

CHAPTER

31

MALNUTRITION

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Malnutrition can cause a specific problem or suppress a bird's immune system, decrease response to therapeutic agents, decrease reproductive performance and prolong the period of surgical recovery.

The diet of every avian patient should be carefully evaluated, even if the bird appears clinically to be well nourished. Marginal nutritional inadequacies frequently occur (see Chapter 8), and correcting the diet will improve a bird's general health and its ability to resist infectious diseases. Gastrointestinal malabsorption, hepatitis or renal disease can increase nutrient requirements so that diets that are sufficient in healthy birds may be insufficient for unhealthy birds. Interestingly, free-ranging granivorous birds that are offered both organic (no pesticides) and pesticide-treated grains will preferentially consume the organic foods. Test birds would not eat the pesticide-treated foods until all of the organic grains were gone.

Birds with signs of malnutrition have often developed strong preferences for unbalanced diets. Most seed diets, for example, contain excessive levels of fat and may be deficient in vitamins A, D₃, E, B₁₂ and K₁, plus riboflavin, pantothenic acid, niacin, biotin, choline, iodine, iron, copper, manganese, selenium, sodium, calcium, zinc and some amino acids (eg, lysine and methionine).⁶

Birds can be encouraged to accept new foods by offering them first thing in the morning when the appetite is strongest. Favorite items can be withheld until later in the day. New foods may also be mixed with the bird's normal familiar diet. Gradually decreasing the quantity of old food items and increasing the quantity of new foods in the mixture will allow for a smooth transition in the diet. The size of food items offered should be appropriate to the species of bird.¹⁰

Some birds may be encouraged to eat an unfamiliar food if they can observe its consumption by other birds (Figure 31.1). Converting birds housed in large groups to a new diet is often easier than converting individual birds.⁵⁵

Nutritional requirements vary depending on a bird's species, age, reproductive state, molting status, the external temperature and amount of daily exercise. Formulated diets supplemented with some fresh

fruits and vegetables will improve the nutritional state of birds (see Chapter 3).

While a diet change is occurring, it is important that the bird be carefully monitored for weight loss. Radical, unsupervised changes in the diet can lead to starvation. Most birds will have some degree of diarrhea or polyuria during a diet change. Ketosis was seen in some cockatoos that refused to eat during the transition to formulated diets. Affected birds showed acute weight loss, diarrhea, weakness, lethargy and possible vomiting. Ketonuria can be demonstrated by a reagent strip examination of the urine. Therapy should include dextrose, supportive alimentation and placing the bird back on its regular diet. The diet should then be changed gradually to prevent anorexia.

Clinical Conditions Associated with Malnutrition

Avian veterinarians encounter a different type of malnutrition today than was described five to ten years ago. Nutrient deficiencies were historically common, but with the use of formulated diets in combination with vitamin and mineral supplementation, many malnutrition problems noted today are a



FIG 31.1 A bird that is already adapted to a specific diet can be used to “teach” a reluctant eater to accept a new diet. In this case, a cockatiel is being used to acclimate a parakeet to a formulated food.

result of excessive nutrients.³⁰ In some cases clinical signs believed to be caused by malnutrition are actually complex diseases that involve nutritional, environmental and species-specific factors.

Obesity

Obesity is the most common and the most severe malnutrition-related problem recognized in avian practice (see Color 8). Obesity occurs if the energy content of the diet is excessive for the energy demands created by normal metabolic functions and the amount of exercise. In some cases, obesity will be secondary to the over-consumption of food in a bird attempting to consume missing nutrients. However, in most cases, obesity in companion birds is a result of feeding excess quantities of improper foods (eg, cookies, crackers, sweets) or high oil seeds (sunflower, safflower, hemp, rape, niger), a lack of exercise and increased food intake due to boredom (Figure 31.2).

Because companion birds frequently have limited opportunities for exercise, the energy content of their diet needs to be monitored closely. In species prone to obesity, it is important to avoid offering foods that have high caloric densities and to avoid excessive quantities of attractive, palatable food. Fats have twice the caloric density of either carbohydrates or proteins, and foods containing high levels of fats (such as peanuts or sunflower seeds) should be limited. Fresh fruit and vegetables have lower calorie densities than dried foods or seeds and should make up a sizable portion of a low-energy diet. Decreasing caloric intake can also be achieved by restricting feeding times (eg, ten minutes in the morning and evening) rather than offering food ad lib. Ideally, companion birds should be fed pelleted or extruded foods supplemented with small quantities of fresh fruit and vegetables. Some formulated diets may be helpful in controlling obesity and fatty liver problems.³⁰

Some species, such as Rose-breasted Cockatoos, Sulphur-crested Cockatoos, Amazon parrots and budgerigars, are particularly prone to becoming obese and may develop secondary lipomas, fatty liver degeneration and heart disease (see Colors 14 and 20). Pancreatitis has been associated with obesity and high fat diets.⁴⁰ Hypothyroidism, which can be associated with low dietary iodine, has been correlated with obesity and lipoma formation, particularly in budgerigars. In birds that are confirmed to have hypothyroidism, thyroxine supplementation is recommended (see Chapter 23).^{32,44,48}

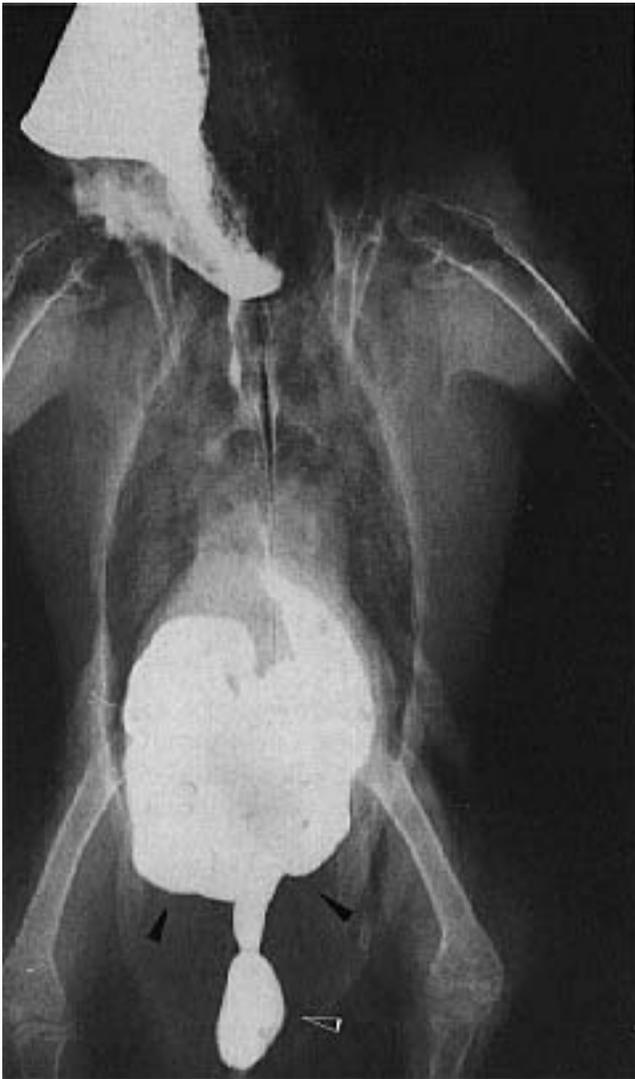


FIG 31.2 A two-year-old female Sulphur-crested Cockatoo was presented with a one-month history of progressive dyspnea (particularly following mild exercise) and abdominal wall masses. The bird was fed a seed diet ad lib and was frequently given treats (eg, crackers, potato chips). Several large pendulated masses were located pericloacally and in the ventral abdominal area. The bird weighed 1050 g. Cytologic evaluation of fine-needle aspirates from a mass was suggestive of adipose tissue. Abnormal clinical pathology findings included lipemic serum and hypercholesteremia. Radiographs indicated a diffuse soft tissue mass in the caudal abdomen. Barium contrast radiography indicated that the mass was displacing the abdominal organs cranially (arrow). Note that the cloaca is being compressed and the colon is oriented in a cranial, rather than its normal right lateral position (open arrow). The bird responded to a diet change (formulated diet, fruits and vegetables and no treats) and increased exercise (an outdoor flight enclosure).

A decrease in photoperiod may induce polyphagia and weight gain in pre-migratory birds. The increased food intake and weight gains appear to be mediated by thyroid hormones, prolactin and gonadotrophins. The role that neurohumoral signals,

such as a changing photoperiod, plays in weight regulation in companion birds is unknown.⁵⁶ It is common for pre-ovulatory females to have up to a ten percent increase in weight.

Low Body Weight/Poor Growth

Low body weight or poor growth can be the result of inadequate food intake, which in turn can be caused by an insufficient quantity of food, inappropriate diet, unfamiliar food items, infrequent feeding, weaning onto solid foods too early, or loss of appetite, maldigestion or malassimilation of food caused by medical problems.²⁰

Poor growth and low body weight occur with nutritionally deficient diets when the energy content of the food is insufficient to meet the energy demands of ongoing metabolic processes.

