



A cockatoo owned by one of the authors that has been "stable" with respect to this condition for the last two to three years.



A less advanced case. Breast feathers still present but body feathers exhibiting typical changes.



Advanced case of Psittacine Beak and Feather Syndrome. Note abnormal feathering and deformed beak.

the Vet's Corner

by
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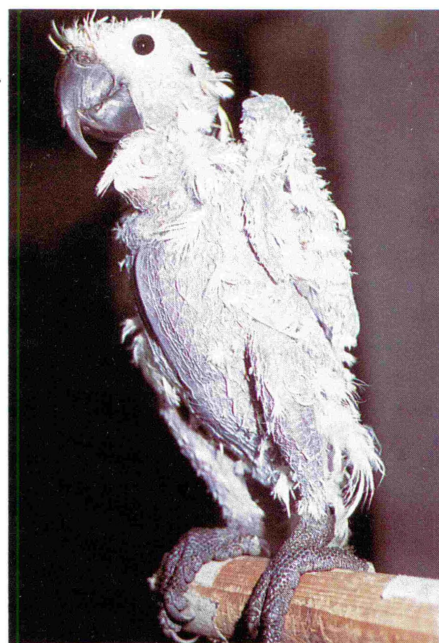
Cockatoo Feather Loss Syndrome: An Update

For the last 15 plus years, in clinical practice, we have seen countless pitiful cockatoos with mild to severe feather (and sometimes beak) abnormalities, presented by heartbroken owners for diagnosis and treatment. Because these birds and their medical affliction is so common to us we have had a tendency to assume, over the years, that most lay people have equal familiarity with this condition. Clearly such is not the case, as evidenced by the continued large number of bewildered clients we see with these birds and more recently, by correspondence with a distraught cockatoo owner in New York who had sought the help of several veterinarians and the advice of several aviculturalists regarding this problem with no resultant satisfaction. It is for these reasons that we elected to write this article.

The Cockatoo Feather Loss Syndrome has several other symptoms. It has been called "Cockatoo Rot," "Beak Rot," "Cockatoo Endocrine Syndrome," "Psittacine Beak and Feather Disease Syndrome (PBFDS)," "White Cockatoo Disease," "Cockatoo Feather Loss and Malformation Syndrome," "Feather Maturation Syndrome," "Cockatoo Apterylosis," and "Feather and/or Rhamphothecal and Ungual Keratodysgenesis."

Formerly, these birds were usually

Photos by authors

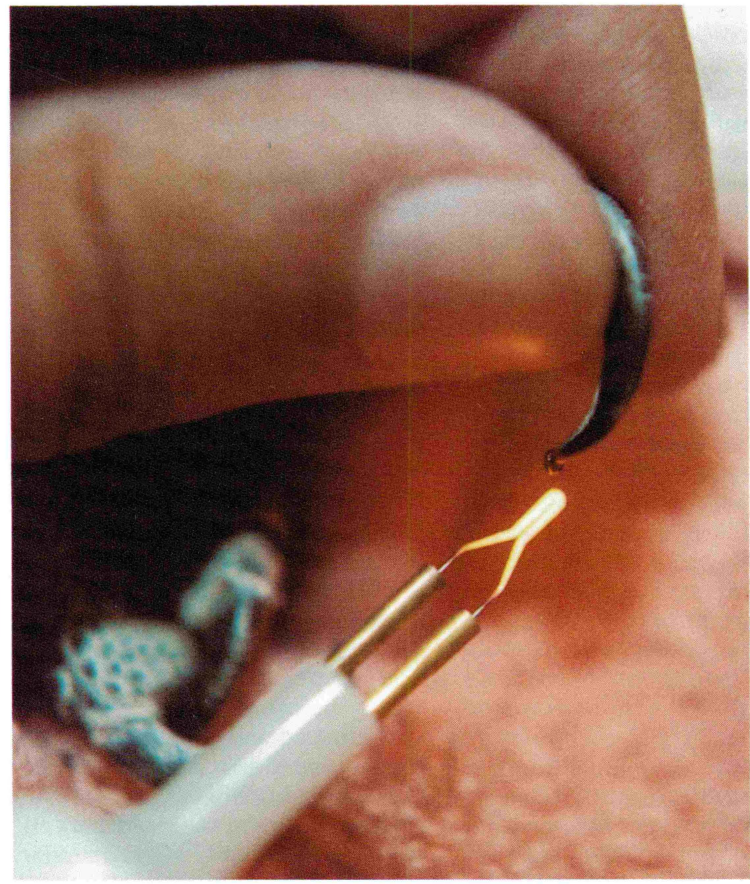


A very advanced case. Note nearly naked condition and the overgrown beak.

