A Diagnostic Approach to Avian Self-Mutilation Syndromes

Ross A Perry*

Avian Self-Mutilation Syndromes (ASMSs) - the last "s" is to remind us that there are many Avian Self-Mutilation Syndromes - for the purposes of this paper, are characterised by the bird inflicting damage to its own integument (feathers, skin, claws) and/or deeper tissues (subcutaneous tissues, muscle, tendons, ligaments and bones, for example) by use of its beak. (Note: ASMSs associated with damage inflicted by the bird's feet and claws occurs occasionally but in practice would be considered rare and likely to be associated with underlying disease affecting the head region of the bird.)

ASMSs may be characterised by one or a combination of behavioural patterns: "stripping", "barbering", plucking or biting or nibbling feathers, biting or nibbling skin and/or deeper tissues. "Feather picking" is a commonly used term which means any form of self-mutilation behaviour directed towards the feathers.

Whereas ASMSs are not confined to psittacine birds, it is with these birds that we have most clinical experience, and so in this paper there is a bias towards them. The paper is also written with a bias from a veterinary surgeon similarly trained to the primary audience of veterinary surgeons with a traditional Western philosophy towards health and disease. However some mention of other philosophies and diagnostic approaches is included.

The importance and significance of ASMSs

The effects of ASMSs may be perceived by us as purely an aesthetic problem. However the affected bird may be inadequately insulated and secondary bacterial infections may occur. In addition, the chronic feather loss and replacement, coupled with blood loss from broken or bitten pin feathers, can compromise the bird's health (GMC p31). ASMSs affecting the patagial membrane and/or the axillary region can be associated with a vicious circle of pain further self-mutilation and restriction of movement from direct damage and from contracting scar tissue. ASMSs affecting the remiges and rectrices can prevent the affected bird from being able to fly and exercise sufficiently to keep fit, even when sufficient space and opportunity is provided.

On another level ASMSs can represent a prompt, a question or a signal to us about what we are doing with and to birds in captivity.

Philosophical Considerations re a Diagnostic Approach to ASMSs

The title of this paper begs the question "why?". Why do we want to learn or even think about a diagnostic approach to ASMSs? Why do we need a diagnostic approach to ASMSs? If we don't need a diagnostic approach they why do we think we need it? I guess the answers each of us immediately jump to are many but generally mostly they relate to :

- ASMSs can be and often are a diagnostic challenge;
- veterinary surgeons want to prevent and cure disease and relieve suffering in birds; and
- veterinary surgeons want to identify "causes" in the hope that they can modify disease by treatment and management.

* **Homebush Animal Hospital, 195 Parramatta Road, Homebush NSW 2140**

These questions may at first appear to be stupid questions with obvious answers, but I would beg to differ with such a conclusion. Perhaps in the "end" we will learn that we don't need a diagnostic approach to whatever it may be in order to heal, but we'll create one for the heck of it, to help each other along the way, and so that future generations have a record of our "thinking" at this point in time.

One of our fundamental or core beliefs can be encapsulated as *the right treatment depends on the right diagnosis*. Yet there are those in our global community who can and do heal a multitude of diseases in animals and people without such knowledge in our terms. Why can't we, or can we? Why don't we, or do we? How can we? How do we?

So let us continue thinking, re-evaluating, questioning ..... why? .....How? Please carefully appraise these notes and observe your thoughts as you do. Perhaps it may prove interesting and informative for us all to read them again in 5 and 10 years time. Certainly ask yourself: is this true, is this just a theory or is it likely to be false or incomplete, why do I and you disagree where we do, as you try to digest what I have to say.

Ask yourself how can I test or investigate those areas which you question (there should be many!) and how can I help better focus our understanding, our thinking and our awareness.

Remember a tortoise doesn't get anywhere without sticking its neck out!

A diagnostic approach to ASMSs as traditionally adopted by veterinary surgeons is based on knowledge and/or theories about the different causes of, and associations with, ASMSs and disease, the perceived species differences and other factors considered likely to predispose ASMSs. Whereas in practice, when a veterinarian is presented with a case of ASMS, thought processes usually call upon recall of this knowledge and opinion, and quickly arrive at a "short list". ASMSs are frequently a diagnostic challenge to us which continue to leave many questions unanswered and many theories open to challenge and review. For example many birds that exhibit ASMSs may be infested with mites and/or lice, (many birds also eat seeds) but to rush to that conclusion without detailed investigation that these are the cause of or are in some way associated with the development of ASMSs is, in my opinion, not sound practice or thinking, since the probability of these being the cause is considered low in terms of our wider experience.

Therefore in practice, certainly make a mental short-list if you will, but develop and use a systematic approach which is open-minded but likely to encourage recognition of signs and clues to other aetiological factors.

Most of us recognise that many incorrect diagnoses and explanations about the cause and pathogenesis of disease have been made by not looking and therefore not seeing what is there to be seen. Other incorrect diagnoses are made through not listening, not feeling, not asking, not examining and not thinking. Then again we may be guilty of none of the above, but still arrive at incorrect diagnoses due to our wearing mental dark glasses or having tunnel vision, only being able to see one perspective. For example if we still think of birds as having "bird brains" (as the community implies in the derogatory sense), stupidity, then this attitude will act as a blind to our thinking. We may contrast this sort of thinking with an attitude that recognises or at least countenances the possibility that birds have far more complex emotional needs and more "intelligence" than suggested by the term "bird brain." The latter perception of birds opens a Pandora's box of factors which we might now investigate as contributing to ASMSs which we would fail to seriously consider if we still think of them as having "bird brains".

As an aside at this point, I recommend to everyone to take the time to read and think about *The Human Nature of Birds*" by TX Barber and available through Bookman Press Melbourne, Freecall 008 060

555. And no I have no financial incentive to make this recommendation!

Our traditional approach to diagnosis and healing may prove to be very limited and very restrictive and yet it still plays an important role in veterinary medicine at this time. Let us continue to use our present framework of beliefs, but not without constant questioning, as a major tool for living and communication at this time. Let us countenance the possibility if not the probability, that many of our traditional beliefs and values will be considered to be significantly different from our perceptions later in this lifetime, let alone the lifetimes of future generations. Let us tolerate, and hopefully examine other frameworks of belief to find and assimilate the essences of truth in each so that we may better serve our calling.

Philosophically thinking, traditionally as veterinary scientists and veterinary practitioners, our methods are very largely based on traditional scientific methodology: examine, recognise, process of elimination, positive findings, trial and error, testing etc. We have been taught (and this has been reinforced by learning), to ignore intuitive approaches. We have not been taught how, for example, to use a pendulum to answer yes/no questions, we have not as a rule either as individuals or as a group dared to or bothered to investigate "scientifically" whether or not there is a statistically significant validity to this methodology. We have not really studied either the value or the limitations of this diagnostic tool. (I have read in the writings of Lyall Watson, I think, that in Japan there are people who use a pendulum over poultry eggs aligned with the earth's magnetic field to determine the sex of unhatched fertile chicken eggs with a success rate and speed to match that achieved by experienced vent-sexers. We are gradually becoming more familiar with acupuncture-associated concepts of medicine and we have started to incorporate aspects of these into our traditional Western Medicine. Most of us are ignorant of metaphysical and iavetic medicine and perhaps of quantum healing methods. (Refer Quantum Healing by Dr O. Chalkla). We largely condemn herbal and homoeopathic medicine, Bach and Bush Flower Remedies etc without having undergone similar "training" and without having applied criteria equally biased as those we apply to traditional medicine. (For those who think otherwise, I apologise for apparently including you in the royal "we", if it has stimulated within you, offence!)

