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Nutrition and Nephrolithiasis

By Mansi Mehta. *Mansi was an Outpatient Renal Dietetic Technician at Loyola University Medical Center. She is currently a full-time dietetic internship student at Loyola University Chicago and may be contacted at mmehta2@luc.edu.*

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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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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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

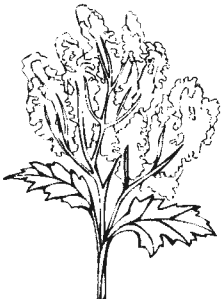
Goals	Avoid or Limit
Ample hydration: 3 - 4 L/day Urine volume goal: 2.0 - 2.5 L/day 250 ml of fluid at each meal, between meals, at bedtime, and when arising to void at night. At least one-half of fluid taken as water. Moderate amounts of tea (about 2 cups/day), diluted, and with milk	Grapefruit juice 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

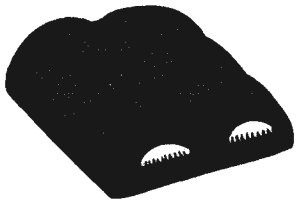
Rhubarb



Berries

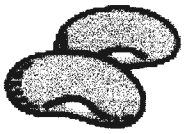
Spinach

Beets



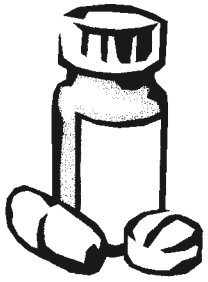
Chocolate & Cocoa

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