“The Aging Process: Why pets Age and How we can influence the process.”

In the last AVMA pet owner survey, more than 39% of the owned pet population were 7 years of age or older and these percentages continue to grow. This change in pet population demographics is in part due to advances in the control of infectious disease, improvements in nutrition, newer surgical techniques, newer medications for controlling chronic disease, plus more comprehensive understanding of the aging process. Changing owner attitudes toward elderly pets (human animal bond / humanization of pets), has also contributed to potential increases in life expectancy.

An animal’s life can be divided into four stages; pediatric, adult, senior, and geriatric. The senior years represents the transition period between the adult and the traditional geriatric period. Although the exact time of each stage could be argued, everyone would agree that smaller breeds live longer than that the giant breeds and each life stage would have a corresponding chronological difference. Human / Pet Age analogy charts reinforce this concept and are excellent client education and marketing tools. The chart also emphasizes the idea of comparable “time compression” in pets as it relates to disease progression, wellness testing intervals, and chronic drug monitoring. (See the age analogy chart on the last page).

Aging is a complex subject influenced by numerous interrelated causative factors. The current causes / theories of aging can be broken down into four general categories; 1. Accumulations of toxic substances or compounds within cells; 2. Cumulative cell damage from ionizing irradiation, oxygen derived free-radical-mediated damage, and/or environmental pollutants; 3. Immune mediated or immune compromised processes; and 4. Genetically preprogrammed cell death initiated by a portion of the gene responsible for the cell’s lifespan. Every cell is genetically preprogrammed for a specified number of divisions and will die at a predetermined point in time dictated by a specific cellular gene.

"Old age" is not a disease but represents the effects of time upon the physical, mental, and internal organs, but bears no absolute relationship to actual chronological age. Unfortunately each internal organ system will age at a different rate. While it is appropriate to use age as a benchmark of organ decline, any assessment of a patient health should be based on a complete health screening of mental and organ function because the organs degenerate at different rates.

The generalized changes associated with aging changes include dryness of all tissues, progressive degeneration of organ function, tissue hypoxia, cellular membrane alterations, decreased enzyme systems, decreased immune competence, and definite personality alterations. These progressive changes represent the complex interactions aging has on bodily functions however considerable individual variation exists even with litter mates. Three reference books one should consider owning when dealing with the older patients include Geriatrics and Gerontology of the Dog and Cat by Dr. Hoskins, the Veterinary Clinics of North America, 2003 Geriatrics edition, and Veterinary Drug Handbook by Plumb.

Gradual changes in the overall body condition are not easily detected by the owner. Regular weight monitoring combined with body condition scoring allows the veterinarian to better assess minor or earlier changes in overall weight status especially in multi-doctor practices. Based on ideal weight, older patients can be placed in three classifications; pets within the normal weight range, animals that are too thin, and
patients that are overweight or obese. Decreases in activity and basal metabolic rates without a corresponding decrease in caloric intake produces the overweight pet.

Several studies have shown that geriatric pets are often too thin. The issue is whether this low body condition score is the result of some underlying disease state or just “normal” aging seen in dogs, cats and humans. Common age-related causes of insidious weight loss include metabolic diseases, cardiac failure, cancer, and malabsorption. Decreases in muscle mass and partial age-related inappetence can be “normal”. Despite the category, a decreased or picky appetite needs to be investigated. Pathologic etiologies associated with a decreased appetite include dental disease, metabolic dysfunctions, a gastrointestinal disorder or cancer. In addition to the normal loss of olfactory neurons, normal loss of taste buds, masticatory muscle atrophy, a lack of sufficient saliva to swallowing dry food, or “senility” can also contribute to deceased appetite in the “healthy” older pet. Feline food aversion is often associated with environmental changes, palatability issues, nausea from IBD, chronic pain, stress, and/or medication administration with food.

Following a thorough physical examination and medical evaluation attempting to rule out cancer cachexia or various metabolic disease, this author advocates one or more of the following non-medical options for encouraging the pet to eat; more frequent meals; hand feeding the pet; adding water to the dry food; feeding canned food; warming the food; adding commercial flavor enhancers; and mild exercise prior to mealtime. Bowls used for feeding elderly cats should be wide and shallow so that the sides do not touch the cat’s whiskers. Dietary fat helps make foods more palatable, an important consideration in older animals that may have a diminished sense of smell or taste. Benzodiazepine derivatives are commonly used to stimulate appetite and are effective in up to 50% of patients. Unfortunately, there uses have been associated with drug related hepatopathies especially in cats. Diazepam may be used in dogs or cats and is most effective when administered intravenously. Fresh, palatable food should be offered immediately following administration (0.2-0.5 mg/kg administered intravenously, a maximum dose of 5 mg per patient). Feeding usually starts within one minute and may continue for up to 20 minutes. Longer lasting oral benzodiazepine derivatives such as Oxazepam produce results within 20 minutes following oral dosing (2.5 mg per cat administered orally). Cyproheptadine (Periactin) 2-6 mg/dog PO BID and 1-2 mg/cat PO BID has been used as an effective appetite stimulant but may take several days before the desired effect. Alpha-2a Interferon (Plumb) has also been used effectively for long term appetite management as have anabolic steroids. In cats and humans weekly B complex injections has also been shown to be effective in increasing appetite in some cases.

Effective thermoregulation is decreased in the aging dog. Their decreased ability to pant and decreased cardiac output make them more prone to heat stroke. Age-related cold intolerance is often attributed to less Sub Q fat, decreased basal metabolic rate, decreased cardiac output and decreases in peripheral vaso-constriction. Cold intolerance can also be a sign of hypothyroidism. The resulting response to cool ambient temperatures may manifest as behavioral problems including reclusiveness, trembling, and reluctance to go outside for eliminations, and/or sleep cycle disturbances. In addition to suggested environmental changes and thyroid evaluation, warm bedding and outdoor garments may help alleviate some of the abnormal behavior.

