



BUMBLEFOOT SURGERY AND MANAGEMENT

NEIL A FORBES BVETMED RFP DIPECAMS FRCVS

Great Western Exotic Vets

Unit 10 Berkshire House, County Park,
Shrivenham Road, Swindon, SN1 2NR

www.gwexotics.com

INTRODUCTION

Bumblefoot is a condition recognised as an inflammatory, and typically infected lesion of the plantar aspect of the foot, affecting either the ball of the foot or one or more toes. Bumblefoot is a common disease of large species of raptors maintained in captivity and tends to become chronic, progressive, invasive and eventually disabling, which is often either unresponsive to traditional therapy or recurrent. The condition most commonly affects falcons (long-wings) and larger owls, whilst hawks (short-wings) (e.g. goshawk (*Accipiter gentilis*), and broad wings (Harris' Hawk (*Parabuteo unicinctus*), buzzards (*Buteo* spp.)) are very rarely affected. The condition is very common in captive birds but is rare in free living birds.

AETIOLOGY

Raptor's feet are protected by a thick layer of stratified squamous epithelium, which in turn is covered by a layer of keratin. On the plantar surface is a covering of hard papillae, these are thought to help to disperse the pressure of the weight bearing function of the foot.

Bumblefoot is caused by one of two main aetiologies. Firstly any penetration of the foot, caused by a talon, thorn or other sharp foreign body, or abrasive object may simultaneously introduce infection either into the dermis or sub dermal tissues. Secondly and far more commonly, captive birds, whether tethered on perches or free in aviaries, may suffer from an 'avascular necrosis' of the plantar aspect of the ball of the foot or a toe. The enforced use of unsuitable perches, or simply the effect of greater periods of inactivity, during which time they are taking excessive weight on their feet, leading to pressure necrosis (similar to a human bed sore) (Griner 1983, Harcourt-Brown 1994). Once such an area of avascular necrosis occurs the normal epidermal defense mechanisms fail, facilitating the migration of pathogenic bacteria through the skin.

The organism most commonly implicated is *Staphylococcus aureus*, this bacteria has a complex pathogenesis, frequently producing endotoxins or exotoxins, which serve as virulence factors (Anderson 1986). The organism is considered to be an opportunist, taking advantage of a breakdown of host immune defences. Malnutrition such as vitamin A or vitamin E deficiencies or any cause of immuno-suppression, such as stress

or intercurrent disease, predisposes to bumblefoot infection. However foot trauma in other species or individuals with similar deficiencies do not result in the same clinical scenario, hence some other reason or mode of immune suppression has been postulated (Oaks 1993). Oaks (1993) attempted to stimulate falcon's immune response to *S. aureus*, by immunisation of bumblefoot affected falcons however there was no evident improvement in recovery rates. Other organisms have been implicated, *E coli*, *Pasturella* spp., *Klebsiella* spp., *Clostridium* spp., *Corynebacterium* spp., *Bacillus* spp., *Diplococcus* spp., *Nocardia* spp., *Actinobacillus* spp., *Actinomyces* spp., *Aeromonas* spp., *Proteus* spp., *Pseudomonas* spp., *Candida* spp. and *Aspergillus* spp..

In the author's experience the nature of the pathogen has a great influence on the outcome of the case. The pathogens with the gravest prognosis are the last four listed above. It is of interest that *Aspergillus* spp. and *Candida* are not recognised as significant pathogens in the Middle East, where much of the current falcon research is carried out. The author postulates that this relates to differing temperature and humidity rates. Remple *et al* (1993), demonstrated perivascular cellular reactions, some leading to perivascular inflammatory infiltrate, oedema, degeneration of collagen and a necrotising vasculitis, in many bumblefoot cases. In all cases there was a marked nodular vascular proliferation/fibrous thickening with endothelial and smooth muscle hyperplasia with consequent luminal narrowing, representing endarteritic obliterative vascular changes. Such changes will inevitably reduce cellular perfusion at the site of the lesion and hence to perpetuate the disease. The formation (within 3 -5 days of initial trauma, (Cooper 1978)) of a fibrin barrier, (which is catalysed by staphylococcal coagulase), insulates the pathogen from phagocytosis and antimicrobial action, which may explain the progression, failure to self heal, and reoccurrence rate of many cases. Although it has been shown that avian heterophils can ingest *S. aureus* in vitro, Glick *et al* (1964) demonstrated that heterophil activity in vivo in the presence of *S. aureus* to be markedly reduced. The later observation suggests a phagocytic paralysis that may result from staphylococcal toxins or a vague immunosuppressive response somehow induced by the presence of the pathogen.

