Maximising mobility with nutrition

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Authors' Profile

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Introduction

Although the definition of 'quality of life' might be controversial, nearly everyone agrees that having freedom of movement is a key component. In our pets, mobility is associated with health and positive interactions with us (fig1). When our pets lose any of their capability for comfortable physical activity, it can be interpreted as a decrease in their quality of life and deserves attention to correct or minimise the primary cause.

Figure 1. Maximising the mobility of our pets is important for our health as well

Potential physical causes of mobility loss are often regional, or local musculoskeletal changes, but can also be a central neurological or metabolic disease. The most common musculoskeletal aetiologies involve trauma or degenerative changes to joints, bones and muscles. The 'trauma' may be an obvious athletic or accidental injury but it also includes the adverse insidious physical effects of poor skeletal growth and the long-term effects of excess body weight. Regardless, the

physical factors tend to manifest themselves ultimately with decreased joint function. From a nutritional standpoint, there are considerations that can help to manage the condition and improve the pet's quality of life.

Factors during growth that can affect mobility

One of the truly remarkable characteristics of the canine is the variation of mature body weights across breeds. Breed descriptions indicate mature body weights ranging from less than 3kg for the Chihuahua and Pomeranian to greater than 75kg for the Mastiff, a 25-fold difference. In practice; however, the range in mature size often greatly exceeds these reference boundaries. Application of genetic selection pressure favouring a large mature body size within specific canine breeds concurrently selects genotypes with a remarkable ability for a rapid rate of growth. For example, 20 years ago a typical Great Dane grew to approximately 60kg mature body weight by 12 to 18 months of age. Today, mature body weights of 80kg or more during that same 12- to 18-month growth period are often achievable.

Unfortunately, it is often not fully appreciated that the genetic propensity for rapid growth rate, co-selected with mature body size, is associated with potentially negative consequences during growth and latent effects later in life. It is well documented that the incidence of skeletal disease, including osteochondroses, hypertrophic osteodystrophy and hip dysplasia, are markedly increased if management practices are such that this maximal genetic potential for growth rate is realised. Of particular importance is the influence of nutritional management on growth rate and skeletal disease. Three dietary components - energy, protein, calcium (Ca) (and phosphorus P) - have been implicated as primary contributors to an increased incidence of skeletal disease in the growing large-breed puppy. Understanding these factors can help us to improve the mobility of growing puppies and contribute to enhanced skeletal health as adults.

Energy

Hedhammer and colleagues[1] investigated the issue of dietary energy intake by feeding growing Great Dane puppies either *ad libitum* or 66% of *ad libitum* intake and observed a dramatic increase in the incidence of skeletal pathology in puppies consuming the higher level of intake. Apparently, the high plane of nutrition effectively supported the genetic potential for rapid growth and predisposed the large-breed puppy to the development of skeletal disease. Dammrich[2] provided further support for this growth rate response by feeding Great Danes either *ad libitum* or restricted (60% to 70% of *ad libitum*) intake from weaning through 6 months of age. Maximal growth (*ad libitum* intake) resulted in subchondral spongiosa that was less dense and weaker per unit area. The resulting osteopenia and biomechanically weak subchondral bone could not adequately support the articular cartilage of the joint. In addition, the increased growth rate of *ad libitum* feeding more rapidly subjected the joint surface to stresses due to increased body mass. Clearly, a high level of energy intake promotes an excessive rate of growth in the large-breed puppy and increases the potential for skeletal disease.

Protein

Dietary protein level has also been implicated as influencing the incidence and severity of skeletal disease in the growing large-breed puppy. Controlled research, however, does not support the hypothesis of an association between high dietary protein intake and skeletal abnormalities. Nap and colleagues[3] fed Great Dane puppies isocaloric diets that provided a broad range of dietary protein (31.6%, 23.1%, or 14.6% protein 'dry matter') from weaning for 18 weeks and observed no treatment effect on calcium metabolism or skeletal development. Protein, within a range of practical levels and aside from the calories it provides, does not contribute to developmental bone disease.

