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CLINICAL APPROACH TO ALOPECIA IN DOGS – WILL THE HAIR GROW BACK?

Peter Hill, BVSc, PhD, DVD, DipACVD, MRCVS
The Royal (Dick) School of Veterinary Studies
The University of Edinburgh, Scotland

Let's face it. When it comes to skin cases, you spend most of your time dealing with itchy dogs, lumps and ear infections. In comparison, hair loss is not in the same league, especially when you forget about hair that is simply ripped out by scratching, chewing or licking. This talk is not concerned with those cases because they were sorted out in the previous lecture. This manuscript deals with non-pruritic, spontaneous hair loss. Although not necessarily every day stuff, alopecia is irritatingly common enough to tax the brain on many an evening clinic.

Before we start, let's make sure that everyone is up to speed on terminology. Hair comes from follicles which may be in the anagen phase (period of active growth) or telogen phase (resting period). Normally, this process is nicely balanced so that the animal is covered in hair all of the time. Very rarely, animals can end up with excessive amounts of hair. However, it is much more likely that when this process goes wrong, they end up with alopecia which means loss of hair in any amount up to complete baldness. It may be partial, in which the hair density is merely reduced, or total, in which the area of affected skin is devoid of hair.

Hypotrichosis means less than the normal amount of hair. Because it means the same as partial alopecia, I don't see any point in using it. Defluxion and effluvium refer to a sudden and widespread loss of hair. As far as I can tell, they mean the same thing. Easy epilation refers to the ability to easily remove excessive quantities of hair with little resistance during physical examination. This implies that many hairs are in the telogen phase. This can be physiological (due to shedding) or pathological. When an animal is shedding, it is not possible to remove enough hair to leave a patch of alopecia. Hence, regardless of the amount of hair being removed, the owner can be reassured that nothing is wrong. On the other hand, if the epilation leads to the development of a bald patch, there is an underlying abnormality that is leading to spontaneous hair loss.

So, the dog is on the table and part of it is missing. An important part according to the owner because they don't like to see their pet without a beautiful and complete coat. After obtaining some history and performing a dermatological examination, the first thing to do when presented with alopecia is to determine the predominant pattern. Although many factors and disease processes can influence the hair follicle directly and can be involved in the pathogenesis of non-pruritic alopecia, the animal nearly always presents with one of three morphological patterns. These three patterns are helpful clinically because they automatically generate a list of differential diagnoses (Table 1).

Table 1 - Causes of non-pruritic alopecia in dogs

Localised alopecia (a single patch of alopecia)	Multifocal alopecia (appears as patchy, approx. circular areas of alopecia scattered over the body. Coat looks "moth-eaten")	Symmetrical or diffuse alopecia
Demodicosis Dermatophytosis Scars Steroid injection site Medication application site Post-clipping alopecia Alopecia areata	Demodicosis Staphylococcal pyoderma Dermatophytosis Sebaceous adenitis Dermatomyositis	Demodicosis Dermatophytosis Hypothyroidism Hyperadrenocorticism Gonadal sex hormone alopecia Pituitary dwarfism GH/adrenal sex hormone alopecia/Alopecia X Cyclic flank alopecia Follicular dysplasia Colour dilution alopecia Black hair follicular dysplasia Pattern baldness Congenital alopecia Telogen / anagen defluxion Sebaceous adenitis Epitheliotropic lymphoma Post-clipping alopecia Paraneoplastic alopecia

To prioritise these differential diagnoses, careful assessment of the skin and coat can provide further clues. The hairs around the areas of alopecia should be carefully assessed to see if they are easily epilated (endocrine disease) or if they are broken off at the skin surface (dermatophytosis). Comedones may be seen with demodicosis, hypothyroidism and hyperadrenocorticism but are uncommon with the other conditions. Follicular casts are a common feature of sebaceous adenitis. The skin should also be carefully examined for the presence of other lesions such as papules or epidermal collarettes which can indicate the presence of staphylococcal pyoderma. Non-pruritic, diffuse erythema in addition to alopecia can be a hallmark of epitheliotropic lymphoma. If secondary hyperpigmentation is present, a diffuse blackening of the skin may suggest endocrine disease whereas a slate-grey pigmentation is more suggestive of demodicosis. The skin thickness should also be assessed because it can appear atrophic in hyperadrenocorticism and alopecia X. The colour of the coat should also be noted as this is important in colour dilution alopecia and black hair follicular dysplasia. In some cases, the history and clinical signs are so suggestive of a particular condition that it may be possible to make a diagnosis without any further tests (medication site application, injection site reaction, pituitary dwarfism, cyclic flank alopecia, black hair follicular dysplasia, congenital alopecia).

