THE GERIATRIC CAT: COMPLEX MANAGEMENT IF MULTIPLE DISORDERS

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Elderly cats often present with multiple concurrent conditions. "Age-associated" or "age-appropriate" illnesses that we expect to see in older cats include problems related to the urinary tract (chronic kidney disease (CKD), pyelonephritis, calcium oxalate ureteronephroliths, bacterial cystitis), endocrine system (hyperthyroidism, diabetes mellitus, hyperaldosteronemia), degenerative joint disease (DJD) and other musculoskeletal conditions, dental diseases and neoplasia. Constipation may become an ongoing concern. Certain infectious diseases become more likely in the older individual (e.g., FIP). A decline in functioning of the special senses occurs frequently and behaviour changes suggestive of cognitive dysfunction may be seen in some individuals.

Making management recommendations may be challenging as treatments, at first glance, may appear to be in conflict. Thankfully, the body is complex and all systems relate to one another. The four most important therapeutic considerations that must be incorporated in caring for every patient, especially those who are older, are optimizing comfort through analgesia, hydration, nutrition and ensuring that the environmental needs are met so they can perform normal behaviours.

MANAGING A CAT WITH STAGE 2-3 CHRONIC KIDNEY DISEASE, HYPERTHYROIDISM, HYPERTENSION AND DEGENERATIVE JOINT DISEASE

Renal disease is fairly common in untreated hyperthyroid cats. It may be masked due to increased cardiac output, renal blood flow and glomerular filtration rate (GFR). The effects of muscle wasting exacerbate the lower Cr concentrations. Monitoring renal parameters and muscle condition during therapy is advised. Similarly, hypertension may become evident only during the course of therapy or even after the patient is euthyroid. It is well recognized that amelioration of the hyperthyroid state by any method (i.e., medical therapy, 131I treatment or surgery) can result in decreased GFR, elevations in serum urea nitrogen and creatinine (Cr), and, in some cases, overt azotemia. The decline in GFR stabilizes by approximately four weeks (Adams, Becker, Boag, DiBartola, Graves).

Numerous studies have attempted to identify parameters that predict declining renal function before correcting hyperthyroidism. Glomerular filtration rate can be measured using plasma clearance of exogenous creatinine exo-iohexol or endo-iohexol; N-acetyl-beta-D-glucosaminidase index and retinol-binding protein have been assessed as possible biomarkers (Lapointe, Riensche, Slater). Symmetric dimethylarginine (SDMA) is a promising biomarker for estimating GFR (Hall JA). It has been shown to be an accurate and precise biomarker for calculating estimated GFR in humans. It is a more sensitive biomarker than serum Cr for assessing early renal dysfunction. By reducing the lower limit of the Cr reference interval and monitoring this parameter longitudinally to look for upward trends, earlier detection in GFR decline can be achieved. False positive elevations occur with SDMA just as they do with Cr with prerenal azotemia, therefore pre-renal causes of decreased GFR and causes of acute kidney injury have to be eliminated before interpreting the result. What role it, or any other early predictive biomarker, will play in the management of CKD in cats is uncertain.

Using common clinical measures, cats with hypertension and/or an increase urine protein: creatinine ratio (UPC) are more likely to develop problems while cats with elevated plasma globulins, a high urine specific gravity (usg) and hematocrit are less likely to.

A practical approach to a patient with concurrent hyperthyroidism and CKD is to treat medically until the serum T4 is adequately controlled at which time the effect of permanent therapy may be predicted. If renal decline becomes apparent once euthyroidism has been achieved, exogenous thyroid hormone can be supplemented to support the kidneys. A balance must then be struck between creating iatrogenic hyperthyroidism and maintaining renal function as iatrogenic hypothyroidism appears to contribute to azotemia and decreased survival. (Peterson 2013, 2013, 2014, Williams, Aldridge) It is recommended to monitor serum TSH levels post radioiodine or during medical therapy to avoid iatrogenic hypothyroidism (Peterson ACVIM 2014, Peterson Enrollment in Clinical Study, 2016).

These cats should either receive a lower dose of medication or be supplemented with thyroxine before the develop overt disease or CKD.

