Unique, Clinically Significant Features of Avian Dermatological Disorders

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Among the most common presenting clinical complaints in the avian veterinarian's daily experience are those related directly or indirectly to the integument.

The avian integument differs in several significant morphological and functional aspects from that of mammals. The epidermis and dermis are, over most of the body, considerably thinner and less substantive than those of mammals. Except for the uropygial (preen) gland, the skin of birds is aglandular. Feathers, considerably more structurally complex than hair, are the product of the feather follicles. The follicles occur in feather tracts (pterylae), leaving significant areas of skin glabrous (apterylae). Scaled skin covers portions of the lower leg and foot in many species. The tough, heavily and densely keratinised *stratum corneum* of the upper and lower beak - the rhinotheca and gnathotheca, respectively - finds no functional mammalian counterpart, and structural counterparts only in claws, fingernails, and hooves. Thus, free extrapolation from the commonly accepted principles and tenets of mammalian dermatology is risky, and assumptions and expectations derived therefrom are often erroneous.

Skin neoplasms, topical hypersensitivities, and generalized dermatitides are far less frequently encountered in avian patients than in mammals. Other than inflammation of the skin integral to or in immediate association with the feather follicle, dermatitis *per se* is a relatively infrequent clinical problem in birds.

The most frequently presented dermatological diagnostic challenges are, by far, those manifested by a variety of structural and pigmentary abnormalities of the plumage. Abnormal plumage is a common complaint registered by the pet bird owner and aviculturist. Plumage abnormalities may be the result of local trauma to the follicle, excessive wear or mechanical disruption of the structure of disclosed feathers, pigmentation abnormalities, or dysplasia of the feather's structural components.

Domestic mammals are subject to a spectrum of skin disorders that are of unusual or rare occurrence in their wild-type progenitors. This suggests that selective breeding has wrought more genetic departure from the original genotype than was intended by the

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unnatural selections imposed to merely enhance productivity and show-standard phenotypes. The relative paucity of dermatopathies to which caged birds are genetically predisposed when compared with domestic mammal breeds is probably accounted for to a significant degree by closer propinquity of caged birds to their wild-type ancestors.

For example, the long-domesticated and intensively and selectively bred domestic fowl (*Gallus domesticus*) does exhibit many genetically determined plumage abnormalities such as frizzle feather, silky feather, leg feathering, continual pterylogenesis in certain pterylae, and many color mutations. It should also be noted that the canary (*Serinus canarius*), the caged bird with the longest history of domestication and subjection to unnatural breeding selection, is that species exhibiting the greatest incidence and variety of genetically determined feather abnormalities. To wit: feather cysts (Yorkshire and crested breeds), coronal crests (Norwich, Crests), frilled feathers (Dutch and Parisian Frills), and tract apterylosis (Gibber Italicus) as well as many color mutations.

Pterylogenesis/Feather Formation and Development:

An understanding of the basic process of pterylogenesis is the foundation upon which rational diagnosis, treatment, and prognosis of plumage abnormalities can be based.

In the depths of the follicle, where the follicle-lining epidermis reflects onto the fibrovascular protrusion of the dermis (which becomes the feather pulp), is an annular thickening of pterylogenic tissue termed the epidermal collar. The germinative epithelium at the upper edge of the collar- the edge directed toward the follicle orifice - gives rise to the growth foci of the rachis, barbs, barbules, hooks, and hooklets. It is easier to visualize feather growth if we figuratively remove the collar from the depths of the follicle, cut the collar opposite the point of growth of the rachis (Fig.1), unroll it, and lay it flat (Fig.2.).

The rachis's growth point remains at the center of the upper edge of the open collar; those of the barbs are arrayed uniformly along the remainder of the upper edge of the collar. The growth points of a new pair of barbs first appear at the ends of the edge of the open collar; as each of the barbs increases in length its growth point moves progressively closer to the center of the collar. Note that the distal end of each barb remains equidistant from that level of the rachis that was forming at the same time the barb?s growth began (Fig. 2).

The growing feather is imbued with all its structural and pigmentary characteristics at the moment each of its components (rachis, barbs, barbules, hooks, and hooklets) is formed at the edge of the epidermal collar. Those portions of the feather?s components forming at a given moment are said to form *isochronously* (at the same time). Note that if an experimenter were to tattoo with black pigment the growth points of all structures being formed isochronously, the result would be a fine black line, a *segmental* exogenous pigmentary defect, across the mature feather, to be revealed when it was fully grown and disclosed (freed from the feather sheath and unfurled). Likewise, naturally deposited pigment is incorporated into the feather when it is formed at the collar's edge.