In my opinion, we are likely to expand our diagnostic and therapeutic repertoires as we open our minds to and undertake training in non-traditional modalities such as the above. We need to gain familiarity, confidence and competence and we need to test and appraise the results achieved. These are perceived aspects of our future.

Personally, my methods, my diagnostic approaches, are in a state of flux. They are changing and constantly being assessed and re-assessed. At this point in time I conclude that with respect to ASMSs each of us still has the opportunity to make "discoveries" and to contribute to our combined "knowledge".

If I could caricature two extremes, using alternative methods one might take a feather from the bird, psychically tune into it and its source body and "intuitively", "psychically" arrive at an explanation as to why and what to do.

Another extreme would be where if we were to attempt to investigate every considered reported possible cause of feather picking we would be performing so many pathology tests and subjecting the bird to so many procedures that we would be likely to lose the bird as a patient and the "owner" as a client.

Conceptually we traditionally like to attribute a particular cause to a particular disease. We traditionally classify infectious diseases as being due to prions. viruses, chlamydia, bacteria, fungi, protozoa, metazoa, nematodes, cestodes, trematodes, various arthropods etc. For example traditional

thinking says psittacine circovirus causes Psittacine Beak and Feather Disease, yet when we are prompted to think a little we recognise that many factors interact with psittacine circovirus to determine whether or not an individual develops Psittacine Beak and Feather Disease (PBFD), and in what form and with what severity the PBFD is manifest. However it seems that few as yet acknowledge that thoughts, attitudes and behavioural patterns can or might contribute to patterns of disease in ways that have many parallels with traditional infectious diseases. We often allow our thinking to fall into the traps of dismembering and oversimplification rather than trying to see the whole . A piece of blue in a jigsaw puzzle could represent many things until it is fitted into the whole picture. So it is with our approach to the diagnosis of ASMSs. Try to keep looking beyond the findings that you have made so far to see if they represent the complete picture or just one fragment of it.

So it is with varying degrees of openmindedness that we practice veterinary medicine and healing. There seems so much to learn, remember and recall scientifically, just from what our profession has already learnt and documented in the field, that to try to incorporate other frameworks of beliefs, of thinking and of conceptualisation (is there such a word?) is mind boggling. The cost of "basic tests" such as chlamydia tests, bacteriology and haematology is already prohibitive to many clients. It appears that if one were to pursue all the investigations we could theoretically undertake the costs would prove totally counterproductive. Already many birds with ASMSs are euthanased because of costs and the prognosis without detailed expensive investigation. Perhaps in future some will increasingly use intuitive and metaphysical methods for diagnosis and treatment, and be accepted by their peers.

Having said all that let us return to a traditional scientific based approach to ASMSs with which hopefully most of us will feel comfortable, despite its limitations.

Recognising ASMSs and distinguishing ASMSs from various feather, beak and claw dystrophies, "wear and tear", unkempt plumage, normal feather loss and regrowth (moulting at breeding - reproductive picking pattern on thighs or vent) and other conditions or syndromes with which ASMSs may be confused.

To recognise ASMSs we have to first recognise and distinguish from similar clinical signs evidence of self-inflicted damage to feathers skin or claws.

Features of ASMSs

1. **Distribution of lesions**

   A key aid to recognition of ASMSs is the distribution of the feathers and/or skin affected by signs indicative of stripping, barbering, plucking, biting and/or nibbling. By definition it is implied that these must be confined to those areas that the bird can reach with its own beak unless the bird cohabits with another which is also exhibiting one or more of these behavioural patterns which is directed towards the affected bird. (This is seen occasionally with pairs of Gang Gang cockatoos, and with captive cockatoos in aviaries and zoos.)

   Therefore typically most birds exhibiting ASMSs will have normal plumage on the upper neck and head unless they are affected by systemic disease or malnutrition of some form. In advanced and chronic cases of ASMSs affecting primarily the plumage there is often a clean line of demarcation approximately midway up the neck dividing normal from abnormal plumage.

   Note: An often overlooked aid to the recognition of ASMSs involves inspection of the feathers and feather fragments in the bird's cage or environment and examination of these for

signs of malformation and mechanical damage. Mechanical damage of at least some of the feathers is usually evident in cases of ASMSs.

2. **Features of "stripping"**

In this context "stripping" is used to describe that form of self-mutilation of feathers in which the bird splits the feather longitudinally (in a manner almost identical with that employed by birds to "chew" along the length of succulent grass stems so as to expose the tasty and/or nutritious "pulp" therein). By so doing this, the bird predisposes the split components to fracture and separation through normal "wear and tear". The bird may or may not then proceed to chew off or pull of the components of the feather. The primary and secondary remiges, the covert feathers and the rectrices are those feathers most commonly targeted by birds which "strip".

3. **Features of "barbering"**

In this context "barbering" is used to describe that form of self-mutilation of feathers in which the bird bites or nibbles through the distal component of the feather, this may involve the barbs and/or the shaft of the feather, and step wise shortens the affected feathers. The contour feathers of the body, particularly over the pectoral and shoulder regions are usually affected first by this behaviour but this behaviour may also be directed towards remiges and coverts along the wings.

4. **Features of "plucking"**

In this context "plucking" is used to describe that form of self-mutilation of feathers in which the bird actually biters onto but not through the feather then pulls the whole feather from its follicle. Usually this starts with easily reached mature contour and down feathers but it may also involve the removal of immature quills. If the powder downs are affected the bird needs to be carefully examined for signs of PBFD and some would argue that PBFD tests should be done, anyway. In Australia at present this involves the submission of appropriate blood and feather samples to the Department of Animal Health, University of Sydney, Private Mail Bag 3, Camden NSW). Birds that have been "plucking" usually have many short blood quills growing in the denuded or affected areas unless the problem is either very acute or very chronic.

5. **Features of "nibbling"**

In this context "nibbling" is used to describe that form of self-mutilation of feathers in which the bird damages and breaks off small irregular pieces of the vane of the feathers. This usually involves the guard or contour feathers of the body and may be a precursor to or concurrent with barbering. It needs to be distinguished from conditions in which similar pieces of feather break off at fault lines during normal "wear and tear" without abnormal preening. Fault lines have a characteristic alignment with the shaft of the feather and fraying of feathers associated with them gives the feather a "cut" appearance, as if it had been cut with scissors, but on close or microscopic examination of the barbs and barbules, anatomical abnormalities can be detected along the edge of the line.

Skin that has been nibbled may appear to be flaky or ulcerated. Often on close inspection affected areas will be found to have multiple or irregular layers missing. That is, the skin won't be all flaking off at the same level or layer. There may be signs of biting. There may be an impression of a transverse or longitudinal orientation to the edge of the missing layers in

relation to the position that the bird's beak would line up with the area during normal preening.

