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Nutrition and Nephrolithiasis

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Nephrolithiasis, or the presence of kidney stones, is a significant health problem in our population. It occurs most frequently in males between 30 and 50 years of age (1). Roughly 10% of people in the United States will have at least one stone episode in their lifetimes (2). Approximately 50% of patients with previous urinary calculi have a recurrence within 10 years (3). The risk doubles in those with a family history of kidney stones (4). Kidney stone disease occurs more often in adults than in elderly persons, and more often in elderly persons than in children. Whites are affected more frequently than persons of Asian ethnicity, who are affected more often than blacks (3).

Etiology & Pathophysiology

Stones range in size from microscopic to as large as light bulbs, and come in an assortment of colors and shapes (2). Kidney stone formation is a complex process that consists of saturation, supersaturation, nucleation, crystal growth or aggregation, crystal retention, and stone formation in the presence of promoters, inhibitors, and complexors in urine (4). Kidney stones are formed when the concentration of components in the urine reaches a level which promotes crystallization. Stones are generally composed of calcium salts, uric acid, cystine, or struvite (1). Calcium stones are most common, with frequency of composition being calcium oxalate (60%), calcium oxalate and calcium phosphate (10%), and calcium phosphate (10%). Urate (5-10%), struvite (5-10%), and cystine (1%) stones follow in frequency of occurrence (4). Decreased fluid intake and consistently strong urine concentration or low urine volume are the most important risk factors for urolithiasis (3, 4). In addition, urolithiasis occurs more often in hot, arid areas than in temperate regions. Certain medications, such as triamterene (Dyrenium), indinavir (Crixivan) and acetazolamide (Diamox), are associated with urolithiasis (5, 6).

Diagnosis

Urolithiasis should always be considered in the differential diagnosis of abdominal pain. The classic presentation of renal colic is excruciating unilateral flank or lower abdominal pain of sudden onset that is not related to any precipitating event and is not relieved by postural changes or non-narcotic medications. With the exception of nausea and vomiting, secondary to stimulation of the celiac plexus, gastrointestinal symptoms are usually absent. It is generally believed that a stone must at least partially obstruct the ureter to cause pain. Distal ureteral stones may be manifested by bladder instability, urinary frequency, dysuria, and/or pain radiating to the tip of the penis, labia or vulva. Increasingly, calculi are encountered in asymptomatic patients and are found incidentally on imaging studies or during evaluation of microhematuria (3).

The diagnosis of urinary tract calculi begins with a focused history. Key elements include past or family history of calculi, and signs and symptoms of sepsis. The physical examination is often more valuable for ruling out non-urologic disease. Urinalysis should be performed in all patients with suspected renal calculi. Apart from the typical microhematuria, important findings to note are urinary pH and the presence of crystals, which may help to identify stone composition. Patients with uric acid stones usually present with acidic urine, and those with stone formation resulting from infection have alkaline urine. Identification of bacteria is important in planning therapy, and a urine culture should be routinely performed. Limited white blood cells in the urine is a fairly common response to irritation caused by the stone and, in absence of bacteruria, is not generally indicative of coexistent urinary tract infections (3).

Renal colic may be suspected based on history and physical examination, but diagnostic imaging is essential to confirm or exclude the presence of urinary calculi. Several imaging modalities are available, and each has advantages and limitations: abdominal ultrasonography, plain radiography, intravenous pyelography, and non-contrast helical computed tomography (CT). The superior sensitivity and specificity of helical CT allows urolithiasis to be diagnosed or excluded definitively and expeditiously without potential harmful effects of contrast media (3).

Medical Management

Uric acid stones are the only type amenable to dissolution therapy. Extracorporeal Shock Wave Lithotripsy (ESWL) and

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From the Editor's Desk

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

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In This Issue

Frequently people compare the passing of kidney stones to the pain of childbirth, so patients experiencing pain have a strong incentive to follow lifestyle changes. There is some disagreement among physicians about approaches to kidney stone management. Some practitioners concentrate on oxalate, some calcium, and still others protein and sodium. Our feature article examines many of these tactics in kidney stone management. The author, Mansi Mehta, designed the accompanying table as a reference for professionals, summarizing MNT strategies. The dietitian and physician must decide which dietary measures to emphasize for individual patients. I especially thank Linda Brinkley, RD, at the Center for Mineral Metabolism and Clinical Research at the University of Texas Southwestern Medical School for providing expert review. As an adjunct to the article, please find a patient handout on oxalate that Carolyn Cochran, MS, RD, from Dallas Transplant Institute, was kind enough to share.

Phillipa Norton Feiertag provides a thorough update on bone disease management in her "Advances in Practice" column. On a similar note, Joyce Vergili attended the National Kidney Foundation's Spring Clinicals and summarized a lecture on secondary hyperparathyroidism given by Dr. Geoffrey Block. Both articles introduce calcimimetic therapy for hyperparathyroidism.

Sharon Schatz presents a tutorial on citrus fruits, considered by many staff as "dirty words" in hemodialysis clinics. She provides useful potassium information that may allow more of our patients to partake in customarily "forbidden fruits."

In the Rehab column, Stephanie McIntyre explains the Life Options Advisory Council's "5 E's" campaign. Stephanie would like to bring recognition to other dietitians who are involved in patient rehabilitation, so please contact her to share your story and inspire others.

Please review the Area Coordinator map. Two positions for Area Coordinators are still open. Anne Ishmael's chair message invites us to find new resources. A wonderful benefit of being a RPG member is access to the lending library. You may check out up to three references for 28 days with a $30 deposit. If you reside in area I, II, or IV, please contact Jane Louis at louisjl@att.net. Members in areas III, V, VI, and VII should contact Heather Ohlrich to request a lending library form. Her email is hohlrich@renalcaregroup.com. This issue of the Forum contains a listing of library materials, so tap into this free resource before you purchase expensive materials!

As RPG members, we face nutritional challenges daily. Please make me aware of new research, new ideas, and new products that would aid practitioners in the delivery of renal MNT. Let's work together to improve patients' lives and their understanding of the foods they consume!

Sarah Carter

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endourologic techniques have almost replaced the open surgical procedures of stone removal used 20 years ago. Now, management strategies are aimed at initial or recurrent kidney stone prevention that include patient evaluation, metabolic workup, ample hydration, avoidance of infection, and good voiding habits (2,4).

MEDICAL NUTRITION THERAPY FOR ALL STONES

After corrective treatment for medical disorders, patients should receive nutrition counseling for diet and fluid modification to reduce urinary stone risk factors. The efficacy of a specific regimen based on comprehensive metabolic evaluation, repeated medical nutrition therapy, and metabolic monitoring was found to be more effective in reducing stone recurrence than nonspecific measures and limited screening (4).

Fluid and Volume

A high fluid intake is the one strategy that can be applied to all types of kidney stones. The objective is to maintain urinary solutes in the under-saturated zone by both an increase in urine volume and a reduction in solute load to inhibit nucleation. High urine flow will tend to wash out any formed crystals (4). A five-year randomized, controlled, prospective study involving first-stone-episode patients revealed lower rates of recurrence (12%) in subjects with a higher intake of water (intake that produced a urine volume $\geq$ 2 L per day) compared to those without (27%) (7).

To achieve dilution, the goal for urine volume should be 2.0 – 2.5 L/ day. Intake of 250 ml of fluid at each meal, between meals, at bedtime, and when arising to void at night is recommended. Hydration during sleep hours is important to break the cycle of "most-concentrated" morning urine. At least one-half of the fluid should be taken as water. Higher fluid intake should compensate for gastrointestinal fluid loss, excessive sweating from strenuous exercise or an excessively dry environment (such as a commercial airplane cabin). Patients who form idiopathic calcium stones with low urine volume and who are unable to increase urine volume may have altered thirst sensitivity and vasopressin release (4).

A recent epidemiological study examined the effects of particular beverages on the risk of symptomatic kidney disease in women. Consumption of tea and coffee, both caffeinated and decaffeinated, was associated with a risk reduction of 8-10%, while white wine decreased risk by 59%. The authors speculated that the protective effects of coffee, tea, and wine were caused by urinary dilution, determined by the ability of caffeine and alcohol to inhibit antidiuretic hormone. Therefore, the decreased risk for decaffeinated coffee may have another conferred mechanism (8). Bioavailability and observational studies do not demonstrate brewed tea with added milk as a risk factor, despite tea's high oxalate content. The recommendation for tea drinkers is to drink only a moderate amount of tea (about two cups per day), diluted, and with milk (4).

In the same epidemiological study, grapefruit juice ingestion was associated with a 44% increased risk for stone formation (8). In a separate study, grapefruit juice ingestion caused no changes in the lithogenicity and no net change in calculated supersaturation (4). Thus, the basis of observations from epidemiological studies with grapefruit juice remains unexplained. Other citrus juices, such as orange and lemon, apparently prevent, or at least fail to stimulate, stone formation because of their citrate content. Currently, good clinical practice recommends avoidance of grapefruit juice and limited intake of soft drinks that contain phosphoric acid (4, 7).

MANAGEMENT OF CALCIUM STONES

Calcium

Hypercalciuria is defined as a mean value of calcium in excess of 300 mg (7.5 mmol)/ day in men or 250 mg (6.25 mmol)/ day in women, or 4 mg (0.1 mmol)/ kg/ day for both genders in random urine collections of outpatients on unrestricted diets (1). Hypercalciuria, leading to increased supersaturation, is the most common metabolic abnormality in patients with calcium containing stones. Thirty to fifty percent of patients with kidney stones have idiopathic hypercalciuria, absorbing and excreting more calcium than normal persons (9). Hypercalciuria is idiopathic when serum calcium is normal and common causes can be excluded. Idiopathic hypercalciuria can result from decreased renal tubular reabsorption of calcium, prolonged bed rest or exaggerated dietary calcium intake. Hypercalciuria can also result from a low serum phosphorus level caused by a renal phosphate leak that stimulates 1,25-dihydroxy vitamin D_3 production. Calcium loading studies show that urinary calcium rises with an increase in dietary calcium of up to 800 mg/day. Beyond that point, animal protein may be responsible for the rise in urine calcium (4).

A reduction in dietary calcium has been considered a logical way to prevent recurrent stones, based on the assumption that a diet low in calcium would reduce urinary calcium and lower relative supersaturation with respect to calcium oxalate. However, a reduction in dietary calcium not only reduces urinary calcium but also increases urinary oxalate. This increase may result in supersaturation related to the calcium oxalate solid phase. Dietary calcium could bind to intestinal oxalate and prevent its absorption and subsequent urinary excretion (9). Thus, patients should be advised to consume age and sex appropriate amounts of calcium. Chronic prolonged calcium restriction may damage the bones of calcium-stone patients because of deficient calcium intake and increased losses of calcium in the urine (4). Moreover, patients with idiopathic hypercalciuria frequently have decreased bone mineral density (9).

In a recent randomized trial, a calcium intake of 1,200 mg, coupled with restriction of both animal protein and salt intake, was found to be more effective than calcium restriction (400 mg/day) and low oxalate intake (less than 50 mg (2.2 mmol)/day) in preventing recurrent calcium oxalate stones. After five years, only 20% of the men on the normal calcium, reduced-salt, and reduced-protein diet had at least one episode of stone recurrence, whereas 38% of the men on the low-calcium diet had at least one stone episode (10).

