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Effects of Restricted Feeding on Onset, Incidence, and Severity of Hip Dysplasia and Osteoarthritis in Dogs: Diagnostic, Therapeutic, and Genetic Ramifications

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Osteoarthritis (OA) is a common, often-debilitating disease of both animals and humans. In dogs a highly prevalent form of OA, known as canine hip dysplasia (CHD), is capable of causing pain and dysfunction in affected animals. The prevalence of CHD is as high as 75% in some breeds of dogs. Canine hip dysplasia is a developmental disease recognized to have complex inheritance, a so-called polygenic disease. It is understood that the incidence and severity of such genetic diseases can be influenced considerably by environmental factors,1–4 meaning any “non-genetic” factor, such as diet, lifestyle, housing, or trauma. Dogs that develop CHD are born with apparently normal hip joints, but after they have reached a few weeks of age the earliest signs, such as hip joint laxity, can be demonstrated by necropsy studies.1,5,6

It is well accepted that hip laxity plays a prominent role in the pathogenesis of CHD.7,8 Initial phenotypic expression of CHD is recognized radiographically as femoral head subluxation, often followed by progressive joint degeneration. Femoral head subluxation, subjectively scored on the hip-extended radiograph, and the distraction index, directly measured on the distraction radiograph, are two types of passive hip laxity. They are static phenomena. Osteoarthritis, however, is thought to be caused by “functional” hip laxity, the dynamic phenomenon associated with the femoral head slipping partially out of the acetabulum during weight bearing.8 It has been shown that joints with excessive passive hip laxity are susceptible to traumatic (functional) subluxation of the femoral head.9 Tight joints are not similarly susceptible. Hip joint laxity and femoral head subluxation, if functional, produce pathologic consequences that include abnormal loading during weight bearing leading to cartilage damage and bony remodeling. The end result of this self-perpetuating cycle of abnormal loading and remodeling is osteoarthritis, characterized by synovitis, increased joint fluid volume, joint cartilage erosion, subchondral sclerosis, elongation and edema of the round ligament, thickening of the joint capsule, and osteophyte formation.6,7,10

Degenerative changes develop in joints of many dogs as they age; however, more severely affected dogs will show radiographic signs of OA well before the geriatric period, often by 1 year of age or younger. Joint laxity associated with CHD has been shown to be the most important risk factor for OA in canine coxofemoral joints.8,9 Trauma or metabolic dysfunctions are also thought to affect disease expression.8,9 One theory of pathogenesis for OA includes the idea that excessive body weight leads to mechanical stress on joints, facilitating the transformation of passive hip laxity to functional hip laxity, thereby initiating OA. Excessive body weight has been documented as a risk factor for OA development in humans, guinea pigs, mice, and dogs.9,11–16

The studies summarized here were conducted to evaluate the effect of food restriction on development of hip joint laxity during growth and subsequent occurrence of OA in coxofemoral joints during adulthood. In addition, the effect of food restriction on development of OA in joints other than hips was evaluated.

Effect of Limited Food Consumption on Hip Dysplasia in Growing Dogs

In this study,17 48 8-week-old Labrador retriever puppies from seven litters were allotted by pairing to two groups of 24 dogs each. This pairing created two groups of genetically similar dogs. One group was fed ad
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libitum (full-fed) and each member of the other group (restricted) was given 25% less of the same food given to the full-fed pair mate. Radiography of the hip joints was done when the dogs were ages 30, 42, 54, 78, and 104 weeks. Subluxation was measured using the Norberg angle on radiographs made with the dog in the standard, hip-extended position. The same radiographs were evaluated for evidence of CHD by independent, blinded radiologists, one using the Swedish scoring system and the other, the Orthopedic Foundation for Animals (OFA) scoring system.

Independent of the age at which radiography was performed, hip joint quality was better in the diet-restricted dogs as a group. Radiographs taken when dogs were age 104 weeks revealed less subluxation and less OA in restricted-fed dogs, irrespective of how the hips were scored, using the Norberg angle, Swedish scoring system, or OFA scoring system. Using the Swedish method, CHD at 2 years of age was diagnosed in 5 of 24 restricted dogs and 18 of 24 full-fed dogs. Using the OFA method, CHD was diagnosed in 7 of 24 restricted dogs and 16 of 24 full-fed dogs. These findings supported a clinical recommendation to avoid excessive food intake in growing dogs (at least up to 2 years of age), particularly in breeds prone to CHD, such as the Labrador retriever.

**Effect of Limited-Food Consumption Over 5 Years on Coxofemoral Joint Osteoarthritis**

The same Labrador retrievers from the 2-year growth study were continued on test for an additional 3 years and additional radiographic evaluations were recorded yearly. At 3.25 years of age two adjustments were made in the feeding protocol. All dogs were switched from a 27% protein puppy growth formula to a 21% protein adult formula, and the amount of food was reduced to prevent insidious development of obesity in full-fed dogs. Ideal body weight was estimated for each full-fed dog on the basis of skeletal size in reference to other dogs of the same breed. These dogs then were fed 62.1 kcal metabolizable energy/kg of estimated ideal body weight. Restricted dogs continued to receive 75% of the amount fed to the corresponding full-fed pair-mate, and thus the intake relationship between the two groups remained the same as prior to the diet adjustment.

Radiographic evaluations of coxofemoral joints for frequency and severity of OA were done at yearly intervals up to age 5 years. Three investigators evaluated each radiograph without knowledge of group assignments, using a scoring system based on radiographic features of OA. The median value obtained by the three investigators was assigned as the score for each dog.

Frequency and severity of OA were greater in full-fed dogs. By age 52 weeks, differences between the groups for frequency and severity of OA were statistically significant. The prevalence of OA in hip joints increased through age 5 years. By age 5 years, 12 of 23 full-fed dogs and 3 of 23 restricted dogs had radiographic signs of OA. Body weight correlated significantly with OA scores at age 5 years.

Dogs varied considerably with respect to individual maintenance energy requirements, and so it is not feasible to specify a universal energy intake to achieve the benefits observed in the restricted dogs. It is recommended, however, that growing puppies and adult dogs be fed throughout their lives to maintain lean body conformation to minimize the development of coxofemoral OA with advancing age.

**Effect of Limited Food Consumption on Multiple-Joint Osteoarthritis**

In this study, restricted dogs continued to receive 75% of the amount fed to the corresponding full-fed pair-mate through age 8 years. Hip, shoulder, elbow, and stifle joints were evaluated radiographically at this time. One radiologist without knowledge of dietary treatment evaluated the radiographs.
Osteoarthritis affecting multiple joints was significantly more common in full-fed dogs. Ten of 22 (45%) full-fed dogs had OA in two different joints, and seven (29%) had OA in three joints, whereas only one of 21 (5%) restricted dogs had OA in two joints, and one (5%) had OA in three joints. Five restricted dogs had no evidence of OA in any evaluated joint, while only two full-fed dogs lacked evidence of OA in the evaluated joints.

Theoretically development of OA at a primary site such as the hip could produce aberrant compensatory biomechanical forces acting on other joints, the altered weight bearing and ambulation leading to multiple joint OA. Hip joints were not always affected first; some dogs had radiographic evidence of shoulder or elbow lesions without hip joint involvement. Thus, alternatively, it may be hypothesized that OA has a systemic cause, with variable expression in different joints. This might explain, in part, the development of OA in joints that are not subject to large forces associated with weight-bearing, such as lumbar intervertebral joints in dogs and joints in the skeleton of sharks. Alternatively, a humoral substance from an affected joint may affect tissues in other joints. This concept is supported by the recent finding that extract of human arthritic bone tissue induced abnormalities in disease-free articular cartilage. Additional support for the concept of variability in tissue susceptibility is derived from a recent report that shoulder joints in young adult dogs at high risk for CHD have histopathologic articular cartilage abnormalities similar to those found in the corresponding hip joints. Yet another hypothesis might be that OA among different joints represents phenomena that are not interrelated or interdependent.