6. **Features of "biting"**

Feathers which have been bitten exhibit mechanical damage which may appear to be relatively random in distribution in terms of the part of the feather affected, but the mechanical damage usually has a transverse orientation. Pieces of feathers may or may not be missing. Feathers are often bent or angled at that point where they have been bitten, but this needs to be distinguished from damage associated with severe fault lines and damage associated with excessive "wear and tear" such as occurs when birds are kept in small cages, in cages where the perches are positioned such that there is insufficient space for the bird to turn around without damaging its feathers, and through predation, fighting, panic and mishandling.

Skin which has been bitten exhibits signs of discrete to more generalised trauma and arbitrarily will involve the deeper structures of the skin and often subcutaneous tissues as well. There may be evidence that layers of the skin have been pulled off. There may be evidence of deep tears.

7. **Symmetry**

Self-mutilation (SM) may be approximately bilaterally symmetrical or it may have an asymmetrical distribution. As a rule of thumb an asymmetrical distribution suggests an increased probability of finding a nearby underlying physical cause such as a tumour, abscess, granuloma, foreign body, or perhaps something pressing on nerves from the area. An example of the latter is a bird biting one foot and found to have kidney pathology or something pressing on the ipsilateral kidney and/or sciatic nerve. An approximately bilateral symmetrical pattern of SM suggests an increased probability of involvement of one or more of the following types of aetiological or predisposing factors being involved: emotional, nutritional, hormonal, metabolic, toxic, hypersensitivity ("allergic"), "genetic", systemic infection and a reduced probability of a localised physical cause.

One should remember that ASMSs may and in fact in certain species often does occur in the presence of other "apparently" unassociated diseases, including those that are commonly linked with feather dystrophies, ie PBFD psittacine circovirus infection, and probably psittacine papovavirus infection (psittacine polyomavirus infection).

Note: several texts have been used as sources of "current information" for the preparation of parts of these notes. "RHH, p. x" denotes Ritchie, Harrison and Harrison's latest textbook Avian Medicine: Principles and Application, published by Wingers Publishing, and the page number in the book supporting or sourcing my notes. Similarly GMC refers to Garry Cross's Bird Veterinary Medicine Veterinary Science V notes. See references and suggested reading for additional information.

Factors considered to be "causes of" and/or "contributing to", "predisposing" and/or "aggravating" ASMSs

1. **Emotional, Management and Stress Factors**

These are many and complex and perhaps the concepts are more important than the examples I have created below. We should keep in mind that a bird normally spends considerable time

preening, maintaining the alignment of feathers, removing keratin sheath from maturing blood quills, dislodging old feathers from follicles in which a new feather has already started to grow, removing ectoparasites and so on. 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 inadequate preening. Over-preening ("feather picking", some forms of ASMSs) occurs when what is a normal part of feather maintenance becomes a pathological condition (RHH p633).

Many chronic feather-picking birds display neurotic behaviour. They are agitated, hop from perch to perch, continually shake their heads in a figure of eight from side to side or just continually dip their heads from side to side (GMC p31). These signs may reflect frustration and fear in birds unable to cope with some forms of captivity, changes in the environment or social pressure. Neurosis is diagnosed in people who exhibit non-adaptive behaviour and attitudes, apparently in an attempt to come to terms with reality rather than escape it. Similarly, anxiety in a bird might be expressed as feather picking and self-mutilation (SM) (GMC, p31). Spoiled, improperly socialised, hand-raised birds of any species may also be prone to self-mutilation (RHH, p634).

It is thought that birds can manifest displacement activity as a response to chronic stressful situations. Grooming or preening is considered to be a common form of displacement activity, and may be attributed to frustration, motivational conflict, arousal and physical thwarting of desire or performance. Thus an acute stress may cause birds to take alarm and cry out and attempt to escape, but on the removal of the stress or having escaped from it, the birds then display "comfort behaviour", which includes a brief period of preening.

Thus if a bird is faced with an inescapable stressful situation, it will begin preening or feather picking, and this results from inhibition of the expected response to the situation (flight), and expression of displacement activity. If the situation is not resolved, frustration and anxiety might result in the exaggerated feather picking and SM of the "psychological" feather picker. This displacement preening in stressed birds differs from normal preening. It is of shorter duration, consists of frantic movements and an altered pattern of preening. Areas of plumage within easy reach receive more attention than in the normal situation (GMC, p31).

GMC. P31 notes that birds are gregarious and require constant and changing environmental stimulation to maintain mental health. Thus confinement in a small cage and an inability to escape confinement is considered likely to frustrate a captive bird. Isolated birds, in view of or in hearing distance of other birds of the same (or different) species, but unable to make physical contact, would conceivably be tormented by their imprisonment. Habit, boredom, attention-seeking, fear, nervousness, frustrations, including inappropriate pair-bonding, lack or loss of a mate, chronic egg laying have been implicated in chronic feather picking. It is postulated that these conditions place stress on a bird, in an environment from which it cannot escape, and therefore, cannot react instinctively (GMC, p31-32).

With regard to Emotional, Management and Stress Factors let's think in terms of the following A-Z examples to help us remember:

a. aggression from other birds, pets, family members particularly children
b. bonding with same or opposite sexed bird of same or different species, people
c. competition with other birds
d. depression
e. exercise: enforced lack of exercise as in confinement to small cage, wing clip, obesity.

f. frustration: unfulfilled needs in the broadest sense.
g. gaolhouse boredom
h. hygiene and holidays: separation from bird's "loved one" when humanised, changes to routine, boarding etc.
i. insecurity: anything which may make the bird feel nervous or insecure for extended periods. Eg confinement to small cage with all-wire sides placed below shoulder level or in an exposed area where it is likely to be seen by predators. Imprisonment.
j.
k.
l. lighting?
m.
n.
o. Oppression: domination by other birds, being low in pecking order
p. photoperiod irregularities: exposure to artificial light for irregular periods and cage being totally covered before dusk and/or after dawn.
q. quality of life
r. rest and retreat and lack thereof.
s. sex and sexual frustration, solitude, spoiled, improperly socialised
t. teasing by humans, torment.
u. unrecognised awaiting your discovery
v.
w. water under which to shower or in which to bathe; wing clipping has been loosely associated with ASMSs in species prone to this behaviour (RHH, p38)
x.
y. YTOTBIDN (You thought of this but I did not)
z. zinc poisoning from poor management and utilisation of unsuitable and/or inadequately treated and protected caged wire, for example. May occur with lead poisoning.

Some will consider much or perhaps all of the above to be anthropomorphous and non-scientific, but with those I beg to differ.

Diagnostic aids: detailed questioning and history taking, response to environment enrichment and modification of management (this is usually essential and most important), response to treatment with antidepressant drugs (this is band-aid treatment).

It is noted that "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, serum chemistries. feather pulp culture and cytology, skin lesions culture and cytology, radiographs, endoscopy and direct microscopic examination and biopsies of affected feathers. If no aetiology can be determined for the over-preening, then behavioural abnormalities should be considered." (Cooper and Harrison, RHH, p 634) (Stick this up on your wall and justify a fortune! RAP) (Do I need to say any more, or does this say it all: a diagnostic approach to ASMSs??!!!)

