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# Renal Nutrition Forum

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## Feature Article

# Renal Tubular Dysfunction and Failure to Thrive in Children: A Case Study

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**This article has been approved for 2 CPE units. The CPEU insert can be accessed in the Members Only Section of the web site from the CPEU Inserts link.**

### Case Presentation

This article presents a case study of a young boy with a previous diagnosis of nephrogenic diabetes insipidus and a new question of kidney stones. He was an ex-26 week premie (BW: 853 g) with a history of polyuria and polydypsia since one year of age. He was being managed with thiazide diuretics, which caused significant fatigue according to the parents. After 1 week of treatment the medication was stopped. The parents were seeking further evaluation.

He presented in clinic at 3 and 10/12 years old with failure to thrive (FTT), height of 84.8 cm (3rd percentile), weight of 9.8 kg (< 3rd percentile), Body Mass Index (BMI) of 13.6 (<3rd percentile), polyuria (4-6 liters per day), polydypsia (4 liters per day), and intolerance to milk. The parents reported a very good appetite claiming he ate 4 to 6 times a day and drank water constantly. A 24-hour diet recall suggested an intake of 1700 kcals (150% of RDA) and high sodium (Na) intake. Renal ultrasound showed nephrocalcinosis. Initial labs were all within normal limits and ruled out the diagnosis of nephrogenic diabetes insipidus. A 24-hour urine litholink (an extensive and specific urine analysis) showed severe hypercalciuria, mild hyperoxaluria, hyperuricosuria and very low urinary citrate excretion. The pediatric renal dietitian was consulted to instruct the parents on a high fluid, low Na and low oxalate diet to lessen his risk for stone formation. Plans were made to do further testing to make a differential diagnosis.

Before additional testing could start, the boy was emergently hospitalized with severe hand and foot cramps and stiffening of the arms and legs. Initial labs were significant for hypocalcemia, hypokalemia, hypophosphatemia and hypomagnesemia. 25-hydroxy vitamin D was low but 1,25-dihydroxy vitamin D and parathyroid hormone (PTH) were elevated (Table 1). Consults were obtained from Ophthalmology to detect any signs of cystinosis, Endocrinology to evaluate vitamin D levels and Gastroenterology to evaluate for malabsorption. Nutrition was consulted for a calorie count and evaluation for FTT. His calorie intake initially was poor, but once electrolyte replacement began his appetite returned and was eating adequately. FTT due to inadequate calorie and protein intake was ruled out. Ophthalmology ruled out cystinosis. Endocrinology found normal parathyroid function but a significantly delayed bone age of 2 years old. Gastroenterolgy determined there was no malabsorption.

A working diagnosis of Bartter Syndrome was made, but the parents refused further genetic testing to determine the variant of Bartter

**RPG**

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*The views expressed in this publication are those of the author and are not necessarily those of The American Dietetic Association. Publication of an advertisement in the Forum should not be construed as endorsement by the RPG of the product or the advertiser.*

Articles about successful programs, research interventions, evaluations and treatment strategies, educational materials, meeting announcements and information about educational programs are welcome and should be emailed to the editor by the next deadline.

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***Remember to update your profile electronically in the 'members only' section of ADA's website. You will need your registration number and web password. Keeping ADA informed of your name and contact information will help avoid delayed issues of your Renal Nutrition Forum.***

# From the Editor's Desk

This time of year brings many changes to anticipate, including the start of the school year, change in seasons, the holidays, and even a U.S. presidential election. Additionally, you now have a new editor for the Renal Nutrition Forum. It is my honor to serve as your new editor, and I look forward to the challenges that come with producing an informative, high-quality, peer-reviewed publication.

What changes can you expect in the Forum? You may notice that the editorial team has been making significant efforts to provide a greater diversity in topics to reflect the wide variety of the patient populations our members serve. For example, enclosed you will find an excellent case study on renal tubular necrosis in a pediatric patient, as well as advice on how to interact with the media. Other recent features have also included cardiovascular disease in transplant patients, as well as a case study in an obese, early chronic kidney disease patient.

Additionally, in this issue, please see the "Errata" for the Summer 2008 article, "Nephrology Nutrition and the Nutrition Care Process: A Renal Nutrition Forum Series with Practice-Based Examples of the NCP," which summarizes its misprints. You may recall that these corrections were also highlighted in an RPG members' e-blast in early September, which hopefully helped dispel any questions that arose. We apologize for any inconvenience or confusion.

What recent changes do you need to be aware of? With the recommendation and implementation of the Nutrition Care Process (NCP) and the new Medicare guidelines coming into effect this fall, there will certainly be no shortage of activity for renal care providers! Be sure to read this edition of the NCP series and CRN chair message for more information.

What does all this change mean to you? The greater diversity in Forum topics means I need to hear from you, the reader, regarding topics you would like to learn more about, authors you would like to hear more from, and recent research you find fascinating. Most importantly, if you would like to publish an article, case study, research, or quality improvement project… look no further. No project is insignificant to us!

> "It is not the strongest of the species that survive, nor the most intelligent, but the one most responsive to change."
> -Anonymous

If you are a fledgling author, please consider this request to challenge yourself. Take a step in a new direction and make a leap of faith to try your hand at something completely new. This is a terrific opportunity to support your profession and patient populations by furthering research, exploring current literature, and solidifying our practice into one of sound research, and not simply opinion. ◆

Rachael R. Majorowicz

**Table 1**
Laboratory Values

| | Reference Range | Clinic Visit | Hospital Admit | Hospital Discharge | 2 Wks Post D/C | 10 Wks Post D/C |
|---|---|---|---|---|---|---|
| Na (mmol/L) | 135-145 | 136 | 143 | 134 | 137 | 140 |
| K (mmol/L) | 3.5-5.2 | 3.6 | 2.4 | 3.9 | 3.3 | 4.5 |
| Cl (mmol/L) | 95-109 | 102 | 104 | 106 | 101 | 102 |
| Ionized Ca (mmol/L) | 1.14-1.32 | N/A | 0.7 | 1.16 | 1.23 | N/A |
| Mg (mg/dL) | 1.6-2.6 | 1.7 | 1.3 | 2.1 | 2.1 | 2.1 |
| P (mg/dL) | 4.5-6.5 | 5.1 | 4.1 | 6.0 | 4.98 | 6.3 |
| Albumin (g/dL) | 3.5-5 | 4.1 | 3.2 | N/A | 3.6 | 4.4 |
| 25-hydroxy vitamin D (ng/mL) | 20-57 | N/A | 7 | N/A | N/A | N/A |
| 1, 25 vitamin D (pg/mL) | 15-75 | N/A | 128 | N/A | N/A | N/A |
| PTH (pg/mL) | 15-75 | N/A | 283 | N/A | 20 | N/A |

N/A = not available

Syndrome. A second diagnosis of nutritional vitamin D deficiency was also made based on his history of milk intolerance and low 25-hydroxy vitamin D. Medications during his hospital stay included indomethacin, spironolactone, calcium carbonate, potassium chloride (KCl), ergocalciferol, magnesium oxide, neutra-phos, and IV saline fluids. Prior to discharge the neutra-phos was discontinued as his serum phosphorous (P) normalized. All other medications and mineral/vitamin supplementation were continued at discharge. He was also put on a high Na, high potassium (K) diet and high fluid diet. No particular level of Na or K was identified, but rather, foods high in Na and K content were reviewed. The parents identified favorites of the child and were advised to increase the portions and frequency. The family was Korean and used many high Na sauces. This practice was encouraged. Finally a minimum goal of at least 2 liters of fluid per day was advised.

**Table 2**
Anthropometrics

| | Clinic Visit | Hospital Admit | 2 Wks Post D/C | 10 Wks Post D/C |
|---|---|---|---|---|
| **Height (cm)** | 84.8 | 86.36 | 87.4 | 89.4 |
| **Weight (kg)** | 9.8 | 9.8 | 11.1 | 11.3 |
| **BMI** | 13.6 | 13.1 | 14.5 | 14.1 |
| **BMI percentile** | < 3rd | <3rd | 15th | 8th |

Within one month after discharge, spironolactone and calcium carbonate were discontinued. The ionized calcium (Ca) was rising rapidly and the child was eating yogurt and cheese daily. He continued on indomethacin, ergocalciferol, KCl, and magnesium oxide. He began to achieve catch-up growth in height and weight. BMI increased from less than the 3rd percentile to the 8th percentile by 10 weeks post discharge (Table 2).

## Discussion

This case report is an excellent example of how disturbances within the renal tubules can clinically present as FTT. This particular patient was diagnosed with Bartter Syndrome which led to excessive excretion of K, Na, Ca, and water. His secondary diagnosis of vitamin D deficiency exacerbated his condition due to insufficient absorption of Ca and P. The initial dietary intervention of salt and oxalate restriction, implemented to prevent kidney stones, may have exacerbated the condition by increasing urine output further. Bartter Syndrome is comprised of several closely related disorders of renal electrolyte transport. It is most often characterized by Na, K and chloride (Cl) wasting, hypokalemic metabolic alkalosis, hyperreninism and hyperaldosteronism. There are 5 variants of the syndrome, Types I through V, with variations in the types and severity of electrolyte disturbances that can occur. Detailed descriptions of these variants go beyond the purpose of this discussion; however, excellent reviews of the variants can be

**Table 3**
Renal Tubular Disorders that can Present with Failure to Thrive

- Fanconi Syndrome (proximal tubular dysfunction)
- Chronic Chloride Deficiency (metabolic alkalosis due to dietary chloride deficiency)
- Primary Proximal Renal Tubular Acidosis
- Distal Renal Tubular Acidosis
- Nephrogenic Diabetes Insipidus
- Bartter Syndrome
- Gitelman Syndrome
- Pseudo-Bartter Syndrome

found in Shaer (1), Proesman (2) and Unwin (3).

It has been established that defective sodium chloride (NaCl) transport at different segments of the distal tubule leads to the variants of Bartter Syndrome (1). The primary abnormality is defective Cl re-absorption in the thick ascending loop (TAL) of the loop of Henle (1). This leads to increased delivery of NaCl to the distal tubule which increases K and hydrogen losses leading to hypokalemic metabolic alkalosis and impaired urine concentrating. Another consequence of decreased Cl re-absorption is decreased Ca and magnesium (Mg) re-absorption leading to hypercalciuria and increased Mg losses (1). Clinically, the patient almost always presents with polydypsia, polyuria and FTT. Most patients presenting with Bartter Syndrome are pediatric, however, it has also presented in adult patients whenever damage to the tubules occur. Pseudo-Bartter syndrome presents more frequently in adults if there is abuse of thiazide and loop diuretics. These diuretics act at the site of Cl re-absorption and inhibit it, leading to excess Cl excretion. It differs from Bartter's because there is no renal tubular dysfunction. Once the diuretics are stopped and excessive excretion of Cl and Na ceases, the metabolic abnormalities resolve (4).

Of particular interest is the association with FTT and growth failure in conjunction with adequate calorie and protein intake. This pattern is more commonly seen with other pediatric renal disorders that affect the tubules' ability to reabsorb electrolytes, cause excessive excretion of electrolytes, or lead to inadequate re-absorption/excessive excretion of water (5). Table 3 lists some of the other disorders affecting the renal tubules. Although the tubular dysfunction will differ among those listed, the common overall features are the loss of one or more nutrients (Na, K, Cl, Ca, P, Mg and/or water) and FTT is a presenting factor at the time of diagnosis. In some disorders, FTT occurs despite evidence of adequate calorie and protein intake (e.g. Bartter, Gitelman, Chronic Chloride Deficiency and Pseudo-Bartter) (6). In other tubular disorders such as Renal Tubular Acidosis and Fanconi, FTT is accompanied by either no anorexia or mild to severe anorexia (7, 8, 9).