The foundation of any treatment plan includes attending to hydration, nutrition, analgesia, and addressing environmental comfort. In this scenario, maintaining euvolemia will be reflected in coat and stool character as well as subjective measures of well-being including grooming, interaction and posture. Daily subcutaneous fluids (warmed to body temperature) may be appropriate. A client handout is included at the end of these notes. Appetite may be negatively affected by renal disease from uremic toxins or, less likely, uremic gastritis. Unlike humans and dogs, cats with CKD are more likely to have gastric fibrosis rather than uremic gastropathy (McLeland)). Nausea associated with uremic acidosis may be alleviated with famotidine 5 mg PO q24h or another H2 antagonist. However, the proton pump inhibitor omeprazole has been shown to provide better acid suppression than famotidine in cats (Parkinson). Additionally, twice-daily omeprazole is more efficacious at suppressing acid production than once daily dosing or ranitidine therapy (Šutalo).

Appetite stimulation with mirtazapine (1.88 mg PO q48h) has been shown to benefit cats with CKD (Quimby 2013). Interestingly, maropitant, while effective in palliating vomiting, did not significantly improve appetite or support weight gain in cats with Stage II and III CKD (Quimby 2015). Cats with untreated hyperthyroidism may have an increased appetite yet, due to increased metabolism, lose weight and muscle. Once euthyroid, their appetites generally normalize.

It is generally recommended to feed cats with Stage 2- 3 kidney disease, a protein-restricted, renal diet. We must consider several questions when treating each individual. Do cats at every stage of CKD have identical nutritional needs? Do all cats at the same stage have the same etiologic cause for their decline in renal function? Could this cat, perhaps, benefit from being fed a protein enhanced diet, a recuperative diet, a growth diet, a senior diet or a maintenance diet?

Protein: calorie malnutrition results from ingesting adequate (or excessive) total calories, but insufficient protein calories. Weight loss may or may not be occur, however there will be muscle wasting as well as a deterioration in the hair coat quality. Additionally, because antibodies are protein, immune function may be compromised; anemia may be exacerbated due to the lack of building components for hemoglobin; albumin levels may decrease and tissue healing may be affected. If a cat is inappetant, restricting protein may result in inadequate intake of all nutrients, because amino acids are key in palatability, resulting in the protein intake falling below that required for normal function.

It appears that some loss of weight and lean body mass (LBM) is part of normal aging (Armstrong, Dora-Rose, Bellows), and is not caused by illness but we may be able to address it nutritionally, potentially improving longevity as well as quality of life. In part this is associated with decreased digestion of fat and protein, (Perez-Camargo) in part due to increased requirements for protein and calories that may not be met. (Perez-Camargo, Harper, Laflamme X2, Villaverde) Maintenance of weight and condition prolongs life. (Cupp) Given that cats with CKD often live for a long time, (King 2007, Boyd) the nutritional needs of older cats should be taken into consideration in treatment of those with CKD.

Weight loss with poor muscle condition is common among cats with CKD. Weight loss begins before 1 to 3 years before a diagnosis of CKD has been made (Freeman) and is associated with decreased survival. (Boyd, Freeman) A decrease in muscle mass is associated with increased morbidity and mortality in human CKD patients (Wang). Protein synthesis is reduced in these individuals compared to healthy human patients. While inadequate calorie intake contributes to weight, fat and LBM loss, reduction in dietary protein exacerbates the CKD-related muscle loss (Wang) in attempts to meet the needs for protein turnover and ongoing metabolic needs. Increasing dietary protein helps to regulate acid-base balance through excretion of hydrogen ions associated with ammonium. (Remer) This is of clinical relevance when we try to design the optimal nutritional regime for our older feline patients: protein and fat restriction may well be contraindicated. Especially if underweight, older cats will benefit from a more energy-dense, highly digestible diet to help offset these age-related digestive and metabolic changes.