Pterylodysgenesis

Just as the characteristics of the normal mature feather were determined by the events at the edge of the epidermal collar, so the effect of any noxious influence or agent upon pterylogenesis will be exerted upon the activity and function of the edge of the collar. If the noxious effect is momentary or short-lived, the effect will be *segmental* on one or more of the feather components being formed during the brief time of the agent's influence (as was the effect of the tattoo mentioned above). A mild disturbance may cause slight to moderate dysplasia of hooklets, hooks, and barbules; a more severe one may result in segmental dysplasia or even agenesis of the barbs and (rarely) dysplasia of the rachis. If the offending agent affects all regions of the collar's edge, the effect would be observed in all structures formed isochronously with the effect of the agent.

The most commonly observed of such effects is the so-called "stress mark", "fret mark", or "hunger trace" of segmental barbule or barbule-and-barb dysplasia. Isochronous barbule and barb dysplasia can be expected to occur in all feathers growing at the moment of such stressful events as: episodes of hunger, temporary chilling or heat stress, acute infectious disease, rough handling or capture, and acute trauma. The effect is particularly pervasive in nestlings engaged in the daunting task of growing all their feathers at once (the only time in life that they will be required to do so). A mature bird in molt exposed to similar stressors or to the administration of exogenous corticosteroids would be expected to develop segmental dysplasia only in those feathers growing at the time of the insult.

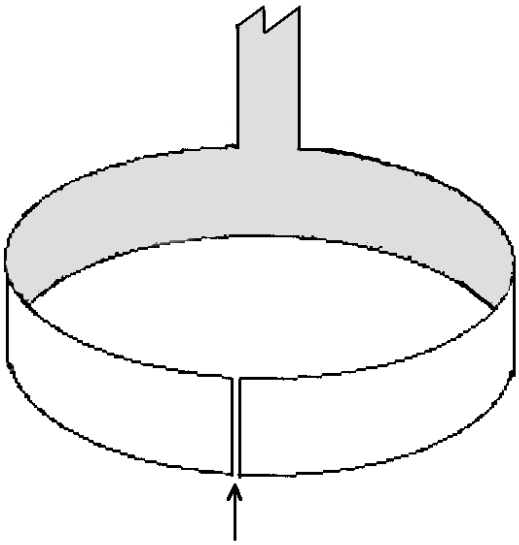

Figure 1. The epidermal collar has been removed from the base of the follicle and cut (arrow) opposite the growth site of the rachis.

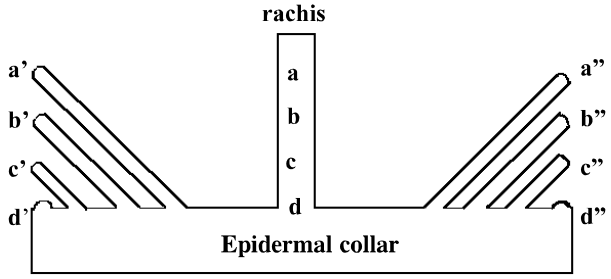

Figure 2. The epidermal collar has been "unbuttoned" and laid flat. Observe that the tips of barb pairs a'a", b'b", c'c" and d'd" remain at the same level and same horizontal distance from their isochronous growth points in the rachis (a, b, c, and d respectively) even as their own growth points move progressively closer to that of the rachis, with which they will ultimately fuse. The diagram has been considerably simplified to illustrate only barb and rachis growth; barbules, hooks, and hooklets are not shown.

Other possible causes of pterylodysplasia reported in the fowl include: pantothenic acid deficiency, niacin deficiency, selenium deficiency, and, in some raptors, iatrogenic hyperthyroidism. The last-mentioned occurs as a result of attempts to induce molt by administration of exogenous thyroid hormone.

Infectious agents such as parrot circovirus and parrot polyomavirus may not affect the entire edge of the collar uniformly; their effects thus tend to be irregularly local to diffuse, rather than discretely segmental. In addition, these virus infections may also cause chronic, non-destructive irritation of the outer layer of epithelium which surrounds the growing feather and forms the feather sheath. This results in the sheath becoming hyperkeratotic, thickened, and unable to flake away normally when the ensheathed feather is extruded from the follicle orifice.

Loss of Follicle function:

Local apterylogenesis is the result of loss of function of individual follicles. Severe septic pterylopulpitis may result in secondary folliculitis of sufficient severity that the follicle bud and epidermal collar are destroyed. Local trauma with intra- and perifollicular hemorrhage, sometimes inflicted by a cagemate but most often the result of exuberant automutilative behavior, can lead to post-traumatic scarring of the follicle bud and/or perifollicular fibrosis and cicatrization. Such loss of follicle function is irreversible.