No clear evidence has been found to explain the definitive link between electrolyte and/or fluid wasting and FTT. Renal K wasting has been implicated in Bartter Syndrome as a growth depressing factor (10, 11, 12). However, only experimental studies have demonstrated that K depletion plays a role in growth retardation as it is accompanied by reduced growth hormone (GH), reduced response to GH releasing factors and reduction of insulin growth factor (IGF) (13, 14). The importance of K in the proper utilization of carbohydrate in insulin release and in growth is also well known but the mechanisms are unclear (1). In an early study, correcting the K deficiency in children with Bartter Syndrome did not always correct growth failure, suggesting other mechanisms are also involved (15). A recent case report of a girl with primary aldosteronism whose growth failure was treated successfully with K supplementation concluded that "serum electrolytes should be included in the evaluation of children with impaired growth" (16).

The hypophosphatemia seen in some tubular disorders results in severe bone changes, bone age delay, and poor growth (9). Good catch-up growth has been reported with P supplementation and normalizing of serum phosphorous (17).

Metabolic acidosis, accompanied by negative Na balance, occurs in numerous tubular dysfunction syndromes such as renal tubular acidosis, and has been identified as an important determinant of FTT (8, 9). Acidosis is known to interfere with major aspects of the GH-IGF axis and ultimately results in the blunting of GH release in children (7). Metabolic acidosis is not a feature of Bartter syndrome.

It is known that successful GH therapy in children with chronic kidney disease (CKD) requires correction of metabolic acidosis and adequate calorie and protein intake (18). GH utilization also requires an adequate nutrient supply (18), and in cases of the various tubular disorders, the supply of minerals, particularly Na, K, Cl, Ca, P and/or Mg is impaired. Inadequate supply of minerals may not only prevent synthesis of IGF, but also impairs growth. Therefore, sufficient supply of the minerals lost by the kidneys may be crucial to providing catch-up growth and resolution of FTT (19, 20).

One of the mainstays of treatment common to the variety of tubular disorders is to provide supplementation of the minerals lost. NaCl, KCl, Ca, P and Mg supplements were all utilized at the initiation of treatment in this case study. Vitamin D also was supplemented, however vitamin D deficiency was a secondary diagnosis not related to Bartter Syndrome. However, it did exacerbate the condition. Also, osteopenia is common with prematurity and may have intensified his Ca and P deficit further. Usual treatment for Bartter Syndrome utilizes indomethacin to treat elevated urinary prostaglandins. Elevated prostaglandins

can lead to suppression of water re-absorption in the collecting duct and NaCl transport in the TAL of the loop of Henle (1). Indomethacin acts to reduce renal excretion of prostaglandin and attenuate the hyperreninism and hyperaldosteronism seen with Bartter Syndrome (1). In addition, indomethacin decreases polyuria, salt wasting and hypokalemia (1).

Finally, appropriate dietary intervention is a part of treatment. Encouraging a diet that is mineral dense, particularly in the minerals that are being wasted is important. The Na restricted diet initially advised at the patient's first clinic visit (i.e. due to the working diagnosis at that time of kidney stones and nephrocalcinosis) was the wrong advice to give in light of the subsequent diagnosis of Bartter Syndrome! Instead, a high Na diet was implemented during the hospitalization and was included in the discharge diet counseling. Indeed, the NaCl supplementation initially provided could be discontinued because the child was able to increase Na content in his diet easily to the amount necessary to normalize his biochemistry. An increased K diet was also encouraged; however, additional KCl supplementation was still required.

## Conclusion

Defects in the re-absorption and/or excessive excretion of minerals and water by the proximal and/or distal renal tubules can lead to FTT in children. This growth failure may or may not be accompanied by inadequate calorie and protein intake.

Pediatric renal dietitians working with children presenting with FTT need to also consider renal tubular dysfunction as a cause of FTT and adjust diet interventions accordingly. By far, most children presenting to a pediatric nephrologist have CKD as a reason for FTT. Strategies for treatment should include correction of acidosis, growth hormone therapy, adequate nutrition and control of Ca and P levels. However, children with renal tubular dysfunction may have no signs of CKD initially, may be eating quite well and yet present with significant FTT. Diet therapy should be aimed at maintaining a higher fluid intake and high mineral diet in these children. ◆

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# If the Media Calls, Are You Ready?

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You're about to break for lunch when you get a call from your organization's public relations office asking if you would be willing to do a last minute TV interview later that afternoon. Will you say "yes" and seize the opportunity to positively portray yourself, and your profession, to the public?

Although we have little to no formal training on media relations, who else should discuss nutrition related topics in the media? For many of us, doing interviews is out of our comfort zone, so we may shy away from them. There is an increased awareness about the increased incidence of chronic kidney disease linked to diabetes and hypertension in many of our communities. Thus, nephrology nutrition professionals and RPG members can – and should – speak up as the experts, instead of allowing unqualified spokespersons to take the lead.

## Working with the media

The "media" include newspapers and magazines, radio and TV, and Internet-based material. There's often overlap, such as when newspaper articles are also posted on the Internet, or when radio interviews become podcasts.

Take a simple approach and start local. Read your community newspaper and watch local television. Offer your congratulations and eventually your own expertise in the future when you read a well-done article on a nutrition topic. Send a note or an email to the health reporter at your local TV/radio station with timely story suggestions. Or call the radio or TV station, ask for the show's producer and pitch your segment idea.

Here's a recent example. I heard a nutrition interview on WGN-AM, Chicago's top radio station. Although the professor did a great job, I noticed the host had to ask her to define what she referred to as "CLA." Although I knew she was referring to conjugated linoleic acid, a naturally occurring fatty acid that may increase the oxidation of fat while slowing fat buildup, the host did not (and neither did the listeners). Sensing an opportunity, I emailed a pleasant note to the host, said how much I enjoyed the interview, and to keep me in mind if he ever wanted a fresh voice. Three weeks later, his executive producer called and booked me for the next day! And guess who is a regular nutrition expert on The Noon Show?

## Preparation

When preparing for a broadcast interview, think about what will be your key messages and "take home" points. Once you've identified them, write them up in the form of questions and send them to the producer. Busy producers will love you because you've made their job easier – and you'll be more comfortable knowing what questions will be asked. Don't forget to also send background information on the subject matter.

Some questions to ask the producer ahead of time include: How long is the interview? Who will conduct it? Will it be live or taped? Will you be sitting or standing?

If possible, get acquainted with the station by watching or listening to the reporter or host. What's her style? If it's a print interview, read the publication and get to know the writer by reading his/her articles.

Remember that the more you prepare the more confident and at ease you will feel – and that will be evident in the interview. Have a colleague or your spouse ask you questions so you can rehearse the responses prior to the interview.

Learn and practice how to use effective communication techniques such as hooking, bridging and flagging by viewing the video referenced in the American Dietetic Association Resources. They really work!

Hooking sets up a question you hope the interviewer will pose. You: "For some people, buying organic is a priority, but during these challenging economic conditions, certain organic foods simply aren't worth the extra expense." (Stop talking.) Interviewer: "Could you give us some examples of what foods you mean?"

Bridging helps you smoothly guide the interview back to your subject. Interviewer (during a segment on practical ways of reducing dietary sodium): "Isn't the medical community conflicted about the role of salt in preventing hypertension?" You: Answer the initial question briefly, and then convey your message. "Although there isn't complete agreement, most physicians recommend lowering the amount of sodium in the diet. One way is to cook more meals at home, rather than eating out."

Flagging highlights your key messages, which alerts the audience to your most important points. Flag by starting out your sentences with, "What renal patients really need to remember when going out to eat is …." or "The most important steps to prevent renal disease are…."

What to wear? Do you have an outfit that brings you compliments every time you wear it? Wear that. Take notice and pay attention to what the national and local TV anchors wear as a guide to appropriate style. The best part of a radio or print interview is that it is done from the comfort of your office, and you can wear anything!

### During the interview

Present your key messages at the beginning. For each point, give examples, specific details, or surprising statistics. Tell a dramatic story about how Medical Nutrition Therapy saved significant money – or someone's life. Give your audience an "ah-ha" moment.

Keep your sentences short. Practice snappy "sound bites." Try to limit your responses to 10 to 20 seconds.
In a print interview, assume anything you say can be used. There is no such thing as "off the record."

### When it's short notice

What if you get a last minute request for an interview? If it's a print interview and they're "on deadline," tell them you'll get back to them in 15 or 20 minutes. Then clear your desk and prepare.
If you are asked to do a last minute broadcast interview, try to accept the opportunity. If you turn it down, they may ask a pseudo-nutrition expert instead!

### Afterwards

You've just conducted an effective interview. Are you done? No, you have one more task to complete. Sit down and compose a handwritten thank you note to the producer and the host. Good manners never go unnoticed or out of style and you will become memorable.

The rest of the world is finally catching up to what we have always known: Food is medicine, and proper nutrition is vital for the prevention and treatment of chronic kidney disease. To be involved with the media you need passion, energy, knowledge and a desire to accept new challenges. You have it all, so why not try it?

### Additional Tips

1. Send press releases about the chronic kidney disease epidemic and what it means to the community to local TV and radio stations and newspapers. Quote yourself. Don't forget to include your contact information.
2. TV is a visual medium, so props and cooking demos make the biggest impact.
3. Watch how the pros conduct interviews on national shows such as Good Morning America, Today and The Early Show.
4. Magazine and newspaper editors and writers change positions or affiliations frequently. Once a great relationship is made, they'll remember you in their next position. ◆

### Resources from the American Dietetic Association

- Working with the Media: A Handbook for Members of the American Dietetic Association. Free to members at: www.eatright.org/media
- Brown D. Becoming a Media-Savvy Registered Dietitian. J Am Diet Assoc. 2006; 106:1163-1164.
- Updates from the ADA Media Spokespersons in the ADA Times
- Video on hooking, bridging and flagging: http://youtube.com/watch?v=1wRa3n8Y0B0

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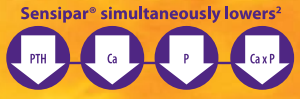


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**References: 1.** Moe SM, Chertow GM, Coburn JW, et al. Achieving NKF-K/DOQI™ bone metabolism and disease treatment goals with cinacalcet HCl. *Kidney Int.* 2005;67:760-771. **2.** Sensipar® (cinacalcet) prescribing information, Amgen.

