Skeletal diseases may be induced by malnutrition that involves certain nutrients, notably calcium, phosphorus, and vitamin D, but also iodine, copper, zinc, manganese, fluorine, silicon, vitamin A and perhaps vitamin C, as well as protein and energy. The number of these nutrients and their interactions is so large that dietary intervention in bone abnormalities is likely to be most effective if directed at consumption of a diet that is completely balanced for growth, maturity, old age, or stress. Substitution of a good diet for a bad one is also likely to be more convenient and safer than most attempts at partial supplementation of the faulty diet in order to bring it into balance.

Underfeeding a good diet decreases size-for-age of bones during growth. Total body calcium per unit body weight is increased, however, and cortical thickness of long bones is also increased, apparently as a result of a lower rate of endosteal resorption. Bone strength as well as density may also be increased by underfeeding during growth. On the other hand, underfeeding may engender osteoporosis in older animals. Starvation induces calciuria in most species, especially humans, but to an almost negligible extent in dogs and, presumably, cats. Carbohydrate-deficient diets also induce calciuria at the expense of bone in susceptible species, again excluding dogs and cats, since these species appear to have no essential dietary requirement for carbohydrate (excepting the bitch toward the end of pregnancy).

Overfeeding a balanced diet induces rapid growth in the young and obesity in adults. Obesity places an unnecessary burden upon the skeleton that is potentially damaging, especially to joints that are already imperfect. The possible contribution of excessive weight-for-age to skeletal abnormalities during growth has attracted much attention. Unfortunately, much of the hue-and-cry about overfeeding large breeds of dogs has been based on experiments that had major limitations in designer Thus, the hypothesis that a diet perfectly balanced for growth may induce skeletal abnormalities when fed ad libitum during growth remains in need of rigorous investigation.
Confusion has arisen regarding the relationship between diet and feeding management. Some investigators have assumed that a sound diet should be designed to be fed ad libitum. My view is that any optimal diet for a dog or cat is likely to be very palatable; hence, it should never be fed ad libitum; instead, it should be fed deliberately by hand and eye to achieve a desired rate of growth.

PHYSIOLOGIC NUTRITION OF BONE

Bone cells elaborate a characteristic crystal structure around a framework of fibrous protein abundant in elastin and especially collagen. This protein is rich in glycine, lysine, and proline. The synthetic hydroxylase enzymes require ferrous iron and ascorbic acid. Impaired collagen synthesis also occurs during deficiencies of vitamin D and E and zinc.

The organic intercellular matrix of bone is rich in mucopolysaccharides. Their synthesis involves enzymes that require manganese, zinc, and vitamin A.

Minerals comprise about 15% to 20% of bone weight on a moist basis or 60% to 70% on a dry basis. Bone mineral is largely hydroxyapatite, Ca\(_{10}(PO_4)_6(OH)\_2\). Adventitious metals, such as strontium, radium, and lead, tend to sequester in the crystal interior. Fluoride ions substitute for hydroxyls and increase stability. Moieties of hydroxyapatite exchange with other ions at the crystal surface, such as sodium, potassium, magnesium, chloride, citrate, and carbonate. Silicates may also be essential for optimal bone growth, mineralization, crystalline structure, and strength.

REQUIREMENTS FOR CALCIUM, PHOSPHORUS, AND VITAMIN D

Homeostatic regulation of the interrelated calcium, phosphorus, and vitamin D systems allows wide optimal ranges of dietary contents or daily intakes. The interactions become important only when one or more of these nutrients is marginal, low or high. The National Research Council (NRC) has recommended dietary contents of 1.1% calcium, 0.9% phosphorus, and 500 IU/kg vitamin D for dogs, and 1% calcium, 0.8% phosphorus, and 1000 IU/kg vitamin D for cats.

The NRC also gives a second estimate for the calcium requirements of dogs: 242 mg/kg body weight/day for maintenance, and 484 mg/kg/day for growth. The two estimates for growth are compared in Table 58-1. The estimate based on body weight equals the theoretically better estimate based on diet fed as a function of metabolic body size (the three quarter power of body weight) only when body weight is 5 kg. Otherwise the estimate of calcium requirement based directly on body weight understimates the better estimate by 21% for a 2-kg pup and overestimates by 19% for a 10-kg pup.

The above estimates of requirements are reasonably sound, but the range of calcium requirement is affected profoundly by several intrinsic and extrinsic factors that are poorly quantitated, notably the animal's ability to alter the efficiency of calcium absorption.
absorption and the effects of certain dietary components on bioavailability of calcium.

When calcium carbonate is added to a highly digestible, low-calcium diet, the dog controls the efficiency of absorption of calcium in such a way as to regulate the amount of calcium absorbed. In such a study of mongrel pups gaining about 40 g body weight/day, calcium retentions were 90%, 46%, and 27% when calcium intakes were 0.63g, 1.62g and 2.67 g/day, respectively, corresponding to dietary calcium contents of 0.11%, 0.63%, and 1.23%. Dietary phosphorus was 0.39%. No pathologic lesions attributable to calcium deficiency were found, despite a calcium/phosphorus ratio of 1:3.5 in the low calcium diet.

In another study, adult beagles were fed a diet containing 0.12% calcium and 1.2% phosphorus, a calcium/phosphorus ratio of 1:10. Progressive loss of alveolar bone became so severe that incisor teeth became detached. The differences between these two studies illustrate the importance of the calcium/phosphorus ratio when calcium is low, the possible influence of age in limiting the efficiency of calcium absorption, and the likely influence of the rest of the diet.

The bioavailability of calcium is affected not only by phosphorus and vitamin D but also by protein, fat, lactose, fiber, and acidity or alkalinity. The efficiency of absorption of phosphorus is affected by concurrent absorption of calcium, hence indirectly by all of the above list of factors that affect calcium. Phosphorus present in phytic acid or phytin (inositol hexaphosphate) is unavailable for absorption and depresses absorption of calcium, iron, and zinc.

Staples for both dogs and cats contain sufficient phosphorus but not sufficient calcium: meat contains about 0.02% calcium and 0.4% phosphorus (dry basis), and corn grain contains 0.03% calcium and 0.3% phosphorus. Thus, both need to be supplemented with calcium.

Corn is also deficient in protein. It is commonly supplemented with soybean meal that contains abundant phytin. Calcium absorption is depressed by phytin and food fiber. Therefore, calcium is usually supplemented heavily in corn-soy-based diets. Excessive calcium impairs absorption of zinc and copper and thyroid uptake of iodine. Excessive calcium may also potentiate vitamin D toxicity. Obviously, balancing the minerals in a corn-soybean diet is difficult.

In practice, I formulate predominantly meat-based diets to contain 0.5% to 1% calcium and phosphorus. Corresponding figures for predominantly grain-based diets are 1.5% to 2% calcium and phosphorus. The calcium/phosphorus ratio is usually between 1:1 and 1:0.7, but this is not crucial. Vitamin D content is kept between 500 IU and 1000 IU/kg dry matter.

METABOLIC BONE DISEASE

The term metabolic bone disease was popularized by Albright. For several reasons that will be addressed below, it has become "at best confusing" in veterinary orthopaedics.

The concept categorized bone disease with reference to the metabolic events of formation and resorption:

1. Defective formation of bone
   a. Defective formation of osteoid, osteoporosis
   b. Defective mineralization of osteoid, osteomalacia
2. Excessive resorption, osteodystrophy fibrosa

A general term, osteopenia, denoted any deficiency of calcified bone. The first source of confusion was the persistence of the use of osteoporosis as the general term, synonymous with osteopenia.