**Lifelong Benefits of Restricted Feeding: Diagnostic and Therapeutic Implications**

(Unpublished Results as of February 2003)

It is generally agreed that there has been slow progress in reducing the incidence of CHD by selective breeding of normal dogs. The conventional diagnosis of CHD has been based on subjective radiographic findings of subluxation of the coxofemoral joint, or secondary OA as seen on evaluation of the hip-extended, ventrodorsal radiographic view of the pelvis. In the United States this analysis is performed by the OFA after dogs are 2 years of age. In much of Europe a similar analysis is made after 1 year of age. In the published reports cited above it was shown that the condition of excessive hip laxity (Norberg angle measured from the hip-extended radiograph) can be marginally reduced (approximately 4 degrees) by reducing caloric intake and this tightening of the joint prevents or delays the expression of OA in some dogs predisposed to the condition. It has been generally assumed that the subjective scoring of hip phenotype at 1 or 2 years of age accurately reflects the true phenotype of the dog. No lifelong studies have been conducted to document the accuracy of the one- or two-year evaluation to predict the end-of-life hip phenotype. It was the purpose of the present investigation to test the lifelong influence of food restriction on hip phenotype and to compare end-of-life hip phenotypes with OFA scores, PennHIP® scores, and OA scores at 2 years of age.

**Materials and Methods.** Forty-eight 8-week-old Labrador retriever puppies from seven litters were allotted by pairing to two groups of 24 dogs each. One group was fed ad libitum (full-fed) and each member of the other group (limit-fed) was given 25% less of the same food given to the full-fed pair-mate. Radiography (ventrodorsal, hip-extended) of the hip joints was done when the dogs were ages 30 and 54 weeks and yearly thereafter for life. Subluxation was measured using the Norberg angle on radiographs made with the dog in the standard, hip-extended position. The same radiographs were evaluated for evidence of CHD and OA by a board-certified radiologist using the scoring system of the OFA. The OFA scoring system is a subjective seven-point scale (Excellent → Severe hip dysplasia) applied to the hips as viewed on the hip-extended radiograph. At 2 years and 8 years of age the hips were scored using the PennHIP method.
includes two other radiographic views with the legs in a stance-phase, weight-bearing position: the compression view and the distraction view. The amount of passive laxity in the hip is maximized and measured from the distraction radiographic view yielding the so-called *distraction index* (DI).

A few dogs in the study died of one or more causes prior to the geriatric years. Most dogs, however, survived into old age. Dogs were humanely euthanatized based on uniform criteria assessing comfort and quality of life. Gross and histopathologic evaluations of articular and perarticular tissue were performed on 45 of the 48 dogs.

**Results.** Limit feeding had a profound positive effect on the hip phenotype of Labrador retrievers. Limit-fed dogs had less joint laxity as measured from the hip-extended radiograph and significantly lower incidence and severity of CHD and OA compared with full-fed pair-mates. This health benefit continued for the life of the dogs. In the pooled sample of 48 dogs, the prevalence of radiographic hip OA increased linearly throughout the study, from 15% at 2 years of age to 67% at end-of-life. Focusing on dietary effects, end-of-life OA prevalence for the full-fed dogs was 83% and for the limit-fed dogs, 50%. At 2 years of age, OFA-type scoring found 19 of the 48 dogs in the study to be “dysplastic” while 29 dogs were scored as “normal.” The 19 dysplastic dogs remained dysplastic for life, with OFA scores increasing in severity for many of the dogs. However, of the 29 dogs scored “normal” at 2 years of age, 16 (55%) were scored dysplastic by end-of-life, representing a high false-negative rate of diagnosis. PennHIP results showed that all the dogs included in this investigation had a DI greater than 0.36 (range 0.36–0.92), indicating that all were susceptible to OA. Kaplan-Meier curves of disease-free interval showed that for dogs with a low DI (better hips) the onset of OA was much later in life than in dogs with a large DI. For dogs with DI ≤0.4 the median disease-free interval was 12 years of age compared with dogs with DI >0.6, whose median disease-free interval was only 3 years of age.

Results of histopathologic evaluation were available on 45 dogs in the study. Forty-three of the 45 dogs were found to have histopathologic evidence of OA at death (median life span 12.1 years). Of the 29 dogs scored OFA “normal” at 2 years of age, 92% developed histopathologic evidence of OA by the end of the study. PennHIP DI at 2 years of age predicted that all dogs in the study were susceptible to developing OA. By the end of the study 96% of the dogs showed histopathologic evidence of OA (DI positive predictive value = 96%). None of the 48 Labrador retrievers had tight hips, DI<0.3, and therefore a negative predictive value could not be calculated from the results of the investigation.

**Discussion.** Results of this lifelong study proved that by keeping dogs lean the onset of OA was delayed and its severity and prevalence was reduced significantly. In addition, OA prevalence in other joints of the lean dogs was decreased. The difference in OA prevalence equated to greater comfort for the restricted-fed dogs: mean therapy-free interval for control-fed dogs was 10.3 years while mean therapy-free interval for the restricted-fed dogs was 13.3 years. Such findings are both statistically and clinically significant. The dogs in this study derived from lines of dogs with hip dysplasia and therefore there was a high probability to express OA. Dogs that were permitted to become overweight (mean body condition scores of 6.7) expressed radiographic OA by end of life much more, 83%, than those kept lean (OA 50%, mean body condition score of 4.6). The linear increase in OA incidence over the life of these Labrador retrievers refutes popular dogma that holds that hip OA occurs either early in life, in the case of dysplastic dogs, or much later in the geriatric years in the case of “old age or idiopathic OA.” The principal risk factor, if not the cause, for the development of hip OA has been shown to be joint laxity irrespective of age. In the hip-extended view this laxity is underestimated and often masked completely, leading to false-negative diagnoses. Many dogs appear phenotypically normal although they are genotypically abnormal. Evidence for this impression derives from the continued high frequency of CHD in many breeds of dogs despite systematic mating of presumed normal parents. The lifelong study reported here provides conclusive evidence for this observation. In this sample of Labrador retrievers...
subjective hip phenotypes scored from the conventional hip-extended radiograph were much worse at the end of life than at 2 years of age, with the prevalence of hip OA increasing in a fairly linear manner over time. Subjective assessment of hips at 2 years of age was not an accurate predictor of either radiographic or histopathologic OA at the end of life. In contrast to subjective hip scoring, the PennHIP distraction index, performed when dogs were 2 years of age, indicated that all dogs in this study were susceptible to OA and therefore genotypically abnormal. This predicted susceptibility was borne out by the observed pattern of OA incidence later in life, both radiographically and histopathologically. An ideal screening test for heritable diseases would not be influenced by environmental factors, such as diet. Unlike the Norberg angle and OFA score, the distraction index was not confounded by the effects of the two feeding regimens imposed in this study, suggesting that the DI more accurately reflects the genotype.

**Clinical Significance.** The results of this body of work have profound clinical significance for the practicing veterinarian, orthopedic surgeon, dog breeder, and pet owner: Dogs suspected to be susceptible to CHD should be kept lean for life and dogs selected for breeding should have hip evaluations at regular intervals throughout life. Although the studies were performed in one breed of dog, the Labrador retriever, it is reasonable to consider the results clinically applicable to similar breeds, such as other retriever breeds and even Rottweilers and German Shepherd dog breeds. This opinion finds support from studies that have shown excess body weight to be a risk factor for hip OA irrespective of breed. Owners of dogs of OA-susceptible breeds should strive to keep body condition score between 4 and 5 (on a scale of 1 to 9 where 9 represents extreme obesity). For tight-hipped breeds of dogs known not to be susceptible to hip OA, such as performance greyhounds and Borzois, it is unnecessary to adhere to these dietary recommendations for musculoskeletal reasons. As discovered and reported separately, however, maintaining body condition scores between 4 and 5 was associated with other health benefits beyond the amelioration of OA, including increased longevity and delayed onset of a variety of chronic conditions.