***Please see brief summary of prescribing information on next page.***

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Sensipar® is contraindicated in patients with hypersensitivity to any component(s) of this product.
**WARNINGS**
**Seizures:** In three clinical studies of CKD patients on dialysis, 5% of the patients in both the Sensipar® and placebo groups reported a history of seizure disorder at baseline. During the trials, seizures (primarily generalized or tonic-clonic) were observed in 1.4% (9/656) of Sensipar®-treated patients and 0.4% (2/470) of placebo-treated patients. Five of the nine Sensipar®-treated patients had a history of a seizure disorder and two were receiving anti-seizure medication at the time of their seizure. Both placebo-treated patients had a history of seizure disorder and were receiving anti-seizure medication at the time of their seizure. While the basis for the reported difference in seizure rate is not clear, the threshold for seizures is lowered by significant reductions in serum calcium levels. Therefore, serum calcium levels should be closely monitored in patients receiving Sensipar®, particularly in patients with a history of a seizure disorder (see PRECAUTIONS, Hypocalcemia).
**Hypotension and/or Worsening Heart Failure:** In postmarketing safety surveillance, isolated, idiosyncratic cases of hypotension and/or worsening heart failure have been reported in patients with impaired cardiac function, in which a causal relationship to Sensipar® could not be completely excluded and may be mediated by reductions in serum calcium levels. Clinical trial data showed hypotension occurred in 7% of Sensipar®-treated patients, 12% of placebo-treated patients, and heart failure occurred in 2% of patients receiving Sensipar® or placebo.
**PRECAUTIONS**
**General**
**Hypocalcemia: Sensipar® lowers serum calcium, and therefore patients should be carefully monitored for the occurrence of hypocalcemia. Potential manifestations of hypocalcemia include paresthesias, myalgias, cramping, tetany, and convulsions**. Sensipar® treatment should not be initiated if serum calcium is less than the lower limit of the normal range (8.4 mg/dL). Serum calcium should be measured within 1 week after initiation or dose adjustment of Sensipar®. Once the maintenance dose has been established, serum calcium should be measured approximately monthly (see DOSAGE AND ADMINISTRATION). If serum calcium falls below 8.4 mg/dL but remains above 7.5 mg/dL, or if symptoms of hypocalcemia occur, calcium-containing phosphate binders and/or vitamin D sterols can be used to raise serum calcium. If serum calcium falls below 7.5 mg/dL, or if symptoms of hypocalcemia persist and the dose of vitamin D cannot be increased, withhold administration of Sensipar® until serum calcium levels reach 8.0 mg/dL, and/or symptoms of hypocalcemia have resolved. Treatment should be re-initiated using the next lowest dose of Sensipar® (see DOSAGE AND ADMINISTRATION). In the 26-week studies of patients with CKD on dialysis, 66% of patients receiving Sensipar® compared with 25% of patients receiving placebo developed at least one serum calcium value < 8.4 mg/dL. Less than 1% of patients in each group permanently discontinued study drug due to hypocalcemia. Sensipar® is not indicated for CKD patients not on dialysis. In CKD patients with secondary HPT not on dialysis, the long-term safety and efficacy of Sensipar® have not been established. Clinical studies indicate that Sensipar®-treated CKD patients not on dialysis have an increased risk for hypocalcemia compared to Sensipar®-treated CKD patients on dialysis, which may be due to lower baseline calcium levels. In a phase 3 study of 32 weeks duration and including 404 subjects (302 cinacalcet, 102 placebo), in which the median dose for cinacalcet was 60 mg at the completion of the study, 80% of Sensipar®-treated patients experienced at least one serum calcium value < 8.4 mg/dL compared to 5% of patients receiving placebo. **Adynamic Bone Disease:** Adynamic bone disease may develop if iPTH levels are suppressed below 100 pg/mL. One clinical study evaluated bone histomorphometry in patients treated with Sensipar® for one year. Three patients with mild hyperparathyroid bone disease at the beginning of the study developed adynamic bone disease during treatment with Sensipar®. Two of these patients had iPTH levels below 100 pg/mL at multiple time points during the study. In the three 6-month, phase 3 studies conducted in CKD patients on dialysis, 11% of patients treated with Sensipar® had mean iPTH values below 100 pg/mL during the efficacy-assessment phase. If iPTH levels decrease below the NKF-K/DOQI recommended target range (150-300 pg/mL)[1] in patients treated with Sensipar®, the dose of Sensipar® and/or vitamin D sterols should be reduced or therapy discontinued. **Hepatic Insufficiency:** Cinacalcet exposure as assessed by $AUC_{(0-inf)}$ in patients with moderate and severe hepatic impairment (as indicated by the Child-Pugh method) were 2.4 and 4.2 times higher, respectively, than that in normals. Patients with moderate and severe hepatic impairment should be monitored throughout treatment with Sensipar® (see CLINICAL PHARMACOLOGY, Pharmacokinetics and DOSAGE AND ADMINISTRATION). **Information for Patients:** It is recommended that Sensipar® be taken with food or shortly after a meal. Tablets should be taken whole and should not be divided. **Laboratory Tests:** ***Patients with CKD on Dialysis with Secondary Hyperparathyroidism:*** Serum calcium and serum phosphorus should be measured within 1 week and iPTH should be measured 1 to 4 weeks after initiation or dose adjustment of Sensipar®. Once the maintenance dose has been established, serum calcium and serum phosphorus should be measured approximately monthly, and PTH every 1 to 3 months (see DOSAGE AND ADMINISTRATION). All iPTH measurements during the Sensipar® trials were obtained using the Nichols IRMA. In patients with end-stage renal disease, testosterone levels are often below the normal range. In a placebo-controlled trial in patients with CKD on dialysis, there were reductions in total and free testosterone in male patients following six months of treatment with Sensipar®. Levels of total testosterone decreased by a median of 15.8% in the Sensipar®-treated patients and by 0.6% in the placebo-treated patients. Levels of free testosterone decreased by a median of 31.3% in the Sensipar®-treated patients and by 16.3% in the placebo-treated patients. The clinical significance of these reductions in serum testosterone is unknown. **Drug Interactions and/or Drug/Laboratory Test Interactions:** See CLINICAL PHARMACOLOGY, Pharmacokinetics and Drug Interactions. Effect of Sensipar® on other drugs: Drugs metabolized by cytochrome P450 2D6 (CYP2D6): Sensipar® is a strong in vitro, as well as in vivo, inhibitor of CYP2D6. Therefore, dose adjustments of concomitant medications that are predominantly metabolized by CYP2D6 and have a narrow therapeutic index (e.g., flecainide, vinblastine, thioridazine and most tricyclic antidepressants) may be required. Desipramine: Concurrent administration of cinacalcet (90 mg) with desipramine (50 mg) increased the exposure of desipramine by 3.6 fold in CYP2D6 extensive metabolizers. Amitriptyline: Concurrent administration of 25 mg or 100 mg cinacalcet with 50 mg amitriptyline increased amitriptyline exposure and nortriptyline (active metabolite) exposure by approximately 20% in CYP2D6 extensive metabolizers. Midazolam: There were no significant differences in the pharmacokinetics of midazolam, a CYP3A4 and CYP3A5 substrate, in subjects receiving 90 mg cinacalcet once daily for 5 days and a single dose of 2 mg midazolam on day 5 as compared to those of subjects receiving 2 mg midazolam alone. This suggests that cinacalcet would not affect the pharmacokinetics of drugs predominantly metabolized by CYP3A4 and CYP3A5. Effect of other drugs on Sensipar®: Sensipar® is metabolized by multiple cytochrome P450 enzymes, primarily CYP3A4, CYP2D6, and CYP1A2. Ketoconazole: Sensipar® is metabolized in part by CYP3A4. Co-administration of ketoconazole, a strong inhibitor of CYP3A4, increased cinacalcet exposure following a single 90 mg dose of Sensipar® by 2.3 fold. Dose adjustment of Sensipar® may be required and PTH and serum calcium concentrations should be closely monitored if a patient initiates or discontinues therapy with a strong CYP3A4 inhibitor (e.g., ketoconazole, erythromycin, itraconazole; see DOSAGE AND ADMINISTRATION). **Carcinogenesis, Mutagenesis, and Impairment of Fertility: Carcinogenicity:** Standard lifetime dietary carcinogenicity bioassays were conducted in mice and rats. Mice were given dietary doses of 15, 50, 125 mg/kg/day in males and 30, 70, 200 mg/kg/day in females (exposures up to 2 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). Rats were given dietary doses of 5, 15, 35 mg/kg/day in males and 5, 20, 35 mg/kg/day in females (exposures up to 2 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). No increased incidence of tumors was observed following treatment with cinacalcet. **Mutagenicity:** Cinacalcet was not genotoxic in the Ames bacterial mutagenicity assay or in the Chinese Hamster Ovary (CHO) cell HGPRT forward mutation assay and CHO cell chromosomal aberration assay, with and without metabolic activation or in the in vivo mouse micronucleus assay. **Impairment of Fertility:** Female rats were given oral gavage doses of 5, 25, 75 mg/kg/day beginning 2 weeks before mating and continuing through gestation day 7. Male rats were given oral doses 4 weeks prior to mating, during mating (3 weeks) and 2 weeks post-mating. No effects were observed in male or female fertility at 5 and 25 mg/kg/day (exposures up to 3 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). At 75 mg/kg/day, there were slight adverse effects (slight decreases in body weight and food consumption) in males and females. **Pregnancy Category C:** In pregnant female rats given oral gavage doses of 2, 25, 50 mg/kg/day during gestation no teratogenicity was observed at doses up to 50 mg/kg/day (exposure 4 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). Decreased fetal body weights were observed at all doses (less than 1 to 4 times a human oral dose of 180 mg/day based on AUC comparison) in conjunction with maternal toxicity (decreased food consumption and body weight gain). In pregnant female rabbits given oral gavage doses of 2, 12, 25 mg/kg/day during gestation no adverse fetal effects were observed (exposures less than with a human oral dose of 180 mg/day based on AUC comparisons). Reductions in maternal food consumption and body weight gain were seen at doses of 12 and 25 mg/kg/day. In pregnant rats given oral gavage doses of 5, 15, 25 mg/kg/day during gestation through lactation no adverse fetal or pup (post-weaning) effects were observed at 5 mg/kg/day (exposures less than with a human therapeutic dose of 180 mg/day based on AUC comparisons). Higher doses of 15 and 25 mg/kg/day (exposures 2-3 times a human oral dose of 180 mg/day based on AUC comparisons) were accompanied by maternal signs of hypocalcemia (periparturient mortality and early postnatal pup loss), and reductions in postnatal maternal and pup body-weight gain. Sensipar® has been shown to cross the placental barrier in rabbits. There are no adequate and well-controlled studies in pregnant women. Sensipar® should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. **Lactating Women:** Studies in rats have shown that Sensipar® is excreted in the milk with a high milk-to-plasma ratio. It is not known whether this drug is excreted in human milk. Considering these data in rats and because many drugs are excreted in human milk and because of the potential for clinically significant adverse reactions in infants from Sensipar®, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the lactating woman. **Pediatric Use:** The safety and efficacy of Sensipar® in pediatric patients have not been established. **Geriatric Use:** Of the 1136 patients enrolled in the Sensipar® phase 3 clinical program, 26% were ≥ 65 years old, and 9% were ≥ 75 years old. No differences in the safety and efficacy of Sensipar® were observed in patients greater or less than 65 years of age (see DOSAGE AND ADMINISTRATION, Geriatric Patients).
**ADVERSE EVENTS**
***Secondary Hyperparathyroidism in Patients with Chronic Kidney Disease on Dialysis:*** In 3 double-blind placebo-controlled clinical trials, 1126 CKD patients on dialysis received study drug (656 Sensipar®, 470 placebo) for up to 6 months. The most frequently reported adverse events (incidence of at least 5% in the Sensipar® group and greater than placebo) are provided in Table 1. The most frequently reported events in the Sensipar® group were nausea, vomiting, and diarrhea.
**Table 1. Adverse Event Incidence (≥ 5%) in Patients On Dialysis**

| Event*: | Placebo n=470 (%) | Sensipar® n=656 (%) | Event*: | Placebo n=470 (%) | Sensipar® n=656 (%) |
|---|---|---|---|---|---|
| Nausea | 19 | 31 | Hypertension | 5 | 7 |
| Vomiting | 15 | 27 | Asthenia | 4 | 7 |
| Diarrhea | 20 | 21 | Anorexia | 4 | 6 |
| Myalgia | 14 | 15 | Pain Chest, Non-Cardiac | 4 | 6 |
| Dizziness | 8 | 10 | Access Infection | 4 | 5 |

*Included are events that were reported at a greater incidence in the Sensipar® group than in the placebo group.