"foisted onto" unsuspecting bird buyers by unscrupulous or simply ignorant sellers who usually told the prospective buyer that the bird had been "caught in tar" and that the feathers "would surely grow back," or that the bird was suffering from a "dietary deficiency" which would "straighten out" with improved husbandry, or that the bird was undergoing a "severe moult." These birds would instead exhibit a chronic, progressive deterioration of beak and feathers and most would eventually succumb to a variety of diseases.

We began an intensive study of this condition about seven years ago which has included post-mortem examinations (autopsies) and clinical pathology (blood, etc.) work ups. We have not been aware of any large number of these birds that has lived more than four years, but we do know of two that have remained stable beyond this period of time. After performing over 50 autopsies, we found an extraordinarily high incidence of adrenal gland lesions with evidence of Pacheco's Disease or other causes of Inclusion Body Hepatitis in the tissues. It now appears that these lesions may have developed later in the course of the disease and were apparently secondary to the initial disease process. One of these cases was pub-

Continued on page 26



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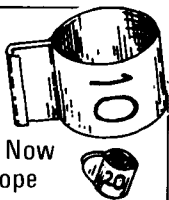
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lished in the Journal of The American Veterinary Medical Association.¹ It is equally likely that some of the inclusion bodies noted were not associated with any disease but, instead, resulted from the storage of metabolically important materials.

Drs. D.A. Pass and R.A. Perry, Australian veterinarians practicing in New South Wales, have recently studied the disease as it occurs in the wild.² They have diagnosed the condition in sulfur-crested cockatoos, Major Mitchell's cockatoos, little corellas, galahs (rose-breasted cockatoos), peach-faced lovebirds, Nyassa lovebirds, hooded parrots, budgerigars, mallee ring-necked parakeets, Port Lincoln parrots, red-rumped grass parakeets, lorikeets, and western rosellas. Veterinarians in the United States have seen the disease primarily in the umbrella cockatoo, moluccan cockatoo, red-vented cockatoo, Goffin's cockatoo, and in the sulfur-crested cockatoos. Drs. Pass and Perry have seen the disease in predominantly young birds and often recognize it with the arrival of the first true feathers after the loss of their down feathers. They say that occasional cases do occur in adult birds that have not previously been noticed as having abnormal feathers. Local Australian trappers have told Dr. Pass that up to 20% of wild sulfur-crested cockatoos may be affected in some flocks. The sulfur-crested cockatoos seem to be the most commonly affected wild hosts.

Abnormal feathering (most notably incomplete maturation of new feathers) occurs in all species but changes in the beak are the most prominent in the white cockatoos and may not be as noticeable in many of the other species. We have seen severe beak changes in most of the cockatoo species we have observed in practice.

Affected birds lose down and contour feathers over most areas of the body. Drs. Pass and Perry described feather loss as roughly symmetrical with the normal plumage replaced progressively by abnormal feathers exhibiting one or more of the following characteristics: retained feather sheaths, blood retained within the shaft of the feathers, short clubbed feathers, curled and deformed feathers, stress or fault lines involving the feather vanes, and newly emerging feathers with a circumferential constriction at the base of the soft quill. This constriction of the soft feather base is what we most commonly notice in our practice and is what the client may describe as "feathers

pinching off half way and breaking off." Lesions also occur in primary and secondary flight feathers, and major tail feathers but these may not be evident until the bird has lost many contour feathers. Essential crest feathers of sulfur-crested cockatoos are often the first to be lost.²