Whilst *S aureus* is the predominate pathogen in raptor bumblefoot, Cooper and Needham (1976) demonstrated that the incidence of naturally occurring *S. aureus* on raptor feet is rare. Raptors may be poorly adapted to human *S. aureus* (Satterfield and O'Rourke, 1980) and that when challenged with these organisms the bird may be unable to mount the appropriate immune response. Turk and Stamm (1970) reported that the introduction of *S. aureus* into the skin can initiate a hypersensitization reaction with considerable resultant tissue damage. Hypersensitisation is likely to be an Arthus Type III reaction, which would result in thrombosis and vasculitis, as demonstrated by Remple (1993).

CLINICAL SIGNS

Response rates are greatly improved if treated early. The severity of cases are graded (Oaks 1993).

- Class I.** Early devitalisation of a prominent plantar area without disruption of the epithelial barrier, subdivided into :-
- a. Hyperaemia (bruise) or early ischaemia (a blanched area with compromised capillary perfusion).
 - b. Hyperkeratotic reaction (an early callous).

Favourable prognosis, as there is no evidence of infection. The changes generally respond to conservative, husbandry changes, including changing perching surfaces and application of topical emollients eg. Preparation H (Whitehall Labs).

Class II. Localised inflammation/infection of underlying tissues in direct contact with devitalised area, with no gross swelling, subdivided into :-
a. Puncture wound.
b. Ischaemic necrosis of epithelium (a penetrating callous or scab)

Good prognosis as infection is localised. Such lesions respond well to surgery as the total affected area is easily resected and epidermal defects are characteristically small, hence the architecture of the weight bearing structures of the plantar aspect are maintained intact. This class will generally not respond to conservative treatment.

Class III. More generalised infection with gross inflammatory swelling of underlying tissues. The origin may be puncture wounds or ischaemic necrosis, however by this stage the initial cause is of minor significance in comparison with the gross on-going pathology. Subdivided into :-
a. Serous (acute)- oedema and hyperaemia of the tissues.
b. Fibrotic (chronic)- attempt at encapsulation and confinement.
c. Caseous - accumulation of necrotic debris.

Traditionally carries a fair to guarded prognosis, as infection is well established, and structural changes have affected the foot. The majority should be treated by complete surgical removal of all affected tissue, followed by first intention healing.

Class IV. Established infection with gross swelling and involvement of deeper vital structures. Radiology and surgical exploration will often be required to differentiate types III from IV. Class IV is a chronic condition causing tenosynovitis and occasionally arthritis and osteomyelitis.

Carries a guarded to poor prognosis as infection is harbored in and affected deeper vital structures, making surgical debridement difficult or impossible. In view of the chronicity pockets of encapsulated infective tissues are often present, which if not cleared will result in latter reoccurrence (see treatment regimes later).

Class V is an extension of Class IV and is characterised by crippling deformities, such cases have previously been considered to be inoperable and best euthanased without treatment.

Until recently the treatment of Class IV and in particular Class V cases carried a poor to grave prognosis. Remple and Forbes (1998) described the use of antibiotic impregnated methymethacrylate beads (AIPMMA), in the treatment of bumblefoot. The use of AIPMMA beads, following aggressive surgical debridement, offers an alternative method for the delivery of antibiotics to an infected site (Klemm 1993). This technique has been used safely and efficaciously in birds for the treatment of cellulitis and osteomyelitis (Wheler *et al* 1996). By this method higher local concentrations of antibiotic can be achieved than with systemic administration without relying on vascular supply and soft tissue. Ototoxic and nephrotoxic serum levels, and other undesirable

side effects such as allergies and nausea, are avoided as minimal systemic uptake occurs. AIPMMA beads are easily made and may be sterilised with ethylene oxide or gamma irradiation (Tobias *et al* 1996). In their study AIPMMA beads, with a range of different antibiotics and antibiotic combinations, were used in a number of cases of bumblefoot ranging in severity from Class II to Class IV, following the classification of Oaks (1993), in a range of raptor species, resulting in a marked improvement in long term resolution and cure rates.

CLINICAL MANAGEMENT

The first step in bumblefoot management is the education of one's clientele. The routine of daily handling and monitoring of foot health is invaluable. If cases can be detected as Class I stage, then virtually all will immediately respond to conservative therapy. Inevitably as a consequence of poor husbandry, or in cases where breeding birds are in secluded aviaries, regular close inspection may not occur. In such situations birds may be presented in more advanced stages. Conversely, at whatever stage a case is diagnosed, if the underlying husbandry cause is not addressed, the case will inevitably reoccur.

