



Dietary management of renal failure in the dog and cat

Peter J. Markwell BSc, BVetMed, MRCVS
WALTHAM Centre for Pet Nutrition, UK



Peter Markwell is the Senior Clinical Nutritionist at the WALTHAM Centre for Pet Nutrition, Waltham-on-the-Wolds, Melton Mowbray, Leicestershire, UK.

KEY POINTS

- Dietary therapy is a key element in the conservative medical management of chronic renal failure (CRF) in dogs and cats.
- Appropriate dietary modifications can alleviate clinical signs of uremia and may help to slow progression of renal damage.
- Although the restriction of dietary protein is of clinical benefit in uremic patients, excessive protein restriction can cause protein malnutrition in both dogs and cats.
- Feeding an energy-dense diet, in which the energy content is derived primarily from nonprotein sources, helps to avoid tissue catabolism and to reduce nitrogenous waste production.
- A staged approach to the dietary management of CRF is currently recommended for dogs.

INTRODUCTION

Chronic renal failure (CRF) is the most common manifestation of renal disease in the dog and cat and represents the end stage of a number of renal diseases. It is primarily a disease of the older animal and there is a strong clinical impression that the condition is progressive, with most patients ultimately dying of uremic complications.

Although there is no cure for CRF and since existing renal damage is irreversible, measures to correct biochemical abnormalities, alleviate clinical signs, and slow the progression of renal damage are of considerable clinical importance.

The kidney performs many vital functions, which may broadly be grouped into three categories:

- Excretory – e.g., waste products of protein metabolism.
- Regulatory – e.g., acid base balance.
- Biosynthetic – e.g., erythropoietin.

These may be variably affected in CRF. Clinical and laboratory signs reflect a progressive decrease in these functions; but, as the kidney has substantial functional reserves, overt clinical signs become apparent only after extensive destruction of renal tissue. Screening, particularly of elderly patients, may permit early detection of renal failure prior to the onset of overt clinical signs.

Azotemia is the finding of increased concentrations of urea, creatinine or other nonprotein nitrogenous compounds in blood. It may, however, be the result of pre- or post-renal factors, as well as of primary renal disease. Azotemia may be present in animals without overt clinical signs of renal failure. **Uremia** is the polysystemic toxic syndrome that develops with more advanced renal failure and is characterized by the presence of clinical signs in association with azotemia. Clinical signs may include:

- Polyuria and polydipsia.
- Anorexia and weight-loss.
- Lethargy.
- Pallor and/or ulceration of the mucous membranes.
- Vomiting.

Dietary manipulation is a cornerstone in the conservative medical management of CRF, but this represents only one aspect of the therapeutic strategy. Where an underlying primary disease has been identified, or if prerenal components are involved, specific therapy to correct these may be possible. Additional measures may include:

- Maintenance of normal hydration through the provision of unlimited access to drinking water or with fluid replacement when there is persistent vomiting.
- Avoidance of stress.
- Administration of sodium bicarbonate to correct metabolic acidosis.
- Anabolic agents.
- Intestinal phosphorus binders.
- Supplementation with calcium and calcitriol.
- Demulcent mouth washes and H_2 antagonists, such as cimetidine, to alleviate gastrointestinal disturbances.
- Erythropoietin.
- Anticonvulsant therapy.
- Avoidance of nephrotoxic drugs.
- Institution of antihypertensive therapy.

While it is not possible to effect a cure in CRF patients, appropriate medical management can result in a good quality of life for the patient for months or even years. For this reason, the early diagnosis of CRF is desirable. Detection of early cases may be facilitated through routine blood and urine analyses in the older animal.

Dietary therapy

The goals of dietary management may be summarized as follows:

- To meet the patient's nutrient and energy requirements.
- To ameliorate clinical signs of uremia, where present.
- To minimize electrolyte, vitamin, and mineral disturbances.
- To try to slow the progression of renal failure.

Diets formulated to meet these goals require modification of the following dietary components: phosphorus, protein, calcium, sodium, potassium, and water-soluble vitamins, along with the dietary energy content and fat.

Phosphorus

Dietary phosphorus restriction has been shown to slow the progression of renal failure in dogs (1, 2), although the mechanism for this remains unexplained. It may be assumed that this effect results from one or more consequences of reducing the phosphorus retention (not always manifested as hyperphosphatemia) that results from reduced renal function.

Hyperphosphatemia is a common finding in patients with CRF. It occurs when the glomerular filtration rate (GFR) falls to approximately 20% of normal, resulting in impaired renal phosphate excretion. Phosphorus retention can cause renal mineralization, secondary hyperparathyroidism, and, potentially, an increase in renal damage.

Renal mineralization appears to be common in dogs and cats with CRF and may be an important factor in progression. Soft tissue mineralization occurs when the concentrations of calcium and phosphorus in plasma exceed the solubility product of calcium phosphate salts.