2. **Nutritional and dietary imbalances (deficiencies and excesses) both past and present**

Think in terms of direct deficiencies and imbalances as in of being of dietary origin, for example a high oil or fat diet in a bird confined to a small cage is likely to result in obesity fatty liver disease and liver dysfunction as well as many other problems.

Think in terms of indirect deficiencies and imbalances as in of being of non-dietary origin

where the diet offered is balanced but for reasons such as intestinal, pancreatic, hepatic disease the bird is unable to utilise all components of the food appropriately.

Often nutritional imbalances and deficiencies will be associated with changes in the colour and development of the feathers before or concurrently with the development of associated ASMSs, so look for these.

Feather picking may be initiated by dry, flaky, pruritic skin, which in turn can be caused by nutritional deficiencies, particularly deficiencies of Vitamin A, sulphur-containing amino acids, arginine, niacin, pantothenic acid, biotin, folic acid and salt. Excessive dietary fat has been incriminated as a possible cause of ASMSs. Deficiencies of minerals such as calcium, zinc, selenium, manganese and magnesium may be associated with brittle, frayed feathers and dermatitis. White streaks (usually associated with breakage) in pigmented feathers may be associated with a hypovitaminosis B (RHH, p846-847).

Parts of normally green feathers changing to yellow or black, blue feathers changing to black or white, red feathers changing to black, grey feathers changing to black, white or pink (eg Galahs) after signs often encountered which may sometime be attributed to "malnutrition" (RAP).

A deficiency of vitamin E and selenium has been associated with discolouration and atrophy of flight feather calami, and degenerative changes following haemorrhage in the pulp of immature feathers in turkey poults GMC, p33).

Abnormal feathering in pheasant chicks has been cured by the addition of zinc to the diet (GMC, p33).

Riboflavin deficiency has been reported to produce short, abnormal natal down feathers (clubbed down) in the domestic fowl (GMC, p33). Whether or not these deficiencies are or can be also linked to ASMSs is not documented here.

Diagnostic aids: Maldigestion/malabsorption: iodine staining of starch granules, ? orange staining of oil droplets; treatment trial with digestive enzymes eg crushed Gastrizyme tablets in food vs other brands available through health food store. Provision of processed formulated diet to which digestive enzymes have already been added eg Vetafarm Finch Budgie and Canary Crumbles, Vetafarm parrot Pellets; weight reduction diets, restricted sunflower seed intake. Addition of vitamins and minerals to diet concurrently with and following say 3 multivitamin injections including Vitamin A at several day intervals.

3. **Infections of the avian integument (feathers, skin and associated structures)**

a. **Prion-associated diseases**

None recognised yet. If Psittacine Pruritic Polyfolliculosis and Self-Mutilation in lovebirds are not viral-associated they may be candidates for investigation in terms of a prion-associated aetiology. (RAP considers the viral aetiology to be more probable.)

b. **Viral**

Psittacine Circovirus (PBFD), not commonly but perhaps through facilitating or predisposing other factors. Diagnostic aid: collect blood and blood quills eg powder downs for submission to Dept Animal Health, University of Sydney. Consider skin

biopsy if bird being anaesthetised. Consider holistic as well as "traditional Western" immunostimulant therapy. Consider preventive vaccination of future acquisitions prior to introduction to premises. Consider preventive vaccination of future acquired birds when vaccine gets registered).

Psittacine Papovavirus (Polyomavirus infection). Consider skin and feather follicle biopsy. Consider holistic as well as "traditional Western" immunostimulant therapy. Consider preventive vaccination of future acquired birds (when vaccine becomes available).

Poxviruses (Avian Pox). There are several strains of poxvirus that affect psittacine birds, one of which has been reported to cause itchy skin lesions in lovebirds (*Agapornis* spp.). This strain has yet to be recognised in Australia. Consider diff-quick stained impression smears and cytology, skin biopsy. Consider application of iodine. The cutaneous form ("dry pox" of avipoxvirus infections must be considered in the differential diagnosis of ASMAs as the lesions may easily induce a misinterpretation of self-mutilation. Remember that (with credit to RHH, p870, 872) the cutaneous form is the most common form of avipox disease in many raptors and passerine, but not in psittacine, birds. Changes are characterised by papular lesions mainly on the unfeathered skin around the eyes, beak, nares and distal to the tarsometatarsus. The interdigital webs are most commonly affected in waterfowl. As lesions progress, papules change colour from yellowish to dark brown and develop into vesicles that open spontaneously, dry and form crusts. Spontaneous desquamation may require weeks and occurs without scarring in uncomplicated cases. however pigmented skin will frequently be discoloured following an infection and secondary bacterial and fungal colonisation of lesions can substantially alter the appearance and progression of the disease.

Papillomavirus Infection. Consider skin biopsy to differentiate from herpesvirus-associated "warts" on feet.

? other Herpesvirus?. Consider skin biopsy. Consider Stoxil and Acyclovir.

c. **Bacterial**

Many organisms have been cultured and or recognised in skin and feather biopsies. The interpretation of their significance in terms of being a cause, incidental or a result of self-mutilation is difficult and is often dependent on the results of a treatment trial with an "appropriate" antibiotic. Infections may be associated with dermatitis and/or feather folliculitis. Some of the organisms include mycobacteria and *Staphylococcus* sp. Consider Gram Stains, Acid Fast stains, culture and sensitivity tests, skin and feather biopsies.

d. **Fungal**

Various dermatophytes similar to those documented in mammalian medicine have been associated with ASMSs. Consider examination under Woods Lamp (very few fluoresce). Consider in addition to Gram and Acid-fast stains, the use of potassium hydroxide/Parker Ink stain overnight. Consider *Fungassay,* fungal culture and submission of specimens to the Australian Reference Laboratory in Medical Mycology at Royal North Shore Hospital, St Leonards, NSW. Consider skin and/or feather biopsy. Consider systemic griseofulvin via Grisovin mixed in vegetable oil

through food. Consider topical thiabenzole (?Tecto 90, plant fungicide).

e. **Protozoan**

Protozoan infections of the skin and avian integument are probably unusual or rare, and their association with ASMSs probably more so. Trichomoniasis infections of the skin have been occasionally encountered in pigeons by this author (ears, umbilicus, perforating crop lesions, axillae) but not linked with much if any ASMSs. Protozoan infections of internal structures ie visceral organs, are far more common and some have been linked to ASMSs (See internal parasites).

f. **External parasites**

Many species of biting lice affect the skin of birds. A mild to moderate pruritus is accompanied by mild hyperkeratosis with minimal feather damage (GMC, p 8). Consider microscopic examination of feather, of feather pulp, of skin scraping. Many species of mites can infect birds. Some may be associated with ASMSs. Consider microscopic examinations as for lice. Consider white cloth test at night of bird's environs (Fowl Mites). Consider Pyrethrins, Permethrin, Carbaryl, Ivermectin, Avomectin, experimental Moxadectin 100 µg/mL oral solution at 200 µg/kg once every 6 weeks on 2 or 3 occasions.

g. **Unproven suspected infections of the avian integument**

Psittacine Pruritic Polyfolliculosis (Budgerigars, African Lovebirds, ?others) postulated viral "aetiology". Refer to paper by RAP in proceedings Avian Diagnostics.

