

**New Evidence in the Management of Chronic Renal Failure**  
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**Nutrition**

**Diet choices:** The first management modification in animals with mild to moderate renal failure is utilization of a diet formulated to have restricted protein, phosphorus, and salt. There are several options available. The most common diet is Hill's K//D (canned and dry), Waltham's Renal LP, Purina CNM NF and Eukanuba Feline Multi-Stage Renal Diet (canned and dry) or Early or Late Stage Renal Diet for dogs. The lower protein content of any of these diets decreases the patient's BUN. Renal diets prolong survival in dogs by almost double, and decrease the number of hospitalizations for uremic decompensation. Renal diets also dramatically prolong survival in cats. Dietary therapy in patients with moderate to severe uremia should yield a BUN:Creatinine ratio in the range of 10 to 15 (normal 15-20), and alterations in this ratio can indicate gastrointestinal bleeding or poor patient or client compliance with dietary recommendations.

Phosphate restriction in the diet will help reduce renal secondary hyperparathyroidism and renal mineralization, and can thus help slow progression and provide a better quality of life. Decreasing the ratio of omega-6 to omega-3 essential fatty acids may have some renoprotective effect; this area deserves further study.

**Anorexia:** Many patients with CRF have a diminished appetite due to the illness, and a sudden diet change is problematic. Inadequate caloric intake forces the animal to metabolize muscle for energy, which is, in effect, a high protein diet. Because of the decrease in renal function, dietary changes need to be made over 2 to 4 weeks to allow the kidneys time to adapt. Trying to introduce the new diet to a patient who is still hospitalized and is still severely uremic may lead to a food aversion, so diet changes should usually be reserved for those patients who are home and have resumed eating. Changing to the renal diet may take 6-8 weeks, and weekly phone calls help to encourage clients to remain compliant. In the interim, several methods of nutritional support exist.

**Enteral Feeding:** Syringe feeding a soft diet like A/D or MaxCal is acceptable for some patients, but it is very difficult to ensure adequate caloric intake by this method due to resistance by the patient. Nasoesophageal or nasogastric feeding tubes are temporary support methods that allow adequate caloric administration. Because the size of a NE tube is limited (5 French), only a liquid diet can be fed through them. Clinicare or Clinicare RF Liquid diet is formulated for this method of administration. Other liquid diets available in the grocery store or human pharmacy must be evaluated for protein content and suitability. The volume of the diet necessary for adequate calories can be a limiting factor. For most animals, after an acclimation period, to allow the stomach a chance to expand to accept the volume and to avoid diarrhea from the diet change, the fluid load is helpful in maintaining hydration and diuresis. Practically speaking, however, these diets must be fed 5 to 6 times a day, which can be difficult for the owner. For animals that do not begin eating adequately within a short period of time, an esophagostomy tube or a PEG tube should be considered. By placing one of these tubes, the animal's nutritional needs can be met by 3 to 4 times a day feedings of a blenderized diet (i.e. canned K/D mixed with ½ to 1 part water). The PEG tube will not interfere with the animal's attempts to eat on its own, and can be used to supplement animals with a fair but inadequate appetite. A PEG tube can remain in place long term. An additional advantage of having a feeding tube is that it simplifies administration of the multiple medications that are frequently prescribed.

**Appetite Stimulants:** Appetite stimulants can be used as a temporary support, to encourage the transition back to home life. One dose of intravenous diazepam (0.5 mg) can induce feeding behavior in many cats. The food needs to be immediately available (onset of action is about 10 seconds), and for some cats, this one dose can “jump-start” the appetite sufficiently to result in sustained eating. If the cat eats with the Valium trial, but results are short-lived, an oral Valium derivative, oxazepam (Serax, 2 mg PO BID) can be used. Unfortunately, Serax is supplied as 15 mg tablets, making dosing inconvenient. Because oxazepam is a controlled drug, cyproheptadine (Periactin) is utilized more frequently. This is an antihistamine that is fairly effective at stimulating the appetite of cats. It is easier to dose than Serax, in that it is supplied as 4 mg tablets, with a dose of 2-4 mg per cat PO SID to BID. Remeron (Mirtazapine) has been used in some cats (3.75 mg/cat twice weekly), although may cause some CNS side effects.