Unfortunately, even the most stunning clinician is not able to make a definitive diagnosis of all cases of alopecia just by looking at them. Even though an astute dermatological examination will allow some of them to be sorted out, in many cases we are still left with a list of dreaded differential diagnoses. Figures 1, 2 and 3 provide a basic diagnostic approach for the three major patterns of alopecia that should allow a definitive diagnosis to be made in all cases.

Two tests that are particularly useful in the investigation of alopecia are the trichogram and skin biopsy. The trichogram can provide diagnostic information in cases of demodicosis (mites seen around the hair bulb), dermatophytosis (spores and hyphae seen on the hair shaft), endocrine alopecias (all the bulbs are in telogen), colour dilution alopecia (macromelanosomes in the hair shaft) and sebaceous adenitis (follicular casts seen around the hair shaft). Skin biopsy is also very helpful in determining the cause of alopecia. It can characterise the pathological changes that are occurring in and around the hair follicle and provide useful prognostic information. However, in order to get valuable information, clinicians must provide biopsies from affected and non-affected areas and ensure that the samples are correctly oriented so that the pathologist sections the biopsy in the direction of hair growth. This can be achieved by marking a line either on the skin itself, or on a piece of cardboard that the biopsy has been attached to.

After a diagnosis has been made, the most important considerations are the prognosis and treatment options. Knowing the underlying mechanisms that lead to hair loss in these various diseases may appear to be an esoteric exercise. However, a tiny bit of pathological and physiological knowledge provides one vital piece of clinical information that the client will be desperate to know – can and will the hair grow back?

REVERSIBLE ALOPECIAS

- In demodicosis, the mites multiply within the follicle, stimulate follicular hyperkeratosis and folliculitis, and force the hair shaft out. This process is reversible as long as it is reversed before large numbers of hair follicles have ruptured leading to furunculosis and scarring.
- Superficial staphylococcal folliculitis leads to hair shedding secondary to neutrophilic exudation and hyperkeratosis of the infundibular portion of the hair follicle. As with demodicosis, the process is reversible as long as the infection doesn't burst out of the follicles leading to scarring furunculosis.
- In dermatophytosis, fungal hyphae invade the hair shaft resulting in fracture and breakage. Once the infection has been eliminated, the anagen bulbs beneath just produce new hair.
- In endocrine diseases including hypothyroidism, hyperadrenocorticism, gonadal sex hormone abnormalities and GH/adrenal sex hormone abnormalities (alopecia X), the alopecia is caused by interruption of the normal hair growth cycle. Hairs are arrested in the telogen phase and are eventually shed without being replaced. A new anagen hair will not regrow until the underlying hormonal abnormality has been corrected. Post-clipping alopecia is likely to be due to clipping of hair when all the hairs are synchronised in the telogen phase and it usually resolves following subsequent growth phases.
- In sebaceous adenitis, destruction of the sebaceous glands leads to severe follicular hyperkeratosis and follicular malfunction. Early control of the disease is required in order to prevent a degree of permanent partial alopecia.
- Stressful events such as high fever, pregnancy/lactation, shock, severe illness, surgery or anaesthesia can cause abrupt cessation of growth in anagen hair follicles, resulting in many hair follicles being synchronised in the telogen phase. Two to three months later, a new wave of follicular activity begins and all the telogen hairs are shed leading to a temporary alopecia. This phenomenon is known as telogen defluxion. A similar phenomenon occurs when cases of endocrine disease are started on treatment. The new wave of hair growth pushes out all the telogen hairs which initially makes the animal look worse. Don't panic – the dog will get better after it has got worse.
- Certain drugs (cyclophosphamide, cytotoxic agents) can have an anti-mitotic effect on the hair bulb. The weakened hair shaft then breaks off below the surface resulting in a phenomenon known as anagen defluxion. This only occurs in dogs with an anagen dominated hair coat. How do you know if a dog has an anagen dominated hair coat? Well, if it needs to be clipped every 6 – 8 weeks, then it has (Poodles, Bichon frises etc). When the chemotherapy stops, the hair will regrow at the next cycle.
- Although alopecia may be seen with a number of autoimmune diseases, it is not the predominant sign. The only skin disease that results directly from an immune-mediated assault on the hair follicles is alopecia areata. In this disease, activated lymphocytes attack certain components of the hair follicle and bulb leading

to loss of the hair shaft. This is the usual cause of sudden onset baldness in man. Although the condition can be reversible in dogs, it may be permanent.