"*Stress-dermatitis" self-mutilation in Agapornis spp*. (African Lovebirds) postulated viral "aetiology". (Refer to paper above). Diagnostic aids: conduct search for "polyfollicles", Consider skin biopsy.

4. **Internal Parasites**

(Note: Internal parasites rarely directly affect the avian integument). "Worms" (nematodes, cestodes, trematodes etc) of various sorts could theoretically predispose or contribute to the development of ASMSs in a variety of ways.

Giardiasis is particularly documented in Cockatiels (quarrions, *Nymphicus hollandicus*) in California, USA. GMC, p32, as well as others have reported an association between giardiasis and ASMSs in cockatiels. GMC, p32 notes 80% of cockatiels presented to Walter Rosskopf in Los Angeles, California, were diagnosed with having giardiasis. (In contrast I have provisionally diagnosed giardiasis in cockatiels of approximately 6 clients this year, (add another 2 in the week beginning 4/7/94!) by finding highly motile "speed-boat" protozoa rushing about within the liquid phase of fresh warm faecal smears under coverslip with condenser lowered. This represents a very low incidence but I fear yet a higher one than observed previously in Sydney. In some cases the protozoa have been as plentiful and obvious as commonly seen in fresh crop washings of birds with severe trichomoniasis. More often one has to search diligently (take 5 minutes) through a wet smear to find one organism. They tend to remain out of focus as a blurred shadow which if unobstructed by faecal debris can move across a 400x field in a matter of a second or two. I have only just (6/7/94) diagnosed an

ASMS in a Cockatiel with apparent Giardiasis. I suspect one of our past errors is to use tap water with its associated disinfectants, for diluting our faeces on the microscope slide. I suspect this probably inactivates many *Giardia* before we find them. Therefore try looking with warm sterile water, saline or Hartmans as the diluent.) GMC notes that birds with giardia infections may be asymptomatic or subclinical carriers of *Giardia* sp. Clinically affected birds were pruritic and had oily greasy feathers. It was postulated that gastrointestinal tract pathology might affect absorption and therefore the nutritional status of the bird, resulting in abnormalities of moulting and regrowth of feathers. Two to three weeks after treatment with Ipronidazole, the birds stopped the feather picking. Consider *Spartrix* (Carnidazole, Janssen), *Emtryl* (Dimetridazole, ?Cyanamid Websters), *Ronivet-S* (Ronidazole, Vetafarm), *Flagyl*, Metrozine.

Coccidiosis: I have not associated this with ASMSs.

Hexamitiasis in crimson rosellas, mountain lowries. I have not associated this with ASMSs but postulate that could be involved in some chronic cases.

Diagnostic aids: examine series of warm wet direct fresh faecal smears under 100x for oocysts, ova and, if you look carefully, *Giardia*. Need condenser lowered. Examine same smears at 400x magnification with condenser lowered. Perform faecal flotation test. Consider treatment trial. Consider *Giardia* antigen tests and other tests for *Giardia*.

5. **Genetic Factors**

Certain species appear to be particularly prone to exhibiting ASMSs behaviour. These include Gang Cockatoo, Major Mitchell Cockatoo, Mollucan Cockatoo, Galahs, Rosellas, Sulphur-crested Cockatoos, African Grey Cockatoos, Eclectus parrots and perhaps captive Australian ravens (crows) and various Mynahs. As ASMSs in these species are rarely, if ever, documented in individuals that have not been subjected to some form of captivity, it must be emphasised that the term "genetic factors" is probably being applied loosely, and it may be that these birds represent "the primates" in the avian world and that their needs when placed in captivity are more complex and less easily satisfied than with other species. Then again, other species may have similar "intelligence" but respond by dying for example rather than by ASMSs if their needs are not met.

6. **Hormonal influences, endocrine imbalances or deficiencies**

Many cockatoos show a seasonal incidence in their ASMSs associated behaviour. It is particularly common in female sulphur-crested cockatoos that have been tamed and/or are maintained in physical isolation from a companion during the breeding season and the behaviour often ceases or reduces at the completion of the breeding season. Most of us recognise variations of the "randy budgie syndrome". This form of ASMSs is probably loosely part of the female equivalent of the "randy cockatoo syndrome". Perhaps we might be more accurate if we substitute the term "frustrated maternal cockatoo syndrome" but what a mouthful!). The usefulness of medroxyprogesterone acetate may outweigh its hazards in these birds.

I suspect that some birds may be adversely affected by human pheromones such that these may contribute to ASMSs in some circumstances in view of the apparent rapid recognition of the sex of people by many hand raised and tame parrots and cockatoos (this is most obvious during the breeding season) and their responsiveness to people of the opposite sex to them (the parrots and cockatoos), and their aggression towards people of the same sex. It may be

that the birds recognise our sex on just visual and auditory cues but I think not. GMC, p32, notes hormonal imbalances can lead to feather problems. Thyroid hormone is important in the growth, differentiation and patterning of the plumage because it increases the metabolic activity of feather-forming cells and initiates feather growth in most adult feather follicles. Thyroidectomy of pigeons, ducks and chickens stops moulting and causes feathers to grow at a slower rate, become fringed and elongated with loss of barbules and colour. Hypothyroid parrots will pick at their feathers but respond well to thyroxine therapy. L-thyroxine sodium can be helpful. Oestrogen retards feather growth. Affected birds also appear pruritic, since they scratch and lose feathers. Progesterone will induce moult in the domestic fowl.

7. **Physical factors**

Trauma and fractures are a source of pain and can cause the bird to continually pick at the area, and often neglect the preening of feathers over other parts of the body.

Deformities such as scoliosis can prevent the bird from preening certain areas of the body and perhaps cause it to focus excessive attention on other areas of the body.

Wing Clipping has been loosely associated with ASMSs in species prone to this behaviour but this is unsubstantiated (RHH, p38).

Wounds

Burns both chemical and thermal
Injuries
Iatrogenic

Colour: the colour red on or amongst the plumage tends to encourage or promote feather picking and ASMSs in some of those birds which normally lack red plumage, for example with blood stains left in the plumage.

8. **Immune-Mediated Conditions**

Allergies - ?stings and bites from insects. ?Contact-type dermatitis. ?Photosensitization has been suspected in many cases of vesicular dermatitis, but the precise aetiologic agents are frequently undetermined. Beak deformation consisting of loss of normal epithelium on the surface of the beak, upturning of the tomia and shortening of the upper beak have been reported secondary to photosensitisation in ducks following ingestion of seeds from *Ammi visnaga*, *A. majus* and the plant or seeds from *Cynopterus watsonii* and *C. longipes* (RHH, p 485). The ingestion of parsley was associated with a photosensitization reaction with skin lesions in an ostrich, and experimentally in ducks (RHH, p1317).