Calcium

In contrast to dietary protein, calcium concentration has been demonstrated to have a significant effect on development, morphology, and pathology of the skeleton in the large-breed puppy. Hazewinkel and colleagues[4] and Goedegebuure and Hazewinkel[5] evaluated the effect of feeding either a typical calcium diet (1.10% Ca/0.90% P) or a high-calcium diet (3.30% Ca/0.90% P) to Great Dane puppies from weaning through 6 months of age. The effect of the high-calcium diet (3.30%) on endocrine status (hypercalcaemia, hypophosphataemia, less active parathyroid glands, increased activity of thyroid C cells), skeletal development (increased osteoblasts, decreased osteoclasts, decreased osteoclast activity, more retained cartilage cones, increased bone mineral mass, delayed bone remodeling), and skeletal disease (increased radiographic irregularities, more osteochondritic lesions) clearly demonstrated the negative impact of excess dietary calcium on skeletal health of the large-breed puppy. This conclusion was further supported by the finding that the large-breed puppy was ineffective in reducing intestinal calcium absorption when provided a high-calcium diet and was therefore unable to protect itself from a chronic high dietary calcium intake.[6]

Goodman and others conducted a study to establish recommendations for dietary calcium and energy levels.[7] Great Dane puppies (total of 36) were assigned to 3 treatment diets differing in calcium (Ca) and phosphorus (P) concentration as follows: 0.48% Ca/0.40% P (low calcium), 0.80% Ca/0.67% P (medium calcium), and 2.70% Ca/2.20% P (high calcium). All diets were formulated to contain 14% fat (reduced energy), and 26% protein to maintain an appropriate dietary protein:energy ratio. The three diets were fed pre-weaning through 18 months of age. The results indicated that while managing growth rate in all treatment groups the medium-calcium diet (26% protein, 14% fat, 0.80% Ca, 0.67% P) produced more controlled growth and increased body weights relative to the other two diets utilised in these studies.[7] The high calcium diet (2.70%) consistently produced poorer body conformation,[7] was associated with 86% of all clinical lameness observed and produced all cases of hypertrophic osteodystrophy. Repeated kinetic gait analyses conducted throughout growth (4, 6, 8, 12, and 18 months of age) on 4 puppies per treatment group indicated that all the puppies consuming the low-calcium (0.48%) or high-calcium diet (2.70%) had some evidence of gait asymmetry. In contrast, 3 of 4 dogs on the medium-calcium diet (0.80%) had normal gait symmetry at all examinations.

Factors during adulthood that can affect mobility

Osteoarthritis is a common disease and is the most prevalent joint disease in dogs affecting as many as 20% of adults – and a recent study in a population of cats revealed 34% had radiographic evidence of degenerative joint disease, including appendicular osteoarthritis.[8] However, our understanding of the diagnosis and management of dogs and cats with osteoarthritis is still developing. Veterinarians often consider osteoarthritis as a specific disease entity, when it should be thought of as a disease process. Although many aetiologic factors can initiate disease development, they all result in the common disease process of osteoarthritis (fig 2).

Figure 2. Examination of joint mobility to help asses for osteoarthritis.

The main diagnostic procedure for osteoarthritis is radiography (see the text 'Diagnostic imaging of orthopaedic problems in small animals: a practical guide' in these proceedings pages 7-13) and radiographic diagnosis of osteoarthritis is based upon the classic features of joint effusion, periarticular osteophyte formation, subchondral bone sclerosis and altered joint shape. Radiographic screening of joints such as the elbow, shoulder, stifle and hip will often reveal a high prevalence of osteoarthritic change, particularly in certain breeds of pedigree dogs in which developmental joint disease, such as hip dysplasia or elbow dysplasia, are common. However, many of these dogs will have no clinical signs of joint disease. In human beings, it is recognised that radiographic change is not a good marker for clinical outcome.

Age-related joint change is likely to explain much of the osteoarthritis that can be identified radiographically in dogs. However, in contrast to human beings, epidemiological data is limited in evaluating the effect of age on osteoarthritis in dogs. Joint stresses associated with cyclic loading can lead to development of osteoarthritis with time, and if cyclic loading is excessive, or occurs at high stresses, development of osteoarthritis may be more rapid. Impact loads from a fall or other trauma also

may induce damage in subchondral bone and cartilage leading to osteoarthritis.