One of the key effects of elevated serum phosphorus is the inhibition of activity of the enzyme 1- α -hydroxylase in the kidney, contributing to a decrease in production of 1,25-dihydroxycholecalciferol (calcitriol), the most active form of vitamin D_3 (Figure 1). In turn, decreased calcitriol concentrations (together with hypocalcemia where this occurs) result in stimulation of

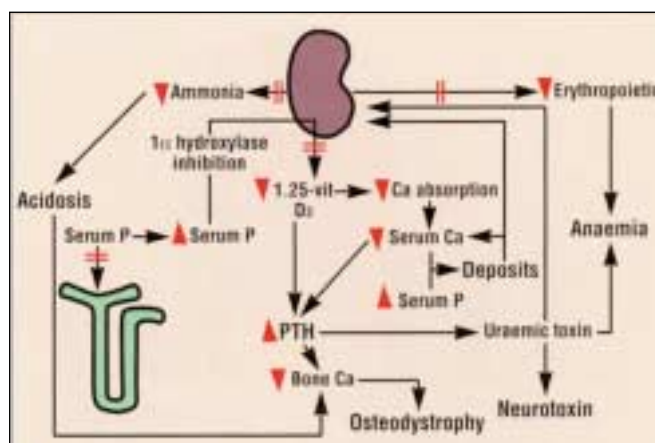


Figure 1 Development and possible consequences of renal secondary hyperparathyroidism.

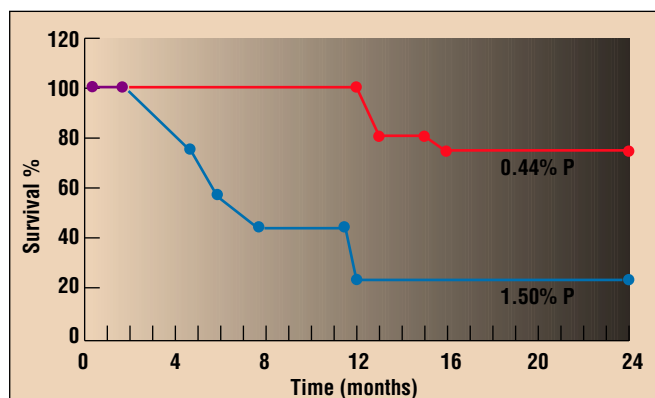


Figure 2 Effect of dietary phosphorus content on survival of dogs with induced renal failure (data from Brown et al) (1).

parathyroid hormone (PTH) synthesis and release, leading to secondary hyperparathyroidism (3). Parathyroid hormone *may* be an important uremic toxin, which could contribute to anemia, neurotoxicity, dyslipoproteinemias, insulin resistance, promotion of soft tissue calcification, renal osteodystrophy, and, most importantly, progression of renal damage (4, 5).

The most severe toxic injuries due to PTH may be inflicted because of high concentrations of PTH receptors in renal cells. These may promote the uptake of calcium by the cells, initially causing cell death and eventually the precipitation of calcium phosphate into tubule lumens. Hyperparathyroidism may thus promote a vicious cycle of cell death leading to decreased renal ability to contribute to phosphate homeostasis, increased PTH levels, and the loss of more renal tissue (3, 6). Supporting data for this detrimental role of PTH are provided by some clinical studies, which have indicated a possible slowing of the progression of renal failure in dogs whose plasma PTH was suppressed (7). This remains unproven, as recent experimental studies failed to demonstrate a clear benefit of reducing PTH (8).

The most complete study to date of the effect of phosphorus restriction was made in dogs with induced renal failure (1, 2). The key finding in this study was that the phosphorus content of the diet influenced mortality, the time period for which renal function was stable, and its subsequent rate of decline (Figure 2). The results of this study provided good evidence of the benefits of phosphorus restriction in the management of CRF. It is of interest that the benefits were noted despite the fact that renal mineralization was not influenced by diet; PTH levels, although related to diet in the early stages of the study, later tended to rise in all groups. Clearly, further studies are required to elucidate fully the mechanism of action of phosphate restriction.



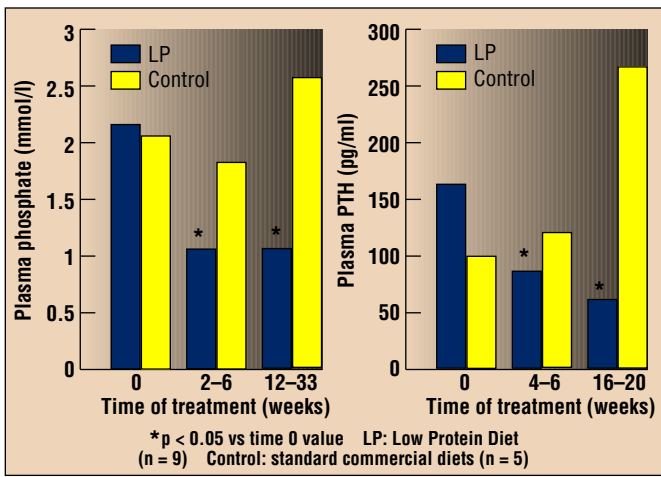


Figure 3 Effect of dietary phosphorus restriction on plasma phosphate and parathyroid hormone concentrations in cats with naturally occurring chronic renal failure (data from Barber et al) (10).