?**Auto-immune diseases**. The obese strain chickens produce antibodies against thyroid cells, thus causing hypofunction and thyroiditis. Although to date, ASMSs do not appear to have been linked to auto-immune diseases, it seems likely that in future some will, such as those involving the integument directly, those involving the thyroid glands, the liver and the intestines, and perhaps last but not least, those involving nerves and/or the sense organs in the skin. It is likely that these conditions together with their association with ASMSs will be recognised and documented within the foreseeable future. See Amazon Foot Necrosis Syndrome below.

9. **Poisoning and toxins**

Heavy metal poisoning resulting in altered sensation and/or pain. I increasing link heavy metal poisoning with ASMSs and whenever there is not some other obvious cause then the possibility of heavy metal poisoning should be considered, investigated and/or included in a symptomatic treatment regimen. Biting and nibbling the skin of the toes and sometimes the toenails is highly suggestive of heavy metal poisoning in those birds and those cases where leg paresis and leg paralysis are common features of more severe poisoning. I postulate that the bird experiences something similar to "pins and needles", some altered sensation, which elicits this behaviour. (Treat metal poisoning systemically (Calsenate injections), remove/push through ingested metal with Metamucil, peanut butter, Maxolon, given calsenate orally as well, prevent re-access to source of poisoning, and try DMSO mix including xylocaine.)

Plant poisoning and other poisons are likely to be documented as being associated with some cases of ASMSs.

10. **Causes of cutaneous and/or internal pain and discomfort**

The following are examples of causes which should be considered but not all have necessarily been documented to be associated with ASMSs.

Feather cysts may become secondarily infected with bacteria and have been seen on Sulphur-crested cockatoos, macaws and Amazon parrots.

Tumours. There are many types of these, and depending on size and location, one would think any might initiate ASMSs nearby. Tumours affecting gonads or the endocrine system might have more generalised effects. Perhaps, similarly, any tumours which may cause the release of prostaglandin-like substances (theoretical thought at present), foreign bodies such as migrating grass seeds, splinters, ingested needles, pins, fragments of wire.

Abscesses
Granulomas
Localised infections
Adhesions

11. **Metabolic and organ diseases associated with ASMSs**

Liver disease. Clinical signs suggestive of pruritus and feather picking have been reported in birds with liver disease. other integumentary disorders that are loosely discussed in association with liver disease include pigment changes of feathers, abnormal moulting and softening, flaking and overgrowth of the beak and nails (JT Lumeij in RHH, p572) and interlaminal haemorrhages in the beak and claws (RAP). Diagnostic aids: PCV, WBC and differential, A:G, AST, LDH, Bile acids, Total body radiographs. Questions re contact with other birds.

Air sacs. Feather picking over the air sacs may be an indication of irritation that requires further investigation (Tully and Harrison, RHH, p572).

12. **Syndromes of unknown aetiology.**

One might say that most cases of ASMSs could be placed in this category, tongue in cheek!

Amazon Foot Necrosis Syndrome: Pruritic ulcerative lesions have been described on the feet and legs of Amazon parrots, particularly Yellow-naped, Double Yellow-headed Amazon parrots. The lesions start with a bird chewing at the feet and legs followed by the formation of hyperaemic lesions, sometimes within minutes of the initial pruritic episode. An ulcerative dermatitis occurs as the bird continues to chew on the feet and legs. Characteristic histopathological findings associated with this syndrome include ulcerative dermatitis that may contain coccoid bacteria and fungi. The role of these is undetermined. Immune mediated and allergic reactions with secondary involvement of autochthonous (love that word!) skin flora have been proposed as aetiologies for these lesions. (Credits to and Refer to RHH p632 for more details). (See also RHH, p965, in which an Amazon parrot with this ASMS healed when the owner, a smoker, started washing her hands after smoking.)

"**Segmental dysplasia**" is another term for "Stress lines" or "stress Marks" in feathers.

So in developing an approach to the diagnosis of ASMSs one needs to consider all of the above plus a whole heap of factors which I no doubt have omitted.

Therefore, as usual the first steps are:

1. **A thorough history-taking and a thorough clinical examination physically**

A thorough history-taking and clinical examination is a prerequisite for recognising many of the potential causes of ASMSs. Translating that from recognition of a potential cause to validly attributing significance to it in a particular instance is fraught with the possibility of error.

Remember that in many cases there appear to be a multiplicity of factors which might be contributing to ASMSs. The more of these that can be identified, and attended to, the more likely will be a successful outcome.

The methodology of performing a thorough clinical examination has been described in detail by this and other authors in various Proceedings of PGCVSc, Uni of Sydney, in avian textbooks, and in Proceedings of AAV.

My methodology as a matter of routine in all but the most obvious cases includes microscopic examination of a fresh "hot and steaming" bird dropping as a wet smear under a cover slip at low medium and high dry magnifications with condenser lowered as soon as possible after the dropping has been passed by the bird. It also usually includes similar examination of a fresh warm crop wash and aspirate, followed by examination of gram stained smears of both crop and faeces. In terms of ASMSs I am interested in both abnormal and abnormal findings. Among other things, I am particularly interested if I find any internal parasites, megabacteria, actively dividing yeasts in increased numbers, evidence of active bacterial infections, reduced total bacterial count in the faeces without recent antibiotic treatment or the use of such products as Avi-clens (Chlorhexidine, Vetafarm) or Halasept or Halamid (chloramine, Cenvet) in the drinking water (suggesting a toxin may be present in the gastro-intestinal tract, think heavy metal poisoning first).

The art of obtaining a thorough history in the context of a consultation in practice and the limitations of restricted time involves in part targeting the client with questions that as quickly as possible help you eliminate or identify those possible causes of ASMSs that are considered most applicable to the species of bird under examination.

Some questions which you might find helpful in various circumstances include:

- How long have you had the bird?
- Do you keep other birds? (What experience do you have with aviculture? What is the risk of acquired infectious disease? Do you show or mind (baby-sit) other peoples' birds? Where is this bird in the pecking order? Does it have a mate? How do they interact?)
- Have you bought any other birds recently? (Have you altered your routine lately, could the bird have been exposed to infectious diseases recently?
- Where is this bird kept? (Does it have exposure to wild birds and infectious diseases? What size is its cage? What is its cage made of? Ascertain with what furniture perches toys the bird is provided.)
- Is the bird allowed out of its cage? (This question is particularly relevant to various poisonings, particularly with heavy metals and indoor plants, and contact with or exposure to irritant chemicals.
- When, ie what time of day or night, and under what circumstances do you see the bird exhibiting the (insert the appropriate descriptive term for the ASMSs) behaviour?
- What treatments have you already tried? (At what dose rate, for how long etc ?) What changes in management have you already tried? What medications is the bird currently receiving?
- What do you offer the bird to eat? Of this, what does it actually eat? What is the bird's diet? How often is the bird fed?

At this time make a mental list if not a written list of your differential diagnosis, then re-arrange that list in priority of considered probable causes. Discuss these with the client while you are mentally listing the samples you wish to collect. Discuss these with the client, illustrating the benefits to be obtained in helping you help the bird and guide the client, as well as noting the costs of such tests and getting permission to proceed or instruction to limit your examination to a treatment trial or whatever.

Perform another physical examination focusing on high priority possibilities and review your differential diagnosis and the samples you want to collect and/or the procedures you wish to perform.

