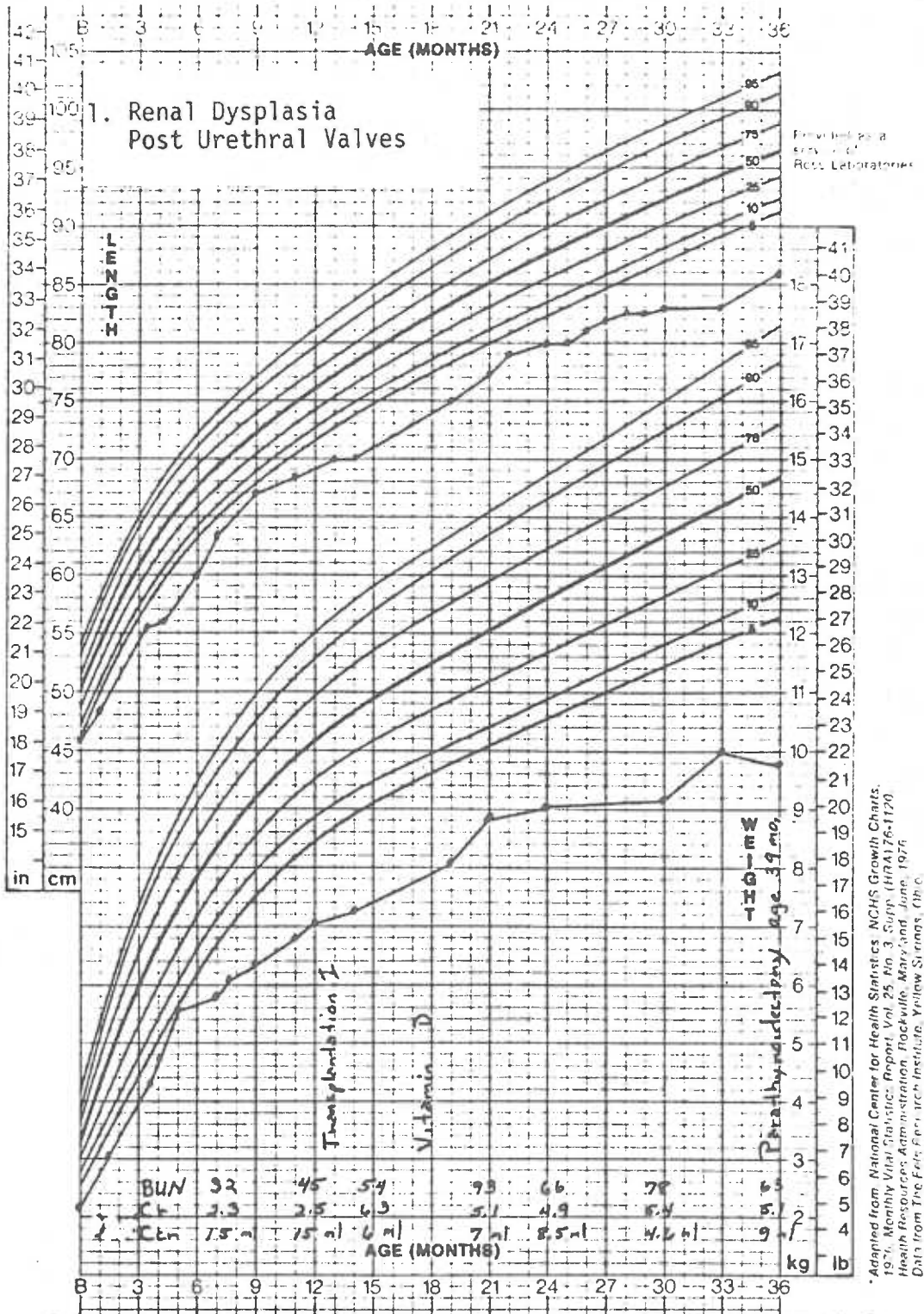
Mean Growth of Three Girls Followed 30  
Months or Longer - Figure 15

Comparison of Growth Velocity in Two Girls  
and Five Boys Followed 12 Months or  
Longer - Figure 16

**BOYS: BIRTH TO 36 MONTHS**  
**PHYSICAL GROWTH**  
**NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



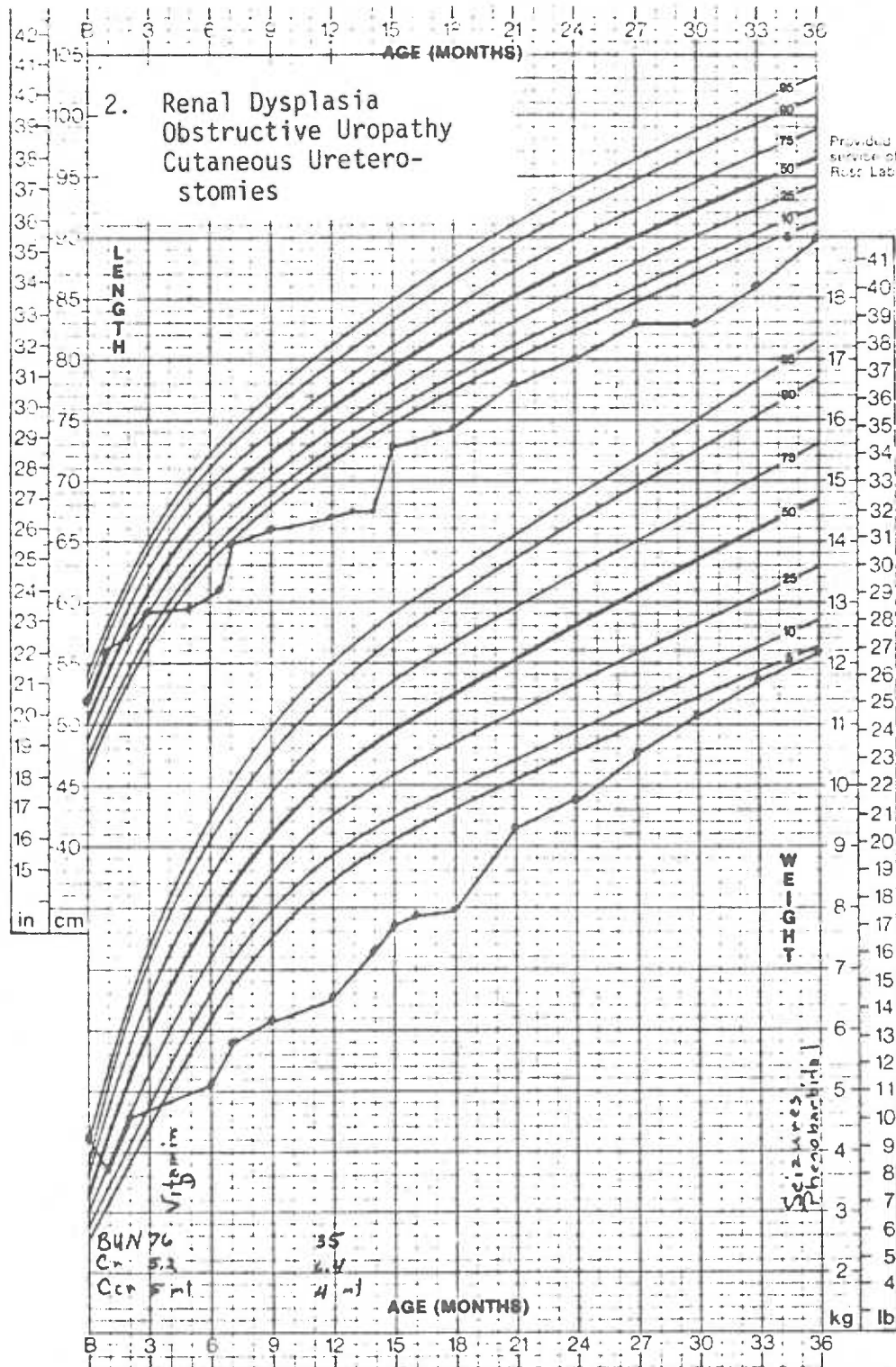
\* Adapted from: National Center for Health Statistics NCHS Growth Charts.  
 1976. Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (HRA176-1120)  
 Health Resources Administration, Rockville, Maryland, June, 1976.  
 Data from The Fels Research Institute, Yellow Springs, Ohio.  
 © 1976 NCHS, IAPCA, NCHS

Figure 1

**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



\* Adapted from: National Center for Health Statistics, NCHS Growth Charts, 1976, Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (HR9A)76-1170. Health Resources Administration, Rockville, MD, 20851, June, 1976. Child from The Reser Laboratories, 14106 Greenway Drive, Reser, NC 27577.