The foregoing four studies demonstrated that controlling body weight delayed, lessened the severity, or prevented the development of OA in some dogs. Consistent with these findings was a parallel benefit in clinical signs within this pool of dogs. Dogs that were kept lean required less pain medication, and required medications starting later in life than dogs in the full-fed group. The mean therapy-free interval (therapy for OA) for the full-fed dogs was 10.3 years, and for the lean dogs, 13.3 years ($P<0.01$). Related evidence to support this experimental finding was published recently showing that reducing body weight and body condition score in obese dogs already showing clinical signs of hip dysplasia resulted in a substantial improvement in clinical lameness and in gait (as demonstrated by force plate testing). These findings add to the growing pool of data in humans showing similar beneficial effects of weight reduction in alleviating the discomfort of OA.

Hip dysplasia has long been understood to be a disease of complex inheritance (so-called polygenic disease). Environmental factors can influence the expression of diseases of complex inheritance and in this investigation, food consumption and body weight were found to be potent factors. Keeping dogs lean certainly did not change the genes of dogs predisposed to hip dysplasia. Rather, leanness was shown to delay or prevent the expression of radiographic signs of CHD in dogs prone to CHD, likely by antagonizing the conversion of passive hip laxity to functional hip laxity that ultimately leads to OA. Some would argue that such environmental manipulation is contraindicated because it “masks” the disease phenotype, thereby confounding diagnosis and related breeding recommendations. Others, however, would counter that for the comfort of the dog, one should invoke all known environmental measures to delay the onset or lessen the severity of CHD. It is conceivable if the dogs in this study were stressed even more (environmentally) or if they had lived longer, that all of them might have expressed OA. Clearly a diagnostic test for hip dysplasia that is not
confounded by environmental factors, such as diet, is desirable. The measure of such a test is embodied in the concept of “heritability” and the higher the estimate of heritability the better. Comparative studies estimating the heritability of hip phenotype have shown the distraction index to have higher heritability than subjective hip scores. In this study the DI was not confounded by the diet and, consistent with observed lifelong hip phenotypes, the DI scored all of the dogs as OA-susceptible. These findings support the previously published figures showing heritability of the DI to be higher than subjective hip score, confirming the DI as a more effective screening tool to make genetic change toward better hip phenotype.

The distraction radiographic procedure was not clinically available until the dogs were 2 years on study; therefore it could not be ascertained whether DI scores at younger ages would have been similarly predictive, although previous research has indicated good reliability of DI from 16 weeks of age. A reliable screening test capable of detecting susceptibility to CHD early in a growing dog’s life would permit the implementation of preventive or palliative measures (eg, dietary, pharmaceutical, or surgical) before the onset of OA.

Longitudinal studies, such as this one, are essential to understanding the true biological behavior of a disease as complex as CHD. To the authors’ knowledge no similar studies in dogs have been published. Of particular interest and importance is the observation that OA prevalence and OFA score changed in a linear fashion long after 2 years of age, the accepted convention for phenotypic expression. Hitherto unappreciated, these studies draw critical attention to the magnitude of change in hip score with aging. Conventional subjective hip scoring at 2 years of age, therefore, underestimates the frequency of hip dysplasia in dogs and this observation alone could explain the recognized slow progress in reducing the frequency and severity of CHD by selective breeding. These new findings emphasize the need for clinical recommendations for hip films well beyond 2 years of age, and in the case of breeding dogs, hips should be evaluated at regular intervals for life.

References

Effects of Restricted Feeding on Onset, Incidence, and Severity of Hip Dysplasia and Osteoarthritis in Dogs


Systemic Arterial Blood Pressure:
The Silent Killer that Never Should Be!

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The prevalence, pathophysiology, and diagnosis of systemic arterial hypertension in dogs remain problematic.
We assume that a blood pressure exceeding 165 mmHg in systole or 135 mmHg in diastole is above
normal. Most animals with hypertension have abnormal renal function, but it is not known whether it is the
cause or the consequence of hypertension. Hyperadrenocorticism is a common cause of hypertension.
Hypertension results in renal (glomerular disease with proteinuria), ocular (retinal hemorrhage, edema and
detachment), and possible cardiac consequences (ventricular hypertrophy). Dogs fed a diet restricted in calories
tend to have lower blood pressure, lower pressure pulse, slower heart rates, and much lower rate-pressure
products—a prime determinant of myocardial oxygen consumption. Caloric restriction also tends to decrease
pulse pressure, indicating a decrease in arterial impedance. Considering extrapolations from experience with
human hypertension and presuming that all dogs would respond as the Labrador retrievers in the Purina Life
Span Study did, caloric reduction in dogs and reduction in arterial pressure and arterial pulse pressure should
translate into decreased morbidity and mortality.

What is Arterial Blood Pressure?
Arterial blood pressure usually refers to systemic arterial blood pressure (SAP), although a similar discussion
may be applied to pulmonary arterial pressure. The SAP is the force responsible for perfusion of blood through
the systemic arterioles and into the systemic capillaries, but it is also a force that hinders the flow of blood
from the left ventricle into and through the systemic arterial tree.\(^1\) It is essential to sustain SAP at levels
elevated enough to perfuse appropriately, but not so elevated as to hinder inappropriately.

How Is Arterial Blood Pressure Expressed?
Systemic arterial pressure is expressed in millimeters of mercury (mmHg). This is the force that tends to
expand the systemic arterial tree when the left ventricle pumps blood into it, but is also the recoil force of the
systemic arterioles as it recoils and pumps blood through the systemic arterioles. There are three well-
known expressions of SAP: systolic, diastolic, and mean. **Systolic SAP** is the greatest pressure recorded from
the systemic arteries, **diastolic SAP** is the lowest pressure recorded, and **mean SAP** is the average pressure.\(^2\) At
normal heart rates mean pressure is usually diastolic pressure plus one third of the pressure difference
between systolic and diastolic. The pulse pressure is the difference between diastolic and systolic pressures.\(^3\)

The maximal rate of rise \((dP/dt_{\text{max}})\) in mmHg/s of SAP from its diastolic value to a value achieved early
during the ejection of blood from the left ventricle into the aorta is also a clinically important expression of SAP.\(^4\)

What Determines Arterial Blood Pressures?
Mean SAP may be understood by applying Ohm’s law \((I = E/R)\) for the flow of current \((I)\) through a wire, the
current being driven by a voltage \((E)\) and hindered by a resistance \((R)\). Comparable designations for the
relationship among cardiac output \((CO)\), the pressure \((\text{mean SAP})\) driving \(CO\), and the systemic vascular
resistance \((svr)\) hindering the \(CO\) is: \(CO = \text{SAP}/svr\).\(^5\) Actually the driving pressure should be the difference in
pressure between the SAP and right atrial pressure (RAP)—the pressure gradient across the systemic arterioles; however, because RAP is normally quite low, it can be neglected in the calculations without producing too much error in the calculations. CO, SAP, and svr for blood flow relate, respectively, to I, E, and R for the flow of electrical current. If \( I = E/R \) and \( CO = SAP/svr \), we can rewrite the equations solving, instead, for E or SAP, as

\[
E = I \mu R
\]

for electricity and

\[
SAP = CO \mu svr
\]

for blood flow. Thus if CO and/or svr are elevated, mean SAP will be elevated.

Systolic SAP is determined by three factors, the volume of blood ejected by the left ventricle during a single beat (termed stroke volume or SV), the pressure in the aorta just before the left ventricle begins to eject (diastolic SAP), and the stiffness of the aorta. These factors are under short-term (neuroendocrine) and long-term (renal) control, the length of the term being a function of how rapidly the SAP responds to the determinants.