Supplementation of the water-soluble vitamins (particularly B vitamins) should not be necessary in cats consuming an adequate amount of a balanced diet. However, because losses may be greater with polyuria and many of these cats have a decreased intake, **multivitamins** (i.e. Pet-Tinic, Favor) can be used judiciously, if care is taken to avoid oversupplementation of fat-soluble vitamins.

### **Proteinuria**

In almost every model of proteinuria renal disease in humans, treatment with an ACE inhibitor (i.e., enalapril or benazepril) improved outcome. The presence of proteinuria predicts faster progression in dogs with renal failure. Treatment of dogs with glomerulonephritis with enalapril slowed progression and reduced proteinuria. In cats, a urine protein:creatinine ratio (UPC) over 0.43 predicts shorter survival times, and treatment of cats with a UPC over 1 with benazepril improves survival compared to placebo. However, only a small number of cats with renal failure have proteinuria. The initial database for any dog or cat diagnosed with renal failure should include an accurate assessment of proteinuria (i.e., UPC). If the UPC is > 0.5 in dogs or > 0.4 in cats, an ACE inhibitor should be considered. Relative contraindications to ACE inhibitor therapy include severe azotemia or hyperkalemia.

### **Fluid Therapy**

The ability to concentrate urine is destroyed with CRF, leading to an obligate polyuria. Because of the urinary water loss, polydipsia is necessary to avoid dehydration. With advancing CRF, some cats are unwilling or unable to drink a sufficient volume of water to replace the deficits, and thus they become dehydrated. Dehydration impairs the renal toxin removal capabilities, adding a prerenal azotemia to the renal azotemia. The dehydration and the azotemia can create nausea that diminishes a cat's desire to drink, exacerbating the situation. Regular fluid administration can help alleviate this problem. Encouraging water consumption in early stages may be sufficient. This can be accomplished by providing fresh cold water (or settled room temperature water, according to the individual cat's preferences), multiple water bowls, running water, or even flavored water (water from tuna, etc.) If using anything other than fresh water, pay attention to the salt and electrolyte content.

If voluntary intake is insufficient, subcutaneous fluids may be helpful. In addition to any diuresis it may provide, it can help “smooth over” any periods of stress when the animal's water intake may decrease (visitors in house, owners away, boarding, anorexia from other disease) or when there is extra fluid loss (vomiting, diarrhea). Lactated Ringer's Solution or 0.9% saline are appropriate choices, although either can lead to hypernatremia. Dextrose is irritating when given subcutaneously, so it should be avoided. There is no established dose; rather, dose is dependent on the severity of azotemia and condition of the patient. Alterations in dose are based on subjective response to treatment, as determined by the owner's perception of quality of life, activity level, appetite, and by clinical parameters such as stabilization of weight, creatinine, BUN, state of hydration, etc. For an average sized cat, a reasonable starting dose is 100-150 ml every day to every other day. Clients can learn to administer the fluids at home in most cases. Active cats may need to be restrained in an open topped

box or carrier, or wrapped in a towel to allow one person to administer the dose. The occasional cat is too fractious for subcutaneous fluid therapy.

## Medications

**Potassium:** Hypokalemia is a common consequence of polyuric CRF. It develops because of potassium wasting in the kidney with the polyuria, and because of inappetence and inadequate dietary intake. Signs of hypokalemia are muscle weakness, which differentially affects the muscles of the head and neck first. These cats may present with profound cervical ventroflexion. At lower potassium levels (usually less than 2 mEq/L) respiratory function can be diminished because of paralysis of the intercostal muscles. The chronic hypokalemia promotes metabolic acidosis, and both the hypokalemia and acidosis promote progression of renal damage.