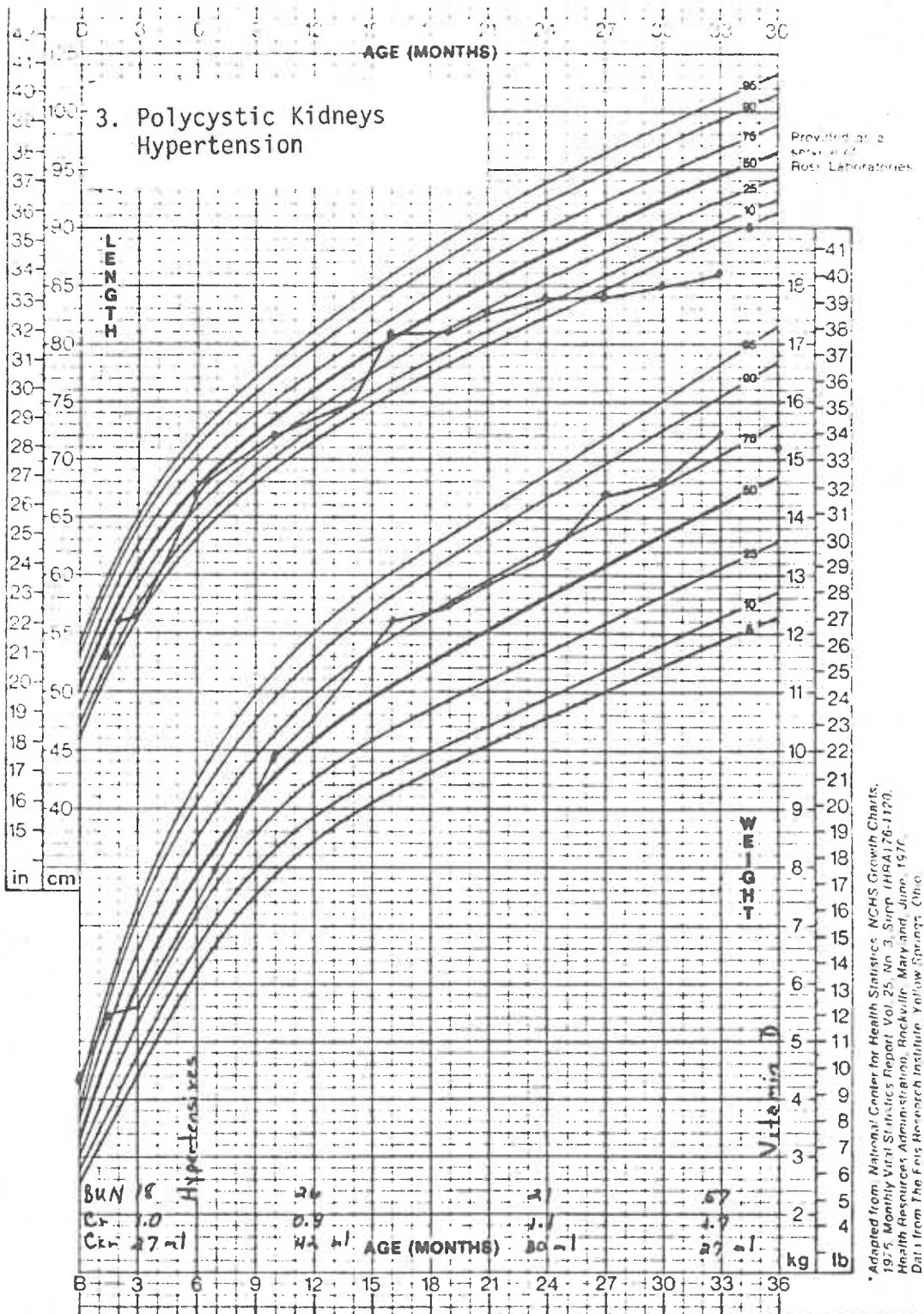
Figure 2



**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



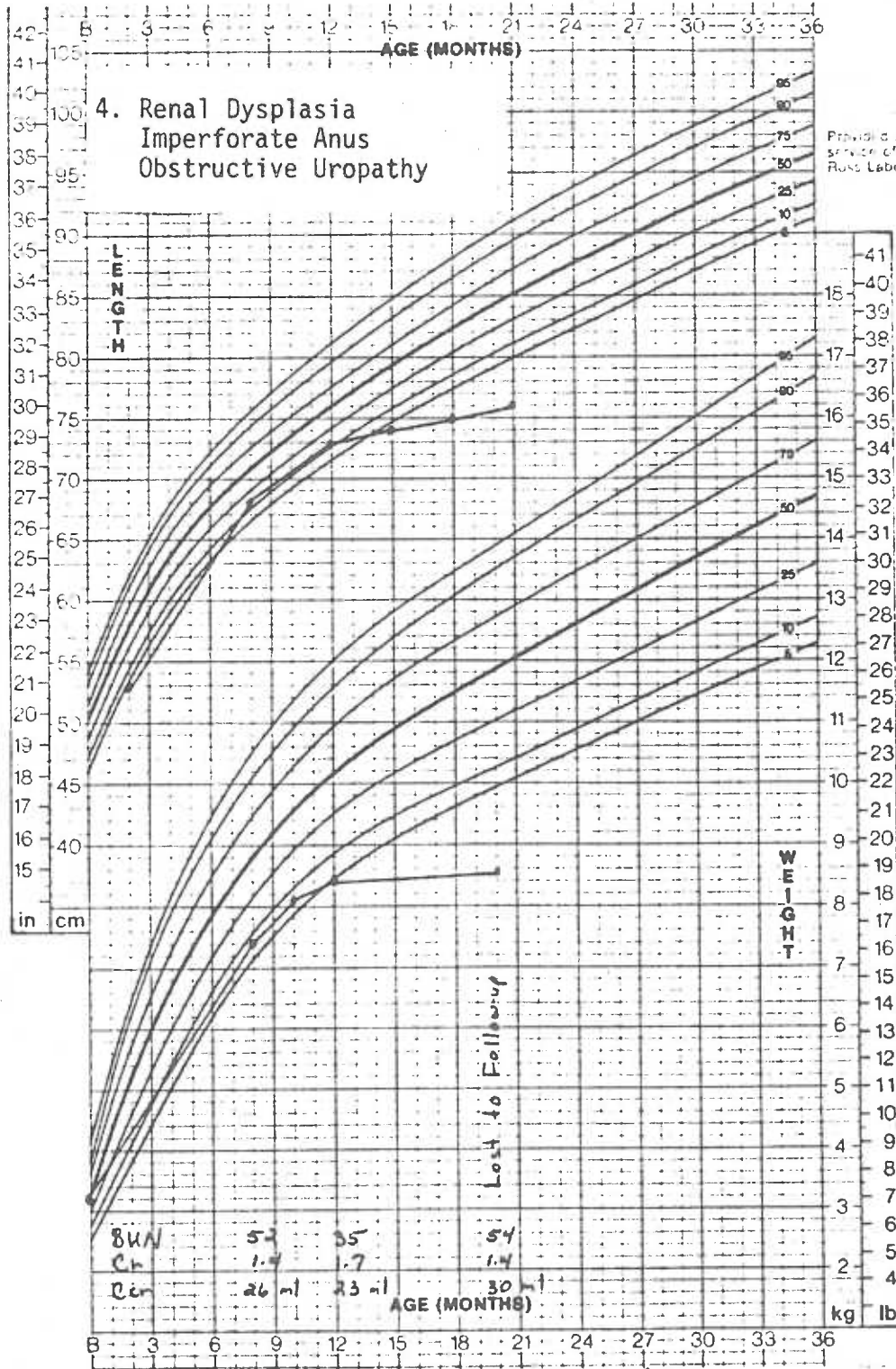
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Health Resources Administration, Rockville, Maryland, June, 1977.  
Data from The Fels Research Institute, Yellow Springs, Ohio.  
© 1978 ROSS LABORATORIES

Figure 3

**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



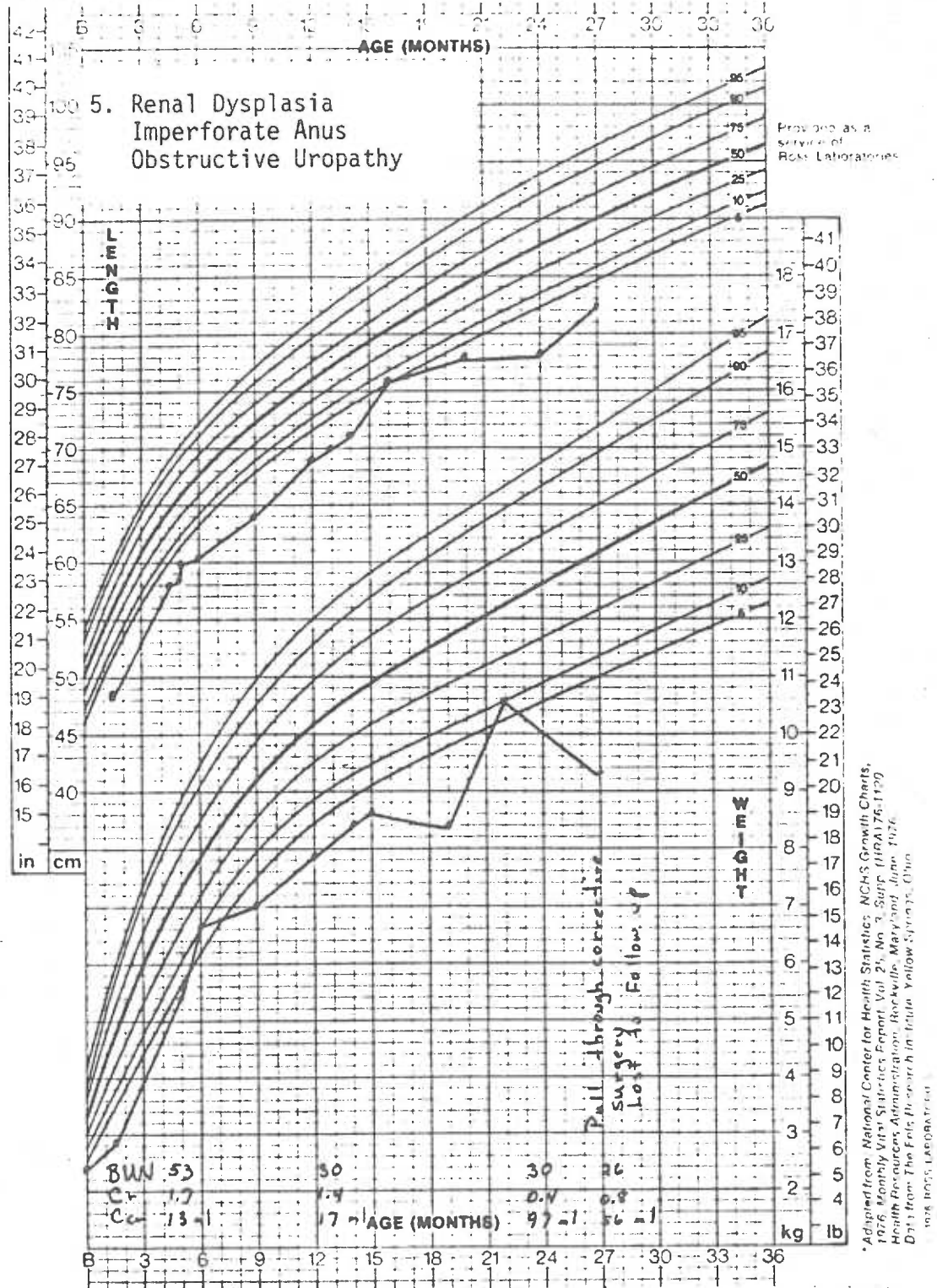
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Figure 4