Data evaluating the effect of phosphorus restriction in cats are less complete. The benefits of phosphorus restriction in cats with induced CRF (although not the mechanism by which they occur) have been reported by Ross et al (9). In this study the effects of phosphorus-restricted (0.42% DM (dry matter)) and normal phosphorus (1.6% DM) diets were compared over periods of up to 343 days. Serum phosphorus concentrations were significantly higher in the normal phosphate group. Serum PTH concentrations were also markedly higher in this group of cats after 6 weeks and subsequently increased further. In the low phosphorus group, serum PTH levels were only slightly higher than clinically normal controls.

Neither diet caused a significant change in renal function during the study. However, renal mineralization, fibrosis, and cell infiltration were demonstrated in the cats on the higher phosphorus intake, whereas the kidneys of those fed on the phosphorus-restricted diet showed little or no change. This study suggested that in cats with CRF, restriction of dietary phosphorus was of value in reducing the development of renal lesions, although clear clinical benefits or effects on progression of disease (as measured by changes in renal function) were not demonstrated.

Preliminary data from clinical studies have shown that WALTHAM Veterinary Diet Feline Low Protein will bring about significant decreases in PTH in cats with naturally occurring CRF (10) (**Figure 3**). In a separate clinical study there were indications that restricting dietary phosphorus and protein may have delayed the progression of renal failure, compared with a control group fed a diet containing 'normal' levels of phosphorus and protein (11, 12).

Dietary phosphorus restriction is thus an important part of the management of CRF that may impact favorably on progression of the condition, even though the mechanism by which its beneficial effect occurs remains elusive (1, 2). Phosphorus restriction, therefore, should be initiated early in the course of CRF and should be considered for any dog or cat in which azotemia is shown to result from primary renal failure.

Dietary therapy aims to normalize serum phosphorus concentration and control secondary hyperparathyroidism. The effect of phosphorus restriction should be monitored by measurement of serum phosphorus or PTH concentrations. If phosphorus is used, the animal should be fasted for 12 hours prior to sampling as postprandial increases in phosphorus concentration may occur. PTH may provide a more sensitive indicator of whole body phosphorus status.

If dietary phosphorus restriction does not correct hyperphosphatemia or hyperparathyroidism, the next stage is to

Table 1
Selected oral phosphorus binding agents

Compound	Starting dose	Comments
Aluminium hydroxide, carbonate, oxide	30–90 mg/kg/d	Liquids most effective. Compliance may be better with capsules or tablets
Calcium acetate	60–90 mg/kg/d	Most effective calcium-containing agent. Risk of hypercalcemia with all calcium salts

commence therapy with oral phosphorus binding agents (**Table 1**). These should be used only in conjunction with phosphorus-restricted diets and after the patient has become accustomed to the diet. They should always be administered with food.

If phosphorus-binding agents do not achieve the goal of normalizing PTH concentrations, supplementation with calcitriol may be considered. A once-daily oral regimen at an average dose of 2.5 ng/kg has been recommended for both dogs and cats (13). If calcitriol therapy is initiated it is essential that serum calcium and phosphorus concentrations are monitored regularly.

Protein

The reduced ability of the kidney to excrete both nitrogenous and non-nitrogenous protein catabolites is considered to be one of the major causes of uremic signs in dogs and cats with renal failure. Several studies have shown that reducing dietary protein intake can bring about clinical benefits in uremic patients. In addition to reducing the level of protein catabolites, dietary protein restriction may also help by:

- Reducing the intake of dietary phosphorus.
- Decreasing the protein-related solute load, thereby lessening the severity of polydipsia and polyuria.
- Decreasing the acid load, which may help to alleviate metabolic acidosis.

There are, however, potential problems with excessive protein restriction. Uremia is a catabolic state which may adversely affect several aspects of protein metabolism. Renal failure may also lead to increased urinary losses of protein or specific amino acids. The protein requirements of dogs and cats in CRF have not been established, but it is likely that they may be different, and probably higher, than those of the healthy animal. It is important, therefore, that high quality protein sources are used in the formulation of restricted protein diets to minimize the risks of essential amino acid deficiency.

Very low protein diets may be poorly accepted and have been associated with protein malnutrition (as indicated by weight loss and decreased serum albumin) in dogs with CRF (14, 15). Other reported side-effects in the dog include hypertension and increased serum ionized calcium and cholesterol (16). These observations have led to the recommendation that protein intake should not be restricted to less than 1.9 g/kg bodyweight/day (approximately 11 g protein/400 kcal metabolizable energy [ME] in the diet for a 10 kg dog), unless further restriction was required to control clinical signs of uremia (15). The cat is at even greater risk of protein malnutrition because of its inability to down-regulate hepatic enzyme activity associated with protein catabolism, even when dietary protein intake

Restriction of dietary protein is beneficial in uremic patients

Table 2
Staged management of canine CRF (after Brown 1995) (24)