Stiffness of the aorta is termed its elasticity modulus \( E \). If all other factors are equal, it is obvious that the greater the stroke volume, the greater the peak systolic SAP. Further it is obvious that if all other factors are constant, the more elevated the diastolic SAP, the greater the systolic SAP. It is less obvious why, if all other factors are constant, the stiffer the aorta, the greater the systolic SAP; this occurs because if a SV is ejected into a stiffer aorta, the pressure increase will be greater. For example, injecting 10 ml of water into a balloon made of hockey-puck material will cause a greater increase in pressure than injecting it into a condom. Thus if SV is great, if diastolic SAP is great, and if the arteries are stiff, then systolic SAP will be elevated. We will discuss further the effects of stiffening of the aorta on both peak SAP and \( dP/dt_{max} \).

Diastolic SAP, if all other factors are constant, is determined by svr, heart rate, and \( E \). It is obvious that the lower the svr, the more rapidly blood will leave the aorta through the systemic arterioles into the capillaries and veins, and the lower the diastolic SAP will be. It is also obvious that the longer the interval between heart beats (ie, the slower the heart rate), the longer the time for blood to exit the aorta and the lower the diastolic SAP will be. Again, the effect of the stiffness of the arterial tree (\( E \)) on diastolic SAP is less obvious. If the systemic arteries are stiffer than normal, they will lack the normal elastic recoil, and it is the elastic recoil of the systemic arteries squeezing on the blood within that tends to sustain SAP. Thus a slow heart rate, a reduced svr, and stiff arteries result in a reduced diastolic SAP.

Because this discussion is principally on elevation of SAP, it follows, then, that a high heart rate, a large SV, and elevated svr will all elevate SAP; while the stiffness of the systemic arteries may elevate systolic SAP, may decrease diastolic SAP, may increase \( dP/dt_{max} \) but may not change mean SAP.

The determinants of each of the determinants of SAP, in the context of how they may elevate SAP, are discussed next.

**What Are the Determinants of the Determinants of SAP?**

If all other factors are held constant, then SV is determined by the force of contraction of the ventricle. Force of contraction can be enhanced by increasing the preload (the volume of blood within the ventricle just before it contracts), or by increasing myocardial contractility (the velocity of cycling of heavy meromyosin heads is responsible for generating either tension or fiber shortening). Preload may be enhanced by increasing blood volume (a balance between water intake and urine production), or by decreasing venous capacity (constriction of venous smooth muscle). Thus preload is under the influence of long-term regulation (water balance), and short-term regulation (venomotion produced by neural activity). Myocardial contractility may be enhanced by an increase in beta-1 adrenergic efferent activity and/or a decrease in parasympathetic (vagal) efferent activity, by enhanced intracellular calcium, or merely by increasing heart rate. Heart rate is accelerated by either decreasing parasympathetic or increasing sympathetic efferent traffic to the SA node, by stretching the atria, or by increasing temperature of the SA node. These constitute rather short-term control.
Both svr and E are determined principally by the degree of constriction or relaxation of smooth muscle within the walls of the vessels. This smooth muscle is under control of neural factors (alpha-1 adrenergic and usually alpha-2 adrenergic constrict, beta-2 adrenergic dilates), endocrine factors (angiotensin–II and anti-diuretic hormone [ADH] constrict, atrionatriuretic factor dilates), local factors (adenosine and nitric oxide dilate, and endothelin constrict, some prostaglandins dilate and some constrict), and oxygen debt, which causes systemic vessels to dilate but pulmonary vessels to constrict. In addition, loading of vessels with fluid may cause them to physically restrict flow, and loading smooth muscle with sodium or calcium may cause them to constrict.

Venous capacity is under the same general control as svr and E. Actions that decrease venous capacity move more blood back to the heart, increase its preload, increase force of contraction, and increase SV. Increasing water intake and decreasing urine production may increase blood volume, increase venous return to the heart, and increase force of contraction.

What Are Causes of Hypertension?

Most hypertension in dogs and cats is probably secondary to diseases of other organ systems—in particular to hyperadrenocorticism. Primary or essential hypertension occurs far less commonly than in humans. Secondary hypertension is observed in dogs and cats with renal disease, in cats with hyperthyroidism, and in dogs with hyperadrenocorticism. Rarely hypertension may be caused by a pheochromocytoma, a catecholamine-secreting tumor of the adrenal.

What Are Normal Values for SAP?

Whereas the literature is replete with references to values for SAP of male and female humans of varying ages, ethnic groups, and conditions, normal values for blood pressure are not known for dogs or cats. A plethora of studies have provided highly variable values, the variations arising from methods (e.g., instrumentation, lateral or end-on pressure, vessel interrogated, number of replications), the restraint (chemical or manual), and the degree of training of the investigator and/or the subject. Systolic SAP for putatively "normal" animals has been reported to vary between 128 and 200 mmHg for dogs, and between 113 and 143 mmHg for cats; diastolic SAP has been reported to vary between 74 and 104 mmHg for dogs, and between 65 and 85 mmHg for cats; and mean SAP has been reported to vary between 90 and 120 mmHg for dogs, and between 87 and 108 mmHg for cats. Standard deviations of these pressures are often as high as 20 to 40 mmHg; therefore these values must be added to the values to include more than 30% of normal animals. This means that systolic, diastolic, and mean SAPs may be, normally, as high as 224, 123, and 145 mmHg, respectively. During these measurements, heart rate—a major determinant of SAP—varied between 90 and 125 beats/minute, while heart rate for quiet resting dogs is usually less than 80, and often less than 60, beats/minute.

No upper limits for SAPs have been established for either dogs or cats. However, many veterinarians use a systolic SAP of greater than 160 to 170 mmHg, and a diastolic SAP of greater than 115 to 120 mmHg as above normal limits for dogs. For cats, systolic SAP of greater than 165 mmHg and a diastolic SAP of greater than 115 mmHg have been used as above normal limits. It must be emphasized however, that there is no consensus for upper limits of normal SAP in dogs or cats. Most agree that a systolic SAP in either species of greater than 180 and a diastolic SAP of greater than 140 mmHg should be considered, at best, above upper limits of normal.

Systemic arterial pressure varies according to where it is measured. A pressure recording from the aorta near the aortic valves may be 120/80, with a mean of approximately 90 mmHg; but a pressure measured near the end of the arterial tree may be 140/60, with a mean of 85 mmHg. Thus there is an average SAP of
approximately 5 mmHg (90–85) driving blood from the aorta proximal to the left ventricle to the distal portion of the arterial tree; however, systolic SAP is greater, and diastolic SAP is lower, in the distal vessel than in the proximal. This might imply that blood flows in systole from the peripheral artery toward the heart, but complicated wave mechanics beyond the scope of this review contradict that intuition.

**How Is SAP Measured?**

The "gold standard" for measuring SAP is to place a hypodermic needle with a closed tip and many side-holes into a major artery, and to attach the open end of the needle to a transducer which converts pressure to an electrical signal that is recorded. This technique is termed **direct manometry**. If pressure is measured through an open tip needle, the pressure will be the sum of the pressure distending the artery plus that imposed by the kinetic energy of the blood flowing toward the open tip. The pressure recorded through a needle with a closed tip but many side-holes is termed the **lateral pressure**, and probably reflects more faithfully the force that the left ventricle must overcome and the force that favors rupture of vessels. The problems with inserting a needle into an artery are numerous: it may injure the artery, it may cause pain, blood may seep out of the artery and produce a large hematoma, the patient must be vigorously restrained or well trained, it requires great skill to puncture an artery, and arteries on cats or small dogs are extremely small.