Potassium gluconate is an oral potassium supplement (2 to 6 mEq/cat/day). Tumul-K is convenient for cats, and comes as powder, tablets, and gel. Potassium gluconate tablets may be purchased at many health food stores. Kaon elixir is a liquid potassium supplement made for humans. It comes in a variety of flavors (grape, cherry) for mixing like "Kool-Aid." It is intended to be diluted prior to use because it is unpalatable, but this makes it less convenient for dosing. Potassium citrate (e.g. Polycitra-K or Urocit-K) can be used as a potassium supplement. Potassium chloride (Lite Salt) is not recommended for oral administration because of its extreme bitterness and unpalatability.

**Histamine Blockers:** Gastric ulceration is a common problem with chronic renal failure. Histamine blockers are routinely used as anti-ulcer medications and as antiemetics. Cimetidine (Tagamet) is associated with several drug interactions. The dose is 5-10 mg/kg TID. Dosage needs to be reduced with renal failure from q 8 hours to q 12 hours. Ranitidine (Zantac) is dosed once a day in renal failure, at a dose of 2 mg/kg. Famotidine (Pepcid) also is a once a day medication. It has fewer drug interactions than cimetidine or ranitidine. The dose for famotidine is 0.5 - 1.0 mg/kg PO SID. These are over the counter medications.

**Other GI Drugs:** Centrally acting antiemetics like metoclopramide (Reglan) can be used as needed, at a dose of 0.1 to 0.5 mg/kg PO TID. Sucralfate (Carafate) is a gastric mucosal protectant. It works best in an acid environment, so it should be given 30 minutes prior to any antacid. It will interfere with absorption of other medications, so medications other than antacids should be given 30 minutes prior to sucralfate administration.

**Alkalinization Therapy:** Alkalinizing therapy is indicated if the bicarbonate level is less than 16 mEq/L, or the pH is persistently below 7.2. Bicarbonate (8-12 mg/kg PO BID to TID) is supplied as tablets (325 mg) or as baking soda, which can be mixed in water and kept in the refrigerator. Most cats do not tolerate this therapy, due to intestinal gas production. An alternative is citrate, which provides buffering for the metabolic acidosis. It is supplied as sodium citrate or potassium citrate (dose, 50-75 mg/kg PO q 12 hours). Using the potassium citrate formulation (Polycitra-K, Urocit-K), hypokalemia can be addressed also.

**Phosphate Binders:** Because the ability to excrete phosphate diminishes with decreased GFR, hyperphosphatemia is commonly seen with CRF. Because methods to increase excretion (i.e., increasing GFR by fluid therapy) are usually of limited efficiency, phosphate binders are used to prevent absorption of phosphorous that is ingested with meals. Salts of aluminum (aluminum hydroxide, aluminum carbonate, aluminum oxide) are available as over the counter antacids. Liquid forms are more effective than tablets or capsules. Excessive absorption of aluminum can lead to toxicity, including anemia and

neurologic symptoms. Calcium based phosphate binders avoid the aluminum toxicity issue, but predispose the patient to hypercalcemia. Of the calcium based products available, calcium acetate is superior due to reduced risk of hypercalcemia with retained efficacy. Dosage of phosphate binders should be titrated based on serum phosphate concentration, but 30-90 mg/kg/day is a starting dose for the aluminum containing preparations. A newer polymer, selavamer (Renagel) has shown promise in people, but experience in animals is limited, and the drug is currently very expensive. Epatikin is a combination of chitan and calcium carbonate. It appears palatable and effective.

**Calcium metabolism:** Calcium is poorly absorbed from the GI tract whereas phosphate is almost entirely absorbed. Absorption of both is enhanced by vitamin D. Most dietary calcium, by virtue of its poor absorption, is excreted via the GI tract. Phosphate, on the other hand is mostly renally excreted. The final activation step in vitamin D (calcitriol, or 1,25-hydroxycholecalciferol) synthesis occurs in the kidney.