Low body weight or poor weight gain independent of organopathy can generally be corrected by placing the bird on a high-energy diet (high in fat and carbohydrates). Digestive enzymes and fiber hemicellulose may increase the digestibility and absorbability of the diet. Procedures for calculating daily energy requirements for birds are discussed in Chapter 15. It is important to note that formulas to calculate energy requirements are based on averages, and the nutritional requirements of individual species and individual birds will vary. A bird's clinical response to a particular diet should be carefully evaluated and adjustments should be made as necessary.^{42,48}

Diets for Birds with Malabsorption and Diarrheal Syndromes

Parasites, bacterial infections, mycotoxins and pancreatic disease may interfere with the absorption of nutrients from the digestive tract. In addition to correcting the primary problem, these birds need foods that are easily digested and absorbed to facilitate healing of the gastrointestinal tract. Lactose and excessive amounts of green vegetables should be avoided. Diets should be moderately low in fiber and provide easily digested carbohydrate (eg, canary seeds, millet, panicum, corn or hulled oats) and a moderate amount of highly digestible protein. Vitamin and mineral supplementation, particularly of vitamins A and E, may be needed. The addition of digestive enzymes to the diet may be useful (see Chapter 18). In some cases, feeding a small quantity of grit may improve digestion and aid absorption, but should be supplied only in low quantities to prevent gastrointestinal impaction.

■ Polyphagia

Occasionally birds will overeat fibrous food or grit, causing crop or ventricular impactions. These problems are more likely to occur if young birds are suddenly introduced to new food items (unhulled seeds, particularly). Birds that are exposed to a number of food items at an early age are less likely to overeat infrequently offered foods. Ostriches may eat constantly following relocation, leading to foreign body ingestion and impaction (see Chapter 48).⁴

Feigned polyphagia, in which a bird hulls seeds and appears to be eating but the crop remains empty, may occur in some birds that are very weak or that are offered inappropriate food items. Vitamin E and selenium deficiencies have been suggested as possible causes of this problem.²⁶ Clients should not rely on the husking of seeds to indicate food intake. Monitoring body weight and fecal output is more effective.

When changed to a formulated diet, older, obese budgerigars and cockatiels may lose weight, yet eat constantly. Obese birds should lose weight slowly to prevent hepatopathies associated with overwhelming fat metabolism. The weight loss can be tempered by adding some millet to the diet. Multivitamin injections and lactulose can be used to suppress progressive hepatopathies in some cases (see Chapter 8).

■ Polydipsia/Polyuria

Nutritional causes of polydipsia and polyuria include hypovitaminosis A, calcium deficiency, excess protein, hypervitaminosis D₃, excessive dietary salt, dry seed diet, formulated diets or a high percentage of dietary fiber.

Polyuria alone may occur in birds fed moist foods such as fruit, vegetables and semi-liquid diets. Urine or urate color may change from a normal white or cream to yellow or brown when birds are supplemented with B vitamins. Consumption of food dyes, berries and other fruits can also alter the urine color (see Color 8).

■ Digestive Disorders

White plaques in the mouth or swelling in the salivary ducts may be associated with hypovitaminosis A (see Colors 8 and 13).

Oral paralysis in cockatiels may be related to vitamin E and selenium deficiencies and a malabsorption syndrome secondary to giardiasis.²⁸

Nutritional causes of crop impaction include high fiber diets, foreign material ingestion (eg, juveniles eating various substrates such as wood shavings) and excess grit consumption. Cold food, a cold environment or infrequent feeding of large amounts of food may increase the risk of crop impaction in juvenile or debilitated birds. Repeated crop impactions may result in an atonic, pendulous crop. Degeneration of ventricular musculature has been associated with vitamin E and selenium deficiencies and calcinosis due to hypervitaminosis D.^{28,52} Crop liths may develop in birds on marginal diets. The etiology is undetermined (see Color 19).

Diarrhea may occur in birds fed low-fiber or high-fat foods, particularly highly processed human foods (eg, cakes, desserts, crackers). Bacteria or parasitic enteritis may occur in birds that eat foods contaminated with excrement. Food and water containers should be positioned so that contamination with droppings will not occur.

Nutritional cases of malabsorption or maldigestion (passing undigested food) include vitamin E and selenium deficiencies (sometimes associated with giardia infection), excess oil in the diet or dehydration. A lack of grit has been frequently discussed as a cause of maldigestion; however, companion birds on formulated diets do not appear to require grit. Studies in poultry indicate that the addition of grit increases the digestibility of feed by as much as ten percent,¹⁴ but similar studies have not been performed in companion birds. Given that obesity is more of a problem than maldigestion in companion birds, increasing the digestibility of a formulated diet that exceeds suggested nutritional requirements is probably unnecessary. Charcoal that is used in some grit mixtures may interfere with the absorption of vitamins A, B₂ and K and contribute to deficiencies of these compounds.

Birds should not be offered grit on an ad libitum basis. If offered free choice, some birds may over-consume grit, leading to crop, proventricular or ventricular impactions. This problem is reported commonly in North America but appears to be uncommon in Australia. The cause for a regional variation in the occurrence of this condition is unknown. Birds showing compulsive grit consumption should be evaluated for hepatopathy, pancreatitis, renal dysfunction and general malnutrition.

There is a difference between grit and crushed shell. Grit is composed of minute stones and commonly

contains silicates and sandstone. Crushed shell is almost entirely composed of limestone (calcium carbonate) and is readily digested by acids in the proventriculus. Crushed shell will provide a source of calcium, and is not effective in aiding in the mechanical breakdown of dietary plant material. Heavy metal toxicity has been associated with feeding crushed shell derived from contaminated sources (oysters raised in polluted waters).

Regurgitation has been associated with feeding high-protein diets to cockatiels.⁴⁶

Respiratory Disorders

Dyspnea (extended neck) and wheezing may be associated with goiter, particularly in budgerigars.⁵³ Hypovitaminosis A leads to squamous metaplasia of epithelial surfaces causing obstruction of respiratory passages or sinusitis (see Color 8). Dyspnea may be caused by calcium or vitamin D₃ deficiency if severe enough to demineralize bone, causing thoracic or spinal deformities.

Asphyxiation may occur from aspiration of feeding formula into the respiratory tract. This can occur if a tube is accidentally placed in the trachea when attempting crop feeding or if a bird (particularly a weak bird) is fed large amounts or excessively thin formula.

Plumage Abnormalities

Dark, horizontal lines (stress marks) on feathers have been associated with nutritional deficiencies (particularly methionine) and indicate that a release of corticosteroid hormone occurred while the feather was developing. Stress lines are common in neonates that have had a disrupted feeding schedule or in raptors that are molting while in a training period (see Color 24). Molting abnormalities, retained feather sheaths and dry flaking beaks have also been associated with overall nutritional deficiencies (Figure 31.3).

Feather picking may be initiated by dry, flaky, pruritic skin, which in turn can be caused by nutritional deficiencies, particularly deficiencies of vitamin A, sulfur-containing amino acids, arginine, niacin, pantothenic acid, biotin, folic acid and salt. Excessive dietary fat has been incriminated as a possible cause of self mutilation (Figure 31.4).

Deficiencies of minerals such as calcium, zinc, selenium, manganese and magnesium may be associated



FIG 31.3 An 18-month-old Amazon parrot on an all-seed diet was presented for a beak trim. The bird was maintained indoors and had no exposure to sunlight or water for bathing. Horny beak material that is dry and flaky, as well as black discoloration of the feathers are typical of malnutrition. This bird responded to a change in diet and daily exposure to direct (unfiltered through glass) sunlight.

with brittle, frayed feathers and dermatitis.²⁹ Arginine deficiency may cause wing feathers to curl upward in chicks. In broilers, pantothenic acid deficiency causes the formation of ragged feathers, while a deficiency in growing cockatiels has been associated with a lack of contour feathers.⁴⁶

The association between diet and feather pigment has long been recognized by canary breeders. Carotene and xanthophyll pigments, which originate from plant material, are found in fat globules in the feathers and give rise to yellow, orange and red colors (see Chapter 24). Birds lacking a dietary source of carotenoids may develop muted feather or skin colors, while dietary supplementation of carotenoids in birds with suitable genetic backgrounds will result in increased depth of color.

Prolonged feeding of bacon rind and bone marrow has been associated with an oily feather and stool texture (steatorrhea) and an increase in depth of the pink feathers in Rose-breasted Cockatoos. Raptors fed laboratory rats and mice (reduced carotenes) may lose the yellow coloration of their cere, feet and legs that is characteristic in free-ranging birds.^{32,38,39} Porphyrins are aromatic compounds synthesized by birds that may produce colors such as red, green or brown. Porphyrins are less sensitive to dietary influences than carotenoids, but both are present in edible blue-green algae, and enhanced feather coloration would be expected in birds fed a diet containing this material.



FIG 31.4 Feather picking and dry, thin, pruritic skin are common signs of malnutrition in Psittaciformes. Note the loss of papillae on the dorsal surface of the feet and toes.