The integument changes are the most obvious to the owner. The skin and hair undergoes some degree of pigment loss (graying), hyperkeratosis, follicular atrophy, and decreased sebum production. The nails become longer and more brittle. The dryness of the skin and coat can be helped with increased grooming, less bathing, post-bathing conditioners, topical emollient (oils) or humectants (moisture) sprays, and essential fatty acid nutritional supplements.
Progressive loss of muscle mass is a normal finding in the older pet especially the geriatric patient. This change is related to a combination of inactivity combined with a decrease in muscle cell numbers due to fibrosis, atrophies of existing muscle cells, and decrease sensitivity to ATP. Additional muscle atrophy can also be attributed to decreased dietary protein when using a “low protein” prescription diets. Unfortunately this muscle wasting may be responsible for or exaggerate hind limb weakness. The resulting difficulty climbing stairs or jumping can be confused with the symptoms of arthritis and disuse atrophy. In dogs systemic or oral anabolic steroids combined with increased dietary protein and a sensible exercise program has been advocated in management of the generalized muscle weakness and muscle wasting conditions. The use of creatine / phosphocreatine supplements is also indicated.

Decreases in hearing and vision are common age-related changes that may have associated changes in behavior patterns. Unfortunately these sensory dysfunctions may not be recognized as such and are often mis-diagnosed or mis-interpreted as senility or Cognitive Dysfunction Syndrome as the symptoms are often similar. Hearing loss associated with decreased sound wave conduction from the external ear to the cochlea (conduction deafness) can be helped with amplification. However specific loss of nerve function (neurogenic deafness) is the most common cause of deafness in older dogs. The use of a high frequency dog whistle is a temporary solution until the higher frequency recognition is finally lost. The normal aging changes in the lens called nucleus or lenticular sclerosis should be always differentiated from opacities of the lens/capsule called cataracts. All cataracts should be staged (immature, mature or hyper mature) to better assess the cataracts impact on vision and the appropriateness of lens extraction. Neither nuclear sclerosis nor an immature cataract should affect a pet’s vision.

Progressive functional renal nephron loss may impact drug and anesthetic selections. Advancing progressive renal nephron loss can be easily determined by urine specific gravity, BUN/ Creatinine, and perhaps persistent micro-albuminuria. Once more than 66% of renal function is lost, the patient loses the ability to concentrate their urine. The bun/creatinine start to rise after more than 75% real function is lost. With the remarkable adaptation of nephron in slowly progressive renal disease, the patient can survive and a very small percentage of kidney tissue. This baseline testing is also helpful in trending, the concept of repeated monitoring / charting of a biological parameter that is helpful in “predicting” the patient’s future health status.

Since significant renal proteinuria (persistent microabuminuria or overt proteinuria) can be the first laboratory finding with chronic progressive real disease. A complete urinalysis with specific gravity, dip Stix, and sediment examination is an essential part of a senior care program. Microabuminuria using ELISA based technology is a very sensitive test but not very specific. Conversely a urine protein/creatinine ratio while not as sensitive a test, is much more specific. Both Heska and IDEXX have proteinuria algorithms charts available that include step by step workup and management options.

Liver function maybe altered by fatty infiltration and/or hepatocyte damage as indicated by elevated alkaline phosphates. Mild elevations (2-3X high normal) in ALP could be considered normal in the older dog. The implications are at what point should the “healthy” patient with an elevated ALP be worked up or just periodically monitored (trended)?

Decreases in both cellular and humeral immunity are associated with the aging process and explain the increase prevalence of tumor growth and infection rates in the senior and geriatric patients. That said, what should the vaccination schedule be for the older pets…. none, more, less or the same?

The four most common causes of death in older dogs, based on a study funded by the Morris Animal Foundation for Animals, re cancer, cardiovascular disease, renal failure, with epilepsy & hepatic diseases tied for 4th. In cats, the top four fatal diseases in order
include cancer, renal failure, cardiovascular disease, and diabetes mellitus. These facts are important when educating your clients and staff on the early warning signs of serious life shortening common diseases in the elderly pet and in determining what tests should be included in a geriatric health screening. Considerable research in the area of interrupting the aging process is ongoing. Technologies such as gene splicing may someday allow each of us to live longer. Until then, exercise, senior diets and antioxidant supplements are generally recognized as valuable anti-aging strategies.

### Human/Pet Age Analogy

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*Age Analogy Chart: W. Fortney, R. Goldston*
Managing Common Behavioral Changes in Older Pets

Definite personality changes and behavioral problems can be extremely challenging to the practitioner and extremely frustrating to the client. Some problems are mild and acceptable, while others are major concerns initiating euthanasia discussions.

General behavioral changes are elderly patient’s desire more attention, are more jealous, are more irritable, are less mentally alert, and have altered sleep cycles. 60% of pets sleep in the owner’s bed or in the bedroom according to an AAHA sponsored study. Altered sleep patterns are common geriatric "behavioral" dilemmas especially in those situations where both the patient and owner are affected. The owners report one or more of the following complaints; pacing at night, periods of panting, awaking the owners to go outside for no obvious reason, inability to get comfortable or constantly "fluffing" their bed. Various causes include an underling painful condition such as osteoarthritis, spondylosis or a chronic IVD; sleeping on a hard surface; an altered biological clock where they sleep all day then can't get to sleep at night; cold from poor circulation or a basal metabolic rate or a poor thermoregulation; or a phobia of the dark. This complaint can also be linked with Canine Cognitive Disorder. The author would advocate the following options; a warm soft bed, a night light, a radio playing softly, a brief walk before bedtime, hydroxyzine 1-3 mg/kg prior to bedtime, melatonin 1-3 mg, and/or a 2 week trial of a short term pain management program.