<i>Clinical category</i>	<i>Recommended treatment</i>	<i>Recommended Diet</i>
Azotemic renal failure	Free access to fresh water. Normal intake of protein. Restrict phosphorus $\pm$ phosphorus binders. Restrict sodium if hypertensive. Supplement potassium to maintain eukalemia. Alkalinization to maintain plasma bicarbonate	WALTHAM Veterinary Diet Canine Medium Protein
Uremic renal failure	Free access to fresh water. Restrict protein. Restrict phosphorus $\pm$ phosphorus binders. Supplement potassium to maintain eukalemia. Alkalinization to maintain plasma bicarbonate. Consider erythropoietin if anemic. Consider calcitriol	Depending on the severity of uraemia: WALTHAM Veterinary Diet Canine Low Protein or: A mixture of WALTHAM Veterinary Diet Canine Low Protein and WALTHAM Veterinary Diet Canine Medium Protein (Table 3)

Table 3
Protein and energy contents of mixtures of WALTHAM Veterinary Diets Low and Medium Protein

<i>Amount of LP g</i>	<i>Amount of MP g</i>	<i>Protein content % ME</i>	<i>Energy content kcal ME/100 g</i>
0.00	100.00	18.30	170.00
20.00	80.00	16.80	176.00
40.00	60.00	15.50	182.00
60.00	40.00	14.20	188.00
80.00	20.00	13.00	194.00
100.00	0.00	11.90	200.00

ME = metabolizable energy

LP = Low Protein Diet

MP = Medium Protein Diet

is low (17).

Dietary protein restriction is justified in dogs and cats showing clinical signs of uremia. Restriction of dietary protein at an earlier stage, before the onset of clinical signs, would be appropriate if it played a significant role in delaying the progression of renal damage. The available evidence does not, however, support this concept in dogs; the case is less clear in cats.

The proposal that protein restriction could slow progression of renal failure came from experimental studies in partially nephrectomized rats. Following a substantial reduction of renal tissue, a series of structural and functional changes occurred in the surviving nephrons, including glomerular hypertension, hyperfiltration, and hypertrophy. It was thought that glomerular sclerosis, and hence progression of renal damage, occurred as a result of these sustained adaptive changes. High protein intake in the presence of renal injury contributed to the increased perfusion, but it was found that a reduction of dietary protein from 24% to 6% reduced the hemodynamic changes and slowed the progression of tissue destruction (18, 19).

Results from studies in dogs suggest, however, that it may not be appropriate to extrapolate data from rats to the canine species, although this remains an area of controversy. Dietary protein (unlike dietary phosphorus) was not found to influence the progression of renal failure in a number of studies (2, 20, 21). In addition, studies of the effects of dietary protein on single nephron glomerular filtration rate, glomerular capillary pressure, and glomerular volume identified several differences between rats and dogs in response to renal ablation (22, 23).

Finally, limited data suggest that very high protein intakes may

be undesirable in dogs with CRF. Robertson et al. found in a four-year study of dogs with induced renal failure that diets providing over 50% of ME from protein were associated with more renal lesions in some dogs than those providing either approximately 31% or 18% of ME from protein. It is of interest, however, that mortality was not correlated with diet (21).

In summary, it is evident that *moderate* protein restriction is desirable in the management of CRF in dogs, as high-protein diets may be associated with more severe clinical problems, laboratory abnormalities, and renal lesions. Care should be taken, however, to avoid excessive restriction of protein because of the risks of protein malnutrition and other possible side-effects. The need to reduce protein intake as far as possible is also questionable given the lack of evidence for progression of renal failure with moderate protein restriction. It seems logical, therefore, to recommend individualization of therapy based on the severity of disease and the response of particular cases.

The approach of using different nutritional strategies for different stages of renal failure in dogs has been recommended (24) (**Table 2**). Use of a diet restricted in phosphorus (but not in protein) is recommended for dogs that are azotemic but not uremic. WALTHAM Veterinary Diet Canine Medium Protein would be appropriate for this stage of disease. It is restricted in phosphorus, while avoiding the high levels of protein that may be detrimental in dogs with CRF. Clear benefits were shown in a clinical study with this diet in dogs with mild to moderate renal insufficiency (25).

Recommendations for dogs that are uremic and have more advanced disease are to restrict dietary protein as well as phosphorus. In these cases WALTHAM Veterinary Diet Canine Low Protein may be the most appropriate choice, although a mixture of the two diets may be fed to individualize protein intake (**Table 3**). All cases should be carefully observed to assess the response to diet therapy and monitor for changes in the patient's condition that may require modifications of therapy.

The situation is less clear-cut in cats. Controversy currently exists regarding the influence of dietary protein restriction on the progression of feline renal dysfunction (26, 27). One recent study showed that dietary protein and energy restriction limited proteinuria and glomerular injury in cats with induced renal failure, although there was no effect on glomerular filtration rate (26). Unfortunately the study did not differentiate between the effects of protein and energy restriction.

Given the inconclusive nature of these data, it is not clear whether protein restriction will be of benefit in nonuremic cats with early renal failure. Studies with WALTHAM Veterinary Diet Feline Low Protein have confirmed that its protein content is adequate to



maintain nutritional status in cats with naturally occurring CRF; thus risks of protein malnutrition with this diet are minimal (11, 12). Pending further research to confirm whether reducing dietary protein intake has an impact on the progression of renal failure in cats, it is recommended that protein restriction should be considered for cats with azotemia and hyperphosphatemia if they persist despite good hydration, even when they are not showing signs of uremia.