Although incipient osteoarthritis is identified frequently in dogs, there is limited data examining risk factors for the disease progressing to severe osteoarthritis associated with lameness. This is in contrast to human beings where there have been many epidemiological studies of osteoarthritis and it is well accepted that factors, such as obesity and physical activity, are important determinants of risk of initiation of osteoarthritis and also the risk of disease progression. Because radiographic evaluation of osteoarthritis is a poor marker for clinical significance and disease progression, use of various biochemical markers in synovial fluid and serum has been widely studied in human beings and is beginning to be studied in dogs. Markers, such as cartilage oligomeric matrix protein[9] may well prove to be superior to radiography for determining the risk of disease progression in dogs. Pathological change in subchondral bone is currently considered an important part of the pathogenesis of osteoarthritis, particularly for risk of disease progression. Therefore, markers of pathological change in periarticular bone, such as serum markers of bone turnover, or measurement of subchondral bone mineral density may also become important for assessment of dogs with osteoarthritis.

Another very important measure is mobility itself. While radiographs will provide objective data on pathological changes, the goal is mobility. Descriptive terms can be used but experience has shown that a 'visual analogue scale' (VAS), or a numerical rating score, tend to work best (fig 3). With a VAS, both the owner and attending veterinarian can score the patient easily. To do this, the VAS is used and a mark is made on the line where the observer feels the dog's mobility is represented. To turn this into numeric data, the length of the line is measured from the 'clinically sound' end. This number and the VAS can be included in the patient's record.

Clinically sound ——— **Extreme lameness**

Numerical Rating Score	
Score	**Criteria**
0	Sound
1	Barely detectable lameness
2	Mild lameness
3	Moderate lameness
4	Severe lameness (carries leg when trotting)
5	Extreme lameness (not ambulatory)

Figure 3. The Visual Analogue Scale (top) and the numerical rating score (bottom)

Primary and secondary joint disease: treatment

Treatments of primary and secondary joint disease includes weight reduction, exercise modification, and administration of nonsteroidal anti-inflammatory drugs (NSAIDs), glucocorticoids, or polysulphated glycosaminoglycans (GAGs). Several of these treatments either directly require, or may be influenced by, nutritional management. Nutritional objectives include optimising growth, managing obesity, controlling eicosanoids, and minimising the effects of injury through chondroprotective agents.

Weight

The propensity for companion animals to become obese continues to increase each year. Several studies have documented this rise and estimate that 25 to 44% of dogs and 20 to 35% of cats are obese.[10,11] While a majority of dogs and cats do maintain a more normal body weight through sustaining an optimal balance between energy intake and energy expenditure, there is a growing subpopulation of animals that are influenced by several predisposing factors. Examples include excessive overfeeding, decreased activity level, genetics, and even having an overweight owner. In addition, pet obesity is associated with a large number of disease states that can exacerbate this condition by negatively impacting health, wellness, longevity and mobility.

Objective measures of a patient's weight status are necessary. Clearly, weight as measured by an accurate scale is necessary, but other means such as girth measurements, are also available that allow both the owner and clinician to assess additional characteristics. Comparing the dog's weight to breed standards where appropriate can be a first step to evaluating the degree of obesity. Given the wide ranges within breeds and the popularity of mixed breed dogs, assigning a Body Condition Score

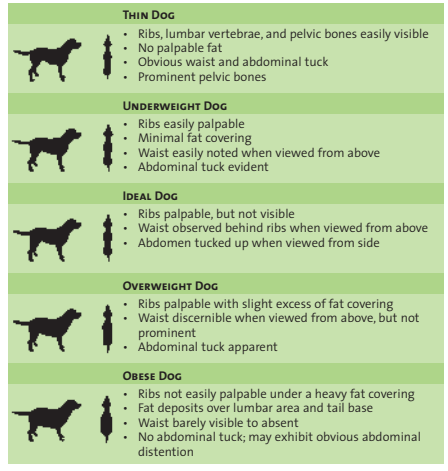

Figure 4. Body Condition Score for Dogs

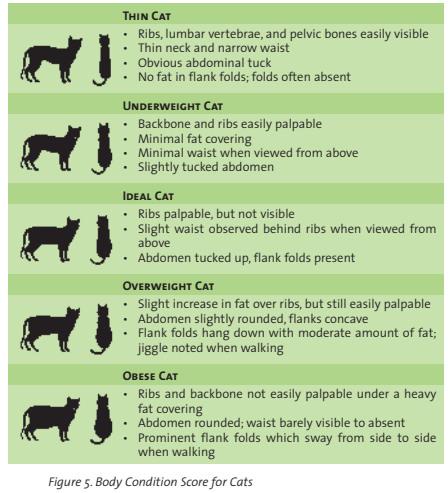

Figure 5. Body Condition Score for Cats

(BCS) may be a more practical approach to determining level of obesity (figs 4 and 5). Determining a BCS requires a visual and tactile evaluation of the ribs, spine, pelvis, waist, and abdomen. If you don't put your hands on the dog you have not completed an accurate BCS!

