

The condition of the skin and feathers of a bird provides a clinical window to the nutritional plane and environmental conditions to which the patient is exposed. Additionally, systemic diseases (hepatic, renal, pancreatic, gastrointestinal, hematopoietic) can alter the condition of the integument. These changes are frequently detected by observant owners and should be carefully evaluated by the veterinarian. In addition to responding to systemic abnormalities, the feathers and skin are subject to a group of organ-specific diseases.

The unique structure and adaptations of the avian integument have long attracted interest.^{26,32,67} The avian integument consists of the skin, scales, feathers, four sets of glands, beak, cere, nails and foot pads. Some species (particularly Galliformes) have highly adapted integumentary appendages that are used for defense or mating rituals. These include wattle, ricti, ear lobes, comb (chickens); dewlap, snood (turkeys); casque (cassowaries); shields (coots and gallinules); knob (goose) and various modifications of the head plumage into crests and bristles. Through selective breeding the comb of the red junglefowl has been modified into dozens of unique shapes, sizes and colors. These unfeathered appendages are particularly susceptible to traumatic injuries and infectious agents. Not all skin appendages are found in any one bird. The only common elements are skin, beak, nails and feathers, which vary in pigmentation, shape, texture, function, location and number, depending on evolutionary adaptations.

CHAPTER

24

DERMATOLOGY

■
John E. Cooper
Greg J. Harrison

Anatomy and Physiology of the Avian Integument

The epidermis of birds consists of three layers including the basal (germinative) layer, intermediate layer and outer cornified layer. The germinative layer is thin (two to four cells thick) in the feathered areas of the body and may be much thicker and interdigitate with the dermis in unfeathered areas of the legs and feet.²⁶ Striated muscles located in the epidermis move the skin.

The dermis is divided into superficial and deep layers, with the former containing loosely arranged layers of collagen in interwoven bundles and the latter containing fat, feather follicles, smooth muscles that control movement of the feathers and large blood vessels and nerves that supply the dermis and epidermis.³² A complex mechanoreceptor system (Herbst's corpuscles) occurs in various parts of the avian body including the integument, bones, tendons, muscles, joints and vessels.

The skin overlying the head, extremities and sternum is firmly attached to underlying skeletal structures. Over the remainder of the body, the skin is loosely attached to the underlying muscles. The areas with the most subcutaneous tissues include the dorsal cervical, midline, axillary and groin regions. Footpads are present in many birds, primarily terrestrial species (Figure 24.1). The feet of some birds indigenous to areas with inclement weather are covered with feathers or contain projections (spikes) to facilitate movement in ice and snow.

During the breeding season, many avian species will develop a thickening and increased vascularization of the skin on the ventral abdomen called a brood patch. Depending on the species, one or both genders may develop this brood patch, which should not be mistaken for a featherless, hyperemic skin lesion.

Birds lack sweat glands and most of the skin over the body is thin, dry and inelastic. The feet and, to varying degrees among species, the legs are covered with thick scales. The skin is glandless except for the uropygial (preen) gland, glands of the ear canal and pericloacal glands. The uropygial gland is involved in maintaining feather condition in those species that have this structure. The presence or absence of these

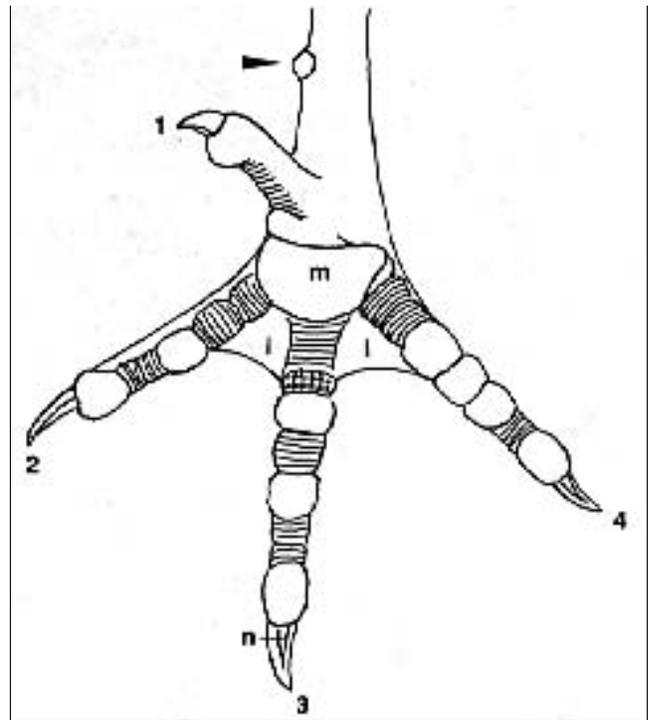


FIG 24.1 Well developed footpads are common in terrestrial birds. Digits 1, 2, 3 and 4 from the right foot of a gallinaceous bird. The interpad spaces are shaded and the digital pads are unshaded. Metatarsal pads (m), interdigital web (i), nail (n) and metatarsal spur (arrow) (modified from Lucas and Stettenheim³²).

glands varies widely among species. Pericloacal glands secrete mucus.²⁸

The feathers serve a protective function and the unfeathered areas of the integument (cere, beak, face, legs and feet) are common sites for primary skin disease (poxvirus, *Knemidokoptes* spp.). The skin is capable of dissipating some heat through evaporative cooling but the lack of sweat glands makes birds particularly sensitive to hyperthermia. The functional capacity of the evaporative cooling mechanism may be adversely affected by an essential fatty acid deficiency.³⁵

Some birds maintain feather quality through dusting, sunning or bathing. "Anting," or the intentional allowing of ants to cover the body, may serve a grooming role in some species. Other species, most notably cockatoos, have specialized feathers (powder down) that produce a fine keratin debris that is involved in maintaining feather condition. Presence of this normal keratin debris should not be confused with a pathologic condition. Additionally, the exsheathed portion of the keratin shaft from a developing feather should not be confused with dandruff. Retention of the sheath is common in birds with systemic disease

and these birds may be pruritic or appear hypersensitive when stroked.

Cere

The wax-like cere is composed of keratinized skin at the base of the upper beak. Many Anseriformes lack a cere and their nostrils are located in the tip of the soft beak (see Figure 46.7). The cere is affected by a number of conditions, and its appearance can change with the health of the bird. In raptors, the cere may change from bright yellow to pale yellow based on the quantity of carotenoids in the diet.

Brown hypertrophy of the cere may occur in male budgerigars, presumably due to changes in the ratio of sex hormones, and is frequently associated with testicular tumors (Color 24.18). The discolored hyperkeratotic material can be moistened and gently peeled away or removed by scraping or rasping.³ Hyperkeratosis and flaking of the skin around the cere may be pronounced in malnourished birds. Some hypertrophy is normal in reproductively active hens.

Beak

The beak (rostrum) consists of the bones of the upper (maxilla) and lower (mandible) jaws and their horny covering (rhamphotheca). The beak (or bill) functionally replaces the lips and teeth of mammals and varies in function, shape, size and length among species. The bone underlying the rhamphotheca is covered by periosteum. The periosteum is covered by the dermis and epidermis. The dermis of the beak does not appear to be divided into layers as it is in the skin, but rather is a single layer of dense connective tissue.³²

The consistency of the rhamphotheca varies among species. It is horny and firm in Psittaciformes, but soft and pliable in Anseriformes. The rhamphotheca can be viewed as the stratum corneum of the beak, and the dermis is well vascularized and connected to the periosteum of the underlying bone. Trauma or necrosis of the dermis will frequently result in a lesion that induces a beak deformity (inability of the damaged area to regrow) (Figure 24.2).

The beak, nails and spurs grow continuously and are worn down by digging, eating or chewing hard objects (Figure 24.3). The beak is used as a tool, weapon and as a tactile exploratory organ for food discrimination, plumage care, nest-building and feeding of the young.



FIG 24.2 Depending on the severity and location of a beak injury, defects in the rhamphotheca can be permanent or they can heal. In this case, a traumatic beak wound in a Great-billed Parrot has been repaired with cyanoacrylic resin (arrows) (courtesy of Louise Bauck).

The beak is modified to rip (raptors), tear or crush (psittacine birds), sift (flamingos) or probe for food (avocets). The beak of some gallinaceous birds is serrated and resembles teeth in both form and function. The buccal surface of the upper beak of some Psittaciformes has a number of rasp-like ridges (Figure 24.4). These are believed to function in holding nuts, filing down fruits and maintaining a sharp edge on the lower beak.²⁸

The skin and beak of birds are sensitive to heat, cold and various degrees of pressure. Cutting the beak of a goose will elicit an increase in blood pressure, heart rate and respiratory rate and initiate a tear flow (pain response).²⁸ Herbst's corpuscles found in the tip of the beak may serve a tactile function that is independent of the tongue and eyes in the exploration for and sorting of food.

Birds that use the beak to search, catch or select food have a well developed "bill tip organ" that can be recognized as papillae that originate in the dermis and end in crater-like structures at the distal tip of the beak.²⁸ The location and degree of development of the bill tip organ vary among species. In parrots, the organ is best developed in the lower beak (Figure 24.5). The upper beak in Psittaciformes can be viewed as a probe that is used to move items over the bill tip organ in the lower beak. Granivores that obtain food by pecking (Columbiformes and Passeriformes) do not appear to have this organ. The bill tip organ should be presumed to be extremely sensitive,

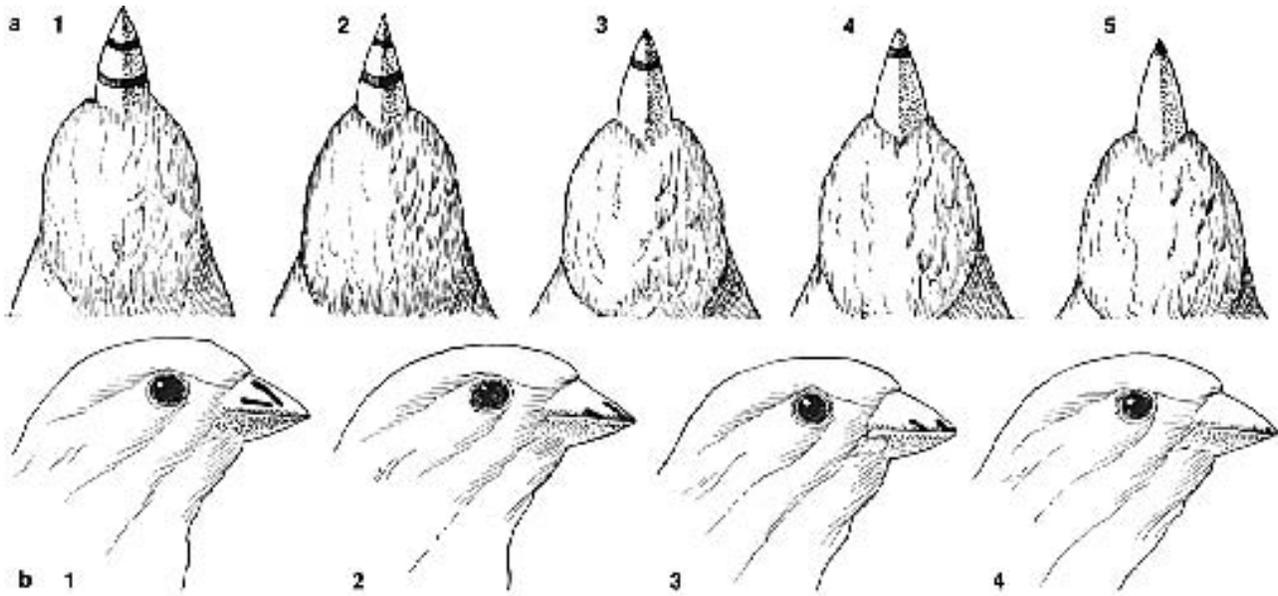


FIG 24.3 Based on beak migration patterns, the clinical progression of rhamphothecal hyperkeratosis secondary to malnutrition must involve dysfunction of multiple growth plates. **a)** Lines were placed on the dorsal rhinotheca to demonstrate the rostral migration of the upper layer of the rhinotheca during growth. Note that the dorsal plates migrate straight to the tip. 1) Marks were placed on the beak and evaluated at: 2) two weeks, 3) one month, 4) two months and 5) ten weeks. **b)** Marks were placed on the lateral rhinotheca to demonstrate the difference in migration of the lateral and dorsal plates of the beak. Note that the lateral plates migrate in a curvilinear fashion toward the cutting edge of the rhinotheca. 1) Marks were placed and evaluated at 2) one month, 3) six weeks and 4) two months (modified from Lüdicke³³).

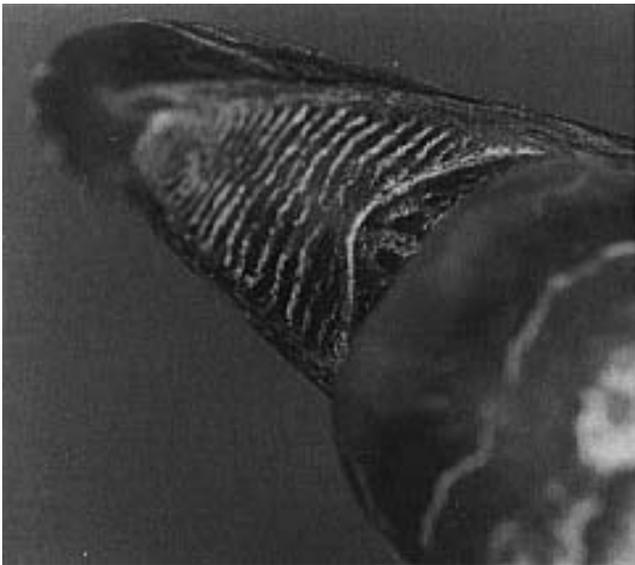


FIG 24.4 Ridges on the occlusal surface of the upper beak of some psittacine birds are believed to function in holding food and sharpening the edge of the lower beak.

which should be considered when manipulating the tip of the beak.

The horny tissue of the beak is generated from two locations. The hard outer horn is produced by the epidermis and grows toward the rim (cutting surface). A softer keratin that surrounds the papillae of the bill tip organ originates from the keratinized epidermis (Figure 24.3).²⁸ The beak should remain in proper condition without trimming in birds that are maintained on a formulated diet supplemented with fresh fruits and vegetables, exposed to adequate periods of sunlight, allowed to bathe regularly and provided with hard woods to chew.

Any companion bird that requires repeated beak trimming should receive a thorough diagnostic evaluation to detect the underlying management, nutritional or systemic abnormality that is causing excessive beak growth or improper beak wear. Overgrowth of the lower beak may lead to occlusion of the openings to the bill tip organ and a loss of function. To improve the sensory capacity of the bill tip organ, the lower beak should be included in routine grooming activities if the beak is overgrown.