Potassium

Hypokalemia has been shown to be the most common electrolyte abnormality in cats with CRF. In one study, hypokalemia was present in approximately 30% of cases (28). It has been suggested that increased urinary potassium losses may represent a primary renal abnormality peculiar to cats with CRF, which may lead to potassium depletion if compounded by decreased dietary potassium intake. The dietary potassium content of the diet used in the study leading to this observation was 0.34% DM (29). These data suggest that this level of dietary potassium may be inadequate for many cats with CRF. Diets designed for the management of CRF in cats should contain at least 0.6 g potassium/400 kcal ME. This level of dietary potassium has been shown to be adequate to maintain normal serum potassium concentrations in cats with naturally occurring CRF (11, 12).

However, not all cats with CRF show hypo- or normokalemia. In a recent report of 116 cases of feline CRF, 30% showed hypokalemia, but a further 13% showed *hyperkalemia* (30). In one clinical study involving 35 cats with CRF (11, 12), two of three cats which were hyperkalemic were the most severely affected in the trial (serum creatinine concentrations 5.4 [477] and 6.0 mg/dl [530 μ mol/l]). The presence of hyperkalemia may therefore be a reflection of the severity of renal insufficiency.

These observations emphasise the need to monitor potassium status in cats with CRF and the possible requirement for supplementation or adjustment of intake on an individual basis.

Calcium

Calcium concentrations in the blood of patients with CRF may be low, normal, or high (14, 31, 32). Decreased ionized calcium, where present, may contribute to increased PTH release (3). A number of factors may contribute to hypocalcemia, including hyperphosphatemia, poor gastrointestinal absorption associated with decreased levels of calcitriol, and reduced intake following anorexia. It has been recommended that calcium intake should be normal or supplemented in patients with CRF (33).

Conversely, if the product of the concentrations of calcium and phosphate ions in the blood exceeds the solubility product of calcium phosphate (estimates from human patients suggest the calcium $\times$ phosphorus product should not exceed about 55 mg/dl) (34), soft tissue calcification may occur, leading to the progression of renal damage. Calcium supplementation, which would be desirable in the presence of known hypocalcemia, would be contraindicated in the presence of hypercalcemia.

Sodium

Sodium homeostasis is maintained primarily by the kidneys. As GFR falls in the diseased state, surviving nephrons increase their fractional excretion of sodium to cope with the increased load

Dietary potassium levels should be enhanced for most cats with CRF, but regular monitoring of potassium status is essential

Dietary calcium levels should be adjusted to suit the individual patient

Dietary sodium levels should be moderately restricted

delivered to each one. In general, this response is adequate to maintain sodium balance until the condition is very advanced. However, the ability of the kidney to adapt to changes in sodium intake becomes progressively limited (35).

There is controversy over the prevalence of hypertension among dogs with chronic renal disease: Some reports suggest that it is between 58–93% whereas others suggest a much lower figure (36–38). Although less well documented than in other species, systemic hypertension has also been reported in association with chronic renal insufficiency in cats (39).

Hypertension, where it occurs, may be important for two reasons:

- It can result in a variety of pathophysiologic consequences, including left ventricular hypertrophy, neurologic abnormalities, and ocular lesions.
- It may contribute to the progression of renal damage.

Reduction of blood pressure in patients with documented hypertension is thus a desirable goal of therapy (40). It has been reported that expansion of extracellular fluid volume, hypertension, and edema may occur in uremic dogs receiving normal or high sodium intake (41). These findings suggest that the traditional recommendation to supplement with sodium is not appropriate for most cases of CRF.

Conversely, severe sodium restriction should probably also be avoided. This could promote volume depletion in some dogs with CRF that are unable to adapt to varying sodium intake. It may also result in a decreased capacity to reabsorb bicarbonate, thus contributing to metabolic acidosis. However, this latter effect was not seen when diets containing 0.25% DM sodium were fed (35, 42).

For these reasons, most recommendations are for diets with 'normal' to 'moderately restricted' sodium content for dogs with CRF. Suggested ranges include 0.25 to 0.8% DM (35), and 0.1 to 0.3% DM (40, 41). Diets designed for the management of CRF are likely to have lower sodium contents than the normal foods that a patient may previously have been fed. This difference is one important reason for recommending a gradual introduction of dietary therapy (over 1 to 2 weeks), because the capacity to adjust sodium excretion rapidly in response to changes in intake becomes impaired as renal insufficiency progresses (41).

Specific pharmacologic therapy should probably be reserved for those cases with documented hypertension.

B-complex vitamins

A tendency toward development of water-soluble vitamin deficiency has been noted in human uremic patients, particularly in those with poor dietary intakes (43). Dogs and cats with CRF are also potentially at risk of water-soluble vitamin deficiency through reduced intake from inappetence and increased urinary losses in polyuric cases.

If present, water soluble vitamin deficiency may contribute to anorexia. Supplementation with B-complex vitamins is therefore likely to be beneficial, and at least twice the maintenance level is recommended.