Cognitive Dysfunction Syndrome

Many of these common “old dog or old cat” behaviors are often grouped into a syndrome called Cognitive Dysfunction Syndrome (CDS). CDS is a progressive disease syndrome of older dogs and cats associated with some brain pathology that results in commonly recognized group of behavioral changes. The clinical signs in older patients are related to impaired mental function commonly referred to as "senility, dementia, or doggy Alzheimer’s disease". Impairments in memory, learning, perception and/or awareness are common. CDS is a progressive disease of the brain in older dogs and cats associated with changes in behavior…not a behavioral disease!

The pathogenesis of CDS is complex and is associated with four categories of pathology each resulting in altered behavior. Initially any one of the following agents will initiate varying degrees of neuronal dysfunction that can be reversible if treated early. Except with alterations in neurotransmitters levels, with time the damaged neurons will eventually die leading to permanent abnormal behavior. This explains why some treatments may be not be beneficial at all or become less effective with time.

One cause of CDS is the accumulation of metabolic waste products in the neurons. The primary offender in humans, dogs and cats is Beta amyloid that exhibits characteristic plaques when viewed on histopathology sections. Initially any behavioral changes associated with Beta amyloid can be attributed to neuronal dysfunction but eventually the affected cells will die. Another category of causative agent is hypoxia. Decreased oxygen to neurons will result in neuronal dysfunction and eventually neuron death. The hypoxia can result from decreased cardiac output, chronic pulmonary disease or vascular compromise from arterial constriction or sclerosis. Another area of current CDS research focuses on the age-related decreases in various CNS neuro-
transmitter levels i.e. ACH, GABA, dopamine, nor-epinephrine, or serotonin. The confirmatory diagnosis of a specific neurotransmitter deficiency is based on the patient’s behavioral response to any replacement therapy. The last category of a cause for CDS involves the deleterious affect of oxygen derived free radical on brain neuronal function. There has been considerable research into the effects of positive effects of anti-oxidants on brain function in patients with CDS. Antioxidant packages are becoming common place in most quality senior diets and are currently being investigated as possible preventatives for many age-related diseases.

The common clinical signs of Canine Cognitive Dysfunction fall into four distinct categories found in the acronym DISH.

D= disorientation / confusion
I = decreased interactions with family or housemates
S= Sleep cycle disturbances (sleeping more or at the wrong times)
H= house soiling

It is important to note that most cases of CDS have symptoms in only one or two of the four categories. In the feline, the common clinical signs of CDS include one or more of the following; aggression, inappropriate elimination, increased vocalization, sleep cycle disturbances, excessive grooming, and/or disorientation/ confusion.

Since certain metabolic diseases, endocrinopathy, sensory dysfunction, or brain tumors can also present with similar if not identical symptoms, the patient should undergo a complete work up prior to initiation of any therapy. The minimum data base for CCD should include a behavioral history, a complete physical examination, neurological examination, CBC, chemistry profile, UA, and thyroid evaluation. CSF tap, and brain imaging is also recommended to run out other causes of organic brain disease.

The therapy depends on the underlying pathological cause. One of the management strategies of CDS involves increasing the oxygen to the brain by increasing cerebral blood flow. A product approved for use in Europe called propentofylline (Vivitonin) increases the blood flow to the brain and therefore the oxygen supply without increasing the brain’s glucose demand. Another European product Nicergolin (Fitergol) is a competitive antagonist to norepinephrin at the alpha-adrenergic receptors. This drug effectively increases the cerebral blood flow via decreasing the vasoconstriction associated with excessive norepinephrin production.

Numerous other products are advocated as effective neuroprotectants agents (those nutritional supplements that protect the neurons from the effects of oxygen derived free radicals) including nutritional supplements vitamins E, C and zinc; other antioxidant compounds; anabolic steroids; and selegilline. Specific agents such as selegilline, Nicergolin, Cholodin® & Dyna-load® alter specific neurotransmitter levels often associated with CDS.

Until the advent of selegilline, the management of CDS was extremely frustrating. Dr. Sheffy advocated mild exercise in older dogs in1985. He reported that mild forced exercise was conducive to increased alertness, plus improved the dog’s spirit and enthusiasm in response to human socialization. Dr. Mosier reported positive clinical cognitive benefits in dogs using oxygen therapy on a weekly schedule. In addition The lay press is filled with testimonials of the benefits of nutritional supplements (vitamins, antioxidants, and Cholodin) on brain function.
Selegilline also known as L deprenyl is a specific B monoamine oxidase inhibitor (MOA-B). The animal form of Selegilline is marketed as Anipryl by Pfizer Animal Health. The generic form of the compound is D-deprenyl. In some dogs and cats with signs of CDS, prolonged Selegilline therapy resulted in significant clinical improvements in greeting behavior, client / patient interactions, inappropriate urination and defecation, activity levels, confusion/disorientation, and/or sleep cycle disturbances. These positive behavioral effects are usually attributed to restoration of depleted Dopamine levels via MOA activity and/or the facilitation of Dopaminergic transmission by some other related mechanism. However, another suggested mechanism of action includes the CNS stimulatory action of two of the metabolites, L-amphetamine and L-methamphetamine. Selegilline has some neuro-protecting properties against amyloid deposition and oxygen derived free radicals.

Regardless of the exact mechanism(s) of action, Selegilline can be beneficial in managing certain age-related behavioral problems in dogs. The initial dosage of Anipryl for use in Cognitive Dysfunction Syndrome is 1.0 mg/kg PO SID each morning for at least a 60-day trial. Adverse reactions including restlessness, vomiting, diarrhea, diminished hearing, pruritis, shivering/shaking/trembling and disorientation can occur at the lower dosage but are much more commonly reported at the 2.0 mg/kg PO SID range or when given at bedtime. The contraindications of Anipryl therapy include concurrent use with meperidine (and other Apodes), tramadol, or use in combination with other MOA inhibitors. Expense will become a major issue with the life long administration.