As obligate carnivores, cats have evolved to manage a high dietary protein load (Hewson-Hughes X2, Plantinga), but adapt to various intake as long as their minimum needs are met. (Green) Dietary protein is not toxic to kidneys. Creatinine and urea result from metabolizing ingested protein as well as from turnover of endogenous stores in health or in protein deficiency. In excess of 60 purported uremic toxins are reported. (Vanholder 2003) Interactions between many endogenous metabolites (Lisowaska-Myjak) resulting from inflammation, malnutrition, increased concentrations of protein-bound solutes and hypoalbuminemia as well as non-nutritional toxins contribute to the clinical spectrum of uremia. (Vanholder X2, Stenvinkel) Thus, while uremic toxins can result in malnutrition, malnutrition itself results in inflammation, morbidity and mortality in human patients with CKD. (Vanholder 2002)

Despite numerous experimental studies and clinical trials, questions about feeding and managing the cat with CKD remain. Some of these are:

1. Do we over-rely on diet? Are there other approaches we could utilize to reduce uremic toxin production or absorption? The renal effects of endogenous protein breakdown have not been studied but may potentially be no different than those of dietary protein.
2. Do different types of kidney disease require different dietary therapies? Fibrosis associated with interstitial changes is the end-point for most cats, however what etiology initiates the process is generally unknown in an individual cat.
3. At what point in disease progression should dietary therapy be implemented, if at all? In theory, would it be better to address acid-base balance initially, and then phosphorus binding, or vice versa?
4. What is the optimal amount of protein for cats with CKD? How much restriction, if any, is necessary? Similar to cats, protein malnutrition, sarcopenia, and iron deficiency are clinical problems in human CKD patients following low protein regimes.
5. Does the type of protein, or the amino acid composition of the protein, make a difference in cats? There is evidence in humans and rats that types of protein can differentially influence the effect of protein on GFR, acid-base and other effects. (Williams, Kontessis, Pecis)
6. Will a cat in IRIS stage 3 or 4 benefit adequately if phosphorus is restricted by means other than diet? No controlled clinical trials address this question.
7. Might some cats with advanced disease benefit from increased dietary protein levels? As discussed earlier, regular reassessment of the patient enables evaluation of muscle and body condition, which is helpful in changing dietary treatment recommendations if warranted. As loss of lean mass is detrimental as well as predictive of progression, increasing dietary protein and using alternate methods to restrict phosphorus or uremic toxins should be considered. When patients fail to eat adequate calories (protein, fat or carbohydrate), then feeding support is required.
8. What are the actual uremic toxins that cause adverse effects in cats and what can we do about them?
9. Should we be investigating phosphatonins (e.g., fibroblast growth factor-23 [FGF-23]) and their role in phosphate homeostasis in cats and potentially seeking ways to block or correct FGF-23 as GFR declines? Finch et al (2013) reported an inverse relationship between FGF-23 concentrations and GFR and demonstrated that FGF-23 is increased in cats that go on to become azotaemic before phosphate concentrations increase. PTH also changes before serum phosphorus. Would these make better markers of progression of renal dysfunction?
10. Progression of renal fibrosis is thought to be related to the ongoing production of pro-inflammatory and pro-fibrotic cytokines. Proteinuria, hypoxia, hyperphosphatemia, ageing and chronic inflammation have been investigated and are believed to maintain this state (Lawson). Should the focus of early identification (e.g., SDMA) and treatment be modification of the inflammatory mediators?
11. Is it appropriate to restrict protein in cats with proteinuria? While protein in urine may initiate an inflammatory response that ultimately progresses to interstitial fibrosis (Lawson), muscle wasting and a perceived decreased quality of life may result in an earlier death, either due to general decline in health or earlier requested euthanasia. Would this be better addressed pharmacologically rather than risking malnutrition? Malnutrition also results in inflammation and mortality, therefore preventing malnutrition, (as well as sarcopenia), is critically important when managing the feline CKD patient.

Protein-restricted therapeutic diets are not identical; there are some marked differences in their composition, not just in protein sources and quantities, but also in the calorie source, in their phosphorus, potassium, and sodium

content. They are variably supplemented with potassium and fatty acids, restricted in phosphorus. Every patient's response to a given diet may differ and each cat should be rechecked to assess the effects and suitability of the recommended diet. Table 1 compares reduced protein and phosphorus foods as of December 2012: composition of these diets will have changed since this time.