**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



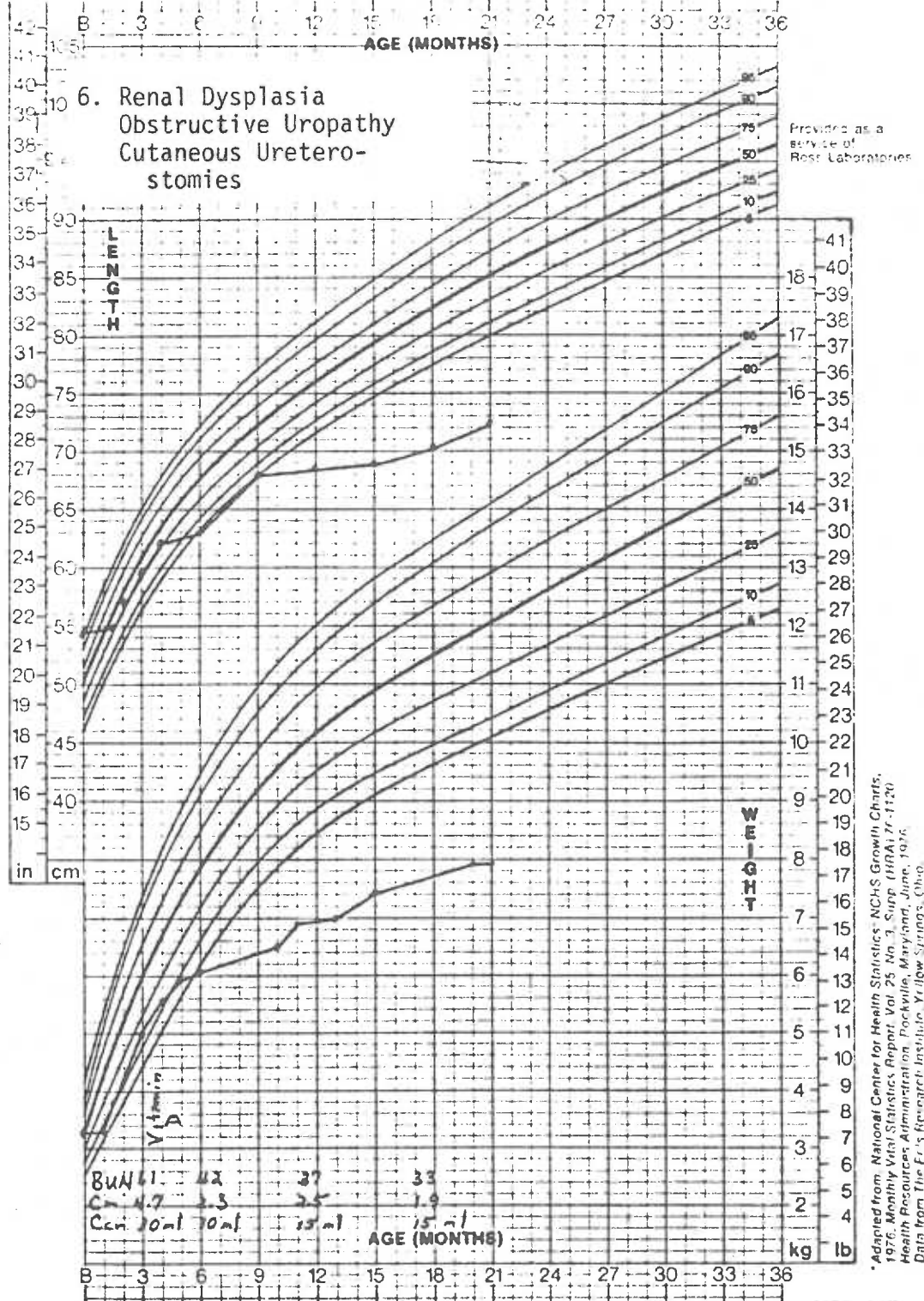
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Figure 5

**BOYS: BIRTH TO 36 MONTHS**  
**PHYSICAL GROWTH**  
**NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



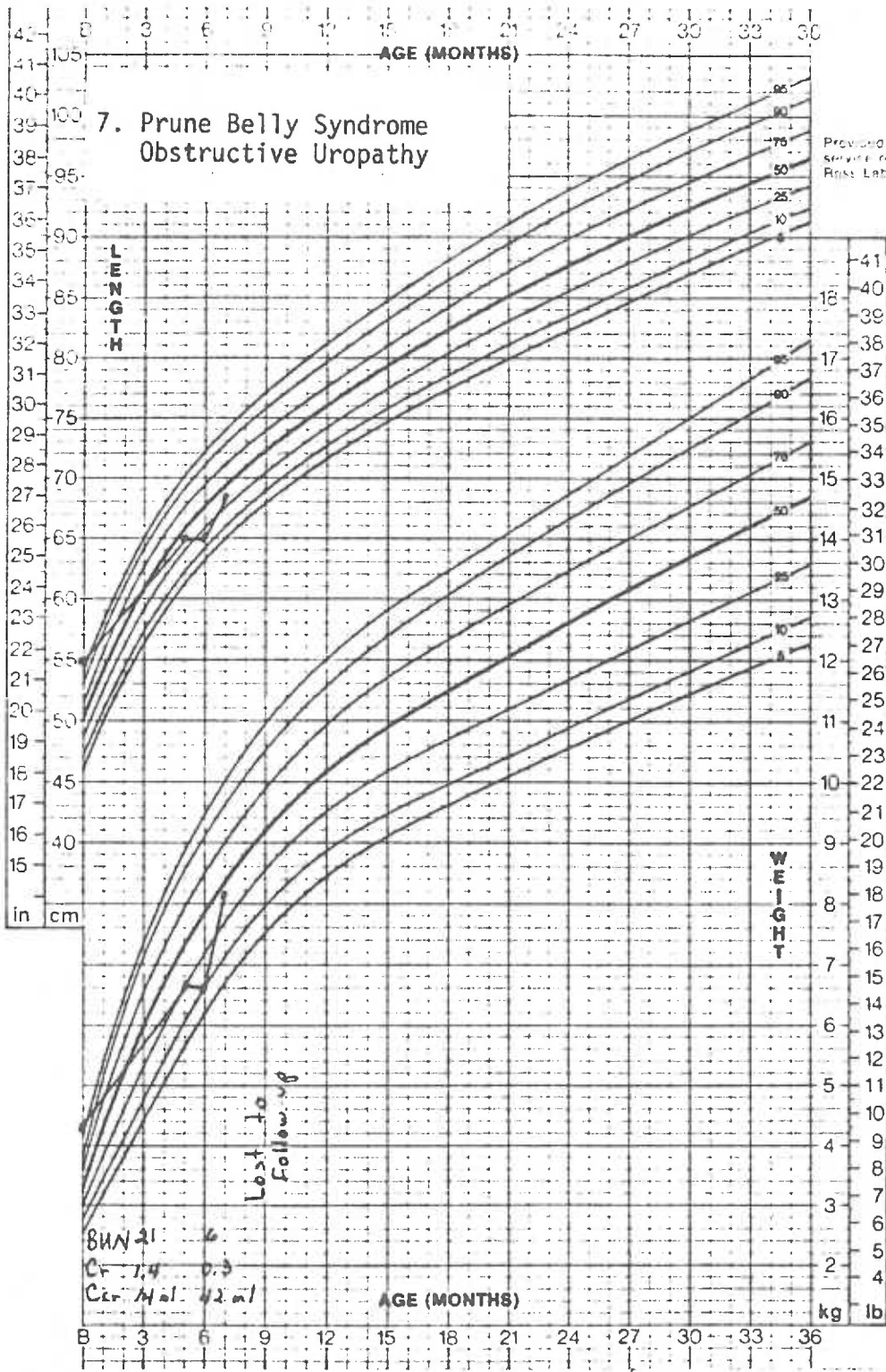
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Figure 6