Another major source of confusion about metabolic bone disease was the shattering of Albright's key hypothesis that primary osteoporosis in older humans may be due to a primary decrease in osteoblastic formation of osteoid. Applications of sophisticated methods using radioisotopes and intravital stains showed that calcium kinetics are normal on a body weight basis but increased in relation to skeletal mass. Moreover, both the formative and resorptive surfaces are increased, the latter more markedly. These findings indicated that osteoporosis should be regarded as a disorder of remodeling or the coupling that usually exists between formation and resorption, with resorption becoming more active than formation,
regardless of their absolute rates.

Another potential source of confusion lies in temporal changes. For example, the local osteoporosis that follows tendon section involves first an increase in resorption with a slight decrease in formation, followed by an increase in formation with a greater increase in resorption, then a secondary reduction in resorption followed by a fall in formation.(13,23)

Further confusion has arisen from the characterization of metabolic bone disease in terms of distinctive histopathologic characteristics.(17,22) This practice becomes especially contentious when a dynamic series of events is inferred from a single static picture.

In medicine generally, the term metabolic disease is becoming confined to inborn errors of metabolism.

GENETIC BONE DISEASES
Genetic bone diseases include the mucopolysaccharidoses,(15,19) hemophilia,(8) familial osteopetrosis,(39) and several forms of dwarfism.(29) This category may also include multicentric periarticular calcinosis(9) and possibly osteochondrosis.(32)

Nutrition may influence the expression of some of these inherited disorders. The earliest instance is the zinc-responsive dwarfism of malamutes.(5) Other possibilities include osteochondrosis and malresorptive disorders, such as osteopetrosis and hypertrophic osteodystrophy.

ENDOCRINE BONE DISEASE
Osteoporosis develops in the course of several primary endocrine disorders, including pituitary dwarfism, cretinism, and hyperadrenocorticism. Some forms of rickets or osteomalacia involve faulty metabolism of vitamin D, namely, the secosterol hormones. Osteodystrophy fibrosa is a manifestation of hyperparathyroidism.(4) Overmineralization of bone may involve hypercalcitoninism. (17,22)

Iodine deficiency and calcium excess or other goitrogenic factors may contribute to hypothyroidism, calcium deficiency, and, perhaps, phosphorus excess may contribute to hyperparathyroidism; and excesses of calcium and vitamin D may contribute to hypercalcitoninism.

BONE DISEASES RESPONSIVE TO NUTRITIONAL INTERVENTION
Under experimental conditions, it is possible to determine the primary event or independent variable, be it genetic, endocrine, nutritional, or metabolic. A natural disease, however, may present multiple perturbations, and questions concerning which event came first or which factor is most important currently may be less relevant to clinical management than the question of feasibility of intervention. Nutrition is most amenable to change and is often the first, perhaps the only, choice for intervention.

Attention will now be given to those bone diseases amenable to nutritional intervention. Emphasis will be placed on nutritional aspects; more details on clinical and pathologic features of these diseases are presented elsewhere,(4,22,29,32,33)

ZINC-RESPONSIVE CHONDRODYSPLASIA
Zinc-responsive chondrodysplasia is a form of dwarfism seen most commonly in Alaskan malamutes, but it may occur also in other northern breeds. The legs, especially the forelimbs, become short and bowed. Elevations in serum alkaline phosphatase activity and urinary acid mucopolysaccharide concentration suggest defective bone maturation.(5) Affected dogs have mild normochromic, hemolytic anemia and delayed sexual maturity. A high percentage of acrosomal defects in spermatozoa was corrected in 2 months by daily oral administration of 45 mg zinc.(5)

Plasma levels of $^{65}$Zn following its oral administration were reduced to one fourth normal levels in affected dogs.(5) In vitro studies indicated that radioactive zinc entered intestinal mucosal cells, bound to a protein, then failed to release to a nonprotein fraction for transport into blood.(5) Affected dogs need to be supplemented with dietary zinc throughout life. The NRC recommends 0.11 mg zinc/kg body weight for an adult and twice this level during growth.(31) Thus, a 20-kg malamute
pup or a 40kg adult needs 4.4 mg zinc/day. The 65Zn absorption studies indicate a need for four times the normal zinc intake, giving 17.6 mg zinc in this case. About twice this amount, 35 mg zinc, is needed if the diet contains abundant phytin or calcium, as in most dry dog foods based on corn grain and soybean meal, since phytin binds zinc, and calcium impairs its absorption. About 35 mg zinc is provided by 150 mg of zinc sulfate or 250 mg of zinc gluconate. Clinicians have prescribed 200 mg to 300 mg of these salts daily without observing adverse effects. The first signs of overdosage of zinc are vomiting and anorexia. Humans report nausea and abdominal cramps. Other symptoms may include diarrhea and fever. Critical lesions, such as gastric ulcers and pancreatitis, occur at sites of higher concentrations of zinc in tissues.

RICKETS
In most species, rickets is usually due to inadequate dietary phosphorus or vitamin D or to lack of sunlight. It is also usually regarded as a juvenile form of osteomalacia. This kind of rickets may occur rarely in dogs and cats suffering from malabsorption of vitamin D due to excessive chronic administration of mineral oil.

One of the most dramatic diagnoses among experiences as a student was made as a woman carried a pup into the examination room. She was told to stop at the door. Could we diagnose the condition? No, we replied. "The pup has rickets," we were told. My hands soon felt the rosary around its ribs. The diagnosis had been prompted by the characteristic odor of mineral oil leaking from the owner's digestive tract.

Calcium deficiency might be expected in dogs and cats because the most common staples, meat and grain, contain only 0.01% to 0.03% calcium on a dry-matter basis. Studies on pups that developed rickets when fed a wheat-based diet contributed to the Nobel prize won by Sir Edward Mellanby.(26) More recently attention focused on unsupplemented "all-meat" canned dog foods. Although alleged to be consistently deficient in calcium,(7,28) the meat byproducts in these cans included much bone, such as veal ribs, so that in fact the calcium contents were usually adequate although highly variable. In one series of analyses on five unfortified all-meat products, the calcium content ranged from 0.07% to 3.76% and averaged 1.13%. In order to demonstrate bone abnormalities associated with calcium deficiency, it was necessary to screen many batches of cans to identify those that were actually deficient in calcium.(28) In another ad hoc demonstration, pups were switched suddenly to a highly palatable product and consequently developed severe diarrhea that became self-propagating. (11) Multiple deficiencies developed and resulted in some bone abnormalities. These lesions should not be attributed to calcium deficiency, nor even primarily to the diet, but rather to faulty feeding management.

Under experimental conditions, a diet deficient in calcium, phosphorus, and vitamin D induced rickets complicated by osteoporosis.(6) Supplementation with vitamin D reduced the rickets but not the osteoporosis. In these studies, the deficient diet contained 0.10% and 0.15% of calcium and phosphorus, respectively, and the control diet contained 0.5% calcium and 0.3% phosphorus; the vitamin D supplement was 100 IU/kg body weight/day.(6)

Nutritional intervention in rickets includes the identification and cessation of undesirable feeding practices, such as chronic administration of mineral oil, and provision of an appropriate balanced diet, as discussed in more detail in the Section II.

SECONDARY NUTRITIONAL HYPERPARATHYROIDISM
Histologic changes more typical of osteodystrophy fibrosa, excessive resorption, have been observed in dogs and cats fed calcium-deficient diets. These have been attributed to chronic hypersecretion of parathyroid hormone due to transient, small decreases in plasma calcium concentration.(4,22) This naturally occurring disease is probably identical to experimental osteoporosis induced by calcium deficiency.(6)

An alleged epidemic of this condition related to a rapid increase in the market share by all-meat dog foods received much publicity from 1969 to 1974 (7,11,28) These products more than doubled their share of the market in the Philadelphia area from 1965 to 1971, but no increase occurred in the percentage of cases of nutritional hyperparathyroidism among total accessions (over 5000 per year) in the Veterinary Hospital of the University of Pennsylvania.(20) Following an exhaustive investigation, I concluded that the alleged epidemic was fictitious.