With muscle wasting, Cr levels decrease making it difficult to know how much of a Cr decrease seen in a cat fed a renal diet is from improvement in renal function and how much is because there is less functional muscle producing Cr. Given the smaller number and size of the studies in veterinary medicine, we are unable to perform meta-analysis. It seems prudent to make the following recommendation: when prescribing restricted protein renal diets practitioners must carefully monitor their patients' protein and energy intake and nutritional status, as evidenced by body and muscle condition as well as enjoyment of meals/quality of life. If deterioration in any of these is noted with no other apparent reason, alternate diets or means to reduce phosphorus should be considered. The WSAVA nutrition tools are a useful resource for the BCS and MCS.
http://www.wsava.org/sites/default/files/Body%20condition%20score%20chart%20cats.pdf
http://www.wsava.org/sites/default/files/Muscle%20condition%20score%20chart-Cats.pdf
The MCS may be found at the end of the notes.

Because of inherent progression of CKD, **IRIS staging focuses on factors which, when managed, are known to slow progression**: azotemia, metabolic acidosis, hyperphosphatemia, proteinuria and hypertension.

Control of **hypertension** is important for its negative effects on cardiac function as well as progression of CKD. Amlodipine is the first choice (0.625 mg PO q24h, titrate as needed). In cats with proteinuria, telmisartan may be the best therapeutic choice (Jenkins) especially in cats that are also hypertensive (1 mg/kg PO q24h). (Sent)

Addressing the **degenerative joint disease** in a patient with CKD requires thought but is not insurmountable. The most common concern regarding drug therapy are side effects of using NSAIDs in a dehydrated patient and their effects on gastric mucosal health or on renal function.

Opioids are safe for pain relief in any age group and are excellent when used at the same time as other agents, especially NSAIDs. They are not, however, a first drug of choice for cats with arthritic pain as they are not very effective for DJD. This is not to suggest that they shouldn't be used for "break-through" pain or for comfort during diagnostic testing. If they produce adverse side-effects (e.g., euphoria, constipation and inappetence) in an individual patient they may be reserved for palliative hospice care.

Pharmacokinetic data is lacking for safe, long-term use of many NSAIDs in cats. Carprofen half-life varies from nine to over 40 hours in cats (Taylor, Parton). As most NSAIDs have long half-lives in cats when compared to other species, one precaution to avoid toxicity is to reduce the frequency of administration. Interestingly, despite having a short half-life of under 2 hours in blood, robenacoxib (Onsior®) its effect persists for 24 h in clinical studies.

Metacam® 0.5 mg/ml oral suspension has been granted a licence in the EU for the alleviation of inflammation and pain in chronic musculoskeletal disorders in cats. The registered dose is 0.1 mg/kg on the first day followed by 0.05 mg/kg orally once daily. This is the first NSAID licensed for long-term use in cats.

Numerous efficacy studies have been performed regarding both of these NSAIDs. Two studies have evaluated long-term safety of this agent in older cats; one concluded that this agent is safe, efficacious and palatable for musculoskeletal pain at 0.01-0.03 mg/kg PO q24h for a mean treatment duration of 5.8 months; no deleterious effect on renal function was detected in cats studied. Gastrointestinal upset in 4% of cats was the only adverse effect noted (Gunew). The second, reviewed the medical records of cats over seven years of age treated for a minimum of 6 months with a daily maintenance dose of 0.02 mg/kg meloxicam and concluded that this dose does not hasten progression of renal disease in aged cats or aged cats with pre-existent stable IRIS stage 1-3 renal disease (Gowan).

A paper published in 2015 reported on the safety of robenacoxib (1–2.4mg⁄kg) for daily, month long treatment of

DJD in cats including 40 with chronic kidney disease IRIS stages 2-4. There was no evidence of increased risk in the frequency of reported adverse events, or in deterioration in renal variables in the subgroup of cats with concurrent CKD (King 2015). Despite being similar to meloxicam (class and mechanism of action), at this time (September 2016) it is licensed only for short-term use.

A comprehensive review of the long-term use of NSAIDs in cats was published in 2010. This document may be accessed free-of-charge at: http://www.catvets.com/guidelines/practice-guidelines/nsaids-in-cats
A suitable protocol for a cat with pain from DJD might be baseline NSAID (such as meloxicam or robenacoxib) with intermittent use of an opioid (such as burprenorphine) when break-through pain is evidenced by a decrease in appetite or mobility or social withdrawal. A recent study by King et al has shown that robenacoxib (1.0–2.4 mg/kg PO q24h) is safe in cats with, or without, CKD when administered for DJD for 30 days.