In another study, the effects of five different dietary protocols (low calcium, high oxalate, vitamin C, high salt, and lacto- vegetarian) were examined on urinary risk factors for calcium oxalate stone formation. The low

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calcium diet excluded all dairy products and contained 400mg calcium. The high oxalate diet added 230g of beetroot each day for an average intake of 510 mg oxalate. The vitamin C diet included a 1000 mg ascorbic acid supplement daily. The high salt diet added 15 mg of sodium chloride each day. In 10 black and 10 white South African males, the low calcium diet caused statistically significant changes in the black subjects as it increased urinary oxalate, decreased relative supersaturation of calcium oxalate, and increased relative saturation of brushite, a calcium phosphate salt (11). High brushite concentration can increase the propensity for calcium phosphate crystallization (12). In summary, there are many reasons why calcium restriction should be avoided in hypercalciuric patients (7):

- No prospective studies are available to support the belief that calcium restriction leads to a reduction of stone recurrence.
- Calcium restriction induces secondary hyperoxaluria.
- Calcium restriction prompts bone loss due to negative calcium balance, which could lead to osteoporosis or other deficiencies.
- Chronic calcium restriction might upregulate vitamin D receptors allowing, 1,25 dihydroxy vitamin D_3 to intensely stimulate both intestinal calcium absorption and bone resorption.
- Other nutrients such as protein, sodium, oxalate, and potassium may affect calcium absorption as well.
- A normal calcium intake may be more palatable, possibly improving patients' adherence (2).

Oxalate

Dietary oxalate intake affects urinary oxalate. Hyperoxaluria plays an important role in calcium stone formation and is observed in up to 20% of recurrent stone formers. The oxalate content of a normal diet is in the range of 80 – 100 mg / day and absorption does not usually exceed 10 – 20% of the amount in food (13). Oxalate cannot be metabolized in the body, and the renal route is the only mode of excretion. Oxalate in urine originates from both the absorption of dietary oxalate and synthesis endogenously. Glyoxylic acid accounts for 50 – 70% of urinary oxalate, and ascorbic acid accounts for 35 – 55%. Several amino acids are precursors of oxalate via glyoxalate and glycolate. Pyridoxine acts as a precursor in the conversion of glyoxalate to glycine, and its deficiency could increase endogenous oxalate production (4).

The ability of oxalate rich food items to augment oxalate excretion depends not only on the oxalate content, but also on its bioavailability, solubility, and salt form (7). Limited data are available on oxalate content of foods because of inconsistent values from different methodologies used for analysis. Only a few foods have been tested for oxalate bioavailability (4). Spinach and rhubarb are considered high-risk food items, due to the high amounts of bioavailable oxalate. Peanuts, almonds, pecans, instant tea, and chocolate are considered moderate risk food items. Finally, the effect of dietary oxalate on urine oxalate critically depends upon calcium intake, since decreasing calcium load in the intestinal lumen will increase the concentration of free oxalate anions available for absorption (7).

Animal Protein

Epidemiological studies find a correlation between improved standard of living, high animal protein intake, and rising incidence of kidney stones (4). High protein intake of animal origin contributes to hyperuricosuria due to purine overload, hyperoxaluria due to higher oxalate synthesis, and hypercitraturia due to the higher tubular reabsorption (14). Additionally, protein–induced hypercalciuria may be caused by higher bone resorption and lower tubular calcium reabsorption needed to buffer the acid load, or by the elevated filtered load of calcium and the presence of non-reabsorbable calcium sulfate in the tubular lumen. An acute moderate protein restriction reduces urinary oxalate, phosphate, calcium, and uric acid and increases citrate excretion (7). A five-year randomized trial of men on a diet composed of normal calcium, low salt, and reduced animal protein (52 g with 21 g from meat or fish and 31 g from milk and derivatives), reduced stone incidence by 51%. This dietary regime decreased urine calcium and oxalate and decreased calcium-oxalate saturation (10).

Citrate

Citrate is a well-established inhibitor of calcium oxalate crystallization, acting as a chelator and modifier (11). By complexing with calcium in the urine, there is less calcium available to bind urinary oxalate. This, in turn, prevents the formation of calcium oxalate or calcium phosphate stones. Distal renal tubular acidosis, acidosis accompanied by hypokalemia, malabsorption syndrome with hyperoxaluria, and excessive meat intake (acid-ash) are associated with decreased urinary citrate levels. Normal daily urinary citrate levels should be more than 640 mg. Lemonade made with lemon juice (4 oz.) diluted to 2 L with water, should be encouraged in patients with low urinary citrate (15). The use of potassium or magnesium citrate to reduce the incidence of kidney stones is currently under investigation (4). Finally, a high calcium intake is expected to lower citrate excretion as well (7).

Sodium

There is a correlation between 24-hour urine sodium and hypercalciuria. A linear increase in urinary calcium occurs as urinary sodium increases from 920-4,600 mg (40 to 200 mmol)/ day. For every 1,380 mg (60 mmol) increase in urine sodium, the relative risk of hypercalciuria increases by 1.63 times. This is due to the fact that sodium and calcium are reabsorbed at common sites in the renal tubule (4). The recommended sodium chloride intake in a recent randomized trial was found to be 1,500 mg (50 mmol)/ day. However, yearly measurements of urinary sodium in these subjects were 2,530 – 2,990 mg (110 – 130 mmol)/day, showing subjects' lack of compliance to this rigid regime (10). Salt intake should be lowered in patients with hypercalciuria to less than 2,300 mg sodium (100 mmol)/day (4).

Potassium

An epidemiological study reported that the lower the potassium intake below 2,886 mg (74 mmol)/day, the higher the relative risk of stone formation. Such an effect is ascribed to an increase in urinary calcium and a decrease in urinary citrate excretion induced by a low potassium intake (7). The level of potassium used in a recent study that reduced stone incidence by 50% was 4,680 mg (120 mmol)/day (10). Stone formers could choose low oxalate

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fruits and vegetables, to increase potassium in their diets (4).

Vitamins

The effect that large doses of vitamin C will increase urinary oxalate is controversial (7, 16). Thus, it is recommended that vitamin C should be limited to < 2.0 g/day (4, 16). Vitamin B_6 intakes of > 4 mg/day in comparison with intakes of < 3 mg/day led to a relative risk reduction of 0.66. This confirms the role of vitamin B_6 in reducing the incidence of kidney stones (4).

Fiber

The bioavailability of oxalate in wheat bran is high and varies per type, although the oxalate in wheat bran does not seem to be a risk factor for hyperoxaluria. No change in urinary calcium or oxalate is observed when fiber intake from fruit, vegetable, and cereal sources is increased up to 25 g/day. Studies of rice bran have shown a reduction in new stone formation (4).

Estrogen

Data from 1,454 adults with idiopathic calcium oxalate nephrolithiasis were analyzed to probe the role of estrogen in calculus formation. The saturation of calcium oxalate and brushite, and excretion of undissociated uric acid in women was less than in men. Compared with men, women had lower urinary calcium until age 50 years (menopause), when the two sexes' urinary calcium became equal. In addition, estrogen treatment was associated with lower urinary calcium and calcium oxalate saturation in postmenopausal women (17). Together these findings support a connection between estrogen status and the propensity for calcium nephrolithiasis.

MANAGEMENT OF URIC ACID STONES

Uric acid is a product of purine metabolism. The sources of purine are food and tissue catabolism. About one-half of the purine load is from endogenous sources and is constant. Exogenous dietary sources provide the other half and account for the variation in uric acid present in the urine (4).

The three major abnormalities causing uric acid stone development are low urinary pH, low urine volume, and hyperuricosuria. Of these three, the most prevalent and invariant is low urine pH (18). Most patients with idiopathic uric acid stones have normal uric acid excretion, but almost all have a persistently low urine pH that promotes uric acid precipitation. Solubility of uric acid in the urine is greatly determined by the urinary pH. At a very low urinary pH, uric acid becomes supersaturated and uric acid crystals precipitate. The subsequent mechanisms for uric acid stone formation remain unclear (19).

Uric acid stones may be idiopathic, congenital, or acquired. Acquired causes frequently influence urinary pH, volume, or uric acid concentration. Low carbohydrate, high-protein (LCHP) diets, such as the Atkins' diet used for weight reduction, deliver an exaggerated acid load to the kidney (19). In a recent study, ten healthy subjects initially consumed their usual non-weight-reducing diet, then a severe carbohydrate-restricted diet for two weeks, followed by a moderate carbohydrate-restricted maintenance diet for four weeks. There was a remarkable increase in net acid excretion. This was likely the result of the combined effects of the high-protein, low-carbohydrate diet, decreased urinary pH and citrate (which can inhibit calcium stone formation) and increased urinary calcium levels. This short-term study suggests that intake of a LCHP weight reduction diet, through delivering an exaggerated acid load and reducing urinary pH, heightens the propensity for both uric acid stone formation and bone loss (20).

Dietary purines should be restricted in patients with uric acid lithiasis and hyperuricosuric calcium oxalate stones. Animal muscle proteins such as meat, fish, and poultry are rich in purines and acid ash; therefore should be used in moderation to meet protein requirements. High purine foods including organ meats, anchovies, herrings, sardines, meat-based broth and meat-based gravies should be avoided. Noncompliance with dietary measures or persistent hyperuricosuria will warrant the use of medication such as allopurinol (4). Urinary alkalization, to a pH of 6.0 to 6.5, is the cornerstone of medical management for uric acid stone dissolution. It should be the primary mode of treatment in the absence of absolute indications for surgical intervention. Furthermore, prophylaxis through urinary alkalization using oral alkali prevents stone recurrence and associated morbidity (4, 20). Potassium citrate has been used as the therapy of choice. Sodium bicarbonate will increase urinary monosodium urate and calcium, thus should not be used (4).

MANAGEMENT OF STRUVITE STONES

Struvite stones are comprised of magnesium ammonium phosphate and carbonate apatite. They are also known as triple-phosphate or infection stones. Unlike most urinary stones, they occur more commonly in women than in men, at a ratio of 2:1. They form only in the presence of bacteria such as Pseudomonas, Klebsiella, Proteus mirablis, and Urealyticum that carry urease, a urea-splitting enzyme. Urea breakdown results in ammonia and CO_2 production, thus raising urine pH and the level of carbonate. Struvite stones grow rapidly to form large staghorn calculi in the renal pelvic area. A staghorn calculus is a large renal calculus with multiple irregular branches. The mainstay of treatment is surgical removal of calculi alone or with ESWL with adjunctive culture-specific antimicrobial therapy that uses urease inhibitors. The goal is to eliminate or prevent urinary tract infections by regular screening and monitoring of urine cultures (4).

MANAGEMENT OF CYSTINE STONES

Cystine stones caused by homozygous cystinuria represent 1-2% of urinary calculi. Most patients with cystinuria will suffer from recurrent stone disease during their lifetime. Normal individuals excrete 20 mg/day or less of cystine in their urine, whereas stone-forming cystinuria patients excrete more than 250 mg/day. Cystine solubility increases when urine pH exceeds 7.0; therefore, an alkaline urine pH must be maintained 24-hours/day, even while sleeping. This is usually achieved by the use of medication; however, in the past, dietary changes were recommended to change urine pH (limiting acid ash foods and increasing alkali ash foods). Calculating the amounts of acid-forming ions and alkaline-forming ions to determine the outcome of a diet can be very complex. The acid ash / alkali ash approach has limited use in today's clinical practice. Fluid intake of more than 4 L daily is recommended to prevent cystine crystallization. Lower sodium intake may also be useful in reducing cystine in urine (4).

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Nutrition and Nephrolithiasis

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Methionine is the metabolic precursor of cystine. While severe protein restriction to avoid methionine is impractical, avoidance of excesses may be beneficial. D-Pencillamine is commonly used as a cystine-binding agent to treat cystinuria. The cystine-pencillamine product is 50 times more soluble than cystine itself (4).