Mild hypocalcemia induces PTH secretion, which induces the kidney to increase conversion of 25-hydroxycholecalciferol to calcitriol. Calcitriol increases serum calcium concentration by increasing intestinal absorption of calcium and increasing resorption from bone stores. The increased serum calcium concentration then feeds back on PTH production, suppressing it.

In renal failure, the diminished GFR leads to phosphate retention. Because of the law of mass action, the increase in phosphate decreases the calcium level, stimulating PTH. PTH increases calcitriol, which increases serum calcium and decreases phosphate. With renal failure, the kidney is unable to make calcitriol. The bone is less responsive to PTH in the face of the hyperphosphatemia. Because of the lack of calcitriol, there is no negative feedback on the parathyroid gland, and the parathyroid gland hypertrophies, causing renal secondary hyperparathyroidism. This is relatively common. A relatively uncommon manifestation of renal failure is renal tertiary hyperparathyroidism, in which the parathyroid gland hyperplasia becomes adenomatous and secretes PTH without regulation. This condition is associated with hypercalcemia, and would be treated in humans via parathyroidectomy.

Signs of renal secondary hyperparathyroidism are uncommon, and are more frequently seen in young animals with renal dysplasia. Bones of the skull are preferentially affected, causing rubber jaw, or mandibular fractures. Soft tissue mineralization occurs predominantly in the lungs, kidneys, arteries, stomach and myocardium.

**Calcitriol:** Calcitriol directly blocks PTH secretion and lowers the setpoint for calcium to decrease production of PTH. It also reverses parathyroid gland hyperplasia and increases GI absorption of calcium, which decreases PTH. However, to avoid massive soft tissue mineralization with calcitriol use, serum phosphate levels MUST be normalized. Phosphate binders are used to prevent absorption of phosphate in the diet. With calcitriol, hypercalcemia can occur if used in conjunction with a calcium containing phosphate binder. Accurate PTH assays exist, and it is highly recommended that the PTH level be checked prior to instituting therapy. In dogs, the starting dose for calcitriol (Rocaltrol) is 2.5 ng/kg once daily, and the dose is adjusted based on serial measurements of calcium and PTH. The drug must be reformulated by a specialty pharmacy to allow accurate dosing in small animals (note the ng/kg dose, whereas it is supplied in mcg doses). Monitoring therapy is essential to avoid hypercalcemic problems. Calcium and phosphorus should be checked every week initially until stable. Thereafter, routine checks (approx. monthly) must be maintained. If hypercalcemia develops, drug administration must be stopped, not merely decreased in dose. Calcitriol improves survival in dogs (360 days vs 250 days with placebo). A randomized placebo controlled study is ongoing in cats. Until evidence exists that this drug is helpful in cats, I do not recommend its administration in this species.

**Anemia:** The anemia of CRF can be treated with human recombinant DNA erythropoietin (EpoGen<sup>®</sup>, ProCrit<sup>®</sup>, Aranesp<sup>®</sup>), but therapy is usually withheld until the anemia is severe and

symptomatic, due to the risk of antiEpoGen antibody formation. Symptoms of anemia can include tachycardia, heart murmur, heart failure, exercise intolerance, and anorexia. If there is evidence of recent blood loss (i.e. GI bleeding) or a concurrent inflammatory process in a cat with a previously acceptable PCV, the decision to start EPO can be delayed for 2 weeks to allow control of the underlying process and improvement in the PCV. See the accompanying proceedings notes for updates.

Several **drug interactions** are relevant. **Phosphate binders** can interfere with absorption of other drugs and should be separated from other medications by an hour. **Sucralfate** works better in an acid environment and thus should be given 30 minutes prior to **H<sub>2</sub> blockers** or phosphate binders.