Disease-Modifying Osteoarthritis Agents: Over the past two decades, research has been aimed at attempts to slow the progression of cartilage degradation as well as to promote rebuilding of healthy matrix. Products have been developed that, in research on humans and dogs, have been shown to be beneficial in enhancing hyaluronic acid production, inhibiting catabolic enzymes in osteoarthritic joints, and encouraging normalization of the synovial fluid and joint cartilage matrix. There is no evidence for efficacy in cats.

Studies using radio-labeled compounds have shown that 87% of orally administered glucosamine is absorbed and is incorporated into the cartilage matrix. Glucosamine provides raw materials for synthesis of glycosaminoglycans. Since chondrocytes obtain preformed glucosamine from the circulation (or synthesize it from glucose and amino acids), adequate glucosamine levels in the body are essential for synthesis of glycosaminoglycans in cartilage. Glucosamine is required for the production of hyaluronic acid by synoviocytes*. In vitro* studies indicate that administration of glucosamine may normalize cartilage metabolism and stimulates the synthesis of proteoglycans. In one study, glucosamine stimulated synthesis of glycosaminolgycans, prostaglandin and collagen by chondrocytes and fibroblasts, suggesting it may actually up-regulate their synthesis.

Chondroitin Sulfate (CS) is a long chain polymer of repeating disaccharide units. It is the predominant glycosaminolycan found in articular cartilage and can be purified from bovine, whale, and shark cartilage sources. Bioavailibilty studies in rats, dogs and humans have shown that 70% of orally administered CS is absorbed, some of it intact. Studies in rats and humans using radiolabeled CS have shown that CS reaches synovial fluid and articular cartilage. Hyaluronate concentrations and viscosity were increased, and collagenolytic activity was decreased, in the synovial fluid of human osteoarthritis patients treated with CS for 10 days.

Both oral preparations (Cosequin) and parenterally injected preparations (Adequan) have been shown to have therapeutic benefit in *in vivo* studies. One caution is that a polysulfated glycosaminoglycan, such as Adequan, is a heparin analog, resulting in a transient prolonged partial thromboplastin time. Avoid using it in cats with bleeding disorders or pre-operatively and do not use it concurrently with either omega 3 fatty acids or NSAIDs.

Diet: Weight loss should be encouraged in the obese, arthritic cat to reduce the pressure on joints. The addition of omega 3 fatty acids may be beneficial by blocking the production of prostaglandins from arachadonic acid in the inflammatory cascade. Dietary modulation should be considered in the therapy of cats with DJD-associated pain (Lascelles). A therapeutic prescription diet for joint health: Hill's j/d with omega 3 fatty acids is available in both a canned and a dry formulation (504 kcal/cup; 152 kcal/156g can); to date the author is not aware of peer-reviewed papers on this diet. Royal Canin has developed Mobility Support (391 kcal/cup) for cats. Both of these diets may not have sufficient protein for some older patients. A recently published study comparing the Royal Canin diet to the identical diet without the added high levels of eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) and supplemented with green-lipped mussel (GLM) extract and glucosamine/chondroitin sulfate. Forty cats with no detectable systemic disease but with radiographic evidence of at least one affected joint and who showed an aversive response to manipulation of that joint were fed one of the two diets (control or test diet) for nine weeks. Cats on the test diet showed significantly greater improvements including an increased ability to jump, increased time spent eating, less time sleeping, increased playing and interacting with other pets. In an earlier study that

looked at efficacy and tolerance of an extract of GLM in dogs with DJD, a similar subjective improvement was noted by clients whether the dogs were receiving placebo extract or GLM extract.

Therapeutic exercise - Moderate exercise has benefits in maintaining range of motion in the face of joint capsule fibrosis, to maintain/build muscle mass and maintain/build healthy articular cartilage. In acute flare-ups, restricting activity may be warranted.