The incidence of serious adverse events (29% vs. 31%) was similar in the Sensipar® and placebo groups, respectively. **12-Month Experience with Sensipar®:** Two hundred and sixty-six patients from 2 phase 3 studies continued to receive Sensipar® or placebo treatment in a 6-month double-blind extension study (12-month total treatment duration). The incidence and nature of adverse events in this study were similar in the two treatment groups, and comparable to those observed in the phase 3 studies. **Postmarketing Experience with Sensipar®:** Rash, hypersensitivity, diarrhea and myalgia have been identified as adverse reactions during post-approval use of Sensipar®. Isolated, idiosyncratic cases of hypotension and/or worsening heart failure have been reported in Sensipar®-treated patients with impaired cardiac function in postmarketing safety surveillance. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure. **Laboratory values**: Serum calcium levels should be closely monitored in patients receiving Sensipar® (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).
**OVERDOSAGE**
Doses titrated up to 300 mg once daily have been safely administered to patients on dialysis. Overdosage of Sensipar® may lead to hypocalcemia. In the event of overdosage, patients should be monitored for signs and symptoms of hypocalcemia and appropriate measures taken to correct serum calcium levels (see PRECAUTIONS). Since Sensipar® is highly protein bound, hemodialysis is not an effective treatment for overdosage of Sensipar®.
**DOSAGE AND ADMINISTRATION**
Sensipar® tablets should be taken whole and should not be divided. Sensipar® should be taken with food or shortly after a meal. Dosage must be individualized. ***Secondary Hyperparathyroidism in Patients with Chronic Kidney Disease on Dialysis:*** The recommended starting oral dose of Sensipar® is 30 mg once daily. Serum calcium and serum phosphorus should be measured within 1 week and PTH should be measured 1 to 4 weeks after initiation or dose adjustment of Sensipar®. Sensipar® should be titrated no more frequently than every 2 to 4 weeks through sequential doses of 60, 90, 120, and 180 mg once daily to target iPTH consistent with the NKF-K/DOQI recommendation for CKD patients on dialysis of 150-300 pg/mL. PTH levels should be assessed no earlier than 12 hours after dosing with Sensipar®. Sensipar® can be used alone or in combination with vitamin D sterols and/or phosphate binders. During dose titration, serum calcium levels should be monitored frequently and if levels decrease below the normal range, appropriate steps should be taken to increase serum calcium levels, such as by providing supplemental calcium, initiating or increasing the dose of calcium-based phosphate binder, initiating or increasing the dose of vitamin D sterols, or temporarily withholding treatment with Sensipar® (see PRECAUTIONS). **Special Populations: Geriatric patients:** Age does not alter the pharmacokinetics of Sensipar®; no dosage adjustment is required for geriatric patients. **Patients with renal impairment:** Renal impairment does not alter the pharmacokinetics of Sensipar®; no dosage adjustment is necessary for renal impairment. **Patients with hepatic impairment**: Cinacalcet exposures, as assessed by $AUC_{(0-inf)}$, in patients with moderate and severe hepatic impairment (as indicated by the Child-Pugh method) were 2.4 and 4.2 times higher, respectively, than in normals. In patients with moderate and severe hepatic impairment, PTH and serum calcium concentrations should be closely monitored throughout treatment with Sensipar® (see CLINICAL PHARMACOLOGY, Pharmacokinetics and PRECAUTIONS). **Drug Interactions:** Sensipar® is metabolized in part by the enzyme CYP3A4. Co-administration of ketoconazole, a strong inhibitor of CYP3A4, caused an approximate 2-fold increase in cinacalcet exposure. Dose adjustment of Sensipar® may be required and PTH and serum calcium concentrations should be closely monitored if a patient initiates or discontinues therapy with a strong CYP3A4 inhibitor (e.g., ketoconazole, erythromycin, itraconazole; see CLINICAL PHARMACOLOGY, Pharmacokinetics and PRECAUTIONS).

**Storage:** Store at 25°C (77°F); excursions permitted to 15-30°C (59-86°F). [See USP controlled room temperature]. Rx Only: This product, or its use, may be covered by one or more US Patents including US Patent Nos. 6313146, 6211244, 6031003 and 6011068, in addition to others, including patents pending.

**References:** 1. National Kidney Foundation: K/DOQI clinical practice guidelines: bone metabolism and disease in chronic kidney disease. American Journal of Kidney Disease 4 2:S1-S201, 2003

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# Nutrition Assessment and Management of Children with Chronic Kidney Disease

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## Epidemiology of Kidney Disease

The most common causes of chronic kidney disease (CKD) in children are obstructive uropathy, renal dysplasia, reflux nephropathy and focal segmental glomerular sclerosis. In the United States, the age breakdown for children with CKD, (excluding dialysis patients) between 1994 and 2006 was 20% infants (n=1,287), 16% toddlers (n=1,031), 32% 6 to 12 years of age (n=2,065) and 28% over the age of 12 years (n=1,788) (1).

The incidence of pediatric end stage renal disease (ESRD) in the United States in 2004 was approximately 15 per million population (2). This number reflects those children who start dialysis. It does not reflect those children who develop kidney failure but do not initiate dialysis or children who develop CKD in adolescence but present with ESRD as young adults (2).

It is estimated that by the year 2010, 650,000 individuals in the United States will have ESRD. Medicare costs are expected to exceed $28 billion per year to care for this population. Many recent studies have demonstrated effective strategies for slowing decline in kidney function, treating complications and improving outcomes. An important goal for the National Institutes of Health is identification of individuals affected in the early stages of CKD (3).

This update on nutrition assessment and management of children with chronic kidney disease will focus on the stages prior to initiation of dialysis. The pediatric registered dietitian (RD) may be involved with patients in the early stages of CKD, prior to management by a pediatric dialysis/transplant center. Pediatric RD's can make important contributions to the prevention and treatment of nutrition issues and improve outcomes of infants and children with CKD.

## CKD Staging

The National Kidney Foundation (NKF) Disease Outcome Initiative (K/DOQI) devised a staging system for CKD. The goals of the system are to establish a common nomenclature for patients and healthcare providers in discussing CKD, for anticipating comorbidities, and for developing treatment plans for progressive kidney disease (Table 1). The NKF recommended the word "kidney" be used instead of "renal" in order to facilitate communication in easily understandable language. There are five different stages of kidney disease based on age, gender, and serum creatinine level. These stages are applicable to children ≥ two years of age. The stages correspond to increasing severity of CKD and

**Table 1**
NKF-K/DOQI Classification of Chronic Kidney Disease (≥ two years of age) (4)

| STAGE | DESCRIPTION | GFR ML/ MIN/1.73 $M^2$ | ACTION |
|---|---|---|---|
| 1 | Kidney damage with normal or increased GFR | ≥90 | Treat primary and comorbid conditions. Slow CKD progression, CVD risk reduction |
| 2 | Kidney damage with mild reduction of GFR | 60-89 | Estimate rate of progression of CKD |
| 3 | Moderate decrease in GFR | 30-59 | Evaluate and treat complications |
| 4 | Severe decrease in GFR | 15-29 | Prepare for kidney replacement therapy |
| 5 | Kidney failure | <15 (or dialysis) | Kidney replacement therapy |

Normal GFR is 133 ±27 mL/min/1.73 $m^2$ for a 2-12 year- old male or female, 126 ±22 mL/min/1.73 $m^2$ for a 13-21 year-old female, and 140±30 mL/min/1.73 $m^2$ for a 13-21 year- old male (4).

declining kidney function as defined by an estimated glomerular filtration rate (GFR). The Schwartz formula is frequently used to estimate GFR in pediatric patients.

**Schwartz Equation**: (4)
GFR (mL/min/1.73m$^2$) = (k x height (cm)/ creatinine (mg/dL))
k= constant (children and adolescent females 0.55, adolescent males 0.70)

Kidney function may begin to decline when clearance is in the range of 50% to 75% of normal. Other kidney functions may cease when the GFR is less than 10 % of normal. However, in children even a small variation in solute and water balance, acid-base status, calcium, and phosphorus can alter growth rate and normal development. Production of renal hormones such as active vitamin D (1,25-dihydroxyvitamin D), and erythropoietin (EPO) may decrease. The kidney can lose the ability to excrete nitrogen waste and the ability to concentrate or dilute fluids or excrete acid load. The consequences of uremia including anorexia, nausea, and decreased sense of taste and smell are major contributors to the poor nutritional intake for many children with CKD (5).

## Nutrition Assessment

Standard assessment parameters including reviewing dietary intake, weight, length or height, weight/height index or body mass index (BMI), head circumference (through 36 months of age), and midarm anthropometry measurements (triceps and subscapular skinfold thickness) have been proposed for nutrition assessment of the child with CKD. The recommended frequency of assessment is every six months, increasing to every one to three months as CKD stage increases. Infants and toddlers will likely require more frequent assessment at all stages of CKD. Fluid overload, as characterized by increased weight, may result in overestimation of nutritional status. In children with significant pubertal delay, comparison to parameters for height age may be preferable (6,7).

## Nutrient Needs

Energy and protein needs may be estimated using standard guidelines according to the Dietary Reference Intake (DRI). The DRI is used as a starting point, and frequent adjustment can be expected due to disease progression as predicted by the stage of CKD (8).

## Special Considerations

***Energy***

Children may require supplemental calories for optimal growth. If a child's oral intake is low, standard recommendations for adding calories and use of modular supplements may be considered. Choice of standard or special formula will depend on the disease and CKD stage. Some children, especially those treated with steroids, may be overweight or obese and not require supplementation.

***Protein***

Protein restriction below the DRI is not recommended in children with CKD due to the deleterious impact on growth. Children with advanced CKD do not tend to choose high protein diets as uremia commonly limits overall calorie and protein intake. If blood urea nitrogen levels are significantly elevated, review protein intake and consider reducing protein if intake exceeds DRI.

***Fluid***

Children's fluid needs should be individually assessed, with goals based on diagnosis and ongoing monitoring of urine output. Urine electrolytes may be helpful in determining mineral losses. Fluid goals may vary greatly and change with progression of kidney disease. For example, a child with renal dysplasia may require two to three times maintenance fluids.

***Sodium***

Children with congenital diseases, such as obstructive uropathy and renal dysplasia, commonly waste sodium. Intravascular volume depletion and poor growth are common until sodium and water supplementation are instituted. Trials of providing fluids of 180-240 mL/kg and sodium supplementation of 2 to 4 mEq (as NaCl or NaHCO3) per 100 mL of hypo-caloric formula demonstrated improved growth rates (9). Conversely, children with glomerular diseases or nephrotic syndrome commonly retain sodium and may benefit from sodium restriction. The edema of uncontrolled nephrotic syndrome can be so severe that mobility may be limited until there is a treatment response. Hypertension is also common in CKD, so a low sodium diet may be indicated.

