

# Management of feline chronic renal failure

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possible, the cause of the renal disease. Any disease process that destroys renal tissue can cause CRF. Indeed, there are many known causes of CRF (2, 3) in cats (Table 1). Identifying the cause of CRF in an individual patient will generally involve evaluation of the results of the serum biochemical panel, urinalysis, and urine culture, plus abdominal radiography and/or ultrasonography. In those patients with CRF and a normal or enlarged kidney, renal biopsy or fine needle aspiration of the kidney for cytological evaluation should be considered.

Unfortunately, for most cats with chronic renal disease, particularly those with bilaterally small kidneys, the cause of the renal disease may not be established. The most frequent histological finding in cats with CRF is chronic interstitial fibrosis (also referred to as chronic tubulointerstitial nephritis or chronic interstitial nephritis) (2). In this disorder, the kidneys are small and firm with irregular surfaces on abdominal palpation and are characterized microscopically by extensive interstitial fibrosis accompanied by variable amounts of tubular atrophy, nephrocalcinosis, and glomerulosclerosis. Chronic interstitial fibrosis is a morphological, not an etiological, diagnosis and probably does not reflect a characteristic response of the kidney to a specific disease. It is more likely that chronic interstitial fibrosis is the typical finding for end-stage kidneys, representing the final common pathway for progression of any feline renal disease toward terminal failure.

## KEY POINTS

- The first step in the management of feline CRF is to determine, if possible, the cause of the renal disease.
- The most frequent histological finding in cats with CRF is chronic interstitial fibrosis.
- Specific therapy for the primary renal disease should be employed whenever a cause is identified.
- In the management of cats with CRF, the amount eaten is as important as what is eaten.
- Dietary protein restriction should be employed to reduce the clinical signs of feline uremia.

## INTRODUCTION

Chronic renal failure (CRF) is a frequent clinical finding, with increasing prevalence as cats age; it affects perhaps one-third of our geriatric feline patients (1). Given the complexity of the abnormalities observed in these patients, the management of feline CRF poses many diagnostic and therapeutic challenges. Despite this, the refinement of old treatments and the availability of new ones address some of the problems faced by veterinarians treating cats with CRF.

## ETIOLOGY OF CHRONIC RENAL FAILURE

For the purposes of this article, CRF is defined as the presence of azotemia of renal origin for a minimum duration of 2 weeks. Azotemia is the presence of elevated serum concentrations of blood urea nitrogen (BUN) and creatinine (SCr).

The first step in the management of feline CRF is to determine, if

## SPECIFIC THERAPY

Specific therapy for the primary renal disease should be employed whenever a cause is identified. In cats with glomerulonephritis and nephrotic syndrome, associated diseases (e.g., feline leukemia or immunodeficiency virus infection) should be identified. Therapy directed at reducing the extent of proteinuria and associated edema should be implemented, specifically to reduce the extent of glomerular inflammation (3). Hypoalbuminemia due to protein loss across the glomerular barrier is the primary cause of the edema, and the clinical management of this requires documentation of the extent of proteinuria. This is best accomplished in clinical patients by measuring serum albumin concentration (Salb), SCr, and the urine protein-to-creatinine (UP/Cr) ratio. As proteinuria is usually variable in these cats, two baseline determinations of the UP/Cr ratio are desirable.

The proteinuria may decline in response to dietary protein restriction and/or the administration of an angiotensin-converting enzyme inhibitor (ACEI). These therapies can be started sequentially, beginning with dietary protein restriction, at 1 month intervals. Their efficacy should be assessed by checking UP/Cr ratio, SCr, and Salb concentration every 2 weeks. Once the appropriate diet is chosen, the efficacy of an ACEI (e.g., 0.5–2.0 mg enalapril/kg orally every 24 hours or 0.25–2.0 mg benazepril/kg orally every 24 hours) should be evaluated. In a small number of cats with CRF the