Additional therapeutic considerations – It is important to make environmental changes in the home as well as having empathic awareness in order to make appropriate changes in handling in a clinic setting. Decreased mobility can result in constipation due to retention, positional stiffness resulting in elimination outside of the litter box, falling when jumping onto or off the bed, inability to climb stairs, inability to crouch to eat resulting in weight loss. Raising bowls for cats with lumbosacral and hip DJD makes it easier to eat. Trimming nails regularly helps to maintain proper joint relationships. Ramps and steps onto favourite sleeping spots gives the cat access to safe places for sleeping and observation. Warm, soft, padded beds for stiff, painful, possibly bony joints should be considered. Adding a litter box so that kitty doesn't have to walk as far may reduce inappropriate elimination as well as encourage regular voiding and defecation. Making sure that the rim of the litter box is not too high, and that the opening into the box is not too small is helpful. Scooping the litter several times a day and making sure that the litter isn't too deep or too sparse will encourage regular use.
Regular nail trimming helps by maintaining proper joint relationships. Ramps and steps to favourite sleeping spots may be helpful. Warm, soft, padded sleeping places for stiff, painful, possibly bony joints should be considered. Adding a litter box to reduce the distance between boxes may reduce accidents as well as encourage regular voiding and defecation. The rim of the box mustn't be too high, nor the opening into the box too small. It should be scooped several times a day to encourage use.

Acupuncture may be considered and can safely be combined with pharmacologic approaches. While efficacy has been shown for acupuncture in a few conditions in humans, there is no solid scientific evidence at the time of writing that clearly supports its efficacy in cats. Peer-reviewed publications for efficacy of laser therapy and stem cell therapy are also lacking.

MANAGING A CAT WITH STAGE 2 CHRONIC KIDNEY DISEASE STAGE, DIABETES MELLITUS, PERIODONTAL DISEASE AND INFLAMMATORY BOWEL DISEASE

One of the challenges with these comorbidities is nutritional (protein quantity and source, meeting all nutritional requirements). Another is achieving immunomodulation for IBD in the face of glycemic instability. Treatment with corticosteroids takes priority over glycemic effects because inflammation is recognized as a predisposing factor for susceptible individuals to develop diabetes. Franchini has shown at a molecular level that the inflammation induced by bacterial or viral infection can, via molecules recognized by toll gate receptors, damage endocrine pancreatic tissue. In some individuals, IBD can be managed using chlorambucil and metronidazole without corticosteroids.

The oral disease adds to difficulties controlling the diabetes. Inflammation and/or infection at any site (dental, urinary tract, etc.) interferes with the ability of all cell receptors to interact with insulin. Anaesthesia for dental care should be performed before the diabetes or IBD are controlled. Before the procedure, ½ of the current dose of insulin is given on the morning of the procedure. The cat should only be fasted for 4 hours with water freely available. Blood glucose measurements should be made during and after the anaesthetic until the cat is able to eat on his own.

Concern regarding safety of performing anaesthesia in elderly cats should not be over-emphasized. Two studies have verified that in cats, as in people, age is not a risk factor and should not be a limiting factor in determining whether or not to undertake a medically beneficial procedure. (Hosgood, Brodbelt)

The American Society of Anesthesiologists (ASA) characterizes risk using the following physical status classification system that is based on the physical status of the patient. Five categories are defined as follows:

- Class 1: Normal, healthy patient

- Class 2: A patient with a mild systemic disease
- Class 3: A patient with severe systemic disease
- Class 4: A patient with a severe systemic disease that is a constant threat to life
- Class 5: A moribund patient not expected to survive without the operation

Feeding strategies for diabetes include a high protein, low carbohydrate diet or a high fiber diet. However, a diabetic cat can be controlled with insulin as long as any diet and treats fed remain consistent from day to day. Neither carbohydrates nor dry extruded diets are cause of diabetes or obesity. However, exchanging dietary carbohydrate for protein appears to be useful for weight loss treatment and management of non-insulin dependent diabetes in cats.

In a prospective, randomized, double blinded 10-week study (Hall TD), 12 cats (7/12 obese) of whom six were newly diagnosed and six were poorly controlled diabetics evaluated standard maintenance diet vs. lower carbohydrate, higher protein (LCHP) diets. The cats ate dry or canned based on their preference. All were treated with glargine and assessed at weeks 1, 2, 4, 6, and 10 with fructosamine, BG curve and clinical signs. One cat from each diet group achieved remission by week 10. All cats improved clinically, increased weight and achieved good glycemic control. Those fed the LCHP had a significantly greater decrease in fructosamine. The conclusion, based on this small study was that using insulin, "frequent monitoring is key to achieving glycemic control in diabetic cats; potential benefits of dietary modification require further evaluation". The author summarizes all of the preceding studies and approaches: high fiber & low fat, high insoluble fiber vs. low fiber, LCHP canned, low carbohydrate diet vs. low carbohydrate diet plus acarbose, low carbohydrate & low fiber diet vs. moderate carbohydrate & high fiber diet. None of these approaches appears to make a meaningful difference in the small numbers of cats in any of the reviewed studies.