While data describing the impact of obesity on the prevalence of osteoarthritis and the risk of disease progression in dogs is not available, obesity is recognised as an important factor influencing the clinical significance of osteoarthritis in human beings.

Many dogs with lameness because of hip osteoarthritis are overweight. Although long-term objective analysis of results of medical treatment of immature dogs with hip dysplasia has been published,[12] alterations to lameness severity in overweight dogs with hip osteoarthritis following weight reduction were only recently described. It is known that limited food consumption to maintain optimal weight during growth and during adulthood does reduce development and severity of hip osteoarthritis measured radiographically in hip dysplasia susceptible dogs.[13] Impellizari and colleagues found that lameness severity in overweight dogs with hip osteoarthritis was significantly influenced by body condition.[14] Weight reduction was associated with 76% improvement in lameness scores as assessed by both owners and attending veterinarians, and analgesic medications were able to be decreased in many patients.

Management of the overweight pet involves

- Diet
- Exercise
- Behavior modification

Diet

The severity of caloric restriction is not universally agreed. A very successful method is to provide 25% fewer calories than the overweight dog or cat's current weight. This amount is adjusted as weight is lost and can result in steady, safe 1-1.5% weight loss per week, which is desirable. The alternative is to estimate the pet's ideal body weight (IBW) and give fewer calories than would be needed to maintain that IBW. This latter method is awkward in that it requires a very experienced clinician to know each breed's or mixed breed's body characteristics and to be able to accurately estimate IBW. For severely overweight patients, it also places an equally severe restriction on the caloric intake that might result in exceeding 1 – 1.5% weight loss per week.

In addition to caloric restriction to reduce weight (fig 6), several important nutrients, or diet composition characteristics, help to both lose weight and to keep weight gain from recurring (recidivism).

Figure 6. Veterinary Nurses can provide invaluable support to owners whose pets are undergoing a weight loss programme

Carbohydrate Source

One such new approach is managing blood glucose postprandially in dogs and cats. Improper metabolic management of the glycaemic response (response of both blood glucose and insulin) is associated with becoming overweight. Mattheeuws et al. reported that glucose as well as insulin levels, and secretion rates, were related to an animal becoming obese.[15,16] Thus, choosing the appropriate source of starch in a weight loss diet will affect an animal's glycaemic status.[10]

The source of carbohydrate in a weight loss diet can highly impact blood glucose concentrations (fig 7). Several studies have demonstrated that starch sources are not uniform with regard to the postprandial glucose and insulin response in dogs (fig 8) or cats. Rice in both species produced a high glycaemic index (rapid increase in blood glucose and a rapid, exaggerated increase in serum insulin). However, in dogs and cats, barley, sorghum and maize (corn) produce a reduced glycaemic response compared to rice. Therefore, in dry weight loss diets a combination of barley and sorghum for dogs, and maize and sorghum for cats, will give good glycaemic control to facilitate weight loss.

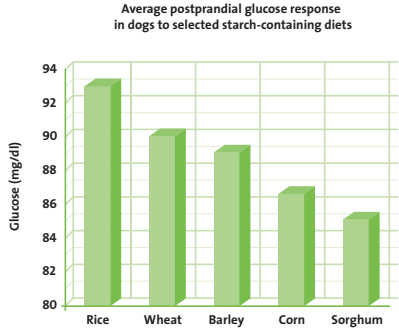

Figure 7. Carbohydrate effect on canine postprandial glycaemia

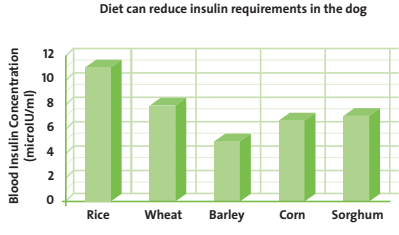

Figure 8. Postprandial effect of carbohydrate on canine blood insulin

L-Carnitine

L-Carnitine, a vitamin-like compound, helps to transport fatty acids into mitochondria for conversion to energy, which ultimately results in better body composition and desired weight loss. Studies conducted in dogs fed diets supplemented with L-carnitine resulted in greater weight loss while also losing fat mass compared to diets not supplemented with L-carnitine. Lean body mass is lost at a lower rate in L-carnitine supplemented diets compared to identical diets without L-carnitine. Similar results in cats have been demonstrated by Center et al.[17] These data have proven the efficacy of L-carnitine use in weight loss diets for dogs and cats to promote desired weight loss and improved body composition.