Secondary nutritional hyperparathyroidism does occur in dogs or cats that are fed exclusively or almost exclusively meat or meat byproducts that include no bone. A young dog or cat is presented lame and evinces pain upon palpation of the skeleton. Folding fractures may be present in long bones, compression fractures in vertebrae. Older animals may have loose teeth.
Nutritional intervention is achieved best by providing the affected animal with a complete and balanced diet. Good examples are mixtures of dry and canned commercial pet foods or properly supplemented home-made diets (Chapter 20). If the owner persists in wanting to feed only meat, a calcium supplement should be provided. Either the supplements shown in Table 58-3 may be used to supply the calcium requirements shown in Tables 58-1 and 58-2, or tablets containing calcium, phosphorus, and vitamin D may be prescribed according to label recommendations after checking against requirements.

Warnings have been issued against excessive dietary phosphorus, accompanied by treatment with aluminum containing antacids that render phosphorus unavailable.(4,27) The phosphorus content of commercial pet foods is unlikely to have any adverse effects, except in animals that have chronic renal failure and hyperphosphatemia.

Overdosage with calcium, vitamin D, or any other nutrient or pharmacologic preparation is unlikely to accelerate the repair of osteopenia. There is currently no acceptable mode of therapy that stimulates bone formation.(2) I add only one qualification to this admonition from Avioli: in addition to the provision of a balanced diet, another mode of therapy, broadly speaking, is a course of graded, progressive physical exercise at a level appropriate to the animal. Exercise has been shown to increase (47)Ca kinetics and "bone formation rate" in humans.(35) Moderate, deliberate loading of the skeleton supplies a positive physiological stimulus to bone formation and remodeling. The provision of a sound diet is probably best regarded as permissive, enabling the animal to respond optimally to this stimulus.

EXCESSIVE DIETARY CALCIUM
Several syndromes have been attributed to excessive dietary calcium. The outcome depends on other factors in the diet.

Short thick bones and osteoporosis were part of a fatal disease in pups oversupplemented with tricalcium phosphates. It was suggested that calcium impaired the absorption of copper, and hence induced osteoporosis. Also, it was suggested that it interfered with thyroid uptake of iodine, causing hypothyroidism and the short bones.

A collection of bone abnormalities was induced in inbred Harlequin Great Dane pups by feeding a highly palatable diet ad libitum.(17) No abnormalities were found in controls fed the same diet at a rate restricted to about twice maintenance, that is, the rate recommended by the NRC.(31) Skeletal abnormalities, including coxa valga, enostosis, hip dysplasia, hypertrophic osteoarthropathy, osteochondritis dissecans, and wobblers, were observed in dogs fed ad libitum but not in restricted controls. The Krook group attributed these skeletal abnormalities to "excessive intake of a food rich in protein, energy, calcium and phosphorus."(17) The experimental design allows only the first part of this conclusion, an association between skeletal abnormalities and excessive food intakes. It does not permit identification of specific nutrients in the only diet fed to both groups of dogs. This diet was satisfactory when not fed ad libitum. In my opinion, its protein content and energy density were in optimal ranges for growing pups. The argument that these features conferred excessive palatability, which evoked overfeeding, brings into question the need to distinguish between feeding management and diet formulation when addressing practical problems in nutrition. The only rigorous conclusion that may be drawn from this study is that a highly palatable diet should not be fed ad libitum.

Speculation about the diet in the overfeeding study draws attention to very high levels of manganese and vitamin D. Excessive vitamin D tends to overwhelm the body's first defense against a high calcium intake by promoting calcium absorption in a situation that usually is associated with decreased efficiency. This concept is consistent with the initial elevation in plasma calcium concentration, which would be expected to release calcitonin. The Cornell group postulated a key role for hypercalcitoninism but neglected any mention of possible synergism between high intakes of calcium and vitamin D.

The prospect that diets rich in protein and calcium might damage bones has attracted much attention. Measurements of bone mineral density using photon densitometry have shown no difference among dogs fed 28%, 33%, and 39% metabolizable

<table>
<thead>
<tr>
<th>SUPPLEMENT</th>
<th>$/LB</th>
<th>(%)</th>
<th>(%)</th>
<th>(%)</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone meal, steamed</td>
<td>1.50</td>
<td>22</td>
<td>14</td>
<td>3.7</td>
<td>1.1</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>.40</td>
<td>24</td>
<td>18</td>
<td>5.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Limestone, ground</td>
<td>.03</td>
<td>36</td>
<td>—</td>
<td>7.2</td>
<td>2.7</td>
</tr>
</tbody>
</table>

* Because commercial calcium supplements vary in composition, attention should be given to the label; common contents of capsules or tablets are 600 mg calcium, 600 mg phosphate, and 400 IU vitamin D.
energy as protein. These three levels are representative of those found in dry puppy foods, semimoist products, and canned meat dinners, respectively. Thus, it is doubtful that commercial dog foods are likely to produce adverse interactions between protein and calcium when fed properly, that is not ad libitum.

The anamnesis of dogs and cats exhibiting skeletal abnormalities involving overmineralization should include questions about diet, especially the use of supplementary calcium and vitamin D.

OSTEOCHONDROSIS
Osteochondrosis is a disturbance of endochondral ossification. When it leads to degenerative changes in a joint, the condition is called osteochondritis dissecans. On the basis of the Harlequin overfeeding study, Krook has attributed this disease to hypercalcitoninism resulting from too much dietary calcium. On the other hand, controlled comparisons in pigs showed an association between the incidence of osteochondrosis and caloric intake but not dietary calcium. A high caloric intake favored rapid growth.

The disease in pigs appears to involve an inherited predisposition that is manifested only in association with rapid growth. In such a situation, liberal feeding of a sound diet would play a permissive role.

Osteochondrosis in horses appears also to have an inherited component. Rapid growth is not a sufficient cause, or nearly all young thoroughbreds and quarterhorses would be affected. Whether overfeeding is necessary for expression of the predisposition, as it appears to be in the pig, or whether it may merely favor its expression, remains in question. The disease has also been associated with zinc intoxication in both horses and pigs. Nutritional intervention again consists of providing a diet balanced for the animal's situation: growth, maintenance, old age, or stress. This diet should be fed at a rate that achieves a desired rate of growth in a young animal; this rate is likely to be slower than the rate of growth previously experienced. Specific warning should be given against supplementation with calcium and vitamin D or with zinc.

HYPERTROPHIC OSTEODYSTROPHY (HOD)
HOD involves imbalance in remodeling, with apposition outweighing resorption, that is, the opposite to osteoporosis. It has been attributed in part to three types of malnutrition:

- Too much protein, energy, and minerals
- Vitamin C deficiency
- Excessive calcium and vitamin D

None of these malnutritions should be considered to be sufficient to cause HOD; clearly the first two are not. Also, none has been shown to be a necessary causative factor. Instead, it appears that any one of these three malnutritions may be associated in some instances but not others with HOD.

Similarly, overfeeding and rapid growth have been associated with HOD, but they are certainly not sufficient causes (or many more dogs would have HOD). Whether they are necessary conditions for the expression of HOD remains in question.

Low plasma ascorbate concentrations in naturally affected dogs were interpreted to suggest "hypovitaminosis C." In contrast, low plasma ascorbate concentrations in dogs suffering from HOD induced by overfeeding were taken to represent an effect of the HOD rather than a cause. Oral administration of 0.5 g ascorbic acid twice daily resulted in "relative hypercalcemia": 12.2 mg to 12.6 mg/dl compared with 12.1 mg to 12.4 mg/dl in unsupplemented controls. This meager difference served as the basis for suggesting that the ascorbic acid supplement enhanced calcium absorption, which would be undesirable, and for issuing a warning against treating HOD by administration of vitamin C.