IRREVERSIBLE ALOPECIAS

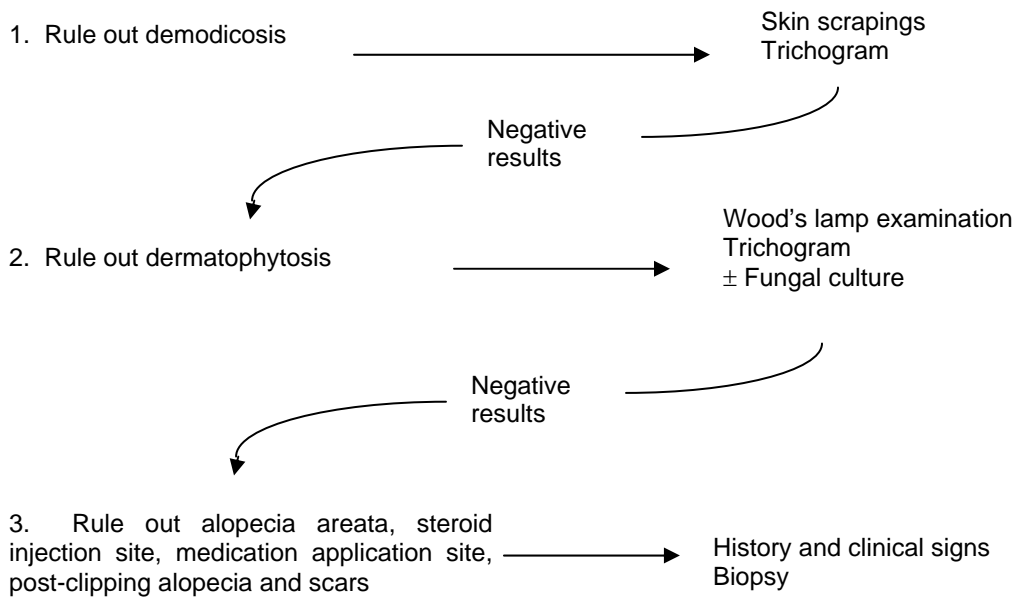
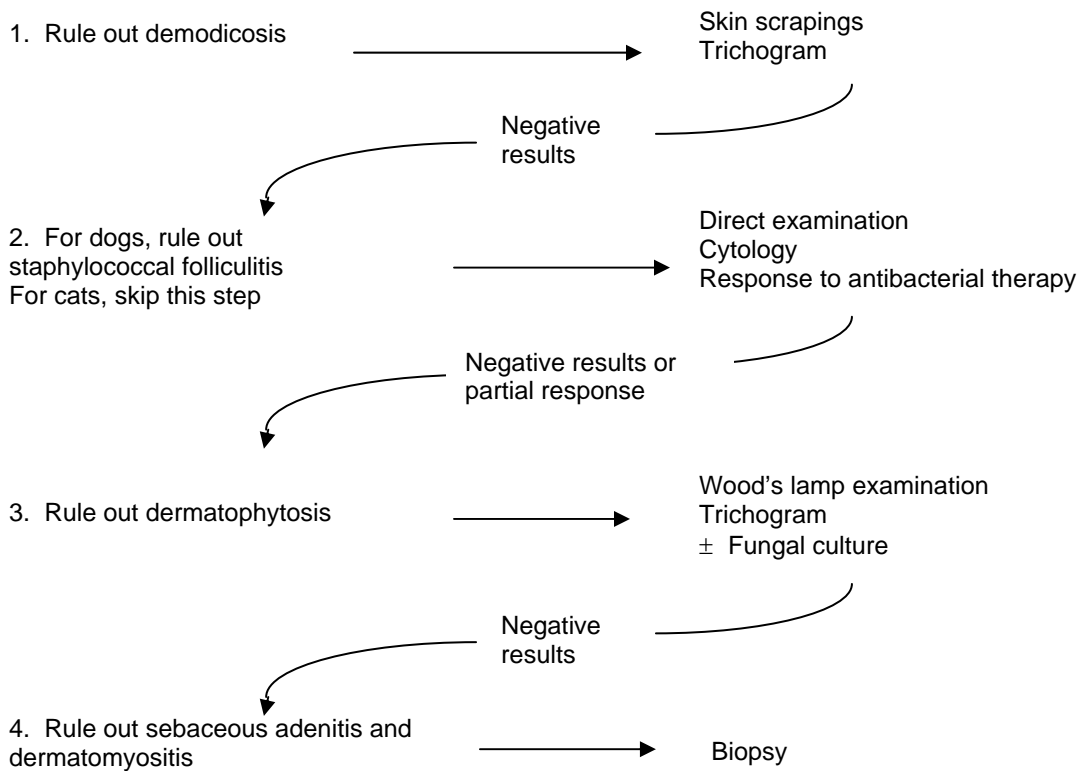
- Hairless breeds, congenital alopecia, follicular dysplasias and colour dilution alopecia all represent genetically programmed alopecias that are presently irreversible. The defect can range from dysplastic hair follicles to total absence of hair follicles.
- Deep bacterial infection (furunculosis), purulent demodicosis, burns, radiation therapy and ischaemia can cause necrotising folliculitis that results in permanent destruction of hair follicles and scarring.
- Dermatomyositis of Collies and Shetland Sheepdogs is thought to be an immune-mediated disease that leads to follicular atrophy and perifollicular fibrosis following vascular insufficiency. In many cases, the alopecia that results is permanent because of complete lack of hair follicles.
- Cutaneous neoplasms that involve the dermis can destroy hair follicles. Cutaneous T-cell lymphoma (mycosis fungoides) can cause generalised alopecia as the follicular epithelium and epidermis is invaded by neoplastic lymphocytes.