Recently the use of dietary “anti-oxidants” and essential fatty acids (hills b/d) has been shown to be effective in the managing some clinical aspects of CDS especially when used in combination with environmental enrichment activities.

**Osteoarthritis: Management Strategies for the Arthritis Pet**

Osteoarthritis (OA) is a chronic, non-infectious, progressive disorder of any synovial joint. OA is characterized by deterioration of the articular cartilage, synovitis, with secondary bony changes. Osteoarthritis is classified as being primary or secondary in nature. **Primary** arthritis (age-related) results from abnormalities within the cartilage (abnormal quality) and is considered an intrinsic problem with the cartilage homeostasis associated with aging. In this form of OA, normal joint forces impact abnormal cartilage. The affected cartilage has limited ability to regenerate and maintain itself in the face of the cumulative effects of ongoing trauma and/or inflammation. Primary osteoarthritis has a slow progressive course, generally involves one or more joints, and is most common in geriatric patients.

**Secondary** arthritis is a more common cause of clinical lameness in the younger dog. It is the consequence of some external event or force affecting the articular cartilage adversely. In this form there are abnormal forces affecting normal cartilage. Examples would include overt trauma, joint incongruity (instability), and joint malalignment. Secondary arthritis can be seen in any age animal and usually involves a single joint. In order to prevent, halt or minimize the degeneration process, the underlying joint incongruity must be eliminated.

The pathophysiology of primary arthritis involves a series of steps that eventually becomes a progressive self-perpetuating process. The mediators of the inflammation, pain, and joint destruction include prostaglandins, cytokines, leukotriens,
The clinical response of the pharmacological agents used in treating OA reflects the specific effect on each of these specific inflammatory mediators or pathways. Articular cartilage is a very complex and dynamic structure. The cartilage matrix is composed of proteoglycan macromolecules (hyaluronic acid and GAGs), type 1 collagen, and 80% water. The matrix is synthesized and maintained primarily by the chondrocytes. In primary arthritis, there is a shift in the cartilage homeostasis towards catabolism. Initially there is decreased production of proteoglycan by the chondrocytes, a loss of chondroitin sulfates and water from the matrix. The resultant articular cartilage is less elastic with less shock absorption. Any minor trauma produces cartilage fissures and chondrocytes damage. Surface cracks produce "flaking" exposing the collagen fibers to wear and tear. Some cracks extend into the deeper layers and bone produce fibrillation. Degrading enzymes are released which further break down cartilage matrix and collagen. These enzymes include serines, collagenase, cathepsins, metalloproteinase’s, hyaluronidases, stromelysins, plus the cartilage breakdown products, initiate a mild synovitis. The resulting inflamed synovial membrane releases primary inflammatory mediators IL 1, IL 6, tissue necrosis factor, proteases, and prostaglandins that further increasing cartilage destruction and inhibiting new matrix production. The further decreased resiliency of the cartilage results in sclerosis of the subchondral bone and osteophyte development in the periarticular margins in advanced cases.

The clinical signs of osteoarthritis are similar regardless of whether the disorder is primary or secondary. The onset is often insidious but progressive. Early in the course of the disease, the animal may sporadically be reluctant to perform previous tasks or activities, i.e., jumping into the car. In the next stage, a lameness or stiffness occurs following periods of excess activity or overexertion. These signs often disappear after several days of rest. As the degeneration progresses, the stiffness and lameness may be most pronounced following periods of rest. The pet typically "warms out" of the signs with activity. Any cold or damp weather will increase the severity and duration of the symptoms. Continuous stiffness, lameness and chronic pain typify the final stage producing an irritable, reclusive and restless pet. In a recent study, 90% cats > 12 years had radiographic evidence of osteoarthritis (Hardie, E., JAVMA 2002). The most common location in cats was the spine followed by the elbow and the hip. Common feline symptoms include grooming difficulties, inappropriate eliminations, less jumping, aggressive when handled, and lameness.

There is no cure for arthritis, only control. The goals in the treatment should be to alleviate patient discomfort, minimize further degenerative changes, and to restore the affected joints to as near normal and pain-free as function possible. The management of arthritis involves the following strategies and should be tailored to best meet the patient and clients needs; 1. Client education on the progressive nature of the disease is critical; 2. Set realistic outcome goals (expectations); 3. Adequate rest periods; 4. Sensible exercise program; 5. Weight assessment and reduction if necessary; 6. Analgesics and anti-inflammatory therapy for rapid results; and/or 6. Chondroprotective agents; 7. Anti-oxidant nutritional therapy (dietary); 8. Complementary therapies, i.e. message therapy, physical therapy, acupuncture, chiropractic, etc.

When choosing between NSAIDs and Chondroprotective agents we initially select the NSAIDS because NSAIDS have a more consistent, predictable and faster
response our client expects. In addition NSAIDS have more research backing the products and generally the product quality control is much better. Unfortunately there are potentially serious side effects that must be communicated and assessed. Conversely the chondroprotective are less reliable in there effect, slower to work, but are much less toxic. NSAIDS have a variable combination of anti-inflammatory, antipyretic, and analgesic effects. The variation in clinical response of the each pharmacological NSAID agent used in treating arthritis reflects the specific effect on various mediators of the inflammation via selective inhibition of pyrogens, COX1, COX2, and pain mediators. While all NSAIDS have some common toxicity, each has unique pharmacological and dosing advantages and should never be considered identical or interchangeable. In fact when switching NSAIDs, I always recommend a “WASH OUT” period of at least 7 days. Managing feline osteoarthritis involves the judicious use of chondroprotective agents, steroids and/or a non-approved long term NSAIDs such as metacam.