**Table 1**  
**Primary disorders that may lead to chronic renal failure in cats**

<i>Primary site affected</i>	<i>Disorder</i>
Tubulointerstitium	Chronic interstitial fibrosis*
	Pyelonephritis (bacterial or mycotic)
	Hypercalcemic nephropathy
	Potassium depletion nephropathy
	Feline infectious peritonitis (non-effusive form)
	Neoplasia
Vasculature	Amyloidosis
	Polycystic kidney disease
	Systemic hypertension
	Glomerular hypertension
	Intravascular coagulation
Glomeruli	Polyarteritis nodosa
	Hyperthyroidism (unproven)
	Glomerulonephritis
	Glomerulosclerosis
Collecting system/pelvis	Nephrolithiasis
	Perirenal pseudocysts

\*Chronic interstitial fibrosis probably represents the microscopic change observed as many, or all, feline renal diseases progress through a final common pathway toward end-stage failure.

**Table 2**  
**Abnormalities observed in uremic cats**

<i>Abnormality</i>	<i>Therapeutic intervention</i>
Weight loss	Encourage food intake (see text)
Vomiting	Antiemetics
	Dietary protein restriction
	Correct electrolyte and acid/base disorders
Hyperphosphatemia	Dietary phosphorus restriction
	Intestinal phosphorus binding agents
Renal osteodystrophy	Dietary phosphorus restriction
	Intestinal phosphorus binding agents
	Calcitriol
Metabolic acidosis	Dietary alkalinizing agents
Hypokalemia	Switch to nonacidifying diet
	Dietary potassium supplementation
Anemia	Maintain adequate caloric intake Erythropoietin
Systemic hypertension	Antihypertensive agents ± dietary sodium restriction
Urinary tract infection	Antibiotic therapy
Progression of renal failure	Dietary phosphorus restriction
	Antihypertensive agents (unproven)
	Calcitriol (unproven)

ACEI reduces renal function and should therefore be discontinued.

Long-term efficacy of the ACEI and/or dietary protein restriction should be assessed with sequential determinations of the UP/Cr ratio, SCr, and Salb every 2 to 6 months. If edema is a problem, a loop diuretic (e.g., furosemide 1–2 mg/kg orally every 8 to 12 hours) may be beneficial, although this may cause dehydration and worsen renal function.

Unfortunately, very little is known about the efficacy of immunosuppressive or anti-inflammatory therapy in cats with nephrotic syndrome and immune-mediated glomerulonephritis. A therapeutic trial with these agents, aimed at reducing glomerular inflammation and resultant proteinuria, can be considered. The current recommendation, however, is to initially consider dietary protein restriction and ACEI. While markedly proteinuric dogs may develop a hypercoagulable state leading to thromboembolism, this is rarely observed in cats with nephrotic syndrome. Consequently, the routine use of anticoagulants, such as aspirin or coumarin, is not indicated.

Other feline renal diseases associated with CRF include bacterial infection of the kidney, perirenal pseudocysts, nephrolithiasis, and renal lymphosarcoma. In such cases specific therapy should be given for the primary disease.

## PROBLEM IDENTIFICATION

All metabolic and clinical abnormalities must be identified as early as possible. These problems frequently remain subclinical for several weeks, or even months, before the owner presents the cat to the veterinarian. It is essential for the veterinarian to identify a developing abnormality with a sensitive test, such as a serum biochemistry screen, rather than institute reactive therapy after the owner presents the animal with severe clinical signs. In all cases, supportive therapy must be individualized on the basis of routine physical and laboratory evaluation of the patient. Patients should be evaluated every 2 to 6 months and more frequently if they are unstable or uremic.

## NUTRITIONAL CONSIDERATIONS

Adequate fresh water and caloric intake must be assured. In the management of cats with CRF, the amount eaten is as important as what is eaten. Cats with CRF often have reduced appetites, and special diets formulated for this condition have variable palatability. Changes in bodyweight should be carefully documented by the veterinarian. An accurate log of food intake should be maintained by the owner and reviewed by the veterinarian at each visit. Caloric needs vary dramatically among cats. An ideal caloric intake is one that will sustain a normal activity level and normal bodyweight. Inadequate intake can be loosely defined as <50 kcal/kg/day with weight loss or poor body condition. Provision should also be made to assure adequate intake of water-soluble vitamins, which may be depleted in polyuric animals.