Most measurements of SAP are made by indirect sphygmomanometry, with a blood pressure cuff like that used most commonly for measuring SAP in humans. With this method a pneumatic cuff is placed around a limb (leg or tail in a dog or cat) containing a large artery. The pressure within the cuff is measured by a mercury or an aneroid manometer attached to the cuff. A stethoscope chest piece or device (Doppler crystal or plastic tubing to detect oscillations of the artery) is placed on the skin over the artery down-stream from the cuff. The devices are used to detect the flow of blood through the artery. The cuff is inflated to a pressure great enough to fully collapse the artery thus prohibiting flow through it. Of course no flow will be detected downstream from the cuff. The pressure within the cuff is decreased continuously but slowly. When the pressure decreases to a level just below the systolic (peak) SAP, a small “puff” of blood will squirt through the artery because arterial pressure exceeds the pressure within the cuff. This little “puff” of blood may be detected by a tapping sound heard with the stethoscope. With some machines, it is detected by a Doppler signal or oscillations produced when the artery pulses towards and away from the emitter/sensor. With other devices, the “puff” is detected when the red blood cells travel towards the emitter/sensor. The pressure recorded within the cuff at this instant is termed the **systolic SAP**. This pressure is determined by reading the manometer attached to the cuff.

As cuff pressure continues to fall, SAP exceeds the pressure within the cuff by longer and longer periods, thus blood will flow through the partially compressed artery for longer and longer periods. This will convert the tapping sound, produced when just a short burst of blood squirts through, to a murmur which is produced by the turbulent blood traversing the partially compressed vessel. The Doppler or oscillometric device will register a greater and greater signal, but will usually plateau when the pressure within the cuff is equal to approximately the mean SAP. Finally as cuff pressure decreases to a level below the diastolic SAP, the cuff no longer compresses the artery, the blood through the uncompressed (normal) artery is no longer disturbed, and the murmur disappears. With the Doppler or oscillometric signal, a new plateau occurs. This occurs at the diastolic SAP.

Unfortunately, SAP measured by indirect sphygmomanometry is an approximation at best, and at worst may be terribly misleading, while direct measurements of blood pressure by arterial puncture may be in error because of pain and restraint.
What Is the Prevalence of Hypertension?

The exact—or even approximate—prevalence of hypertension is not known for dogs or for cats, principally because normal limits are not known and methods of measuring are fraught with error.36 It is estimated that 40% to 90% of cats with non-prerenal azotemia have hypertension, but there is truly no comparable figure for dogs.37,38 It is estimated that 30% to 40% of hypertensive dogs have renal impairment, but it is not known whether the renal impairment caused the hypertension or the hypertension caused the renal impairment. However, if dogs are like humans, in terms of sensitivity to what appears to be trivial elevation of blood pressure, it is highly likely that a significant (truly unknown) number of dogs manifest morbidity and/or mortality because of elevation of SAP. For example if maximal, normal systolic SAP for humans is 140 mmHg, a population exceeding that by only 5 mmHg may have a 5% increase in morbidity. In fact, in humans, the lower the systolic SAP—within limits—the longer the life span of the person. Therefore a systolic SAP of 100 mmHg would carry less morbidity than a systolic SAP of 120 mmHg, even though 120 mmHg may be considered quite acceptable.

When we state that a dog’s life span should be 12 years, we merely state that the typical dog of that breed and sex and with a mean SAP of 135 mmHg—if it is known—dies on average at 12 years of age. As will be suggested later, it is reasonable that life could be prolonged by modifying hundreds of factors that we do not today believe need to be modified. As an example, humans with postprandial blood glucose concentrations of between 60% and 140 mg% are considered normal. Yet humans whose postprandial blood glucose concentrations deviate from 101.5 mg% by less than 1% do not have dental caries. Later it will be shown that by modifying diet and/or SAP, a dog’s life may be prolonged, and its quality of life improved as well.

Why is Hypertension so Bad?

Hypertension is an adverse state for a number of reasons, probably the most important of which are its effects on the heart, eyes, and kidneys.39,40–42 Quite obviously if SAP is elevated, the left ventricle must work harder—actually generate additional wall tension—to eject its stroke volume. This increased tension is given the name increased afterload. Increased afterload initiates a cascade of biochemical and mechanical events leading to hypertrophy (increased muscle mass). Although this helps the heart eject against the increased afterload, it actually results in a decrease in myocardial contractility. Furthermore, the capillaries carrying blood to the hypertrophied wall do not increase in proportion to the increased muscle mass; therefore there may be inadequate delivery of oxygen to the myocardium. To make matters worse, afterload is one of the three major determinants of myocardial oxygen consumption; therefore the myocardium has increased demand for oxygen and less oxygen is delivered. This imbalance between demand and delivery may lead to oxygen debt, and since oxygen is essential for the production of ATP—the source of energy to fuel both contraction and relaxation—both systolic (contractile) and diastolic (relaxation) function may diminish. Whereas this is absolutely known to affect humans with coronary arterial flow restricted partially by arteriosclerosis, the applicability to dogs or cats with arteriosclerosis is less certain.

It is well-known that cats with hypertension may develop retinal edema, hemorrhages, and/or detachment.39,41 The edema and hemorrhages are thought to occur because of elevation of systemic arterial pressure which is transmitted directly to the choroidal capillaries that do not possess the high resistance arterioles possessed by most systemic capillaries. The retinal detachment is thought to occur because of transudation of serum weeping from the choroidal capillaries into the space between the choroid and retina.

The kidney is both a cause of hypertension and a target of hypertension.36,44–46 Renoprival hypertension—elevation of pressure due to reduced renal blood flow and release of renin—has been used as a model to study other features of hypertension, and has been known to produce hypertension in humans. Elevation of pressure
increases transudation across the glomerular capillary membrane, thus producing polyuria and polydypsia. It results in glomerulosclerosis, glomerular atrophy, and loss of nephrons. This results in hyperalbuminuria, damage to the ductal network, and renal failure.

The Role and Possible Impact of Caloric Restriction in Modulation of Systemic Arterial Pressure in Labrador Retrievers

A 14-year-long study in which Labrador retriever dogs were control-fed or restricted-fed, the Purina Life Span Study, was recently completed. The study is described elsewhere in these proceedings and the majority of the findings of this study have been, or will be, reported elsewhere. In this section, the results as they apply to SAP are summarized. The dogs were fed the diets for up to 14 years, and results from year 7 to year 14 are reported here. Plots of mean SAP and heart rate (HR) for the two groups (control-fed and restricted-fed) are shown in Figures 1 and 2. The product of HR and mean SAP for the two groups is shown in Figure 3; the rate-pressure product is a method used to approximate myocardial oxygen consumption because it includes two of the three prime determinants of myocardial oxygen consumption. There are no differences of statistical significance for HR or SAP between the groups, although in every instance but for SAP at year 11 and for HR at years 11 and 13, values for the restricted-fed dogs appeared to be lower than values for control-fed dogs.

Rate-pressure product for the restricted-fed dogs was substantially and significantly lower than for the control-fed dogs at all times except for at year 13. Thus, excluding year 13, it can be assumed that the myocardial oxygen demand was 4% to 23% lower for restricted-fed dogs. This indicates that the demand for oxygen for the restricted-fed dogs should have been substantially lower than for the control-fed dogs for years 7 through 12. Restricted-fed dogs weighed less and had longer median life span than control-fed dogs. Data for year 13 may be less valid because many of the control-fed dogs had died, and those dogs that did survive to year 13 might have had lower SAP for reasons other than their diet regimen.

The average pulse pressure (systolic minus diastolic) over years 7 through 14 for restricted-fed dogs was 57.8 mmHg, whereas the average pulse pressure was 63.2 mmHg for the control-fed dogs over the years 7 through 13. This difference was significant ($P = 0.019$). The pulse pressure is proportional with the relative stiffness of the systemic arterial tree and the stroke volume. Stroke volume was not measured in these dogs, and if we presume that it did not differ between the groups, then differences in arterial stiffness must
Systemic Arterial Blood Pressure: The Silent Killer that Never Should Be!

account for the differences in pulse pressure. The stiffness reflects both degree of smooth muscle tone and the physical properties of the systemic arterial tree. The left ventricles of the restricted-fed dogs would be functioning against a decreased arterial impedance. Both systemic vascular resistance and systemic arterial impedance make up the afterload; therefore both fractions of the afterload appear to be reduced in dogs with the caloric restriction. In humans a reduction in afterload translates to a reduction in myocardial oxygen demand which, if all other factors are equal, translates to a longer life and decreased morbidity.