NSAIDS toxicity ranks at the very top of the geriatric adverse drug reaction list. Primarily this is because of their wide spread usage and potential life threatening side effects. Adverse Drug Events (toxicity) are usually associated with over dosages, misuse by owners, combined therapy with steroids, failure to “wash-out” a NSAID before switching to another one, concomitant disease, and the lack of pharmacovigilance on the veterinarians part. If necessary, during the washout period, tramadol at 2-5mg/kg q12h can be used in dogs. It stimulates the opiate, adrenergic, and serotonin receptors. This pain reliever is compatible with all the COX inhibiting NSAIDS drugs but NOT with serotonin reuptake inhibitors, tricyclic antidepressants, or monoamine oxidase inhibitors such as Anipryl. The induced enzyme Cyclooxygenase 1 (COX1) is responsible for maintaining the gastrointestinal mucosa health, renal and hepatic blood flow. Nonselective inhibition of COX1 can result in gastrointestinal damage, decreased renal blood flow and/or hepatopathies. The resultant toxicity(s) includes gastro-duodenal hemorrhage, gastro-duodenal ulcerations, bleeding dyscrasias, and analgesic nephropathy/hepatopathies. Most toxicity associated with the administration of NSAIDS results from Prostaglandin E inhibition by blocking the COX1. This is often dose-related adverse drug event. Prostaglandin E is gastric cytoprotective by inhibiting gastric acid secretion, maintaining mucosa blood flow, stimulating epithelial cell renewal/restitution, increasing gastric bicarbonate and mucus secretion. Cyto-protective strategies are aimed at eliminating or reducing the possible side effects with prolonged NSAID administration. Using more selective COX2 inhibitors (COX1 sparing), accurate dosing, administering NSAIDs with a meal, the use of concomitant antacids, the use of H2 receptor blockers can all aid in prevent most of the untoward side effects of nonsteroidal anti-inflammatory medication. Misoprostal, a synthetic PGE1 replacement, was first advocated to combat the side effects of NSAIDS. The dosage is 2-5 ug/kg PO TID and is contraindicated in pregnancy. Glucocorticoids are very effective at decreasing the inflammation associated with arthritis however they should only be used in those advanced cases that are unresponsive to NSAIDS since steroids hasten the cartilage degeneration via chondromalacia. PU/PD, GI bleeding and ulceration are common adverse reactions which are amplified when used concomitantly with NSAIDs.

Chondroprotective Agents: Chondroprotective agents are a group of compounds that directly benefit and/or support the cartilage matrix. As a group, these compounds
act to decrease the breakdown of articular cartilage and/or provide the building blocks to up regulate cartilage synthesis. Some actually decrease inflammation and increase beneficial synovial fluid secretion. Therefore chondroprotective agents may not only relieve some symptoms but they may also decrease the degenerative process thereby may actually “reverse” the degeneration. Although chondroprotective agents are not considered analgesics, recent studies indicate they may have a direct anti-inflammatory effect on the synovial membrane. Principle components of chondroprotective agents include one or more of the following; PSGAG’s, glucosamine, and chondroitin sulfate. Unfortunately clinical failures with chondroprotective therapies are common and could be attributed to minimal cartilage remaining (bone to bone), unresponsive inflammation, time /dose dependant responses, and the lack of analgesia. The low daily dosage of chondroprotective in some diets, there efficacy in treating arthritis is questionable. While more expensive, the injectable PSGAG’s give a faster and longer lasting response than the oral forms. Although more expensive, the combination of NSAIDs plus chondroprotective is attractive to many owners, especially if the chondroprotective agent decreases the NSAID dosage and frequency of usage.

Of recent interest is the use of nutrition as part of an overall arthritis management strategy. Dietary glucosamine and/or dietary n-3 fatty acids, especially DHA and EPA, can reduce production of the pro-inflammatory factors and result in clinical improvement is some arthritic patients (Purina L, Hills j/d, and IAMS).

A wide variety of other drugs have been used in the management of DJD in small animals including Vitamin C, cryotherapy (gold injections), free radical scavengers orgotein, MSN, hyaluronate sodium, and copper collars. In most cases the exact mechanisms of action is poorly understood and little scientific data exists to determine any efficacy.

Complementary therapies continue to gain favor among the veterinary community and pet owning public in managing arthritis. At Kansas State University we have a faculty member Board Certified in acupuncture, chiropractic, and holistic medicine that I common refer patients to for complimentary therapy when western medicine has failed, is too toxic, or the owner wants to take another approach.

**Common Dermatological Problems in Older Pets**

Senior patients are to be more prone to infections, food allergies, endocrine-related skin changes, pressure point calluses, adult onset demodicosis, and cutaneous neoplasia. Sebaceous gland tumors “senile warts” account for up to 35% of all canine skin tumors but are rare in cats. They can be single but are usually multiple and occasionally ulcerate. Rarely they do they become malignant. Clinical management options include surgical excision, cryotherapy, electro surgery, laser surgery and topical chemotherapy agents including 5 FU. To the owner, integument changes are the most obvious sign of aging. Aging of the skin and adnexa is a complex subject influenced by a combination of interrelated causative factors including wear and tear, cumulative cell damage from ionizing irradiation, and genetically preprogrammed cell changes.

The decrease in the number of active hairs and the follicular atrophy results in a thinner hair coat. Graying of the hairs especially on the muzzle results from the decrease in melanocyte replication within the hair root. The nails become longer, malformed and more brittle. There is a progressive hyperkeratosis of the skin and hair
follicles. This is especially obvious on the patient’s foot pads and nose. The application of keratolytic agents or softeners is usually helpful in managing this problem.

The altered sebum production and cystic dilation of the apocrine glands results in a dry, lack luster hair coat and dry skin. The dryness of the skin and coat can be helped with increased grooming, less bathing, post bathing conditioners, topical emollient (oils) and/or humectants (moisture) sprays, and essential fatty acid nutritional supplements.