In conclusion, pain from renal colic may provide the initial motivation to patients to prevent stone recurrence. Unfortunately, when symptoms subside, the dietary and pharmocologic regimen often becomes suboptimal (7). Nevertheless, diet and fluid modification should be advocated as a first step for prophylaxis of kidney stone disease to improve the urinary risk profile and reduce recurrence rate (4). These dietary and fluid modifications are summarized in table 1.

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Table 1. Summary of Recommendations for MNT for Kidney Stones

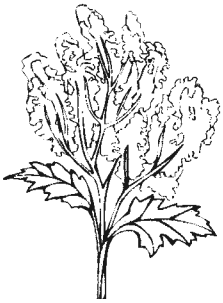
Goals	Avoid or Limit
Ample hydration: 3 - 4 L/day Urine volume goal: 2.0 - 2.5 L/day 250 ml of fluid at each meal, between meals, at bedtime, and when arising to void at night. At least one-half of fluid taken as water. Moderate amounts of tea (about 2 cups/day), diluted, and with milk	Grapefruit juice Limit soft drinks containing phosphoric acid.
Good voiding habits	Urinary tract infections.
Adequate calcium intake (Check RDA for age /sex appropriate recommendations.)	Vitamin D supplementation. Calcium supplementation unless recommended by physician.
Moderate protein intake (21g from meat, fish, and poultry and 31g protein from milk and derivatives)	High purine/High protein foods: organ meats (liver, kidney, sweetbreads), anchovies, herring, sardines, mackerel, meat-based gravies (from meat drippings or extracts). High protein low carbohydrate diets (Atkin's type). Excessive intakeof other high purine foods, such as mushrooms, asparagus, peas and lentils
Use low oxalate fruits and vegetables	Foods with moderate to high bioavailabiloity of oxalate: Spinach, rhubarb, peanuts, almonds, pecans, instant tea, and chocolate.
Sodium intake $\leq$ 2,300 mg/d; Increase potassium intake to $\geq$ 4,000 mg/d	High sodium intake $>$ 2,300 mg/d.
Increase intake of vitamin B6 to $\geq$ 4 mg/d. Limit vitamin C supplementation to $<$ 2.0 g/d	Excessive vitamin C supplementation.
Use lemonade made with lemon juice (4 oz), diluted to 2 L, with water to increase excretion of citrate $\geq$ 640 mg/d. Take medications as prescribed by physician.	

High Oxalate Foods

(may increase oxalate in your urine &
make more calcium-oxalate kidney stones)

Tea

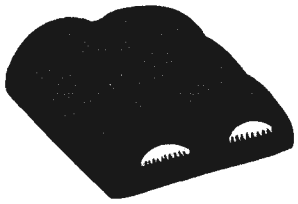
Rhubarb



Berries

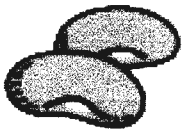
Spinach

Beets



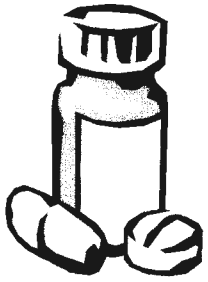
Chocolate & Cocoa

Dried Beans



Nuts

Wheat Bran



Vitamin

Vitamin C (ascorbic acid), in large doses, can be part of making more oxalate!

References: Journal of the American Dietetic Assoc 93(8): 901, 1993; and DFW Hospital Council Diet Manual, Irving, TX. 1997.

Remember: Always take your medicines as your doctor prescribes. Drink plenty of liquids to help dilute your urine, so that it is less likely to make kidney stones. Ask your dietitian for more information about cutting back on salt and salty foods.

Flesch-Kincaid grade level 10.2

C. Cochran 6/2004

Advances in Practice

New Approaches for Managing Bone Metabolism and Disease in Chronic Kidney Disease

By Philippa Norton Feiertag, MEd, RD, CSR, LD *Philippa is a Clinical Analyst/Renal Nutrition Specialist with Clinical Computing, Inc. in Cincinnati, OH. She can be reached at feier@fuse.net*.

Publication in 2003 of the National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (NKF-K/DOQI) Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease (CKD) focused attention on managing serum calcium, phosphorus and parathyroid hormone (PTH) in this population (1). If these parameters are not effectively controlled, their impact on morbidity and mortality in patients with CKD may be severe.

Serum phosphorus, PTH and calcium-phosphorus product all increase when kidney function declines. These changes promote bone disease, as well as the calcification of soft tissue and changes in lipoprotein metabolism that are associated with cardiovascular disease (CVD) (2-4). If dietary phosphorus restriction, phosphate binders and vitamin D analogs are unsuccessful in controlling serum phosphorus and PTH, patients may require surgical parathyroidectomy (1).

Data from the United States Renal Database indicate that rates of parathyroidectomy declined by approximately 30% between 1995 and 1999, suggesting an improvement in the effectiveness of therapy for elevated PTH (5). However, PTH and serum phosphorus remain elevated in many patients undergoing maintenance dialysis therapy, and results from a recent study indicate that phosphorus restrictions are abused more commonly than other dietary restrictions (2,6,7).

Clearly, there is a need to explore new strategies for managing calcium, phosphorus and PTH to improve health outcomes in patients with CKD. This column will review the pathophysiology of bone disease, examine recommendations for appropriate serum calcium, phosphorus and PTH levels, and explore interventions for controlling these parameters in patients with CKD.

Pathophysiology of bone disease in CKD

CKD is defined as kidney damage, or glomerular filtration rate (GFR) below 60 mL/min/1.73 m2 for ≥ 3 months reflecting decreased kidney function (8). Five stages of CKD are recognized based on the level of kidney function. Table 1 summarizes the disturbances in mineral and hormonal balance that accompany each stage of CKD.

Phosphorus excretion starts to decrease during Stage 2 and hyperphosphatemia is evident at Stage 4 (1). When GFR approaches 35 mL/min/1.73 m2 (Stage 3), synthesis of 1,25- dihydroxyvitamin D declines and serum vitamin D levels drop below normal (9). PTH subsequently begins to rise, and this process is exacerbated by hyperphosphatemia and a drop in serum calcium (1,9).

Elevated PTH, or hyperparathyroidism, impacts bone metabolism and causes the high turnover bone diseases osteitis fibrosa and uremic osteodystrophy (1). In osteitis fibrosa, abnormal bone formation results from increased activity of cells involved in modeling bone. Osteodystrophy is characterized by defects in bone mineralization. Net bone loss in high turnover bone disease leads to reduced bone strength and increased risk of fracture.

Persistently elevated PTH also contributes to cardiovascular morbidity and mortality. When mineral storage in bone decreases, extraskeletal mineralization may result in calcification of vascular tissues. In patients undergoing continuous ambulatory peritoneal dialysis (CAPD), hyperparathyroidism and high calcium and phosphorus levels are associated with calcification of the aortic and mitral valves in the heart (10). Calcification of the cardiac valves, aorta and carotid arteries have been linked to increased risk of CVD.

PTH also suppresses the lipid regulating enzyme hepatic triglyceride lipase (HTGL), resulting in increased intermediate-density lipoprotein (IDL) and decreased high-density lipoprotein (HDL). These changes in lipoprotein levels are strongly associated with progression of atherosclerosis in patients undergoing dialysis (4). Furthermore, animal studies show that PTH promotes thickening of arteriole walls in the heart, which may impair vasodilation when there is a need for increased blood flow to the heart wall (11).

Recommendations for appropriate serum calcium, phosphorus and PTH levels in patients with CKD

The NKF-K/DOQI Clinical Practice Guidelines for Bone Metabolism and Disease in CKD are directed at adults, age 18 years and older, with CKD (1). Goals

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Table 1. Stages of Chronic Kidney Disease (CKD) and associated changes in mineral and hormonal balance (1,8,9)

Stage	Level of Kidney Function	GFR (mL/min/1.73 m^2)	Changes in mineral/ hormonal balance
1	Kidney damage with normal or increased GFR	≥90	
2	Kidney damage with mild decrease in GFR	60-89	Decrease in phosphorus excretion
3	Moderate decrease in GFR	30-59	Decrease in serum vitamin D and calcium
4	Severe decrease in GFR	15-29	Hyperphosphatemia and hyperparathyroidism
5	Kidney failure	<15	Hyperphosphatemia and hyperparathyroidism

GFR = glomerular filtration rate

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for managing bone metabolism and disease include:

- Measuring calcium, phosphorus and intact PTH (iPTH) in all patients with GFR<60mL/min/1.73m^2 (CKD stages 3 – 5);
- Measuring vitamin D and correcting deficiency if iPTH is elevated;
- Implementing dietary phosphorus restriction and phosphate binders for serum phosphorus control;
- Maintaining Ca x P < 55mg^2/mL^2. Precise targets for serum calcium, phosphorus and iPTH levels depend on the stage of CKD. Target levels, and recommended strategies for achieving them, are summarized in Table 2.

Interventions for controlling serum calcium, phosphorus and PTH in CKD

Concern regarding the potential for adverse outcomes from therapeutic management of renal bone disease predates publication of the NKF - K/DOQI Clinical Practice Guidelines for Bone Metabolism and Disease in CKD. Vitamin D analogs may increase risk of soft tissue calcification by raising serum calcium and phosphorus levels (12). Calcium-based phosphate binders can also contribute to hypercalcemia (13). For these reasons, alternative strategies for managing serum calcium, phosphorus and PTH have been investigated.

Serum phosphorus levels must be controlled early in the course of CKD if hyperparathyroidism and renal bone disease are to be reduced. However, dietary phosphorus restrictions are difficult to follow, calcium-based binders can increase the risk of soft tissue calcification and use of aluminum binders is limited by their potential toxicity (7,14).

Sevelamer hydrochloride (GelTex Pharmaceuticals, Inc., Waltham, MA), a phosphate-binding, calcium-free polymer, decreases rates of vascular calcification when compared with phosphate binders containing calcium (15). Nevertheless, results from an 8-week randomized double blind study of hemodialysis patients suggest that sevelamer hydrochloride may be less effective than calcium-based binders in controlling serum phosphorus and calcium-phosphorus product (16).*

Lanthanum carbonate (Shire Pharmaceuticals Group plc, Basingstoke, UK) is undergoing evaluation for treatment of hyperphosphatemia in patients with CKD Stage 5 on maintenance dialysis therapy. One 16-week study assessed effects of lanthanum carbonate on serum phosphorus, calcium, calcium-phosphorus product and PTH in 126 hemodialysis (HD) patients aged ≥18 years (17). During dose titration, patients received divided doses of lanthanum carbonate with meals to achieve serum phosphorus ≤ 5.9 mg/dL. Patients were then randomized to receive either lanthanum carbonate or placebo during a

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Table 2. Strategies for achieving recommended levels for serum calcium, phosphorus and intact parathyroid hormone (iPTH) in Chronic Kidney Disease (CKD) (1)