**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



Provided as a service of Risk Laboratories

\*Adapted from National Center for Health Statistics, NCHS Growth Charts, 1976 Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (1976-1120). Health Resources Administration, Rockville, Maryland, June, 1976. Data from The Fels Research Institute, Yellow Springs, Ohio. © 1976 RISK LABORATORIES

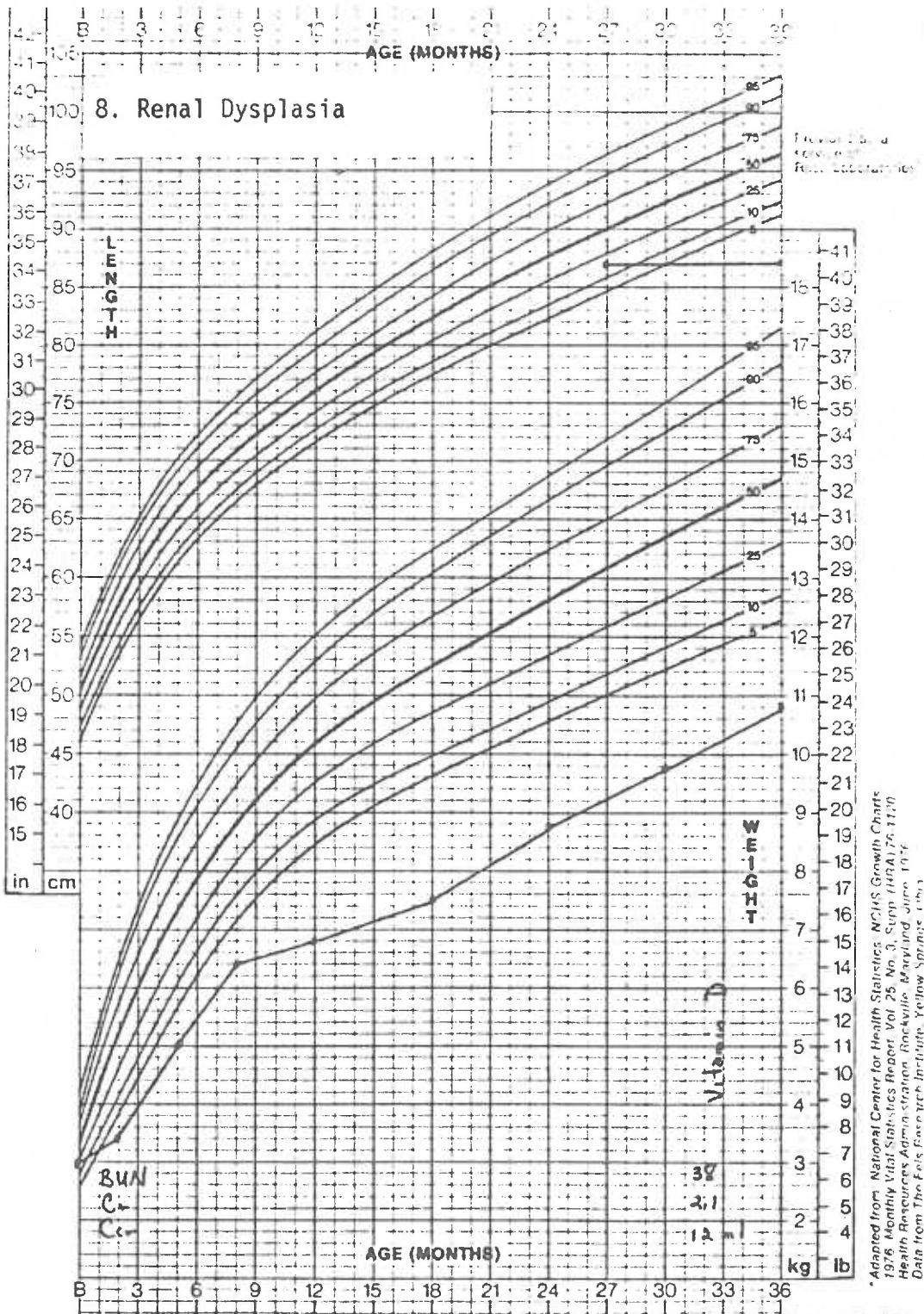
Figure 7



**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



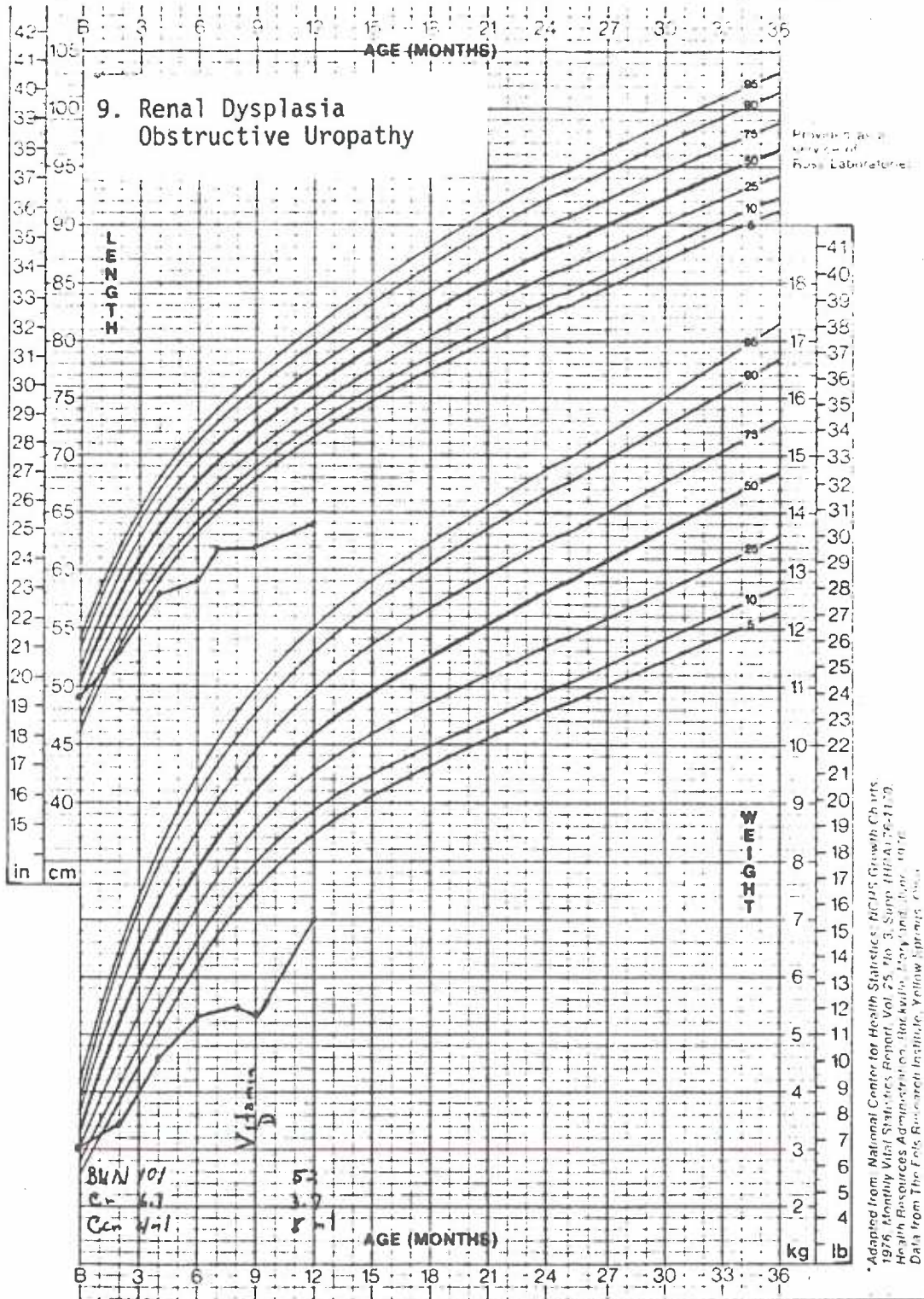
\* Adapted from: National Center for Health Statistics: NCHS Growth Charts 1976. Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (11/11/74):11-17. Health Resources Administration, Rockville, Maryland, June 1977. Data from The Fels Research Institute, Yellow Springs, Ohio. © 1977, PHS, LAG0041-1115.

Figure 8

**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



\* Adapted from: National Center for Health Statistics, NCHS Growth Charts, 1976, Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (11)(A), 76-1179, Health Resources Administration, Rockville, Maryland, Jan. 1976. Data from The Fels Research Institute, Yellow Springs, Ohio. © 1976 NCHS, Lathrop Co., B.I.