Food intake may be increased in various ways. These include:

- Making all dietary changes gradually.
- Offering different forms of the diet (e.g., dry versus moist formulations).
- Warming the food.
- Providing fresh food daily (uneaten canned food should be discarded every 6–12 hours).

Frequently offering small amounts of food, often by hand, may be helpful in some patients. The addition of a small amount of a

palatability, enhancing agent, such as anchovy or fat, may increase caloric intake. However, care should be taken to avoid excessive supplementation as this will alter the nutrient balance of the diet, particularly if certain nutrients have been restricted. Increasing the activity of the animal may enhance its appetite. Other therapies may indirectly enhance activity level, such as specific therapy for anemia, electrolyte abnormalities, or uremia (see below). Pharmacological efforts to increase food intake (e.g., 0.2–0.3 mg diazepam/kg IV every 12–24 hours, 0.2–0.4 mg oxazepam/kg orally every 24 hours, 0.2–0.4 mg flurazepam/kg orally every 4–7 days, or 1–3 mg cyproheptadine per cat orally every 12–24 hours) may be tried. Some cats introduced to new diets during hospitalisation or during an uremic crisis develop an aversion to that particular diet, apparently similar to some food aversions observed in human beings. The transition to new diets intended for long-term ingestion should therefore be carried out at home and not while the cat is hospitalized and/or uremic.

## UREMIC SYNDROME

Regardless of cause, similar abnormalities generally affect all cats with CRF and produce a constellation of clinical signs and biochemical findings commonly referred to as the uremic syndrome or uremia (**Table 2**). Many of these clinical signs are nonspecific, including depression, lethargy, weakness, inactivity, lack of social interaction, and weight loss. Others involve primarily the gastrointestinal tract; these include inappetence, vomiting, and the accumulation of dental tartar. Laboratory and clinical findings associated with the uremic syndrome include:

- Marked azotemia.
- Hyperphosphatemia with concurrent renal secondary hyperparathyroidism.
- Hypokalemia.
- Metabolic acidosis.
- Systemic hypertension.
- Anemia.
- Progressive loss of renal function.

Management should be directed towards identifying, characterizing, and properly treating any abnormalities.

## UREMIA: DIETARY AND ADJUNCTIVE THERAPY

To reduce the clinical signs of uremia in cats, dietary protein restriction should be employed once blood urea concentration reaches the range of 10–15 mmol/L. The reduced protein diet should contain approximately 26% to 32% protein on a dry matter basis and supply approximately 3.8–4.5 g/kg/day. In some cats, intermittent administration of a balanced electrolyte solution (e.g., 20–40 ml lactated Ringer's solution/kg SC every 24–72 hours) may be helpful.

Hyperphosphatemia is common and directly related to the degree of renal dysfunction and the level of dietary phosphate intake. To delay the progression of renal injury, dietary phosphorus restriction should be instituted in all cats with azotemic CRF (4, 5). The diet should contain approximately 0.5% phosphorus on a dry matter basis (supplying 65–85 mg/kg/day). The goal of therapy is normophosphatemia.

After an initial dietary trial with a restricted phosphorus diet of 2–4 weeks, the addition of intestinal phosphorus binders dosed to effect (initial dose of 30–180 mg/kg/day with meals) will usually be necessary to achieve normophosphatemia. Aluminium- or calcium-

containing salts can be used as phosphorus binders at the same initial doses. Although the former may be associated with osteodystrophy or encephalopathy in humans, there is little evidence of these complications in cats. Calcium-containing phosphorus binders will produce hypercalcemia in some cats. Mix the phosphorus binder with moistened or canned food and change doses gradually to limit food aversion.

Hyperphosphatemia and reduced renal production of 1,25 dihydroxyvitamin D (calcitriol) contribute to the genesis of renal secondary hyperparathyroidism in animals with CRF (6–8). A variety of clinical abnormalities observed in animals with the uremic syndrome have been attributed to the presence of excess parathyroid hormone, including uremic osteodystrophy, anemia, arthritis, cardiomyopathy, encephalopathy, glucose intolerance, hyperlipidemia, immunosuppression, myopathy, pancreatitis, pruritus, skin ulceration, and soft tissue calcification. While the role of excess PTH in uremic osteodystrophy is widely accepted as pivotal, the importance of parathyroid hormone in the pathogenesis of other abnormalities in uremia is unclear in many cases.