Dietary Fat

Eicosanoids are local chemical mediators (e.g. leukotrienes, thromboxanes, prostaglandins, and prostacyclins) produced by the actions of the enzymes cyclooxygenase and lipoxygenase on arachidonic acid (an omega-6 or n-6 fatty acid) and eicosapentaenoic acid (an omega-3 or n-3 fatty acid). Eicosanoids mediate inflammation, pain and fever, blood pressure, blood clotting, several reproductive functions, and the sleep/wake cycle.

Manipulation of eicosanoid production can also be obtained by altering the types of fatty acids present in the diet. Rather than effecting the enzymes responsible for eicosanoid production, this approach alters the substrates presented to these enzymes, which dictates the families of eicosanoids produced. For instance, feeding n-3 fatty acids results in the replacement of the 20 carbon arachidonic acid (n-6) in the cell membrane with the 20 carbon n-3 eicosapentaenoic acid. The eicosanoids derived from arachidonic acid (prostaglandin E_2, leukotriene B_4, leukotriene C_4, and thromboxane A_2) are pro-inflammatory, whereas eicosanoids synthesised from eicosapentaenoic acid (prostaglandin E_3 and leukotriene B_5) are less inflammatory.

Chondroprotective Agents - Chondroitin sulphate and glucosamine

Oral chondroprotective agents may modulate joint structure and physiology.[18] Two such agents which are included in the majority of nutritional joint health supplements are glucosamine and chondroitin sulphate. A number of controlled, short-term studies have found glucosamine and chondroitin sulphate (both together or individually) have some degree of chondroprotective activity.

Glucosamine is a ubiquitous amino sugar used in the synthesis of the disaccharide units of glycosaminoglycans (GAGs). In humans, an oral dose of glucosamine sulphate is 90% absorbed and 26% bioavailable. Another study found the bioavailability, pharmacokinetics and excretion pattern in the human to be consistent with those of the dog.[19] Chondrocyte studies *in vitro* found that glucosamine has a stimulatory effect causing increased production of normal collagen and proteoglycans.[18] Using rat models of subacute inflammation and subacute mechanical arthritis, oral glucosamine was found to be 50–300 times less potent than indomethacin (a non-steroidal anti-inflammatory drug used to reduce fever, pain, stiffness, and swelling, working by inhibiting the production of prostaglandins). But glucosamine's chronic toxicity is 1,000–4,000 times less toxic than indomethacin which suggests a therapeutic margin 10–30 times more favorable for using glucosamine in prolonged oral treatments.[20]

Chondroitin sulphate is a GAG which can be sulfated on the fourth or sixth carbon.[18] Chondroitin-4-sulphate is the predominant GAG in growing mammalian hyaline cartilage, but with age, production of chondroitin-4-sulphate decreases and more production of other types of GAGs is increased by chondrocytes.[18] Two chondroprotective activities produced by chondroitin sulfate, but not by glucosamine, are prevention of thrombi formation in microvasculature and inhibition of metalloprotease enzymes via the modulation of interleukin-3.[18] A 1998 study in humans with uni- or bilateral knee osteoarthritis found that both the chondroitin sulphate dosing regimens used (40 people received a single daily dose of 1,200 mg of chondroitin sulphate in an oral gel and 43 received 400 mg capsules TID) produced improvement of the subjective symptoms and improved mobility.[21]

In a dog study, 13 Beagles (age <1year, weight 4.2–5.1kg) received 2 capsules of glucosamine, chondroitin sulphate, and ascorbate orally twice daily for 30 days (daily dose of 800 mg sodium chondroitin sulphate, 1,000 mg glucosamine hydrochloride, and 152 mg manganese ascorbate).[22] Serum GAGs (12 dogs) were elevated by 37% with no change in circulating hexosamine (3 dogs). Calf cartilage segments were incubated in serum collected (9 dogs) before and after treatment. Following treatment, the biosynthetic activity in the cartilage increased by 50% and proteolytic degradation decreased by 59%. Glucosamine and chondroitin sulphate have not produced reports of safety concerns. Another study found that

glucosamine and chondroitin sulfate (2 capsules, PO, q 12; daily dose of 800 mg sodium chondroitin sulphate; 1,000 mg glucosamine hydrochloride; and 152 mg manganese ascorbate) administered to 13 clinically normal Beagles for 30 days produced statistically significant but clinically unimportant changes in haematocrit, haemoglobin, white blood cells, segmented neutrophils, and red blood cells; however, no changes were seen in the prothrombin time, activated partial thromboplastin time, mucosal bleeding time.[23]