Melanin occurs in granules in the skin and feathers and produces black, brown and red-brown colors. This pigment is derived from tyrosine in an enzymatic reaction requiring copper. Consequently, deficiencies of tyrosine (or other related amino acids) or copper could interfere with melanin production and cause dark-colored feathers to become lighter.²⁵

Blue and white are structural colors in feathers. In most cases, their occurrence depends on a scattering of light caused by the structure of the keratin in the spongy layer of the feather rami rather than on the presence of pigments. Essential amino acids that occur in keratin include methionine, histidine, lysine, tryptophan, threonine, isoleucine and valine. It is possible that amino acid deficiencies could alter the structure of keratin and consequently alter feather color. A change in feather color from green to yellow is usually caused by a loss of structural blue color, which may be associated with essential amino acid deficiencies. While this color change is commonly seen in nutritionally deficient Psittaciformes, the exact nature of the deficiency has not been clarified, and it is possible that more than one amino acid could be involved (see Color 24). Lysine deficiency has been discussed as one possible cause of green-to-yellow feather discoloration because many affected birds are consuming all-seed diets that are low in lysine.

Feather color may change from blue to black, green to black or grey to black in birds that are sick or malnourished. These color changes are associated with altered keratin structure in the spongy layer that prevents normal light scattering. When this

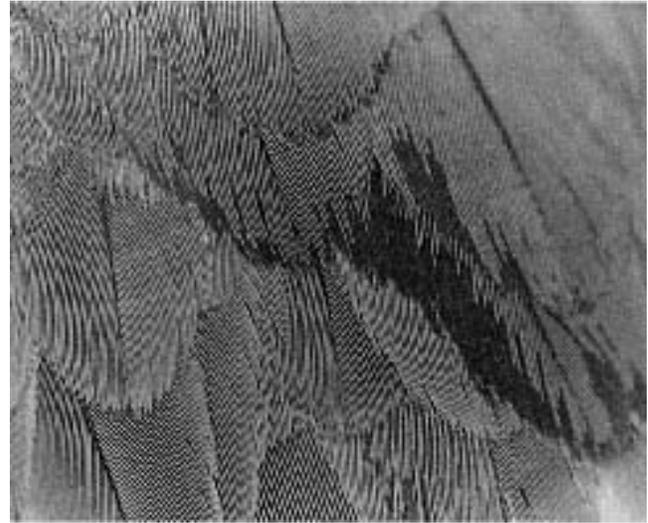


FIG 31.5 Feathers that turn black are an indication of altered keratin structure in the spongy layer of the feather. The black feathers in this Amazon parrot resolved with a change in diet (seeds to formulated diet) and correction of chronic active hepatitis.

occurs, melanin granules in the middle of the feather, if present, would absorb all wave lengths of light, giving the visual effect of black (Figure 31.5).

Nutritionally related alterations in feather color may vary based on the species of bird, specific nutrient deficiency, timing of the deficiency in relation to feather development and the initial color of the affected feathers. While lysine deficiency in chickens, turkeys and quail produces achromatosis, there was no loss of feather color in young cockatiels fed a lysine-deficient diet. However, choline and riboflavin deficiencies produced feather changes in young cockatiels that resembled achromatosis caused by lysine deficiency in poultry.⁴⁶ White streaks (usually associated with breakage) in feathers may be associated with a hypovitaminosis B (Figure 31.6).²⁹

■ Skin Changes

Plantar corns and pododermatitis have been associated with biotin and vitamin A deficiencies, particularly in obese birds (Figure 31.7).²⁹ Edema of subcutaneous tissues has been seen with vitamin E and selenium deficiencies. Exfoliative dermatitis on the face and legs has been associated with biotin, pantothenic acid, riboflavin or zinc deficiency¹ (see Color 48.). If a formulated diet is not available, a diet can be supplemented with multivitamins to compensate for any nutritional deficiencies. Several kiwis in a New Zealand zoo developed a scaly dermatitis over their necks and legs when a multivitamin supple-



FIG 31.6 White streaking in feathers that are normally colored may be associated with a malnutritional deficiency. These feathers are frequently brittle and may break at the site of abnormal coloration.

ment that was routinely included in their diet was omitted. The clinical problem resolved when the multivitamin supplement was again added to the diet.⁵ Formulated diets should not be supplemented with vitamin products. Over-supplementation may cause problems with excess vitamin, mineral, fat or protein consumption.

■ Skeletal and Muscular Disorders

Demineralized, bent bones and pathologic fractures may occur in birds with hypovitaminosis D and calcium, phosphorus or magnesium deficiencies or imbalances.

Airplane wing in waterfowl (rotation of the distal carpi due to heavy, developing feathers being supported by non-rigid, developing bones) may be caused by rapid growth or excessive levels of protein or low levels of calcium (see Chapter 46).¹⁷

Slipped tendon of the hock (perosis) may occur with manganese, biotin, pantothenic acid or folic acid deficiencies (see Color 8). Obese birds that are not allowed sufficient exercise and birds fed high-mineral diets may be prone to this condition. There is gross enlargement of the tibio-metatarsal joint, twisting and bending of the distal tibia and slipping of the gastrocnemius muscle from its condyles. Young gallinaceous birds, cranes and ratites are particularly susceptible to this condition.³⁴ The author has seen a similar condition in raptors, cockatiels and rosellas. In some cases, surgical correction is possible (see Chapter 46).



FIG 31.7 A mature female cockatiel was presented with a complaint of intermittent lameness. The bird was on an all-seed diet with no supplementation and was kept in a small enclosure. The bird weighed 138 g and had severe ulcerative lesions on both metatarsal pads. Changing the diet, increasing the exercise (outdoor flight enclosure) and standard treatment for grade 4 bumble-foot were effective in resolving the lesions.

Enlargement of the hock, without tendon slipping, may occur with zinc deficiency.

Tibial dyschondroplasia is characterized by uncalcified masses or plugs of avascular hyperplastic cartilage in the proximal metaphyses, particularly of the tibiotarsus. The condition is seen in poultry and ratites. A genetic predisposition along with electrolyte imbalances involving sodium, potassium and chloride are thought to be involved in the development of tibial dyschondroplasia. A relative excess of chloride may increase the incidence of disease.³³

■ Neurologic Signs

Seizures or localized paralysis have been associated with salt toxicity and low levels of thiamine, calcium and vitamin E. Leg paralysis has been associated with calcium, chloride or riboflavin deficiency.

Cervical paralysis has been associated with a folic acid deficiency. Jerky leg movements have been associated with pyridoxine deficiency.

Sudden collapse or fainting has been associated with hypoglycemia in raptors or in other species when a bird has not eaten and is acutely stressed. Syncope is characteristic of advanced hypocalcemia in African Grey Parrots.

Behavioral changes including aggressiveness (biting), nervousness, rejection of food and regurgitation occurred in cockatiels placed on a high-protein diet.⁴⁶

Frequently, companion birds that are switched from an unbalanced all-seed diet to a balanced formulated diet will undergo a corresponding change in behavior characterized by decreased biting, screaming and chewing, and increased activity and playfulness.³⁰

Reproductive Disorders

Many dietary deficiencies or excesses may result in reduced reproductive performance due to infertility, poor hatchability or nestling deaths. Calcium, vitamin E and selenium deficiencies may be associated with egg binding.

General Ill Health or Sudden Death

Fatty liver infiltration may occur due to high fat diets, fatty acid or B vitamin deficiencies and high-energy diets in exercise-deprived birds (see Color 20). Gout may be a precipitating cause or an end result of systemic diseases (see Color 21). Ascites may be associated with excessive dietary levels of iron in birds susceptible to iron storage disease (hemochromatosis). Atherosclerosis may be associated with diets high in fat and cholesterol (see Color 14).

Aortic rupture has been associated with copper deficiency in poultry and is suspected to occur in ratites (see Color 48).

Immune Response

Adequate levels of both B complex (particularly pantothenic acid and riboflavin) and vitamin E have been shown to improve the body's response to pathogens. In poultry, vitamin C and zinc are involved in T-cell response, and vitamin C stimulates macrophages and helps to counter the immunosuppressant effects of stress. Low vitamin A levels may result in a sub-optimal immune response and have been associated with the occurrence of aspergillosis in psittacines.^{3,13}

Deficiencies of Specific Nutrients

When one considers the array of ecological niches to which different species of birds are adapted, it is not surprising that there are major species differences in

the ability to metabolize proteins, fats, carbohydrates and other nutrients.

To date, comprehensive nutritional requirements have been established only for domestic fowl. In spite of the absence of complete data for companion birds, anecdotal findings and scientifically supported investigations suggest that general health and reproductive success will be greater in birds fed "balanced" formulated diets supplemented with limited fresh fruits and vegetables compared to birds fed seeds supplemented with fresh fruits and vegetables (Figure 31.8).^{30,54}

Research findings and clinical experience suggest that there is considerable interspecies variation in nutrient requirements and in clinical signs of malnutrition. For example, some finches may consume up to 30% of their body weight, budgerigars, 25% of body weight and domestic chickens, 6% of body weight on a daily basis. These differences point to the dangers of extrapolating nutrient requirements, particularly of minerals, from poultry data when the level of food consumption varies dramatically. Requirements will also vary depending on the bird's age and physiologic state, interactions with other dietary components and the presence of concurrent diseases.