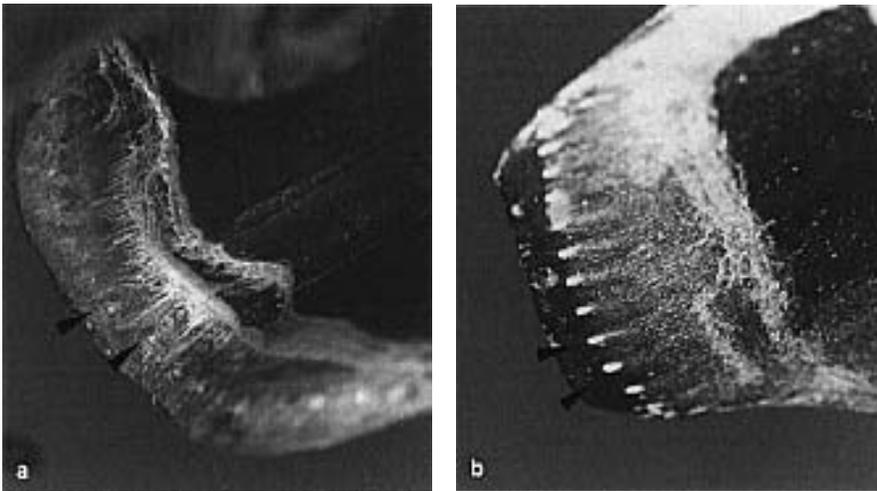


FIG 24.5 a) The bill tip organs are barely visible on the occlusal surface of an overgrowing lower beak in a Hyacinth Macaw. b) After trimming, the bill tip organs are distinct (arrows).

The recently hatched neonates of most bird species have a small pointed eminence on the dorsal surface of the upper beak (egg tooth) that is used during the hatching process to penetrate the shell (see Color 30). The egg tooth regresses in Galliformes, Psittaciformes and Passeriformes during the first week of life. Some birds, megapodes for example, lack an egg tooth; these neonates are believed to use the feet to kick their way out of the egg.

Abnormalities of the beak are caused by:

- Malformation (often due to nutritional disorders)
- Primary viral infection
- Overgrowth (associated with a high-protein diet in some frugivorous birds, believed to be secondary to malnutrition or organopathy [liver] in many species)
- Fracture or puncture (usually traumatic).

Color changes in the beak of some species (toucans, lorikeets) may be associated with malnutrition or systemic disease. Bacterial or fungal infections of the beak are usually secondary to injuries that result in damage to the horny layer of the beak³ (see Chapter 42). Bragnathism and scissors beak occur commonly in some neonatal psittacines (see Chapters 30 and 42). A discussion of the diseases of the beak is provided in Chapter 19.

■ Skin

Developing dermal cells (keratinocytes) undergo a metamorphosis from a cuboidal to squamous nature, and in the process lose cellular organelles, produce

lipids and fibrous proteins (keratin) and finally dehydrate and lyse.⁶² Although avian skin is noted for its paucity of glands, it has been suggested that the lipid production by the keratinocytes (a function unique to birds) makes the entire skin an oil-producing holocrine gland.^{37,44} The lipids produced by the keratinocytes are combined with oils secreted by the uropygial gland to form a thin film that is deposited over the feathers.⁶² In poultry, lipid production has been found to be higher in thin skin that must be kept supple than in thicker skin that is relatively rigid.

In combination, lipids from the keratinocytes and uropygial gland secretions are believed to waterproof the feathers, inhibit the growth of bacteria and fungi and maintain proper moisture content and pliability of the feathers.³⁷ It can be theorized that the severe and generalized feather pathology associated with systemic diseases (eg, organopathy, malnutrition) is a result of improperly functioning keratinocytes.

■ Patagia

Skin may be reflected into flat, membrane-like structures (patagia) in areas where the wings, legs, neck and tail join the body.³² The wing has four patagia: the propatagium (wing web), where the neck and wing join the thorax; the postpatagium, formed at the caudal angle of the wrist; the metapatagium, at the caudal junction of the wing and the thorax; and the alular patagium, at the interspace between the alula and the metacarpus (hand) (Figure 24.6). A cervical patagium is located anterior to the shoulder in the angle between the neck and the scapula.

A similar structure is formed by the skin connecting the knee to the prolateral region of the paralumbar area (knee web). This is a transitory structure that is formed when the leg is in certain positions. It is called a web to differentiate it from a patagium, which is always present regardless of the position of the limb.³² A groin web may be formed by the skin extending from the sternal region to the medial surface of the thigh (Figure 24.6).

Patagia and webs represent sites of major skin flexion and can be used clinically for subcutaneous injec-

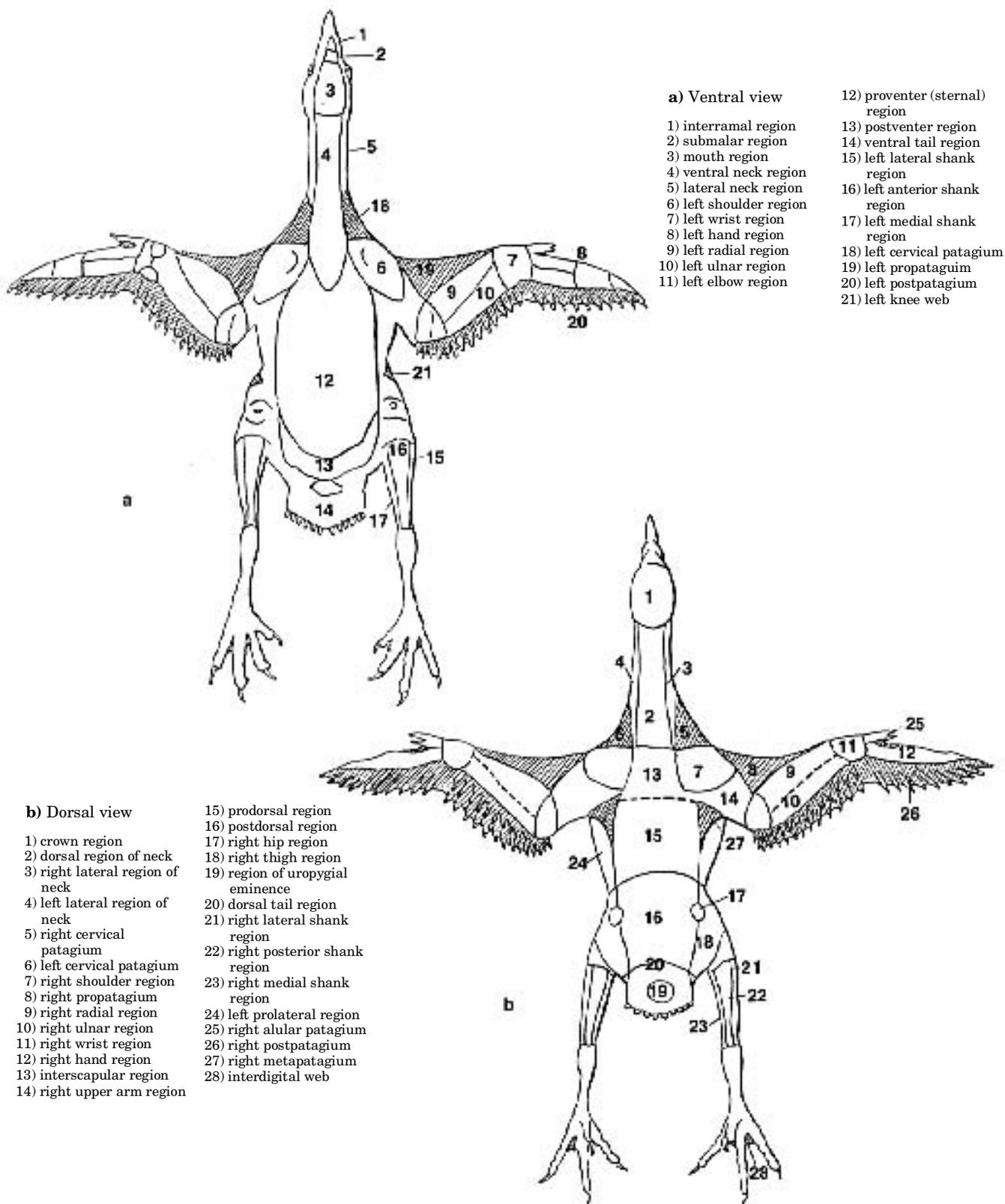
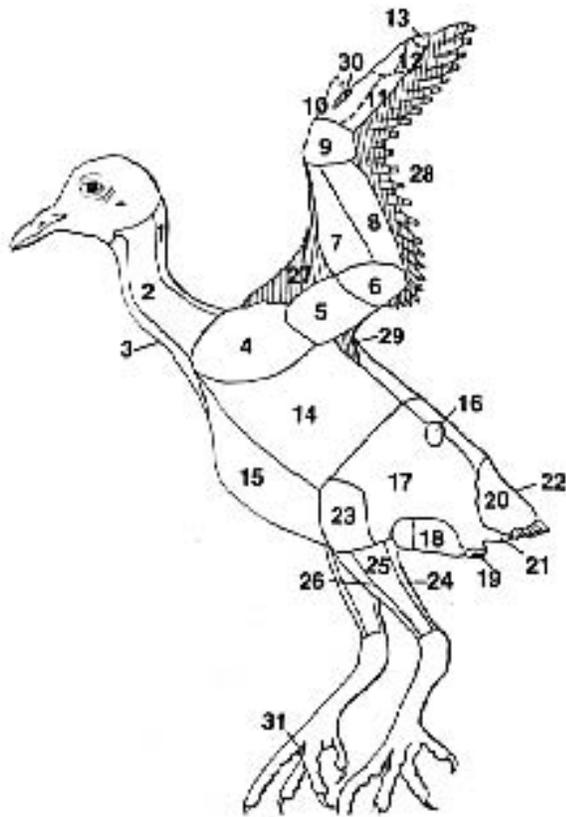


FIG 24.6 a) Ventral, b) dorsal and c) lateral (next page) drawings of the pigeon showing the location of patagia, webs and regions of the body that should be used in describing integumentary lesions. Some of the patagia are common sites of chronic ulcerative dermatitis lesions (modified from Lucas and Stettenheim³²).



c) Lateral view

- | | |
|--|---------------------------------|
| 1) dorsal region of neck | 15) proventer (sternal) region |
| 2) lateral region of neck | 16) hip region |
| 3) ventral region of neck | 17) thigh region |
| 4) left shoulder region | 18) postventer region |
| 5) left upper arm region | 19) vent |
| 6) left elbow region | 20) dorsal tail region |
| 7) left radial region | 21) ventral tail region |
| 8) left ulnar region | 22) uropygial eminence |
| 9) left wrist region | 23) left knee region |
| 10) left region of metacarpus, digit III | 24) left posterior shank region |
| 11) left region of metacarpus, digit IV | 25) left lateral shank region |
| 12) left region of P1, digit III | 26) left anterior shank region |
| 13) left region of P2, digit III | 27) left propatagium |
| 14) left prolateral region | 28) left postpatagium |
| | 29) left metapatagium |
| | 30) alular patagium |
| | 31) interdigital web |

tions or tattooing. These anatomic areas as well as the ventral tail region appear to be frequent sites for the occurrence of ulcerative dermatitis.

Uropygial Gland

The uropygial gland is a bilobed gland located at the base of the tail dorsal to the pygostyle. The gland is absent in many Columbiformes, Amazon parrots and other Psittaciformes. This holocrine gland opens to the outside through a caudally directed nipple that is frequently surrounded by a tuft of feathers (Figure 24.7). Its secretions are spread by preening (grooming) and are considered to serve a waterproofing



FIG 24.7 The uropygial gland is located on the dorsal surface of the bird at the base of the tail (arrow). The opening to the gland is frequently surrounded by a tuft of feathers (open arrow).

function. Additional secretions from the skin and the uropygial gland are believed to suppress the growth of microorganisms. Uropygial gland secretions contain vitamin D precursors that are spread through the feathers, converted to an active form following exposure to ultraviolet light and ingested with subsequent preening activity.

Abnormalities associated with the uropygial gland include neoplasm (primarily squamous cell or adenocarcinoma), abscessation and impactions. A presumptive diagnosis of uropygial gland abnormalities can be based on microbiological culture and cytologic examination of exudate, an aspirate or a biopsy.

Impacted glands are frequently discussed in the literature but appear to be uncommon clinically. The gland is normally swollen and appears as though it may need expressing. In some birds, hyperkeratotic plugs may form in the gland. These cases will generally respond to removal of the plug and improving the bird's diet. An African Grey Parrot with widespread feather loss and a cystic uropygial gland failed to

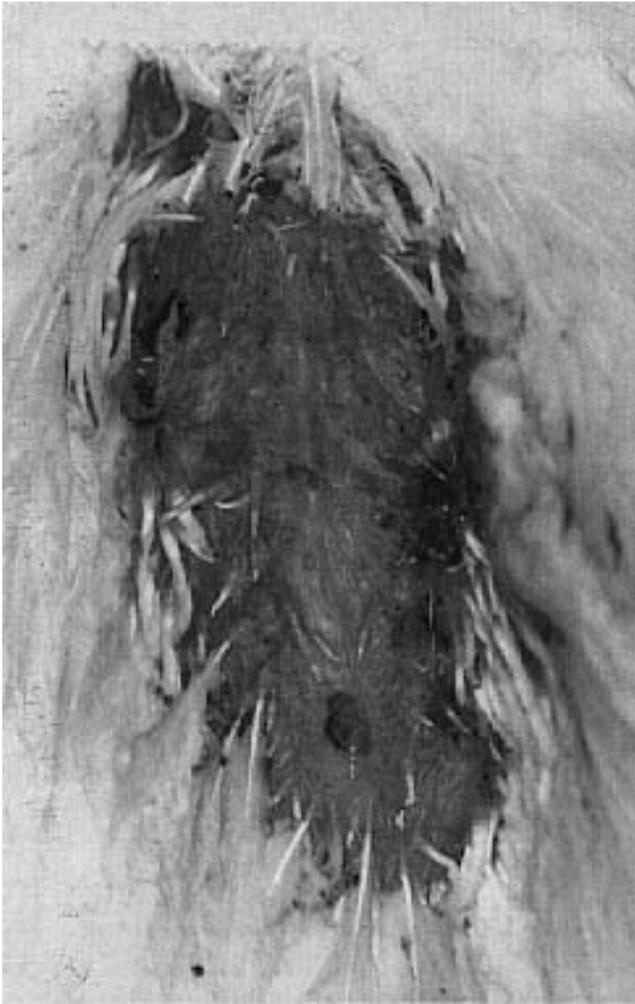


FIG 24.8 The body is divided into areas that contain feather tracts (pterylae) and areas that do not contain feather tracts (apteria). The division between feather tracts is evident on the back of this lutino cockatiel with pruritic dermatitis (courtesy of Louise Bauck).

respond to extensive treatment that included laser therapy, but recovered three months later after a deficient diet was corrected.³² Uropygial gland rupture has been described in Gentoo Penguins and in free-living seabirds in Europe (Cooper JE, unpublished).³⁴

Surgical extirpation of the gland may be necessary if neoplasia occurs (see Chapter 41). In ducks, removing the gland will cause the birds to lose the ability to waterproof their feathers. In other birds, removal of the gland seems to have few clinically detectable effects.³²

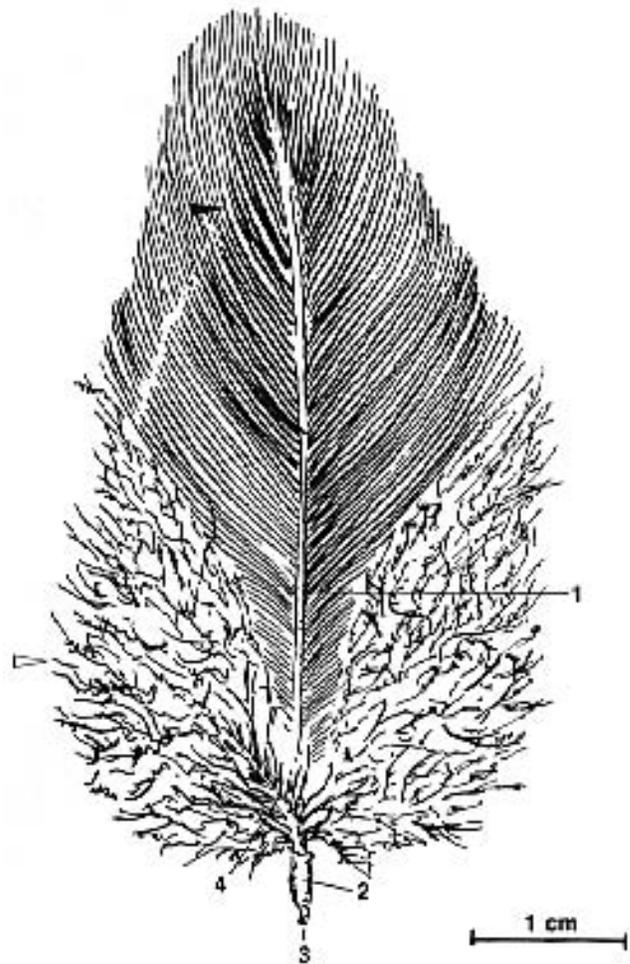


FIG 24.9 Anatomy of a contour feather: 1) rachis 2) calamus 3) posterior umbilicus and 4) afterfeather. The pennaceous portion of feather (arrow) and plumaceous portion of feather (open arrow) are also evident. The rachis and calamus form the shaft. The pennaceous and plumaceous portions of the feather form the vane (modified from Lucas and Stettenheim³²).