It is impossible from this study to determine if there is a cause and effect relationship between longevity and the estimate of myocardial oxygen demand. As already mentioned in this article and in other reports of these studies, restricted-fed dogs weighed less and were more active, so it might have been that the lower weight and greater activity prolonged life independent of their effects on the rate-pressure product.

It is known that body weight reduction tends to normalize blood pressure, enhances baroreceptor control of heart rate, and increases natriuresis. This should translate into reduction in heart rate and systemic arterial pressure both because heart rate is decreased and because reduced sodium content of vascular smooth muscle decreases systemic arteriolar tone.

In summary, systemic arterial hypertension in dogs is a disease of unknown etiology, prevalence, and pathophysiology. The percentage of dogs that have essential hypertension (ie, primary hypertension) is probably low, and most appear to have hypertension secondary to renal impairment or hyperadrenocorticism. It is difficult to determine if the renal impairment contributed to or was caused by the hypertension. Body weight reduction by caloric restriction appears to decrease blood pressure, heart rate, and the rate-pressure product—all of which should decrease morbidity and mortality from hypertension.

References


We live to eat and eat to live. When food is plentiful, we store “fuel” as fat. Glucose is absorbed from carbohydrate sources in food and insulin is secreted to allow storage of glucose as fat and glycogen. During periods of starvation, however, our metabolic machinery changes from “storage phase” to “starvation phase.” Insulin secretion is diminished and fat deposits are used as an energy source to conserve glucose for insulin-independent organs such as the brain. Do we have the mechanisms for preventing obesity and its many detrimental effects? Does body composition—lean versus fat mass—make a difference? Is the onset of disease heralded by the onset of perturbations in carbohydrate metabolism? What are the lessons learned from the Purina Life Span Study on the effects of diet restriction on life span and age-related changes? This article addresses the clinical implications of the Purina Life Span Study for today’s veterinary practitioners.

Obesity
Humans are not the only species beset by the problem of obesity. It is estimated that approximately 35% to 40% of cats, 25% to 35% of dogs, and more than 50% of humans are obese.1–3 In humans, and probably in cats, obesity is a precursor to diabetes mellitus (type 2). Obese cats are four times more likely to develop diabetes, five times more likely to develop lameness, and three times more likely to have non-allergic skin conditions compared with cats of optimal body condition.4 To say that obesity in the pet population is a problem is a gross understatement. Until only recently, however, veterinarians had no real “smoking gun” to convince owners to watch their pet’s weight.

The Set Point
Studies have shown that weight loss and neuroendocrine mechanisms revolve around a body set point. Perturbations from the “normal” set point, which may be genetically determined, result in weight gain and redistribution of body mass (lean versus fat). This set point can be altered by changes in diet caloric content and composition (protein versus carbohydrate). Studies in humans have shown that subjects consuming a high-glycemic index diet for 9 days have higher levels of serum leptin, a greater decrease in resting energy expenditure (REE), and more negative nitrogen balance than those consuming a low-glycemic index diet.5

Obese diabetic cats fed a restricted carbohydrate diet lost body fat (based on dual energy X-ray absorptiometry [DEXA]), gained lean body mass (DEXA), became normoglycemic, and lost insulin dependence.6 Overweight dogs fed 20% calories from protein lost twice as much lean body mass as overweight dogs fed 30% or 39% calories from protein.6 Obese cats fed 35% metabolizable energy as protein showed weight loss consisting of 20% as lean body mass (LBM) and 79% as fat; however, obese cats fed 45% energy as protein lost only 11% as LBM and 88% as fat.7 Clearly, shifting to a diet which is lower in carbohydrates, lower in glycemic index, and higher in protein has a beneficial effect on metabolism leading to maintenance of lean...
body mass and loss of body fat. Finally, as shown in the Purina Life Span Study, dogs fed on a restricted calorie regimen had a lower percentage of body fat, improvements in carbohydrate metabolism, and significantly longer life span than their full-fed litter-mates.8

**Perturbations in Carbohydrate Metabolism**

Some of the more fascinating findings in the Purina Life Span Study were the perturbations in carbohydrate metabolism noted in the control-fed dogs. This group of dogs was fed ad libitum until 3.25 years of age, and thereafter received 62.1 kcal/kg estimated ideal body weight for the remainder of life. The control-fed dogs exhibited a reduction in insulin sensitivity that was highly correlated (0.8) with fat mass. Age-associated increases in fat mass (especially visceral fat) suppress hepatic glucose metabolism leading to decreased insulin sensitivity in rats,9 humans,10 and cats.11 In the Purina Life Span Study, lower basal glucose levels and higher insulin sensitivities were seen in the restricted-fed dogs and were highly correlated with survival; furthermore, the age at which 50% of the dogs were treated for a chronic condition (such as osteoarthritis) was increased by nearly 20% (9.94 years for control-feds, 12.0 years for restricted-fed, \( P < 0.01 \)).8

The question is: Why was altered carbohydrate metabolism the only predictor of onset of chronic disease and survival? The answer to that question may lie with the composition of the diet. Although restricted-fed dogs consumed an average of 22.5% fewer calories throughout the study, the calculated proportion of reduction in calories consisted of approximate 50% from carbohydrates, but only 25% each from protein and fat. Could it be that restriction of carbohydrate, rather than protein or fat, had a significant effect on insulin and glucose metabolism? Certainly, others have observed similar effects in cats with type 2 diabetes mellitus fed low carbohydrate diets.5,12

**Clinical Implications**

One of the problems associated with obesity management is the lack of an objective, practical method for accurately assessing body composition (LBM and percent body fat). The most useful research tool for assessment of body composition is DEXA. Most veterinarians, however, do not have access to such tools. Therefore, body condition score systems have been developed for both cats and dogs and correlated with laboratory methods such as DEXA.13,14 The nine-point system developed by Laflamme provides a simple, repeatable method of assessing body composition in small animals [Figures 1, 2, and 3].13,14 (See Appendix 1 at end of these proceedings for Nestlé Purina’s canine and feline body condition score charts.) Another method of assessing body fat in cats is through the use of the feline body mass index. Percentage body fat can be calculated by the following formula:

\[
\text{Percentage body fat} = \frac{(\text{RCC (cm)})/0.7062 - \text{LIM (cm)}}{0.9156} - \text{LIM}
\]

where RCC = rib cage circumference and LIM = lower hind limb measurement.

One of the easiest and most practical methods is to take a digital or Polaroid photograph (head on and profile view) of the dog or cat on a yearly basis. Photographic images can be imported into the animal’s medical record thus providing serial images of body condition over the years. Body weight and body condition scores should be recorded on a yearly, if not quarterly, basis. Dogs and cats with a heavy haircoat should be palpated carefully to assess changes in body condition, which may not be visually apparent.

Changes in insulin, glucose, and thyroid metabolism were evident in the Purina Life Span Study.8 Restricted-fed dogs had lower fasted serum insulin concentrations and lesser area under the curve (AUC) for...
glucose after an intravenous glucose tolerance test (IVGTT) than control-fed dogs. From a practical standpoint, IVGTTs are not easily performed in a practice setting for routine annual examinations. However, a serial record of annual fasting blood glucose values for an individual animal may be very illuminating. Particularly in cats, because the risk of type 2 diabetes is so great, annual fasting and postprandial blood glucose values might be of assistance in identifying early type 2 diabetic cats. Dietary intervention at this point could prevent the development of insulin dependence.

Another parameter that might be beneficial, particularly in cats, is serum fructosamine. Serum fructosamine is composed of albumin and other serum proteins which have become glycosylated by a nonenzymatic process; chronic hyperglycemia will accelerate the process of glycosylation. Increased serum fructosamine values may presage the onset of overt diabetes mellitus in cats. Again, serial fructosamine values on an annual basis may provide a more accurate reflection of normal versus abnormal for a particular patient.