Protein and Amino Acids

Protein in the diet is broken down into component amino acids before being absorbed by the intestine.



FIG 31.8 Birds appear to be healthiest when supplied a formulated diet supplemented with some fresh fruits and vegetables. Over-supplementation with fresh foods, as is the case with this daily vegetable bowl for a cockatoo, can actually cause malnutrition through insufficient consumption of a formulated diet. For a bird the size of an Umbrella Cockatoo, the formulated diet should be supplemented with the equivalent of several slices of carrot (or dark squash or sweet potato), one-eighth cup of spinach (or broccoli or endive) and several small slices of favorite fruits as a treat.

Amino acids are needed by the body to reconstruct proteins that make enzymes, hormones, muscles, bones and feathers. Birds are uricotelic, ie, the product of protein breakdown is uric acid, which is excreted as a slurry (urates) by the kidneys.

Excess Dietary Protein

Dietary protein requirements vary dramatically between species. Broiler chickens and turkeys have been genetically selected for rapid growth and are fed high protein levels to achieve maximum growth rates. These feeding practices are rarely appropriate in other species. Starter rations for turkey poults or pheasants may contain nearly 30% protein, but young ratites, waterfowl and psittacine birds require much lower levels. Using a high-protein diet in these latter species may result in clinical problems such as airplane wing in ducks, deformed legs in ratites, poor growth rates in psittacine birds and increased susceptibility to disease in all species.

Inappropriate calcium levels in the diet may compound problems caused by excessive dietary protein. A group of macaw neonates being fed a human, high-protein baby cereal with added vitamins and calcium showed suboptimal growth rates. When the protein level in the diet was reduced by adding pureed fruits and vegetables, the growth rate and the chick's general health improved dramatically (see Chapter 30).

Nutritional data collected in juvenile cockatiels indicated that a protein level of 20% was optimal for this species. Levels of 10% produced stunting, poor growth and high mortality; levels over 25% produced transient behavioral changes such as biting, nervousness, rejection of food and regurgitation.⁴⁶

In budgerigars, one study showed that a protein level of 17 to 20% was optimal. Birds on low-protein seed diets increased their food intake and gained weight in the form of excessive body fat. Those on low protein (12%) mash diets lost weight, but some died with their crops packed with food. This finding suggests that the birds unsuccessfully attempted to consume enough food volume to compensate for the protein deficiency. Birds fed high-protein diets were very thin.⁵⁵ Other studies in budgerigars indicate that a diet with 2% lysine and 10% protein (13 kcal/kg of body weight) is ideal.¹²

Gout is the deposition of uric acid crystals on body organs (visceral gout), in joints (articular gout) or in the ureters (renal constipation) (see Color 21). High dietary levels of protein and calcium, hypervitaminosis D₃, poor kidney response, dehydration, cold

weather and other stress factors work in concert to interfere with the kidney's ability to adequately excrete uric acid.^{15,35} Hypervitaminosis D₃ causing renal calcinosis or vitamin A deficiency causing squamous metaplasia of the ureters may exacerbate blockage of the ureters.

Diets for Birds with Renal Disease or Gout

Birds with renal disease or gout should be provided diets that decrease the workload of the kidneys and slow the loss of renal function. These diets should be lower in protein and meet energy needs with non-protein calories. Calcium, phosphorus, magnesium, sodium and vitamin D₃ levels should be reduced to avoid renal mineralization. Vitamin A should be present in adequate amounts to ensure proper function of the mucous membranes lining the ureters. B vitamins should be increased to compensate for losses associated with polyuria.

Protein and Amino Acid Deficiencies

Protein or specific amino acid deficiencies are occasionally encountered in companion birds, particularly in insectivorous birds (softbills). Insectivorous birds require higher protein levels than granivores and generally require live food such as crickets or mealworms. If these insects are reared exclusively on bran, their total body protein may be low, and consequently the level and quality of protein that they provide to birds will also be low. Clinically, insectivores receiving low-protein insects will have a history of recurrent disease problems. Feeding crickets that have been raised on dried dog food or encouraging insectivores to consume artificial diets with appropriate levels of high quality protein prevents the problem.

Many seeds are relatively low in total protein and may also be deficient in some essential amino acids such as tryptophan, methionine, arginine or lysine. Free-ranging, seed-eating birds will frequently eat insects, particularly during the breeding season and when raising young.

Deficiencies of individual amino acids may cause abnormal feathers as well as suboptimal growth and poor breeding performance. Deficiencies of essential amino acids are most likely to occur if birds are fed a diet restricted to one or two individual types of seeds.

Serine, glycine and proline are the most abundant amino acids in feather keratin while methionine, histidine, lysine and tryptophan occur at lower levels. Methionine content of chicken feathers decreases

with age, while that of threonine, isoleucine and valine increases. Lysine deficiencies have been associated with impaired feather pigmentation in poultry, but not in cockatiels.⁴⁶

Methionine deficiency has been associated with stress lines on feathers and fatty liver change. Cystine and methionine act as sources of glutathione, which has a sparing effect on vitamin E.

■ Fats and Essential Fatty Acids

Fats provide a concentrated source of energy. Linoleic and arachidonic acids are essential fatty acids needed for the formation of membranes and cell organelles. Deficiencies of linoleic acid may be associated with decreased metabolic efficiency, decreased growth, hepatomegaly, increased fat storage, decreased reproduction, embryonic mortality and decreased hatchability. In mammals, lipogenesis occurs mainly in adipose tissue while in birds, it nearly all occurs in the liver. T₃ is believed to be associated with lipogenesis and calorogenesis, especially during migration, while T₄ is associated with reproduction and molt (see Chapter 23).⁵⁶

Lipogenic liver function in birds predisposes them to the occurrence of conditions involving excessive accumulation of liver fats, for example, fatty liver and kidney syndrome in young chickens and fatty liver hemorrhagic syndrome in laying hens (FLHS).³³ Geese that are force-fed cream and not allowed to exercise in preparation for *pate de foie gras* may have a six-fold increase in liver weight with only a two-thirds increase in weight.²⁴

Fatty liver syndromes of undetermined etiologies are common in companion birds (see Color 20). In addition to fatty liver, excessive levels of fat in the diet are known to cause obesity, diarrhea and oily feather texture, and to interfere with the absorption of other nutrients such as calcium. Paradoxically, lack of fatty acids can also result in fatty liver infiltration because essential fatty acids are needed for lipid metabolism. Poor growth and reduced resistance to disease also occur with essential fatty acid deficiencies. FLHS in poultry is associated with high carbohydrate, low-fat, selenium-deficient diets given ad lib.

If fats become rancid, essential fatty acids may be destroyed, amino acid availability may be reduced and peroxidases may be produced that interfere with the activities of fat- and water-soluble vitamins (biotin). Rancid foods have been shown to reduce growth

and egg production in poultry. Levels of peroxide exceeding 15 mEq/kg were found to be toxic. Changes in the taste or odor of rancid food stuffs did not occur until the peroxide level reached 90 mEq/kg. Rice and oats are particularly susceptible to becoming rancid and are processed for foods through extrusion, rolling or roasting. Many commercial diets contain antioxidants (propylene glycol or ethoxyquin) to prevent foods from becoming rancid. The long-term effect of these products on birds is unknown.

Ventricular erosion may occur in birds fed highly polyunsaturated fatty acids (such as those present in cod liver oil), if the fatty acids are not protected by an adequate dietary level of vitamin E. "Gizzerosine" has been associated with ventricular ulceration in poultry fed heated fish meal. Because of these problems, fish liver oils are not recommended as dietary components in companion birds.^{26,49} Soybean oil is a good source of fatty acids that is less likely to spoil.

Atherosclerosis may be induced by diets high in saturated fats and cholesterol. This problem is occasionally seen in aged Psittaciformes and may be associated with long-term feeding of high-oil seeds such as sunflower and safflower (see Chapter 27).³⁴

Young cockatiels were able to tolerate fat levels from 1 to 60% of the diet with no effect on growth. However, about half of the birds fed a 60% fat diet developed a necrotic crop infection and died.⁴⁶

■ Carbohydrates

Carbohydrates are a source of energy in the diet and are readily converted into fats in the liver. Exercise-deprived birds on high-energy diets may develop fatty liver infiltration even though carbohydrates, rather than fats, form the major component of energy consumed.

Clostridial infections, in which gas fermentation occurs along the gastrointestinal tract, have been associated with high-sugar diets in nectivorous birds.^{20,23}

Birds have blood glucose levels that are several times higher than those of mammals. Some species, such as penguins and sea birds, are adapted to tolerate long fasting periods during molting, egg incubation or migration. Small companion birds (eg, finches) may collapse from hypoglycemia if they are deprived of food for even short periods. Food restriction prior to anesthesia should not exceed several hours. Raptors that are fed small quantities of food as part of their training program may experience hypoglycemic col-

lapse and may require emergency therapy with oral or parenteral glucose. Glucagon, rather than insulin, is the principal director of carbohydrate metabolism in birds.⁵⁶

Diets for Birds with Hypoglycemia

Birds prone to hypoglycemia should be fed frequently with nutrients that are slowly converted to glucose (a high-protein, high-energy diet). In most cases, hypoglycemia is dietary-induced, and placing the bird on a diet appropriate for that species is all that is required.