Vitamin C is not required in the diet of dogs or cats. Growth and reproduction have not been improved in these species by supplementing vitamin C free diets with vitamin C. However, the rate of ascorbic acid synthesis in dogs and cats may be less than in most species, and the possibility exists that it may be marginal in rare animals. Such an animal is likely to be brought to the attention of a veterinary practitioner but not a research worker in animal science. Over a dozen instances of "skeletal scurvy" or HOD associated perhaps with vitamin C deficiency are recorded. The NRC disposes of these clinical observations as "equivocal reports" and concludes "that there is no adequate evidence to justify recommendation of routine vitamin C additions to the diet of the normal dogs . . ."
Ascorbic acid concentrations in plasma and adrenal gland decline during stress. These responses are often taken to suggest that the requirement for vitamin C is increased during stress. Many dogs are subject to chronic stress that is overt, such as showing and racing, or insidious but nevertheless cumulative, such as deprivation anxieties or exposures to infection. I recommend reasonable supplements for dogs at risk of stress. Megadoses of vitamin C are discussed in Section 11.

Nutritional intervention in the management of HOD again involves feeding a balanced diet at a rate to achieve a desired rate of gain in body weight in a growing animal or a desired final body weight in one that has reached maturity. Warnings should be issued against supplementary calcium and vitamin D. The question of supplementation with vitamin C remains dependent on each case; moderate supplementation, about 2 mg of sodium or potassium ascorbate/kg body weight/day, is unlikely to be harmful in general and may be helpful in certain animals.

**HIP DYSPLASIA**

Hip dysplasia is a progressive instability of the hip joint that develops in cats and dogs that are born apparently normal. It is partially inherited, and its expression may be favored by rapid growth, overfeeding, and, perhaps, malnutrition. No nutritional factor has been established as a sufficient or necessary causative factor, but the following types of malnutrition have been suggested to play a role:

- Excessive protein, energy, calcium, and phosphorus
- Protein deficiency
- Vitamin C deficiency

The first of these suggestions has been discussed above. It may be valid only insofar as it relates to overfeeding in general promoting a growth rate so rapid that it exacerbates any instability that is developing in the hip. The proposition that rapid growth rate enhances the likelihood of phenotypic expression of an inherited predisposition to develop hip dysplasia remains in question.

The protein insufficiency hypothesis is based mainly on the observation of hypoalbuminemia in association with hip dysplasia. It may relate to a protein/energy imbalance or to imbalances among amino acids, such as those involved in collagen synthesis (see Physiological Nutrition, above).

The postulation of vitamin C deficiency is based on an apparent benefit from megadosage of ascorbic acid in uncontrolled trials. No support was afforded by the only published controlled trial, which was limited in scope.

Nutritional intervention again consists of feeding an appropriately balanced diet at a rate that achieves the desired growth rate, usually a slower rate than heretofore. Dietary protein deserves attention. I recommend a dietary protein content of 25% to 30% of metabolizable energy for growing pups and 30% to 35% for kittens at risk of hip dysplasia.

Megadosage of ascorbic acid is not recommended. It may threaten acid-base balance, alter uric acid metabolism, or induce erythrocyte fragility. It has also been shown to depress tests of immunocompetence. Extreme megadosage leads to bowel intolerance, the most common overt sign. On the other hand, I am not adverse to moderate supplementation of vitamin C (see HOD above).

**VITAMIN A EXCESS OR DEFICIENCY**

Skeletal changes attributable to hypovitaminosis A have been observed both experimentally and clinically in dogs and cats. The animals are usually lame. The long bones appear bulky and ill-shaped. This disease is probably rare, since vitamin A is abundant in virtually all commercial pet foods and most recipes that include liver. Associated clinical signs are not specific, and plasma retinol is not a good index of vitamin A status: thus, confirming any diagnosis of vitamin A deficiency is not easy. The clinical and radiologic responses to moderate vitamin A supplementation may be among the most convenient indicators.

Nutritional intervention includes the daily oral administration of vitamin A, about 200 IU/kg body weight. This supplement should be added to a balanced diet that already has an adequate vitamin A content of 5,000 IU to 10,000 IU/kg dry matter. The dog is able to convert carotene to retinol, but the cat has an essential requirement for vitamin A.
Hypervitaminosis A occurs in cats and, rarely, dogs. It usually follows predominant feeding of liver for 1 to 5 years. The condition in cats is characterized by extensive, confluent exostoses of cervicothoracic vertebrae and has been named "deforming cervical spondylosis." (41) It also affects long bones, particularly the forelimbs. The clinical appearance and dietary history of the animal should secure the diagnosis.

Exostoses are not common in hypervitaminosis A of 5 dogs. There are malformations of long bones, cranial bones, and vertebrae. (4) Remodeling is accelerated, and cortices are thinner. The dog is usually lame but without fractures. (31)

Vitamin A intoxication due to oversupplementation is often associated with vitamin D intoxication. Certain glucocorticoids may be used to treat vitamin D intoxication, especially those that tend to reduce the plasma calcium concentration. These corticosteroids are contraindicated in vitamin A intoxication.

Nutritional intervention in vitamin A intoxication consists of feeding a balanced diet, preferably one known to contain vitamin A close to the level recommended by the NRC: 5,000 IU/kg dry matter for dogs and 10,000 IU/kg for cats. (30,31) Many commercial products contain appreciable "overage" of vitamin A to cover anticipated losses during processing and storage. Thus it may be propitious to use a recipe for home-cooking, perhaps with only one half of the routine amount of liver, for 1 or 2 months.

OBESITY
Gross obesity increases the risk of death in humans. Causes of these excess deaths are, in descending order of frequency, diabetes mellitus, gallstones, appendicitis, liver cirrhosis, essential hypertension, and atheromatous heart disease; these diseases are rare or not found at all in cats and dogs. Next most frequent is exacerbation of joint and bone problems. Thus, among veterinary medical specialists, orthopaedic surgeons should be the most concerned with obesity.

Obesity and body weight reduction are discussed in detail elsewhere. (21) A brief summary of my preferred weight reduction program for dogs follows:

1. The owner should decide if the dog is overweight or obese by reference to charts as well as personal preference.

2. The dog should be examined by a veterinarian to exclude causes of overweight other than overfeeding and to help decide upon a target weight.

3. The general objective of the program should be to change the life style so that weight reduction will persist. First, the owner should establish one or more rewards other than feeding, preferably rewards that involve exercise. Examples are playing with balls or sticks, tug-of-war with socks or rags, and walking.

4. A progressive, graded exercise program should be introduced. Its three aims are to provide a reward other than food, to ensure that physical condition is maintained or improved during weight reduction, and, least of all, to burn off a few calories. This exercise program may need to begin very gently in a habitually slothful animal, but it should be maintained for at least a week before the next step.

5. The amount of food should be measured daily. It should be decreased by about 5% per week for 4 weeks. The dog should be weighed weekly. The nature of the diet need not be changed. If it is changed, then the change should be a reduction in "empty calories" but not essential nutrients. Commercial weight-reduction diets are high in fiber content so that water and bulk are retained in the digestive tract to give a feeling of fullness. These diets are claimed to reduce begging, the chief complaint about dogs on weightreduction programs. Begging is also averted by diminishing the daily food intake by only 5% per week and by distracting the dog with other rewards.

6. The 4 weeks of food reduction, reaching a total decrease of 20% of daily intake, are followed by 4 weeks at this level. Then the whole program should be reevaluated by the owner and the veterinarian. A decision should be made to hold at this daily intake or to decrease it further.