**Infectious Diseases**

Because of decreases in cellular immunity, except for fleas and ticks, infectious diseases of the skin continue to be the most common skin disease of older pets. Recurrent bacterial folliculitis, pyoderma, or otitis externa can be problematic in the older pet. The use of antiseptic shampoos, antiseptic rinses, and long term / low dose antibacterial therapy is often required to manage the problem. In older patients, Demodicosis is not usually considered in the differential diagnosis of a skin problem, but adult-onset demodex does occur and is often missed because of omitting a routine skin scraping in the initial work up. Remember demodex is a great imposer and can look like a pyoderma, an allergy, a dermatophytes infection, and even an auto-immune skin disease. The decreased cell mediated immunity may also suppress the usual inflammatory changes seen. With generalize adult-onset demodicosis cases, some underlying immunosuppressive systemic disease such as hyperadrenalcorticism, hypothyroidism, renal failure, diabetes mellitus, or neoplasia should be ruled out.

**Adverse Skin Reactions to Food, Food allergy and food hypersensitivity** are terms used to describe the immunological mediated disease reaction induced by food ingestion. Conversely **Food intolerance** is the term used for an abnormal physiological response of foods or food additives that does not have an immunological basis. In a clinical setting, food hypersensitivity and food intolerance are rarely differentiated and refer to a non-seasonal pruritis skin disorder associated with ingestion of a substance usually found in the diet.

Numerous dietary allergens exist including beef, pork, chicken, cows’ milk, horsemeat, chicken egg, wheat, oats, fish, corn, and soy protein. Although any dietary protein is capable of causing pruritis, beef, dairy products, wheat and soy proteins are the most common cause of adverse cutaneous reactions. Dogs and cats can react to one or more dietary allergens.

Non-seasonal pruritis is the most common clinical sign in dogs with food-related dermatitis. In dogs the age of onset of food-related dermatitis is variable. Food-related dermatitis should be considered a differential for pruritis in any age of dog and especially senior dogs with no previous “allergic” history. Unfortunately, the onset of food-related dermatitis is not usually associated with a recent change in diet. In one study 68% of dogs had been fed the offending diet for at least two years before onset of clinical signs. Dogs with food-related dermatitis generally will not respond as well to conventional anti-inflammatory doses of glucocorticoids, antihistamine therapy, or essential fatty acid supplements as dogs with other allergic diseases. Therefore the lack of response to these medications in a dog with mild to moderate pruritis would make Atopic dermatitis less likely than food-related dermatitis.
The primary lesion of food-related hypersensitivities is erythema. A papular eruption, urticaria, and angioedema have also been reported but are rare. The secondary lesions associated with pruritis include alopecia, excoriations, hyperpigmentation, and lichenification. The distribution is usually similar to that seen in Atopic dogs with facial, ears (pinnae and external ear canals), feet, axilla, and ventral involvement. Some patients with food related dermatitis will present with clinical tail head lesions which are identical to those associated with flea allergy dermatitis.

In cats, food-related dermatitis is a major cause of pruritis and self-mutilation in young to middle age cats. As in dogs, non-seasonal pruritis is the most common clinical sign in cats. The pruritis tends to be localized to the head and neck. Only half of the cases are considered “steroid responsive”. The often intense pruritis and self-trauma results in erythema, alopecia, erosions, ulcerations, and crusty lesions. GI signs are present in 10-30% of confirmed cases. Some of the cases of feline miliary dermatitis, eosinophilic granuloma complex, and a symmetrical alopecia caused by excessive grooming, have also been associated with food hypersensitivity.

In dogs and cats the histopathology is consistent with a non-specific hypersensitivity plus often demonstrates the pathology of a secondary pyoderma or Malassesia infection. Since intradermal skin testing and serologic testing are considered marginally diagnostic, the best way to confirm a diagnosis of food-related dermatitis is evaluating the clinical response to a single novel protein test diet or a diet containing protein hydrolysates, then a subsequent challenge with the patient’s original diet. A single novel protein ingredient dietary trial involves feeding a diet that contains a single protein and a single carbohydrate source based on the dietary history that has not been previously fed to the patient. The single protein limited ingredient diet should be fed for at least six to eight weeks before a decision is made regarding efficacy. If no response is seen in 6 to 8 weeks, then it is unlikely that food is playing a major role in the patient’s dermatitis and the diet is discontinued. However, if even a partial response is noted then the diet should be continued for an additional four weeks since some cats and dogs may take as long as 10 weeks for a maximum beneficial response. A follow up challenge with the patient’s original diet is always recommended to be sure that the improvement was not coincidental with other factors. A food allergic dog or cat will typically break with pruritis and dermatitis within seven days of the challenge.

Dietary trials with protein hydrolysates are gaining favor with many veterinary dermatologists. This process of protein hydroxylation involves cleavage of protein peptide bonds into smaller units. The theory is that lower molecular weight “proteins” will not elicit an immunologically mediated response. Avoidance is the most important aspect of managing food allergies.

Superficial Necrolytic Dermatitis (SND) / Hepatocutaneous Syndrome (HS)
Superficial necrolytic dermatitis (SND), hepatocutaneous syndrome (HS), necrolytic migratory erythema (NME), metabolic epidermal necrosis (MEN), diabetic dermatopathy are terms used to describe a skin disease with a multifactorial underlying etiology. This syndrome has been associated with diabetes mellitus, glucagon secreting tumors of the pancreas. However the syndrome is most often seen with a variety of canine and feline hepatopathies using the term hepatocutaneous syndrome (HS) for those conditions.
Superficial necrolytic dermatitis is generally seen in middle-aged to older dogs with the average being 10 years old. The history of skin lesions is weeks to months with a typical waxing and waning but slowly worsening course. Skin lesions are usually noted to first affect the feet and or foot pads. The lesions description includes hyperkeratosis, interdigital erythema, some erosion with crusting and occasionally intact bulla will be observed. The lesion pattern progresses to a more generalized pattern include the face, hocks, genitals, interiginous zones, lower abdomen and/or groin area. With the feet and muco-cutaneous junctions on the face the most common sites leading to the presumptive diagnosis of immune mediated skin disease.