Vitamins

Vitamins are a mixed group of organic compounds that are essential for a variety of metabolic processes. Most birds require the same vitamins as mammals with the exception that vitamin D₃ (not vitamin D₂, as in mammals) is the active form of this compound. Exogenous vitamin C is required in fruit-eating birds such as bulbuls, but seed-eating species are generally able to synthesize vitamin C. Debilitated birds may have higher requirements and a reduced ability to synthesize vitamin C, and should be supplemented orally or parenterally.

Birds with vitamin deficiencies may have life-threatening clinical signs (eg, seizing associated with thiamine deficiency) or simply appear ruffled and in poor condition. Vitamins A, C, E and B complex are all involved with immune responses, and deficiencies in these compounds may increase the severity of infectious diseases.

Antibiotics may induce vitamin deficiencies by interfering with normal intestinal microflora. In most cases, birds given long-term antibiotics should also receive multivitamin supplementation. Protozoan infections such as coccidiosis or giardiasis may interfere with the absorption of vitamins (such as vitamins A or E) from the intestinal tract. Vitamins are sensitive to heat and light, so overheated or outdated commercial foods may be vitamin deficient.

Hypervitaminosis, particularly with fat-soluble vitamins, is becoming increasingly common as clients over-supplement improved, formulated avian diets.

Fat-soluble Vitamins

- **Vitamin A:** Vitamin A is formed in the liver from beta carotene. It is involved in mucopolysaccharide biosynthesis and is needed for the formation of normal mucous membranes and epithelial surfaces, for

growth, for vision, for the development of the vascular system in embryos, for the production of adrenal hormones and for the formation of red and orange pigments in feathers. Beta carotene and vitamin A, themselves, are colorless. It is their derivatives that are responsible for feather pigmentation. Low vitamin A in the diet may result in a suboptimal immune response.³

Numerous clinical problems may be associated with hypovitaminosis A. Squamous metaplasia of mucous membranes may occur, altering the function of the respiratory, gastrointestinal or urogenital systems. Hyperkeratosis, a related condition, may affect epithelial surfaces (Figure 31.9).¹³

Small white pustules may be seen in the mouth, esophagus, crop or nasal passages. If squamous metaplasia causes blockage of salivary ducts, small swellings (often symmetrical) may be noted dorsally around the choana, around the larynx and laterally under the tongue or mandibles (see Figure 19.3). White caseous material may accumulate in the bird's sinuses, particularly if hypovitaminosis A is associated with a concurrent sinus infection. Squamous metaplasia may also lead to thickening and sloughing of part of the lining of the syrinx with subsequent partial or complete tracheal obstruction (see Color 8). Xerophthalmia occurs if squamous metaplasia affects the eyes. There may be lacrimation, and caseous material may accumulate under the eyes (see Color 26). In chicks, acute hypovitaminosis A has been associated with weakness, incoordination and ataxia. These symptoms must be differentiated from "crazy chick disease" caused by hypovitaminosis E.

In mild cases of hypovitaminosis A, particularly in budgerigars, the only clinical signs may be polyuria and polydipsia, but squamous metaplasia may be seen histologically along the gastrointestinal and urinary tracts. Kidney damage and gout may occur if squamous metaplasia causes partial or complete occlusion of the ureters.

Reduced egg production, egg binding or poorly formed egg shells (pitted) are common in hens with hypovitaminosis A. In cocks, hypovitaminosis A may cause decreased sperm motility, reduced sperm counts and a high level of abnormal sperm.

Hypovitaminosis A may cause hyperkeratosis of the plantar skin of the metatarsal and digital pads (see Color 8). The normal papillary scale structure is lost and the corneum is thickened. Focal hyperkeratosis (corns) often occurs on the metatarsal pads (see Fig-

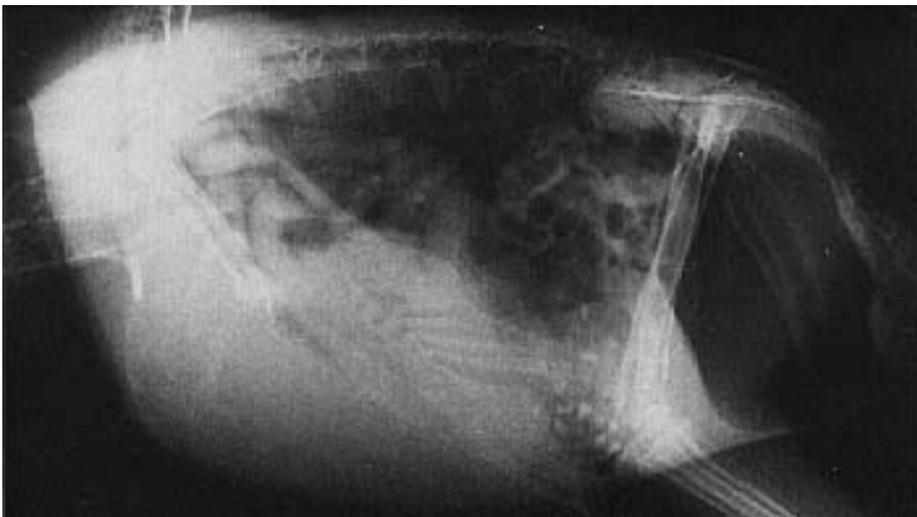
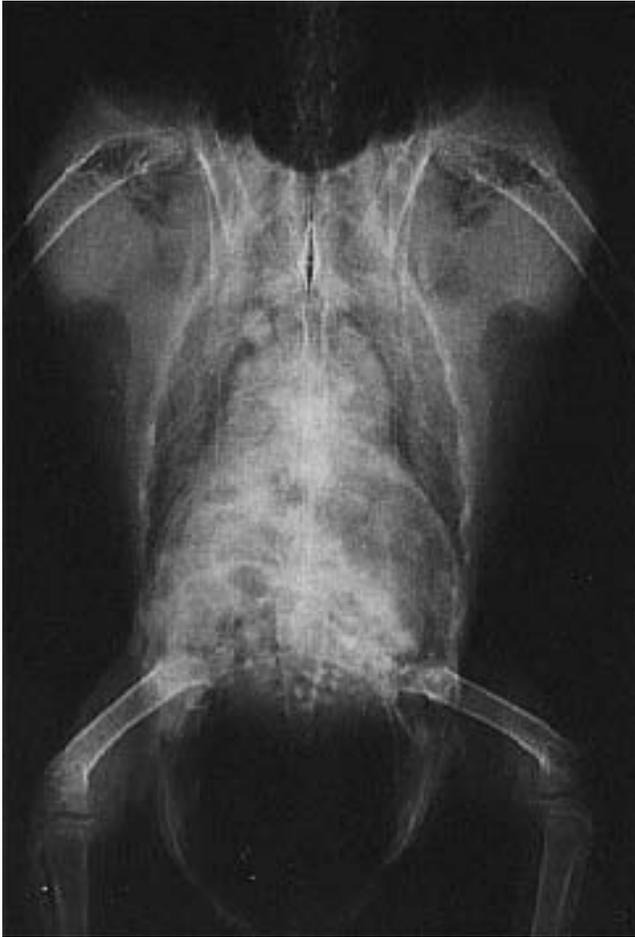


FIG 31.9 An adult female Amazon parrot was presented for severe dyspnea (open-mouthed gasping) and diarrhea. A large, ulcerated pharyngeal mass was evident on physical examination. Radiographic changes included gaseous distension of the crop, gastrointestinal tract and cloaca. The gaseous distension (aerophagia) was related to gasping for air associated with an occluded glottis. The bird did not respond to emergency care. The oval mass was characterized by marked epithelial acanthosis and parakeratosis (see Color 8). The lungs were congested and hemorrhagic. Cystic hyperplasia was evident in the pancreatic ducts. The diagnosis was severe hypovitaminosis A and syringal granuloma.

ure 31.7). These changes predispose birds to pododermatitis (bumblefoot). Other factors apart from malnutrition are also associated with this condition.^{22,27} In young ducks, hypovitaminosis A has been shown to cause retardation of endochondral bone growth.

In a number of avian species, vitamin A levels in the liver of less than 50 IU/gm have been found to correlate with the occurrence of squamous metaplasia elsewhere in the body.³⁴

Hypovitaminosis A should be initially treated with parenteral supplementation, which establishes rapid blood levels and does not rely on intestinal absorption. In limited clinical trials, some birds may respond just as quickly to supplementation of the diet with spirulina (Harrison GJ, unpublished). Oral administration in the food and modification of the diet to include natural sources of beta carotene is recommended. Zinc levels in the diet should be sufficient to allow for normal vitamin A function. Liver disease may decrease the bird's ability to store vitamin A.