Frequency of **monitoring** depends on the clinical condition of the patient. If there are ever questions about the cat's stability or a change in the status, a recheck is always in order. I have created a set of arbitrary guidelines for monitoring. For cats with low-grade azotemia (e.g. creatinine <3.5 mg/dl) who are relatively asymptomatic (good appetite, maintaining weight, on minimal treatments), I recommend **rechecks (physical exam, weight, chemistry panel, CBC)** every 3-6 months. For cats with moderate azotemia (e.g. creatinine 3.5-6 mg/dl) that are stable on therapy should be checked every 2 months. Cats with a creatinine over 6 mg/dl or who need SQ fluids on a daily basis should be checked on a monthly basis. **Urinalysis and urine culture** should be performed at least twice a year in all cats with CRF. **Blood pressure** measurement should be performed every 3 months in normotensive cats, and monthly in hypertensive cats who are controlled on therapy.

## **Anemia of Chronic Renal Failure**

Anemia is a common abnormality in patients with chronic renal failure. It causes many signs, including exercise intolerance, fatigue, anorexia, tachycardia, heart murmur and congestive heart failure. Causes include blood loss from a gastrointestinal ulcer or uremia induced platelet function defects, iron deficiency, decreased RBC survival and bone marrow inhibition by uremic toxins. The major cause of anemia in chronic renal failure is lack of erythropoietin.

Erythropoietin is a hormone produced by the kidneys that stimulates red cell precursors in the bone marrow to proliferate. Production of erythropoietin decreases as the kidneys fail. Insufficient new RBC's are produced and a non-regenerative anemia slowly develops.

Blood transfusion is an appropriate therapy if the anemia is causing clinical signs that need rapid correction, particularly dyspnea or heart failure. The availability of blood for transfusion is limited in some practices and there may be a short delay before the blood product is available for transfusion. The transfused cells will have a shortened lifespan due to the uremic environment and due to minor incompatibility reactions. If frequent transfusions are necessary, cross-match incompatibility can become a limiting factor and it is usually not possible to restore the RBC mass to normal due to volume limitations.

Supplementation of B vitamins necessary for erythropoiesis may be beneficial in CRF patients but will have little effect on restoring RBC mass. Anabolic steroids have been recommended for the treatment of the anemia of CRF. Results have been variable and generally disappointing. Their use cannot be recommended due to the lack of efficacy and the potential for hepatotoxicity.

Human erythropoietin has been produced using recombinant DNA technology and is currently marketed under the trade names of Epogen (Amgen) or ProCrit (Ortho Diagnostics). It differs from canine and feline erythropoietin by only a few amino acids. The functional site is similar enough to have biologic activity in the bone marrow. The usual starting dose is 100 units/kg subcutaneously three times a week until the PCV is at the low end of the target range (25-30% for cats, 30-35% for dogs). The dose is then reduced to twice weekly administration until the PCV is in the middle of the target range. The maintenance dose is usually 50-100 units/kg once to twice a week, and is titrated to maintain the PCV in the middle of the target range.

Darbepoetin (Aranesp, Amgen) has more carbohydrate groups attached to the protein backbone, in order to slow clearance and decrease the frequency of administration. It is less immunogenic in laboratory animals, but a clear idea of the risk in dogs and cats is not available. Based on my initial cases, I suspect the rate of symptomatic antibody formation is less than 10%. The starting dose (0.45 mcg/kg) is initially administered weekly, and then decreased to every 2-3 weeks as needed to maintain the PCV. I find the dose frequently needs to be increased to 1 mcg/kg. Other management as with epogen should be followed.

The PCV should be checked weekly until the PCV has been stable for 4 weeks on twice weekly therapy. Monthly monitoring is usually sufficient once the PCV has been stabilized. A complete blood count should be checked monthly to bimonthly for the duration of therapy.