Intervention	CKD Stages 3 and 4	CKD Stage 5
Dietary phosphorus restriction	Restricted dietary phosphorus to 800 - 1000 mg if serum phosphorus >4.6 mg/dL or iPTH elevated [>70 (Stage 3) or> 110 pg/mL. (Stage 4)].	Restricted dietary phosphorus to 800-1000mg/day if serum phosphorus >5.5 mg/dL or iPTH >300 pg/mL.
Phosphate binders	Implement phosphate binders if serum phosphorus/iPTH remain elevated despite dietary phosphorus restriction; Calcium (Ca)-based phosphate binders may be used as initial therapy.	Implemented Ca-based or non-Ca, non-aluminum, non-magnesium binders for elevated serum phosphorus; If serum phosphorus remains > 5.5 mg/dL, use a combination of phosphate binders: • Total Ca intake from binders and diet should not exceed 2000 mg/day; • Avoid Ca-based binders if serum Ca>10.2 mg/dL or iPTH<150 pg/mL on consecutive measurements or if there are vascular and/or soft tissue calcifications; • If serum phosphorus>7.0 mg/dL, use one 4-week course of aluminum-based binder and consider more frequent dialysis.
Vitamin D therapy	Initiate oral vitamin D therapy (0.25 mcg calcitriol or alfacalcidol/day or 2.5 mcg doxercalciferol 3x/week) when serum 25(OH) - vitamin D>30 ng/mL and iPTH > 70 pg/mL (Stage 3) or > 110 pg/mL (Stage 4). Hold oral vitamin D for: • iPTH < target range for CKD stage. Resume therapy at 50% previous dose or use alternate day dosing when iPTH > target range for CKD stage; • Serum Ca > 9.5 mg/dL. Resume therapy at 50 % previous dose or use alternate day dosing when Ca < 9.5 mg/dL. • Serum Phosphorus > 4.6 mg/dL. Initiate/increase phosphate binder and resume prior oral vitamin D dose when serum phosphorus ≤ 4.6 mg/dL.	Initiate vitamin D therapy the iPTH > 300 pg/mL; • 0.5-1.5 mcg calcitriol, 2.5-5 mcg paricalcitol or 2 mcg doxercalciferiol IV each treatment for hemodialysis patients; • 0.5-1 mcg calcitriol or 2.5-5 mcg doxercalciferol by mouth 2-3x/week, or 0.25 mcg calcitriol by mouth daily for peritoneal dialysis patients. Adjust vitamin D therapy based on changes in iPTH, serum Ca and phosphorus according to algorithms provided in K/DOQI Clinical Practice Guidelines for Bone Disease and Metabolism in CKD.

* Editor's note: FDA-approved labeling for sevelamar hydrochloride does not mention use in CKD stage 1-4

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4-week maintenance phase. At the study end-point, there was a mean difference in serum phosphorus of 1.91 mg/dL between the lanthanum carbonate and placebo groups. Calcium-phosphorus product and serum PTH were also significantly lower with lanthanum carbonate than placebo.

In a multi-center study of 98 maintenance dialysis patients, bone biopsies were taken at baseline and after one year of treatment with either lanthanum carbonate or calcium carbonate (18). The incidence of hypercalcemia was much lower in the lanthanum carbonate group (6%) than in the calcium carbonate group (49%). The percentage of patients with bone abnormalities decreased from 36% to 18% in the lanthanum carbonate group, while renal osteodystrophy increased from 43% to 53% in those who received calcium carbonate.

Mixed metal hydroxy-carbonate (MMHC) compounds, based on magnesium and iron, have also been evaluated for their effectiveness as phosphate binders. MMHCs were found to be much more effective at binding phosphorus than established binders including calcium carbonate, calcium acetate, aluminum hydroxide, magnesium hydroxide and lanthanum carbonate (19). Thus, MMHCs may provide a better alternative to existing and emerging phosphate binders for managing hyperphosphatemia.

Nonpharmacological intervention to improve serum phosphorus control was the focus of a recent small study in HD patients (20). Weekly dialysate phosphate removal was the end-point of an investigation into the impact of dialysis duration on serum phosphorus levels in nine stable patients. In a separate investigation, the effect of exercising with a bicycle ergometer on dialysate phosphate removal was measured in 12 different patients. Both increased dialysis time (five hours versus four hours) and exercising before or during dialysis increases dialytic removal of phosphorus and is anticipated in the long term to improve serum phosphorus control.

Vitamin D analogs have been used with varying degrees of success to suppress production of PTH in patients with CKD. However, vitamin D therapy may raise serum calcium and phosphorus by increasing intestinal absorption, placing the patient at increased risk for soft tissue calcification (21). Use of vitamin D therapy has also been associated with increased frequency of low turnover bone disease (1).

Identification of a specific calcium-binding receptor in cell membranes of parathyroid tissue has allowed the development of a new therapy for hyperparathyroidism. Organic molecules, called calcimimetics, increase sensitivity of this receptor to calcium leading to decreased PTH secretion (22,23). Calcimimetics can be administered orally and do not elevate serum calcium and phosphorus levels.

The calcimimetic cinacalcet hydrochloride (Amgen, Thousand Oaks, CA) decreases both iPTH and calcium-phosphorus product in HD patients with secondary hyperparathyroidism (24,25). Efficacy of this calcimimetic was demonstrated in a double-blind, randomized, placebo-controlled study of 78 HD patients with secondary hyperparathyroidism (26). Patients received either calcimimetic or placebo, and baseline iPTH was similar in both groups (632 ± 280.1 pg/mL versus 637 ± 455.9 pg/mL respectively). However, during the course of the 18-week study, iPTH decreased by 26% in the calcimimetic group compared with a 22% increase in the placebo group. Patients receiving calcimimetic had an 11.9% decrease in calcium-phosphorus product compared with a 10.9% increase in those receiving placebo.

Calcimimetics are also effective in reducing PTH in HD patients with severe secondary hyperparathyroidism (27). Twenty-one patients with iPTH up to 1200 pg/mL were randomly assigned to receive either a calcimimetic or placebo for 15 days. iPTH levels decreased by 70 ± 3% at four hours on the first treatment day in the calcimimetic group, declined progressively until day nine and remained below pre-treatment levels until the end of the study. Blood ionized calcium levels also decreased after the first dose of calcimimetic, but neither iPTH nor blood calcium levels declined in the placebo group.

Summary

Renal bone disease and soft tissue calcification remain important contributors to morbidity and mortality in patients with CKD. The NKF-K/DOQI Clinical Practice Guidelines for Bone Metabolism and Disease in CKD provide guidance for treating these disorders. Therapeutic strategies continue to evolve, and novel approaches including calcium-free phosphate binders and calcimimetics offer the potential to control hyperparathyroidism without elevating serum calcium. Nonpharmacological interventions, including exercise regimens and longer dialysis times, are also receiving attention as strategies for improving serum phosphorus control.

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Stipend Report from National Kidney Foundation's Spring Clinical Meetings, April 30, 2004

Secondary Hyperparathyroidism: Clinical Consequences, New Therapeutic Targets and New Therapies

Presented by Geoffrey A. Block, MD. Summarized by Joyce M. Vergili, MS, RD, CDE. *Joyce is the renal dietitian for Northern Hudson Valley Dialysis Center in Catskill, NY. She is a doctoral student in the Nutrition Education Program at Teachers College, Columbia University in New York, NY. She can be reached at jvergili@earthlink.net.*

Prevalence of Secondary Hyperparathyroidism

Most of the 300,000 people on dialysis have secondary hyperparathyroidism (2° HPT) and abnormal mineral metabolism. However, 2° HPT begins during stage 3 chronic kidney disease (CKD), which affects seven to eight million people. According to a study by Winkelmeyer and others, only 3% of CKD patients had their parathyroid hormone (PTH) level checked during their pre-ESRD care (1). This appalling statistic may explain why at dialysis treatment initiation, 50% of patients have an intact parathyroid hormone (iPTH) > 300 pg/mL (with nearly 80% having an iPTH of > 100 pg/mL); 65-70% have serum phosphate levels > 4.5 mg/dL; and 25% have a serum calcium level < 8 mg/dL. Geoffrey Block, M.D. states, "If they come to us looking like this, things [can only] go down hill from there with regard to mineral metabolism…We have a lot of areas where we can improve."

K/DOQI Guidelines: How Are We Doing?

According to the K/DOQI guidelines, the target range for serum calcium is 8.4 – 9.5 mg/dL, i.e., the lower end of normal. In all cases, serum calcium should be <10.2 mg/dL. Serum phosphate (PO_4) should be 3.5 – 5.5 mg/dL. Block points out that, even though this range is lower than the target ranges we have used in the past, this is not a normal serum PO_4 (which is about 2.5 - 4.5 mg/dL). The goal for the calcium x phosphorus product (CaxP) is <55; and for iPTH, 150- 300 pg/mL. Block says that according to data compiled by a national dialysis chain, we have a long way to go in terms of achieving these goals. During the first 6 months of analysis:

- 25% of patients achieved target PTH ranges
- 60% achieved the CaxP target of <55%
- 40% achieved the phosphorus goal
- 50% achieved calcium goal ranges

Within the next 6 months, all percentages dropped dramatically, with only 4% of patients meeting all four guidelines. Clearly, the work of the clinician is cut out for us!

Failure to Meet K/DOQI Targets Is Associated with Increased Mortality Risk

According to an unpublished study of over 40,000 patients, Block and colleagues found a positive relationship between increasing serum PO_4, serum calcium, CaxP product, and PTH levels (as the independent variables), and mortality risk (as the dependent variable). Block interprets these findings to mean that thousands of lives every year could potentially be saved by optimizing mineral metabolism.

What is the Proposed Mechanism to Explain Why Abnormal Mineral Metabolism Increases Mortality Risk?

Block references Hujairi and associates to support his contention that part (but probably not all) of the mortality risk associated with mineral metabolism has to do with the minerals' ability to stimulate vascular calcification (2). The reader is referred to the diagram in the Hujairi article for a comprehensive review of the very complex set of events by which abnormal calcium and phosphorus metabolism increase mortality risk. Briefly, calcium and phosphorus seem to be involved in two separate and distinct ways. First, calcium-phosphorus disturbances seem to modify the phenotypic expression of smooth muscle cells such that they de-differentiate into calcifying vascular cells and produce mineralizing proteins. Second, calcium and phosphorus levels play a role with regard to calcification in non-skeletal environments.

Fetuin, a protein that has received much attention lately, is a circulating protein that inhibits calcification, and appears to increase the solubility of calcium and phosphorus, thereby preventing the circulation of calcium-phosphorus crystals in the blood. Fetuin is negatively affected by one's state of inflammation, i.e., when inflammation increases, fetuin levels decrease. This mechanism may be one way (of many) by which inflammation reduces survival. Additional promoters of calcification, besides inflammation, include vitamin D (possibly), warfarin (interestingly enough), and a high CaxP product.

A vitamin that we may have been overlooking with regard to bone metabolism and arterial calcification is vitamin K. According to the results of the Nurse's Health Study, there appears to be a relationship between vitamin K and osteoporosis, at least in the general population, as well as a relationship between this vitamin and arterial calcification. Matrix GLA protein (MGP), which depends upon vitamin K for its formation, is an inhibitor of calcification. Like fetuin, MGP inhibits the calcification process. Accurate assessment of patients' vitamin K nutriture is difficult, but it may be an unexplored opportunity by which the calcification process might be mitigated.

In summary, there are physiological attempts to achieve a balance among mineral metabolism disturbances, stimulators of calcification, and inhibitors of calcification. The net result of that balance determines whether and how aggressively dialysis patients calcify, which in turn, determines outcome.

Cardiovascular Disease ≠ Luminal Obstruction

Block believes that assessing cardiovascular disease (CVD) solely by using cardiac catheterizations may be inappropriate in the dialysis population. Calcification of the central aorta is, according to Block, "perhaps the biggest culprit lesion when assessing for CVD risk in our patients." Calcification of the central aorta, even without luminal

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obstruction, is highly predictive of CVD. Block reminds us that the 100-fold increase in CVD risk seen in CKD patients is not accounted for by the "usual" risk factors, i.e., hypertension, smoking, diabetes, family history, hypercholesterolemia - yet these factors continue to dictate much of the prevention and research efforts. He strongly believes that it is necessary to assess our patients for central aortic calcification. Based on the work of Blacher and others, Block states that calcification causes the aorta to become "stiff" or "pipe-like" (3). This "stiffness", in turn, decreases the artery's capacitance or compliance, reduces coronary perfusion during diastole, and decreases the patient's ability to handle increased oxygen demand. The greater the degree of aortic "stiffness," the lower the chances of survival. Therefore, efforts to reduce aortic calcification are warranted and, in fact, overdue, according to Block.