On presentation a full history should be taken and the bird should receive a full clinical examination. Any concurrent disease, leading to excessive weight bearing on either foot, or causing pedal problems (e.g. pox, frost bite, spinal or pelvic limb injuries etc.) or general illness (e.g. nutritional deficiencies, aspergillosis, tuberculosis etc.) should be evaluated. Some authors have considered Vitamin A deficiency to be significant, any bird fed regularly on day-old chicks (so long as the yolk sack is left intact at least once a week) is highly unlikely to suffer such a deficiency. Vitamin E deficiency has also been implicated, this is most often caused by the storage of frozen food for excessive periods prior to feeding. Food should not be stored frozen for more than three months.

Bumblefoot never only affects one foot. As soon as one foot is affected additional weight is born by the good foot, the latter rapidly leads to a pressure necrosis bumblefoot in the good foot. In view of this on the occasion of the initial examination, if one foot appears unaffected, action should be taken to dissipate weight over the whole surface of the good foot, including the toes, rather than simply the ball of the foot, in order to prevent disease of the second foot.

Each case should be classified and the appropriate investigative procedures should be carried out, e.g. microbiology, radiology, surgical investigation. In the author's hospital microbiology swabs are taken for bacteriology and sensitivity testing, prior to surgery. The importance of this is that the fibrotic reaction can encapsulate infective organisms within 3 - 5 days (Cooper 1978). If sensitivity testing is carried out at the time of surgery, the results may not be available in time to be effective prior to fibrotic encapsulation of infective organisms. Such a scenario would render the surgery ineffective. Prior to the results of sensitivity testing being available a first line antibiotic should be administered. In this situation the author favours marbofloxacin or B lactamase potentiated penicillin. Some clinicians administer Vitamin A prior to surgery (30,000 iu Vit A/kg body weight). In cases where there is extensive oedema and swelling of the tissues of the foot, a ball bandage soaked in a combination of Dimethyl sulphoxide (DMSO), dexamethasone and piperacillin is placed for 2- 3 days prior to surgery. Such therapy reduces the swelling of the foot very rapidly, controlling any hypersensitivity reaction that may be present, which in turn it is hoped will improve

vascularity. DMSO when applied topically will cause a 'garlic like' odour on the birds breath, this can depress the birds appetite, such therapy should not normally exceed 4 days duration.

SURGERY

The aim of surgery is to reduce the antigen loading, were possible removing infected and fibrotic tissue in toto thereby converting a necrotic infected area into a clean, vascular surgical site that may be closed to heal by first intentions. The skin is carefully prepared to remove any bacterial contamination. A tourniquet is applied to the leg, for a minimal period during surgery to facilitate a 'good blood free' operative site. The bird is placed in lateral or dorsal recumbency, with the talons extended by a stationary device as described by Remple (1993). The initial incision should include all devitalised or ischaemic areas of the plantar aspect of the foot. The direction and size will take into account directions of tension and the requirement for normal functional post surgical anatomy. All fibrotic, ischaemic, necrotic, exudative and caseous material is removed. All surrounding tissues are debrided. Some authors favour the use of enzymatic-debridement following physical debridement. Prior to closure of the wound, cavities are explored between phalanges 1 and 2, between 2 and 3 and on the lateral aspect of the foot between phalanges 3 and 4 (in a position above the plantar surface, over which the jessie cannot rub). Into each of these cavities an AIPMMA bead is placed (as described by Remple and Forbes 1998). These beads will continue to release antibiotics into the local area surrounding the previously infected tissue for a period of months. These beads may typically be left in situ, although the position of their placement is such that they may be removed from the dorsal aspect of the foot, if that should prove necessary.

In Class IV and V cases, following full assessment it may be considered that first degree healing is not practical. Such a decision will be based upon the bacterial isolate and the degree of tissue damage and ischaemia. In such cases the wound edges are drawn together, (trapping AIPMMA beads inside), often using a purse string suture but are not closed. Topical applications of antibiotics, anti-inflammatories, cleansing agents (eg. proflavine) or epitheliogenic agents are used.