In considering oral supplementation of glucosamine and chondroitin sulphate for management of joint problems we must be aware of the route of supplementation. If capsules or pills are utilised owner compliance may be an issue of concern. Inclusion of these compounds in a total diet will eliminate any concerns for compliance.

Exercise

Exercise regimens for dogs vary from easy walks to running, and from hide-and-seek to elaborate agility courses. Studies have not been done in dogs to evaluate the benefit of one versus the other, but it is known that sustained activity will burn fat better than short or intense exercise, which relies on muscle and liver glycogen more than free fatty acids. Experience has taught all of us that any exercise is better than none and that 20 to 30 minutes daily seems to be a good target.

Behaviour Modification

Behaviour modification is a significant component and a key to any successful weight control programme. The real target for the behaviour modification is the owner! With the increasing association between an owner's body composition and their pet, it seems obvious that the human lifestyle is affecting the pet's lifestyle, as well (see the text 'Can canine mobility benefit human health?' in these proceedings: pages 41-45). Although exercise represents a behaviour modification, the emphasis should also be placed on non-fattening patterns of interaction. Many owners interact with their pets by giving food treats or rewards that are not considered in the daily caloric allocation. A 'begging' dog or cat does not necessarily need to eat; many times the pet is simply asking for some play time. If play is provided, it gives the pet the attention being sought as well as burning a few calories and toning muscles.

Combining Efforts

Compliance with a weight control diet or an exercise regimen is often poor. Since the likelihood of an overweight owner is now three times more likely to have an overweight dog than a normal weight owner, combining the pet's needs with those of the owner can be very beneficial for both. Motivation for either can be a limit to successful weight control. A study by Green and others examined the effect of both human and dog participating in a medically supervised weight control programme together.[24] The results showed that

- 90% of the dogs in the study lost weight
- 54% of humans participating in the study lost weight
- Dogs that lost weight had a mean body weight loss of 10%
- Humans that lost weight had a mean body weight loss of 3%

Among other observations, some were specific to mobility. The combination of diet, exercise and owner involvement

- allowed the dog to be less stiff when getting up in the morning, and
- positively affected the animal's ability to run more, go for more walks, and use the stairs more

Nonsteroidal anti-inflammatory drugs and Corticosteroids

Nonsteroidal anti-inflammatory drug (NSAIDs) or corticosteroids are frequently included in treatment regimens for traumatic and degenerative joint problems. Both drug therapies are aimed at reducing the production of arachidonic acid metabolites (eicosanoids), which are released as a result of cell membrane damage and cause inflammation and pain. These drugs act directly to inhibit the enzymes that help produce these eicosanoids.[25] Some NSAIDs have contraindications for use in growing animals or with cartilage damage; the drug information should be reviewed for details.

Recommendations

We are all striving to enhance the quality of life of our patients in many ways. Mobility is a key part. Although genetics, the bumps and knocks of normal life and other diseases can affect mobility, there are nutritional means by which we can help our patients have the best opportunity to be as mobile as they can be. Long-term provision of premium nutrition is associated with an increased range of motion.[26] With the benefits available, we should be encouraging all of our patients to take advantage of these options to improve their quality of life with the maximum mobility possible (fig 9).

- Encourage healthy growth with controlled rates of growth by controlling calories and for properly developed bones by limiting calcium to 0.8% in large breed puppies.
- Maintain a healthy, ideal body weight by controlling caloric intake, selecting a diet made to control weight and by paying attention to the patient's BCS to catch and manage weight gains quickly.
- Provide nutritional support for healthy joints with glucosamine and chondroitin sulphate.
- Conduct regular, appropriate exercise to burn extra calories, maintain muscle tone and encourage an interaction between the owner and their pet.

Figure 9. Dogs with an ideal body weight will have a better chance of maintaining their mobility.

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