Feathers

The three principal functions of the feathers are flight, insulation and waterproofing. Feathers may also function in courtship, defense (color mimicking) and aggressive territorial behaviors. In most birds, the body is divided into areas that contain feather tracts (pterylae) and areas that do not contain feather tracts (apteria) (Figure 24.8). The location of feather tracts varies among avian families. By originating from tracts rather than being scattered randomly over the body, feathers can smoothly overlap each other and conform to the natural contours of the

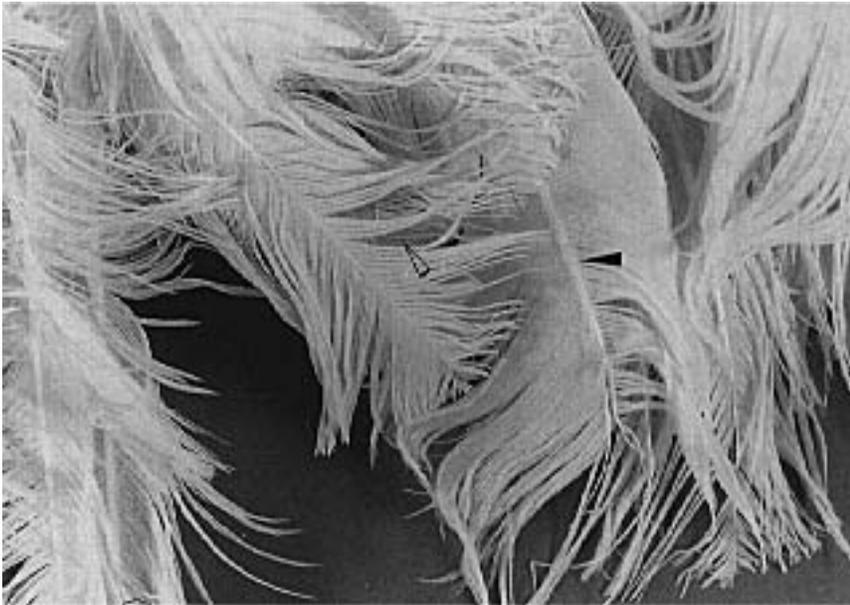


FIG 24.10 Damaged feathers in a malnourished cockatoo. These lesions are frequently blamed on an enclosure of insufficient size. The central shaft of the feather is called the rachis (arrow). The barbs (open arrow) branch from the rachis. The barbs are connected to each other by the barbules. Where the barbs are connected, the barbules are intact. Those that are not connected have damaged barbules.

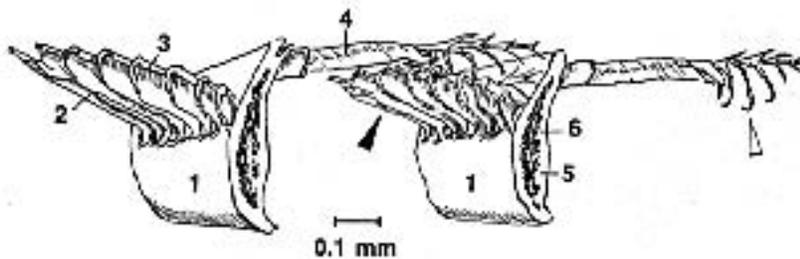


FIG 24.11 The 1) cut end of two barbs showing the interlocking nature of the barbules (arrow). The 2) posterior barbules contain 3) ridges that connect with the hooks (open arrow) found on the 4) anterior barbules. This interlocking mechanism makes the feathers waterproof and improves their insulating capacity. 5) Cortex and 6) pith (modified from Lucas and Stettenheim³²).

body. The spaces between the tracts can facilitate the clinical evaluation of the skin. A single featherless region and its underlying integumentary components is called an *apterium*.

Terms used to describe parts of a feather are listed in Table 24.1.

The feather is composed of a long, central tapering shaft that is divided into the hollow base (quill, calamus) and an angular central shaft (rachis) (Figure 24.9). Barbs branch from both sides of the rachis, and barbules branch from both sides of the barbs (Figure 24.10). The anterior, middle and posterior portions of the barbule vary in structure. The anterior and mid-

dle barbules contain barbicels (hooks), which are missing in the posterior barbules. The posterior barbules contain ridges, to which the anterior barbicels are attached in a zipper-like fashion.

A feather appears as a unified sheet of tissue because of the interlocking barbules that hold the barbs together to form the vane on either side of the feather shaft. The interlocking nature of the barbules serves to waterproof the feathers, forming a type of thatched roof (Figure 24.11). The interlocking barbules also serve to improve the insulating capacity of the feathers and create an aerofoil to facilitate flight (see Chapter 8).

The feathers can be characterized based on the structure of the rachis, barbs and barbules, and are divided into ten feather types.³²

- **Contour feathers** represent the predominant feathers that cover a bird's body. They are the largest feathers and have a well developed shaft, pennaceous and plumaceous components of the vane and an afterfeather.
- **Coverts** are the small contour feathers that are found in rows on the wing and tail.
- **Remiges** are large, stiff, well developed feathers found in the wing and are principally responsible for flight. These feathers are generally asymmetric in form and have an entirely pennaceous vane. The remiges that arise from the periosteum of the metacarpus are called primaries, and those that arise from the periosteum of the ulna are called secondaries. The primaries are counted from proximal to distal (digits), while the secondaries are counted from distal (carpus) to proximal (elbow) (Figure 24.12). The number of primary and secondary feathers varies among species.
- **Rectrices** are large flight feathers found in the tail. They are structurally similar to the remiges. Tail feathers are counted from the center laterally.

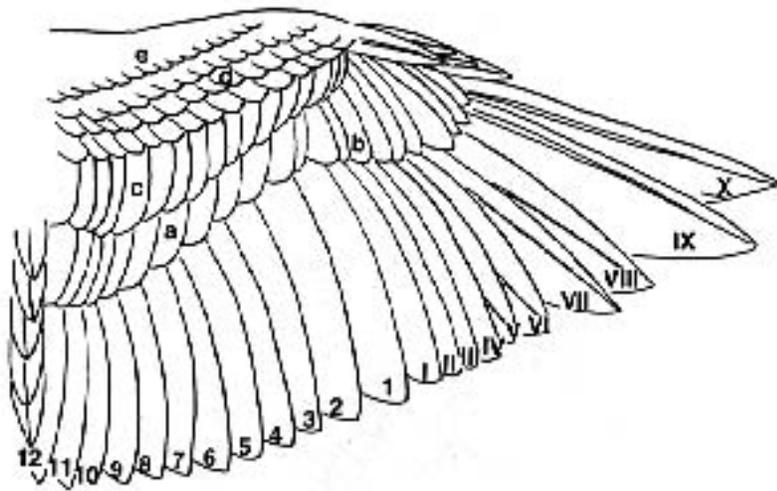


FIG 24.12 Dorsal view of the feathers of the wing: primaries (Roman numerals); secondaries (numbers). a) greater secondary coverts b) greater primary coverts c) median secondary coverts d) lesser secondary coverts and e) marginal coverts (modified from Lucas and Stettenheim³²).

- **Downs** (juvenile and definitive) are small, fluffy, wholly plumulaceous feathers with a short or absent rachis. Natal downs are present at or soon after hatching and are replaced during the first molt. Definitive down feathers occur on various parts of the body as part of the adult plumage. They are evenly distributed in parrots and waterfowl, confined to tracts in gallinaceous birds and sparse or absent in some pigeons and passerines.
- **Powder down** are specialized down feathers that disintegrate and produce a powder (keratin) that is spread through the feathers during preening. They are found throughout the body among the down and contour feathers. African Grey Parrots, cockatiels and cockatoos have the most abundant powder down feathers. Birds with damaged powder down feathers frequently have soiled-appearing feathers, suggesting their involvement in the maintenance of normal feather condition.
- **Semiplumes** have a long rachis and entirely plumulaceous vane. They occur in feather tracts of their own or are found along the margins of contour feather tracts. They provide insulation.
- **Hypopnea** (afterfeathers) are structures attached to the underside of a feather at the superior umbilicus. They may consist only of barbs or have a shaft and plumulaceous barbs.
- **Filoplumes** are fine, hair-like feathers with a long rachis and a tuft or barb on the tip. They

generally accompany contour feathers in most species. They are believed to serve a proprioceptive function.

- **Bristles** are characterized by a stiff, tapered rachis with no barbs except at the proximal end. They are usually found around the mouth, nostrils and eyes and are believed to serve a sensory function.

The feather follicles are formed by invaginations of the skin. The follicular wall has an abundant supply of sensory nerve fibers, and the papillae, pulp and feather muscles are also well innervated.³² Smooth muscles at the base of the feather follicles help maintain body temperature by increasing or decreasing the elevation of the feathers from the skin.

Herbst's corpuscles at the base of feather follicles are believed to detect subtle ground vibrations and changes in air current. Changes in

TABLE 24.1 Common Terms Used to Describe Portions of a Feather

Calamus	The short, tubular, unpigmented end of the mature feather that is inserted into the feather follicle and is thus present below the skin level.
Rachis	The long, solid, tubular portion of the shaft above the skin. It is a thickened continuation of the calamus external to the skin margin. The rachis contains pith, which is composed of air-filled keratinized epithelial cells surrounded by a solid keratinized outer cortex.
Shaft	The longitudinal central axis of a feather that is composed of the calamus and rachis. The calamus and proximal portion of the rachis are vascularized in the developing feather (pin feather).
Vane or vexillum	The portion of the feather that extends to either side of the rachis and is composed of the barbs and their associated structures. The vane is either plumulaceous (soft, downy) or pennaceous (compact and closely knit) depending on the individual type of feather.
Pulp	The mesodermal component of the growing feather consisting of vascular connective tissue. The pulp regresses as the feather grows and is absent in the normal mature feather.
Pulp caps	Keratinizing epidermis that covers the distal extremity of the pulp. As the pulp regresses, the keratinized caps remain and are visible as horizontal bars crossing the lumen of the calamus.

TABLE 24.2 Feather Coloration as a Result of Pigments and Structural Features**Pigments**

- **Melanins**
Alone, dull. When combined, create black, brown, reddish brown, yellow, red, purple and chestnut red-appearing colors. These pigments make feathers more dense and resistant to wear.
- **Carotenoids**
Bright red, orange, yellow. Cannot be synthesized and must be derived from ingested plants. Carotenoids have growth-promoting properties and impart a green color.
- **Carotenes and xanthophylls**
Xanthophylls are more readily absorbed from food than carotenes. Yellow = lutein.
- **Porphyryns**
Red and brown, true green.

Structured Color

- Due to the physical separation of the components of white light reflecting from or passing through the feather:
Iridescent colors change with the angle of view; eg, blue.
Noniridescent colors do not change with the angle of view (eg, green, purple and violet).

electroencephalographic activity following the removal of feathers suggest that it is a painful procedure.¹⁶ Clinically, the removal of a feather will frequently stimulate movement in an anesthetized bird at the same anesthetic plane that can be used to perform surgery.

Feather Color

The color of feathers is determined by two factors: the pigments that are deposited at the time of development, and structural features of the feather that alter the absorption or reflection of light (Table 24.2). These structural features of the feather can be inherent in the development of the feather or can be induced by materials that are placed on the feathers after development. If a feather reflects all wavelengths of light, it appears white; if it absorbs all wavelengths of light, it appears black. Dark-colored feathers appear to be more durable than light-colored ones.

The pigmentation of feathers may serve to absorb or repel heat (light), warn predators, act as a camouflage or function in mating displays. The capacity of the barbs and barbules to scatter and reflect varying wavelengths of light causes the iridescent glow of the feathers. Blue colors are created by the barbs interacting to reflect blue light while allowing other wavelengths of light to be absorbed by darker melanin granules. Green colors may be created by pigments, or more commonly through the combination of blue (from structural characteristics) and yellow pigments.

Colors tend to be brightest and boldest on the exposed surfaces of the feathers and paler on the ventral surface. Some red coloration in the appendages of birds is caused by vascularization and not pigment disposition. Pinching the tissue and observing for blanching can be used to determine if an area is vascularized.

The normal iridescent glow of the feathers may be induced in part by lipids derived from the keratinocytes. This “glow” is frequently absent in birds with clinical abnormalities and returns as a bird responds to therapy. The sheen of dark feathers has been suggested to be caused by the fat-soluble red and green pigments that are either synthesized by the bird (melanins and porphyryns) or absorbed from food (carotenes and xanthophylls).⁴⁴

It is interesting to note that abnormally colored feathers may return to normal without a molt. This is particularly common in cockatiels with feathers that are stained yellow secondary to chronic biliverdinuria (liver disease) (see Color 8). As birds respond to therapy for hepatitis, these feathers will return to a normal white coloration, presumably because biliverdin-laden, keratinocyte-produced lipids are replaced with lipids that do not contain biliverdin.

Yellow or red pigments derived from the uropygial gland can be spread on the feathers where the pigment remains bright until it fades due to oxidation from exposure to air and light. In a healthy bird, feathers maintain their bright pigmentation through the addition of newly synthesized oils during preening. These mechanisms for imparting color to a feather would allow changes in feather pigmentation to occur without a bird undergoing a molt.³² Birds receiving higher fat diets would be expected to produce a lipid-rich, keratinocyte-derived uropygial gland secretion that may enhance the color and sheen of the feathers.

In poultry, a lack of pigmentation (achromia) has been associated with dietary deficiencies in lysine, folic acid and iron.⁶³ Lysine deficiency has not been found to alter the pigmentation of cockatiel feathers but deficiencies of choline or riboflavin will cause abnormal pigmentation (see Chapter 31). Both melanism and albinism have been reported in a variety of captive and free-ranging species.

Peach-faced Lovebirds may develop red patches on their normally green plumage, and both diet and blood parasites have been suggested as a cause of

this condition. Abnormal yellow, red and pink feathers may be noted in Amazon parrots and African Grey Parrots, and it has been suggested that these are associated with hepatopathies, renal dysfunction or systemic disease. Psittacine beak and feather disease has been implicated in some cases of the abnormal occurrence of red feathers in African Grey Parrots.²⁹ Excess dietary levels of beta carotene can cause a similar feather change.