Skin lesions may become mildly pruritis and usually painful. Systemic signs of illness including lethargy, anorexia, and weight loss are reported in less that 20% of the cases.

Laboratory changes in dogs with hepatocutaneous syndrome (HS) include increased liver enzymes especially serum alkaline phosphatase. Unfortunately many “normal” older dogs have elevated liver enzymes.

Hepatic ultrasonography is often helpful diagnostically. With the hepatocutaneous syndrome (HS) form of superficial necrolytic dermatitis the hepatic ultrasound generally shows a characteristic "Swiss cheese" or “honeycomb” pattern that some feel is pathognomonic for the syndrome. However in dogs with idiopathic hepatocellular collapse as the cause for their liver disease, the liver has a nodular appearance with variable sized hypoechoic regions of regeneration surrounded by an echogenic borders. There is a lack of fibrosis and reduced liver size which is seen in cirrhosis. In cases involving the glucagon secreting tumors of the pancreas, the ultrasound findings are less consistent. The pancreas and liver maybe normal or a mass may be identified in one or both organs. If a glucagonoma is suspected and ultrasonography is normal, a glucagon assay should be performed. Plasma glucagon levels are characteristically 5-10 times above the normal range.

The differential diagnosis for the cutaneous lesions is extensive however they typically have an immune mediated disease “look” and distribution pattern. However the rule outs for the hyperkeratotic lesions on the feet and face would also include zinc responsive dermatosis and generic dog food dermatosis.

Initial diagnostics tests should include skin surface cytology, cytology of vesicular fluid if present. A complete blood count, a biochemical profile are indicated. Multiple skin biopsies from several affected areas are essential. With H&E staining, the epidermal parakeratotic hyperkeratosis is red, the epidermal edema as white and hyperplastic basal layer is blue. This characteristic “red, white & blue” histopathologic change is pathognomonic for superficial necrolytic dermatitis. However in older lesions, this typical pattern may not be present, which may result in misdiagnosis or failure to diagnose the condition.

Numerous treatments have been used in dogs with hepatocutaneous syndrome. A 10% crystalline amino acid solution (Aminosyn, Abbott Labs) may be administered intravenously at either 25 ml/g body weight or 500 mls total volume per dog slowly over six to eight hours every 7 -10 days. The response has been variable. If minimal to no response is noted, the infusions are repeated every 7-10 days for four treatments. If an individual dog does not respond by this time, they will generally not respond. For those that respond, the amino acid infusion is repeated with each exacerbation of the skin lesions. Dogs may go several months between amino acid infusions, but some
dogs will require monthly infusions to maintain remission. As the disease progresses, the need for amino acid infusions will more than likely increase. This AA solution is hypertonic and must be administered via a central vein such as the jugular vein to reduce the chance of thrombophlebitis.

An oral amino acid supplementation (Promod, Ross Laboratories) has been noted to be of some benefit, perhaps because of the specific amino acid profile provided and ease of use. Some dogs seem to benefit from daily supplementation with three to six egg yolks for specific amino acids. The use of human body builder muscle protein supplements has been advocated by some authors. Regardless of the options, oral nutritional support with a high-quality protein diet such as Hill’s Prescription Diet a/d or Eukanuba Recovery Formula is highly recommended if the dog is eating or via an enteral route. Zinc supplements (2 mg/kg daily of zinc methionine) and essential fatty acid supplementation (high in omega 3 fatty acids) should also be used.

Temporary improvement of skin lesions and hyperkeratotic footpads may be seen with topical and systemic glucocorticoids (prednisone starting at 1 mg/kg daily). However, effects are usually transient and will eventually become refractory to these therapeutic dosages plus steroids complicate the hyperglycemia.

The prognosis with Superficial Necrolytic Dermatitis is grave. The mean survival time in one study was 1.6 months. The prognosis for dogs with idiopathic hepatocellular collapse is generally poor. However, there have been reported cases which have been medically managed for two to three years after diagnosis of the disease.

**IMPLEMENTING A SENIOR CARE PROGRAM: A PRO-ACTIVE APPROACH TO SENIOR WELLNESS**

A Senior Care Program provides a proactive comprehensive health care platform that addresses the older patient’s special needs in a marketable package. This specialized medical service is based on two premises; first there are fundamental differences in the specific diseases, behavior problems, and the nutritional needs of the older pet; secondly that prevention, early detection, and timely intervention of medical problems can have a significant impact on the longevity and quality of life of an older pet. Recently the AAHA came out with Senior Care guidelines. A copy can be found on their website; aahanet.org

Senior Care changes the way veterinarians traditionally approach the senior pet. Senior Care is a more inclusive health care program starting at seven years of age and advocating twice-yearly evaluations. However, the major benefit of Senior Care is the Industry support form companies like IDEXX, IAMS and recently Fort Dodge. IDEXX ‘s continued commitment to the program provides your practice with age related in house and referral laboratory diagnostics plus all the marketing and implementation tools necessary to make Senior Care successful in your practice. Tools for client education, health data gathering and health reporting tools, patient diagnostic and management charts plus program implementation tips are currently available.

Why build a Senior Care program in your practice? By advocating more comprehensive histories, performing more complete physical examinations, and recommending more diagnostic testing of older pets, you are providing higher quality veterinary medicine for your senior patients. A great deal of professional satisfaction
for you and your staff comes from helping those long established senior patients live longer healthier lives. Plus managing most age related disease in the early phases is far more rewarding than the “end stage”. Senior Care can be a major profit center for your practice. With over 39% of the pet population considered seniors, tremendous financial opportunities exist.

While the veterinary profession has been very successful at providing comprehensive health care programs for the puppies and kittens, there are almost 2 1/4 times as many senior pets as puppies and kittens and dogs are puppies for one year but are seniors for four to ten years. By taking a more detailed history on the senior pet, performing a more complete physical examinations, and submitting more diagnostic tests, not only are you increasing the standard of care you provide your senior patients, but approximately 40% of those patients will now require additional diagnostic testing, medication or therapy. Ultimately a properly implemented Senior Care program can represent 35% or more of your gross annual income.

