APPRAOCH AND MANAGEMENT OF HAIR LOSS –
AN UPDATE FOR THE FAMILY PHYSICIAN

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ABSTRACT

Hair loss is a common problem and can result in deleterious effects on self-esteem. With a thorough history, which includes addressing the patient's concerns and expectations, and directed physical examination, most causes of hair loss can be diagnosed. Laboratory investigations are used when needed to further refine the diagnosis. Androgenetic alopecia is the commonest cause of hair loss in males and females. Pharmacological and non-pharmacological methods are available to treat androgenetic alopecia. Other causes of hair loss include alopecia areata, telogen effluvium. Scarring alopecias are trichologic emergencies and should be promptly referred to the dermatologist for further management.

Keywords

Hair loss, alopecia, approach, management, androgenetic alopecia, alopecia areata

INTRODUCTION

Hair loss (Alopecia) is a common problem. Human society has placed a high importance on hair, which has been called our “crowning glory”. It is not surprising that affected individuals perceive hair loss with embarrassment and resultant deleterious effects on self-esteem, psychological wellbeing, and body image. It is therefore important that, as part of the management of hair loss to explore the psychosocial impact on the patient who sees the doctor.

TREATMENT

A thorough history and a directed physical examination of the body and the hair of the patient will allow the cause of hair loss to be made most of the time. The commonest causes of non-scarring alopecia are androgenetic balding, telogen effluvium and alopecia areata. Scarring alopecia is commonly due to trauma, infections or systemic illnesses such as lupus erythematosus. Rare causes include congenital defects of the hair shaft.

Laboratory investigations are employed where indicated to help in defining the final diagnosis.

HISTORY

Start by asking the pattern and duration of the hair loss. Ask the patient if hair is thinning (diffuse hair loss) or hair is lost in patches. Androgenetic alopecia (male pattern balding) typically presents with thinning of the hair, which starts as a ‘M’ shaped pattern of hair loss as described by Hamilton. As this is hereditary, this problem may also be present in another member of the family. Patchy hair loss is seen in alopecia areata or telogen effluvium. Telogen effluvium is often preceded by events such as high fever, childbirth, surgery, drugs or other stressors.

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Next, ask if hair is lost in its total length with the roots or hair is lost in broken pieces. The latter is usually seen in hair infections or trichotillomania. It is also important to ask the patient if hair is lost from other parts of the body: Axillae, groin, pubic area, eyelashes, eyebrows and body hair. Conditions such as alopecia areata and trichotillomania can result in hair loss in any hair bearing area.

In the systemic review, a history of medication use (such as cyclophosphamide, warfarin, carbimazole, colchicine, lithium, oral contraceptives, retinoids etc) should be elicited. Endocrine problems such as underactivity of the thyroid, pituitary or adrenals can cause diffuse alopecia. A diet history is helpful as nutritional factors such as iron deficiency can also cause hair loss. Females who have male-pattern balding, menstrual problems and acne problems may have polycystic ovaries (PCO). Cancer patients undergoing chemotherapy may also present with hair loss.

Practices such as hairdressing, hair dyes, hair treatments and hair maintenance habits should be asked as some of these procedures can also result in some form of hair loss.

The impact of the hair loss on the psychosocial wellbeing of the patient should also be explored. Further exploration may be needed if the doctor suspects presence psychiatric problems such anxiety or depression.

**Physical Examination**

There are three parts to the physical examination.

i) Pattern and extent of hair loss

ii) Pull test and gross examination of the hair

iii) Systemic examination

Firstly, examine the scalp and note the pattern and extent of the hair loss. Determine if it is generalised or localised. Generalised diffuse hair loss suggests androgenetic alopecia. The early stages of androgenetic alopecia in males presents with bi-temporal recession and thinning of the vertex. In female pattern balding, the hair loss is usually diffuse. The frontal hairline is maintained. Patchy and localized hair loss suggests alopecia areata. Other hair conditions such as infections (Tinea) and trichotillomania also present with patchy hair loss. In systemic illnesses such as lupus erythematosus, scarring of the scalp is also seen. A localised, ‘moth eaten’ loss pattern suggests syphilis. Look out for other clues such as rashes or a relevant history.

Next look for follicular openings in the areas of balding. Follicular openings are visible in non-scarring alopecia. In scarring alopecia, the follicular openings are not seen.

Perform the “pull test” which is an easy technique for assessing active hair loss. The patient should not shampoo the hair at least 1 day prior to the test. Approximately 60 hairs are grasped between the thumb and the index and middle fingers. The hairs are then gently but firmly pulled. If 6 or less hairs are pulled, this constitutes a negative test and is normal. A positive test (more than six hairs obtained) indicates a process of active hair shedding.

The hair is examined grossly noting the characteristics such as length, diameter, colour, shape and whether the hair is fragile. Observe for hair that is intact along the whole length or broken. Hair loss due to alopecia areata has an abnormal “exclamation mark”. The use of a microscope/magnifying glass will help in gross morphological examination.

The rest of the systemic examination can be performed to screen for systemic causes as mentioned above in particular looking out for nutritional deficiencies, endocrine problems or other medical conditions.

**Laboratory Investigations**

Blood tests are usually not required for the commonest causes of hair loss diagnosed with history and physical examination alone. However, when indicated selected laboratory blood investigations (hormonal profiles, connective tissue screening, VDRL etc), microbiological examination (such as KOH preparation), special examination techniques (Wood's lamp) and punch biopsy of the scalp may be done.