Traditional Approaches to Treating Secondary Hyperparathyroidism

Traditionally, elevated PTH and abnormal serum calcium levels are managed with vitamin D, calcium supplements (either the initiation or discontinuation of), and perhaps changing the calcium concentration of the dialysate bath. Hyperphosphatemia is controlled with modified phosphorus intakes and binders.

Treating hyperparathyroidism with vitamin D can perpetuate hyperphosphatemia and hypercalcemia. However, vitamin D therapy should not be abandoned, even in this era of new treatment options for hyperparathyroidism. Block reports that vitamin D has many "non-classical" (i.e., non mineral related) roles that may benefit dialysis patients. He notes that vitamin D receptors occur diffusely throughout our bodies, having been found in blood vessel and immune system cells, and that vitamin D is involved in cell differentiation. "It is not a stretch to think that there are pathways through which Vitamin D might affect survival independent of mineral metabolism," says Block.

A historical cohort study involving over 60,000 patients found that the 36-month mortality rate was 16% lower among paricalcitol-treated patients than among calcitriol-treated patients (4). In those patients who switched from calcitriol to paricalcitol, survival rate was 73%, as compared with 64% among those who switched from paricalcitol to calcitriol. All results were statistically significant. In other words, patients who received paricalcitol appear to have a significant survival advantage over those who received calcitriol. Because this study was not a randomized controlled clinical trial, it does not prove that paricalcitol improves survival, but it is certainly an interesting observation that needs to be explained and confirmed. One possible explanation is that, since a vitamin D receptor (VDR) promoter has been found on the renin gene, vitamin D may be a negative regulator of renin expression. If this is true, and if low levels of 1,25 $(OH)_2$ vitamin D stimulate the renin-angiotensin system, as some researchers believe, then administration of vitamin D, perhaps regardless of PTH level, may be one potential pathway for affecting positive outcomes.

New Approaches to Treating Secondary Hyperparathyroidism

One half to two-thirds of patients have hyperparathyroidism by the time they have their first hemodialysis treatments. As the left side of figure 1 illustrates, hypocalcemia not only stimulates PTH secretion, it also stimulates hyperplasia of the parathyroid gland itself. As depicted on the right side of the diagram, vitamin D levels affect the transcription of PTH by partially determining the number of calcium-sensing receptors (CaR) on the parathyroid gland. Once PTH is synthesized, it is stored inside the cell. The most important regulator of its secretion is serum ionized calcium. Serum calcium is the "second-to-second" regulator of PTH secretion, mediated by the CaR. "The amount of PTH secreted is exquisitely regulated by the amount of serum calcium," says Block. Very small changes in ionized calcium produce abrupt changes in PTH levels. When serum ionized calcium levels increase to 1.21-1.22 mmol/L (i.e., the "set point"), PTH levels decline rapidly.

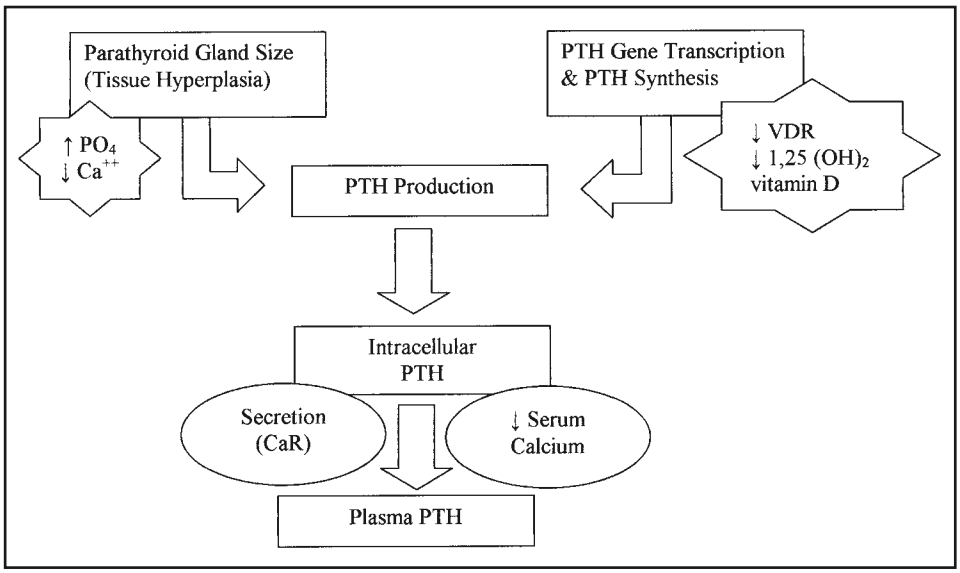

Figure 1. Relationships amoung serum calcium, serum phosphorus, calium sensing receptors (CaR), serum 1,25 $(OH)_2$ vitamin D, vitamin D receptors (VDR), PTH production, and PTH secretion.

The observation that the CaR resides on the surface of the parathyroid cell and detects changes in serum ionized calcium is the basis for the development of calcimimetics. "Calcimimetic agents increase the sensitivity of the calcium-sensing receptor to extracellular calcium ions" (6). These agents shift the calcium sensitivity curve to the left by modulating the sensitivity of the CaR. Therefore, at any given ionized calcium level, and in the presence of a calcimimetic agent, PTH secretion is much lower than would otherwise be the case.

A total of three Phase-3 trials have been conducted on the new calcimimetic agent Sensipar® (cinacalcet) – one in North American, one in Europe and one in

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Australia. In all the trials, a dramatic and abrupt fall in PTH level was observed after administration of the drug. This decline results because the CaR is "tricked into thinking" that the serum calcium level is higher than it truly is. Therefore, PTH secretion shuts off and PTH levels drop 60–80% 4 hours later, almost independently of the initial PTH level. In other words, if the PTH is 1000 pg/mL, it drops to 400 pg/mL; if it is 600 pg/mL, it drops to 240 pg/mL – a 60% drop in either case. Over 24 hours, the PTH level increases again, but not to pre-medication levels. After the next dose of Sensipar® the PTH decreases again. The overall result is intermittently high and low, or pulsating, PTH levels, which is very different from the gradual and continuous decrease in PTH levels seen with vitamin D administration. Vitamin D primarily effects transcription, whereas Sensipar® primarily affects secretion. There is some evidence from animal studies that this new medication may also be able to prevent parathyroid gland hyperplasia and PTH oversecretion in the early stages of CKD – yet another reason to be optimistic regarding the potential to improve long-term outcomes in patients.

The phase-3 trial that was conducted in North America, for which Block is the primary investigator, was recently published in the New England Journal of Medicine (6). Over 700 hemodialysis patients with inadequately controlled secondary hyperparathyroidism were randomly assigned to receive either cinacalcet or a placebo, and were followed for 26 weeks. The study protocol placed no restrictions on phosphate binder use (either type or dose); however, it did place restrictions on vitamin D use. Vitamin D dosages could be increased if serum calcium levels were < 8.4 mg/dL or if PTH increased by > 25%; and they could be decreased if the CaxP product increased or if there were sustained reductions in the PTH level to < 100 pg/mL. Results were that 43% of the patients receiving cinacalcet and 5% of those in the placebo group attained an iPTH level of < 250 pg/mL. (A primary endpoint of iPTH of < 250 pg/mL was chosen via the "educated guess" method, as the "optimal" PTH level for dialysis patients is still unknown). Mean PTH levels decreased 43% in the treatment group and increased 9% in the placebo group. PTH levels decreased fairly rapidly in the treatment group, and these lower levels were sustained over the six months of the clinical trial. CaxP product declined by 15% in the cinacalcet group but remained unchanged in the placebo group. CaxP product reductions were due to mild reductions in serum calcium and moderate reductions in serum phosphorus levels. Ninety percent of the patients who achieved the PTH goal also had a reduction in their CaxP product, i.e., attainment of target PTH levels was not at the expense of a worsening CaxP product! For all reported statistics, the P value was <0.001. In summary, Sensipar® lowers calcium, phosphorus and iPTH levels.

Serum calcium levels did not continue to decrease with continued treatment. Because Sensipar® shifts the sensitivity curve of the parathyroid gland, serum calcium falls as the dose is titrated upward, but a steady state in which calcium stabilizes at the low end of normal is eventually reached.

The safety data from all 1200 patients in all three phase-3 trials indicated no difference in adverse events between cinacalcet and placebo, no difference in serious adverse events, and no difference in deaths. Only one patient in each arm of the North American trial had symptomatic hypocalcemia. Some gastrointestinal side effects were reported – those of mild, transient nausea and vomiting. Nausea was not dose related, whereas vomiting did seem to be. In cases where these side effects occurred, a decrease in the dose until symptoms resolved, and then a gradual increase, was usually quite effective.

So Where Does Sensipar® Fit Into the Treatment Algorithm?

Sensipar® allows much greater flexibility with regard to vitamin D use. It may allow for:

- increases in the vitamin D dose to achieve better PTH control without causing hypercalcemia or hyperphosphatemia;
- continuous administration of the same dose of vitamin D, thereby avoiding the "on-off-on-off" phenomenon that is so detrimental to CKD patients;
- some reductions in vitamin D use.

The starting dose is 30 mg by mouth daily. Block emphasizes that it is not 30 mg only if the PTH is below a certain level and 60 mg if the PTH is very high – the starting dose is 30 mg, regardless of the PTH level. The dosage can then be titrated every 4 weeks, to a maximum dose of 180 mg daily. The pills come in 30, 60 and 90 mg strengths and they should be taken with food or shortly after a meal. The most common dose was 30 mg (about two-thirds of the patients in the trials reached K/DOQI targets with 30 or 60 mg). Therefore, it is possible that a 30 mg dose will allow the patient to achieve and maintain the PTH goals. One week after the initiation of the medication, or any dosage adjustment (including discontinuation of the drug), serum calcium and phosphorus levels should be measured. PTH levels should be obtained in one month. During maintenance, serum calcium and phosphorus should be checked once or twice a month, and PTH every 1-3 months. To properly ascertain the medication's effect, PTH levels should be checked 12-24 hours after the patient takes the medication, i.e., PTH should not be checked within 12 hours post-dose. Sensipar® is well absorbed, highly protein-bound and will not dialyze off. Other drugs do not particularly affect Sensipar® levels. Its half-life is 30-40 hours. A steady state is usually reached in about 7 days.

Many questions remain unanswered. What impact will Sensipar® have on clinical outcomes and on survival? What impact will normalizing phosphorus, calcium, their products, and PTH levels have on survival? What will the combined effect of cinacalcet and paricalcitrol be on bone? The "hard outcomes" will become more evident after more studies are conducted. However, based on the studies published thus far, as well as his experience in his own practice (in which he has 40 patients on Sensipar®), Block believes, and certainly hopes, that Sensipar® will help patients achieve the K/DOQI goals and the clinical outcomes desired: controlled PTH levels, prevention of elevated serum PO_4, normalization of serum calcium, and decreased cardiovascular morbidity and mortality.

[Note: As of April 2004, Sensipar® is not covered by Medicare because it is not administered during dialysis treatments.