Molt

Soft keratin structures (skin, comb, wattles, cere) undergo constant replacement through the sloughing of the outer cornified layer (Figure 24.13). Old or damaged outer layers of hard keratin structures (rhamphotheca and metatarsal spurs) are replaced through normal wear. The thick, horny heel pads on the back joints of woodpecker, toucan and barbet neonates are molted at fledging. In cases of malnutrition or systemic disease, hyperkeratotic layers of the rhamphotheca can accumulate and be peeled off with a blunt instrument.

Molting is the process whereby the growth of a new feather causes the shedding of an old feather. The single generation of feathers that occurs as a result of a molt is collectively known as plumage. At any one time, a bird may have feathers derived from more than one molt. This is because some molts involve all of the feather tracts, while others involve only certain tracts or specific feathers. Collectively, the feathers present on the body at one time, regardless of when they first appeared, are called the feather coat.

A new feather that is still enclosed in a feather sheath is called a pin feather (Color 24.5). The physical characteristics and appearance of the feather are controlled by factors that affect the development of the feather at the edge of the epidermal collar. Any infectious agent or systemic abnormality that alters the nutrients or blood supply available to the developing feather will alter its appearance. Additionally, damage to the epidermal collar will be manifested clinically as an abnormal feather.

Feathers grow from the base and mature in an upward and outward fashion (Figure 24.14). The developing feather is composed of the outer epidermis and the inner pulp. The barb ridges, rachis and hyporachis are formed by the epidermis as it grows longitudinally. Lateral growth from a basal layer forms the keratinized sheath.

Once a new feather has been stimulated to grow in the follicle, the molting process is purely mechanical



FIG 24.13 The epidermis of birds is normally replaced on a constant basis. Excessively dry, flaky skin can be an indication of malnutrition or organopathies. In this cockatoo, a heavy molt and sloughing of sheets of the epidermis were induced by changing the bird from a wild-bird seed to a formulated diet.

and is strictly dependent on the developing generation of feathers; thus, the pattern of molt should be defined based on the *developing* feathers (which control the molt cycle) and not on the *shedding* of a feather (which has nothing to do with the molt unless the feather has been mechanically removed).

Molting Periods

The molting process can be divided into periods. The first molt occurs shortly after hatching and involves the replacement of the natal down, resulting in the second plumage (the first plumage would be the natal down). A parrot in its second plumage appears smaller than an adult because the feathers are reduced in length and width at this stage of development. The second molt in a juvenile leads to the third plumage, which is a divided process with many second and third generation feathers being present at the same time. The third molt occurs with the growth of the fourth generation of feathers, which should

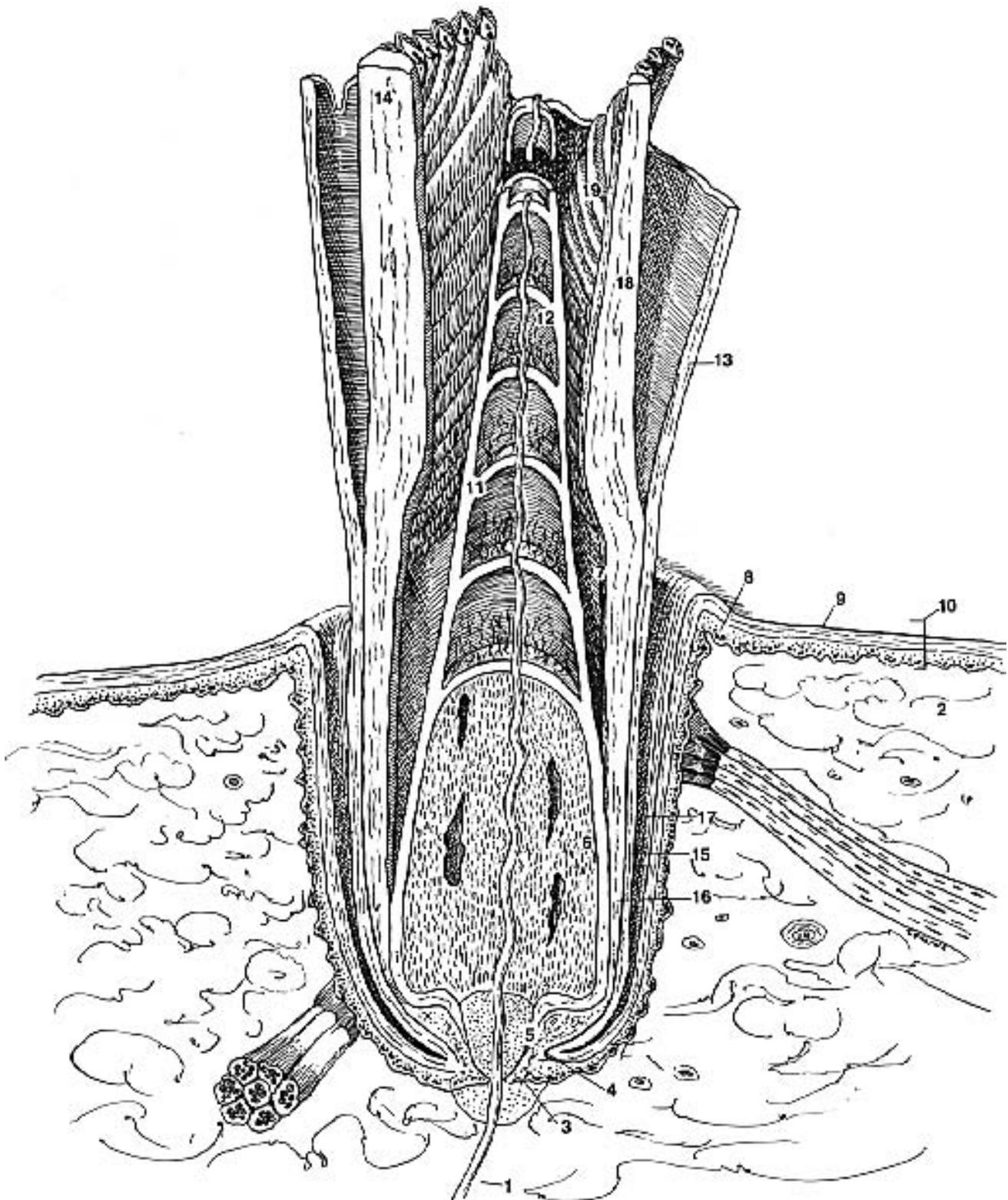


FIG 24.14 Illustration of the layers of feather development: 1) nutrient (axillary) artery 2) dermis 3) inferior umbilicus 4) epidermal collar 5) dermal papilla 6) pulp 7) calamus 8) germinative layer 9) corneous layer 10) epidermis 11) pulp cap 12) remnant of the axillary artery 13) degenerating feather sheath 14) rachis 15) intermediate follicular epithelium 16) basilar follicular epithelium 17) sheath follicular epithelium 18) hyporachis and 19) barb of vane (modified from Lucas and Stettenheim³²).

result in the development of mature plumage in a normal bird. Malnutrition may cause the adult plumage to be incomplete or abnormal.

The molting process in adult birds occurs on a cyclic basis. A molt cycle is defined as the period that runs from the appearance of a plumage to the appearance of its replacement. The cycle length for most birds is one year; however, some species will molt throughout the year, while others will molt annually or several times a year during distinct periods. Large Psittaciformes may have a two-year molt cycle. Powder down feathers are shed continuously. Most authors theorize that replacement of the adult plumage is synchronized with the gonadal cycles and will be longer or shorter than one year based on reproductive activity; however, molting may be more dependent on photoperiod. Domesticated birds that reproduce year round under artificial lighting conditions may not undergo the seasonal molt that would be expected to occur in their free-ranging conspecifics.

In general, the molting process of the flight and tail feathers starts with the proximal primaries on both wings and progresses until about half of the primaries are replaced. The secondary feathers are then molted in a distal to proximal progression. The body feathers begin to molt after the wing feathers are actively being replaced. The tail feathers are replaced from the central feathers outward. By having a progressive molt, birds are able to continue flying while the feathers are being replaced. In some waterfowl and seabirds, all of the flight and tail feathers are replaced at one time, and these birds go through a period of flightlessness. Penguins molt randomly.

Malnutrition can impact the speed of molt and the health of the developing feathers. The molt period increases a bird's metabolic rate and demand for protein. Birds that are on diets that contain insufficient energy or protein might undergo a partial molt of shorter than normal duration.^{5,65}

Companion and aviary birds may have abnormal molts caused by unnatural lighting conditions, malnutrition and environmental or disease-related stress factors. Many companion birds will have a new generation of feathers on the head and neck, with several generations of old feathers on the wings and body (Figure 24.15).

Molting has been suggested to be an autonomous process within the feather papillae, which may be triggered by seasonal changes. If the physiology of the papillae were to change in preparation for feather



FIG 24.15 A malnourished African Grey Parrot with light brown feathers (arrows) in place of the normal slate gray feathers. The bird was placed on a formulated diet and the newly developing feathers are correctly pigmented (open arrows).

growth, it might become increasingly sensitive to certain secretions (thyroid hormone, sex hormones) that could then potentiate the growth of a new feather.³²

The occurrence of a cyclic rather than systemically controlled molt seems clinically feasible given that all the feathers do not molt at the same time from all locations. The feathers appear to molt in sections starting with the head, neck and thorax, followed by the wing and tail feathers. The molt in each anatomic location may occur at varying times, and some pterygiae may undergo several plumage replacements before any feathers are molted from another area.

Control of Molt

The control of molting is extremely complex and only partially understood. The process probably involves a combination of hormonal, seasonal, nutritional and local (feather follicle) factors. The effects of individual hormones on the molt cycle appear to vary widely among avian genera, and information derived from studies in one species should be cautiously applied when evaluating clinical abnormalities in another species.

Specifically, the precise role that thyroid hormone plays in the molting process appears to vary among species. This hormone may affect the shape, structure, formation of pigment, color patterns and rate of growth of feathers. In fowl, administration of thyroid hormone may induce a molt in seven or eight days. If the thyroid is removed, feather formation on the body stops but the molt of wing feathers will continue, suggesting that their replacement is not controlled by thyroid hormone. Administration of high concentrations of thyroxine will increase the speed of the molt cycle. These findings suggest that thyroid hormone is important in initiating a molt; however, other studies indicate that progesterone and prolactin can induce a molt without a change in circulating levels of thyroid hormone.³²

In a study of King Penguins, it was found that thyroxine levels rose significantly (five times resting levels) during the molting period, and corticosterone levels increased at the end of the molt.⁵ In other studies, it has been demonstrated that thyroid activity (as measured by thyroidal uptake of radioactive iodine) did not differ appreciably between molting and non-molting hens. These apparent conflicts in experimental findings may suggest that research protocols, no matter how effectively conceived, may not accurately reflect the natural mechanism of molting.

Feather formation is prevented by circulating estrogens. Progesterone will stimulate feather growth in follicles that are already replacing a feather but will not stimulate feather development. Molts are sluggish and prolonged in fowl exposed to 12 to 14 hours of light. The effect of photoperiod on normal molt in companion birds that originate from widely varying geographic regions is undetermined. In studies in poultry, plasma prolactin, growth hormone and LH levels decrease, and testosterone and thyroxin levels increase, during the molt. Molting activity can be induced by high doses of medroxyprogesterone, decreased exposure to light or administration of thyroxine or prolactin.²⁴ Luprolide has been found to decrease the size of the ovary, elevate circulating testosterone levels and induce a molt.^{37a}

The nervous system may serve as a mediator between the rhythmic environmental events (principally photoperiod) and the hormonal activities of the thyroid and gonads that all combine to facilitate molting. Birds that are stressed by handling during a molt may lose more feathers than birds that are in a relaxed atmosphere. Some birds are able to release feathers when being restrained (fear or stress molt).

General Diagnosis and Therapy

Investigation of Dermatologic Disease

Integumentary diseases can be broadly classified as being caused by infectious or noninfectious agents (Table 24.3). In many cases, dermatologic lesions are secondarily infected with bacterial or fungal agents, and the identification of microbial agents from cultures of the skin does not necessarily implicate these organisms as the precipitating cause of the lesions.

Using a dermatology examination form is a concise way to consistently evaluate and record integumentary lesions. Making drawings or taking photographs is an effective method of recording the precise location and the effects of therapy on skin lesions. By using a standardized form and evaluation system, avian veterinarians and dermatologists can more effectively quantify and compare their findings, which will ultimately lead to improved clinical description, diagnosis and treatment of skin and feather diseases.

The predilection to develop certain types of integumentary diseases may vary among species (Table 24.4). The diagnostic evaluation used for avian dermatologic diseases is similar regardless of the etiology (Table 24.5). The evaluation of feather and skin lesions, particularly in small birds, can be facilitated by the use of a magnifying loupe. Inflammation of the skin can occur as a result of trauma, chemical irritation, bacterial, fungal, viral or parasitic agents. Pericloacal inflammation may be associated with the accumulation of excrement.

Cytology, culture and biopsy are indicated in cases of dermatitis. Cultures should be obtained by removing

TABLE 24.3 An Etiologic Approach to Integumentary Diseases

Infectious	Non-infectious
Viral	Traumatic
Mycoplasmal	Chemical/toxic
Chlamydial	Nutritional
Bacterial	Hormonal
Fungal	Developmental/genetic
Protozoal	Irradiation
Metazoal (parasitic)	Neoplastic
	Immune-mediated
	Behavioral
	Allergic

TABLE 24.4 Common Integumentary Diseases by Order**Passeriformes**

Poxvirus
Knemidokoptes infection
 Papillomatosis (pedal)
 Damaged nails and beak
 Constricted feet and digits
 Hyperkeratinization associated with malnutrition
 Bacterial dermatitis (often secondary to above)
 Loss of feathers around the head and neck - malnutrition
 Dermatophytes
 Trombiculid mites

Galliformes

Poxvirus
Knemidokoptes infection
Dermanyssus and *Ornithonyssus* infection
Echidnophaga gallinacea
 "Bumblefoot" syndrome
 Contact dermatitis
 Malnutrition
 Skin tumors
 Xanthomatosis
 Viral (Marek's disease, reticuloendotheliosis)
 Genetic
 Enlarged sternal bursa

Anseriformes

Malnutrition
 "Bumblefoot" syndrome
 "Wet feather"
 Vesicular dermatitis and photosensitization
 Leech infestation

Raptors

Malnutrition
 Poxvirus
 "Bumblefoot" syndrome
 Gangrene of wing
 Tuberculosis
 "Blain" (bursitis of carpus)
 Damaged nails and beak

Columbiformes

Poxvirus
 Feather defects associated with PMV infection
 Salmonellosis
 Neoplasia including melanomas

Ratites

Poxvirus
 Malnutrition

any scabs, moistening the culturette in the sterile transport media and rolling the tip over the lesion. Moistened swabs will yield better results than dry ones, and it is important that the swab be plated as soon as possible after collection. A quick and inexpensive diagnostic technique in practice is to apply a microscope slide to the affected area and to examine it cytologically (see Chapter 10). Skin biopsies are most diagnostic if collected from the center and the periphery of the lesion.