7. Once the dog has achieved the desired body weight, efforts should persist to keep it at this level. At this time, the advantages of using a good diet during weight reduction and building the program around alternative rewards become even more important. Without a positive change in life style, dieting degenerates into the "rhythm method of girth control."
This weight-reduction program may be adapted to cats, with more attention to two difficulties. One is the susceptibility of cats to struvite urolithiasis, which may be favored by reduction in water available for urine when a cat is fed a high-fiber diet. The other is the difficulty in fathoming rewards for a cat. Unless orthopaedic demands are compelling, it may be better to agree with the client that fat cats are pretty.

SECTION TWO  
FEEDING FOR GROWTH, REPAIR, AND STRESS

- Nutritional Requirements
- Daily Intakes
- The Staples
- A Versatile Supplement
- Convenient Cooking in Home or Hospital
- Commercial Pet Foods
- A Feeding Program Using Commercial Pet Food

Orthopaedic surgeons often express interest in the influence of nutrition on bones, especially during growth or repair, which may be regarded as a form of growth associated with metabolic stress. Any complete and balanced pet food made by a reliable manufacturer expressly for puppies or kittens probably contains nutrients in optimal ranges for both conditions, growth or repair. The first modification of that recommendation would be to add a small supplement of high-quality protein for slightly better prospects of repair if the animal is undergoing stress. It has been found that in dogs the protein requirement is greater for stress than for growth. Any further attempt to advise the orthopaedic surgeon about optimal feeding of growing, injured, or recovering dogs and cats encounters several difficulties. The literature on growth of puppies, for example, is at odds with practical experience, which calls for more narrow optimal ranges of fat, protein, and carbohydrate. The literature often attributes adverse consequences of poor feeding management to poor diets (Fig. 58-1). The orthopaedic surgeon, like the competitive breeder, is interested in optimal nutrition, but the establishment recommends only "adequate" nutrient requirements that are likely to be suboptimal (Fig. 58-2).

FIG. 58-1 Requirements of food energy and nutrients are determined by the nature of the animal and the performance desired by its owner. The diet when consumed in an appropriate daily amount, should provide these requirements. A ration is the amount of the diet offered daily. In our experience, problems that arise primarily from poor feeding management are often attributed erroneously to the diet. For this reason we evaluate feeding management ahead of diet when investigating any health problem that may involve nutrition.
The optimal ranges are not well established; nonetheless, I will offer my own current practical guides. These will be followed by a description of practical feeding regimens of cooking in home and hospital or using commercial pet foods.

NUTRIENT REQUIREMENTS

Nutrient requirements of dogs and cats have been proposed by the NRC. Only one diet on a dry-matter basis has been recommended for each species (Table 58-4). One diet is claimed to provide "adequate nutrition of both growing puppies and adult dogs," and the other is "presumed adequate to support maintenance and growth of the cat." The ration or daily intake is varied by changing the quantity of the diet rather than its quality (composition). The manufacture of foods for pups and dogs in the other stages of the life cycle shows that industry disagrees with the NRC. This is a distinct step away from "adequate" nutrition toward optimal nutrition (Fig. 58-2).

FIG. 58-2 The optimal daily intake or dietary content of a nutrient is represented better by a plateau than by a peak because of imperfections in methods of determination, variation among animals, and homeostatic responses within animals. The upper figure is redrawn from Mertz with added indications of the minimum nutrient requirements and the indeterminate "adequate." The lower graph shows the protein requirements of dogs, comparing optimal ranges for maintenance of adults, growth of pups, and stress. The range is broad for the undemanding situation but becomes more narrow as nutritional demands increase and constrain homeostasis. Ranges covering about 85% of maximal performance are 15% to 65% for maintenance, 25% to 50% for growth, and 30% to 40% for the stress of repeated exhaustive exercise. (These ranges are the best estimates from available data; the lower limits are more precisely determined than the upper.)

The optimal ranges are not well established; nonetheless, I will offer my own current practical guides. These will be followed by a description of practical feeding regimens of cooking in home and hospital or using commercial pet foods.

TABLE 58-4 Comparison of Nutrient Requirement of Dogs and Cats as Recommended by the NRC

Experimental observations made on puppies suggest that very wide ranges lead to no gross differences. In one study, nine diets appeared satisfactory, although protein varied from 20% to 48%, fat from 13% to 76%, and carbohydrate from 9% to 62% of metabolizable energy. The kinds of observations made by highly competitive breeders of show dogs and racing dogs with whom I work indicate much narrower optimal ranges (Table 58-5). Thus, practical experience of feeding dogs runs contrary to the data in the scientific literature. The crux of the discrepancy lies perhaps in the use of different criteria of performance and in the sensitivity of observations.

TABLE 58-5 Tentative Guidelines for Optimal Ranges of Protein, Fat, and Carbohydrate
Requirements of essential nutrients are expressed best on the basis of metabolizable or available energy. If the amount of protein on an energy basis is held constant, say at 25% for growth or 30% for stress, then its content on a weight basis must increase as the amount of fat on a weight basis is increased (Table 58-6). The energy density of the diet is determined largely by its content of fat, since oxidation in the body yields 9 kilocalories/gram of fat compared with only 4 kilocalories/gram of protein or carbohydrate. Ironically, the optimal fat content of the diet is not well established (except for hard work and stress in dogs), but selection of the fat content determines largely two principal features of the diet: its energy and protein contents on a dry matter basis (Table 58-6).

In contrast to protein, mineral requirements on a weight basis probably decrease as energy density increases in commercial pet foods, since higher energy densities are associated with meat and meat byproducts, while lower energy densities are associated with corn, milling byproducts, and soybeans. Corn is low in protein and calcium. Soy is added as a protein supplement, but it is rich in phytin, which binds calcium. Thus, calcium is added in abundance. The efficiency of absorption of many minerals, notably zinc,(63) is suppressed by vegetable fiber, phytin, and calcium. The mineral requirements recommended by the NRC (see table 58-4) relate well to semipurified or mainly meat diets but may be too low for diets based on grains and soybeans.

### DAILY INTAKES

Average food intakes are based on energy needs of healthy dogs and cats (Tables 58-7 and 58-8). Individual variation in dogs and cats probably is more like that of humans (coefficient of variation about 15%) than that of farm animals (about 5%). If the average 25-lb dog requires 8 oz of dry dog food, then one of 20 such dogs will need 6 oz and another one of the same 20 will need 11 oz. This divergence illustrates the need for individual attention to daily food intake and body weight. Injury further complicates the situation, since it may reduce appetite at a time when requirements are increased for food energy and especially protein.

### TABLE 58-6 Influence of Dietary Fat Content on Energy Density and on Protein Contents Expressed on a Dry-Matter (DM) Basis

<table>
<thead>
<tr>
<th>FAT (g/d)</th>
<th>ENERGY DENSITY (kcal/g)</th>
<th>PROTEIN (g/d)</th>
<th>PROTEIN (% E)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>3.4</td>
<td>30</td>
<td>25</td>
</tr>
<tr>
<td>10</td>
<td>3.7</td>
<td>30</td>
<td>28</td>
</tr>
<tr>
<td>15</td>
<td>4.0</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>20</td>
<td>4.4</td>
<td>30</td>
<td>33</td>
</tr>
<tr>
<td>30</td>
<td>5.0</td>
<td>30</td>
<td>37</td>
</tr>
<tr>
<td>50</td>
<td>5.6</td>
<td>30</td>
<td>42</td>
</tr>
</tbody>
</table>

* Lower end of optimal range for cats in general and dogs under stress (see Table 58-5).