Energy

The metabolism of protein (either from dietary or endogenous sources) to provide energy is undesirable in patients with CRF since this increases the amount of nitrogenous waste products that must be excreted through the failing kidneys. An adequate energy supply in the diet is

Dietary vitamin levels should be adequate to compensate for possible losses

Sufficient energy should be available from nonprotein sources to meet the patient's requirements

therefore important to prevent further tissue catabolism and, as far as possible, should be derived from non-protein sources.

Appetite is often poor in affected animals, so the energy density of the diet should be high to enable the animal to obtain its nutritional requirements from a relatively small volume of food. In this respect, fat offers advantages over carbohydrate as a non-protein source of energy: It provides approximately twice the energy per gram and aids palatability in the diet. For this reason, canned diets designed to support dogs and cats with CRF tend to be high in fat. One concern about this design is whether the high fat content could adversely influence lipid metabolism in CRF patients, perhaps contributing to progression of renal damage.

Abnormalities in lipid metabolism have been documented in a variety of human renal diseases and have also been reported in dogs with both spontaneous and induced renal disease (1, 44, 45). Changes in human patients are thought to result (at least in part) from decreased activity of enzymes involved in lipoprotein metabolism (lipoprotein lipase and lecithin:cholesterol acyltransferase [LCAT]) resulting in increased concentrations of potentially atherogenic lipoproteins (partially metabolized low density and very low density lipoproteins).

In addition to creating a more atherogenic environment, these lipoproteins may also be responsible for glomerulosclerosis, a process which may have similarities to atherosclerosis (45–47). Lipid metabolism has not been studied extensively in dogs with CRF, but one report documented increased serum cholesterol concentration and a shift in the distribution of cholesterol from high density to low density lipoprotein fractions in a small group of dogs with spontaneous renal failure (44).

The effect of WALTHAM Veterinary Diet Canine Medium Protein (canned) (which provides over 50% of ME from fat) on lipid profiles and metabolism in healthy dogs and those with naturally occurring CRF was recently investigated (48, 49). Plasma total cholesterol, triglyceride, lipoprotein cholesterol distribution, and LCAT activities were investigated. LCAT is involved in cholesterol metabolic regulation and is associated with high density lipoproteins, the major lipid transport particle in dogs. As a part of reverse cholesterol transport, it is significant in that it catalyses the first important enzymatic step in returning cholesterol to the liver for utilization or excretion (50).

Significant ($p < 0.01$) increases in plasma total cholesterol were seen in both groups of dogs following the change to WALTHAM Veterinary Diet Canine Medium Protein. This was associated with significant increases in α -migrating (high density) lipoproteins in the healthy dogs. A similar trend was observed in the dogs with CRF, although the difference was not statistically significant. There were no changes in β -migrating (low density) lipoproteins or triglyceride concentrations in either group. Plasma LCAT activity increased significantly in the healthy dogs, and, again, a similar trend was seen in the dogs with CRF.

It was not possible to determine whether the abnormalities in lipoprotein metabolism known to occur in human patients with CRF (such as decreased LCAT activity) were present in the dogs in this study because some of them had been switched to phosphorus and protein-restricted, relatively high-fat diets prior to study entry. If similar abnormalities do occur in dogs with CRF, the changes in this study resulting from dietary intervention with WALTHAM Veterinary Diet Canine Medium Protein could actually benefit the patient by reversing abnormalities induced by CRF. Even if this is not the case, the increase in total cholesterol associated with the high-density lipoproteins is unlikely to be atherogenic or promote glomerulosclerosis, indicating that the inclusion of high fat levels to increase non-protein energy in this type of diet is not going to be detrimental to the CRF patient.

CONCLUSION

Dietary management is an essential component of therapy and may help to prolong and enhance the quality of life for dogs and cats with CRF. It should be remembered that CRF is a dynamic condition and regular monitoring of patients is an important component of management. The priorities in monitoring are assessment of physical status (especially hydration and body weight) and evaluation of key laboratory parameters (blood urea nitrogen, creatinine, phosphorus, PTH, potassium, packed cell volume, serum albumin, and total protein). In addition, compliance to dietary and medical therapy should be monitored. The frequency of monitoring depends on the severity of the patient's condition. Regular monitoring provides an opportunity to review status and optimize therapy.