Changes in serum triiodothyronine (T3) concentrations were also noted in the Purina Life Span Study; in fact, higher serum T3 concentrations were evident in the control-fed group. Higher serum T3 has been shown to be a consequence of over-feeding because of an increase in deiodination of thyroxine (T4) to T3. In contrast, carbohydrate restriction and weight reduction cause a decrease in serum T3 concentrations. This brings up the question of whether serial measurements of serum T3 and T4 might be beneficial in monitoring and prevention of obesity in pet animals.

The problem with this strategy, particularly in dogs, is the tendency to over-diagnose thyroid disorders. Thyroid metabolism is complex and the finding of low serum T4 in an otherwise healthy animal may not be of much importance. In dogs, if thyroid hormone is low, endogenous thyroid-stimulating hormone (eTSH) concentrations should be measured. The findings of a high serum eTSH combined with a low T4 will ensure against the misdiagnosis of hypothyroidism. Serial measurement of T4 and T3, while not detrimental, probably do not provide any additional information for the management and prevention of obesity. Obviously, hypothyroid dogs should have these parameters measured on an annual basis.

**Strategies to Cope with Obesity**

In a recent article examining factors involved in feline obesity, the top four main factors contributing to body condition were neuter status, age, frequency of treat feeding, and ad libitum feeding. Because neutering has health benefits and prevents pet overpopulation, it seems unlikely that current recommendations for neutering will change. Age cannot be eliminated as a factor, although increased vigilance in middle age seems appropriate as this age group is most at risk for obesity.

Treat feeding is a source of calories and a known contributor to obesity in small animals. If animals are fed a diet that satisfies them, or treats that satisfy their hunger, overeating will become less of an issue. In cats and dogs, owners should provide a food with adequate protein for maintenance of lean body mass and in the case of cats, enough fat to provide satiety. In dogs, higher protein levels will maintain body mass, even during calorie restriction for weight loss. In cats, the author has had success in reducing body fat in both
diabetic and nondiabetic cats using a high protein, low carbohydrate canned formulation such as Purina Veterinary Diets DM Diabetes Management brand Feline Formula® (Nestlé Purina PetCare Company, St. Louis, MO).  

Just as in humans, portion size should be regulated. A true measuring cup of dry food is a small amount. Clients should not use a jumbo sized cup or giant scoop to measure dry food amounts. Clients need to realize how much food they are feeding—a “Big Gulp” cup contains about four actual cups of food. Portions may be more accurately assessed using canned formulations or by weighing the food. In the author’s experience, cats are more satisfied with foods that are higher in fat and protein and lower in carbohydrate content. This “satiety” factor seems to help limit over-eating in this species.

The most obvious change that veterinarians can promote is a change in feeding regimens and the easiest change to implement is to eliminate free-choice or ad libitum feeding. Ad libitum feeding (which is not available in nature, by the way) has been shown to increase the incidence of osteoarthritis in young growing dogs and increase obesity in cats.  

Veterinarians can now cite the Purina Life Span Study which showed that controlling food and calorie intake to maintain a lean body condition has significant positive impacts—namely, an increase in life span!

Conclusions

If you told your clients there was a way to prolong their dog or cat’s life by 15% would they listen to you? The answer is a resounding “yes.” After all, it’s been shown to happen in dogs in the Purina Life Span Study. It is up to us, as veterinary professionals, to bring the enlightening information of the Purina Life Span Study to the pet-owning public.

References

The goal of many of the diets, nutritional supplements, and other health aids available today is to prolong life span. The only treatment, however, that has been repeatedly and scientifically proven to increase life span is calorie restriction. Calorie restriction has been shown to increase life span in a variety of species and, as long as other nutrients are not deficient, good health is maintained. Recently, the evidence for benefits of calorie restriction has also been extended to dogs.

A recently published paper reported the results of Nestlé Purina’s 14-year-long Life Span Study. In this study, researchers divided 48 8-week-old Labrador retriever puppies into two groups. The control group was fed free choice (initially) and then controlled amounts from 3.25 years of age on (to avoid obesity). The restricted-fed group was fed 25% less food than the control group throughout the study. All 48 dogs were followed throughout their life. Dogs in the control group were not obese but were slightly overweight on average. A standard body condition score (see Appendix 1) from 1 to 9, where 1 is emaciated, 5 is ideal, and 9 is grossly obese, was used. In this study, control dogs had an average body condition score between 6 and 7. Dogs in the 25% restriction group maintained an average body condition score between 4 and 5.

Several papers have already shown orthopedic benefits of calorie restriction from this same study. The new information from this study, however, is that the dogs in the 25% restriction group also lived longer. The median life span (the age when half the dogs were deceased) was nearly 2 years longer in the 25% restriction group. Other health benefits from calorie restriction are also being studied. The results of this study support careful weight management throughout life for dogs and may also extend to cats.

Preventing Obesity in Dogs and Cats

Two issues to address are how to prevent dogs and cats from becoming overweight and how to get an already overweight pet to lose weight. For puppies and kittens, it is important to select a good quality commercial food that has been shown to meet the nutritional requirements of puppies through feeding trials. Most puppies that will have a mature body weight over 60 pounds should be fed a diet designed for large-breed puppies. Feeding directions are required on pet food labels but the quality of the information varies greatly. Some feeding directions are very sketchy while others are quite detailed. Also, some feeding directions vastly overestimate the amount of food a puppy or kitten needs. Therefore, the best method is to calculate the exact resting energy requirements (RER) and maintenance energy requirements (MER), which give an estimate of the total number of calories required/day:

$$\text{RER} = 70 \times (\text{BW in kg})^{0.75}$$

<table>
<thead>
<tr>
<th>Dogs:</th>
<th>Cats:</th>
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<tr>
<td>$\text{MER} = 3 \times \text{RER} (&lt;4 \text{ months of age})$</td>
<td>$\text{MER} = 2.0-2.5 \times \text{RER} \text{ (growth)}$</td>
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<tr>
<td>$2 \times \text{RER} (4-12 \text{ months of age})$</td>
<td>$1.4 \times \text{RER} \text{ (intact adult)}$</td>
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<tr>
<td>$1.6 \times \text{RER} \text{ (neutered adult)}$</td>
<td>$1.0 \times \text{RER} \text{ (obesity-prone adult)}$</td>
</tr>
<tr>
<td>$1.0-1.4 \times \text{RER} \text{ (neutered adult)}$</td>
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Computer programs are available from various pet food companies to help with these calculations but the activity factors used vary between the companies so the usefulness of these programs also varies. Once the MER is calculated, this number of calories is then divided by the caloric content of the pet food selected to determine the number of cups or cans required per day:

\[
\text{Calories/day ÷ calories per can or cup} = \text{cans or cups/day}
\]

This amount should be divided into at least two meals per day. The number of calories in a particular pet food can be obtained from product guides, from direct inquiries to the manufacturer, or for some foods, from the label. The pet food label is now allowed to contain the calorie information but it is still not required for most pet foods, so most foods designed for growth still do not list this information. Pet foods that are "light," "lite," "low calorie," or "reduced calorie," however, must include a calorie content statement on the label.

The owner should be instructed to measure the food exactly at each meal to prevent overfeeding. The most important thing to remember, however, is that this is just a starting point in terms of food quantity. The veterinarian and the owner must monitor the individual puppy or kitten's response. Owners of new puppies and kittens should be taught what optimal body condition looks and feels like, and how to assess body condition on a regular basis. Owners should look at the pet but also feel the pet to assess body condition, particularly in long-haired animals (Figure 1). Just like people, dogs and cats vary tremendously in how easily they gain weight. If it is becoming difficult to feel the ribs or the animal is losing the "waist" behind their ribs, they are becoming too heavy (Figure 2). To keep a puppy or kitten trim throughout the growth period, the food will need to be adjusted during growth spurts and plateaus to maintain a trim body condition (between 4 and 5 on a 1 to 9 scale). Most puppies and many kittens will require meal feeding (as opposed to free-choice feeding) to achieve optimal body condition. Be sure to talk to the owner about treats and table food.