Figure 9

**GIRLS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

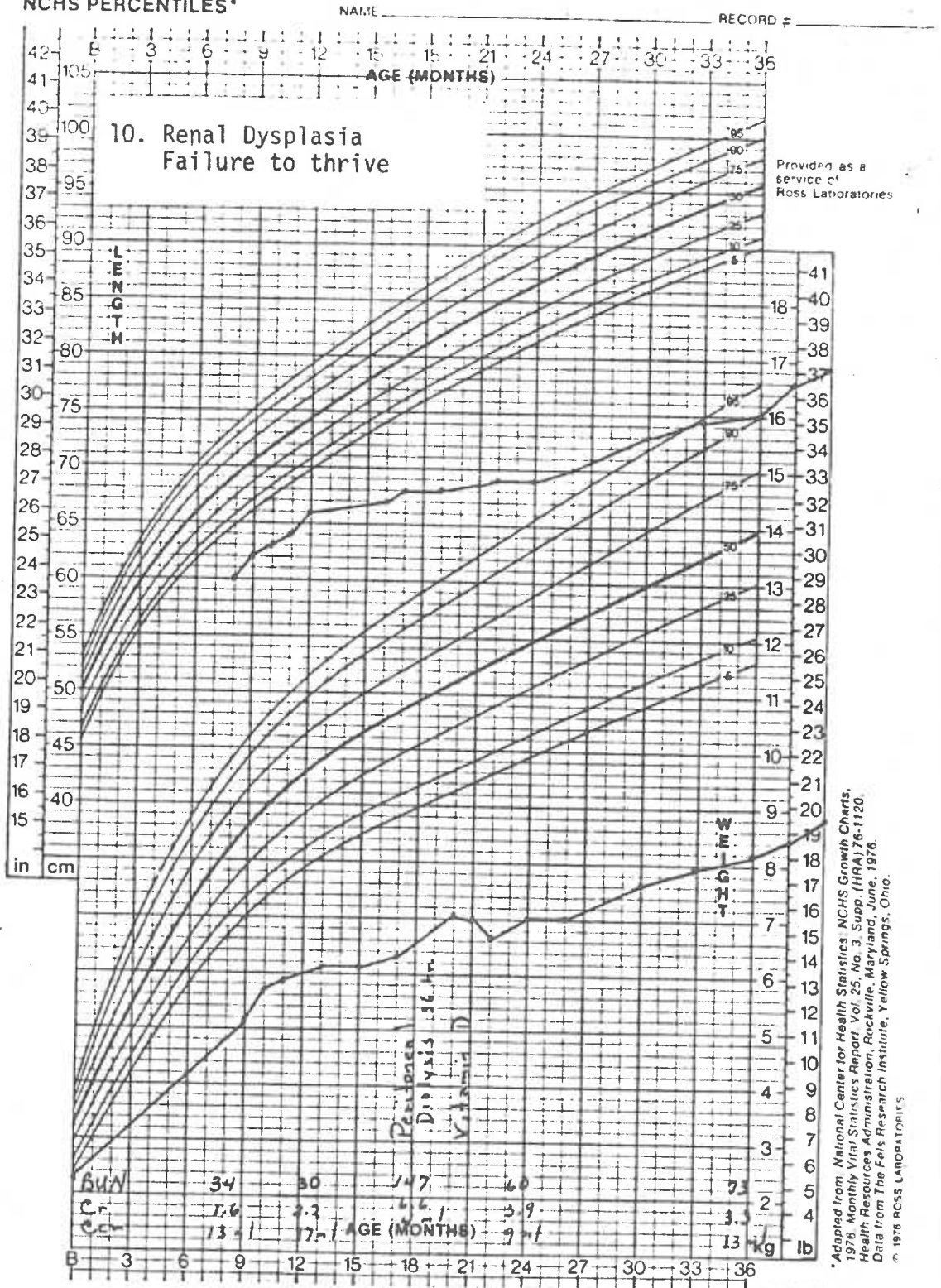


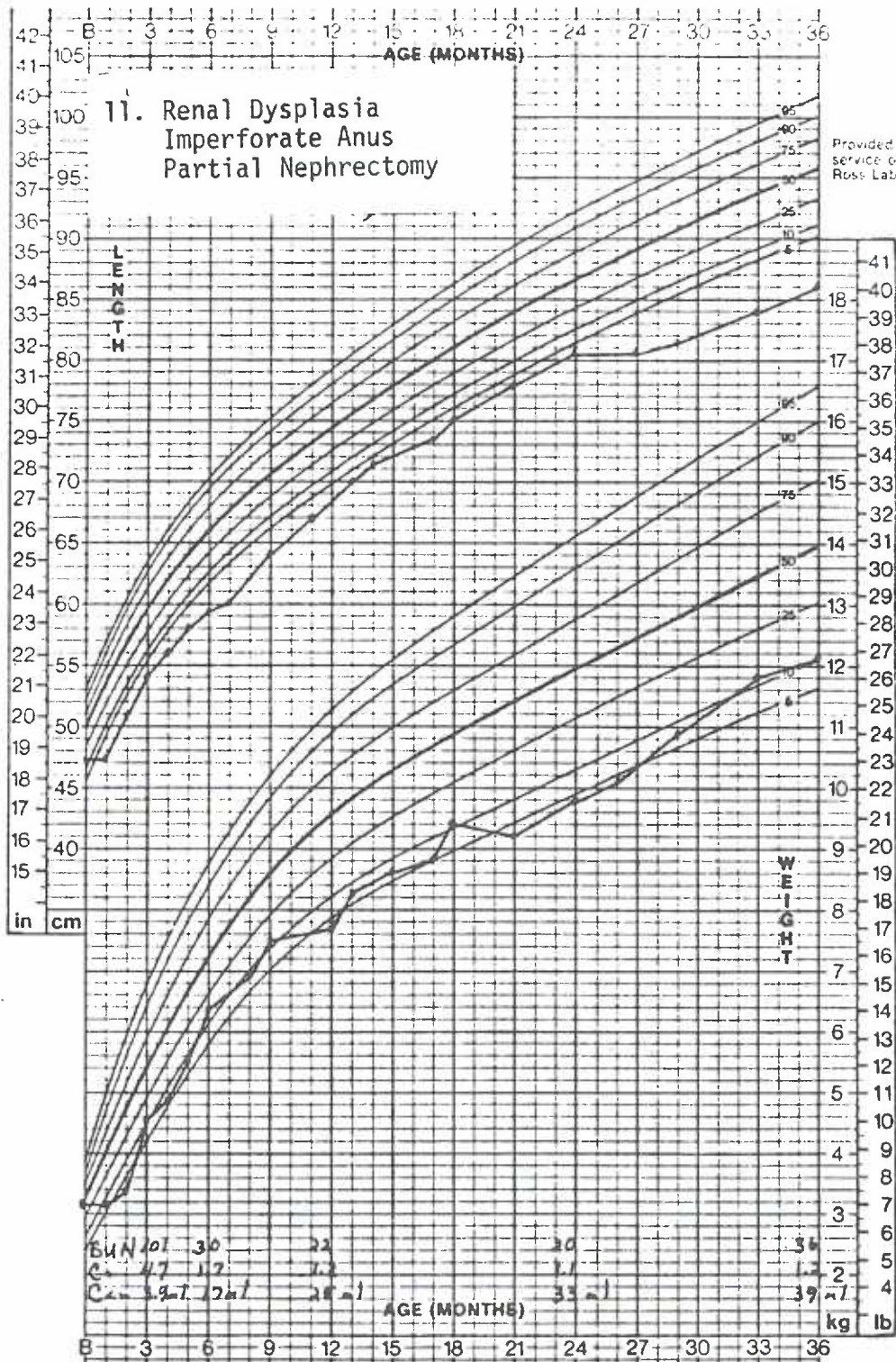
Figure 10



GIRLS: BIRTH TO 36 MONTHS  
 PHYSICAL GROWTH  
 NCHS PERCENTILES\*

NAME

RECORD #



\*Adapted from: National Center for Health Statistics: NCHS Growth Charts, 1976. Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (HRA) 76-1120. Health Resources Administration, Rockville, Maryland, June, 1976. Data from The Fels Research Institute, Yellow Springs, Ohio. © 1976 ROSS LABORATORIES