- **Vitamin D** helps to stimulate gastrointestinal absorption of calcium, has a hormonal effect on regulation of calcium and phosphorus excretion in the renal tubules and may be involved in controlling alkaline phosphatase in the blood. An increase in alkaline phosphatase may be an early indication of hypovitaminosis D₃.

Ingested vitamin D precursors are converted to the active form of the vitamin (vitamin D₃ in birds) in the skin. Alternatively, vitamin D precursors in the uropygial gland may be spread on the feathers, activated by UV light and then consumed during preening activities. This process requires natural sunlight or appropriate artificial ultraviolet light. Low levels of calcium in the diet, particularly if associated with high levels of phosphorus, will precipitate hypovitaminosis D₃.⁵³ If groups of juvenile birds are fed diets low in calcium and vitamin D₃, birds in shady flights may show overt signs of rickets while those in sunny flights will be normal. Hypovitaminosis D₃ can easily occur in birds raised indoors. It is advisable to sup-

plement indoor birds that do not have access to natural sunlight with exogenous vitamin D₃.

Signs of vitamin D₃ deficiency parallel those of calcium deficiency. Adult hens may show thin-shelled or soft-shelled eggs, decreased egg production and poor hatchability. Seizuring or leg weakness may occur due to pathologic bone fractures or if an already low blood calcium level is further exacerbated by metabolic demands of egg laying (Figure 31.10).

Hypovitaminosis D₃ in neonates is characterized by demineralized and easily broken bones. Leg bones will frequently be bent into grossly distorted posi-



FIG 31.10 Metabolic bone disease is common in birds with hypovitaminosis D₃ and hypocalcemia, as well as in birds consuming diets with low calcium to phosphorus ratios. An 11-week-old African Grey Parrot was presented for limb deformities. The bird was being fed a commercial hand-feeding formula that was rendered ineffective with the addition of oatmeal, strained meat, mashed fruit, baby vegetables and neo-calglucon. Radiographs indicated bending type deformities in the right humerus, pelvis and both femurs and tibiotarsal bones. The case was managed by removing the supplements from the hand-feeding formula.

tions. The sternum may be bent laterally or indented. The spinal column may undergo lordosis or fracture easily, causing pressure on nerves and subsequent paralysis. Radiographically, bones will show poor density and pathologic fractures may be apparent (Figure 31.11).

High levels of vitamin D₃ (>10⁶ IU/kg of food compared with recommended levels of around 2000 IU/kg of food) in chickens may cause calcification of renal tubules and arteries, visceral calcinosis, urate nephrosis and visceral gout. Excessive levels of vitamin D precursors may also be toxic. Clinical evidence suggests that young macaws may be particularly susceptible to hypervitaminosis D. Nephrocalcinosis, suspected to be associated with hypervitaminosis D, has been reported in a dove, a toucan, a cardinal and a variety of Psittaciformes (Figure 31.12).³⁰ Home-



FIG 31.11 Rickets in a juvenile Blue and Gold Macaw. Note the lack of bone density, bending of the bones and pathologic fractures (courtesy of Marjorie McMillan).

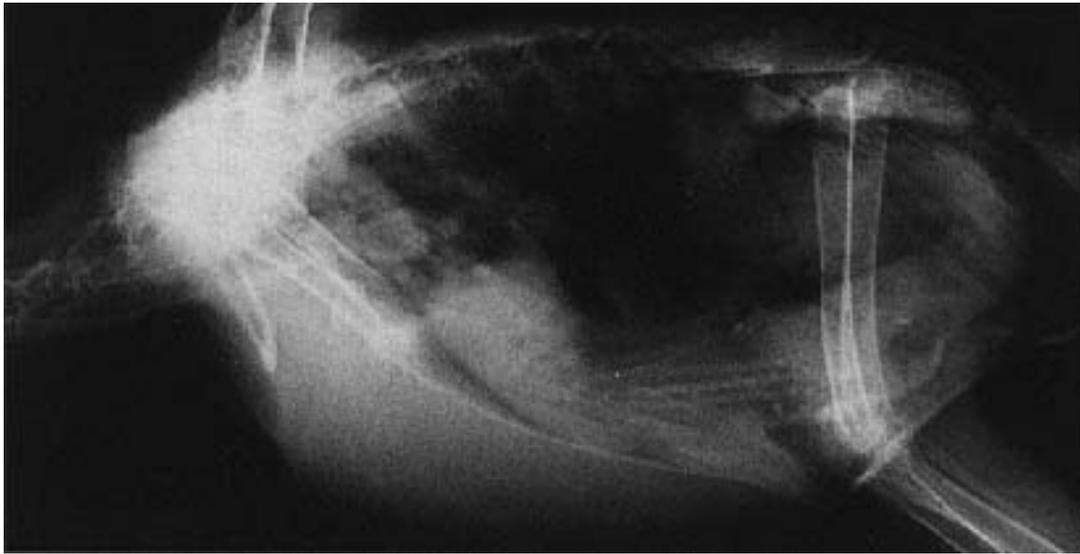


FIG 31.12 An African Grey Parrot was presented for a pre-purchase examination. Physical and clinical pathology findings were unremarkable. The bird was negative for PBFV virus and polyomavirus by DNA probe testing. Radiographs indicated mineral densities in the kidneys. This is a frequent finding in African Grey Parrots and its clinical importance is unknown. However, calcium supplementation and injectable vitamin A and D₃ in a bird with this condition are contraindicated. Note the extensive air sac capacity, which appears to be normal in African Grey Parrots.

made dietary formulas (particularly those for neonates) are likely to contain improper levels of many nutrients.^{41,52} Neonatal birds are best fed proven formulas (see Chapter 30).

- **Vitamin E:** Vitamin E is an antioxidant that acts to prevent fat rancidity and fatty acid degeneration in foodstuffs, as well as acting in concert with selenium and sulfur-containing amino acids to prevent peroxidative damage to cell membranes. Birds on high-fat diets, particularly if the fat has become rancid, require higher amounts of antioxidants, and consequently are more likely to show signs of vitamin E deficiency than birds on diets low in fat.

If accompanied by deficiencies in sulfur-containing amino acids or selenium, hypovitaminosis E may result in skeletal muscle dystrophy as well as muscular dystrophy of the heart or ventriculus. Electrocardiographic changes may accompany heart muscle dystrophy. Undigested seed in the droppings may occur with ventricular muscular dystrophy. Degeneration of the pipping muscle may occur in neonates, resulting in decreased hatchability. Exertional rhabdomyolysis or spraddle legs may be associated with vitamin E and selenium deficiencies (see Chapter 48).^{11,34}

Muscle weakness, localized wing paralysis, poor digestion and embryonic and hatchling mortality have been described in cockatiels that responded clinically to vitamin E and selenium therapy. Giardiasis may

have been a predisposing factor. Elevated levels of serum creatinine phosphokinase may suggest nutritional myopathy.²⁸

Hypovitaminosis E may cause encephalomalacia in poultry and other species. This condition can be prevented by supplementing the diet with linoleic acid but not arachidonate. *Neophema* parrots fed a dog food that contained a high amount of rancid fat and seed soaked in cod liver oil showed incoordination, abnormal body movements and torticollis. At necropsy, affected birds showed cerebellar demyelination and muscular dystrophy of the heart and skeletal muscle typical of vitamin E deficiency.⁸ A similar vitamin E and selenium-responsive syndrome has been reported in *Eclectus* Parrots in Switzerland.⁴⁷

Deficiencies in vitamin E and selenium may cause exudative diathesis, which results in edema of ventral subcutaneous tissue in poultry. Hypovitaminosis E is one of a number of dietary factors that has been associated with enlarged hocks in turkeys. Prolonged hypovitaminosis E may cause testicular degeneration in males, and in hens it may result in infertility or early embryonic deaths.

In mammals, a degenerative disease of fat (yellow fat disease, steatitis) has been associated with hypovitaminosis E. A similar condition has been recognized in birds fed fish with high fat content such as herring, smelt or red meat tuna. Ceroid, a pigment that is

considered pathognomonic for yellow fat disease in mammals, was identified histochemically in the fat from affected birds.⁹

Birds suspected of hypovitaminosis E should receive parenteral supplementation. Mild conditions may respond dramatically to this treatment. In cases where there is irreversible nerve or muscle damage, response is poor (see Chapter 18).

- **Vitamin K:** Vitamin K is required for the synthesis of prothrombin. Deficiency of vitamin K results in prolonged prothrombin time and delayed blood clotting. Affected birds may exsanguinate from minor traumatic injuries. Bacterial flora in the intestine are the natural source of vitamin K. Clinical problems associated with bleeding or petechia from pulled feathers may respond to injectable vitamin K, but naturally occurring hypovitaminosis K has not been proven in companion birds.¹⁸ Sulfaquinolone has been reported to induce hypovitaminosis K in poultry, and it is possible that long-term antibiotics used in aviary birds could do likewise.