Pigmentation abnormalities:

Feather colors and color patterns are determined by: 1) the deposition of pigments (or lack thereof) in feather components at the time of their genesis at the edge of the epidermal collar and 2) microstructural features of feather keratin which affect the optical characteristics of the barbs and barbules. Absence of pigment characterizes white feathers;

they reflect all wavelengths of incident visible light.

Feather pigments include melanins (black, some browns, and some yellow-tans), lipochromes/xanthines (yellow, orange, red), and porphyrins (some reds, browns, and, in turacos, green). Depending upon the species and specific feather considered, pigments are deposited in different feather structures and at different levels within the keratin. For example; the yellow of a sulphur-crested cockatoo?s crest feathers is a lipochrome deposited in the surface keratin of the barbules, but not in the barbs; the melanins in blue feathers are deposited in the deep keratin layers of the barbs and barbules.

Some feather colors (blues, purples, and most greens) occur in the absence of specific blue, purple, or green pigments. These colors, as well as iridescence displayed by some feathers, are so-called "structural" or "optical" colors and are the result of an interplay of pigments with the microstructure of feather keratin which imparts specific optical properties to the feather.

Iridescence is dependent upon specific surface microtopography which permits the phenomenon of *reflective interference* of light to occur, much as iridescence may be observed on the surface of a vinyl record or a compact disk as a result of the fine, regularly repeated, surface irregularities.

Blue feathers are dependent upon melanin deposited only in the deep layers of keratin. The light-absorbtive effects of the deep melanin and the optical effects of the structure of the overlying non-pigmented keratin combine to cause differential absorption and scattering of different wavelengths such that only the relatively short wavelengths of the blue portion of the visible spectrum leave the feather and are perceived as blue by an observer. Green feathers (other than those of turacos which have a true green pigment, *turacin*) are the result of "optical" blue in combination with pigmentary yellow.

The Myth of Post-pterylogenic Pigmentation ("Acquired Brown Spot"):

A common complaint is that blue or green feathers (of a hyacinth macaw, or an Amazon parrot, for example) begin to accumulate dark brown to black pigment some time after having emerged from the follicle as normal green feathers. This seems counter to the principle that the feather is incapable of becoming pigmented after its structural elements are formed by the epidermal collar! In fact, such a discolored feather, when viewed by *incident light*, does, indeed, appear to have dark, melanotic pigment in the dark regions. However, when viewed by *transmitted light* it becomes obvious that *all* regions that are or were originally green contain melanin.

The phenomenon of putative "late pigmentation" is the result of excessive wearing away of the surface keratin which exposes the indigenous, deeply residing melanin present in all blue and green feathers. Loss of the surface keratin and its optical properties results in loss of the "structural" component of blue and thus of the blue component of green. It is then incumbent upon the avian clinician to determine the reason for excessive wear of the

surface of the affected feathers. Excessive autopreening or allopreening, and frequent abrasion of feathers on the cage wires ("large-bird-small-cage syndrome") are common offenders. Excessive feather age with attendant unnaturally prolonged wear from autopreening may be a sequella of interruption or cessation of the normal molt cycle by the effect of inappropriate photoperiod or of hypothyroidism.

Feather achromia (lack of normal pigmentation), other than that which results from mutation, occurs in several species as a result of certain nutritional deficiencies. Some gallinaceous species (chicken, coturnix quail) develop feather achromia as a result of deficiencies of lysine, folic acid, and iron. We should be cautioned against interspecies extrapolation, however, because it has been shown that cockatiels fed a diet devoid of lysine exhibit normal pigmentation, but suffer achromia of feathers growing when being fed a diet deficient in either choline or riboflavin (deficiencies of neither of which cause achromia in the coturnix quail). *Caveat extrapolator!*

Automutilative Behavior:

The spectrum of behaviors that should be included under the rubric "automutilation" range from the least severe tendency to excessive autopreening, thru the more obvious acts of feather chewing and feather plucking, to the most severe compulsion to mutilate the skin and underlying tissues. Indeed, in some cases there is a progression of behaviors thru all these stages; in other cases the bird may enter the behavioral continuum at some intermediate stage. After having eliminated all the routinely treatable primary dermatological and ectoparasitic conditions, the clinician is forced to seriously consider that automutilation is a behavioral abnormality. The behavior may begin as a vice of boredom, or possibly as displacement behavior in response to environmental, social, (or asocial) stress.