Dietary phosphorus restriction ± intestinal phosphorus-binding agents will generally lower but not normalize serum parathyroid hormone concentration in cats with CRF. There is some support for the use of calcitriol to further lower PTH: 2.5–5.0 ng/kg/day orally every 24 hours and given separately from meals will lower PTH in most, but not all, dogs with CRF (5). Similar effects occur in cats. Treated animals should be carefully monitored once every 2 to 4 weeks as calcitriol can cause hypercalcemia in some cats, with resultant hypercalcemic nephropathy.

## VOMITING

In addition to dietary protein restriction, specific therapy to reduce the incidence of vomiting may include antihistamines specific for the H<sub>2</sub> receptor, such as cimetidine (4 mg/kg orally every 6–8 hours), ranitidine (1–2 mg/kg orally every 12 hours), or famotidine (1 mg/kg orally every 24 hours). Centrally acting antiemetics may be required intermittently, particularly when protracted vomiting is present.

## DISORDERS OF POTASSIUM BALANCE

Hypokalemia is most often observed in polyuric cats (2). In feline patients it may indicate inadequate potassium intake and/or the effects of an acidifying diet to promote kaliuresis. Hyperkalemia is uncommon except in the terminal phases of CRF and is often associated with oliguria or anuria.

Hypokalemia has a variety of adverse effects, including a reduction in renal function. In hypokalemic cats, the first step is to switch to a diet with a high-potassium, low-acid content such as those formulated for the treatment of feline CRF. The administration of oral potassium (1–3 mEq/kg/day) as potassium gluconate mixed with food will help to restore eukalemia. Other potassium salts are generally less well tolerated, although some cats will ingest KCl mixed with food. Once eukalemia is achieved, the potassium supplements may be reduced or discontinued, based upon serial measurements of serum potassium concentration.

## METABOLIC ACIDOSIS

Proteins, particularly those of animal origin, are rich in sulfur-containing amino acids; metabolism of these leads to hydrogen ion generation. Consequently, many diets fed to cats provide a net load of acid, which must be excreted by the kidney if acid–base balance is to be achieved. Unfortunately, cats with reduced renal mass are less



able to excrete acid, potentially resulting in metabolic acidosis from acid retention. This is generally associated with an increased anion gap due to the accumulation of unmeasured anions, and the acidosis may cause lethargy and inappetence.

Appropriate control of acid–base status includes routine monitoring of serum total carbon dioxide or bicarbonate concentration. The therapeutic goal is to keep the patient's value within the normal range. Alkalinizing agents should be added to the food with dosage adjusted to achieve this (e.g., initial doses of 15 mg sodium bicarbonate/kg every 8–12 hours or 30 mg potassium citrate/kg every 8–12 hours). The latter also supplies potassium, which will benefit many cats with CRF.

## ANEMIA

The anemia of chronic renal disease is normocytic, normochromic, and nonregenerative and is primarily due to decreased renal generation of erythropoietin (9, 10). Although the accumulation of toxins and endocrinopathies (e.g., renal secondary hyperparathyroidism) depresses erythropoiesis and shortens the lifespan of erythrocytes, these factors make only a minor contribution to the pathogenesis of the anemia.

The clinical signs of depression – inappetence, inactivity, and lack of social behavior – may respond to an increase in hematocrit. Unfortunately, anabolic steroids and blood transfusions are of limited value in the treatment of anemia in these patients. Recombinant erythropoietin (50–100 units/kg SC 2–3 times weekly) will increase the hematocrit in most affected animals (10). When erythropoietin therapy is started, the cats should receive oral ferrous sulfate supplementation (50–100 mg orally every 24 hours). When administering recombinant erythropoietin, there must be frequent monitoring of hematocrit to assure adequacy of response and avoid polycythemia. The therapeutic goal is the low end of the normal range for hematocrit (30% to 35%).