REFERENCES

1. Brown, S. A., Crowell, W. A., Barsanti, J. A. et al. Beneficial effects of dietary mineral restriction in dogs with marked reduction of functional mass. *Journal of the American Society of Nephrology* 1991; **1**: 1169–79.
2. Finco, D. R., Brown, S. A., Crowell, W. A. et al. Effects of dietary phosphorus and protein in dogs with chronic renal failure. *American Journal of Veterinary Research* 1992; **53**: 2264–71.
3. Nagode, L. A., Chew, D. J., Steinmeyer, B. S., Carothers, M. A. Renal secondary hyperparathyroidism: Toxic aspects, mechanisms of development, and control by oral calcitriol treatment. *Proceedings of the 11th ACVIM Forum*, 1993; 154–7.
4. Ibels, L. S., Alfrey, A. C., Haut, L. et al. Preservation of function in experimental renal disease by dietary restriction of phosphate. *New England Journal of Medicine* 1978; **298**: 122–6.
5. Massry, S. G. The toxic effects of parathyroid hormone in uraemia. *Seminars in Nephrology* 1983; **3**: 306–28.
6. Nagode, L. A., Chew, D. J. Nephrocalcinosis caused by hyperparathyroidism in progression of renal failure: treatment with calcitriol. *Seminars in Veterinary Surgery* 1992; **7**: 202–20.
7. Nagode, L. A., Chew, D. J., Steinmeyer, B. S. et al. Low Doses of Oral Calcitriol Retard Progression of Naturally Occurring Renal Disease in the Dog. In: Norman, A., Bouillon, R., Thomasset, M. et al (eds). *Vitamin D-gene Regulation, Structure, Function Analysis and Clinical Application*. New York; De Gruyter, 1991: 879–80.
8. Finco, D. R., Brown, S. A., Cooper, T., et al. Effects of parathyroid hormone depletion in dogs with induced renal failure. *American Journal of Veterinary Research* 1994; **55**: 867–73.
9. Ross, L. A., Finco, D. R., Crowell, W. A. Effect of dietary phosphorus restriction on the kidneys of cats with reduced renal mass. *American Journal of Veterinary Research* 1982; **43**: 1023–6.
10. Barber, P. J., Rawlings, J. M., Markwell, P. J., Elliott, J. Treatment of hyperparathyroidism in feline chronic renal failure. *Journal of Veterinary Internal Medicine* 1996; **10**: 166.
11. Harte, J. G., Markwell, P. J., Moraillon, R. Dietary management of feline renal disease. *Feline Medicine Symposium (in association with North American Veterinary Conference)*. Waltham, Vernon, Ca., 1993: 27–36.
12. Harte, J. G., Markwell, P. J., Moraillon, R. M. et al. Dietary management of naturally occurring chronic renal failure in cats. *Journal of Nutrition* 1994; **124**: 2660S–2662S.