***Potassium***

Most children will not become hyperkalemic until CKD stage 5. Children with renal dysplasia or obstructive uropathy may experience hyperkalemia in earlier stages of CKD which may respond to salt and water repletion. Correction of metabolic acidosis may also decrease potassium level. In addition to a low potassium diet, lower potassium infant or adult formulas such as Similac PM 60/40® and Suplena Carb Steady® (Abbott Laboratories, Columbus, OH) may be chosen. If problems with hyperkalemia persist, exchange resins may be administered as a medication or added to the formula. However, in addition to

**Table 2**
Nutrients per 100 calories (in order of increasing potassium by category)

| Product | K mg | Ca mg | Phos mg | Na mg | Protein g | Comments |
|---|---|---|---|---|---|---|
| Infant | | | | | | |
| Similac PM 60/40 ®§ | 80 | 56 | 28 | 24 | 2.2 | Mineral reduced |
| Breast Milk | 83 | 40 | 21 | 26 | 1.5 | Preferred |
| Similac ®§ | 105 | 78 | 42 | 24 | 2.07 | Standard formula |
| Good Start ®¶ | 108 | 67 | 38 | 27 | 2.2 | Standard formula |
| Enfamil Lipil ® * | 108 | 78 | 43 | 27 | 2.1 | Standard formula |
| Pediatric | | | | | | |
| Resource Just for Kids 1.5 ®± | 87 | 87 | 66 | 46 | 2.8 | Concentrated |
| Resource Just for Kids ®± | 114 | 114 | 80 | 59 | 3 | Standard formula |
| Kindercal ®* | 124 | 96 | 80 | 35 | 2.8 | Standard formula |
| Pediasure ®§ | 131 | 97 | 84 | 38 | 3 | Standard formula |
| Nutren Jr.®¶ | 132 | 100 | 80 | 46 | 3 | Standard formula |
| Compleat Pediatric ®± | 164 | 144 | 100 | 76 | 3.8 | Standard formula |
| Adult | | | | | | |
| Renalcal ®¶ | 0 | 0 | 0 | 0 | 1.7 | Incomplete<br>Short-term use |
| Novasource Renal ®± | 40 | 65 | 32 | 44 | 3.7 | Contains medium chain triglyceride oil |
| Nepro Carb Steady ®§ | 59 | 59 | 39 | 59 | 4.5 | Contains fiber |
| Suplena Carb Steady ®§ | 62 | 59 | 39 | 44 | 2.5 | Contains fiber |
| Nutren Renal ®¶ | 63 | 70 | 35 | 37 | 3.5 | Contains medium chain triglyceride oil |

§ Abbott Laboratories, Columbus, Ohio ¶ Nestle Nutrition, Glendale, California
* Mead Johnson, Evansville, Indiana ± Novartis Nutrtion, Minneapolis, Minnesota

reducing the potassium content of the formula, other minerals are reduced and the sodium load is increased. Alternatively, short term use of a formula without potassium such as Renalcal® (Nestle Nutrition, Glendale, CA) can be considered while waiting for dialysis to start. A summary of commonly used formulas and their respective potassium content is shown in Table 2.

***Iron***

Anemia associated with the inadequate production of EPO is frequently seen by CKD stage 4. Treatment with EPO and iron has been a major advance in the treatment of children with CKD. Epoetin alfa (Epogen®, Amgen, Thousand Oaks, California) is given via frequent injections. Darbepoetin alfa (Aranesp®, Amgen, Thousand Oaks, California), is a newer option requiring less frequent injections.

## Calcium, Phosphorus, Vitamin D, and Parathyroid Hormone

Children with CKD are at very high risk for poor bone health. The K/DOQI guidelines provide recommendations for management of pediatric bone disease based on kidney disease stage (10). Calcium, phosphorus, calcium phosphorus product,

**Table 3**
Signs and Symptoms of Bone Disease in Children with CKD (10)

- Hypocalcemia
- Hyperphosphatemia
- Altered vitamin D metabolism
- Secondary hyperparathyroidism
- Bone pain
- Linear growth failure
- Fractures
- Avascular Necrosis
- Delayed skeletal maturation and epiphyseal closure
- Slipped epiphyses
- Skeletal deformities resembling Vitamin D-deficient rickets
- Progressive muscle weakness, waddling gait
- Soft-tissue calcification of blood vessels, lung, kidney, myocardium, coronary arteries, and cardiac valves

Vitamin D, and parathyroid hormone (PTH) should be assessed.

***Calcium***

Intake and excretion of calcium are decreased in children with CKD. If 1,25- dihydroxyvitamin D is low, hypocalcemia can be severe. Serum PTH will increase in response to hypocalcemia, calcium will be mobilized from bone, and renal osteodystrophy will result. Hypercalcemia can occur due to excess vitamin D or excess calcium supplements resulting in unwanted side effects including soft tissue calcification.

***Phosphorus***

Prevention of renal osteodystrophy requires careful attention to phosphorus control. It is important to use age-specific phosphorus levels. Infants maintained on a low-phosphorus formula may require phosphate supplementation to maintain normal serum phosphorus levels. In older children and adolescents, a low phosphorus diet and phosphorus binding medication may be needed to reduce the amount of phosphorus absorbed. Phosphorus management becomes more difficult as CKD progresses. The calcium-based supplements given to correct hypocalcemia, such as calcium carbonate or calcium acetate, can also provide phosphorus binding. Calcium citrate is avoided due to aluminum concerns with kidney disease. Two non-calcium phosphorus binders, Sevelamar Hydrochloride (Renagel®,Genzyme, Cambridge, Massachusetts) and Lanthanum Carbonate (Fosrenol® Shire Pharmaceuticals, Wayne, Pennsylvania) may be beneficial if the child is unable to use calcium-based binders due to hypercalcemia.

***Calcium Phosphorus Product***

K/DOQI guidelines recommend monitoring the calcium phosphorus product (i.e., serum calcium x serum phosphorous level) in order to minimize the risk for soft tissue calcification. Children less than 12 years old should have a product of < 65 $mg^2/dL^2$, while the older child should have a product <55 $mg^2/dL^2$. Achieving these targets may involve alterations in diet and/or calcium supplements, vitamin D, and phosphorus binding medications.

***Vitamin D***

It is recommended to monitor and treat serum vitamin D levels <30 ng/mL with ergocalciferol in CKD stages 2 to 4 if the PTH level is above target. PTH levels warranting treatment are >70 pg/mL for CKD stages 2 and 3, and >110 pg/mL for CKD 4. The active form of vitamin D (calcitriol) should be provided in stage 5 if the PTH level is above 300 pg/mL. The active form of vitamin D is used as the kidney's ability to convert adequate levels of inactive vitamin D to active vitamin D is markedly reduced or absent in CKD stage 5.

## Monitoring Bone Health

The signs, symptoms, and undesired outcomes of bone disease in pediatric CKD are outlined in Table 3 (10). Acidosis may impact bone health and is a major component of the linear growth failure associated with CKD. It is recommended that serum $CO_2$ levels be maintained at ≥20 mEq/L for infants through two years of age and ≥22 mEq/L for children over two years of age (10).

Although much is written about the utility of dual-energy X-ray absorptiometry (DEXA) in various childhood disorders, there is no data to support the utility of DEXA to evaluate bone status of children with CKD. The DEXA evaluation will underestimate the bone density of shorter individuals. The comparison of a chronically ill child of short stature to a child of equivalent height age will result in comparison to a younger, less mature control and inaccurate conclusions (10).

***Vitamin/Mineral Supplements***

Standard vitamins may be needed if oral intake is inadequate. Analysis of usual intake in comparison to the DRI's will provide the basis for recommendations. Excess vitamin A should be avoided due to the potential for toxicities including hypercalcemia. Serum vitamin A along with retinol binding protein levels can be monitored to assess vitamin A status. Excess vitamin C supplementation should also be avoided due to the potential for oxalosis. Serum zinc levels have been found to be low in children with CKD; monitoring and supplementation of zinc

should be considered. Special vitamins are available that include only B vitamins and vitamin C such as Nephro-vite® (Watson Laboratories, Corona, California), Dialyvite® (Hillestad Pharmaceuticals, Woodruff, Wisconsin), and Nephronex liquid® (Llorens Pharmaceuticals, Miami, Florida). These vitamins, formulated for adults, were designed to replace losses associated with dialysis. Pediatric versions of these vitamin supplements remain under study. The vitamin chosen will depend upon the child's current oral intake and clinical status. Partial dosing of pediatric or adult vitamins may be considered in order to avoid providing excess vitamin A.

## Growth

Children with CKD diagnosed in infancy, or in childhood prior to achieving adult height, will likely be of short stature. Growth hormone has helped many children achieve a normal height, even within genetic potential. The formula for calculating mid-parental height, which may indicate height potential, is presented in Table 4 (8).

**Table 4**

Midparental Height Calculation (8)

**Males:**

$$\frac{\text{Paternal Height (cm)} + \text{Maternal Height (cm)} + 12\text{ cm}}{2}$$

**Females:**

$$\frac{\text{Paternal Height (cm)} + \text{Maternal Height (cm)} - 12\text{ cm}}{2}$$

Consideration of growth hormone should be included in the nutrition evaluation. Loss of height velocity can be detected by determining rate of growth. Prior to initiation of growth hormone, correction of insufficient energy and protein, acidosis, salt-wasting, hyperphosphatemia, and secondary hyperparathyroidism should be accomplished (10). Growth hormone is especially effective if given to toddlers before the age of two years and is most effective within the first year of use. In one study the improvement in height was found to be 10.7±3.1 vs. 6.5±2.6 cm/year for the first year and 7.8±2.1 vs. 5.5± 1.9 cm/year for the second year (11). Growth hormone, which is administered by injection, is covered by most insurance companies when a diagnosis of CKD with growth failure has been documented.

## Cardiovascular Issues

The prevalence of hypertension in children and adolescents is increasing. The development of cardiovascular disease and renal disease later in life is associated with childhood hypertension. Forty-nine percent of children with CKD have hypertension (12). In addition, in the general population, it is estimated that 5% of 10 to 19 year-old children are hypertensive, and it is estimated that 11% of obese children are hypertensive (13). Early identification is essential to minimize the long-term health effects which include kidney failure. Treatment options include a low sodium diet and lifestyle modifications including aerobic exercise and weight loss, if indicated. Family–based intervention is recommended with hopes of increasing likelihood of adherence.

Guidelines for prevention of cardiovascular disease are available for high-risk pediatric patients including those with CKD 5. These guidelines include recommendations for modification of dietary fat, activity goals, and weight loss if BMI is above the 95th percentile (12-14).

## Special Enteral Nutrition

Formula supplementation is often necessary to meet energy and protein goals. The oral route is attempted initially. Feeding therapy is indicated if the infant or child is not achieving age-appropriate feeding skills. If oral intake remains inadequate, naso-gastric and/or gastrostomy feedings are initiated. Permanent feeding tubes are often needed to meet nutrient goals. Jejunostomy feedings may be helpful for infants or children with chronic vomiting not improved by medical management. Nissen fundoplications are occasionally performed due to chronic vomiting.

The choice of formula used will depend upon clinical status, diagnosis, and CKD stage. Infants commonly use Similac PM 60/40® (Abbot Laboratories, Columbus, Ohio) a mineral-reduced formula appropriate for conditions such as hyperkalemia, hypercalcemia, or hyperphosphatemia. Iron supplementation is indicated as this formula is low in iron. Other infant formulas, such as Good Start® (Nestle Nutrition, Deerfield, Illinois) may be appropriate. Good Start® has levels of calcium and phosphorus between those of Similac PM 60/40® and other commonly used infant formulas such as Enfamil Lipil with iron® (Mead Johnson Nutritionals, Evansville, Indiana) and Similac with iron® (Abbott Laboratories, Columbus, Ohio).