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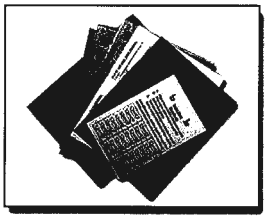
Integrative Medicine: Your Quick Reference Guide, 1st Edition, 1998	2
CLINICAL PRACTICE GUIDELINES	
Handbook of Dialysis, 3rd Edition	4
Handbook of Nutrition and the Kidney, 3rd Edition	5
Urea Kinetics in Nutritional Management of Pre-End Stage Renal Disease, 1993	2
Suggested Guidelines for Nutrition Care of Renal Patients, 2nd Edition	2
Suggested Guidelines for Nutrition Care of Renal Patients, 3rd Edition	2
K/DOQI Clinical Practice Guidelines - Bone Metabolism and Disease in Chronic Kidney Disease	2
Clinical Guide to Nutrition and End Stage Renal Disease, 2nd Edition	2
MEDICAL NUTRITION THERAPY	
Cultural Foods and Renal Diets for the Clinical Dietitian, Section 1, 1988 (Asian Indian, Black American, Chinese American, Filipino American, Japanese American, Mexican American, Southeast Asian)	3
Cultural Foods and Renal Diets: A Multilingual Guide for Renal Patients, Section II, 1988	3
National Renal Diet, ADA, 1st Edition Professional Guide, Kidney Disease, Diabetes, and Kidney Disease, Hemodialysis, Diabetes and Hemodialysis, Peritoneal Dialysis, Diabetes and Peritoneal Dialysis	1
National Renal Diet, 2nd Edition	2
Renal Handbook of Nutrition for Dietitians by CRN of New England, 1993	2
Manual of Clinical Dietetics, 5th Edition	1
Nutritional Management of Renal Disease, 2nd Ed.	1

Products of the ADA Renal Practice Group (Marketed through the Kidney Thinking Company) www.kidneythinking.com

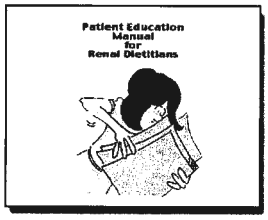
BONE STORE - A motivational program/game startup kit that includes all the materials needed to help start motivating dialysis patients toward better phosphorus control. This is a product of the ADA's Renal Practice Group. All materials in the kit are camera-ready for your copying pleasuer.

$10.00



PATIENT EDUCATION MANUAL - A collection of the "best of" the patient education materials included in the Renal Forum, a quarterly publication of the ADA's Renal Practice Group. All materials in the kit are camera-ready for your copying pleasure.

$10.00



EATING SIMPLY WITH RENAL DISEASE PAMPHLET - Brought to you by the Renal Practice Gruop of the American Dietetic Association is the long awaited simplified version of the National Renal Diet. It is a colorful, low literacy pamplet that provides some basic information about calories, protein, salt and diabetes. T0plet opens up to aproximately 14x22 (fits on the freezer door of the fridge). The inside sections includes the green light "go foods" and "yellow light cautions light foods" for potassium and phosphorus as well as information on fluids, protein and a sample menu sec tion. The pamphlets are packaged in packs of 25's.

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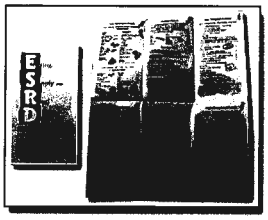

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Stipend Report from ...

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The cost in New York is about $350 for a 30-day supply of a 30 mg dose taken once a day. Block reports that the cost in Denver is $240. Sensipar® does have a "Sensipartners" access and reimbursement program to facilitate third party reimbursement and/or to provide low- or no-cost drugs to qualified patients. The website is www.sensipar.com (accessed June 17, 2004).]

Acknowledgements: I would like to thank the Renal Practice Group for awarding me the scholarship that allowed me to attend this informative, inspiring and empowering conference. I would also like to thank Geoffrey A. Block, MD for graciously providing me for his kind and invaluable assistance with the preparation of this manuscript.

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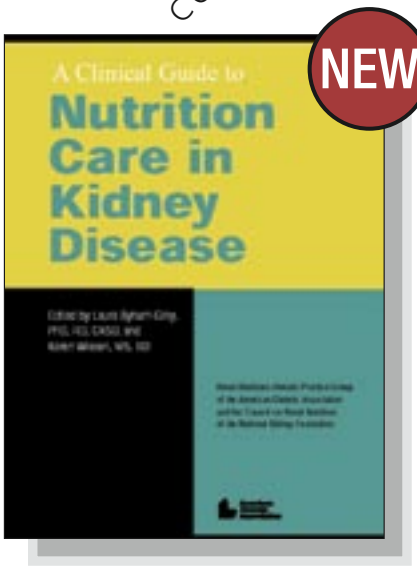
COMING THIS SEPTEMBER



NEW

A Clinical Guide to Nutrition Care in Kidney Disease

Renal Dietitians Dietetic Practice Group of the American Dietetic Association and the Council on Renal Nutrition of the National Kidney Foundation

Laura Byham-Gray, PhD, RD, CNSD, and Karen Wiesen, MS, RD, editors

A Clinical Guide to Nutrition Care in Kidney Disease *is the only text to explore the nutritional needs of patients at* ***all*** *stages and treatment modalities of kidney disease—from CKD in children and adults to patients receiving hemodialysis and peritoneal dialysis to kidney transplantation.*

Who should buy this book: Renal and clinical dietitians, nephrology nurses and technicians, and physicians will find this text an important reference for their day-to-day practice.

Topics include:

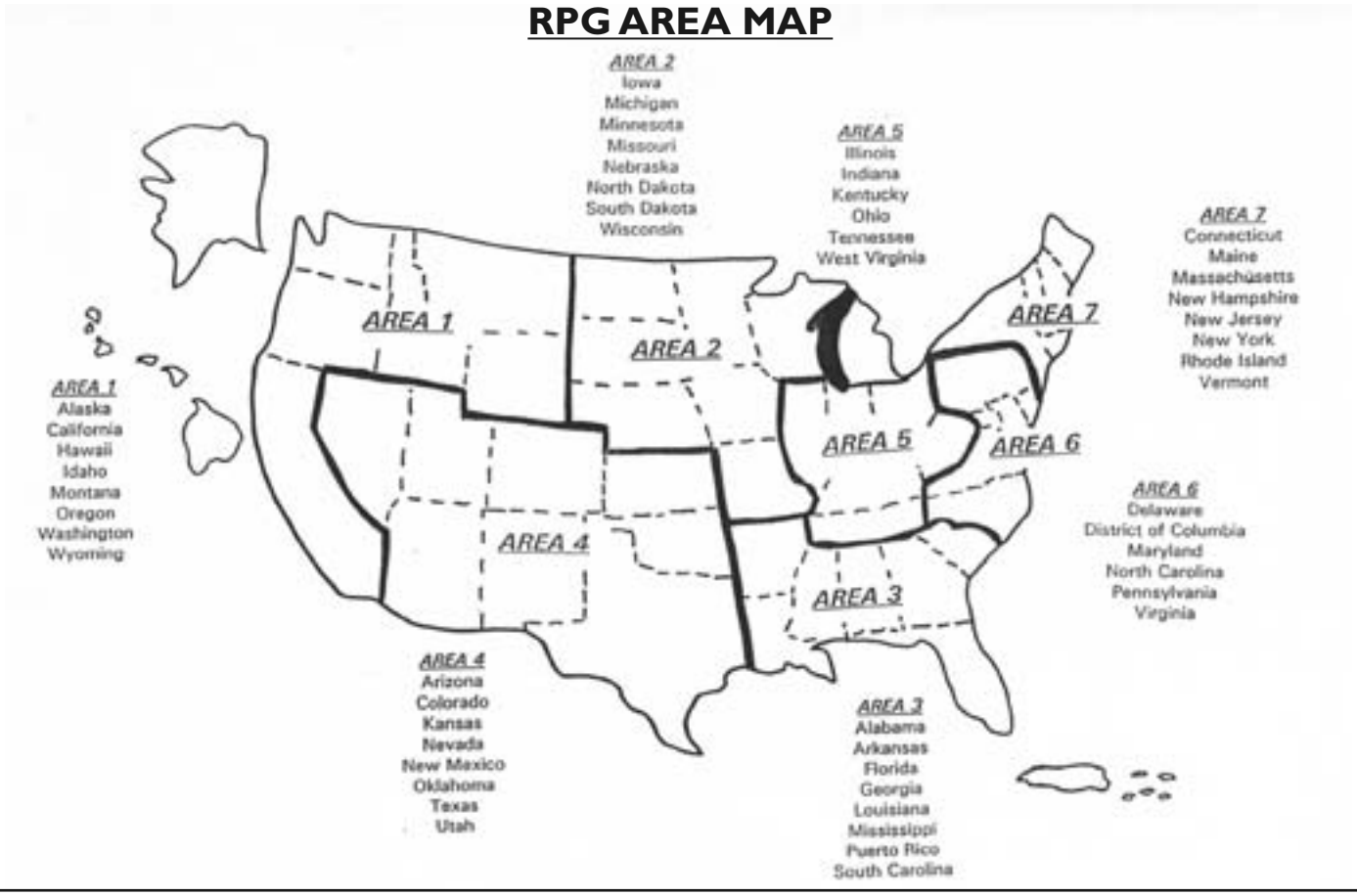
- Kidney pathophysiology—basic structure, function, and types of kidney failure
- Dyslipidemias—classification, assessment, and treatments, including lipid-lowering agents
- Acute renal failure—etiology, metabolic changes, and treatment, including CRRT
- Parenteral nutrition—components, administration, considerations for ARF and CKD patients, intradialytic parenteral nutrition
- Anemia management—identification and management
- Nocturnal home hemodialysis—treatment outcomes and nutrition intervention
- Co-morbidities such as diabetes and HIV/AIDS—recommended treatment options

For a current catalog of ADA's professional reference texts, visit ADA's Web site at **www.eatright.org/catalog** or contact ADA at **sales@eatright.org**, phone **800/877-1600, ext. 5000**, fax **312/899-4899**.

Available September 2004

Thought of the Month

RPG AREA MAP



RPG Chair Message

By Anne Ishmael, MS, RD, LD *Anne is a renal dietitian for Gambro Healthcare, San Jacinto in Houston,TX and can be reached at auto63347@hushmail.com.*

Seek and Find

"It is good to be a seeker but sooner or later you have to be a finder and then it is good to give what you have found, a gift into the world for whomever will accept it." Richard Bach. In the renal nutrition community, we have a number of professionals who reinvent themselves by learning and sharing their expertise.

As a seeker you may want to expand your renal reference library by adding the updated A Clinical Guide to Nutrition Care of Kidney Disease, 3rd Edition; Editors Laura Byhnam-Gray, L.D. Ph.D and Karen Wiesen, MS, RD, LD. [Available from the American Dietetic Association (ADA), 216 West Jackson Boulevard, Chicago, IL 60606-6995; 1-800-877-1600, ext. 5000. Or e-mail sales@eatright.org.] In it, you will find the gifts of many who volunteered their expertise: Patricia DiBenedetto Barba' MS, RD Gambro Healthcare, Andrea Bickford, RD Abbott Laboratories, Ronna Biesecker, PhD, RD Watson Pharmaceuticals, Pamela Charney, MS, RD, CNSD, Carolyn C. Cochran, MS, RD, CDE Dallas Transplant Institute, Lori Fedje, RD Pacific Northwest Renal Services/RCG, Paula J. Frost, RD, CSR Lee Street Dialysis, M. Patricia Fuhrman, MS, RD, CNSD, FADA Coram Healthcare, Paul Garney, MS, RD DaVita Med Center Dialysis, D. Jordi Goldstein-Fuchs, DSc, RD University of Nevada, School of Medicine, Janelle Gonyea, RD Mayo Clinic, Jill Lynn Goode, MS, RD BoneCare International, Inc., Kathleen Hunt, RD Castro Valley, Maria Karalis, MBA, RD, Pamela S. Kent, MS, RD, CSR Ohio Renal Care Group, Susan C. Knapp, MS, RD, CSR DaVita Broken Arrow Dialysis, Carol Liftman, MS, RD Franklin Dialysis Center/DaVita, Linda McCann, RD, CSR Satellite Healthcare, Maureen P. McCarthy, MPH, RD, CSR RCG/ Pacific Northwest Renal Services, Lesley L. McPhatter, MS, RD Lynchburg Nephrology Dialysis, Inc., Joni J. Pagenkemper, MS, MA, RD Creighton University Medical Center, Jessie Pavlinac, MS, RD, CSR Oregon Health & Science University, Julie Rock, MS, RD Abbott Laboratories, Sharon R. Schatz, MS, RD, CSR, CDE Gambro Healthcare, Donna Secker, MSc, RD The Hospital for Sick Children Toronto, Canada, Bruce Smith, MS, RD DaVita Northstar Dialysis, Jean Stover, RD Gambro Healthcare, and Naomi Stuart, MS, RD, East Carolina University East Carolina University Greenville. Please thank them for their contributions.