13. Chew, D. J., Nagode, L. A., Carothers, M. A., Steinmeyer, C. L. Calcitriol treatment of renal secondary hyperparathyroidism in dogs and cats. *Proceedings of the 11th ACVIM Forum*, 1993; 164.
14. Barsanti, J. A., Finco, D. R. Dietary management of chronic renal failure in dogs. *Journal of the American Animal Hospital Association* 1985; **21**: 371–6.
15. Polzin, D. J., Osborne, C. A., Lulich, J. P. Effects of dietary protein/phosphate restriction in normal dogs and dogs with chronic renal failure. *Journal of Small Animal Practice* 1991; **32**: 289–95.
16. Hansen, B., DiBartola, S. P., Chew, D. J., Brownie, C., Nagode, L. Clinical and metabolic findings in dogs with chronic renal failure fed two diets. *American Journal of Veterinary Research* 1992; **53**: 326–34.
17. Rogers, Q. R., Morris, J. G., Friedland, R. A. Lack of hepatic enzymatic adaptation to low and high levels of dietary protein in the adult cat. *Enzyme* 1977; **22**: 348–56.
18. Brenner, B. M., Meyer, T. W., Hotstetter, T. H. Dietary protein intake and the progressive nature of kidney disease. *New England Journal of Medicine* 1982; **307**: 652–9.
19. Hostetter, T. H., Olsen, J. L., Rennke, H. G., Venkatachalam, M. A., Brenner, B. M. Hyperfiltration in remnant nephrons: a potentially adverse response to renal ablation. *American Journal of Physiology* 1981; **241**: F85–F93.
20. Bovee, K. C., Kronfeld, D. S., Ramburg, C., Goldschmidt, M. Long-term measurement of renal function in partially nephrectomized dogs fed 56, 27 or 19% protein. *Investigative Urology* 1979; **16**: 378–84.
21. Robertson, J. L., Goldschmidt, M., Kronfeld, D. S. et al. Long-term responses to high dietary protein in dogs with 75% nephrectomy. *Kidney International* 1986; **29**: 511–9.
22. Brown, S. A., Finco, D. R., Crowell, W. A., Choat, D. C., Navar, L. G. Single nephron adaptations to partial renal ablation in the dog. *American Journal of Physiology* 1990; **258**: F495–F503.
23. Brown, S. A., Finco, D. R., Crowell, W. A., Navar, L. G. Dietary protein intake and the glomerular adaptations to partial nephrectomy in dogs. *Journal of Nutrition* 1991; **121**: S125–S127.
24. Brown, S. A. Dietary management of canine renal disease: a staged approach. *Proceedings of the BSAVA Congress*, 1995; 47.
25. Leibetseder, J. L., Neufeld, K. W. Effects of medium protein diets in dogs with chronic renal failure. *Journal of Nutrition* 1991; **121**: S145–S149.
26. Adams, L. G., Polzin, D. J., Osborne, C. A., O'Brien, T. D., Hostetter, T. H. Influence of dietary protein/calorie intake on renal morphology and function in cats with 5/6 nephrectomy. *Laboratory Investigation* 1994; **70**: 347–57.
27. Finco, D. R., Brown, S. A. Newer Concepts and Controversies on Dietary Management of Renal Failure. In: Kirk, R. W. (ed). *Current Veterinary Therapy X: Small Animal Practice*. Philadelphia: Saunders, 1989; 1198–1201.
28. DiBartola, S. P., Rutgers, H. C., Zack, P. M., Tarr, M. J. Clinicopathological findings associated with chronic renal disease in cats. *Journal of the American Veterinary Medical Association* 1987; **190**: 1196–1202.
29. Dow, S. W., Fettman, M. J., LeCouteur, R. A., Hamar, D. W. Potassium depletion in cats: renal and dietary influences. *Journal of the American Veterinary Medical Association* 1987; **191**: 1596–1575.
30. Lulich, J. P., Osborne, C. A., O'Brien, T. D., Polzin, D. J. Feline renal failure: questions, answers, questions. *Compendium for Continuing Education for the Practicing Veterinarian* 1992; **14**: 127–53.
31. Finco, D. R., Rowland, G. N. Hypercalcaemia secondary to chronic renal failure in the dog and a report of 4 cases. *Journal of the American Veterinary Medical Association* 1978; **179**: 990–4.
32. Polzin, D. J., Osborne, C. A., Hayden, D. W., Stevens, J. B. Influence of reduced protein diets on morbidity, mortality, and renal function in dogs with induced chronic renal failure. *American Journal of Veterinary Research* 1984; **45**: 506–17.
33. Bovee, K. C. The role of dietary protein in the pathogenesis and management of chronic renal failure in the dog. *Proceedings of the 7th Kal Kan Symposium*. Kal Kan, Vernon Ca., 1983; 27–33.
34. Massry, S. G. Requirements of vitamin D metabolites in patients with renal disease. *American Journal of Clinical Nutrition* 1980; **33**: 1530–5.
35. Polzin, D. J., Osborne, C. A. and O'Brien, T. Diseases of the kidneys and ureters. In: Ettinger, S. J. (ed). *Textbook of Veterinary Internal Medicine (3rd edn)*. Philadelphia: Saunders, 1989; 1962–2046.
36. Cowgill, L. D., Kallet, A. J. Recognition and management of hypertension in the dog. In: Kirk, R. W. (ed) *Current Veterinary Therapy VIII*. Philadelphia: Saunders, 1983; 1025–8.
37. Osborne, C. A., Polzin, D. J., Abdullah, S., Klausner, J. S. Role of diet in the management of feline chronic polyuric renal failure: current status. *Journal of the American Animal Hospital Association* 1982; **18**: 11–20.
38. Michell, A. R., Bodey, A. R. Canine hypertension. *Proceedings of the 12th ACVIM Forum*, 1994; 502–5.
39. Morgan, R. V. Systemic hypertension in four cats: Ocular and medical findings. *Journal of the American Animal Hospital Association* 1986; **22**: 615–21.
40. Ross, L. A., Labato, M. A. Use of drugs to control hypertension in renal failure. In: Kirk, R. W. (ed) *Current Veterinary Therapy X*. Philadelphia: Saunders, 1989; 1201–4.
41. Cowgill, L. D. Diseases of the Kidney. In: Ettinger, S. J. (ed). *Textbook of Veterinary Internal Medicine (2nd edn)*. Philadelphia: Saunders, 1983; 1793–1878.
42. Schmidt, R. W., Gavellas, G. Bicarbonate reabsorption in experimental renal disease: effect of proportional reduction of sodium or phosphate intake. *Kidney International* 1977; **12**: 393.
43. Kopple, J. D. Chronic Renal Failure: Nutritional and Nondialytic Management. In: Glasscock, R. J. (ed). *Current Therapy in Nephrology and Hypertension* 1984–85. St Louis: Mosby, 1984; 252.
44. Down, L. K., Krawiec, D. R. Lipid abnormalities in canine chronic renal failure. *Proc 13th ACVIM Forum*. Blacksburg: ACVIM, 1995; 1027.
45. Keane, W. F., Kasiske, B. L., O'Donnell, M. P. Hyperlipidaemia and the progression of renal disease. *American Journal of Clinical Nutrition* 1988; **47**: 157–60.
46. Attman, P.-O., Alaupovic, P. Lipid abnormalities in chronic renal insufficiency. *Kidney International* 1991; **39**: S16–S23.
47. Attman, P.-O., Samuelsson, O., Alaupovic, P. Lipoprotein metabolism and renal failure. *American Journal of Kidney Disease* 1993; **21**: 573–92.
48. Malcik, K., Bauer, J. E., Crocker, R. et al. Lipoprotein metabolism and LCAT activities of dogs with chronic renal failure. *Journal of Veterinary Internal Medicine* 1996; **10**: 194.
49. McAlister, K. G., Bauer, J. E., Harte, J., Rawlings, J. M., Markwell, P. Canine plasma lipoproteins and lecithin:cholesterol acyltransferase activities in dietary oil supplemented dogs. *Veterinary Clinical Nutrition* 1996; **3**: 50–56.
50. Fielding, P. E., Fielding, C. J. Dynamics of Lipoprotein Transport System. In: Vance D. E., Vance J. (eds). *Biochemistry of Lipids, Lipoproteins, and Membranes*. New York: Elsevier, 1991; 427–59.