Older children may tolerate a standard pediatric formula and transition to an adult kidney formula as CKD stage advances. The adult kidney products are used in young children with varying degrees of success. The adult formulas are up to twice as concentrated as standard pediatric formulas at 53 to 60 calories per ounce. Suplena Carb Steady® (Abbott Laboratories, Columbus, Ohio) is the only complete low protein kidney formula. Suplena

Carb Steady® is more convenient than the infant powders and is lower in potassium, but it may provide more protein than desired. Renalcal® (Nestle Clinical Nutrition, Deerfield, Illinois) is also low in protein but nutritionally incomplete. Of the higher protein kidney formulas, Novasource Renal® (Novartis Nutrition, Minneapolis, Minnesota) and Nutren Renal® (Nestle Clinical Nutrition, Deerfield, Illinois) may be better tolerated by those sensitive to the fiber in Nepro Carb Steady®. Tolerance is best achieved by gradual introduction and initiation at a caloric concentration typically tolerated by children of a similar age or weight.

Use of carbohydrate, protein and fat modulars may be necessary to achieve desired levels of nutrients. Standard products such as Polycose® (Abbott Laboratories, Columbus, Ohio), Beneprotein® (Novartis Nutrition, Minneapolis, Minnesota) and Duocal® (Nutricia North America, Rockville, Maryland) as well as oils or Microlipid® (Novartis Nutrition, Minneapolis, Minnesota), may be used depending upon the child's needs.

## Summary

Nutritional care of the pediatric patient with CKD requires frequent assessment and an interdisciplinary approach. Parents of young infants and children have many demands placed upon them. Adolescents often have difficulty adhering to the nutrition and medication regimen. The pediatric RD is a key member of the multi-disciplinary team working with the family and child to improve nutritional status and growth in preparation for the next stage in the continuum of chronic kidney disease. ◆

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# A Renal Nutrition Forum Series with Practice-Based Examples of the Nutrition Care Process (NCP)

**Maureen McCarthy, MPH, RD, CSR, LD & Stacey Evert, RD, CNSD, LD**

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Helpful reviewer comments on the case study that appeared in our previous column made it clear that this series has yet to address an important topic related to Standardized Language (SL): How should the International Dietetics and Nutrition Terminology Reference Manual (IDNT Reference Manual) be used in integrating SL into documentation of patient care (1)? Sure, it provides the lists of terms, but does the manual have more to offer than that? This article will discuss how to use the worksheets in the manual.

Before implementing the NCP and SL, it is critical to prepare for change. Helpful tools and references for navigating the process of change are found on the web pages devoted to NCP, as mentioned in the first article in the series (2). These pages are available with American Dietetic Association (ADA) membership login at http://www.eatright.org/cps/rde/xchg/ada/hs.xsl/home_13848_ENU_HTML.htm (accessed 7-1-08).

Following careful preparation for change, other activities key to successful incorporation of SL into daily practice revolve around practice, practice, practice:

- Find opportunities to discuss case studies and how SL may be used in documentation.
- Use the IDNT Reference Manual regularly to clarify the meanings of terms (1).

## Opportunities to Discuss Case Studies

In my experience, it can be hard to implement SL in a solitary setting. Take advantage of interactions with other dietitians on the job or in professional group meetings to work on case studies. Use regular meetings in the work setting, at local Council on Renal Nutrition (CRN) meetings, at dietetics association and/or practice group meetings, and other venues to practice SL documentation.

At Oregon Health & Science University (OHSU), dietitians on the Nutrition Service staff meet for at least 30 minutes at one meeting a month to practice SL. Each dietitian presents an informal discussion of a case study, with a focus on finding an accurate nutrition diagnosis. Our goals are: 1) to develop the thinking skills needed to write diagnostic statements (problem-etiology-signs/symptoms, or PES, statements), 2) to work through some situations where clinicians have a difficult time finding the correct term in the SL for nutrition diagnosis, and 3) to make sure we use terms as defined in the IDNT Reference Manual. Anytime there are group discussions of case studies and how to use SL, the IDNT Reference Manual should be at hand to look up terms, making sure they are used as intended.

The discussion usually follows the NCP quadrants, beginning with assessment data; then the diagnostic or PES statement; and then intervention and monitoring & evaluation. Then the group of dietitians considers the diagnosis and PES statement: Do they accurately describe the major nutrition problem for the case under discussion? Again, this is where consultation with the IDNT Reference Manual is helpful.

## Use of the IDNT Reference Manual

This manual includes worksheets that define each term and provide examples of how they might be used. In regard to nutrition diagnosis terms, the manual provides:

- The "superbill" listing the Nutrition Diagnostic Terminology (page 39)*
- An index of the terms and their definitions (pages 40-42)*
- A 2-4 page worksheet for each of the 60 terms, which includes the definition, possible etiologies, and possible signs and symptoms for each diagnosis. Additionally, the worksheets include references, which in my opinion, are another valuable feature of the manual.

## Practice Sessions with Other Dietitians

The OHSU dietitians recently considered this case study involving a patient who was being readmitted after a recent stem cell transplant. Presenting symptoms included nausea, vomiting, and diarrhea significant enough to lead to a hospital admission. Despite these symptoms, weight was stable and admitting labs were also stable and within accepted ranges. (Although this is not a nephrology case study, the discussion that followed developed several major themes which are universal in general practice and in specialty areas.) The dietitian presenting the case had prepared the PES statement shown on Table 1.

Themes of the discussion included:

1) Is it appropriate to use a diagnosis, such as graft-versus-host-disease (GVHD), as the etiology in a nutrition diagnostic statement? The reference manual, in a discussion of critical thinking to develop clear PES statements, says the etiology should be "the root cause or the most specific root case that

**Table 1**
Components of PES Statement

| | Definition | Example (P...E...S) |
|---|---|---|
| **P** | **Problem:** the actual nutrition diagnosis, taken from the standardized terms for nutrition diagnosis. Terms from the "intake" domain should be considered first. | Inadequate dietary protein *related to* |
| **E** | **Etiology:** the cause of the diagnosis, or factors contributing to the diagnosis | Knowledge deficit about dietary protein needs for dialysis patients *as shown by* |
| **S** | **Signs and symptoms:** defining characteristics of the diagnosis which will be monitored to evaluate outcomes. | Patient still following meal plans for 45-50 g protein/day |

the RD can address with a nutrition intervention" (1). Many speakers, including one of the authors in this series, have advised that the etiology should not be a medical diagnosis, but should be directly amenable to nutrition intervention. For the diagnosis of "altered GI function" the IDNT Reference Manual lists several etiologies, including: alterations in GI anatomical structure, changes in GI tract motility, compromised GI function (which seems to include GVHD), compromised function of related GI organs, and decreased functional length of the GI tract (1). This seems to suggest that the PES statement as presented in our group discussion met broad guidelines for constructing these statements.

2) Is there a nutrition diagnosis term from the Intake domain that applies to this case? Nutrition diagnosis terms are organized in three domains: Intake, Clinical, and Behavioral. Notably, the reference manual advises "When all things are equal and there is a choice between…. nutrition diagnoses from different domains, consider the Intake nutrition diagnosis as the one more specific to the role of the RD" (1).
This leads to another possible PES statement for the case:
**Problem (or Diagnosis): Inadequate protein-energy intake related to**
**Etiology: altered GI function, as shown by**
**Signs and symptoms: patient report of minimal intake for 3 days prior to admission.**

3) Is there ever a single correct diagnosis and/or PES statement? This question comes up at every discussion of this topic. In my opinion, most often there is no single correct diagnosis or statement for a given clinical scenario. There can be more than one correct nutrition diagnosis because there will always be different approaches at the individual patient care level. Still, SL will serve to tighten up our documentation language so that we can describe what dietitians do more consistently and succinctly. The true test will be the links between diagnoses, interventions and outcomes in large groups. SL is simply a tool to allow us to study and report on those links when we look at larger numbers of cases.

4) Can there be more than 1 diagnosis in a given scenario? Yes, there may be more than 1 diagnosis. But it is essential to have data to support each diagnosis and it is essential to follow through with interventions and outcomes for each diagnosis. My personal preference has always been to focus on one diagnosis at a time, assuming it is safe for the patient to wait to address additional diagnoses after the first problem has been addressed.

## Applications in Nephrology Nutrition

The case study in our previous column described a new peritoneal dialysis (PD) patient with poor glycemic control upon starting PD (for full details, see the Summer 2008 issue of Renal Nutrition Forum). This was due to lack of education as the patient initiated PD (3). In the draft of the column that went out to reviewers, the PES statement was:
**Problem (or Diagnosis): Altered nutrition-related labs due to**
**Etiology: dextrose load of PD, as shown by**
**Signs and symptoms: elevated glucose and A1c since starting PD.**

The reviewers reminded the authors that the Intake domain is preferred, and this made good sense in this case study. So the PES statement was revised in the copy that went to press to state:
**Problem (or Diagnosis): Excessive carbohydrate intake related to**
**Etiology: lack of education to date regarding how to adjust for dextrose load of PD, as shown by**
**Signs and symptoms: elevated glucose and A1c since starting PD.**

Incorporating SL into practice is an on-going process, and it is always helpful to receive input from others, as this episode demonstrates. Using a diagnosis from the Intake domain acknowledges our unique clinical expertise in diagnosing problems with dietary intake.

### What Lies Ahead?

So far at OHSU, the monthly case studies have been devoted to nutrition diagnostic terms and statements. We are busy developing the nutrition components for our new electronic medical record (EMR) system, for which the SL is very helpful in this monumental task. Dietitians in the renal community may also find the SL an essential tool in designing comprehensive nutrition components for EMR documentation.

By the time this article is published, we will have the interpretive guidelines for the new Conditions for Coverage (CfC) that were announced in April 2008. Implementation of the assessment and care plan mandates in the updated CfC will be an excellent opportunity to incorporate SL into nephrology nutrition.

*NOTE: The next edition of the IDNT Reference Manual will be published in Fall 2008 and page numbers will change. ◆

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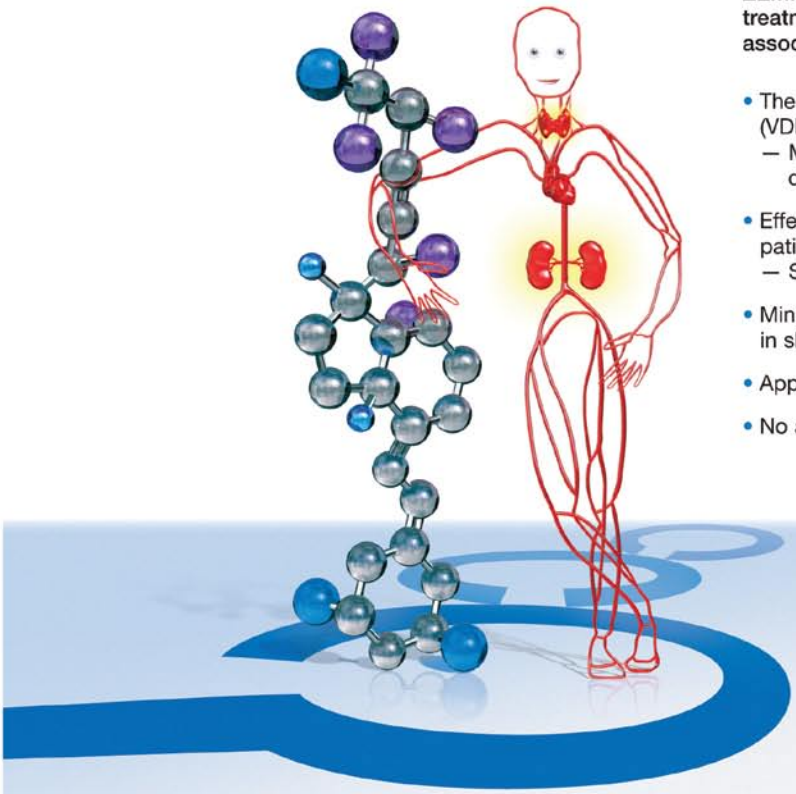

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# ADA Committee Brings Nutrition Coding and Coverage to the Forefront - A Message from ADA

Regardless if you are a registered dietitian (RD) working in a private practice, a hospital, nursing home or other practice setting, the members of the Coding & Coverage Committee (CCC) are some of your biggest advocates. The committee's goal – to empower RDs to expand nutrition services coverage and receive competitive reimbursement-- impacts all RDs. If you've ever wondered what the American Dietetic Association (ADA) is doing about reimbursement or coverage for nutrition services (and the problematic lack of), know that some of the most business savvy RDs in the association are working hard to develop strategies for increasing RD recognition and coverage.