Another opportunity for learning is the updated RPG's website. Thanks to the collaboration of Catherine Goeddeke-Merickel, MS, RD, LD and Andrew Kamm from ADA, a new platform is available as a source of renal information. Access the site at www.renalnutrition.org.

The renal nutrition community is rich in opportunities for personal and professional growth. RPG has a number of ways for you to share your gifts. With your help, we will develop and maintain a current database of renal dietitians with expertise in media, writing, testifying, research, literature review, anemia or bone disease management etc. Authors are needed for the "World Outside of Renal" column. Interested? Then go to the 'members only' section of RPG's website, drill down to 'downloadable forms', select 'RPG Technical Resource Application', complete, and send to Sarah Carter as a way give back to your profession.

2005 Call for Nominations

American Dietetic Association
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The Nominating Committee is accepting nominations for enthusiastic and dedicated individuals for the American Dietetic Association, Commission on Dietetic Registration 2005 ballot positions and the Leadership Database. For candidate qualifications and skills sets, log onto the member-only web site. http://www.eatright.org/Member/85_8064.cfm and select Qualifications and Skill Set. The nomination form is available on the member-only web site at http://www.eatright.org/CallforNom05.doc. **Nominations for President-elect and Speaker-elect were due September 1, 2004; all other nominations are due October 10, 2004**

REMINDER: The 2005 elections will occur by electronic ballot only beginning February 1, 2005. Members will again have the opportunity to request a paper ballot.

CRN Chair Message

By Susan M. Reams, RD, CSR, LD
Susan is Chair of the Council on Renal Nutrition of NKF and is Chief Clinical Dietitian at Mercy Medical Center in Des Moines, IA. She can be reached at sreamswdm@prodigy.net

Let's Celebrate with CRN!

Do you recall some of the events, which took place in 1975? Some of you, however, may not have been born quite yet, but history is always fun to research. Just to refresh your memories, the following are a list of events, which occurred that year:

1. The Teamsters Union President, Jimmy Hoffa, was reported missing.
2. Arthur Ashe won at Wimbledon
3. Tiger Woods was born
4. "Saturday Night Live" and "The Wheel of Fortune" debuted.
5. The "Bee Gees" were the top disco band

However, the most important event of 1975 was the founding of the NKF-Council on Renal Nutrition. Our council became known as "CRN" and was formally launched in 1975 by an advisory group. CRN was one of the three professional councils recognized by the NKF during a 2-year formative period. The mission statement, which was developed in the early years of CRN, still stands strong today; "CRN strives to function as a professional council within the framework of the National Kidney Foundation, and network with other organizations to prevent and eradicate kidney and urological diseases."

Over the last 30 years, the CRN has maintained a focus of outstanding education for renal nutrition professionals and patients and continues to contribute to the wide variety of NKF programs and numerous nutrition-related research grants. CRN also contributes to the professionally sponsored programs of NKF such as the Annual NKF-Clinical Meetings.

The legislative efforts of the NKF have had strong support by its CRN members with numerous legislative-related issues over the past 30 years. Some of these include the prescription drug bill for transplant immunosuppressive therapy, the Medical Nutrition Therapy (MNT) bill, and the most recent Medicare prescription bill legislation. CRN communicates regularly with the Washington D.C. office of the NKF Scientific and Public Policy staff.

Since the founding of the CRN in 1975, a host of very talented renal dietitians have served the NKF to lead this professional council through the avenues of change in our professional status. These leaders have left behind them benchmarks and trails of dust for the advancement of renal nutrition. Membership has grown from the 25 initial members to almost 1700. The very first CRN affiliate chapter was formed in 1976, which was the CRN of New England, and as of 2004, we have 53 Council Chapter Affiliates! Starting in 1981, European chapters were added to the organization with their founding "father" of France.

In the early 1970s, dietitians had extremely limited nutrition information for their patients. In fact, very little was known about how to support the CKD patients requiring dialytic therapy. Much of the information, at that time, indicated that the diet for these individuals necessitated a very strict and largely unpalatable regimen. The results of investigative research were slowly evaluated with the goal of less regimented nutrition protocols for the dialysis patients.

As dialysis was becoming more available, research studies on a larger scale were more prominent, and over time, we are now at the crossroads of newer discoveries and treatment courses for the CKD patient population. Just look at how our terminologies for categorizing our patients has changed, and that's only a small piece of the CKD puzzle. The intensity of the renal dietitian's involvement and growth of the CRN membership can also be credited to the formation of the NKF-K/DOQI Standards for Clinical Practices and their expansions with renal MNT.

The first Chairperson of the CRN was Sherry Barlow, RD, from Houston, Texas. Her leadership skills set the pace for those that followed and led us up to where we are today. These early leaders realized that their first responsibility was to share credible information and thus, formed committees on communication, membership and their involvement with the scientific programs.

Almost immediately, the CRN published its first format for communication, "The CRN Newsletter." Over the years, the evolution of this newsletter developed into the "CRN News", then the "CRN Quarterly," the "CRN News and Briefs," that was connected with an outward branch to the successful Journal of Renal Nutrition. More recently, this newsletter evolution has settled into the "Renalink", which is a polished publication for all three NKF Councils, CRN, CNNT, and CNSW.

Another marvelous feature of the NKF-CRN has been through the development of the CRN Website and the continuous activities of its cyberNephrology committee with a sponsored on-line discussion group for renal dietitians known as RenalRD. This is a list serve where renal dietitians and other interested healthcare team members can ask questions of each other and share discussion with problem solving and fact finding. This communication forum has proven to be a valuable tool to those who choose to participate and has become a further endorsement by the NKF to enhance communication worldwide.

CRN has also established a compilation of recognition awards. These include:

1. The Recognized Council Awards (RCA)
2. The Recognized Renal Dietitian Award (RRD)
3. The CRN-Outstanding Service Award
4. The Dr. Joel Kopple Award for a selected individual that upholds the philosophies of the NKF-CRN and practices in some branch of renal nutrition.

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CRN Chairman Message

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CRN members have also developed an expanded variety of professional and patient/public education materials over the years. Some have been published for purchase purposes and others are available on-line through the NKF-CRN website.

The CRN has recently re-teamed with the American Dietetic Association-Renal Practice Group to collaborate our efforts in developing professional education materials and updating previously published materials. The latest of these includes the recently published monograph, "The Renal Dietitian's Standards for Clinical Practice," and the 3rd Edition of *"A Clinical Guide to Nutrition Care in Kidney Disease"*, which became available during the Summer of 2004.

CRN was involved with funding the CRN Research Grant for its members since its inception in 1985. Within 20 years, nearly 30 renal nutrition studies were funded with outstanding results and manuscript publications. As of recent, the newest cyberNephrology communication avenue was launched for those individuals who are interested in renal nutrition research, the "CRN Research Bulletin Board", which can be found on the NKF-CRN website.

As the CRN moves towards celebrating its 30th founding celebration, plans have begun for a great list of events for the Washington D.C., Clinical Meetings in 2005 (CM.05) The current CRN-Executive Committee and CRN-Program planning committee cordially invites its members and other readers of this publication to earmark your calendar for May 4-8, 2005 to join us with all of our planned festivities.
Like the other councils, the CRN has established a commitment to improve the quality of life for our CKD patient population. We continue to pull together to bring a better understanding of the current medical, psychosocial and nutrition-related issues, which confront us on a daily basis.

Please join us in congratulating CRN for 30 years of excellent work!

With my Best!
Susan M. Reams, RD, CSR, LD
CRN Chairperson

Nutrition and Nephrolithiasis

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Nominations Wanted for American Dietetic Association / Renal Practice Group

The Nominating Committee is accepting nominations for enthusiastic and dedicated individuals for the 2005 ADA/RPG Executive Committee. The positions open are for Chair-Elect, Treasurer, and Nominating Committee.

Please refer to RPG's website at www.renalnutrition.org for descriptions of these positions and the nominations form. Deadline for submitting nominations is October 1. Please contact Susan Knapp, Nominating Committee Chair, with questions regarding nominations at sknapp@davita.com or
918-585-1977 Ext. 374.

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Dialysis Facility Compare Gets a New Look: from the Center for Medicare and Medicaid Service (CMS).

June 29, 2004: CMS is pleased to announce the release of Dialysis Facility Compare in the "next generation compare" format that provides users with simple navigation within the tool. Users will be able to search for dialysis facilities by State, County, City, Zip or Name. Proximity searches are now available when searches are made on City or Zip. In addition, a search can be narrowed to specific services offered by dialysis facilities. Users will find new text explanations and a lower reading level. Users can also search for additional web resources by population (CKD, Children, Transplant, etc.). Dialysis Facility Compare enables users to compare information on three facility specific quality measures: anemia, hemodialysis adequacy, and patient survival.

For more information, please visit the website at www.medicare.gov/dialysis/home.asp (accessed August 14, 2004) or contact Eileen Zerhusen at (410) 786-7803.

Kidney Friendly Food Facts
Citrus - Section I

By Sharon Schatz, MS, RD, CSR, CDE. *Sharon is a renal dietitian with Gambro Healthcare in Lumberton, NJ. She can be reached at Srsmsrd@aol.com or sharon.schatz@us.gambro.com.*

The topic of citrus in the renal diet has many aspects to consider. Patients on potassium-(K+) restricted diets are often advised to avoid citrus in their diets with the exception of lemons and limes. However, the renal patient may actually tolerate potassium from some citrus fruits. A person may want to limit the quantity consumed and work the fruit into the total K+ intake. It is therefore important to know the varieties of citrus fruits and their K+ content. Understanding the derivation of citrus helps the dietitian understand why the K+ content varies. It also provides clues to possible K+ content when nutrient data analysis is unavailable. A table of K+ content in citrus foods is included. Lemons and limes will be discussed in a future column.

Citrus is a botanical genus of the Rutaceae family that is most likely native to the subtropical and tropical regions of Asia and the Malay Archipelago. It has undergone evolution and hybridization to become the fruit that is most common to North America. A detailed history is available at http://lib.ucr.edu/agnic/webber/Vol1/Chapter1.htm, *History and Development of the Citrus Industry* by Herbert J. Webber in The Citrus Industry, University of California, Division of Agricultural Science, 1967. It's a fascinating journey from Biblical times to its spread throughout Europe. Columbus introduced citrus into the Americas in 1493, and Spanish explorers brought the fruits to Florida sometime around 1565. Arizona is actually an older citrus-growing state than California, dating back to 1707. California oranges probably date back to the establishment of the first mission at San Diego in 1769.