TABLE 24.5 Dermatology Database**Systemic**

Physical examination
 CBC, AST, LDH, UA, bile acids, CPK
 DNA probe for PBFV virus
 DNA probe for polyomavirus
 Gram's stain of feces
 Fecal examination for parasites
 Radiographs
 Thyroid levels - TSH test

Specific Integumentary Examination

Microscopic (operating or dissecting) examination of feather for parasites
 Cytology of pulp cavity (bacterial and fungal)
 Bacterial and fungal cultures of feather pulp
 Histopathology of biopsy specimens (skin and follicle)
 Electron microscopy of feather sections

General Therapy for Integumentary Lesions

In most cases, the therapy for feather and skin abnormalities caused by a number of factors is similar, with modifications necessary only to resolve specific disease agents. These general therapeutic considerations include:

- Correcting any nutritional deficiencies by administering parenteral multivitamins, minerals (trace minerals) and placing the bird on a formulated diet supplemented with some fruits and vegetables.
- Removing the bird from all exposure to aerosolized toxins that may accumulate on the feathers and skin and cause irritation (eg, cigarette smoke, kerosene fumes, cooking oils).
- Ensuring that the bird has frequent exposure to sunlight, and that a regular bathing program is instigated.
- Identifying and correcting any behavioral abnormalities that are causing over-grooming (feather picking).

Skin lesions should be kept clean, and creams, lotions or solutions can be used to moisturize and sooth dry, irritated skin and reduce pruritus and discomfort. Any medications placed on a wound should either kill specific target microorganisms or protect healing tissue. Ointments and oily compounds interfere with normal feather function and should be avoided (Color 24.3). Some commonly used lavage agents, povidone iodine compounds for example, are effective in controlling bacteria, but may also impair healing by destroying fibroblasts and white blood cells (see Chapter 40).⁶⁴ Hydrophilic compounds are often of value in birds but should not be used on large open

wounds where they may potentiate dehydration and electrolyte imbalance.

Aloe vera gel, human skin softeners with a vanishing cream base, nystatin-neomycin sulfate ointment^a (for pruritic lesions and moist dermatitis) and silver sulfadiazine cream^b (for moist dermatitis and burns) are particularly effective topical medications. A mixture of Penetran and aloe vera may relieve severe pruritus in some cases (see Chapter 18). This therapy should be discontinued or the solution should be diluted further if a bird becomes depressed or lethargic. If a bird does not improve within 48 hours of initiating therapy, the preparation should be considered ineffective and discontinued.

If an infectious agent is identified, specific antimicrobial therapy should be initiated. In some cases of severe ulcerative dermatitis, surgical debridement and primary wound management may be necessary; however, surgery should not be considered until all other therapeutic modalities have failed to resolve the lesions over a six-month treatment period. Peeling, flaking skin and heavy molts are common for prolonged periods (up to a year) when a diet change is initiated in a malnourished bird.

Lesions should be evaluated regularly (generally on a weekly basis) to determine if prescribed therapy is effective. Trimming the tip of the beak to prevent a bird from self-mutilating or applying a neck brace is justified only as a last resort.

Specific Etiologies of Generalized Dermatopathies

Viral Diseases

Dystrophic feathers may occur in birds infected with PBFD virus, polyomavirus, adenovirus and a parvovirus (waterfowl). Dermatologic lesions may occur with poxvirus, papillomavirus and herpesvirus infections (see Chapter 32).

Young birds are most susceptible to PBFD virus, which is characterized by the progressive appearance of dystrophic feathers after a molt (Colors 24.7, 24.13). The disease progression can be acute or chronic depending on the age and species of bird. A diagnosis of PBFD is

made by demonstrating viral antigens or nucleic acid in affected tissues. DNA probes are available that can be used to detect the virus in circulating white blood cells (see Chapter 32).

Avian polyomavirus (budgerigar fledgling disease) causes feather pathology in some affected budgerigars and occasionally in large Psittaciformes (see Chapter 32).

“French moult” is a descriptive term used to describe feather dystrophy in young psittacine birds, primarily budgerigars.¹ The classic clinical changes include premature molting of the wing and tail feathers and associated hemorrhage and poor plumage (see Color 32). Affected young birds are termed “runners” because they are usually incapable of flying. Feather changes characteristic of “French moult” can be caused by PBFD virus, polyomavirus or both (Color 24.7). It should be noted that any factor (infectious or noninfectious) that damages the epidermal collar can result in a gross lesion resembling that induced by PBFD virus or polyomavirus (see Color 32).

There is no specific treatment for French moult. Techniques that are discussed in the lay literature, including dietary additives and careful selection of breeding stock, are probably futile. Good hygiene is advisable, and birds should be purchased from sources that test free of PBFD virus and polyomavirus (see Chapter 32).

Poxvirus can cause skin lesions in most avian species and may retard wound healing. Uncomplicated lesions are characterized by the formation of nodules on the unfeathered skin. Skin lesions should be kept clean and dry to prevent secondary bacterial or fungal infections (see Chapter 32).

Cutaneous papillomas may occur on the head, neck, beak commissure, feet or uropygial glands. Some of these lesions have been associated with papillomavirus or herpesvirus while others are of undetermined etiology. Therapy is generally limited to removal of the masses in birds in which they cause problems. A herpesvirus has been associated with “feather dusters,” and adenoviral folliculitis has been reported in lovebirds (see Chapter 32).

Parasites

Wasps, bees or other stinging insects will occasionally attack birds causing characteristic hyperemic swellings (Color 24.17). Most affected birds heal with no therapy; however, in severe cases steroids may be

indicated to reduce inflammation. The likelihood of a bird being stung can be reduced by removing uneaten soft foods (particularly fruits) from the enclosure and destroying wasp nests found near the aviary.

Flies, mosquitoes and gnats can cause severe dermatitis on the face, feet and legs, particularly in birds raised in warm coastal areas (see Color 26). Lesions are most common in Amazon parrots and macaws, but can occur in any species. The flies that commonly parasitize cattle and deer can induce small bleeding ulcers on the unfeathered areas of the body (Color 24.17).

Ants (especially fire ants) can be a nuisance to nesting birds. If necessary, five per cent Sevin dust can be used in the nest box to prevent chicks from being eaten alive. Many affected chicks die, and those that survive may have localized necrotic areas that are secondarily infected with *Staphylococcus* spp. Topical application of antibiotic and steroid lotions or creams can be used to reduce swollen or hyperemic lesions. Ant bites also may cause localized necrosis that results in defects in the webs of the feet in waterfowl (Figure 24.16). Some helminths and mites can cause dermatitis (see Chapter 36).

A sarcoptid mite infection was described in a Grey-cheeked Parakeet with feather loss and flaking skin on the head and trunk. Severe pyogranulomatous dermatitis was associated with a sarcoptic mite infection in a Green-winged Macaw. The bird did not respond to ivermectin therapy.⁵⁵ Generalized alopecia and thickening of the calamus occurred in a Red-fronted Parakeet infected with *Knemidokoptes* spp. (see Chapter 36).

Mites are more likely to be a primary cause of dermatitis on the head than are lice. Control of ectoparasites, whether on the head or elsewhere, must be undertaken with caution. Only those parasitocidal agents that are licensed or recommended for use in birds should be applied, and such therapy must be accompanied by other measures to exclude the parasites.

In subtropical and tropical areas, the sticktight flea (*Echidnophaga gallinacea*) can be a problem on many species of birds.¹⁰ This is a sessile flea, and large numbers may attach to the skin of the head, especially around the eyes, and cause anemia (see Color 8). This parasite can be controlled with the topical application of a pyrethrin-based product.



FIG 24.16 Defects in the interdigital webs in a duck secondary to multiple fire ant bites.

Bacterial and Fungal Diseases

There have been remarkably few studies on the bacterial flora of the avian skin other than in poultry and birds of prey.³⁹ Surprisingly, fungi have attracted more attention, and several surveys on the fungal flora of free-ranging birds have been reported. In one study, 6000 fungi were recovered from the feathers, nests, pellets, droppings and organs of 92 species of free-ranging birds.²³ Several of the fungi isolated were potential pathogens and a number were keratinolytic.

Many authors have suggested theories to explain the apparent paucity of primary skin infections in birds, including a high body temperature, which might inhibit the growth of some organisms, and keratinocyte-derived lipids that may inhibit certain pathogenic bacteria or may provide appropriate nutrients for competitive autochthonous flora.⁵⁴ Bacterial and fungal infections of facial skin are usually secondary to trauma or possibly a contact dermatitis. Avian skin abscesses are rare but can be found following wounds or in association with feather cysts. Treatment is routine with surgical drainage or removal.

Although frequently discussed, documented cases of bacterial folliculitis in birds are rare. The pulp can be examined for the presence of bacteria by making impression smears or by culturing the pulp cavity (Figure 24.17). Bacterial pathogens that have been implicated in folliculitis include *Staphylococcus* spp., *Aeromonas* spp. and *Mycobacterium* spp.^{13,44,60} Dermatitis of the head and body was associated with mycobacteriosis in an Amazon parrot.¹⁴ *Staphylococ-*



FIG 24.17 A macaw was presented with dystrophic feathers. Numerous developing feathers were frayed, and the pulp cavities were split and contained dried blood and developing feather components. The pulp cavity of birds with this type of presentation should always be examined cytologically, and cultures should be submitted for bacterial and fungal isolation. The diet should be carefully evaluated in these cases.

cus spp. are frequently isolated from exudative ulcerative skin lesions of the patagial area (Color 24.23). The importance of staphylococci in the occurrence of these lesions has not been defined. Secondary fungal agents may also be recovered from these lesions.¹⁸

Trichophyton spp. (*flavus*) have been associated with scaly, crusty lesions of the wattle, comb and legs in gallinaceous birds (see Color 8). *Trichophyton* spp. and *Microsporium gypseum* have been reported as a cause of dermatitis of canaries and budgerigars, respectively.²⁷ A vesicular dermatitis was reported in chickens following the ingestion of *Cladosporium berbarum* fungus.⁴⁷

Aspergillus spp. have been associated with skin and feather lesions, particularly in pigeons. Affected feathers are generally dry, have yellow spots and are scaly.⁶⁶ Candidiasis has been associated with skin and feather lesions in gallinaceous birds.^{30,66}

Mucor circinelloides was recovered from three pigeons experiencing severe feather picking and self-mutilation problems. *Rhizopus arrhizus* was isolated from a lovebird, *Penicillium chrysogenum* from a par-

rot, *A. candidus* from a cockatiel and *A. phoenicis*, *P. cyclopium* and *M. circinelloides* from parakeets that were mutilating feathers. The birds in these cases responded favorably to fungicidal therapy, suggesting that the fungus was involved in the feather picking behavior. The use of STA (3 g salicylic acid, 3 g tannic acid and 100 ml ethyl alcohol) applied bi-weekly is particularly effective in controlling integumentary fungal infections.⁶⁶

Nutritional Factors

The ability of avian skin to resist infections and to heal properly is related to many factors, the most important of which is the nutritional status of the bird. Malnutrition, particularly hypovitaminosis A, is suggested by the smoothing of the normally papillary surface of the plantar surface of the feet (see Color 8).

Hyperkeratosis of the feather sheath may occur as a result of malnutrition or in association with some infectious agents that affect the developing feather (eg, PBFV virus, polyomavirus). In affected feathers, the sheath on the developing feather is retained, resulting in a bird that appears to have an excess number of pin feathers. The precise effects that malnutrition and organopathy (particularly hepatopathy) have on the quality and pigmentation of feathers remain undetermined; however, their role is suggested clinically by the frequency of abnormal plumage in birds fed marginal diets and with hepatopa-



FIG 24.18 A grackle that had been fed a baby cereal diet was presented for weakness and poor feather formation. The feather barbs were not connected properly, making the vanes appear like they contained holes. The bird was placed on a formulated diet and molting activity started within several weeks. The newly developing feathers were properly formed (arrow).

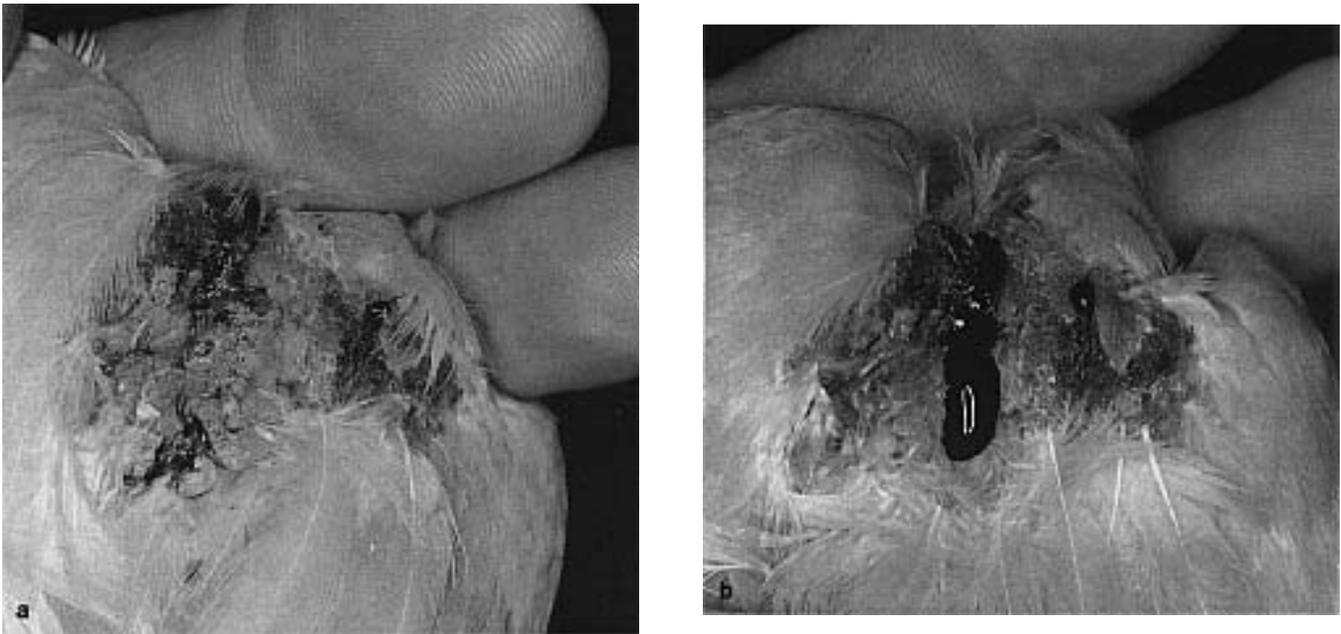


FIG 24.19 a) Chronic ulcerative dermatitis in the cervical patagium and interscapular area of a canary. b) The skin was extremely dry and brittle, and the least flapping motion would cause spontaneous tears and hemorrhage. The etiology of these lesions was not determined, but the bird responded to bandaging, a diet change, frequent exposure to sunlight and keeping the wounds clean with dilute chlorhexidine solution.

thies (Figure 24.18). Further, many generalized feather abnormalities will resolve when a bird is placed on a proper diet or when an organopathy is effectively treated.

A malnutrition-induced loss of feathers on the back of the head and neck is believed to occur in canaries. Affected birds are usually egg-laying females and also may show decreased fertility and produce weak chicks. Dietary changes will usually resolve the lesions.

■ Nonspecific Dermatopathies

Many minor scratches and cuts (that are not caused by animal bites) require no medical attention, especially if they are in the non-feathered areas of a healthy bird. If a severe wound occurs, the feathers can be trimmed or pulled from the periphery of a lesion to prevent the accumulation of necrotic debris. Most companion and aviary birds do not pick at skin injuries (see Chapter 16).

Burns occasionally occur in companion birds (Color 24.1, 24.2) Treatment should include debridement and topical antimicrobial agents (see Chapter 15). A “stress-related” dermatitis has been reported in lovebirds, cockatoos and budgerigars. *Staphylococcus* spp. are frequently recovered from these birds, and

topical drying agents and antibiotics may be effective therapy.