Iron, a major component of hemoglobin, should be supplemented in patients receiving erythropoietin. Ferrous sulfate, a commonly available form, is dosed at 100-300 mg per day for dogs, which corresponds to 20-60 mg of elemental iron. For cats, 50-100 mg per day of ferrous sulfate (10-20 mg elemental iron) is recommended. Gastrointestinal upset is a common problem with oral iron supplements. If oral iron is not feasible, injectable iron dextran can be used as a deep intramuscular injection. The dose is 10-20 mg/kg for dogs, 50 mg total dose per cat, once monthly. There is a risk of anaphylaxis if iron dextran is administered intravenously.

Monitoring iron parameters is recommended prior to and one month after starting

erythropoietin therapy and then monthly to bimonthly thereafter. Serum iron level is a measure of mobile iron. Total iron binding capacity (TIBC) is an indirect measure of transferrin, the carrier molecule for iron in the serum. Ferritin is a water-soluble storage form of iron that is present in the cell cytoplasm and in the circulation. Hemosiderin is a storage form that is present in the bone marrow and can be estimated by evaluating bone marrow cytology when necessary. The transferrin saturation is a measure of the amount of the iron carrier molecule, transferrin, that is bound to iron, and is calculated from the equation: % saturation = serum iron ÷ TIBC. Transferrin saturation is normally above 33%. With iron deficiency, the serum iron level, ferritin level, and the transferrin saturation are low. However, in the anemia of chronic inflammation, iron is sequestered in the body, causing iron levels to be low, but ferritin levels are elevated.

Hypertension has been associated with erythropoietin therapy, although not directly caused by it. Blood pressure should be checked prior to starting therapy and monthly to bimonthly thereafter. Antihypertensive medications may need to be started or doses escalated in patients already being treated for hypertension. Seizures have been reported in association with erythropoietin administration and hypertension may play a role in their etiology.

Injection discomfort or skin reactions reported in people are not commonly recognized in animal patients. Vomiting has been reported but it is difficult to differentiate whether it is related to erythropoietin usage or the underlying uremia. Polycythemia is certainly possible if the dose is excessive and not adjusted appropriately.

Refractory anemia in patients on erythropoietin therapy has many potential causes. A careful review of the patient and the clinical pathology results may indicate iron deficiency, ongoing blood loss, the anemia of chronic inflammatory disease, or inadequate dose. Most animals will respond well to 100 units/kg three times a week, but some may need 150 units/kg.

Human recombinant DNA erythropoietin is not antigenically identical to canine or feline erythropoietin. One quarter of patients will develop a clinically significant immunologic reaction to Epogen or Procrit. Antibodies usually develop within the first 90 days of administration, although a later onset could occur. The primary sign of anti-Epogen antibodies is a precipitously declining PCV. Reticulocyte counts will decrease to zero prior to the drop in PCV and so may serve as an early warning. There is currently no commercially available test for the presence of antibodies. Bone marrow aspiration cytology will usually show a very high myeloid to erythroid ratio (M:E > 10:1), although there are exceptions.

If anti-Epogen antibodies are suspected, discontinue administration of Epogen. Blood transfusions are used as needed to support the patient. It may take 2 to 12 months for antibody titers to decline to a subclinical level and transfusions will be necessary during this time. Because of the cost of repeated transfusions, cross-match compatibility problems, and the fact that PCV will return only to the level that prompted treatment in the first place, the development of anti-Epogen antibodies frequently results in the death or euthanasia of the patient.

## **Nephroliths and Ureteroliths: Should We Leave No Stone Unturned?**

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Nephroliths are calculi that form in the kidney. They are usually located in the renal pelvis or diverticula. Ureteroliths are nephroliths that pass into the ureter. These calculi may pass to the bladder, where they may either be passed out in the urine or remain and cause cystic calculi. Ureteroliths may lodge in the ureter, causing partial or complete obstruction to urine flow. Ureteroliths are being diagnosed much more commonly in cats in the past decade compared to previously.