There are four defined horticultural groups of principal commercial importance: oranges, mandarins, pummelos and grapefruits, and the common acid members which has the three subgroups of citrons, lemons, and limes. For detailed information refer to http://lib.ucr.edu/agnic/webber/Vol1/Chapter4.html, *Horticultural Varieties of Citrus* by Robert W. Hodgson. However, the four original wild species from which the main hybrid species are derived are: Citrus medica (citron), Citrus grandis (pummelo, pamplemousse, shaddock), Citrus reticulata (mandarin, Satsuma, and tangerine), and Citrus aurantifolia (lime) (refer to http://www.steve.wagar.com/stuff/citrus.htm). It should be noted that there is disagreement among botanists, which further complicates the subject.

A trip through supermarket aisles can be confusing as there are many hybrid fruits. Some of these are:

- Orange = pummelo x mandarin
- Lemon = lime x citron
- Common grapefruit = sweet orange x pummelo
- Tangelo (Ugli fruit is one variety) = grapefruit x mandarin
- Tangor (Temple orange is one variety) = mandarin x sweet orange
- Tahiti lime, seedless lime, or Persian lime = lime x citron or possibly lime x lemon
- Chinese lemon, Mandarin lemon, or Mandarin lime = mandarin x lime

The primary members of the orange group are the sweet orange and the bitter orange. The most common is the blood orange of the Mediterranean and its many varieties. The pigmented or blood orange is more predominant in Europe. Bitter oranges are used more often in marmalade. The four types of Florida oranges are Navel, Valencia, Hamlin, and Pineapple. The Temple orange is actually a tangor.

In this country, we tend to use the term tangerine for the group of fruit that is actually Mandarin orange. The three classes are Mandarin, tangerine, and Satsuma. We are more familiar with canned Mandarin oranges (or satsumas) instead of fresh ones. One 11-ounce can of these sections provides 1¼ cups of fruit. Canned mandarin orange sections can be used in fruit salads and desserts, such as in gelatin or on cakes, or they can add zip to salads made with chicken or turkey. Clementines, a class of tangerine imported from Spain, are usually plentiful around Christmas time. Dancy is the leading tangerine in the United States. The Robinson tangerine, actually a hybrid of a Clementine tangerine with an Orlando tangelo, is in the tangerine class and may have slightly more K+. The satsuma originated in Japan but some are grown in California. Honey tangerines, also known as Murcotts, are tangors.

Tangelos were probably first crossbred in California in 1898. Their shape is irregular, the pulp is often colorful, the flavor is tart, and it is very juicy. Minneolas, a cross between the Duncan grapefruit and Dancy tangerine, have a distinctive bulge at their stem end, deep red-orange peel, and orange pulp. Ugli fruit has light-yellow peel or mottled green skin and light-orange pulp. Other varieties include Nocatee, Orlando, Sampson, Seminole, and Thornton.

Pummelos, also known as shaddocks, are larger than grapefruit. The yellow, dimpled skin is thick; and the flesh is pinkish yellow with a sharp, refreshing flavor. These are usually found at specialty food markets.

Grapefruits, originally called "pomelo", came from the West Indies. The term "grapefruit" was coined in 1814 in Jamaica where it was referred to as a special and smaller kind of shaddock (pummelo) whose flavor somewhat resembled that of grapes. The derivation may also be from the fact that the fruits commonly occur in small clusters like grapes rather than singly. The Marsh and the Duncan are the two most commercially available types of the ordinary grapefruit, commonly referred to as the white grapefruit in the produce trade to distinguish it from pigmented varieties. The conditions responsible for the development of lycopene in pink or

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red-pigmented grapefruits are not well understood but seem to be heat related. The most important pigmented varieties are Foster (Foster Pink), Redblush (Ruby, Red Marsh, Red Seedless), and Thompson (Pink Marsh). Sweetie is a grapefruit pummelo hybrid released in 1984 by the Citrus Marketing Board in Israel.

It is important to know what medications the patient takes when advising use of grapefruit and grapefruit juice as they interact with metabolic pathways of various drugs. Detailed information regarding this is available at http://www.powernetdesign.com/grapefruit and http://www.pharmacytimes.com/article.cfm?ID=258.

For a pdf hand-out, go to http://www.powernetdesign.com/grapefruit/general/GJDIsummary.pdf .

Additional information regarding citrus is available:

http://www.thefruitpages.com; Fruits of Warm Climates by Julia F. Morton at http://www.hort.purdue.edu/newcrop/morton; Produce Pete at http://www.producepete.com;

Market Information in the Commodities Area at http://r0.unctad.org/infocomm/anglais/orange/sitemap.htm; Florida Department of Citrus at http://www.floridajuice.com/floridacitrus; Sunkist Foodservice Seasonal Products at http://www.sunkistfs.com/products/seasonal.asp.

These books are great references:

Pijpers D, Constant JG, and Jansen K. *The Complete Book of Fruit.* Gallery Books, NY, 1986; Whiteman, K and Mayhew M. *The World Encyclopedia of Fruit.* Lorenz Books, NY, 1998; and *Dr. Richter's Fresh Produce Guide,* which can be ordered through Try-Foods International, Inc, http://204.117.192.83/consumer.asp. (Editor's note: All websites were accessed July 31, 2004).

K+ Content of Selected Citrus Fruits

Amount	Item	Wt (g)	K+ mg
.5 cup	grapefruit sections in juice, canned	125	210
.5 cup	grapefruit juice canned	124	188
.5 each	grapefruit, pink/red	123	180
.5 each	grapefruit, white 3 3/4" diam	118	174
.5 cup	grapefruit, pink/red, sections fresh	115	169
.5 cup	grapefruit sections, canned in water	122	161
.5 cup	grapefruit sections, raw	115	155
.5 cup	Mandarin oranges canned in juice	125	165
1 each	Mandarin orange, large 2.5" diam	98	153
1 each	Mandarin orange, med 2 3/8" diam	84	131
.5 cup	Mandarin oranges in juice, drained	95	128
1 each	Mandarin orange, small 2.25" diam	70	109
.5 cup	Mandarin oranges in light syrup	126	98
.5 cup	orange juice, fresh	124	248
1 each	orange, Florida, 2 5/8" diam	141	238
1 each	orange, Calif navel, 2 7/8" diam	140	232
1 each	orange. Calif Valencia, 2 5/8" diam	120	216
.5 cup	orange sections, fresh	90	162
.5 each	pummelo, raw	305	657
.5 cup	pummelo sections, raw	95	205
1 each	tangelo, 2 3/8" diam	96	173
.5 cup	tangelo sections, fresh	90	162
1 each	tangerine, large, 2.5" diam	98	153
1 each	tangerine, small, 2.25" diam	70	109

Rehab Corner
The Fantastic Five

By Stephanie McIntyre, RD *Stephanie is Patient Rehab Director for Renal Care Group, Inc. and the renal dietitian at Renal Care Group-Phoenix, Phoenix, AZ. She may be reached at smcintyre@renalcaregroup.com or 602-340-9050.*

The Fantastic Five

"Got Rehab?" Are you actively involved and promoting rehab programs in your facility? Do you have a program that utilizes all of the "5 E's"? Rehab helps our patients return to stable health, positive outlook, and enjoyable activities that make them feel better physically and mentally. A successful rehabilitation program uses medical treatment, counseling, education, nutrition, and exercise with the help from the "5 E's": Encouragement, Education, Exercise, Employment, and Evaluation.

The "5 E's" were developed through a combined effort among patients, dialysis professionals and researchers – Life Options Advisory Council. Utilizing these "5 E's" can help simplify your approach to helping patients with their seemingly complex issues. In one way or another, the "5 E's" provide numerous means to make that connection with patients to help them adjust and adapt to dialysis faster. This article intends to give a brief review of the "5 E's" and encourage you to be involved in promoting them in your facility.

Encouragement

Encouragement is the first step to renal rehabilitation. Encouragement involves having a positive attitude and believing our patients can succeed and live well with kidney disease. Encouragement addresses the questions, "What do you look forward to doing each day?" and, "What gets you through the ups and downs?" There are a variety of rehab encouragement activities you may already be doing in your clinic – bulletin boards, support groups, sharing patient success stories, patient of month type activities, etc. Don't forget that patients can also encourage each other, so don't rely solely on staff.

Education

Education can help patients understand their disease and ways in which they can participate in their own care. Knowledge is power. Patients lead their healthcare team whether they realize it or not. Their decisions outside the dialysis clinic affect how well they will do with dialysis. What and how we get information to patients is critical. Make education fun and simple. Create opportunities for the patients to be actively involved through contests or games. Active participation helps the learner to transfer information into long-term memory. Examples of education rehab activities include monthly or quarterly patient and staff education (bulletin boards, contests, handouts, videos, etc), CQI projects on specific themes, patient education/support groups, family education, education lobby fair, etc.

Exercise

Research has demonstrated that exercise has powerful positive effects on the physical functioning and emotional well-being of dialysis patients. Lack of activity and co-morbidities such as diabetes may contribute to the fact that heart disease is the highest cause of death for dialysis patients. It's no wonder exercise is the rehab activity we look to first to improve patients' health. It has often been a catalyst for patients to make additional improvements in their life, especially when they see their energy level increase. We have featured many articles on dietitians involved in exercise programs and their experiences to help create successful programs. Dietitians play a valuable role, whether or not they are actually leading these types of programs. Here are some examples of exercise rehab activities:

- Patient and staff education on exercise
- Search your community for local fitness centers that offer discounts to your patients
- Invite a fitness expert to visit your facility
- Initiate or maintain facility exercise programs

Employment

Employment is more than just full or part-time employment. It is really about asking our patients, "How do you spend your time away from the dialysis facility?" Patients' habits and activities (or lack of) influence their eating habits and nutritional status. All staff, including the dietitian, can encourage patients to stay active, work, go back to school, pursue hobbies, volunteer, etc. Some employment rehab activities may assist the dietitian within the facility, such as highlighting working patients or patients who participate in volunteer projects, and encouraging hobbies and participation in art contests, fairs, etc.

Evaluation

Evaluation of patient outcomes is an integral part of all healthcare delivery, including successful rehabilitation programming. There are some simple ways to measure outcomes by monitoring labs, tracking patients' treatments, pre- and post-quizzes, and other evaluation tools such as quality of life surveys. As dietitians, we are constantly involved in formal evaluation through lab reports, care plans, but also informally when we are just talking with our patients.

Some evaluation rehab activities may include using quality of life surveys to help measure outcomes as part of your rehab program, looking at ways to include patients and families in care planning, evaluating reading level of educational materials, being a part of a CQI team in your facility, etc.

Rehab plays a significant role in our patients' quality of life and encourages a positive atmosphere in our facilities. There are many ways you can be involved in rehab. You may decide to lead a rehab activity or play a supporting role on a team. Try to incorporate all of the "5 E's" in your facility. This will enable you to reach all of your patients and empower them to live long and live well with dialysis.

For more information about the "5 E's", visit Life Options Rehabilitation Program's website, www.lifeoptions.org (accessed June 14, 2004).

Do you have a story highlighting the role of renal dietitians in the rehab process? Contact Stephanie McIntyre for an interview that may lead to an article in this column.

Missing issues?
Trying to de-clutter your office?

RPG's website (www.renalnutrition.org)
contains back issues in pdf format from Summer 2003.

The 'members only' section also contains:
Nationwide area coordinator map with coordinator introductions
Application for the conference and meeting Stipend Award
Application for the Outstanding Service Award
Mentor form, Reprints order form
ADA's House of Delegates and committee reports
Renal Practice Group's Resource Guide
AND MUCH MORE!

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