### TABLE 58-7 Recommended Daily Intakes of the Four Main Types of Commercial Dog Foods for the Maintenance of Adult Dogs, Expressed in Terms of Ounces, Cans (420g or 14.8 oz) or Patties (85g or 3oz)

### TABLE 58-8 Recommended Daily Allowance of Commercial Cat Foods

An optimal diet should relate not only to the animal but also to feeding management (see Fig. 58-1). Overfeeding that results when all palatable diet is fed free choice(33) is primarily a problem of feeding management. Its correction by lowering the contents of animal fat and protein to reduce palatability leads surely to suboptimal nutrition; two wrongs do not make a right. Young or injured animals should be fed a high-quality diet, and the desired daily intake or ration should be decided by the feeder.
THE STAPLES
The main sources of food energy—staples—for dogs and cats are meat, meat byproducts, cereal grains, and mining byproducts. In this order, these foodstuffs decline in nutritional value but increase in profitability.

Meat is skeletal muscle. It contains a variable amount of fat; very lean is about 5%, very fat about 50%. The remainder is about one third protein and two thirds water. Muscle glycogen breaks down post mortem. Meat is deficient always in calcium and vitamin A, often in copper and iodine.

Corn (maize) grain contains about 2% fiber, 8% protein, and 80% starch. It is deficient in calcium, riboflavin, and niacin, and in protein and fat for carnivores.

Milling byproducts contain relatively more fiber, protein, and phosphorus. Wheat bran, for example, contains about 11% fiber, 17% protein, 1.3% phosphorus, and 55% starch.

Meat byproducts is a collective term for all of the edible parts of the animal carcass that are not meat. Most common in commercial pet foods are lungs, glands, and bone. Any single type of byproduct may have deficiencies, but an assemblage of meat byproducts may be sufficient in all essential nutrients. Bone meal may supply the calcium that is lacking in corn and meat, and liver may supply the needed vitamins and trace minerals. Meat byproducts may be formulated to provide a complete and balanced diet for cats and dogs, but exclusive use of meat, grains, or mining byproducts would induce deficiencies.

A VERSATILE SUPPLEMENT
The traditional approach to diet formulation is to decide upon the staple for reasons of supply and economy, then to assemble a leastcost supplement to make up for deficiencies in the staple. I have abandoned that approach in my system of home-cooking for dogs and cats. I found that a single supplement (Table 58-9) would be suitable for any mixture of staples that contained at least one third meat by volume (the common measure in the kitchen). This much meat is needed to provide minimal to adequate contents of protein for maintenance (basic formula in Table 58-10). Twice as much meat is needed in a diet for maintenance of cats or for growth, stress and, presumably, repair in dogs (meaty formula in Table 58-10).

Among the four ingredients in the staple, three are packaged conveniently: the bone meal, corn oil, and salt. The fourth, liver, may also be handled fairly readily. Each pound is divided into 16 pieces that are placed in the wells of a plastic ice-cube tray. Frozen pieces of liver are popped out as required.

CONVENIENT COOKING FOR HOME OR HOSPITAL
The diets listed in Table 58-6 are prepared conveniently in the home or hospital. The rice, twice its volume of water, the salt, and corn oil are simmered for 20 minutes, ground or chopped meat and liver are added, and the mixture is simmered for another 10 minutes. The food is cooled before feeding. Batches may be refrigerated for 3 to 4 days or frozen indefinitely. For the purposes of growth, repair, and stress, the meaty formula would be used (Table 58-10). It provides about 33% protein, 27% carbohydrate, and 40% fat on an available energy basis. The basic formula has 18% protein, 55% carbohydrate, and 27% fat on an energy basis. Both contain 1.4% calcium and 1.3% phosphorus on a dry-matter basis.

These home-cooking recipes rely on the liver for vitamins A and D. In general liver is rich in these vitamins, but its vitamin content is inconsistent. For this reason, it might be prudent to feed a proprietary vitamin-mineral supplement that contains vitamins A and D sufficient to provide only one or two times the NRC recommendations (see Table 58-4).
COMMERCIAL PET FOODS

The three main forms of commercial pet foods (dry, semimoist, and canned) differ primarily according to their means of processing and preservation. They also differ in regard to composition, that is, predominant ingredients and chemical analysis, especially the proportions of protein, fat, and carbohydrate (Table 58-11). It is often said that they differ most in their contents of water, but they differ more in contents of carbohydrate and air. The diversification of brands during the last decade reflects the drive of market segmentation more than any demonstrated nutritional differentiation of needs for specific purposes, except for the difference between products intended for maintenance only and those intended for growth.

According to US labeling regulations, "complete" or "balanced" means that the product either meets or exceeds the recommendations of the NRC or has passed protocols for the whole life cycle set by the American Association of Feed Control Officials (AAFCO). Complete and balanced for maintenance (only) or for maintenance and growth (only) indicates that the product has passed somewhat less demanding AAFCO protocols than those for the whole life cycle. Veterinary surgeons would almost always prefer products that have passed the tests for the whole life cycle and should scrutinize the label carefully for such wording.

Canning destroys microorganisms and toxins by heat. Excessive heat may also destroy essential nutrients, notably thiamine and methionine. Sealing preserves heat-sanitized food, but solders contain lead in amounts that used to be hazardous and stir could be in instances in which old canning machines remain in use.

Canned foods can be divided into two main categories: predominantly meat, or mixed grain and meat byproducts ("ration"). Market share of the "ration" type has slipped in the last decade; it is uneconomical both as a main energy source and as a protein supplement.

Most "all-meat" products were relabeled "95% meat and meat byproducts" in 1969, and then "meat dinner" in 1974. The dinner designation covers meat and meat byproducts from 95% down to 25%. These products have come to include progressively more texturized vegetable protein (TVP) chunks of tofuleike material made from soy flour. The limit to soy seems to be the social acceptability of the pet; galactosides in soy that resist enzymatic hydrolysis in the small intestine then undergo bacterial fermentation in the lower bowel. Large amounts of soy, rich in phytin, could also compromise the absorption of zinc. Canned meat dinners are holding on to their share of the market. Canned products that have protein contents high relative to fat tend to induce diarrhea in dogs. I recommend products with minimums no less than 11% protein and 6% fat for dogs, and 14% protein and 8% fat for cats. Canned fish or poultry for cats may contain excessive bone, sufficient to diminish digestibility and to cause generalized deficiencies of nutrients and energy. Ash should constitute less than 3% of canned cat foods.

The first wave of semimoist products depended for preservation mainly on a low availability of water for microorganisms (water activity) controlled by humectants, such as propylene glycol, sucrose, and corn syrup. Main ingredients are grain and meat byproducts. These products are convenient for use in the home but perhaps too expensive for a large kennel or hospital.

### TABLE 58-10 Four Mixtures of Meat and Rice Suitable for Use With Supplement in Table 58-9

<table>
<thead>
<tr>
<th>Rice, dry</th>
<th>Meaty</th>
<th>Reducing</th>
<th>Germinating</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Dry</td>
<td>90</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Dry, 7%</td>
<td>92</td>
<td>94</td>
<td>11</td>
</tr>
<tr>
<td>Dry, 5%</td>
<td>94</td>
<td>94</td>
<td>11</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>96</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Energy, kcal</td>
<td>750</td>
<td>750</td>
<td>605</td>
</tr>
<tr>
<td>Protein, % kcal</td>
<td>18</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Fat, % kcal</td>
<td>27</td>
<td>27</td>
<td>27</td>
</tr>
<tr>
<td>Carbohydrate, % kcal</td>
<td>55</td>
<td>55</td>
<td>55</td>
</tr>
</tbody>
</table>

* Measures in cups (9 fl oz).