A significant number (approximately 25–40%) of cats develop antibodies to this human glycoprotein, evidenced by refractoriness to therapy. Other causes of treatment failure, such as feline leukemia, virus infection, or iron deficiency, should be considered. Once antibody formation occurs, further use of the drug will not be possible. As the prevalence of antibody production causes many cats to become unresponsive to this agent after a few months or a year, it should be used judiciously, perhaps only in those animals with a hematocrit <20% and clinical signs attributable to the anemia.

## SYSTEMIC HYPERTENSION

Cats with renal failure usually have systemic hypertension. Severe systemic hypertension can lead to retinal hemorrhages and/or detachment, seizures, and cardiac hypertrophy and may cause progressive renal injury (11–19). Mild to moderate systemic hypertension may be detrimental, but this remains to be established.

Therapy for hypertension should be based upon measurement of blood pressure or the identification of end-organ damage directly attributable to systemic hypertension (e.g., retinal detachment). Measurements should be made by an experienced technician in a quiet setting, taking a minimum of five consistent readings. The preferred indirect method in cats is the ultrasonic Doppler technique, using the median artery. Treatment is generally indicated if systolic pressure exceeds 200 mmHg or 170 mmHg in cats with complications attributable to hypertension. If diastolic pressure exceeds 110 mmHg and there are clinical abnormalities attributable to hypertensive injury, treatment should be instituted (15, 16). The goal of therapy is to reduce arterial pressure by at least 25–50 mmHg while sustaining adequate renal function. Ideally, blood pressure

would be in the normal range for systemic arterial pressure (systolic 100–140 mmHg; diastolic 60–100 mmHg; mean 80–120 mmHg).

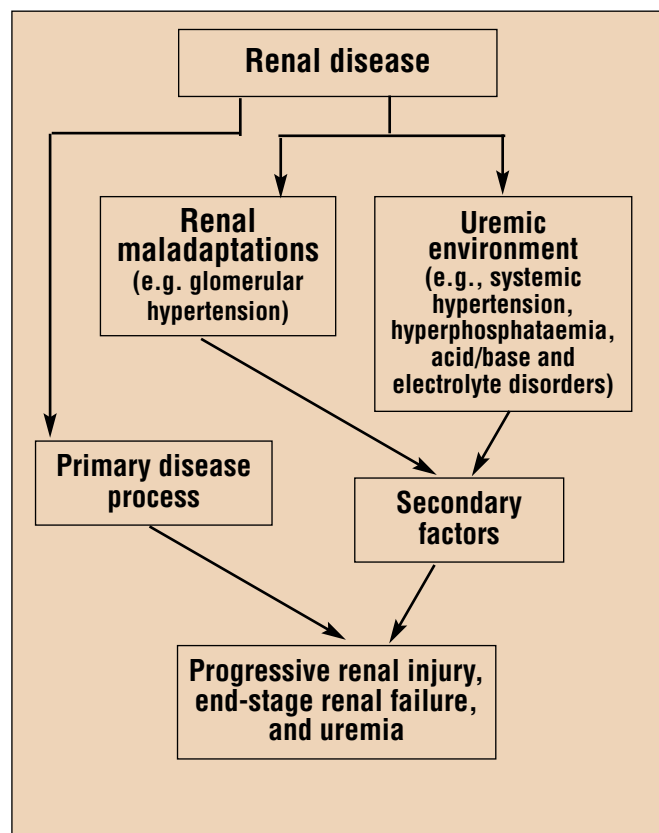
Antihypertensive therapy should include the use of a low-sodium diet combined with either an ACEI (0.5–2.0 mg enalapril/kg orally every 12–24 hours or 0.25–2.0 mg benazepril/kg orally every 12–24 hours) or a calcium channel antagonist (0.625–1.25 mg amlodipine/cat orally every 24 hours). These agents may be combined if necessary to obtain the desired reduction in blood pressure.