Water-soluble Vitamins

- **Thiamine (Vitamin B₁):** Thiamine deficiency may lead to loss of appetite, opisthotonos, seizures and death. Deficiency of thiamine is uncommon in birds on a seed diet because seeds and grains generally contain sufficient thiamine. Thiamine deficiency-induced seizures and neurologic signs may occur in carnivorous birds fed solely on meat or day-old chickens, and in fish-eating birds fed fish containing thiaminase.¹⁷ Free-ranging honey-eaters in urban areas of southern Australia may develop thiamine deficiency during the winter. This is thought to be associated with the planting of exotic ornamental trees that provide inadequate nutrition but encourage the birds to remain in an urban area rather than properly migrate.

Response to treatment in thiamine deficiency cases can be dramatic. Affected birds will respond within minutes to injectable thiamine. Response to oral thiamine may also be rapid.

- **Riboflavin (Vitamin B₂):** In young chicks, riboflavin deficiency causes weakness and diarrhea, but the bird's appetite remains normal. Affected birds have toes curled inward both when walking and resting. The skin is rough and dry. Similar clinical signs thought to be associated with riboflavin deficiency have been reported in young waterfowl, an eagle and ratites (see Color 48).³⁴ Turkey poults with riboflavin deficiency show poor growth with dermatitis around

the face and feet. Cockatiels fed riboflavin-deficient diets failed to incorporate pigment into their primary feathers.⁴⁶

Older birds are more resistant to riboflavin deficiency than juveniles. Breeding hens fed riboflavin-deficient diets may show fatty infiltration of the liver as well as decreased hatchability of their eggs and increased embryo mortality. Heterophil counts may increase and lymphocyte counts decrease. Primary wing feathers may be excessively long.^{36,51} Early treatment with riboflavin will resolve clinical signs; however, in chronic cases permanent nerve damage may occur.

- **Niacin (Nicotinic Acid):** Clinical signs of niacin deficiency are fairly nonspecific and include poor feathering, nervousness, diarrhea and stomatitis. Young chickens, turkeys and ducks with niacin deficiency may show enlargement of the hock and bowed legs similar to those seen with perosis, but the gastrocnemius tendon does not slip from the condyles. Chickens showing signs of hysteria have responded clinically to niacin supplementation in the drinking water.³ Niacin deficiency has not been described in Psittaciformes.
- **Pyridoxine (Vitamin B₆):** Chicks with pyridoxine deficiency may show depressed appetites, poor growth, perosis, jerky movements and spasmodic convulsions. As with riboflavin deficiency, heterophil counts may increase while lymphocyte counts decrease.

Because pyridoxine is involved in amino acid metabolism, signs of deficiency rarely occur unless dietary protein levels are high. In adult chickens, pyridoxine deficiency causes reduced egg production and poor hatchability. Pyridoxine deficiency was suspected in juvenile rheas that developed "goose-stepping" gaits.¹⁶

- **Pantothenic Acid:** Symptoms of pantothenic acid deficiency in chicks are similar to those of biotin deficiency and include dermatitis on the face and feet, perosis, poor growth, poor feathering and ataxia (see Color 48). Severe edema and subcutaneous hemorrhages are signs of pantothenic acid deficiency in developing chicken embryos.¹ Similar signs have also been seen in developing ostrich embryos. High incubator humidity may contribute to this problem. Cockatiels reared on pantothenic acid-deficient diets failed to grow contour feathers on their chests and backs, and many died at three weeks of age. Affected birds had the appearance of feather-picked chicks.⁴⁶

- **Biotin:** Natural sources of the vitamin are the same as those for pantothenic acid, and signs of biotin deficiency may parallel those of pantothenic acid: dermatitis on the face and feet, perosis, poor growth, poor feathering and ataxia. Biotin deficiency may also be associated with swelling and ulceration of the foot pads, and biotin-deficient embryos may show syndactylia and chondrodysplastic changes in the skeleton. Fatty liver and kidney syndrome in chickens has been associated with marginal biotin deficiency. Although egg yolk is a rich source of biotin, uncooked egg white contains a biotin antagonist called avidin, and biotin supplementation of a diet containing raw egg white may not correct the deficiency unless the biotin-binding capacity of the egg white has been exceeded. Mycotoxins may also interfere with biotin uptake.
- **Folic Acid:** In poultry, folic acid deficiency has been associated with embryonic mortality, deformation of the upper mandible, poor growth, macrocytic anemia, bending of the tibiotarsi and perosis. Folic acid, lysine and iron appear to be needed for the production of feather pigments in colored breeds of poultry. Folic acid is synthesized by bacteria in the digestive tract, so antibiotic therapy, particularly with sulfonamides, could induce a deficiency.
- **Choline:** A deficiency of choline caused poor growth and perosis in juvenile turkeys and chickens. In older birds, fatty liver infiltration may occur. Cockatiels reared on choline-deficient diets showed unpigmented wing and tail feathers but no signs of perosis.⁴⁶
- **Vitamin C:** Bulbuls and fruit-eating birds may require exogenous vitamin C (ascorbic acid) but in chickens, and probably most species of seed-eating birds, vitamin C is synthesized in the liver. Birds with vitamin E and selenium deficiencies, heat stress, trace element toxicity or parasitic infections would be expected to have an increased requirement and decreased production of vitamin C.³ In these situations, parenteral supplementation of vitamin C would be indicated. Natural sources of vitamin C include fresh fruits and vegetables (eg, citrus fruits, broccoli, green peppers). High dietary intake of ascorbic acid improves albumen quality and egg shell thickness in chickens. Signs of vitamin C deficiency have not been documented in companion birds.

Minerals

Calcium and Phosphorus

Calcium in the diet is used for bone formation, egg shell production, blood clotting, nerve impulse transmission, glandular secretion and muscle contraction. Phosphorus is important in many body functions including bone formation, the maintenance of acid-base balance, fat and carbohydrate metabolism and calcium transport in egg formation. Separate vitamin D-dependent mechanisms are believed to be responsible for calcium and phosphorus absorption from the intestine.

If calcium utilization exceeds absorption from the intestine over a prolonged period of time, parathyroid hormone excretion will increase and the parathyroid glands will enlarge (see Color 14). This condition, called secondary nutritional hyperparathyroidism (SNH), allows normal blood calcium levels to be maintained. High levels of phosphorus or low levels of vitamin D in the diet may exacerbate SNH. Symptoms of the syndrome may include weakness, polydipsia, anorexia and regurgitation. In breeding hens, SNH may result in decreased egg production, production of soft-shelled eggs, egg binding and fragile bones (see Chapter 23).

Hypocalcemic seizures associated with severe parathyroid enlargement and degeneration occur as a syndrome in African Grey Parrots.⁴⁵ Affected birds are generally between the ages of two to five years. Abnormal clinical pathology findings include leukocytosis and hypocalcemia. Calcium levels may be below 6.0 mg/dl and sometimes as low as 2.4 mg/dl. At necropsy, there is no apparent calcium mobilization from bones as would be expected when blood calcium levels decrease in normal birds. Affected birds have difficulty in mobilizing calcium from body stores, and should be supplemented constantly with dietary calcium. There has been some undocumented discussion that the syndrome is limited to imported birds, and that some naturally occurring environmental factor may initiate the syndrome. Additionally, the problem may be regional in nature because it appears to be more prevalent on the West Coast, as compared to the East Coast, of the United States.

- **Diets for Birds with Hypocalcemia:** Calcium syrup may be used in the drinking water, sprinkled on seeds or soft foods or administered directly. Foods containing high levels of calcium such as bones, cheese or yogurt may be provided. Calcium powder may be sprinkled on soft food. High-fat seeds (eg, sunflower,

safflower) may interfere with calcium uptake from the intestine. Levels of vitamin D₃ in the diet should be evaluated and supplemented if needed.

High-calcium diets are generally required only until normal body reserves are restored. The addition of psyllium to the diet may increase the absorption of calcium.³⁰ Long-term consumption of high levels of calcium may interfere with manganese or zinc absorption and may result in renal calcium deposition, reduced numbers of glomeruli per kidney and subsequent renal failure.⁴¹ Because of these problems, care should be taken to provide correct supplementation levels. Laying hens and rapidly growing juveniles will require higher levels of calcium than non-breeding adults.

Hypocalcemic seizures are rare in species other than African Grey Parrots. Occasionally, companion birds on an all-seed diet will be presented with seizing caused by hypocalcemia. These birds usually respond dramatically (within minutes) to intramuscular calcium and multivitamin therapy.

Because calcium metabolism is closely linked with vitamin D metabolism, many changes caused by calcium deficiency in juveniles are identical to those caused by hypovitaminosis D. Appropriate amounts of calcium, phosphorus and vitamin D are necessary for optimal bone and egg shell formation. The normal calcium to phosphorus ratio for chickens is 2:1. It is likely that a similar ratio would be appropriate for most species of birds although specific research data is lacking. Most available commercial seeds are extremely calcium deficient: corn=1:37, millet=1:6, milo=1:14, oats=1:8 and sunflower seeds=1:7. High-fat content in oil seeds may also interfere with calcium absorption from the intestine and exacerbate the problem.⁴⁶

Muscle meat is low in calcium and high in phosphorus with a ratio of 1:20. Carnivorous birds fed an all-meat diet, day-old chicks or pinky mice may show signs of calcium deficiency and SNH. Feeding whole adult mice, older chicks, quail or rats to carnivorous birds should provide better calcium balance. It is important to provide variety in the type of food fed.³⁸

Long bone deformities in juvenile birds, particularly ratites, may be associated with high protein, low calcium diets; however, reducing dietary protein and supplementing calcium may not always correct the problem. In these situations, the overall suitability of the diet, including the calcium to phosphorus ratio, the level of magnesium and electrolytes and the en-

ergy level in the diet should be evaluated. The birds should be encouraged to exercise more and the rate of weight gain should be reduced (see Chapter 48).