A Senior Care program implies both a preventative and comprehensive therapeutic approaches to management of acute and chronic conditions in aging dogs and cats. The program emphasizes client education, prevention, early detection, and timely medical intervention.

FOUR ESSENTIAL COMPONENTS of any Senior Care program should include; 1. A comprehensive patient health assessment (discovery) 2. A formal 3. A review period where all the findings are communicated to the owner and 4. A formulating specific short & long term action plans A scheduled follow-up

I. HEALTH ASSESSMENT:
When implementing a Senior Care program, a critical decision each practice must make is establishing the boundaries of the recommended health assessment. Exactly how detailed the history should be? Which diagnostic tests should be included in the health assessment? The ultimate programmatic decision is based on several interrelated factors including patient age, presence or absence of disease, current medications, and client resources. Most hospitals use a basic screening strategy however others employ a more extensive comprehensive health evaluation approach.

Historical health assessment utilizing a medical, behavioral and dietary history is the starting point of any program. Utilizing a waiting room questionnaire greatly expedites time, ensures completeness plus can be a very useful in educating the client on the warning signs of disease or behavioral problems. Pet owners can be invaluable sources of information on the overall health of their pets. Observant owners can detect subtle changes in their pet’s activity levels, alterations in elimination patterns, and behavior. Often this critical information is unapparent to the veterinarian in an examination room setting. Owner’s medication and monitoring skills are paramount for success in managing certain chronic diseases. Therefore, our goal must be convince each owner to become a much more active partner in the health care of their aging pet.

A complete age related physical examination is the second part of the health assessment. In addition to a regular physical, the exam should also include a weight assessment, gentle palpation of each joint and digital rectal examination. Extra time should be taken for diligent palpation of the mammary glands, skin and subcutaneous
for any tumors. Each tumor identified should be accurately measured, mapped and recorded in the medical record.

A minimum data base laboratory evaluation (MDBLE) should include a CBC, chemistry profile, complete urinalysis, fecal and thyroid screening. Many Senior Care programs also incorporate additional diagnostic testing such as a lead II ECG, thoracic radiographs, abdominal imaging, blood pressure measurement, adrenal function testing, and ocular pressure determination into the evaluation.

II. REVIEW PERIOD
A formal review period is the time where all the normal and abnormal findings are communicated to the owner. In addition to the important verbal communications of any health concerns, I also suggest you provide a specific written evaluation, a health report card, of their pet’s health. Sending home any appropriate client education materials helps reinforce any health issues. To be optimally effective, this step must occur in a timely fashion and not several days or weeks later.

III. SHORT AND LONG TERM ACTION PLANS
During the review period, you and the client need to formulate specific short and long term plans of action for each problem identified. Specific recommendations for medication, diet, exercise, and dental care should be explicitly communicated. Dietary recommendation should be based on the health needs of the patient and not on cost alone. Those factors influencing the diet selection includes quality of ingredients, specific anti-oxidants shown to modify the aging process and a research based formulation.

IV. FOLLOW-UP
Timely follow-up telephone calls and written reminders are essential components and critical in the overall the success of any Senior Care Program.

MAKING THE PROGRAM SUCCESSFUL
Obviously any professional rewards are proportional to the success of the program. While success is never guaranteed, the follow steps may help you reach your program goals.
1. Convince yourself that a Senior Care Program will become a significant asset to your practice before you invest the time, energy, training and resources necessary in developing and maintaining the program. However, because of the commitment necessary for success and growth, this program is not for every practice and may not appeal to every client.
2. Convince your staff of the significant health benefits the program offers the senior pet. Critical to the success or failure of a Senior Care program is the involvement and buy-in of your staff. In fact, ownership of the program by every staff member is essential. Staff incentive programs will also help the Senior Care program patient base grow and maintain the program’s momentum.
3. Create a very specific and detailed program including age of onset, frequency of visits, scheduling periods, fee structure, educational materials, and marketing strategies. Decide exactly which tests are to be included in the routine program.
Finally ensure every member of the staff is “program proficient” in detailing the specifics.

4. Now convince the owners of the significant health benefits the program offers their aging pet. A percentage of clients will readily accept the program, but the rest will need repeated convincing. Increased client knowledge usually equates to increased client acceptance and compliance. Early and persistent owner education is a long term investment in Senior Care.

5. A well designed market strategy correlates with success. Utilize newsletters, reminder cards, invoices, yellow pages, and the media to educate your clients and prospective clients on age-related problems. Many practices participate in various Senior Month activities. Some practices celebrate a particular older pet as the Senior of the Month. The pet’s picture and life story are placed on the waiting room bulletin board. Client marketing efforts should emphasize all the State of the Art advances in veterinary medicine including newer diagnostic testing, improved anesthetics and anesthetic monitoring equipment, behavioral drugs, newer arthritis therapy options, leading edge cancer chemotherapy, more effective cardiac medications, dental care and nutritional advancements.

6. Bundle the fee structure including a senior citizen discount. Charge one fee for both visits or discount other preventative health care services/products.

7. Start slow be patient and the program will grow. A Senior Care Program is a long-term hospital investment and actually begins when outlining a life long preventative health care program to new puppy or kitten owner.

8. Periodic program review by your clients and staff is essential in maintaining the consistent high standard of care you have established for your senior patients.

The goal of Senior Care Program should be to optimize the quality of life for the older pet, using preventative health care combined with State of the art diagnostics and therapies. Historically, veterinarians have only reacted to diseases / problems in the elderly animals. Implementing Senior Care Program is a pro-active approach to elderly health care and not waiting until overt disease is present. The program should emphasize client education of age related disease, steps for prevention, programs for early detection, and timely medical intervention using state of the art diagnostic and treatment modalities.