Diet recommendations for a cat with a sensitive gastrointestinal tract and a diagnosis of "IBD" are either a limited antigen, a "hypoallergenic" or a hydrolyzed protein diet. Some cats may tolerate a highly digestible, low residue intestinal diet. Omega-3 fatty acids may be beneficial in small quantities. Unless the GI disease is extremely mild, this condition has dietary priority as the diabetes can be regulated on any diet if used consistently and protein restriction may be unnecessary or contraindicated.

Regardless of nutritional recommendation made, we are performing a feeding trial with one subject (n=1). We need to reassess the patient to determine how suitable the diet is for him. How is his weight? How is his coat? Does he eat with enjoyment or vigour? What are his stools like (moist logs or dry pellets, cow patties or coloured water)? How energetic is he since he has been on this diet? Has there been a change in his hematocrit and proteins? Have biochemistries or usg changed? Is he proteinuric and potentially protein deficient? What about his blood pressure? Ultimately, quality of life is more important than quantity.

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SAMPLE CLIENT HANDOUT INSTRUCTIONS ON SUBCUTANEOUS FLUID ADMINISTRATION

HOW TO GIVE YOUR CAT SUBCUTANEOUS FLUIDS

Warming the fluids to body temperature

1. If you are using an unopened bag:
    a. Remove the outside protective bag.
    b. Microwave the bag for two to three minutes, depending on the microwave.
    c. Massage the warmed bag to distribute the heat evenly.
    d. Test the bag on your wrist. It should feel comfortably warm—just about body temperature.
2. If the bag has already been used and has the line attached, do not microwave it as the line will melt and seal shut. Instead:
    a. Boil water in a kettle or pot.
    b. Put the bag into a vase or tall upright container with the bulb portion up so it will remain above the water.
    c. Pour the hot water into the vase, taking care to not reach the bulb.
    d. Set the timer for about five minutes, depending on how much is remaining in used bag.
    e. Massage the warmed bag to distribute the heat evenly.
    f. Test the bag on your wrist. It should feel comfortably warm—just about body temperature.

Connecting a new line to a bag

1. Prepare the line by rolling the wheel to a closed position.
2. Take the cap off the line, being careful not to touch the end of the line.
3. Remove the end from the port on the bag.
4. Insert the pointed end of the intravenous line into the port.
5. Squeeze the bulb of the intravenous line to fill the bulb half full.
6. Open the line by rolling the wheel to the open position, and fill the line with fluids.

Giving fluids

1. Hang the bag of fluids on a curtain rod or shower rod with the still capped line hanging down.
2. Place an unused, covered needle on the line, and place the sterile cap (from the end of the line) close by.
3. Sit somewhere comfortable. (I prefer the floor so that the cat feels secure.)
4. If you want, you can wrap your cat in a towel, leaving its head and shoulders exposed, and cradle your cat.
5. Remove the cover on the needle.
6. With cat facing away from you and while holding the needle, rest your dominant hand on your cat's back with the needle facing toward its head.
7. Lift and make a tent with the skin between your cat's shoulders with your nondominant hand.
8. Exhale and firmly pull that skin tent over the needle.
9. Open the intravenous line wheel, and administer the volume of fluids as directed by your doctor.
10. Because the fluids are warmed, once the needle is in place the cat should be comfortable. Giving treats and praise doesn't hurt either!
11. Close the intravenous line, and remove and discard the needle safely, recapping the line with the sterile cap.
12. Pinch the skin together with your nondominant hand when you remove the needle.

Congratulations! You've done it.

Notes:

1. While you are getting used to this procedure, it may help to have the fur shaved over two places at the back of the neck. That way you can be sure the needle is getting under the skin. The fur will grow back.
2. Your cat will look like it is wearing shoulder pads. The fluids will droop to one side down a leg, even to the paw. These will be absorbed over 12 to 24 hours.
3. If some of the fluids or even a bit of blood leak from the injection site, there is no need to worry.

WSAVA MUSCLE CONDITION SCORING TOOL