Following surgery it is imperative that pressure is effectively relieved from the plantar foot. In a normal stance the total bird's weight is applied to this particular area. In recent years a number of different systems have been advocated for raising the operation site off the ground. A number of dressing or resin based structures have been advised. In essence a soft (pressure sparing) dressing, which achieves weight bearing around the periphery of the foot and hence way from the ball of the foot. Such a dressing will require changing every 7-10 days. Differing structures are required for different species, for small species corn plasters are used, larger species a similar shaped dressing made of rigid foam, or a fabricated circular padded structure. Such dressings are shaped (similar to a ring doughnut) to make contact with the plantar aspect of the foot, at a point half way along the length of each toe. The latter dressings may be fashioned from rigid foam, plastic, wood or metal circle, amply padded with foam or cotton wool. If correctly applied, birds appear comfortable in these dressings. Even birds with severely affected feet no longer spend periods of time lying down to rest their feet. The bird's food does need to be cut up, as bandaged birds are unable to hold food in their talons and so cannot feed in the normal manner. Antibiosis is maintained for 14 - 21 days.

CONCLUSION

Bumblefoot has been a serious affliction of falconer's birds for thousands of years. A detailed description was made by Holy Roman Emperor Frederik II of Hohenstaufen (1194-1250) in his monumental treatise, *De Arte Venandi Cum Avibus*. The earliest reference to surgical treatment of the disease appears in *Falconry, or the Falcons Lure and Cure* published in 1615 (Cooper 1980). However despite its long recognition, the condition remains an all too common affliction of captive raptors.

REFERENCES

- Anderson JC. (1986). Staphylococcus. In : Pathogenesis of bacterial infections in animals. CF Gyles and CO Theon (eds). Iowa State University Press. Ames.
- Cooper JE (1978). Veterinary aspects of captive birds of prey. Standfast Press. Saul.
- Cooper JE, Needham JR (1976). An investigation into the prevalence of *Staphylococcus aureus* on avian feet. *Vet Rec.* 98: 172 - 174.
- Cooper JE (1980). Surgery of the foot in falcons : an historic operation. *Annals of the Royal College of Surgeons of England* ; 62 : 445 - 448.
- Glick B, Sato K, Cobenour F (1964). Comparison of the phagocytic ability of normal and bursectomized birds. *Journal of the Reticuloendothelial Society.* 1: 442-449.
- Griner LA. (1983). Pathology of zoo animals. Zoological Society of San Diego, 117.
- Harcourt - Brown N H (1994). Diseases of the Pelvic Limb of Birds of Prey. FRCVS Thesis. RCVS Library.
- Klemm KW (1993). Antibiotic bead chains. *Clinical Orthopaedic Rel Res* ; 195 : 63 -76.
- Oaks JL (1993). Falcon Staphylococcal Pododermatitis. In : Raptor Biomedicine. PT Redig, JE Cooper, D Remple, DB Hunter and T Hahn (eds). University of Minnesota Press. Minneapolis.
- Remple JD (1993). Raptor Bumblefoot: A New Treatment Technique. In : Raptor Biomedicine. PT Redig, JE Cooper, D Remple, DB Hunter and T Hahn (eds). University of Minnesota Press. Minneapolis.
- Remple JD, Adnan A, AL-Ashbal (1993). Raptor Bumblefoot : Another Look at Histopathology and Pathogenesis. In : Raptor Biomedicine. PT Redig, JE Cooper, D Remple, DB Hunter and T Hahn (eds). University of Minnesota Press. Minneapolis.
- Remple JD, Forbes NA (1998). Antibiotic-Impregnated Polymethylmethacrylate Beads in the Treatment of Bumblefoot in Raptors. In : Raptor Biomedicine II.
- Satterfield WC, O'Rourke KI (1980). Immunological considerations in the management of bumblefoot. In: Recent Advances in the Study of Raptor Diseases. JE Cooper, AG Greenwood, (eds). Chiron Publications. Keighley, England
- Tobias KMS, Schneider RK, Besser TE (1996). Use of antimicrobial-impregnated polymethylmethacrylate. *Journal American Veterinary Medical Association* ; 208 (6) : 841 - 845.
- Turk M, Stamm W (1970). Staphylococcal Infections. In : Harrison's principles of internal medicine, 9th ed., Fudenberg et al (eds). Lange Medical Publications. Los Altos, California.
- Wheler CL, Machin KL, Lew IJ (1996). Use of antibiotic-impregnated polymethylmethacrylate beads in the treatment of chronic osteomyelitis and cellulitis in a juvenile bald eagle (*Haliaeetus leucocephalus*). In: Proceedings of the AAV. Tampa, Florida: Annual Conference of the Association of Avian Veterinarians, 1996: 187-194.