Figure 11

**GIRLS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_

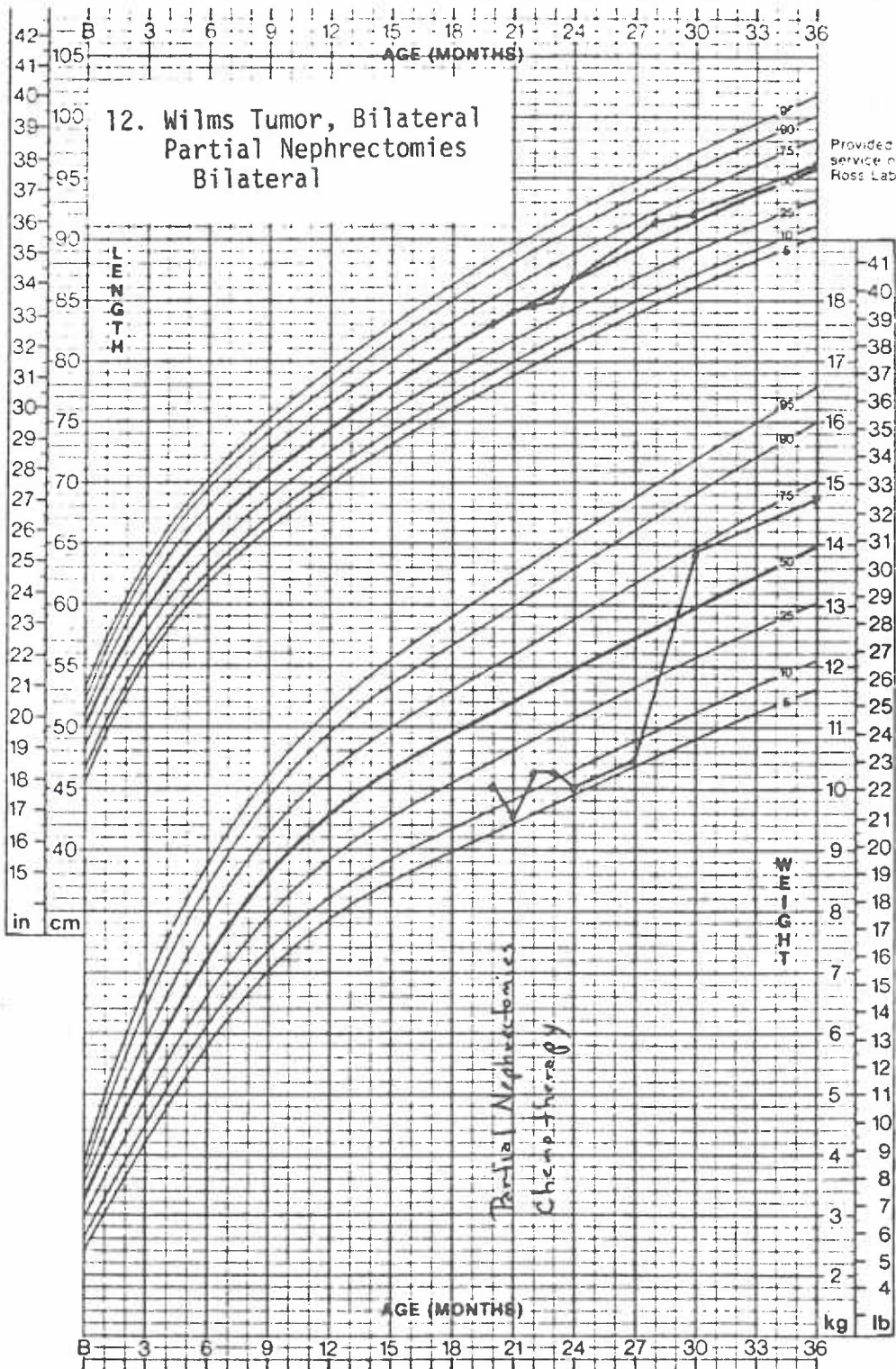


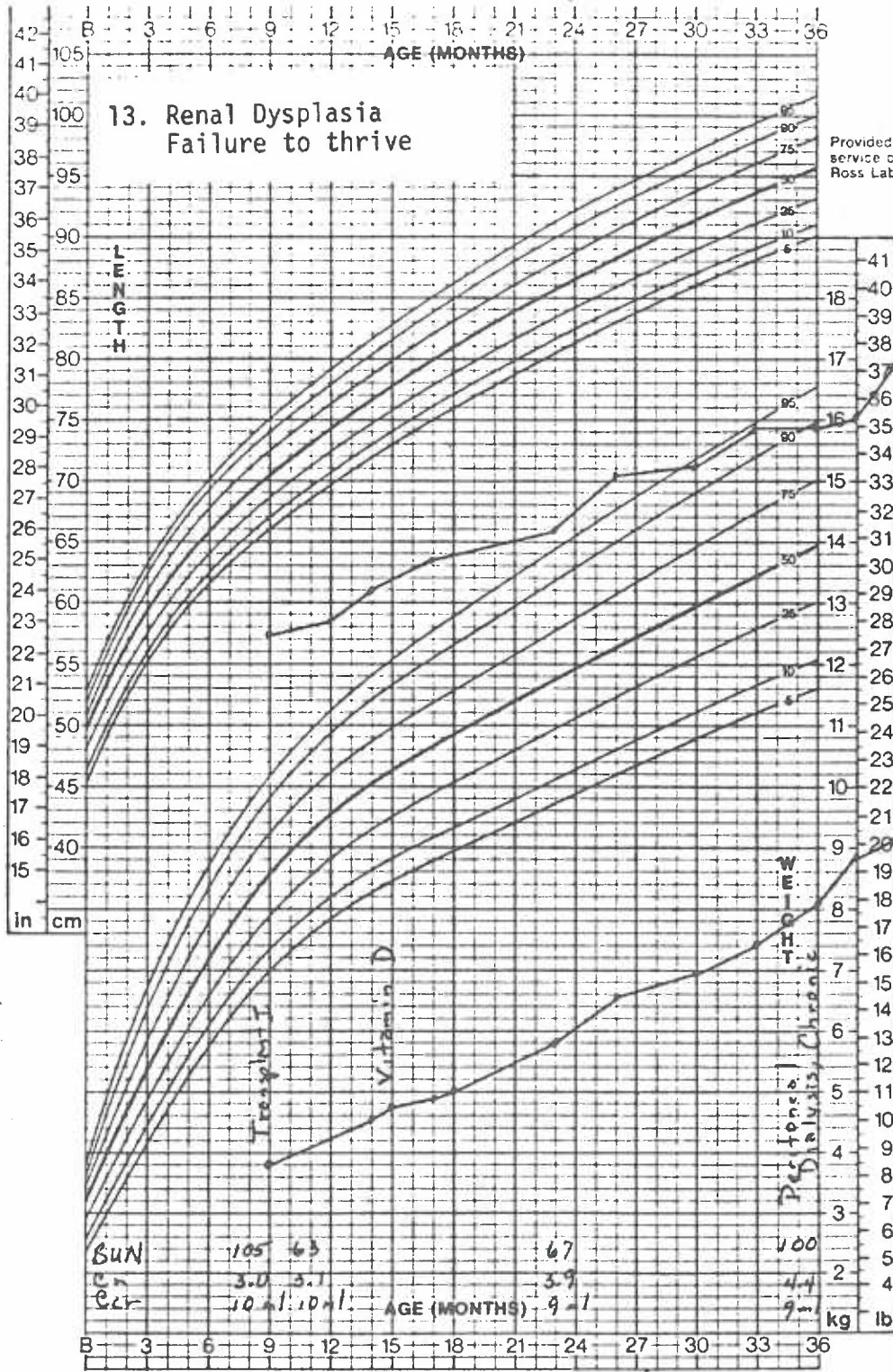
Figure 12



GIRLS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\*

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



\* Adapted from: National Center for Health Statistics: NCHS Growth Charts, 1976. Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (HRA)76-1120. Health Resources Administration, Rockville, Maryland, June, 1976. Data from The Fels Research Institute, Yellow Springs, Ohio. © 1976 ROSS LABORATORIES