If appropriate take samples eg mature feathers, blood quills and blood and perform the procedures eg radiograph the bird, take skin and feather follicle biopsy under isoflurane anaesthesia using radiosurgery (Ellman Surgitron or similar).

Initiate a treatment protocol awaiting results of tests and procedures.

This will review basic management, nutrition and will often include provision of a more interesting stimulating environment, making the bird search and/or work for its food, treatment of any 'treatable" diseases discovered, irrespective of whether or not they are considered likely to be associated with ASMSs, prevention of further ASMSs by Elizabethan collar or neck brace if indicated by self-inflicted damage to flesh until underlying and predisposing factors have been corrected and dealt with.

Record and document the above.

Assess results and revise treatment protocol in light or darkness of findings.

Record and document.

Plan and carry out follow up enquiries and/or progress examinations.

Record and document.

The process is rarely as simple as this but I'm sure you'll get the idea that you will have struck it "lucky" if you get it right the first time!

Quoting from RHH, p635 "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 PBFD virus infections, treatment of other feather abnormalities with an aetiology that was determined by the minimum database was generally successful. Idiopathic cases of feather picking were less likely to respond to therapy."

Management of Birds manifesting ASMSs

Obviously we aim to identify and "treat' as many contributing and predisposing "causes" as we can in the individual bird, its diet, and its environment. In principle, we always have Nature as our teacher, if we so desire. I find that "She" can be very helpful. Certainly I place a great deal of emphasis on addressing sub-optimal management and nutrition, as I usually consider the longer term answers will be found here. Certainly, I usually prescribe a combination of medications at the same time, but I put these in the context of buying time for the bird while we address underlying and predisposing management and dietary factors.

Elizabethan collars, tube collars and other physical restraints are sometimes used to buy time to prevent (further) self-mutilation and to facilitate healing of lesions while the underlying "causes" are being discovered and addressed.

Medications used to treat ASMSs

Note. In the view of RAP medications such as those noted below are usually relatively short term aids for treating manifestations of underlying deficiencies in nutrition, management and welfare. Their prolonged use is discouraged in favour of encouraging the attending veterinarian to fully investigate the ASMS as noted elsewhere, and/or the "owner' of the bird to face the reality that usually MS has a large component of association with mismanagement in a captive environment. For example he or she may not be able to meet the needs of the lone imprinted bird, especially during the breeding season. One might do well to question what is in the bird's best interests: euthanasia, prolonged symptomatic treatment or attempts to rehabilitate and bond the bird with another of the opposite sex but same species with the facilitated opportunity to breed and experience more of Nature's repertoire in captivity vs the wild (after appropriate conditioning, training etc). Similarly sometimes birds are paired by us inappropriately such that a pair may be socially compatible but sexually incompatible, as an example. The answer may then be found in breaking the bond and re-establishing it with another more compatible bird.

Quoting from RHH, p 636, "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 behaviour. 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 behavioural modification (see Chapter 4). If these therapies are determined to be ineffective over a two-month period, then mood-altering drugs may be necessary."

1. **Hormones**

Administration of progesterones, as a cure for feather picking, is debatable. Efficacy declines over time and prolonged use predisposes the bird to side effects. However, it has been successful in a small percentage of cases. Sexual frustrations are thought to contribute to feather picking, and therefore some birds may respond to sex hormones. Side effects of Medroxyprogesterone Acetate (MPA) can include quietness, increased appetite, polydipsia and polyuria, increase in pecking order (in the same hen) and suppression of ovulation. (GMC, p35). A diabetes mellitus-like syndrome may be seen with some birds receiving repeated MPA injections. In one bird, a budgerigar experiencing continuing weight loss and marked glucosuria, and rapidly approaching emaciation and debility, the side effects were apparently reversed by use of a combination of herbal therapies witnessed and recorded by RAP. On the subject of apparent diabetes mellitus in birds, it has been suggested that a diet of predominantly hulled oats can be used to reduce polydipsia and polyuria (Gestier, pers com 1994).

Medroxyprogesterone Acetate: Depo-Provera, Upjohn Pty Limited. MPA. Vials, sterile aqueous suspension, 50mg/mL; 1 mL. Dose: 0.025-0.07mg/g. This is of use only if there is a reproductive endocrine imbalance; the owner should be warned of possible side effects, such as those noted above (GMC, p36). (Other brands and injectable formulations of MPA are also available in Australia.)

Megestrol Acetate: Ovarid, Glaxo, Tabs, 5mg, 20mg. Dose: start with 5 mg/100 mL drinking water and titrate up at double dose or down at half dose if indicated by side-effects vs ineffectiveness, according to response, at about 4 day intervals, as an alternative to MPA injections. Side-effects similar. Risks and indications similar. Failure rate similar, but has advantage that drug can be withdrawn from body by withholding treatment more quickly than with long-acting MPA injections. (RP)

Prednisolone: Solu-Delta-Cortef Solution, Upjohn Pty Ltd - Prednisolone sodium succinate - Injection - vial - 100mg/10 mL. Each mL contains 10mg of prednisolone. Dose: 5-10mg/kg IV. Use only if a bird is truly pruritic. Try lowest dosage, preferably short-term (GMC, p37).

Thyroxine: Oroxine, Wellcome Australia Limited. L-thyroxine sodium. Tablets 50microgram; 100 microgram; 200 microgram. Dose: crush 50 µg in 15 mL water and give 0.4-0.5 mL/kg SID (dose 1.5 µg/kg). This drug may be helpful if hypothyroidism is suspected as a contributing factor to the problem, or if there is a temporary or seasonal hypothyroidism, or together with other therapy to encourage new feather growth regardless of T4 level. (GMC, p36.) RAP has prescribed this drug for periods of 2 to 12 months, over many years at doses of 50 to 100 µg/100 mL drinking water fresh daily as an adjunct to dietary therapy to promote weight reduction in obese budgerigars, galahs and sulphur crested cockatoos. Note that obesity, fatty liver disease and ASMSs often seem to occur concurrently in these birds prior to treatment.

Canitone: Tabs. Contain thyroxine, other hormones, and some vitamins. RAP has substituted these tablets for oroxine at arbitrarily chosen doses from 1/4-1 tablet per 100 mL drinking water fresh daily for periods of 3 to 6 months for some birds on weight reduction diets and with suspected hormonal imbalance associated feathering disorders.

2. **Antidepressant Drugs**

GMC, p35, notes that these are behaviour modifiers and their use requires good communication between the owner and the veterinarian to evaluate effectiveness and to monitor possible side effects or adverse reactions. All tricyclic antidepressants have some antihistaminic, anticholinergic, and local anaesthetic properties, making them useful as adjunct therapy for feather pickers or mutilators.

Monoamine oxidase inhibitors act to inhibit monoamine oxidase, resulting in a rapid rise in noradrenaline, serotonin and dopamine, particularly serotonin, at the synaptic cleft, returning

the hyposensitive postsynaptic receptor-site to normality (GMC, p35).