■ Chronic Ulcerative Dermatitis (CUD)

Chronic ulcerative dermatitis is characterized by septic, edematous and hyperemic ulceration and exudation of the skin (Figure 24.19). Chronic ulcerative dermatitis has been associated with tumors (lipomas, squamous cell carcinomas and papillomas), abscesses, unhealed wounds, hernias, mycobacteriosis, diabetes, nephritis, hepatitis and giardiasis. Biopsies should always be performed on proliferative, chronic skin lesions to determine if they are neoplastic in origin.

Giardiasis and hypovitaminosis E seem to be associated with ulcerative dermatitis in lovebirds and cockatiels (Color 24.24). The precise nutrients that may be missing in the diet have not been defined, but these birds are frequently fed seed-based diets with or without the addition of fruits and vegetables. Many cases of CUD will improve when a bird is placed on a balanced, formulated diet and provided with adequate exposure to sunlight. Complete resolution may not occur for several months after these management changes are initiated.

Propatagial CUD

Lovebirds, cockatiels, Grey-cheeked Parakeets and occasionally Amazon parrots and cockatoos may develop chronic ulcerative dermatitis involving the metapatagium or propatagium (Color 24.23). Lesions may also be noted in the proventer and in the interscapular regions of the body. The lesions appear to be extremely pruritic. Outbreaks of ulcerative dermatitis affecting patagial membranes have been described. In one outbreak, 60% of the lovebirds in a flock were affected, and the progression of the disease suggested an infectious agent.⁴⁹

Treatment for propatagial CUD should include metronidazole for giardiasis (if identified), administration of parenteral vitamin E, removing the feathers from the periphery of the lesion and placement of a figure-of-eight bandage to prevent mutilation. Secondary bacterial or fungal infections should be treated with appropriate topical medications.

Surgical debridement and primary wound closure may be necessary if the lesions do not heal in five to six weeks. Radiosurgery should not be used to debride or control hemorrhage associated with these lesions. Birds with long-term or severe lesions will replace the normally elastic patagial tissue with scar tissue, which may make the bird more susceptible to future lesions.

CUD in Other Regions of the Body

Ulcerative dermatitis of the proventer region may occur in heavy-bodied birds (African Grey and Mealy Amazon Parrots) that have had improper wing trims. A bird that attempts to fly from a high perch and has no lift may land on its sternum, resulting in a bruise or open wound over the cranial portion of the keel. These damaged tissues seldom become infected although cellulitis of the area is common (see Color 8).

The skin wounds should be treated as discussed under general therapy for integumentary lesions, and several of the clipped primary and secondary feathers from each wing should be removed to stimulate replacement of the feathers. These new feathers will provide the bird with the necessary lift to prevent further injury. In severe cases, necrotic portions of the keel must be surgically removed. Supportive care is successful in most minor cases and the lesions generally resolve in six to nine weeks.

Birds with chronic ulcerative dermatitis in the caudal aspect of the postventer region may be presented with a history of blood-tinged excrement. Feathers



FIG 24.20 A mature cockatiel on an all-seed diet was presented for evaluation of bloody diarrhea. An ulcerated lesion was noted in the postventer region. Bilateral ulcerative lesions of the metapatagium were also present. Pruritic skin lesions and ulcerative dermatitis in cockatiels appear to be associated with primary malnutrition or giardiasis. In this case, giardia could not be documented and the bird responded to a change in diet. The tail feathers were transected to reduce the pressure on the postventer skin. The lesions were cleansed daily with chlorhexidine solution and were coated with live yeast derivatives twice a day. Cloaca (arrow).

adjacent to and covering the skin lesion may be stained with blood. This lesion is common in malnourished birds and may begin when a bird with an improper wing clip lands on a hard surface. The impact of the tail with the ground causes a hyperextension of the rectrices and places excessive pressure on the tight skin of the proventer region (Figure 24.20).

Disorders Affecting the Feet and Legs

Skin on the legs may be damaged by bands (rings) or, in the case of falconers' birds, by badly fitted leather jesses. Secondary bacterial infections of skin wounds can occur and impair healing, particularly when a foreign object is constantly in contact with the wound. The application of a self-adherent wound dressing (see Chapter 16) will keep the wound clean and moist and permit regular visual inspection.⁸

Pox lesions on the feet and legs are characterized by dry, brown plaques. Other viral infections appear to be rare, but a herpesvirus has been implicated in skin lesions in Mallard Ducks and cockatoos (see Chapter

Dermatology

Color 24.1

A goose sustained severe burns on the unfeathered portions of the face, feet and legs after being trapped in a yard fire. The wounds were debrided and flushed repeatedly with copious amounts of sterile saline solution.

Color 24.2

Burns on the legs and feet of a goose were cleaned and treated with silvadene cream TID. This photograph, taken two weeks after the initial burns, shows a healthy bed of granulation tissue over the burns, and the bird healed with no complications.

Color 24.3

A mature, male budgerigar with dermatitis was presented for progressive shivering and depression. The bird had been treated with an over-the-counter, oil-based antibiotic. The oil-laden feathers had lost their insulation ability, causing the bird to lose excessive amounts of body heat. The oil was removed with repeated washing in warm dishwashing detergent, and the bird was placed in an incubator (94°F) to dry.

Color 24.4

A proliferative skin mass on the abdominal wall of a cockatoo with PBFD virus. The mass had histologic features characteristic of a papilloma. Part of the diagnostic evaluation for any proliferative skin mass should be a biopsy to rule out neoplasm.

Color 24.5

Normal primary pin feathers (blood feathers) in a developing Moluccan Cockatoo neonate.

Color 24.6

Feather cyst in a budgerigar. The cyst was surgically removed and the bird had no further complications.

Color 24.7

Primary feathers removed from a pionus parrot with PBFD virus. The infection was confirmed by DNA probe testing of whole blood and by histopathologic evaluation of dystrophic feathers. Note the constricted

calamus, areas of necrosis and hyperkeratotic feather sheaths. This bird was exposed to PBFD virus when infected neonates from another collection were introduced to the nursery. The fact that part of the distal feather is normal indicates that there was no damage occurring to the follicular epithelium when this part of the feather was developing.

Color 24.8

Feather cysts are common in canaries, particularly the Norwich, Crested, Crest-bred and new color canaries that have “double-buff” soft feathers. In severely affected birds, feathers emerge in all directions (courtesy of Patricia Macwhirter).

Color 24.9

Split section of a feather cyst showing the accumulation of cellular debris in multiple follicles (courtesy of John Cooper).

Color 24.10

Straw-feather disease has been described in canaries and a few other Passeriformes and is believed to be genetic in origin. There is incomplete development of the feather barb and barbules and there may be retention of the feather sheath in some affected feathers (courtesy of Louis Filippich).

Color 24.11

Segmental discoloration, black lines or transparent areas across the vane of a feather are called stress marks and indicate a dysfunction of the epidermal collar at the time the feather was developing.

Color 24.12

Magnified view of stress marks in a developing feather (courtesy of John E. Cooper).

Color 24.13

Necrotic, dystrophic contour feathers on the body of an adult Umbrella Cockatoo with chronic PBFD virus. Note the dystrophic growth, areas of necrosis and constriction of the feather shaft at its interface with the edge of the epidermis.





Dermatology

Color 24.14

Knemidokoptes spp. infections are most common in budgerigars and passerine birds, but may also occur in other birds, such as this Sulphur-crested Cockatoo. This bird responded to topical ivermectin therapy, but did have some post-infection feather loss and damage to the eyelids.

Color 24.15

Knemidokoptes spp. may cause severe proliferation and deformity of the beak. Note the skin on the face is also infected.

Color 24.16

A mature Yellow-collared Macaw was presented for severe depression. The head was covered with normal feathers but the remainder of the body was featherless. Several areas of self-mutilation were present including both feet and legs and the cervical patagium. An etiology could not be determined for this bird's self-mutilation.

Color 24.17

Hyperemic, indurated masses secondary to wasp stings in a Blue and Gold Macaw.

Color 24.18

Brown hypertrophy of the cere in a male budgerigar. This syndrome is believed to be caused by imbalances in the ratio of sex hormones.

Color 24.19

A mature Amazon parrot was presented with an acute onset of picking at the feet and legs. This photograph was taken several hours following the onset of the picking behavior and is characteristic for the acute phase of the Amazon foot necrosis syndrome. The cause of this bird's problem could not be determined, but it responded to general dermatologic therapy.

Color 24.20

A Golden Eagle was presented with severe bilateral swelling of the metatarsal pads. One pad was ulcerated, and a thick, greenish-yellow discharge was present in the center of the mass. The necrotic material was surgically removed from both feet and the wounds were packed with antibiotic-impregnated gel foam. A healthy granula-

tion bed was produced within three weeks of initiating therapy, and walking bars that were stabilized in a tarsometatarsal cast were used to allow the bird to ambulate without placing pressure on the plantar surface of the foot.

Color 24.21

A mature cockatiel hen was presented for severe feather picking. The bird had removed most of its feathers from the axillary and leg regions. *Giardia* sp. was diagnosed by examining a fresh wet mount fecal sample. The bird responded to therapy with metronidazole.

Color 24.22

Knemidokoptes spp. mites have caused proliferative yellowish-colored lesions on the foot of a canary. The bird was presented with a shifting leg lameness.

Color 24.23

Chronic ulcerative dermatitis in the ventral proapatagium of a mature cockatiel hen. Note that the feathers are melanistic. This bird had biliverdinuria and responded to a dietary change and therapy for liver disease. The feathers returned to normal color with subsequent molts.

Color 24.24

A lovebird was presented for progressive feather picking. The bird would scream as it picked at the tissues of the chest, back and wings. Ulcerative lesions were present on the cranial edge of both proapatagial membranes. *Staphylococcus* spp. and *Candida* spp. were isolated from the wounds, but they were considered secondary pathogens. The bird responded to therapy for chronic ulcerative dermatitis (and wing splinting), but many of the feather follicles had been destroyed and the bird remained featherless in some areas.

Color 24.25

Necrotic digits in adult passerine birds are commonly caused by fibers that wrap around the toe. Diagnosis usually requires examining the proximal edge of the affected digit under a dissecting or operating microscope.

32).⁷⁰ Proliferative, hyperplastic lesions on the feet of canaries and mynahs have been associated with abrasions, aging and malnutrition. A condition involving cracking of the feet that is responsive to high doses of biotin has been documented in flamingos, ratites and waders (see Color 48) (Greenwood A, unpublished).

Keratomas that appear clinically as digit-like projections composed of hyperkeratotic scales have been described in some species. These callus-like growths may predispose a bird to bumblefoot (see Chapter 43). Virus-induced papillomas are common on the feet of finches in Europe.²

“Bumblefoot” or pododermatitis has been reported in many species of birds but is a particular clinical problem in captive birds of prey, Galliformes, Anseriformes, waders, penguins and many Psittaciformes (Color 24.20). In Psittaciformes and Passeriformes, most lesions are believed to be the result of malnutrition, which causes the skin of the foot to become dry and hyperkeratotic. Hepatic dysfunction may also be involved in some cases. Penetrating wounds or bruising of the feet may be predisposing factors in raptors and Anseriformes (see Chapter 16).⁸

A “constricted toe syndrome” has been described in a number of Psittaciforme neonates (see Color 30). The fibrous band can be surgically excised to correct the problem (see Chapter 41). Other causes of ischemic necrosis of the feet or legs may include entangled fibers, hairs, bedding material, leg bands, strings, jesses, dried skin, frostbite or ergot poisoning (Color 24.25).

Pruritic, ulcerative lesions have been described on the feet and legs of Amazon parrots (particularly Yellow-naped and Double Yellow-headed Amazon Parrots). The lesions start with a bird chewing at the feet and legs followed by the formation of hyperemic lesions, sometimes within minutes of the initial pruritic episode. An ulcerative dermatitis occurs as the bird continues to chew on the feet and legs (Color 24.19). Characteristic histopathologic findings associated with this syndrome include ulcerative dermatitis that may contain coccoid bacteria and fungi. The role that the bacteria or fungi play in the pathogenesis of this syndrome is undetermined. Immune-mediated and allergic reactions with secondary involvement of autochthonous skin flora have been proposed as etiologies for these lesions (see Chapter 33).

Staphylococcus spp. are frequently isolated from the lesions, but the birds will usually not respond to antibiotic therapy alone. The syndrome appears to be

more common in the spring (suggesting a seasonal allergen), and many affected birds belong to cigarette smokers. In these latter birds, the lesions may spontaneously resolve when the clients stop smoking or wash their hands before handling the birds. Other cases will respond to a change in diet, frequent exposure to sunlight and a topical antimicrobial cream containing steroids. Topical steroids should be applied with caution to prevent toxicity.

Atarax and oral antibiotics were found to be effective in some cases.²¹ Seasonal recurrence of the lesions may be prevented by the oral administration of prednisolone about one month prior to the time that lesions typically occur.

A hydroactive dressing can be used to facilitate healing of these wounds. Initially, the bandage may require daily changing. The frequency of bandage changes can be reduced as the wound becomes less exudative. Once granulation tissue forms at the edge of the ulcers, scabs should be removed and the lesions should be kept clean to facilitate healing (see Chapters 15, 16).

Some reports detail the use of thyroid supplementation as a therapeutic regimen for foot necrosis syndrome; however, thyroid levels were not determined in the treated birds and the indiscriminate administration of thyroxine, can cause fatal toxicity (see Chapter 23).

Diseases of the Feathers

The appearance of malformed, broken, bent, dirty, stained or unusually colored feathers should be considered abnormal. Feather conditions can be divided into two main groups: those affecting normal feathers and those in which abnormality of the feather is the primary feature. A simple method to determine if a feather problem occurs during or after development is to remove an affected feather (it should be examined cytologically, microscopically and possibly histologically) and evaluate the growth of the new feather over the next one to three weeks. There are three possibilities with respect to the new feather:

- The feather does not regrow (suggests a systemic or follicular abnormality)

TABLE 24.6 Common Pathologic Terms Used in Avian Dermatology

Term	Definition
Acanthosis	Hyperplasia of the stratum germinativum
Acantholysis	Lack of cohesion between epidermal cells, leading to formation of clefts, vesicles and bullae
Atrophy	Decrease in size of a tissue or organ – in dermatology usually refers to thinning of the epidermis
Ballooning degeneration	Intracellular accumulation of fluid (edema)
Depigmentation	Loss of (melanin) pigmentation
Dyskeratosis	Prematurely cornified cells with eosinophilic cytoplasm and small dark nuclei
Excoriation	Secondary ulceration that may occur following self-inflicted trauma
Hyperkeratosis (hyperkeratinization)	Increased thickness of the stratum corneum
Hyperplasia	Thickening of the epidermis
Hypopigmentation	Reduced (melanin) pigmentation
Melanosis	Dark appearance due to increased melanocyte activity and deposition of melanin
Parakeratosis	Retention of pyknotic nuclei in the cells of the stratum corneum, usually associated with defective keratinization
Spongiosis	Extracellular accumulation (edema) causing separation of epithelial cells
Telangiectasis	Persistent vasodilation: skin does not blanch when compressed with a microscope slide

TABLE 24.7 Incidence of Histologic Lesions in a Group of 213 Feather Biopsies⁶⁰

Diagnosis	Number of Affected Birds
PBFD virus	32
Suspect PBFD virus	20
Normal skin and feathers	26
Inactive feather follicle - no lesion	22
Epidermal atrophy	6
Staphylococcus dermatitis	3
Other pyodermas	17
Suspected bacterial pulpitis	9
Dermatomycosis	3
Sarcoptic mange	1
Hypersensitivity reaction	78
Trauma	46
Drug eruptions	2

- The feather regrows but is not normal (suggests a problem in the feather follicle or organopathy)
- The feather regrows normally (suggests that the feathers are being damaged after development, eg, feather chewing, enclosure trauma).