### TABLE 58-11 Representative Values of Available Energy and Proportions of Protein, Fat, and Carbohydrate in the Main Types of Commercial Pet Foods

<table>
<thead>
<tr>
<th>As Fed</th>
<th>Dry Matter</th>
<th>Available Energy (kcal)</th>
<th>Protein</th>
<th>Fat</th>
<th>Carbohydrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dog Food</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dry, general</td>
<td>3.4</td>
<td>3.7</td>
<td>25</td>
<td>25</td>
<td>54</td>
</tr>
<tr>
<td>Dry, puppy</td>
<td>3.5</td>
<td>3.8</td>
<td>27</td>
<td>27</td>
<td>54</td>
</tr>
<tr>
<td>Dog, canned</td>
<td>5.3</td>
<td>4.1</td>
<td>31</td>
<td>31</td>
<td>44</td>
</tr>
<tr>
<td>Canned mixed</td>
<td>3.1</td>
<td>3.1</td>
<td>35</td>
<td>35</td>
<td>31</td>
</tr>
<tr>
<td>Canned meat dinner</td>
<td>3.3</td>
<td>5.0</td>
<td>41</td>
<td>31</td>
<td>5</td>
</tr>
<tr>
<td>Cat Food</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dry</td>
<td>3.7</td>
<td>4.0</td>
<td>32</td>
<td>32</td>
<td>31</td>
</tr>
<tr>
<td>Semimisit</td>
<td>3.0</td>
<td>4.0</td>
<td>31</td>
<td>31</td>
<td>32</td>
</tr>
<tr>
<td>Canned, 4 oz</td>
<td>1.3</td>
<td>4.9</td>
<td>62</td>
<td>47</td>
<td>13</td>
</tr>
<tr>
<td>Canned, 1 oz</td>
<td>1.2</td>
<td>4.4</td>
<td>62</td>
<td>47</td>
<td>13</td>
</tr>
</tbody>
</table>

According to US labeling regulations, "complete" or "balanced" means that the product either meets or exceeds the recommendations of the NRC or has passed protocols for the whole life cycle set by the American Association of Feed Control Officials (AAFCO). Complete and balanced for maintenance (only) or for maintenance and growth (only) indicates that the product has passed somewhat less demanding AAFCO protocols than those for the whole life cycle. Veterinary surgeons would almost always prefer products that have passed the tests for the whole life cycle and should scrutinize the label carefully for such wording.

Canning destroys microorganisms and toxins by heat. Excessive heat may also destroy essential nutrients, notably thiamine and methionine. Sealing preserves heat-sanitized food, but solders contain lead in amounts that used to be hazardous and stir could be in instances in which old canning machines remain in use.
The second wave of semimoist pet foods, sometimes known as softdry, depend mainly on a pH of 4.2 or lower for preservation. In preliminary studies, we have found that one of these products greatly increases the titratable acidity of urine and the fractional excretion of calcium. Experiments are in progress that should determine whether extra urinary calcium derives from the digestive tract or from bone. Until this dilemma has been resolved, I recommend caution in feeding soft-dry pet foods, especially when bone growth or repair is a concern.

Dry pet foods are based on grain and milling byproducts. Protein is supplied as meat-and-bone meal or soybean meal. "Meals" are mixtures of precooked ingredients, and kibbles are broken biscuits, cooked after mixing. These names remain, but nearly all national brand dry foods (in the United States) are extruded biscuits that are "puffed" or expanded during extrusion. The degree of expansion is decided by market research. One major company advertises that the caloric content of one cupful (8 fl oz) of their product ranges from 240 kcal to 410 kcal; this indicates the range of air trapped in the nuggets. This trapped air is undesirable for dogs at risk of acute gastric dilation (bloat).

Overcooking is a major problem of dry dog foods. The close proximity of proteins and carbohydrates favors the formation of amino-aldehyde polymers that are indigestible. The better products may have only about 5% of protein made unavailable in this way, but products from companies with limited quality control in processing may contain over 50% unavailable protein.

The relative risk of urolithiasis in cats was greatly increased in those fed only dry cat food.(62) Hypotheses concern tubular concentrations of magnesium, ammonium, and phosphate, that is, the moieties of struvite. These relate not only to these constituents in the diet but also to the food's titratable acidity or alkalinity and its fiber content, which affects water in feces, hence urine. The dry cat foods continue to gain yearly in market share despite this health hazard.

Contemporary dry cat foods that contain animal digest, an enzymatic degradation product of meat byproducts, poultry byproducts, or fish,(70) may not promote urolithiasis as much as the dry cat foods of a decade ago. The degradation of digest is stopped by the addition of acids, such as phosphoric. Inclusion of digest increases the titratable acidity of dry pet foods. The consequences of this food acid on absorption of calcium from the digestive tract or resorption from bone are currently under investigation. Until results are in hand, I do not recommend the use of dry pet foods that contain digest for animals with broken or repairing bones. I would like to extend this caution to growing puppies and kittens, but a dry puppy food or kitten food that does not contain digest is increasingly hard to find. Digest is used primarily as a flavor enhancer.(70)

A FEEDING PROGRAM USING COMMERCIAL PET FOODS
For safety, use only those products labeled complete and balanced for the whole life cycle, tested by AAFCO protocols.(61) Meeting the NRC requirements(59,60) is not good enough.

For economy, use dry pet foods as the staple or main food energy source. Select a product that contains no digest or the least digest, that is, digest closest to iodized salt in the list of ingredients.

For economy and convenience use dry products made expressly for puppies or kittens during growth or repair.

For higher demands, such as repair or stress, supplement protein. Add whole egg, for example, one medium size egg (50 g) per half pound of dry dog food or per 4 oz of dry cat food. Or mix dry pet food with canned meat dinner for dogs or canned meat, chicken, or fish for cats. Mixtures of dry dog food and canned meat dinner allow adjustment of proportions of protein, fat, and carbohydrate within desired ranges (Table 58-12). A 1:1 mixture of dry/canned by volume is about a 4:1 mixture on a dry-matter basis and 1:3 on available energy basis. For equal parts on an energy basis, the volume ratio is about 1:3 of dry/canned meat dinner. Therefore the usual range of mixtures by volume needed for growth and stress is 1:1 to 1:3 of dry/canned meat dinner. Similar proportions are probably useful for cats.

| Table 58-12 Volumes (Canfuls) of Dry Dog Food and Canned Meat Dinner in Proportions Suitable for Various Levels of Performance or Nutritional Demand |
|---|---|---|---|
| Performance or Nutritional Demand | Dry Dog Food | Canned Meat Dinner | Nutritional Demand |
| Low | Medium | High |
| Growth | Stress | Repair |
| 1:1 | 2:1 | 3:1 | 1:3 | 2:3 | 3:3 |

Although the main nutritional interests of orthopaedic surgeons relate to growth, stress, and repair, systematic discussion of feeding should begin with maintenance then proceed through pregnancy, lactation, weaning, and growth.
MAINTENANCE
Maintenance is the condition in which a healthy adult animal maintains its body weight while relatively inactive in comfortable surroundings. Most cats and dogs fare fairly well on dry pet foods when at maintenance. This is most economical, at least in terms of immediate cost of food. I have reservations, however, about restricting dogs and cats to commercial dry pet foods. Because dry cat food increases the relative risk of urolithiasis,(62) I recommend that dry cat food be restricted to one half or less of the ration (see Table 58-8).

Dogs that are never likely to be used for breeding or competition, such as neuters, may be relegated to dry dog food. I recommend, however, that dogs that retain potential for breeding or competition should be fed mixtures of dry food and canned meat dinner. This establishes a routine in feeding management and a better adjusted starting point for improving the diet when higher nutritional demands arise.