Figure 13

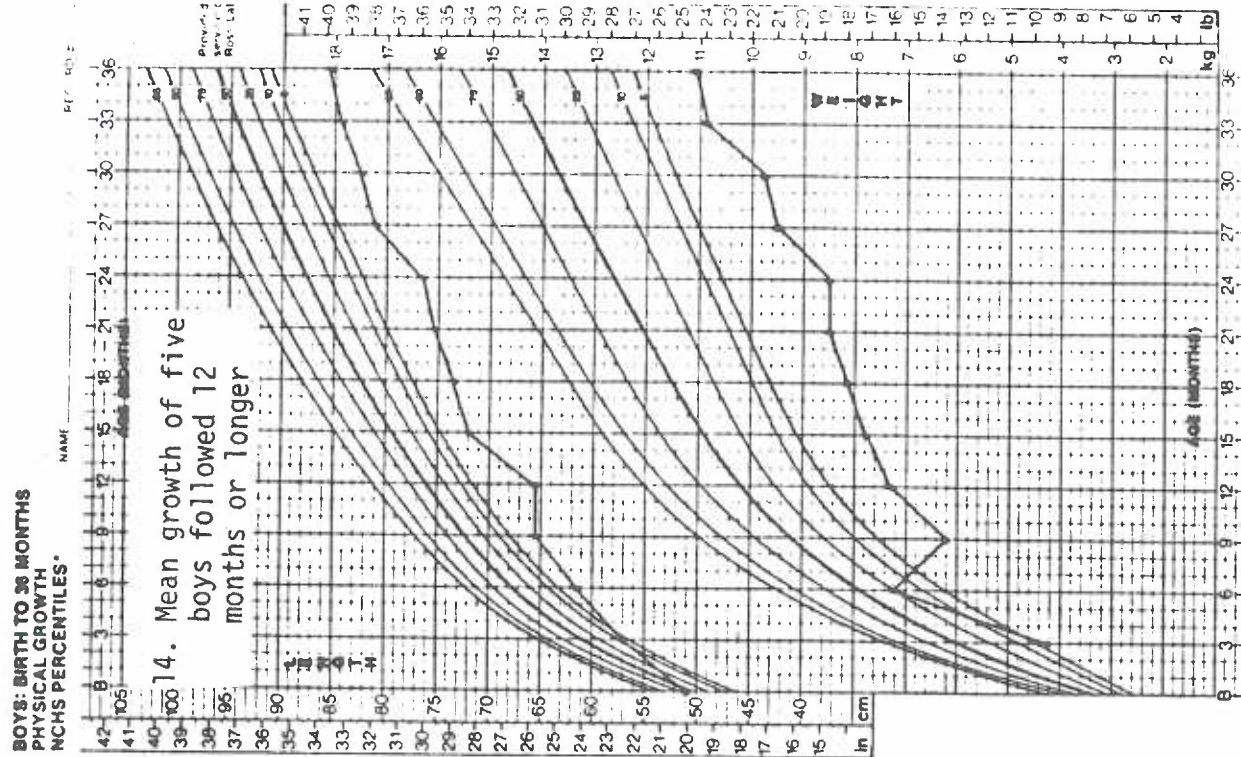
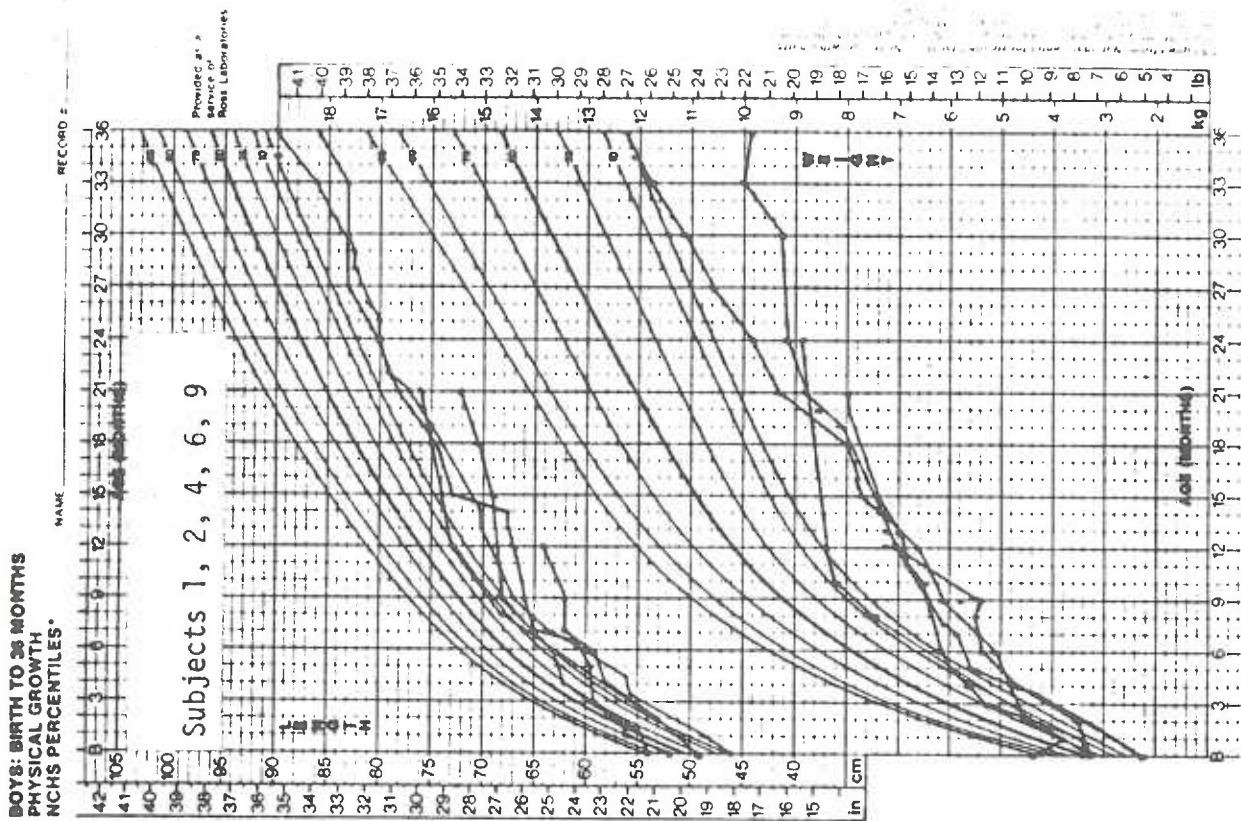