Drs. Pass and Perry have observed that lesions involving the beaks of wild birds tend to start as a change in color from "gray to dark semi-gloss black." This is followed by progressive elongation of the upper and lower beaks, development of transverse fracture lines across the top beak and longitudinal splits in the upper and lower beaks. Uneven wear, chips, and fractures impair eating. The upper beak may become progressively undermined and chronically inflamed leading to constant tongue action and nibbling. The surface begins to bleed easily and has a tendency to become infected. Eventually these birds are unable to crack and eat seed and require a diet of soft foods. Some birds undergo toenail changes, but this isn't as common a finding. The lesions occurring in the feathers, beak, and nails are similar.

The major lesions of the Feather Loss Syndrome occur in the developing feather shaft. Investigators have found cells with cytoplasmic inclusions within the pulp and epidermal layers.^{2,3} Death of cells of the distal pulp and lining cells and bleeding into the distal shaft occurs, effectively choking off the developing feathers.

The exact cause of these changes is not yet known, but the presence of specific types of inflammatory cells and the presence of the aforementioned inclusion bodies strongly suggest the disease is infectious and probably viral in origin. Dr. Linda Lowenstine of the University of California at Davis feels the disease is linked to a parvovirus (not the same one as in dogs or cats).⁴ Dr. Elliot Jacobson, of the University of Florida, is also involved in investigating this intriguing but devastating disease. He has found "intracytoplasmic crystalline arrays" composed of viral particles in the feather follicles of affected Moluccan and triton cockatoos. Attempts are being made to isolate this virus in tissue culture. He warns us that the virus he is seeing may not be the primary cause of the disease and that isolation of a virus in tissue culture and transmission studies would be necessary to confirm a cause and effect relationship.⁵

During our last conversation with Dr. Jacobson, one of the world's foremost

viral researchers, it was learned that he feels strongly that the disease may indeed be viral and may be a very complex problem. He speculates that the causal agent may infect these birds at a young age which results in the deterioration of their immune systems, producing an "AIDS"-like effect with secondary problems. Dr. Pass has found that the thymus and the bursa of Fabricius (tissue responsible for immune functions in birds) undergo necrosis (death of cells) and extreme atrophy in some birds. This also suggests that the immunological functions of the bird's body may be compromised. This would explain why so many of these birds die of bacterial septicemias⁶ and herpes virus infections.^{1,7}

SUMMARY

To summarize the findings on Cockatoo Feather Loss Syndrome, we will list what we know about the disease and what we do not know.

1. The disease appears to be progressive from a very young age.
2. Feather loss and beak deterioration is chronic and progressive (with rare exceptions).
3. Affected birds usually die at a young age from secondary problems which appear to result from lowered resistance: bacterial septicemia, yeast infections, Aspergillosis, Pacheco's Herpes Virus Disease, etc.
4. The disease is most likely caused by an infectious agent, probably a virus.
5. We do not know whether or not this disease is contagious and, if it is, for how long, and how the disease is transmitted.
6. The disease is *not* caused by primary endocrine (hormonal) disturbances. All recent thyroid and adrenal function tests on affected birds have shown normal endocrine function,^{8,9,10} but adrenal gland compromise often occurs prior to death because microscopic lesions are often seen in the adrenal glands.⁷ This is another problem due probably to the action of the herpes virus on the adrenal glands and the septic nature of Psittacines under stress.^{6,7}
7. This disease is not caused by nutritional problems, capture techniques (i.e. tar), moulting, toxicological factors, and behavioral abnormalities such as feather picking.
8. We do know that the disease is

complex. It may be that even if the progress of the disease can be arrested, the damage is more than likely irreparable.

9. A cockatoo (or other species) that has this condition can be expected to progressively lose feathers and feather condition, despite any supportive care, until death eventually results before three or four years of age due to secondary disease complications.
10. All that can be done for these birds is to support them with good diet, exemplary husbandry, and whatever treatment a laboratory work up may indicate (such as immune stimulation, elimination of gram negative pathogenic bacteria, adrenal gland support therapy if indicated by ACTH response tests later in the course of the disease, etc.).

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