The dry food is moistened with sufficient water, perhaps one eighth to one quarter of its volume. Then a little canned meat dinner is added. This moistened and supplemented food is fed immediately or refrigerated in order not to spoil.

If there is no need to establish the moist mixing routine, the dry food may be left dry in the bowl and the canned meat dinner fed separately as a treat.

I recommend a 20:1 to 10:1 mixture, dry/canned, by volume (canful) for maintenance of dogs over 80 lb body weight, about 10:1 to 3:1 for dogs between 80 lb and 20 lb, and 3:1 to 1:1 for smaller dogs.

BREEDING
Bitches and queens who are candidates for breeding should be reduced to medium condition. They should also be up-to-date in health programs for worming and vaccinations.

Both species have a gestation period of 9 weeks. The pregnant cat starts to gain weight appreciably in 1 or 2 weeks, but the pregnant dog gains little until about 6 or 7 weeks. The average bitch may increase her body weight by 35%, the average queen by 25%.

A queen that is fed at least a 1:1 mixture of dry and canned food before mating needs no change in her diet during gestation, only a progressive increase in daily intake. As an initial guide, I recommend about 5% increase per week, starting in the second week. In contrast, a bitch should be held to her maintenance level of intake until 5 or 6 weeks of gestation, then the daily intake is increased about 10% per week.

The real metabolic challenge lies in lactation; thus, part of the feeding program during the last third of pregnancy is concerned with preparation for lactation. In both species, the daily intake is divided into 2 meals from 6 weeks onward. For queens and small bitches, the proportion of protein and fat is increased, changing to a 1:3 ratio of dry/canned by volume (equal parts on an energy basis). Large breeds of dogs may change to about 1: 1, dry/canned. In this way, the digestive and metabolic systems of the animals are completely adjusted to the diet before lactation commences, at which time the ration is increased only by increasing the daily intake of the diet without further change in the diet.

Appetite may be lost during parturition, perhaps 12 hours before. Any unconsumed food is removed. The animal is tempted from time to time with a little fresh food.

After she starts eating again, the animal may need two to three times her maintenance intake. The peak should be reached gradually. She is allowed about one half more than maintenance during the last 10 days of pregnancy, in two meals, and returned to the same regimen for two or three days immediately after parturition. She is then offered increasing amounts of food until given all she wants by the seventh or eighth day. This amount may be so large that three or four meals are easier on her than two. Overwhelmed digestive processes may be manifested as sloppy stools or diarrhea. These signs warrant division of the daily intake into more meals or reduction of the intake. If the quantity must be reduced, the food quality should be improved at the same time, so that her intake of fat and protein does not decrease, that is, less dry food is used with same amount or a little more canned food.
Classic studies in bitches showed that milk production is greatly influenced by dietary protein. The milk of a bitch or queen also contains much fat, so she should achieve her best genetic capability of milk production when fed a diet that contains abundant protein and fat.

CREEP-FEEDING, WEANING, AND GROWING
Pups and kittens are often weaned at 6 weeks of age. Pups may benefit from another week or two with the bitch. I recommend creep-feeding from 3 or 4 weeks onward, offering a little canned food to pups beyond the reach of the bitch. Queens are more likely to share. Creep-feeding reduces nutritional demands on the mother and helps the digestive system of the pup or kitten gradually adapt from the composition of milk (Table 58-13) to the postweaning diet. This approaches 1:3 mixture of moistened dry food and canned food by 10 weeks. The ratio changes to 1:1 by the time the growing animal is about 20% of its intended mature weight.

<table>
<thead>
<tr>
<th>AVAILABLE ENERGY (%)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fat</strong></td>
<td><strong>Protein</strong></td>
</tr>
<tr>
<td>Dog</td>
<td>60</td>
</tr>
<tr>
<td>Cat</td>
<td>40</td>
</tr>
</tbody>
</table>

For home-cooking, kittens and cats all receive the meaty formula (see Table 58-10), that is, a 1:2 ratio by volume of dry rice to meat. They continue on this until nearly fully grown and then are changed gradually to the basic formula (2:1, rice/meat) for maintenance.

No exact amounts of feed are prescribed. Instead, the client is told about adverse effects of underfeeding (poor growth, less activity) and overfeeding (diarrhea, less activity, perhaps bone abnormalities in large breeds). Emphasis is placed on adjusting the ration every week to obtain the desired rate of growth, coat, body conformation, and spontaneous activity.

At weaning, a pup of a large breed may weigh 10 lb and require a daily intake like that of a mature dog of 30 lb (see Table 58-7). By 4 months, it may be half-grown at 40 lb and require daily intake like that of its own expected mature weight of 80 lb. When it is three quarters grown, that is, about 60 lb, its daily intake may be like that of an adult that is 90 lb to 100 lb. Thus, the level of intake is about three times maintenance at first and gradually decreases toward maintenance. This recommendation differs from the NRC,(60) which is two times maintenance throughout growth.

Recommended daily intakes for kittens from weaning to 5 months are shown for various types of cat foods in Table 58-5. At 10 weeks, for example, a kitten should be fed about 1 oz of dry food and 2 oz of canned, or 4 oz of canned alone.

STRESS
Our studies on racing sled dogs have shown that 32% available energy in the form of high-quality protein is needed to maintain red blood cells.(55)

Less dietary protein, like that in the only commercially available "stress diet," leads to anemia.(47) The red blood cell responses connect these field trials to classic experiments on stress in dogs.(52,57,66) Stress anemia is thought to involve release of certain hormones, such as glucocorticoids, that reduce anabolic functions, including red blood cell production.

Protein mobilization and urinary loss of nitrogen are characteristic of stress.(49) The stress response is affected by tissue protein reserves. These become maximal only after feeding diets that contain much more protein than that required for zero nitrogen balance about two times this level in mature young dogs and three times in old dogs.(67) Dietary protein before stress occurs has been shown to be important in metabolic response of rats subjected to bone fractures.(58) Old dogs probably require higher dietary protein than young adults not only to maximize tissue protein reserves(67) but also because, like humans, the efficiency of protein utilization is decreased(69) and the likelihood of stress developing is increased.(68) The latter might include a higher incidence of bone fractures in the aged associated with the bone loss of aging(51)

Little is known about extra demand for nutrients during stress in cats and dogs, except for the high protein requirement.
discussed above. In other species, stress has been shown to increase demands for potassium, calcium, magnesium, copper, iron, and zinc, as well as vitamins A, E, and C. Among these nutrients, only vitamin C has received attention in dogs. The serum concentration of ascorbic acid was decreased in dogs that had hypertrophic osteodystrophy induced by overfeeding. (65) This hypoascorbicacidemia was attributed to pain resulting from the abnormal bone, that is, to be an effect rather than a cause of the bone condition, contrary to previous suggestions that identified the bone abnormality as a form of skeletal rickets.

REFERENCES

NUTRITIONAL ASPECTS OF SKELETAL DISEASES
42. Sheffy BE, Caramichael LC, Appel M: Canine nutrition with special emphasis on the role of vitamin C. Gaines Veterinary Symposium 20:30, 1970

FEEDING FOR GROWTH, REPAIR, AND STRESS
48. Coleman WC: Status of protocols uncertain with revised NRC requirements. Petfood Industry 17:12, 1975
50. Daggs RG: Studies on lactation: I. Production of milk in the dog as influenced by different kinds of food proteins. J Nutr 4:443, 1931
58. Munro HN, Chalmers MI: Fracture metabolism at different levels of protein intake. Br J Exp Pathol 26:396, 1945
88:66, 1966
68. Young VR, Schrimshaw NS: Protein needs of elderly. Nutrition Notes, American Institute of Nutrition 11:6, 1975

All rights reserved. This document is available on-line at www.ivis.org. Document No. B0059.0685.