The CCC is a small but passionate group of RDs who envision a financially bright future for the dietetics profession. Members of the CCC come from a variety of work settings and are appointed by the ADA President-elect to a two or three-year term. Their focus is on both internal and external stakeholders in the nutrition reimbursement arena. During their tenure, they are charged with planning for future nutrition code development and maintenance; educating members on coding and coverage, and creating strategies to promote recognition and the benefits of RD-provided nutrition services.

Former ADA President Susan Laramee, MS, LDN, RD, formed the CCC in 2005 upon recognizing a crucial need within ADA to strategically plan and monitor coding and coverage activities. Since its formation, the CCC has already made significant strides; from educational outreach and awareness efforts at FNCE and affiliate meetings, to third party payer outreach and physician recognition of RD-provided services.

Outgoing committee chair, Keith Ayoob, EdD, RD, FADA serves as a liaison to the American Medical Association's (AMA) Current Procedural Terminology Health Care Professional Advisory Committee (CPT HCPAC). This allows him to participate in the development of CPT (procedure) codes for RDs through the AMA process. "It is a very arduous process," said Ayoob, "but constant presence at such meetings reinforces the value of registered dietitians in the coding and coverage process."

Also representing ADA within the AMA on national coding activities is Jane White, PhD, LDN, RD, FADA, an ex-officio member of the CCC. White is the liaison to the AMA's Relative Value Scale Update Committee (RUC), which has the ability to influence code values, or the rate at which codes, such as the MNT codes, are reimbursed by the Centers for Medicare & Medicaid Services. White sees coding and coverage as the "lifeblood" of the RD profession. "It is important that ADA be present at the AMA coding related meetings so that all members of the health care team are exposed to RDs and understand the vital nature of the work that we do to optimize the nutritional status, health and well-being of the American public," said White. "We must be at the table when the discussions of health care and nutrition services delivery occur so that we can have input into the decisions that impact services provision, code creation and services reimbursement."

Due to ADA's involvement in AMA coding meetings, new codes for team conferences, phone and email services, and education and training are available for RDs. Depending on payer policies, these codes may be used among private insurance companies. White and Ayoob's appointments to the HCPAC ensure ADA will have a strong voice among many competing interests in the health care field.

In addition to these external groups, members of the CCC work closely with internal ADA groups to advance opportunities for RDs. For example, CCC members contributed to the development of performance measures for outcomes reporting to the government and insurance groups. The CCC also funded and is involved with the MNT Effectiveness Evidence-Analysis Workgroup to review research on cost-savings of RD-provided clinical nutrition services and the value and impact of RD nutrition services compared with services provided by other healthcare professionals. (Information will soon be posted on the Evidence Analysis Library.) In addition, members participate on the Evidence-Based Practice Committee and helped draft the August 2008 *Journal of the American Dietetic Association* article, "Referral Systems in Ambulatory Care– Providing Access to the Nutrition Care Process."

During the past year, committee members have reached out to the larger ADA membership in many ways. At affiliate meetings across the country, hundreds of ADA members attended a "Cracking the Code- Billing Potential Beyond Medical Nutrition Therapy" seminar presented by the CCC members. Thousands of ADA members received email requests to participate in an ADA 2008 CPT Code Survey, requests to participate in an ADA/AMA Practice Information survey, and requests to participate in a phone and email procedure code survey, all CCC projects. Also during the year, thousands of ADA members downloaded the "MNT Works Kit" and marketing materials on "How to sell MNT message" to use to expand coverage with local insurance companies. All this, and more, was made possible through the hard work and dedication of the CCC members.

According to Ayoob, the CCC's success during 2007-2008 can

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be attributed largely to the unique perspective and passion that each member brought to the table. Looking ahead, 2008-2009 Chair Charlotte Thiessen, MS, RD, LMNT, hopes to add to the strong foundation by expanding the educational "Cracking the Code" presentation schedule and developing strategies for increasing RD recognition and coverage among private sector third party payers. "The work of the coding committee is critical to the profession," said Thiessen. "Assisting RDs on interacting with insurance companies and understanding code use and billing procedures for nutrition services is fundamental to ensure MNT remains an effective and covered benefit."

To learn more about the Coding & Coverage Committee and to view member coding and coverage resources go to www.eatright.org/mnt. ◆

## 2009 ADA Public Policy Workshop
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Information and registration information are available on the ADA website under the Advocacy and the Profession tab.

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# Member Spotlight

**Ruth Kruk, MS, RD, LDN**
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The 2008 Spring Meeting of the National Kidney Foundation (NKF) was held in Grapevine, Texas at the fabulous Gaylord Texan Hotel and Convention Center. On the day of the "Strategies 2 – Applying the Principles" pre-conference workshop, my flight was unfortunately cancelled. I arrived in the afternoon and heard that I missed an excellent study of the Nutrition Care Process for standardized nutrition diagnosis language, which is something we all need to learn and embrace.

My favorite lecture was "Motivational Interviewing" by Dana Sturtevant, MS, RD, which was an outstanding discussion of motivational interviewing. She described using motivational interviewing as making encounters feel more like dancing than wrestling. The interaction between the provider and the patient powerfully influences patients' resistance or adherence, and one should never underestimate the power of relationships.

Patients who express a motivation to change are more likely to demonstrate "change talk." People who argue against change are less likely to change, which is known as "resistance language." Affirm your clients by thanking them for talking to you, and look at things from the patients' perspective. Encourage patients to present their own arguments for change, which helps them resolve their ambivalence. Also ask patients to list the benefits of making a change.

One should not feel like they are working harder than the patient. Open the conversation, provide feedback in a non-judgmental way, use "open ears," and close the conversation. Open ears are open-ended questions which are used to seek permission before you start a conversation. Listen for two minutes without interrupting, take note of the flow of the conversation, and nurture confidence. Encourage, empathize, affirm and elicit change talk. Summarize what you heard the patient say. Listen with presence-individualized attention using all of your senses. Opportunities to improve our communication skills are always rewarding. Motivational interviewing techniques provide greater opportunities to improve many aspects of our lives and those around us.

Each attendee arrives at NKF with a different set of expectations and growth plans. It is very hard to decide which session to choose because many of your interests can be scheduled concurrently. Unfortunately, I was closed out of several I had hoped to see, but still learned more than I could remember. I do remember the 'heavy suitcase fine' on my return flight and did forget to tell my husband about it!

Since the trip, I've read through most of the NKF presentations, many of which are available online to those who attended the conference. It was great to go through them and pick up what I missed, as well as view references from the slides that I couldn't read during the lectures. NKF had a lot to offer, and I hope to attend more often in the future. Next year, the meeting will be at a Gaylord Hotel in Nashville, Tennessee, so start making your plans to attend in 2009! ◆

# Renal Dietitians Chair Message

**Pamela S. Kent, MS, RD, CSR, LD**
RPG Chair

"The only thing we know about the future is that it will be different." Peter Drucker

The landscape of the nephrology community is constantly changing. The focus of our clinical practice has expanded to the diagnosis, treatment and complications of chronic kidney disease (CKD) well before a patient reaches dialysis, as well as adhering to new legislative requirements for CKD patients.

As you are already aware, the end stage renal disease (ESRD) Conditions for Coverage were published April 15, 2008. These guidelines set the minimum health and safety rules that all Medicare and Medicaid participating dialysis facilities must meet. The revised regulations are patient-centered, reflect improvements in clinical standards of care, the use of more advanced technology, and, most notably, a framework to incorporate performance measures viewed by the scientific and medical community to be related to the quality of care provided to dialysis patients.

The Interpretive Guidelines were available for public comment during late summer. The Renal Dietitians (RPG) leadership reviewed this document and submitted comments regarding various tag numbers of the guidelines to Mary Hager, PhD, RD, FADA, in Regulatory Affairs from the ADA Washington office, who submitted the final comments to Centers for Medicare and Medicaid Services (CMS). The RPG leadership felt it was imperative to step up to the plate and address any critical issues impacting the future of the registered dietitian and our profession.

Speaking of stepping up to the plate, Karen Basinger, RD, LD, has represented RPG on the legislative and reimbursement front for several years. Karen was awarded the RPG Outstanding Service Award, which is the highest designation a member can receive within RPG. Karen was selected due to all of her support representing RPG with CMS, MNT efforts and National Kidney Disease Education Program (NKDEP) activities. The publication of the proposed rules on dialysis units was postponed until February, 2009 largely due to Karen's comments. Karen also represented renal dietitians at the annual ADA Food & Nutrition Conference & Expo (FNCE) in Chicago by attending the NKDEP discussion group on October 25th, 2008. This focus group discussed the role dietitians can play in CKD diet education.

The RPG keynote priority session speaker during FNCE was Marcia Kalista Richards, MPH, RD, CNSD, LDN, who spoke on the Nutritional Management of the Patient with Acute Kidney Injury and CKD. The session highlighted clinical management of CKD. In addition, the RPG hosted the first pre-FNCE workshop on medical nutrition therapy (MNT). The RPG leadership chose both of these programs to promote awareness of CKD and MNT. I hope those of you who attended the RPG FNCE Member reception and dietetic practice group showcase had a chance to meet the Executive Committee and take advantage of the networking opportunity.

The time is now to get involved. Please feel free to contact me with any comments. PamkentRD@yahoo.com

"The world is a dangerous place, not because of those who do evil, but because of those who look on and do nothing." A. Einstein ◆

## 2009 Board Certification for a Specialist in Renal Nutrition

http://www.cdrnet.org/certifications/spec/renal.htm

| Next Specialty Examination Dates | Application Deadlines (postmark) |
|---|---|
| May 4-22, 2009 | March 9, 2009 |
| November 2-20, 2009 | September 4, 2009 |

**To become a Board Certified Specialist in Renal or Pediatric Nutrition, you must first complete the eligibility application and successfully meet the following minimum criteria:**

- Current Registered Dietitian (RD) status with CDR.
- Maintenance of RD status with CDR, for a minimum of 2 years from original examination date (by the time of the specialty examination date).
- Documentation of 2,000 hours of practice experience as an RD in the specialty area within the past five years (by the date the application is due). Please note: certain education and professional experiences can be used as a substitution for the required 2,000 specialty practice hours up to a maximum of 40% (800 hours).

# CRN Chairperson Message

## Conditions for Coverage Released – Implications for Nephrology Dietitians

**Maria Karalis, MBA, RD, LDN**
CRN Chair

The Centers for Medicare and Medicaid Services (CMS) released the final rule of the Conditions for Coverage on April 4, 2008. These are "rules" that dialysis facilities must follow in order to be Medicare certified in the United States. This is the first update to these rules since 1976. The ESRD final rule (116 page document) can be found on http://www.cms.hhs.gov/center/esrd.asp. This final rule focuses on "the patient and the results of care provided to the patient, establishes performance expectations for facilities, encourages patients to participate in their plan of care and treatment, eliminates many procedural requirements from the previous conditions for coverage, preserves strong process measures when necessary to promote meaningful patient safety, well-being, and continuous quality improvement."