What are you going to tell your clients when you are faced with a group of conditions in which some are curable and some are not, some cure themselves whereas others require treatment, and some reflect serious systemic medical problems whereas others are perfectly harmless and the cure is worse than the disease. First, it is important to give the client an accurate prognosis from the outset. The information in Table 2 should help you to navigate through this minefield. Secondly, stick to the adage "Vets advise – clients decide." And lastly, remember that bald isn't always bad.

Table 2 – The prognosis and treatment options for alopecic conditions

Prognosis	Diseases	Treatment options
Hair will come back spontaneously	Post-clipping alopecia	None required
	Telogen defluxion	None required
	Anagen defluxion (as long as insult or disease is removed)	None required
	Juvenile onset demodicosis	95% resolve without treatment
Hair may come back spontaneously but alopecia can become permanent. Treatment has been reported but: <ul style="list-style-type: none"> ➤ Treatment is for cosmetic, not medical, reasons ➤ It may be associated with adverse effects ➤ It may not be effective 	Cyclic flank alopecia	Melatonin
	Alopecia areata	Topical or systemic glucocorticoids
Hair will not come back without treatment. However: <ul style="list-style-type: none"> ➤ Treatment is for cosmetic, not medical, reasons ➤ It may be associated with adverse effects ➤ It may not be effective 	Adrenal sex hormone alopecia	Castration Trilostane Mitotane Testosterone Melatonin
	Testosterone responsive alopecia	Testosterone
	Pattern baldness	Melatonin
Hair may come back with medical treatment. However, the prognosis for full regrowth and a normal coat is poor to fair.	Dermatomyositis	Prednisolone Pentoxifylline
	Pituitary dwarfism	Progestogens Thyroxine
	Sebaceous adenitis	Topical therapy Retinoids
	Paraneoplastic alopecia	Surgical removal of pancreatic tumour. Recurrence is likely due to metastasis
Hair will come back with successful treatment. The prognosis for full regrowth and a normal coat is fair to good.	Demodicosis	Amitraz Ivermectin
	Dermatophytosis	Griseofulvin Itraconazole Malaseb shampoo
	Hypothyroidism	Thyroxine
	Hyperadrenocorticism	Various
	Testicular and ovarian neoplasia	Gonadectomy
Incurable – hair will not grow back	Congenital alopecia	None
	Follicular dysplasia	None
	Colour dilution alopecia	None
	Black hair follicular dysplasia	None
	Epitheliotropic lymphoma	Palliative treatment for other manifestations
	Scars	None Surgical removal