Excess phosphorus consumption can exacerbate SNH. Decreases in egg production, poor egg shell quality and rickets could occur with phosphorus deficiency, but this is unlikely because the mineral is very widely distributed in common food items.

Magnesium

Magnesium is necessary for bone formation, for carbohydrate metabolism and for activation of many enzymes. Its metabolism is closely associated with that of calcium and phosphorus. Deficiencies in young chicks may result in poor growth, lethargy, convulsions and death. Excessive amounts may cause diarrhea, irritability, decreased egg production and thin-shelled eggs.

Iron

Iron is needed for the production of hemoglobin and many enzymes. Iron deficiency may result in hypochromic, microcytic anemia. Normal levels of non-heme iron in the plasma are necessary for feather pigmentation.

- **Diets for Birds with Anemia:** Birds with anemia should receive a diet that is high in energy and protein, and be supplemented with B complex vitamins (including B₁₂, pyridoxine, niacin and folic acid), iron, cobalt and copper.
- **Diets for Birds with Hepatopathies:** Iron storage disorders have been reported in a variety of non-psittacine species, particularly Indian Hill Mynahs, birds of paradise, hornbills and toucans.³⁴ In some cases, the disease has been correlated with diets high in iron, and problems with the condition decreased when dietary iron levels were lowered to less than 40 ppm (see Chapters 20, 47).

Liver disease may decrease the absorption and storage of fat-soluble vitamins A and D and inhibit the synthesis of vitamin C necessitating supplementation.²¹ Other objectives in designing diets for birds with liver disease include reducing the work load on the liver (fat conversion, gluconeogenesis, deamination and nitrogen conversion), preventing sodium retention and hypokalemia, restoring liver glycogen and minimizing the possibility of hepatic encephalopathy. The diet should contain a readily available energy source such as dextrose or other easily digested carbohydrate. Canary seeds, millet, panicum, corn or hulled oats are relatively high in carbohy-

drate and low in protein and fat. These should be used in preference to sunflower seed, rape or niger, all of which are much higher in fat and protein and lower in carbohydrate. Birds with hepatopathy should be offered a variety of fresh fruit and vegetables that are generally high in easily digestible carbohydrates. These fruits and vegetables should be organically grown to prevent exposing the compromised liver to pesticides. The diet should contain a low level of protein of high biologic value such as chopped hard-cooked egg, cottage cheese or cooked chicken. For carnivorous birds, purine-containing foods (offal) should be avoided. The bird should receive a sufficient volume of food to meet caloric needs (see Chapter 20).

Copper

Copper is necessary for heme synthesis and is an important component of several enzymes including lysyl oxidase, an enzyme involved in the formation of cross-linking in elastin and tyrosine o-diphenol oxidase, which catalyzes the early stages of melanin synthesis. Copper deficiency has been associated with aortic rupture in poultry as well as being associated with increased bone fragility and impaired feather pigmentation. In laying hens, copper deficiency may cause decreased egg production and shell abnormalities including shell-less, misshapen, wrinkled eggs and large eggs with thin shells.

Selenium

In addition to having a vitamin E-sparing effect in the prevention of ventricular myopathy, white muscle disease and exudative diathesis, selenium is also linked with exocrine pancreatic function and the production of thyroid hormones. In young chickens, selenium deficiency causes poor growth and poor feathering, impaired fat digestion and pancreatic atrophy. Similar problems are occasionally seen in companion birds, but a link with selenium deficiency has not been established. Excess dietary selenium (above four ppm) in ducks can impair hatching success and may be teratogenic³⁷ (see Chapter 48).

Manganese

Manganese is required for normal bone and egg shell formation and for growth, reproduction and the prevention of perosis. Poultry embryos and young chicks with a manganese deficiency develop chondrodystrophia: short, thickened limbs, parrot beak, protruding abdomen and retarded growth. Ataxia may also be noted. Seed diets may be deficient in manganese.

Zinc

Zinc is needed for the formation of insulin and many enzymes in the body. In poultry, zinc deficiency may cause short, thickened long bones, enlargement of the hock, dermatitis and impaired T-cell function. Ducks may develop hyperkeratosis of the feet and oral cavity. Zinc is also necessary for proper function of vitamin A. Patients with hypovitaminosis A must receive adequate levels of dietary zinc for therapy to be successful. Excess levels of zinc may cause pancreatic cell necrosis secondary to interference with cellular protein synthesis.

Iodine

Iodine is needed for the formation of thyroxine and related compounds in the thyroid gland. Iodine deficiency may result in goiter (enlargement of the thyroid glands). The thyroid glands in birds are located in the thoracic inlet and usually cannot be palpated (see Anatomy Overlays).

Clinical signs of goiter are the result of pressure on organs adjacent to the gland. A loud, wheezing respiration with neck extended may occur if there is pressure against the trachea. Crop dilation and vomiting may occur if the goiter obstructs the outlet to the crop. Iodine-deficient budgerigars are particularly prone to goiter (see Color 19). Goiter has occasionally been reported in other species of birds⁵³ (see Chapter 23).

Birds with goiter must be handled with care. Excessive stress may cause regurgitation and subsequent aspiration of vomitus. Conservative therapy should include the administration of a drop of iodine orally each day. Injectable iodine and dexamethasone may be necessary in more advanced cases. Once stabilized, the bird should be changed to a formulated diet.

Excess dietary iodine has also been reported to induce goiter (eg, birds consuming iodine-based cleaning agents). High levels of iodine may also antagonize chloride, depress growth rates and induce CNS signs. Goitrogens in plants of the genus *Brassica* (eg, broccoli, cabbage or mustard greens) have been implicated as a cause of goiter in mammals; however, companion birds frequently consume these plants with no side effects.

The iodine content of seed depends on the iodine content of the soil on which it is grown. Seed grown in "goiter belt" regions of the world are likely to be deficient. The problem can be eliminated if iodine is

added to the seed. Alternatively and preferably, the birds should be changed to a formulated diet.

Budgerigars with thyroid tumors may have clinical signs identical to those seen with goiter. While goiter will generally respond quickly to iodine supplementation, thyroid tumors will not. It has been suggested that iodine-deficient diets may be associated with signs of hypothyroidism (eg, lethargy, obesity or dermatitis); however, these signs are rarely seen in companion birds with goiter (see Chapter 23).³²

Potassium

Potassium is the principal cation in intracellular fluid and is required for glucose and protein metabolism. The mineral is widely distributed in food of both plant and animal origin. Symptoms associated with deficiency are unlikely to occur, but in chickens these may include decreased egg production, egg shell thinning, muscle and cardiac weakness, tetanic convulsion and death.

Sodium and Chloride

In poultry, salt deficiency causes weight loss, decreased egg production, small eggs and increased cannibalism. In psittacine birds, it has been suggested that salt deficiency may play a role in some cases of self-mutilation. Sodium deficiency alone may cause a decrease in cardiac output, hemoconcentration, reduced utilization of protein and carbohydrates, soft bones, corneal keratinization, gonadal inactivity and adrenal hypertrophy.

Chloride deficiency in chickens produces dehydration and characteristic CNS signs, in which chicks fall forward with their feet stretched out behind them for several minutes. Tibial dyschondroplasia in meat poultry has been associated with excess dietary chloride. This problem is seen occasionally in young ratites, although the cause has not been clearly defined.

Demineralized bone formation was seen in a variety of juvenile Australian parrots fed a homemade mineral block containing apparently adequate calcium and phosphorus levels, but an excess level of salt. The problem stopped when the mineral block was removed.

Excessive amounts of salt may be acutely toxic. Affected birds show intense polydipsia, muscle weakness and convulsions. Ducks are more sensitive to salt intoxication than are poultry. Sea birds have a nasal salt gland that is controlled by the ATPase pump in the gastrointestinal tract, and is used to excrete excessive exogenous salt. Oil contamination may suppress the ATPase pump and cause clinical signs of salt toxicity. The salt gland of sea birds provided fresh water becomes nonfunctional. Prior to release into a marine environment, these birds should receive gradually increasing levels of salt to ensure that their glands are functional.⁵⁷

Water

Water consumption in birds varies dramatically among species and among individuals of the same species. Budgerigars and Zebra Finches (species that evolved in desert regions) have been reported to survive several months without drinking, apparently relying on water derived from metabolic sources.⁷ On the other hand, healthy companion birds may consume significant amounts of water daily and become distressed if water is withheld.

Some birds that have not evolved for desert living (eg, canaries), may die if they do not drink water for 48 hours. The addition of any compound to the drinking water can cause these birds to stop consuming water, resulting in a rapid dehydration and death.

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