The previous rules defined a "qualified dietitian" as a Registered Dietitian (RD) with the Commission of Dietetic Registration and required that the RD meet the practice requirements of the state in which he/she is employed. The new rules include an additional requirement that the dietitian also must have a "minimum of one year of professional work experience in clinical nutrition as a RD in order to qualify to perform the special responsibilities of renal dietitians…." The National Kidney Foundation's Council on Renal Nutrition (NKF CRN) and the American Dietetic Association (ADA), in consultation with the Renal Practice Group, supported this definition as it is consistent with quality standards for dietetic practice.

Last October, CMS released a draft of the interpretative guidelines for the new conditions for coverage. Interpretative guidelines provide guidance to state surveyors in understanding CMS' final rules. Many key stakeholders, including the NKF CRN and ADA, commented on the draft interpretative guidelines. CRN's comments can be found under the member's only section of the NKF website.

Plans are underway to educate nephrology dietitians on the newly released rules and the final interpretive guidelines before their release later this year. Education will focus on all of the major changes impacting RD's, including the Comprehensive Multidisciplinary Patient Assessment (CMPA). The CMPA replaces the requirement for individual assessments by each discipline (ref: § 494.80).

Last year CMS encouraged the NKF and the American Nephrology Nurses Association (ANNA) to establish a task force to develop resources and guidelines to assist facilities in complying with the proposed requirement for a CMPA. CRN participated in this task force. Education will be forthcoming on examples of questions that facilities and dietitians may want to use to meet the requirements for a CMPA. Look for more information in your email inbox from the subject line "CRN Announce."◆

**Do you have an educational handout that you have created and would like to share with others?**

The editorial team is seeking professional resources to compile in an upcoming "electronic only" supplement to the Renal Nutrition Forum. If you are interested, please contact assistant editor Stacey C. Phillips, RD (phillipsc@trinity-health.org) by March 1, 2009.

Congratulations to
**Stacey C. Phillips, RD**
on your appointment of
RNF Assistant Editor!
Welcome to the editorial team!

## ERRATA

Please note the following mis-printed items in the Summer 2008 Renal Nutrition Forum

***Hidden Phosphorus-Feature Article***

- Page 9, Table 3 Footnote addition: SM* (several manufacturers) & AM** (all manufacturers)

***Nutrition Care Process Article***

- Page 22, column 2, CAPD prescription under Clinical Data, should read, "CAPD prescription 4 – 2.5% 2 Liter exchanges qd."
- Page 23, column 2, under Monitoring and Evaluation Carbohydrate intake should be "5-6 carbohydrate choices/meal"

# ADAF Study Grant

## Case Western Reserve University Researcher Receives American Dietetic Association Foundation Grant for Chronic Kidney Disease Study

**Phil Allen, Julia Dombrowski**
ADA Media Contacts
E-mail: *media@eatright.org*

The American Dietetic Association Foundation, with sponsorship from Abbott, has awarded a research grant of $50,000 to support the development of a screening and assessment tool to identify people at nutritional risk with chronic kidney disease.

Alison Steiber, PhD, RD, LD, assistant professor of nutrition at Case Western Reserve University, received the grant for her proposed study "Improving Patient Outcomes with a Nutrition Assessment Algorithm in Chronic Kidney Disease Patients Stages 1-5."

Steiber, a registered dietitian and a member of the American Dietetic Association, is a specialist in nutritional assessment and CKD. She has conducted research in the areas of hospitalization and nutritional intake; hemodialysis and quality of life; subjective global assessment and carnitine content in foods. Steiber is active in the Cleveland Dietetic Association and the Northeast Council on Renal Nutrition. She is a member of the 2008-09 board of the Commission on Accreditation for Dietetics Education; a CADE program reviewer; and a member of the editorial board of the *Journal of Renal Nutrition*.

Steiber's study will include collaboration with other organizations that are also leaders in the area of CKD and nutrition research and treatment including the American Dietetic Association's Renal Dietitian practice group, National Kidney Foundation's Council on Renal Nutrition and the American Society for Parenteral and Enteral Nutrition's Renal Section.

The American Dietetic Association Foundation is a 501(c)3 charity devoted exclusively to nutrition and dietetics. It funds scholarships and awards, public awareness and research projects and ADA strategic initiatives and is the largest provider of scholarships and awards in the field of dietetics. The Foundation's mission is funding the future of the dietetics profession through research and education. Visit the ADA Foundation at www.adaf.org.

The American Dietetic Association is the world's largest organization of food and nutrition professionals. ADA is committed to improving the nation's health and advancing the profession of dietetics through research, education and advocacy. Visit the American Dietetic Association at www.eatright.org.

Abbott is a global, broad-based health care company devoted to the discovery, development, manufacture and marketing of pharmaceuticals and medical products, including nutritionals, devices and diagnostics. The company employs more than 68,000 people and markets its products in more than 130 countries. Abbott Nutrition (www.AbbottNutrition.com) develops and markets a wide range of science-based infant formulas, medical nutritionals, nutrition and energy bars, and related products to support the growth, health and wellness of people of all ages. ◆

## 50 Years Ago—ADA in 1958

Excerpts from article by Karen Lechowich,
Vice President, Diversity and External Relations

In 1958, E. Neige Todhunter, PhD was ADA president and presided over the 41st Annual Meeting in Philadelphia. Dr. Todhunter was Dean, School of Home Economics at the University of Alabama. She told the members that in addition to the accomplishments of the Association that "people have just completed Year One of the Space Age. This has already brought dramatic changes in the thinking and way of life of the American people, but the big changes are just beginning." There was a shortage of qualified dietitians in the country and the sphere of practice opportunities was widening.

ADA membership was 13,287 and one of the issues was the failure to develop leadership among the younger members. (That of course would be those celebrating their 50 years of membership today!) Geraldine Piper was speaker of the House of Delegates and Lendal Kotschevar, PhD from Montana was the first male member to serve as a HOD delegate.

The profession was concerned about the growth of food faddists, quacks and those profiting from the sale of vitamin concentrates and other special preparations. There also was discussion of the problem of follow-up of hospital dietary instruction in the community. A booklet, Food Facts Talk Back was produced to show facts and fallacies in a variety of settings. Some of the fallacies of the day: A grape juice diet is a cure for cancer; Oysters and beer are a poisonous combination and Cucumbers and watermelon cause polio. The booklet was published in Braille, Swedish and the National Science Teachers' Association mailed more than 2,000 copies to US science teachers.

### Resources:

ADA Annual Reports
Cassell, Jo Anne: Carry the Flame: the History of the American Dietetic Association, 1990

# What Does RPG Membership Do For You?

## Go to **www.renalnutrition.org** for all the details!

- Receive the quarterly newsletter, ***Renal Nutrition Forum***, containing:
  - Feature articles on a variety of nephrology nutrition issues for increasing our knowledge and for continuing education.
  - Chair reports provide updates on changes in their respective positions. This includes RPG Chair, CRN Chair, Legislative Chair, and other chairs.
  - Stipend reports contain conference summaries and highlights from members who have attended a conference in which RPG provided a stipend.
  - Rehab Corner provides insights to develop and integrate rehabilitation into our practice.
  - Relevant nutrition tips that can be shared with kidney patients and their families.
  - Book or cookbook reviews.
  - Updates on new membership services like new resources in the Lending Library, etc.
  - ***Plus much more…….***

Subscription for the membership year, begins with the Summer issue of the Forum, and continues through the Fall, Winter, and Spring Issues.

Contact the Membership Chairperson Danielle Frazer, RD (rd813303@gmail.com) if you do not receive all issues.

- Access to the **lending library**.
  - Access to the Lending Library which allows you to check out current texts and other materials free of charge except for the shipping costs. The library contains several of the texts that are recommended for review for the Certified Specialist in Renal Nutrition exam. We are constantly expanding and updating the library to include the latest texts.
- Access to the members only area of the **RPG Web site.**
  - www.RenalNutrition.org members only area features archives of the *Renal Nutrition Forum*
  - downloadable forms and applications
  - other topics of interest
- Are eligible to **apply for stipends and scholarships.**
  - For attending renal education conferences and meetings
  - For a scholarship worth $1,000.00 towards the pursuit of an advanced degree in nutrition.
  - Eligibility begins after 1 year of RPG membership.

Contact Sarah Kruger (kruger_sarah@yahoo.com), ADA-RPG Awards Chair for more information.

- Benefit from **publications,** which **advance technical and clinical skills** enhancing professional and patient education.
  - The National Renal Diet
  - The Guidelines for Nutrition Care of Renal Patients
  - The Clinical Guide to Nutrition Care in End Stage Renal Disease, third edition (in progress)
  - Medical Nutrition Therapy Across the Continuum of Care
- **Renal Dietitians (RPG):**
  - Plans a scientific priority session on renal nutrition topics, suggested by RPG members, at the ADA Food & Nutrition Conference & Expo (FNCE).
  - Provides a collective voice to promote the interest of renal dietitians and affect change in policy and legislation.
  - Has a network with the National Kidney Foundation – Council on Renal Nutrition, to coordinate joint efforts and educational opportunities.
  - Provides an avenue through which RPG members can influence the future direction of the profession through the House of Delegates.
  - Awards an "Outstanding Service Award" to a member who has made significant contributions to the clinical practice and profession of renal nutrition. ◆

# 2008-2009 RPG Executive Committee

**Mission:** Renal dietitians dietetic practice group is leading the future of dietetics by promoting and supporting its members working in nephrology nutrition.
**Vision:** RPG members are a valued source of expertise in nephrology nutrition.

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## RNF Guidelines for Authors

**Article length:** Article length is determined by the Editor for each specific issue. The feature article (including abstract) is approximately 3000 words (not including tables/graphs). Other articles are usually 1000-1500 words; member highlights and reports are approximately 400-500 words.

**Text format:** Times New Roman font, 12 point, double space.

**Tables/illustrations:** Tables should be self-explanatory. All diagrams, charts and figures should be camera-ready. Each should be accompanied by a title and brief caption that clearly explains the table, chart, diagram, figure, illustration, etc.

**References:** References should be cited in the text in consecutive order parenthetically. At the end of the text, each reference should be listed in order of citation. The format should be the same as the *Journal of the American Dietetic Association*.

**Reference citation examples:**
***Article in periodical:***
Knower WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Eng J Med*. 2002;346:393–403.
***Book:***
Institute of Medicine. *Dietary Reference Intakes: Applications for Dietary Assessment*. Washington, D.C.: National Academy Press; 2001.
***Chapter in a book:***
Walsh J. Which insulin to use and how to start. In: *Using Insulin*. San Diego, Calif.: Torry Pines Press; 2003.
***Web site:***
Medscape drug info. Available at www.medscape.com/druginfo. Accessed Feb. 3, 2004.
**Author information:** List author with first name, middle initial (if any), last name, professional suffix and affiliation (all in italics) below the title of the article. Also include the primary author's complete contact information including affiliation, phone, fax and email address.

*All submissions for publication should be submitted to the editor as an email attachment (MS word file). The feature articles from the Renal Nutrition Forum will be posted on the Members Only Section of the RPG website (password protected). Thus, please include a brief abstract along with feature article submissions.*


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