A hallmark of automutilative damage is that the skin and plumage of the head and upper regions of the neck are unaffected. *The bird can't reach these areas even if it sits on a higher perch!* (yes, that was an attempt at humor; a joke, as it were, to see if you were still giving this disquisition the rapt attention it so richly deserves).

The owners of most automutilating birds will staunchly deny that they ever observe their bird exhibiting such behavior and are convinced that there must be some organic cause for the obvious plumage and/or skin lesions. They aren?t intentionally misleading the clinician or offering a fallacious history. In fact, that the bird does not exhibit automutilation when the owner is present is strong evidence that the behavior is a vice of boredom practiced when the target of desired social interaction - the owner - is absent. Were the owner to observe the bird undetected - as thru a one-way mirror or from a dark room adjoining the well-lit room where the bird is caged, for example - he would observe the behavior he was so convinced was a figment of his veterinarian?s imagination.

Excessive preening causes unkempt, rough or slightly tattered feathers, the slow development of which may escape the owner?s notice. If allowed to proceed unchecked,

overpreening often progresses to more severe forms of the automutilative vice. Many early-recognized cases of overpreening can be interrupted simply by providing the bird the opportunity to bathe once or twice daily.

When the behavior becomes more compulsive and results in feather mutilation or worse, merely spraying the bird or providing for bathing is rarely curative. The oft-recommended Elizabethan collar will, indeed, prevent mutilation of the plumage and the bird may well regain normal plumage with the next molt. However, if the underlying cause of the behavioral abnormality is not addressed, automutilation re-appears with removal of the collar.

Common circumstances under which the condition occurs include:

1. solitary bird,
2. owners absent during much of the day,
3. change of owner,
4. change of cage,
5. insufficient opportunity to bathe, and
6. lack of environmental stimuli ("sterile" cage)

Treatment of the abnormal behavior is aimed at behavioral modification thru manipulation of the bird?s physical and social environment. Such include:

1. change location of cage to an area of the house with more traffic,
2. provide regular (daily) bath or spraying,
3. provide a larger cage,
4. offer more handling by, and attention from owner(increase socialization),
5. provide replaceable soft wood perches or pieces of wood for chewing,
6. cage with or near another bird (give the automutilator a pet),
7. find a new home for the bird (some inveterate feather chewers drop the habit immediately upon entering a new household),
8. provide toys such as chain, rawhide, and/or other distracting objects of interest.

The “New York Times” gambit may be surprisingly successful. Newspaper is torn into strips, tightly crumpled, and used to fill the cage to the level of the perch. Food and water containers must be accessible at perch level. The level of crumpled paper must be maintained at perch height in order that the bird to may chew on paper as easily as upon itself. I have found that the *Houston Chronicle, Dallas Morning News,* and the *Chicago Tribune* also serve well and I suspect that the newsprint of the Brisbane, Sydney, Adelaide, and Perth dailies might likewise prove useful in this application.

Attempts to interrupt automutilative behavior with psychoactive agents and tranquilizers have met with inconsistent and unconvincing results because adequately controlled studies were not performed. Most such trials that were deemed successful were accompanied by one or more of the techniques of enriching the bird?s physical and social environment

concurrent with drug administration.

The client should be introduced to the concept that one should wonder not why some psittacine birds exhibit such behavior; rather wonder why more do not! Parrots are highly social and gregarious species dependent for maintenance of normal behavior upon continuous or frequent socialization with conspecifics - or with individuals of other species upon which they may target their social tendencies or requirements. Solitary confinement as an isolated pet, or deprivation of adequate opportunity for normal socialization, should be expected to elicit behavioral abnormalities. The imposition of social deprivation in an unstimulating environment - "solitary confinement" - of members of an equally social and gregarious species (*Homo sapiens*) is coming to be viewed a "cruel and unusual punishment"; why should it be considered otherwise for psittacine birds ?

Recommended Reading on the Avian Integument:

Hodges, R.D. The Histology of the Fowl. Acad. Press. London. 1974.

King, A.S.. Aves Respiratory System. In: Getty, R. (ed.) The Anatomy of Domestic Animals. Vol.2. 5th ed. Saunders, Philadelphia. 1975.

Lucas, A.M. and P.R. Stettenheim. Avian Anatomy-Integument, Parts I and II, U.S. Govt. Printing Office, Wash. D.C. 1972

Nickel, R., A. Schummer, and E. Seiferle. Anatomy of the Domestic Birds. Translated by Siller, W.G. and P.A.L. Wight. Springer-Verlag, New York. 1977