Small Animal - Dermatology

**Figure 1** - General diagnostic approach to localised patches of alopecia**Figure 2** – General diagnostic approach to multi-focal patches of alopecia

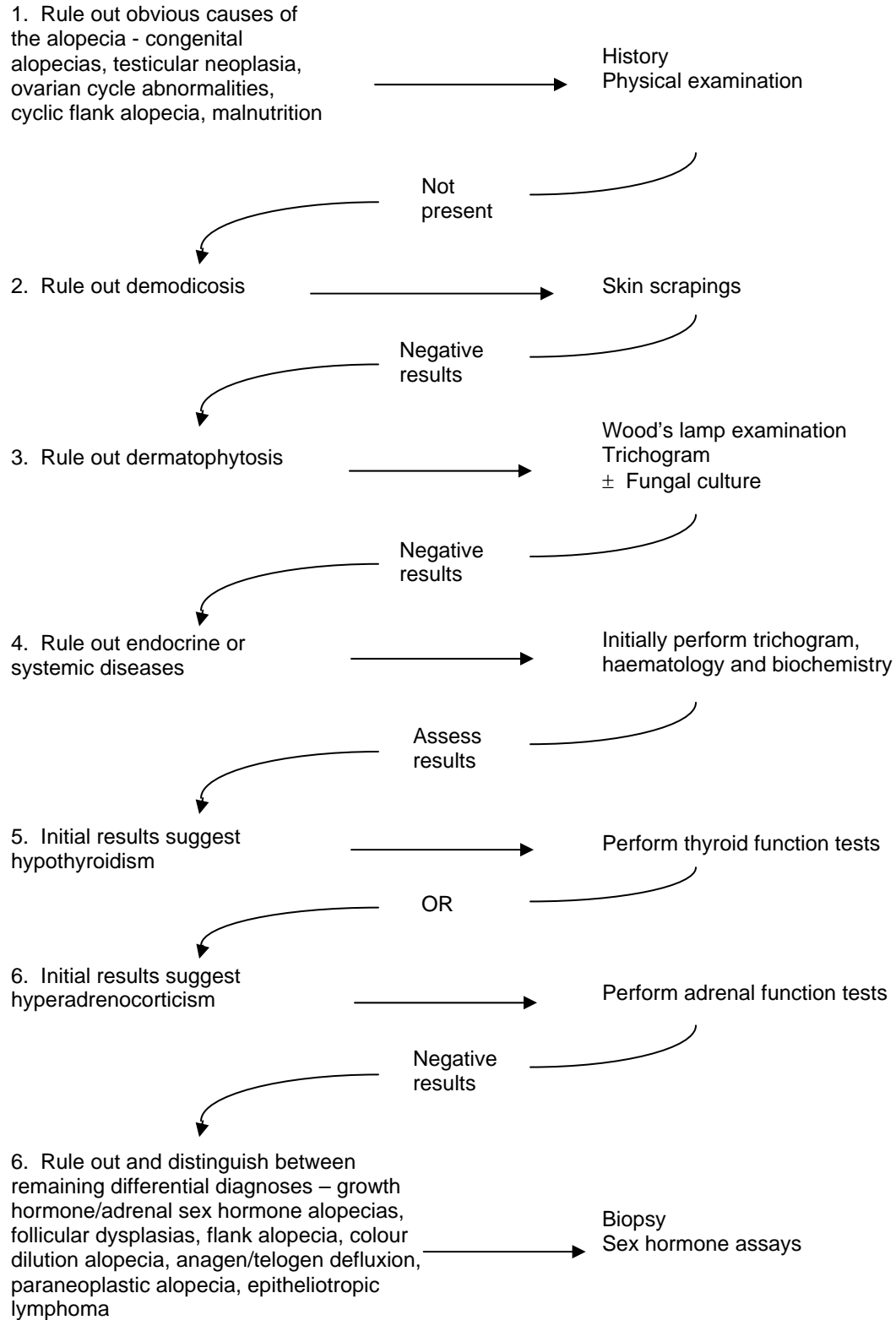


Figure 3 – General diagnostic approach to symmetrical or diffuse alopecia