Biopsy and histopathology are indispensable for diagnosing the cause of feather lesions. Some common descriptive terms that may be needed by the clinician to interpret the results of pathology reports are listed in Table 24.6. The results of 213 feather biopsies from a group of Psittaciformes are listed in Table 24.7.⁶⁰

Stress Marks

Translucent lines across the vane of a feather are frequently referred to as stress marks (Color 24.12). These abnormalities represent segmental dysplasia that occurred in the developing barbs and barbules and represent a brief period of dysfunction in the epidermal collar (Figure 24.21). These marks can be induced by the administration of exogenous corticosteroids, suggesting that they are truly “stress” marks. Restraint, illness, a brief period of food deprivation or exposure to environmental extremes should be expected to induce these lesions. Deficiencies of arginine (curled wing feathers), riboflavin (clubbed down feathers) and pantothenic acid, niacin and selenium (poor feathering) are nutritional causes of poor feather structure in poultry.⁶³

Preening

Much of a bird’s day is spent in feather preening, a natural process for maintaining feather condition. Feather preening appears to be innate, but occasionally a hand-raised neonate will have poor quality feathers or an excess number of pin feathers because of an improper preening response. These birds should be taught to preen the feathers by gently breaking the sheaths while encouraging the bird to pick at an area with its beak. Some wear of the feathers should be considered normal. Over-preening (feather picking) occurs when what is a normal part of feather maintenance becomes a pathologic condition (see Chapter 4).

Some birds may molt feathers on a seasonal basis from the ventral abdomen and lower legs (developing a brood patch). Damage to the feathers of the breast, abdomen and legs during the breeding season may indicate reproductive frustration. Seasonal feather picking associated with breeding activity is usually temporary and no specific therapy is necessary or warranted unless the feather loss is persistent or involves areas other than the lower abdomen.

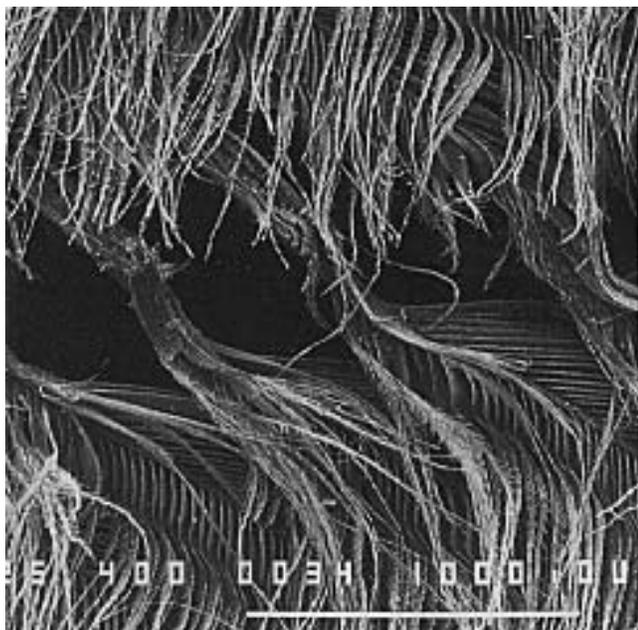


FIG 24.21 Scanning electron micrograph of a feather with segmental dysplasia (stress marks). Note that the barbs and barbules that were present in the epidermal collar at the time the stress occurred are improperly formed (courtesy of John Cooper).

Feather Picking

Feather picking occurs when a bird damages its feathers or skin (or the feather and skin of a companion). Feather picking is a condition of captivity. With the importance of the feathers for thermal regulation and flight, severe, self-induced feather damage would be life-threatening to a free-ranging bird. Clinically, feather picking is characterized by the loss or damage of feathers on the body and neck with normal feathers on the head (Figure 24.22). Feather picking induced by a companion is characterized by loss or damage of feathers around the head and neck. Male cockatiels will occasionally over-preen the orange face patch of the hen.

Feather picking can become an obsessive behavior with a bird progressively damaging all or part of the accessible plumage, leading to abnormalities in normal feather development and molt. Self-mutilation is characterized by over-preening and subsequent damage to the skin or muscle. Mutilation of the skin can cause, or occur secondary to, chronic ulcerative dermatitis.

Many feather-picking or self-mutilating birds are considered to be pruritic, which is difficult to document. Over-preening and scratching an area with the nails is suggestive. Inflammation or irritation associated with internal pathology, including that caused

by infectious agents, has been suggested as a precipitating factor for feather picking.¹⁹ Organopathy, toxins, malnutrition, bacteria, viruses, fungi, parasites (blood or intestinal), boredom, anxiety, lack of sleep, psychosis and undesired contact with strangers or family pets (dogs or cats) have all been implicated in cases of self-mutilation.⁵⁵ Feather loss on the neck of lorries and Hyacinth Macaws has been attributed to contact with conifers.

Mutilation is a commonly discussed problem in poultry and has been associated with improper management, crowding and malnutrition. In humans, hepatopathies have been associated with pruritus, and many self-mutilating birds have clinical changes suggestive of liver disease.²¹

Some birds may be mutilated by other birds (canaries, finches, conures, cockatoos). In colony-breeding flocks, reducing the number of birds in the enclosure, increasing the number of hiding places and nest boxes or removing the offending birds may be necessary for control. Cockatoos may occasionally over-preen a mate, but more commonly a male bird will kill its mate with no previous indication of aggressive behavior (see Chapter 2).

Examination of the Feather-picking Bird

Feather-picking birds should be approached in a systematic fashion.^{7,19} A diagnosis of psychologically induced self-mutilation should be reserved for patients in which no cause for the problem can be identified by physical examination, complete blood count (CBC), serum chemistries, feather pulp culture and cytology, skin lesion culture and cytology, radiographs, endoscopy and direct microscopic examination and biopsies of affected feathers. If no etiology can be determined for the over-preening, then behavioral abnormalities should be considered.

There is an apparent species' predilection to feather-picking behavior. African Grey Parrots appear to be particularly prone to feather picking, perhaps as a result of their sensitive natures or need for a highly stimulated environment. Spoiled, improperly socialized, hand-raised birds of any species may also be prone to self-mutilation. Cockatoos and conures frequently develop feather-picking behavior for which an etiology cannot be conclusively identified, necessitating a diagnosis of psychologic feather picking. By comparison, idiopathic feather picking in budgerigars and cockatiels is rare. In these species, feather picking associated with ulcerative dermatitis of the patagial membranes is most common (Color 24.21).



Treatment of Feather Picking

Once initiated, feather picking can become habitual and continue even though the precipitating cause is no longer present. Chronic feather picking can result in sufficient damage to the follicles to prevent any future feather growth (Figure 24.22). Therapy for self-mutilation of undetermined etiology should be considered effective if the destructive behavior can be reduced. Complete cessation of self-mutilation is rare.

In a retrospective study of 106 feather-picking cases, 31 had no change on follow-up examination; resolution of the problem occurred in 20 cases; 21 showed some improvement; and 34 were lost to follow-up. Amazon parrots and cockatiels appeared most likely to respond to treatment. Excluding birds with confirmed PBFV virus infections, treatment of other feather abnormalities with an etiology that was determined by the minimum database was generally successful. Idiopathic cases of feather picking were less likely to respond to therapy.⁶⁰

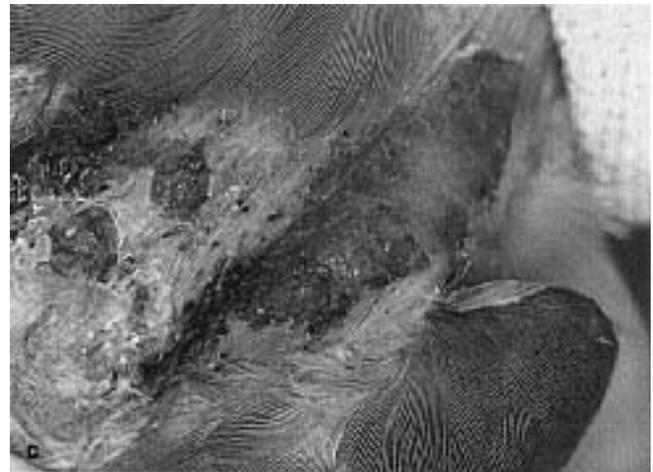


FIG 24.22 a) Feather picking is characterized by normal feathers on the head and neck, with damaged feathers at other locations where over-preening can occur. b) With chronic feather picking, the follicles may be permanently damaged, and feather regrowth cannot occur. c) In severe cases of self-mutilation, a bird may actually induce wounds in the skin or muscle.

Occasionally, a bird will self-mutilate as a result of sexual frustration. Some of these birds will stop mutilating when placed in a breeding situation; however, others will continue self-mutilation activities and may also over-preen a mate. Assuming that idiopathic self-mutilation is a result of some undetectable neurosis, it would be considered unwise for these birds to be added to a breeding collection where they may pass on genes that will predispose their progeny to the same problem.

There are probably as many recommended therapies for the feather-picking bird as there are avian veterinarians. Any underlying medical problems should be identified and corrected. Various foul-tasting substances are frequently applied to the feathers in an unsuccessful attempt to modify the picking behavior. This procedure only masks clinical signs and should not be considered therapeutic. Treatment for feather picking should include the correction of organopathies, specific therapies for folliculitis (bacterial or fungal), improving the diet, removing exposure to cigarette smoke, providing frequent exposure to fresh air and sunlight, providing an 8- to 14-hour photoperiod that varies naturally with the seasons, and behavioral modification (see Chapter 4). If these therapies are determined to be ineffective over a two-month period, then mood-altering drugs may be necessary.

Where feather picking is determined to be psychological (a failure in the ability to diagnose a cause for the problem), a video recorder may be helpful in documenting a bird's behavior in its normal environment. Identifying the specific factors that induce the feather-picking behavior (separation anxiety, a tormenting pet, an unliked child, an abusive adult) can guide the clinician in making specific recommendations to correct the behavior and resolve the problem (see Chapter 4). Striving to improve the human-animal bond may be the most effective therapy in these cases.

Some problems with separation anxiety can be corrected by leaving tape recordings of family activities or a radio or TV playing in the family's absence. With some birds, the addition of new toys or moving an enclosure to a different location will be a stress factor that induces feather-picking, while with other birds these moves are positive and help to keep a bird mentally stimulated. A bird that is properly socialized and adapted early in life to changes in daily routine is less likely to develop emotional problems due to separation anxiety when changes occur later in life.

If psychological feather picking cannot be stopped with behavior modification, drugs may be necessary. Mood-altering drugs that have been suggested for use in feather-picking birds include tricyclic antidepressants and antihistamines (hydroxyzine hydrochloride,^c 2 mg/kg oral). These therapeutic agents are frequently discussed but are rarely effective. Hormonal therapies including thyroxine, testosterone and medroxyprogesterone have also been suggested for some cases of feather picking; however, all of these agents have undesirable side-effects and should be used only to treat specifically identified problems.²¹ Medroxyprogesterone acetate may be effective in stopping some sexually related behavioral disorders including feather picking, aggressiveness and masturbation; however, the drug can have severe side-effects including obesity, polydipsia, polyuria, glucosuria and liver disease.⁵¹

Ongoing studies suggest that haloperidol^d may be effective in some feather-picking cases.³¹ This drug is used to control hyperactive and impulsive behavior in humans. The dose being used in cockatoos is 0.08 mg/kg orally SID. It takes two days to stabilize the dose. Side-effects include loss of appetite, incoordination and vomiting. If there are no side-effects and a bird is still picking, the dose can be increased in 0.01 ml increments every two days. The maximum dose should not exceed two times the initial dose.

Successful treatment is generally reported within two to three days when the bird stops over-preening or self-mutilating and begins to play, sing and interact with the client.³¹ There is also a haloperidol decanoate (50; 100 mg/ml) injectable repositol for IM administration. Dosed at 1-2 mg/kg, the patients respond for up to 14 to 21 days. Both administration forms have to be used continually unless the initiating cause of the feather picking can be corrected. Clinical experience suggests that Moluccan and Umbrella Cockatoos, Quaker Parakeets and African Grey Parrots may respond to a lower dose (half that used for other birds).

Feather damage can be prevented by beak trimming or, as a last resort, by applying restrictive collars (Figure 24.23). These procedures only suppress the clinical signs and do not address the underlying problem.

■ Endocrine-related Feather Disorders

In poultry, hypothyroidism causes black, brown and yellow feathers to become red, longer and more pointed and to have less pennaceous barbules than

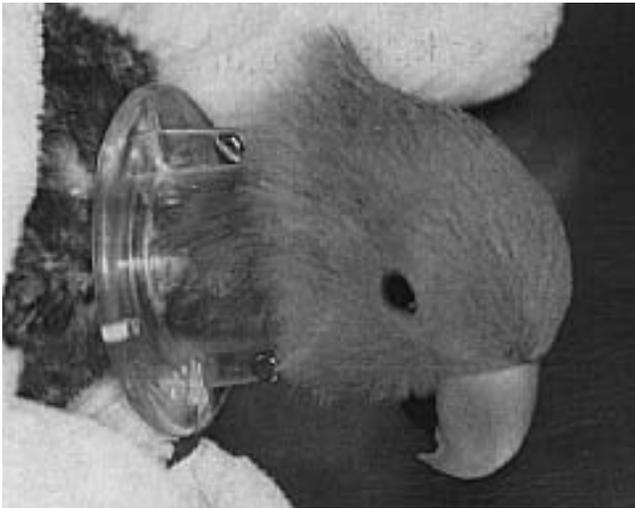


FIG 24.23 Collars should be used only in cases of severe self-mutilation that cannot be diagnosed and resolved with specific therapy (courtesy of Louise Bauck).

normal. The feather vanes have a fringed or lacy appearance. Hypothyroidism has been shown to delay wound healing in pigeons.⁵³ Documented cases of hypothyroidism in companion birds are rare. It should be noted that some species of birds that are deficient in iodine will have a TSH response test that suggests hypothyroidism (see Chapter 23).

In a Scarlet Macaw with reported hypothyroidism, clinical signs included nonpruritic feather loss, mild nonregenerative anemia, mild leukocytosis and heterophilia, hypercholesterolemia, sparse feathers, a history of no molting for over a year, and obesity with fat deposits on the lower abdomen and under the skin of the legs. Although no feather measurements were obtained, photographs of this bird suggest that contour feathers lacked width and were shorter than normal. This bird responded favorably to treatment with 0.02 mg/kg (20 µg/kg) L-thyroxine orally BID.⁴⁰

There are no documented cases of feather abnormalities resulting from hyperadrenocorticism or hypoadrenocorticism in birds although both conditions would be expected to occur. Hyperkeratotic dermatitis and feather loss were reported in a macaw that had histologic evidence of adrenal gland degeneration. This bird was on a poor diet and had staphylococcal abscesses of the occipital bone, bacteremia and bilateral pododermatitis.⁴¹ It is likely that the adrenal gland degeneration in this bird was secondary to other medical problems and was not the primary cause of the noted lesions.

Hyperestrogenism is associated with pruritic hair loss in mammals. Hyperestrogenism has been associated with proliferation of endosteal bone in birds, but has not been associated with feather lesions (see Chapter 23). Up to 60% of the male canaries in some flocks may develop baldness that is responsive to a change in the level of nutrition provided.