Therapy for systemic hypertension is titrated to effect. Efficacy is judged by blood pressure measurements and determination of SCr at 2 weekly intervals initially and evaluation every 3 to 6 months, when the efficacious dose has been determined. Dosage adjustments are common. Adverse effects of antihypertensive therapy include decline in renal function or weakness and syncope due to hypotension.

## PROGRESSION OF RENAL DISEASE

Frequently, cats with renal disease suffer a progressive decline in renal function until terminal uremia develops. The possible causes of this progression include exacerbation of the primary renal disease (20) or secondary factors such as dietary phosphate excess or hypertension in systemic or glomerular vessels (11) (Figure 1).

To slow the progress of renal failure, the primary disease process and secondary factors that may be causing further renal injury should be controlled. For example, dietary phosphate restriction supplemented with intestinal phosphorus binders (see above) should be given to cats with azotemia (4, 5). Dietary protein restriction (see above) can be considered in cats with mild azotemic renal failure, although this remains controversial (20). Dietary protein restriction



**Figure 1** Chronic renal failure is often a progressive disease. The mechanisms responsible for the progression include the primary renal disease (e.g., glomerulonephritis or hypertensive nephropathy) and secondary factors. These secondary factors occur as a result of renal maladaptations and the effects of the uremia on the kidney.

to limit the extent of uremia is indicated in moderate to severe azotemia. Based on results in other species (12), the use of an ACEI may reduce systemic arterial and intraglomerular pressure (13), reduce the extent of glomerular hypertrophy, and interfere with the actions of a variety of growth factors that play a role in progressive glomerulosclerosis and interstitial fibrosis. Appropriate agents to consider for a therapeutic trial would include enalapril (0.5–2.0 mg/kg every 12–24 hours) and benazepril (0.25–2.0 mg/kg every 12–24 hours).

## PATIENT MONITORING

In all cats with azotemic CRF, the urinalysis, urine culture, SCr, serum electrolytes, hematocrit, and blood pressure should be assessed every 2 to 6 months. These evaluations should be more frequent if renal function is unstable, if SCr > 4 mg/dl, or if systemic hypertension is documented. The biochemical panel and complete blood count should be evaluated annually.

# EFFECT OF PHOSPHATE AND PROTEIN RESTRICTION ON PROGRESSION OF CHRONIC RENAL FAILURE IN CATS

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The effect of feeding a diet restricted in phosphates and protein on survival of 37 cats presenting with stable CRF was studied prospectively. A veterinary renal failure diet\* was offered to all cats once the diagnosis had been confirmed. Dietary compliance and appropriateness of the level of phosphate restriction (need for addition of phosphate binding drugs) were assessed at follow-up consultations, which were held at monthly intervals initially and then bimonthly. Phosphate binding drugs were only used in cats that had accepted the Veterinary Diet and then only if deemed necessary based on the plasma phosphate concentration. Complications of CRF (hypokalemia, hypertension, urinary tract infections) were managed as and when they were recognized in all cats.

Twenty-two of the cases were fed the Veterinary Diet and were considered to be adequately phosphate restricted, whereas fifteen owners were not able or willing to introduce the Veterinary Diet effectively over a 4 week period. Both groups were equally matched in terms of initial plasma creatinine concentration, body weight, and age. At the time of reporting, 13 (59%) of the diet-fed cats have died (n = 5) or been euthanatized (n = 8), with a median (1st and 3rd quartiles) survival time of 581 (296.5 and 957) days. These cats were maintained on the Veterinary Diet for 80.5% ± 6.5% of their survival time. Eleven (73%) of the cats that did not have restricted phosphate and protein intake have been euthanatized with a significantly shorter (P = 0.017; Mann Whitney U test) median survival time of 252 (169 and 465) days. Progressive renal failure as judged by rises in plasma creatinine concentration was the reason for death or euthanasia in four (31%) of the cats fed the Veterinary Diet and eight (73%) of the cats that were not protein or phosphate restricted.

These data suggest that phosphate and protein restriction increase survival time and slow progression of clinical cases of feline CRF.

\*WALTHAM Veterinary Diet; WHISKAS Low Phosphorous, Low Protein, Masterfoods Austria

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