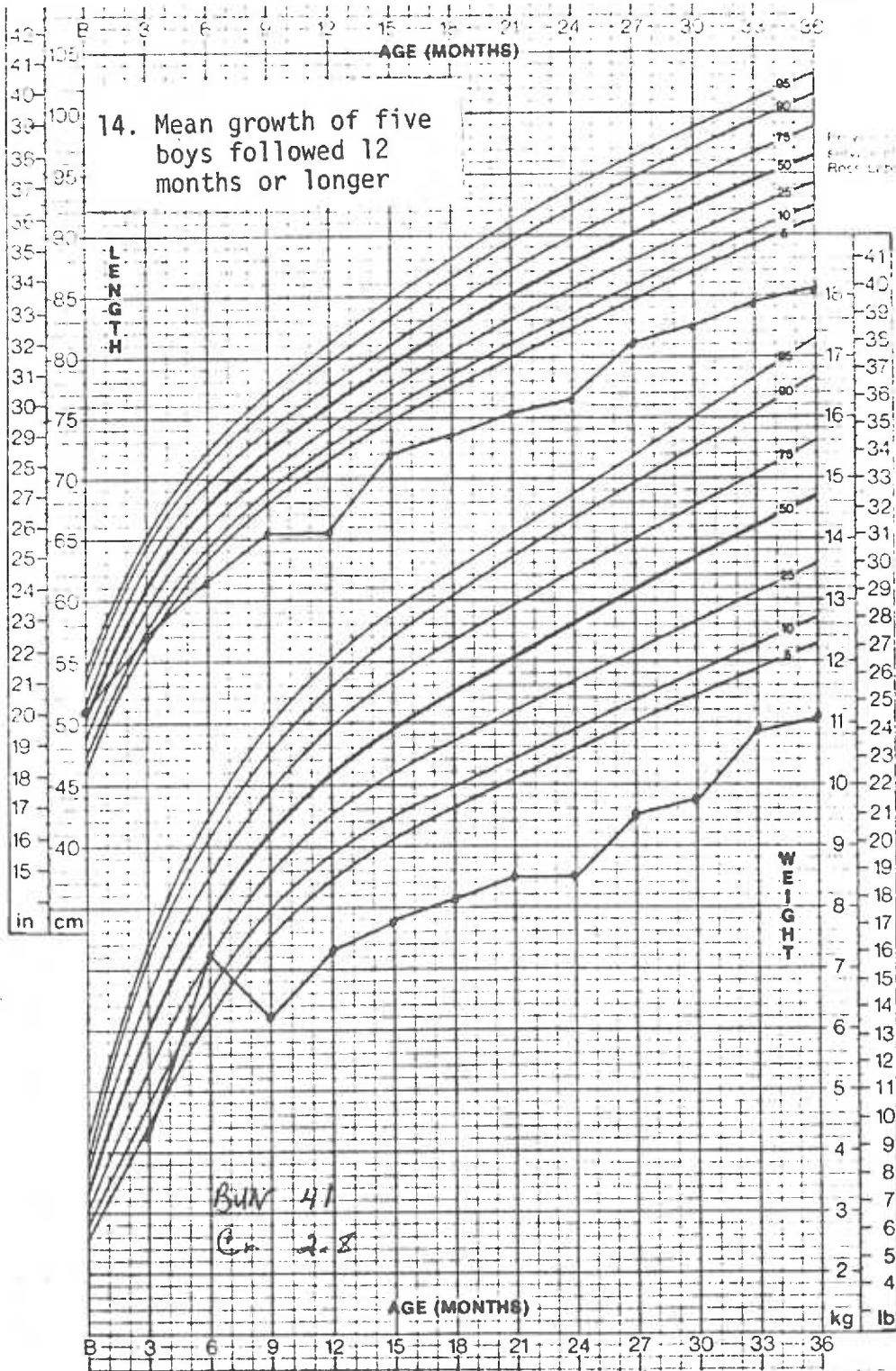
Figure 14



**BOYS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\***

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_

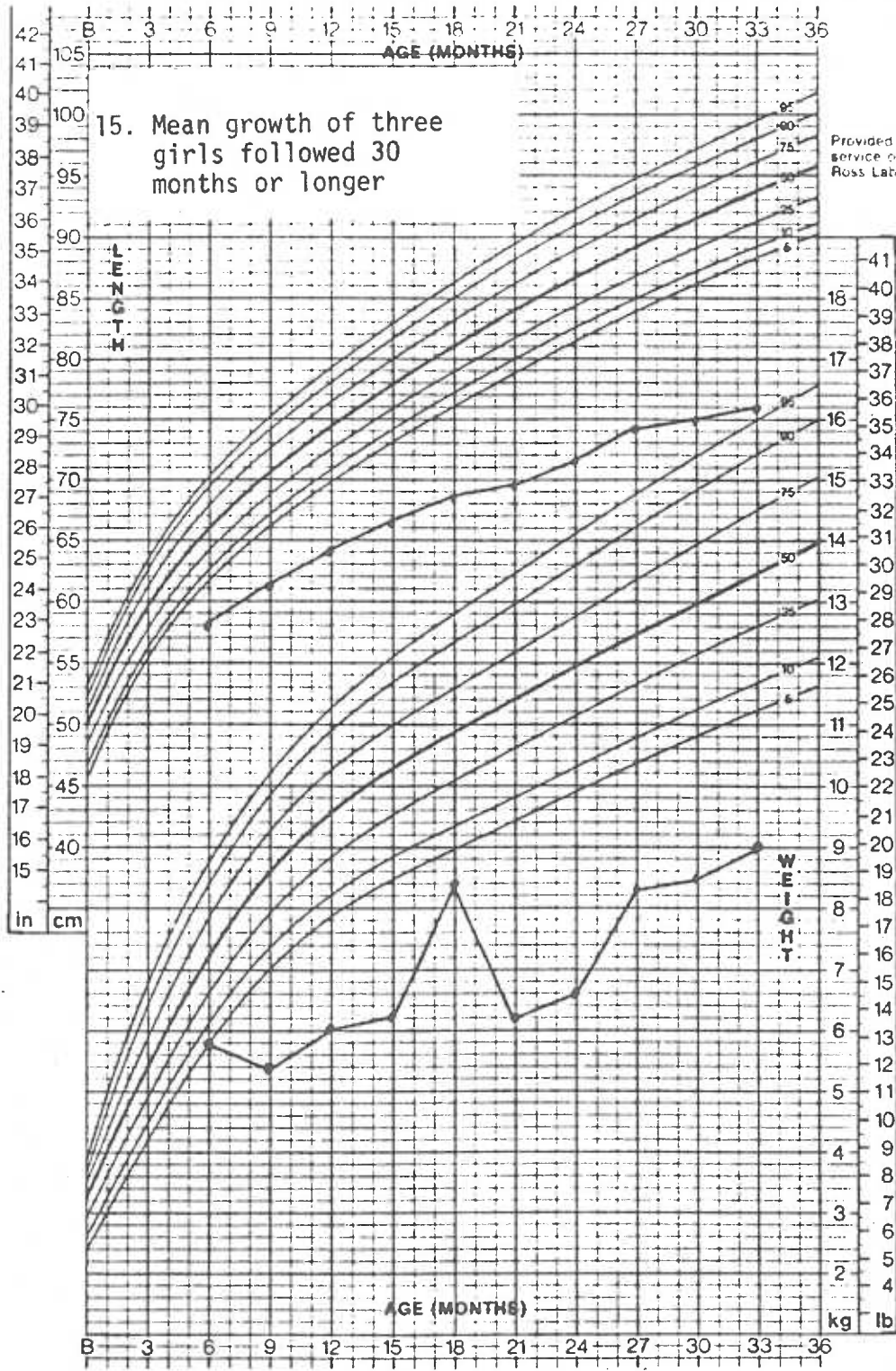


\*Adapted from National Center for Health Statistics NCHS Growth Charts, 1976, Monthly Vital Statistics Report, Vol. 25, No. 1, Supp. (HQ-A176-1170) Health Resources Administration, Rockville, Maryland, June, 1976. Data from The Fels Research Institute, Yellow Springs, Ohio. © 1975, ROSS LABORATORIES

GIRLS: BIRTH TO 36 MONTHS  
PHYSICAL GROWTH  
NCHS PERCENTILES\*

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_



\* Adapted from National Center for Health Statistics: NCHS Growth Charts, 1976. Monthly Vital Statistics Report, Vol. 25, No. 3, Supp. (HRA) 76-1120. Health Resources Administration, Rockville, Maryland, June, 1976. Data from The Felix Research Institute, Yellow Springs, Ohio. © 1976 ROSS LABORATORIES

Figure 15

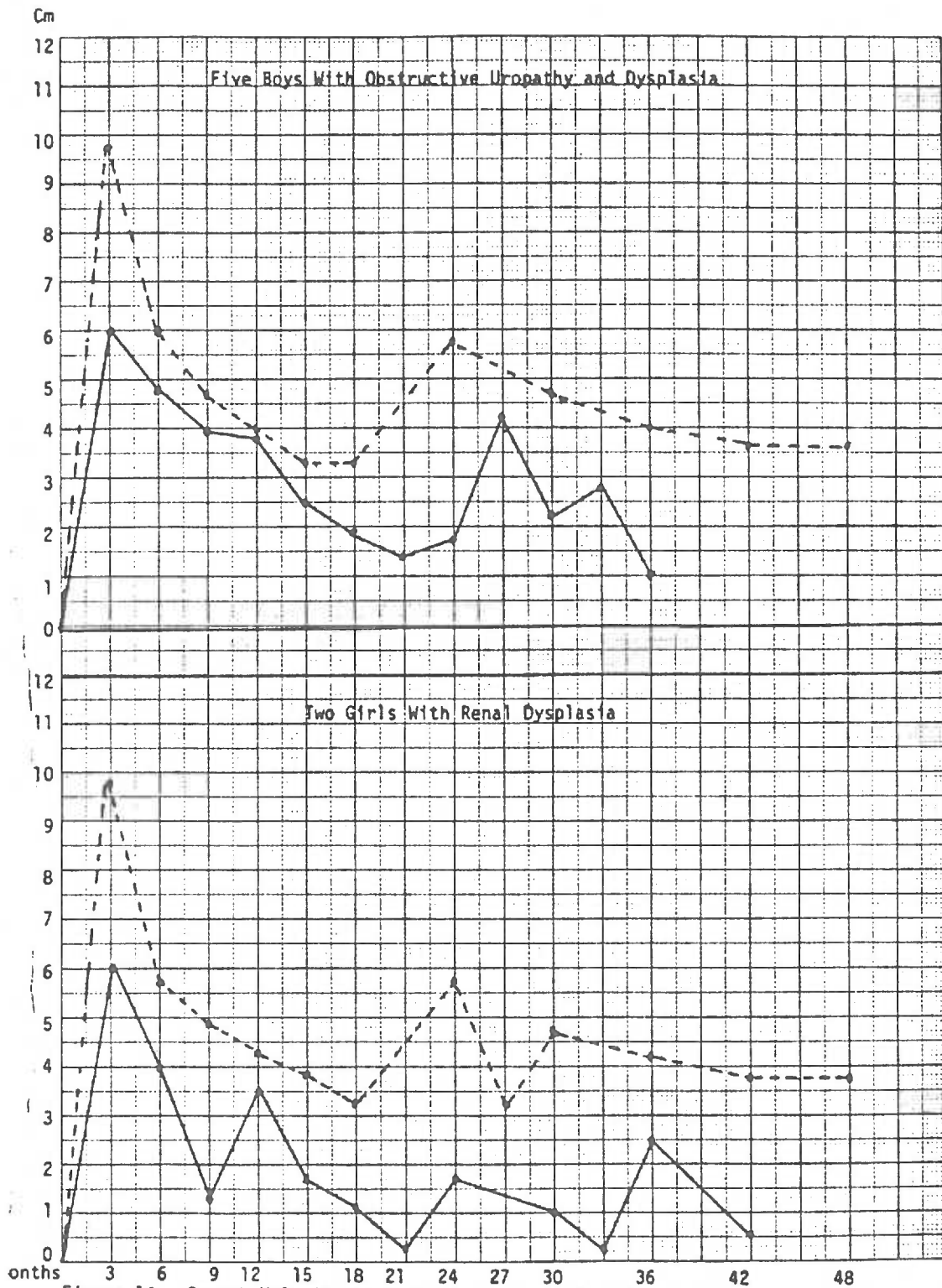


Figure 16: Growth Velocity in Children Followed From One to Three Years and Compared with Velocity at the 50th Percentile.

AN ABSTRACT OF THE  
CLINICAL INVESTIGATION OF

BARBARA LOGGAN FREIBERG, B.S., R.N.  
for the Master of Nursing

Date of Receiving this Degree: June 8, 1979

Title: A DESCRIPTION OF THE GROWTH OF YOUNG CHILDREN WITH CHRONIC  
RENAL FAILURE

Approved: \_\_\_\_\_  
Professor in Charge of Clinical Investigation

The purpose of this clinical investigation was to document and describe linear growth in young children under four years of age who were in chronic renal failure. This descriptive study, through a retrospective chart review, presented thirteen subjects who were patients at the University of Oregon Health Sciences Center during the period between January, 1974 to June, 1978. This sample size, although small numerically, represented all of the cases referred to the University of Oregon Health Sciences Center pediatric nephrology service during the identified time period.

Variables that may affect growth were discussed: osteodystrophy, acidosis, anemia, malnutrition, genetics, and pathology of renal disease. The contribution of the social environment, particularly the family as a support system was included. The children's linear growth patterns were



plotted and compared with growth of normal children by the use of individual growth charts. Further measurements included a comparison of median growth velocity to the 50th percentile for normal height and a Weight-Height Index to demonstrate caloric intake.

The study supported observations made in the literature that linear growth retardation in children with chronic renal failure may be severe. Although some of the children with renal insufficiency, as demonstrated in creatinine clearances of less than 30 ml/1.73, did grow, the velocity was less than that of normal children. None of the 13 subjects followed the normal growth patterns for age and sex outlined on NCHS growth charts. In addition, 11 of the 13 subjects demonstrated malnourishment where their weight was compared to normal weights for age (Weight-Height Index).

Multiple factors with a probable relation to growth failure were identified in this sample, again supporting findings in the literature. Obstructive uropathy and renal dysplasia were seen as major causes of chronic renal failure and occurred more frequently in boys (five out of the nine boys) than girls (one out of four) in this study.

This investigation described growth in children in chronic renal failure under three years of age providing information not previously available in the literature. Further studies are recommended to provide much needed longitudinal information about growth patterns in this population. The long term goal of such studies would be the identification and control of factors that would promote improved growth in these children.