Inactive Feather Follicles

A feather follicle is normally inactive between molts. Persistent generalized inactivity of the feather follicles should be considered abnormal. In one study, many birds with inactive follicles had abnormal bacterial populations, elevated CPK activity and toxic heterophils. Some birds had a leukocytosis and elevated calcium levels; a few of these cases responded to antibacterial therapy. Epidermal atrophy accompanied chronic inactive feather follicles, hyperkeratosis and follicular atrophy in some birds.⁵⁵

Cysts

Cutaneous cysts are characterized by an epithelial wall surrounding keratinaceous contents. Epidermal cysts have been described in the dermis and subcutis of budgerigars (Color 24.6) If the orifice of the feather follicle is occluded from a traumatic or infectious episode, keratinaceous debris will accumulate in the follicle resulting in a follicular cyst (Color 24.9). These lesions are particularly common in canaries. Therapy is excisional (see Chapters 41 and 43). Feather cysts have been reported to occur in free-ranging birds.¹¹

Polyfolliculitis

Pruritic polyfolliculitis and dermatitis that may be caused by a virus have been described in lovebirds and budgerigars. Lesions appear to be particularly common in the feather tracts of the tail and dorsal region of the neck. The newly emerging feathers have short, stout quills with retained sheaths. Some of these birds have been histologically diagnosed with PBFV virus infections, whereas others have not been shown to be infected (Figure 24.24).

Histologically, polyfolliculitis is characterized by the appearance of multiple feather shafts from the same follicle with a thin layer of epidermis separating the shafts. Chronic inflammation occurs beneath the pulp cap, and the feather sheath is thickened. In some cases, large, keratin-filled cysts may also be

noted. Therapy with broad-spectrum antibiotics and corticosteroids is palliative at best.⁵⁰

Other Feather Abnormalities

Bleeding occurs if the protective keratin sheath of a developing feather (pin or blood feather) is injured or the feather is dislodged from the follicle. Hemorrhage can be severe, particularly in birds with coagulopathies. Experimentally, developing feathers that are removed can be rotated and reinserted and will continue to grow. In the clinical setting, it is best to remove damaged pin feathers (see Chapter 15).

Neonates kept in areas with low humidity may have dystrophic feather growth characterized by failure of the developing feather to exsheath. The lesions will usually resolve when the humidity is increased (and the affected feathers are removed).

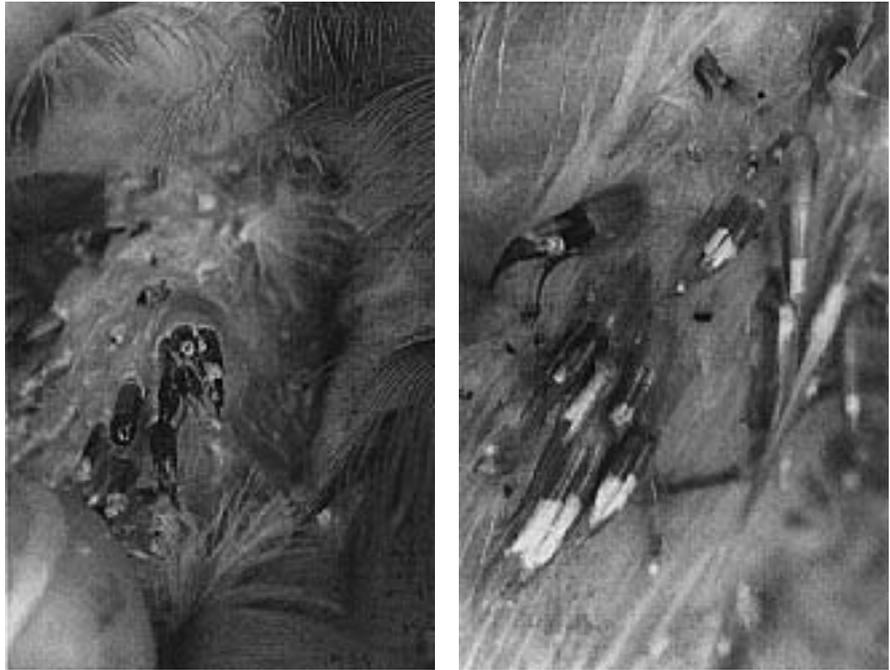


FIG 24.24 Clinical appearance of polyfolliculitis in two lovebirds. Both birds had progressive syndromes that could not be resolved. An etiologic agent could not be detected by histopathology in either case.

Products Mentioned in the Text

- a. Panalog, Solvay Animal Health, Mendota Heights, MN
- b. Silvadene, Marion Laboratories, Kansas City, MO
- c. Atarax, Roerig, Pfizer Pharmaceuticals, New York, NY
- d. Haloperidol, Schein Pharmaceuticals, Port Washington, NY

References and Suggested Reading

1. André J-P: "Les maladies des oiseaux de cages et de volières." Point Vétérinaire, Maisons-Alfort, 1990.
2. Arnall L, Keymer IF: Bird Diseases. London, Baillière Tindall, 1975, p 459.
3. Altman RB: Conditions affecting the integumentary system. In Petrak ML (ed): Diseases of Cage and Aviary Birds. Philadelphia, Lea & Febiger, 1982.
4. Berners, DJ: "The Boke of St. Albans," 1486.
5. Cheryl Y, et al: Fasting in king penguins: Hormonal and metabolic changes during molt. Am J Physiol 252:178-184, 1988.
6. Cooper JE: Introduction to birds. In Poole TB (ed): The UFAW Handbook on the Care and Management of Laboratory Animals 6th ed. Harlow, UK, Longman, 1987.
7. Cooper JE: Caged and wild birds. In Anderson RS, Edney ATB (eds): Practical Animal Handling, Oxford, Pergamon Press, 1991.
8. Cooper JE: Veterinary Aspects of Captive Birds of Prey. Glos, Standfast Press, 1978.
9. Cooper JE, Gschmeissner S, Ion F: The laboratory investigation of feathers. Proc 2nd Europ Symp Avian Med, Utrecht, Netherlands, 1989.
10. Cooper JE, Mellau LSB: Sticktight fleas (*Echinodnophaga gallinacea*) on birds. Vet Rec 130:108, 1992.
11. Couxillon CE, Maslin WA, Montgomery RM: Multiple feather follicle cysts in a wild turkey. J Wildl Dis 26:122-124, 1990.
12. Davidson WR, et al: Feather damage due to mycotic infections in wild turkeys. J Wildl Dis 25(4):534-539, 1989.
13. Dorrestein GM, Van der Hage MH: Veterinary problems in mynah birds. Proc Assoc Avian Vet, 1988, pp 263-274.
14. Drew ML, Ramsey E: Dermatitis with *Mycobacterium* spp. in a blue-fronted Amazon parrot. Proc Assoc Avian Vet, 1991, pp 252-254.
15. Gaskin JM: Psittacine viral diseases: A perspective. J Zoo Wildl Med 20(3):249-264, 1989.
16. Gentile MJ, Hunter LN: Physiological and behavioural responses associated with feather removal in *Gallus gallus var domesticus*. Res Vet Sci 50:95-101, 1990.
17. Ginn HB, Melville DS: Moulting in Birds. British Trust for Ornithology, Tring, England, 1983.
18. Graham DL: The avian integument. Its structure and selected diseases. Proc Assoc Avian Vet, 1985, pp 33-52.
19. Harrison GJ: Feather disorders. Vet Clin No Am Sm Anim Prac 14(2):179-199, 1984.
20. Hier RH, Perry PS, Sperry M: Foot tumor found on juvenile common loon. The Loon 58:41-42, 1986.
21. Hillyer EV, Quesenberry KE, Baer K: Basic avian dermatology. Proc Assoc Avian Vet 1989, 101-121.
22. Hochleithner M: African grey parrot responds to a change in diet. J Assoc Avian Vet 6:146, 1992.
23. Hubalek Z: The distribution patterns of fungi in free-living birds. Acta scientiarum naturalium Academiae scientiarum bohemoslovaca 8:1-51, 1974.
24. Johnson AL: Reproduction in the female. In Sturkie PD (ed): Avian Physiology, 4th ed. New York, Springer-Verlag, 1986, pp 403-431.
25. Johnson BJ, Castro AE: Canary pox causing high mortality in an aviary. J Am Vet Med Assoc 189:1345-1347, 1986.
26. Junge E, MacCoy DM: Amikacin therapy for *Pseudomonas cellulitis* in an Amazon parrot. J Am Vet Med Assoc 187:417-418, 1985.
27. Keymer IF: Mycoses. In Petrak ML (ed): Diseases of Cage and Aviary Birds 2nd ed. Philadelphia, Lea and Febiger, 1982, pp 599-605.
28. King AS, McLelland J: Form and Function in Birds Vol 3. Orlando, Academic Press, 1985.
29. Kray RA: Dermatitis and feather discoloration syndromes in psittacines. Proc Am Assoc Zoo Vet, 1985, p 12.
30. Kuttin ES, et al: Chicken dermatitis and loss of feathers from *Candida albicans*. Avian Dis 20:216-218, 1976.
31. Lennox A, Van Der Heyden N: Haloperidol for use in treatment of psittacine self-mutilation and feather picking. Proc Assoc Avian Vet, 1993.
32. Lucas AM, Stettenheim PR: Avian Anatomy, Integument. Agricultural Handbook 362. US Department of Agriculture, Washington DC, 1972.
33. Ludicke M: Wachstum und Abnutzung des Vogel Schnabels. (Growth and wear of the bird's beak). Zool Jahrb 57:465-533, 1933.
34. MacCoy DM, Campbell TW: Excision of impacted and ruptured uropygial glands in three gentoo penguins (*Pygoscelis papua*). Proc Am Assoc Zoo Vet, 1991, pp 259-260.
- 34a. Mancuso S: African "red" parrots? Bird Talk Dec:85-87, 1990.
35. Marder J, Arieli Y, Ben-Asher J: Defense strategies against environmental heat stress in birds. Israel J Zool 36:61-75, 1989.
36. McOrist S: Some diseases of free-living Australian birds. In Cooper JE (ed): Disease and Threatened Birds. Cambridge, England, Intl Coun Bird Pres, 1989.
37. Menon GK, et al: Fine structural basis of the cutaneous water barrier in nestling Zebra finches (*Poephila guttata*). Ibis 130:505-511, 1988.
- 37a. Millam J, Finney H: Leuprolide acetate can reversibly prevent egg laying in cockatiels. Proc Assoc Avian Vet, 1993.

38. **Muller M, Cooper JE:** Avian dermatology. In von Tscharnner C, Halliwell REW (eds): *Advances in Veterinary Dermatology Vol I*. London, Baillière Tindall, 1990.
39. **Needham JR, Cooper JE, Kenward RE:** A survey of the bacterial flora of the feet of free-living goshawks (*Accipiter gentilis*). *Avian Pathol* 8:285-288, 1979.
40. **Oglesbee BL:** Hypothyroidism in a scarlet macaw. *J Am Vet Med Assoc* 201:1599-1601, 1992.
41. **Onderka N, et al:** Adrenal degeneration associated with feather loss in a macaw. *Cand Vet J* 28:193-194, 1987.
42. **Oppenheimer J:** Feather picking: Systematic approach. *Proc Assoc Avian Vet*, 1991, pp 314.
43. **Palmer RS:** Mechanisms and control of molt. In Farner DS, King JR (eds): *Avian Biology Vol 2*. New York and London, Academic Press, 1972.
44. **Pass DA:** The pathology of the avian integument: A review. *Avian Pathol* 18:1-72, 1989.
45. **Pass DA, Perry RA:** Granulomatous dermatitis in peach-faced lovebirds. *Aust Vet J* 64(9):285-287, 1987.
46. **Payne RB:** Mechanisms and control of molt. In Farner DS, King JR (eds): *Avian Biology Vol 2*. New York and London, Academic Press, 1972.
47. **Perek M:** Ergot and ergot-like fungi as the cause of vesicular dermatitis in chickens. *J Am Vet Med Assoc* 132:529-533, 1958.
48. **Perelman B, Gur-Lavie A, Samberg Y:** Pox in ostriches. *Avian Pathol* 17:735-739, 1988.
49. **Perry RA:** A psittacine combined beak and feather disease syndrome. *Proc Post Graduate Comm Vet Sci, Cage and Aviary Birds, Sydney, Australia*, 1972.
50. **Perry RA:** Pruritic polyfolliculitis and dermatitis in budgerigars (*Melopsittacus undulatus*) and African lovebirds (*Agapornis* spp.). *Proc Assoc Avian Vet*, 1991, pp 32-37.
51. **Perry RA, Gill J, Cross GM:** Disorders of the avian integument. *Vet Clin No Am Sm An Pract* 21(6):1307-1327, 1991.
52. **Phalen DN, Wilson VG, Graham D L:** Epidemiology and diagnosis of avian polyomavirus infection. *Proc Assoc Avian Vet*, 1991, 27-31.
53. **Pilo B, Verma RJ, Shah RV:** Alterations in the lipid content during cutaneous wound healing in hypothyroidic pigeons. *Pavo* 25:9-12, 1987.
54. **Purton MD:** Skin surface topography in the domestic fowl and Japanese quail. *Brit Vet J* 142:446-452, 1986.
55. **Reavill DR, Schmidt RE, Fudge AM:** Avian skin and feather disorders: A retrospective study. *Proc Assoc Avian Vet*, 1990, pp 248-255.
56. **Ritchie BW, et al:** A review of psittacine beak and feather disease. *J Assoc Avian Vet* 3(3):143-149, 1989.
57. **Ritchie BW, et al:** Advances in understanding the PBFV virus. *Proc Assoc Avian Vet* 1990, pp 12-24.
58. **Ritchie BW, et al:** PBFV virus: Disease prevention through experimental vaccination. *Proc Assoc Avian Vet*, 1991, pp 50-56.
59. **Roy NA, Threlfall W, Wheeler TA:** Feather loss of unknown etiology in a gull colony in Newfoundland. *Can J Wildl Dis* 22(4):591-594, 1986.
60. **Schmidt RE:** Avian skin diseases: A pathologist's perspective. *Proc Assoc Avian Vet* 1987, pp 117-124.
61. **Spearman RIC:** Physiology and Biochemistry of the Domestic Fowl. London, Academic Press, 1971, pp 604-618.
62. **Spearman RIC, Hardy JA:** Integument. In King AS, McLelland J (eds): *Form and Function of Birds Vol. 3*. London, Academic Press, 1985.
63. **Scott ML, Austic R, Gries C:** Nutritional deficiency diseases. In Hofstad M, et al (eds): *Diseases of Poultry* 7th ed. Ames, Iowa State University Press, 1978.
64. **Swain SF:** New concepts in wound management. *Tijdschrift voor Diergeneeskunde* 112:56-58, 1987.
65. **Tollefson CI:** Nutrition. In Petrak ML (ed): *Diseases of Cage and Aviary Birds*. Philadelphia, Lea and Febiger, 1982, pp 361-367.
66. **Tudor DC:** Mycotic infections of feathers as a cause of feather pulling in pigeons and psittacine birds. *Vet Med Sm Anim Clin* 78:249-253, 1983.
67. **Voitkevich AA:** The Feathers and Plumage of Birds. London, Sidgwick and Jackson, 1966.
68. **Wilds C:** The terminology of plumage and molt. *Birding*: 148-154, 1989.
69. **Wertz PW, Downing DT:** Glycolipids in mammalian epidermis: structure and function in the water barrier. *Science* 217:1261-1262, 1982.
70. **Wojcinski ZW, et al:** Cutaneous herpesvirus infection in a mallard duck (*Anas platyrhynchos*). *J Wildl Dis* 27:129-134, 1991.