In cats, 98% of ureteroliths are calcium oxalate. In dogs, 40% of nephroliths are calcium oxalate, and 33% are struvite. There is a gender difference in dogs, in that 75% of nephroliths in female dogs are struvite, likely related to the higher incidence of bacterial urinary tract infection in female dogs. Urate nephroliths account for 12% of canine nephroliths.

Nephroliths may be detected serendipitously in patients being imaged (abdominal radiography, abdominal ultrasonography) for unrelated reasons. Other signs that may be encountered include hematuria not associated with dysuria, signs of lower urinary tract infection (e.g., pollakiuria, stranguria), abdominal or flank pain (infrequently reported in veterinary patients), or signs associated with uremia if obstruction is complete and the contralateral kidney is nonfunctional.

Abdominal imaging is a major part of diagnosing nephroliths and ureteroliths. On radiographs, the majority of calculi tend to be radio-opaque, although small size may be below resolution limit of radiography. It is occasionally difficult to differentiate calculi from renal mineralization on ultrasonography. Excretory urography (intravenous pyelography) is a sensitive method of detection, although more invasive than plain film radiography or ultrasonography. Additional recommended diagnostic testing includes serum biochemical profile, urine culture, and individual renal scintigraphy if nephrectomy being considered.

The appropriate treatment course is a matter of debate in many circumstances. In a few cases, such as severe uremia associated with an acute obstruction with normal renal architecture and a nonfunctional contralateral kidney, removal of the obstructing calculus is clearly desirable.

With an asymptomatic calculus associated with no renal dysfunction, treatment is aimed at slowing progression or stone dissolution. If struvite is suspected (e.g., female dog with concurrent UTI), stone dissolution diets (i.e., Hill's S/D diet) with concurrent antibiotics may be attempted. If calcium oxalate is suspected (e.g., cats), avoidance of acidifying diets, addition of potassium citrate, and feeding canned diet should be considered. Medical dissolution is not possible. If urate suspected (e.g., Dalmatians), dietary therapy (restricted protein and purines, alkalinizing, such as Hill's U/D) and allopurinol (10 mg/kg/day) may cause stone dissolution. In such cases, it is important to monitor for progression of calculi and renal dysfunction. If worsening, consider more active management, keeping in mind that surgical intervention will decrease renal function by 20%

If the calculus is associated with significant renal impairment, medical management may be a better alternative than surgery, depending on extent of disease, involvement of contralateral kidney, and stone type. This seems especially true with severe chronic renal failure and a low likelihood of improving function by removing the ureterolith. My clinical experience is that cats with partially obstructing or unilateral ureteroliths can live with medical management of CRF for several months (about 4-8 months, in general).

Calculi causing obstruction or recurrent urinary tract infection, are the best candidates for removal. Surgical removal is via pyelotomy, nephrotomy, or nephrectomy if renal function of kidney is minimal and irreversible. Staged surgery (2-4 weeks apart) may be preferable for bilateral disease. The

risks of surgery include urine leakage and uroabdomen, need for subsequent nephrectomy if leakage occurs, decreased renal function if nephrotomy performed, acute renal failure secondary to ureteral obstruction from post-operative inflammation and swelling. In my clinical experience, not all cats survive the post-operative period (or owners decline a second surgery if complications from the first occur). However, in those that are discharged from the hospital, survival times may exceed many years, and in general are better than survival times with medical management.

An alternative to surgical removal is extracorporeal shockwave lithotripsy. It is a less invasive procedure compared to surgery, had faster results than medical dissolution, but availability is limited. This is a good option to consider in patients with stable renal function, especially those who have recurrent urinary tract infections associated with the calculus.

Regardless of the method employed to control the nephroliths and ureteroliths, monitoring is recommended, to include radiography every 2 to 6 months to assess size and recurrence, a serum biochemical profile every 3-6 months or more frequently, and urinalysis and urine culture every 3 months.

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