If the caloric content of treats and table food are not accounted for in the daily intake of the pet, they can contribute to obesity.

Weight Reduction Programs for Dogs and Cats

The other issue is reducing weight in a dog or cat that is already overweight (Figure 3). Obesity is the most common nutritional problem in dogs and cats today. Predisposing factors include the availability of highly palatable commercial pet foods, free-choice feeding, lack of exercise, and neutering. Benefits of weight reduction have been shown for obese pets, particularly with respect to orthopedic disease. In one study, dogs with lameness secondary to hip osteoarthritis and that were at least 10% over ideal body weight were put on weight reduction diets. Dogs that completed the study lost between 11% and 18% of their body weight and had significant reductions in hind limb lameness.
The failure rate for weight reduction programs in people is over 90%, which is likely similar to the lack of success seen in many dogs and cats. Animals with health conditions such as orthopedic disease, cardiopulmonary disease, or intervertebral disk disease (Figure 4) usually provide the owner with greater incentive to comply with their pet’s weight loss program. The key to successful weight reduction is reducing the total number of calories the dog or cat is eating, increasing exercise (if possible), and changing the behavior of both the animal and the owner. Of greatest importance is a thorough diet history. Most owners will easily admit to the type of pet food and the amount, but it is often more difficult to get a complete story on treats, table food, and other sources of calories without asking very specific questions about these areas. A list of questions to ask owners includes:

- What type of pet food and how much is fed per day?
- Is the owner measuring the food daily (using an 8-oz. measuring cup)?
- How many times per day is the pet fed?
- How many pet treats are given daily?
- What types of “people food” are fed daily (including quantities)?
- Are treats or “people food” fed regularly or in response to begging?
- When does the pet beg for food (eg, at dinnertime, while cooking)?
- Who feeds the pet? Is there more than one person in charge of feeding?
- Does the pet have access to other pets’ food (either dog or cat)?
- Is the pet ever outside unsupervised?
- Are there children or other people in the house that can provide food to the pet?
- What type of exercise does the pet do (and how much)?

Once these questions are answered, a plan can be made that will help avoid the problems encountered with the individual owner/pet. This plan must control the quantity and type of pet food, restrict treats and table food, limit access to all other sources of calories (eg, children, other pets, neighbors, grandparents, mailmen, dog walkers), and provide exercise. First, an initial goal weight should be selected. This should be a reasonable goal for the owner (eg, if a 90-pound dog needs to lose 30 pounds, a reasonable initial goal might be to lose 15 pounds). Owners can easily get discouraged if the initial goal is unreasonable. Also, if they are successful in reaching the initial goal, they are much more willing to continue. If the absolute number of calories currently being eaten by the pet can be determined, a reduction of approximately 25% can be used to calculate a daily calorie requirement for weight loss. Usually, however, exact calorie intake cannot be confirmed so a starting point for daily calorie intake must be calculated. Caloric requirements to reach the
initial weight goal can be calculated using the equation for RER given earlier. I use the RER for the initial goal weight as the daily calorie requirement (i.e., I do not use an activity factor to calculate a MER). A 1% to 2% rate of weight loss per week generally is recommended.7

The total number of calories required per day is then divided by the caloric content of the pet food selected to determine the number of cups or cans required per day, as discussed earlier. The total amount of food per day should be divided into at least two meals per day. The owner must be instructed to measure the foods exactly at each meal to prevent overfeeding. It is recommended to select a diet that is reduced in calories compared with regular diets. Reduced-calorie commercial dog and cat foods vary tremendously in the number of calories they contain and their other nutrient levels so care should be taken to avoid the broad recommendation to feed a “reduced-calorie food.” Reduced-calorie diets may or may not be increased in fiber. Studies have produced conflicting results on the satiety effect of fiber in weight loss programs8–10. Reducing the number of calories eaten requires controlling the treats and table food as well as the pet food. Some owners are able to completely discontinue treats and table food but most will need recommendations for acceptable treats. For dogs, these can include raw or cooked non-starchy vegetables (e.g., carrots, green beans, zucchini, green peppers) and air-popped (not microwave) unbuttered popcorn. If the owner feels strongly about feeding pet treats, a specific number per day should be recommended and the calories provided by the treats should be subtracted from the amount of pet food recommended.

Monitoring is the most important part of a successful weight reduction program. It is critical to have the pet come in to be weighed two weeks after beginning the new diet program. If weight has not changed (or has increased), the owner should first be questioned about possible noncompliance. If compliance is not an issue, then the total amount of pet food should be decreased further. “Weigh-ins” should be done every two weeks until slow, steady weight loss is achieved and then at monthly intervals thereafter until the goal weight and body condition (4 to 5; Figure 5) are achieved.

Figure 5. An adult dog with ideal body condition (4 to 5). The ribs are easily palpable, the dog has a “waist” when viewed from above, and the abdomen is “tucked up” when viewed from the side.

References

**Body Condition System**

1. Ribs, lumbar vertebrae, pelvic bones and all bony prominences evident from a distance. No discernible body fat. Obvious loss of muscle mass.


3. Ribs easily palpated and may be visible with no palpable fat. Tops of lumbar vertebrae visible. Pelvic bones becoming prominent. Obvious waist and abdominal tuck.

4. Ribs easily palpable, with minimal fat covering. Waist easily noted, viewed from above. Abdominal tuck evident.

5. Ribs palpable without excess fat covering. Waist observed behind ribs when viewed from above. Abdomen tucked up when viewed from side.

6. Ribs palpable with slight excess fat covering. Waist is discernible viewed from above but is not prominent. Abdominal tuck apparent.

7. Ribs palpable with difficulty; heavy fat cover. Noticeable fat deposits over lumbar area and base of tail. Waist absent or barely visible. Abdominal tuck may be present.

8. Ribs not palpable under very heavy fat cover, or palpable only with significant pressure. Heavy fat deposits over lumbar area and base of tail. Waist absent. No abdominal tuck. Obvious abdominal distention may be present.


The BODY CONDITION SYSTEM was developed at the Nestlé Purina Pet Care Center and has been validated as documented in the following publications:


LeFemme DP. Development and Validation of a Body Condition Score System for Dogs. Canine Practice July/August 1997; 22:10-15

Kasey, et. al. Effects of Diet Restriction on Life Span and Age-Related Changes in Dogs. JAVMA 2002; 220:1315-1320

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1. Ribs visible on shorthaired cats; no palpable fat; severe abdominal tuck; lumbar vertebrae and wings of ilia easily palpated.

2. Ribs easily visible on shorthaired cats; lumbar vertebrae obvious with minimal muscle mass; pronounced abdominal tuck; no palpable fat.

3. Ribs easily palpable with minimal fat covering; lumbar vertebrae obvious; obvious waist behind ribs; minimal abdominal fat.

4. Ribs palpable with minimal fat covering; noticeable waist behind ribs; slight abdominal tuck; abdominal fat pad absent.

5. Well-proportioned; observe waist behind ribs; ribs palpable with slight fat covering; abdominal fat pad minimal.

6. Ribs palpable with slight excess fat covering; waist and abdominal fat pad distinguishable but not obvious; abdominal tuck absent.

7. Ribs not easily palpated with moderate fat covering; waist poorly discernible; obvious rounding of abdomen; moderate abdominal fat pad.

8. Ribs not palpable with excess fat covering; waist absent; obvious rounding of abdomen with prominent abdominal fat pad; fat deposits present over lumbar area.

9. Ribs not palpable under heavy fat cover; heavy fat deposits over lumbar area, face and limbs; distention of abdomen with no waist; extensive abdominal fat deposits.

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