Tricyclic Antidepressants (TCA)

These seem to be the treatment of choice for chronic feather pickers refractory to traditional modes of therapy. TCA act to inactivate catechol-o-methyltransferase and block the reuptake of neurotransmitters. This increases their concentration at the synaptic cleft, thereby facilitating transmission of neuronal impulses, by prolonging the duration of contact of noradrenaline and serotonin with the post synaptic monoamine receptor site. Once the appropriate TCA and maintenance dose for the alleviation of signs of ASMSs has been determined, treatment must be continued for six to 12 months beyond remission of these signs. Maintenance doses are generally one half to two thirds the dose required to induce remission of signs (GMC, p35-36). Doxepin - Deptran: Alphafarm Pty Limited - Doxepin hydrochloride - capsules, 10mg, 50's; 25mg, 50's. Sinequan: Pfizer Pty Limited - Doxepin hydrochloride - Capsules, 10mg, 50's; 25mg, 50's. Dose: Start with 0.5-1.0mg/kg BID (GMC, p37.) Doxepin HCl, Sinequan, Roerig, USA. Capsules (10, 25, 50, 75, 100, 150mg) or suspension (10 mg/mL) for oral administration (humans). May be helpful in some cases of ASMSs. May cause severe lethargy (RHH, p462).

Non Tricyclic-Non Monoamine Oxidase Inhibitor Antidepressants

These have proved to be equally as effective as the TCA in treating similar psychiatric conditions in people, but this is not so with birds (GMC, p 36). Amitryptyline. Endep: Alphafarm Pty Limited - Amitryptyline HCl - Tabs, 10mg; 25mg; 50mg; all 50's. or Charles E Frosst (Australia) Pty Limited - Amitryptyline HCl. Tabs 10mg; 25mg; both 50's. Dose: PO, SID-BID 1-2mg/kg. Rarely effective. Should not be used in conjunction with monoamine oxidase inhibitors (GMC, p37). Nortriptyline: Allegron - Dista Products (Australia) and company - Nortriptyline hydrochloride. Liquid, 10mg/5 mL; 100 mL. Dose: start with 1 mL/100 mL drinking water. Rarely effective. Should not be used in conjunction with monoamine oxidase inhibitors (GMC, p38).

Neuroleptics

Haloperidol is a neuroleptic drug or tranquilliser, an antagonist for catecholamines. A monolayer of haloperidol blocks endogenous and exogenous catecholamines from reaching their receptor sites and so prevent the transmission of nerve impulses. Low doses specifically block the nigrostriatum of the midbrain, and high doses block the noradrenergic A10 neurones in the median forebrain. Blockage of the nigrostriatum has an antipsychotic effect, while median forebrain blockage causes sedation. Haloperidol is an or perhaps can be an effective drug for feather picking and ASMSs birds. Results are not so good for birds that are feather picking other birds and not mutilating themselves (GMC, p36). Serenace, Searle Laboratories - haloperidol: Liquid, 2mg/mL, 15 mL, 100 mL, 500 mL. Dose: 0.2mg/kg for birds under 1 kg; 0.15mg/kg for birds over 1 kg. Frequency not stated. (GMC, p38).

Narcotics

Naloxone hydrochloride Injection USP - either Commonwealth Serum laboratories, Mini-10-jet Syringe 0.4mg/mL, 1 mL, 2mL, 5 mL or David Bull Laboratories: ampoules 0.4mg/mL 5's. Syringe 0.4mg/mL. Dose: 1.5 mg/kg BID IM (GMC, p38).

3. **Dimethyl Sulfoxide (DMSO)**

Rightly or wrongly this author has frequently used and prescribed mixtures of DMSO, usually 17-19 mL, with injectable dexamethasone solution 2 mg/mL (Azium, Schering, or Colvasone), usually 2 to 4 mL, and injectable trimethoprim sulphonamide solution (Trivetrin injection, Wellcome) and sometimes with added 4% topical xylocaine solution (as for spraying cat's throats) 2 to 4 mL, for topical application sparingly BID to a variety of pruritic, scabby, ulcerated and/or infected lesions affecting the non-feathered avian integument. Such treatment has yet to be submitted to double blind controlled evaluation! (But I think it often helps!)

4. **Miscellaneous**

Aspirin, Bayer Aspirin, Bayer Australia Limited - Tabs. 300mg. Dose: 150mg/kg TID. (GMC, p37).

Diazepam, Diazepam Injection, Astra Pharmaceuticals Pty Ltd, Ampoules 5 mg/mL, 2 mL, 5's, 50's. Diazepam Injection USP, David Bull Laboratories, Ampoules, 5 mg/mL, 2 mL, 5's, 50's. Valium, Roche Products pty Limited, Ampoules, 10 mg/2 mL, 5's. Hypnovel, Roche Products Pty Limited. Midazolam water solution - 15 mg/3 mL(5's), 5 mg/1 mL (10's). Palmin Injection (veterinary). Dose: 2-6 mg/kg IM, IV. it is preferable to use this drug in hospital, since any bird kept on low dose tranquillisers should be supervised or observed frequently (GMC, p36).

Clomipramine HCl - Anafril Baker Cummins, USA. Capsules 25, 50, 75 mg. Used (in humans?) to control compulsive disorders. May be effective in some cases of ASMSs in birds. Initial dose should be low with a gradual increase over a four to five day period. Clinical impressions suggest that this drug is rarely effective in controlling ASMSs in birds. Numerous metabolic side effects. Regurgitation and drowsiness may occur in some birds. Ataxia may develop (RHH, p 461).

Diphenhydramine HCl, Benadryl Elixir, Parke Davis Pty Ltd. Elixir 12.5mg/5 mL - 100 mL, 500 mL. Diphenhydramine HCl Injection, CSL. Single use vial and vial injector, 50mg/mL, 1 mL. Dose: 2-4mg/kg BID, start with lower dosage. Slight sedative effect, and anti-depressant properties. Can be used long-term if necessary (GMC, p37). Also has antihistamine properties. Has an atropine-like action and toxic side effects.

Phenobarbital, Phenobarbitone sodium, Fawns and McAllan P/L, Injection 200mg/mL (5's). 0.05 mL/300 mL DW. Dose: 3-7 mg/kg BID-TID. Be aware that the bird may have droopy eyelids (GMC, p 37)

5. **Alternative and Complementary Therapies**

Rosehip and Camomille Teas etc, Australian Bush Flower Remedies (See book of same name by Ian White, Bantam Press)(for example combined Waratah, Illawarra Flame and Sturt Desert Rose for immunostimulant therapy, 1-7 drops bid PO 14-28 days, then change); herbal preparations (For example Echinacia, Pau D'arco, Resistance Plus, Immuno Plus, IGS 11 for immunostimulant therapy); homoeopathy, others. Acupuncture. Tellington Touch (TEAM).

Recommended Reading

RHH Ritchie BW, Harrison GJ and Harrison LR (1994) Avian Medicine: principles and application. Wingers Publishing, Inc, Lake Worth, Florida, USA. (PO Box 6863, Lake Worth, Florida 33466-6863).

GMC Cross GM (1994). Bird Veterinary Medicine. Veterinary Science V. Department of Animal Health, The University of Sydney.

Harrison and Harrison (1986 and second edition) Clinical Avian Medicine and Surgery. Saunders.