Cicatricial (scarring) alopecias may be difficult to differentiate clinically from each other and almost always requires a scalp biopsy. In a study on 112 cases of primary cicatricial alopecias, the authors recommend that a scalp biopsy is mandatory in all cases with some cases requiring multiple biopsies to reach a definitive diagnosis.

**MANAGEMENT OF SOME COMMON HAIR LOSS CONDITIONS**

**Initial Considerations**

The patient’s ideas, concerns and expectations should be addressed right from the beginning in the management of hair loss problems. Many patients with hair loss problems express frustration when their worries are either ignored or deemed insignificant.

The doctor should explain and discuss the possible diagnoses including prognosis, progression of the condition and the need for further investigations or referral to a dermatologist if necessary. Proper communication could resolve problems without specific intervention. Underlying problems such as depression and anxiety that may occur as a result of the hair condition should be recognised and managed appropriately even before further treatment.

**Androgenetic Alopecia: Male Pattern Balding**

Male pattern balding or androgenetic alopecia is androgen-dependent and has a familial inheritance. Affected individuals have increased susceptibility to the influence of androgen, dihydrotestosterone, which is produced as a result of reduction of testosterone by 5-alpha reductase. The androgen binds to receptors on the hair follicles and activates genes that shorten the anagen phase. This gradually converts normal long, pigmented thick terminal hairs to fine short, non-pigmented vellus hairs with each successive hair growth cycle. This condition affects males from the second decade of life onwards...
and by the seventh decade, about 80% of males are affected. Alopecia that starts in the late teens has a strong familial association and is rapidly progressive. This heralds a poor outcome as these patients lose most of their hair on their scalp by their mid-twenties.

In males, the hair loss starts with a receding frontal hairline. This proceeds to bitemporal recession followed by diffuse thinning at the vertex. Finally what remains is a semi-circular arrangement of hair that runs from the ears bilaterally to the occiput. Norwood Hamilton's classification (Table 2) uses a scale of 1 to 7 to grade the degree of baldness.

**Table 2. Hamilton's Scale**

- **Type I:** M Type frontal hair loss (also most commonly seen in women).
- **Type II:** Contiguous frontal and vertex baldness.
- **Type III-IV:** Vertex hair loss.
- **Type V:** Vertex hair loss.
- **Type VI:** Hair loss begins at the vertex.
- **Type VII:** Vertex hair loss.

Females who have male-pattern balding (androgenetic alopecia), menstrual problems and acne problems may have polycystic ovaries (PCO). A recent study confirmed an association between androgenetic alopecia and PCO. In the study 67% (versus 27% in the control) of women presenting with androgenetic alopecia were found to have PCO. Furthermore, biochemical investigation revealed indices of androgen metabolism typical of women with polycystic ovary syndrome (PCOS). This has important implications, as PCOS is a risk factor for developing type 2 diabetes mellitus. Further hair investigations may not be necessary if the female patient has no hirsutism, no acne and has normal menses, fertility and endocrine function.

In women, the hair loss is diffuse, but more marked in the frontoparietal scalp with relative preservation of the frontal hairline. The analogous grading system is the Ludwig classification (Table 3).

**Table 3. Ludwig Classification**

- **Stage I:** Thinning is minimal. Increased frontal part width compared to occipital part width.
- **Stage II:** More advanced frontoparietal thinning.
- **Stage III:** Obvious thinning or near baldness of the area while hairline remains intact.

**TREATMENT**

This is divided into pharmacological and non-pharmacological which also includes surgical methods.

**Pharmacological Treatment - Medications**

The effectiveness of medications depends on the extent of the loss and individual response. There are two types of drugs currently approved for treatment of alopecia. No studies have directly compared the use of minoxidil with finasteride in patients with hair loss. The is only one case report describing combination use of both agents.

**Minoxidil (Regaine)**

The use of oral minoxidil resulted in widespread hirsutism as a side effect. It was this observation that lead to the development of topical minoxidil for an intended localized effect on hair growth. The mechanism of action of minoxidil appears to be a direct effect on the hair follicles, increasing proliferation and differentiation of epithelial cells in the hair shaft. Minoxidil is a liquid that is rubbed onto the scalp twice daily to re-grow hair and to prevent further loss. Minoxidil is available in a two percent solution (available as over-the-counter medication) and in a five percent solution, which requires a doctor's prescription. Minoxidil is approved for the treatment of androgenetic alopecia (for both males and females) and alopecia areata. Topical minoxidil five percent seems to give better results than two percent lotion. Price et al in a study in 1996 showed that topical minoxidil five percent (compared to minoxidil two percent) resulted in a greater hair mass with the difference being greatest in the early part of the study. A study involving female patients showed that topical minoxidil solution was significantly more effective than placebo in the treatment of androgenetic alopecia.

Using topical minoxidil about 10% of men can regrow cosmetically acceptable terminal hair. In women about 50% show minimal regrowth and 13% moderate regrowth. New hair resulting from minoxidil use is often thinner and lighter in color than previous hair. It is not useful for frontal hair loss. The recommended treatment period is about 6 months. If no significant results are achieved, other methods of treatment is advised. Side effects include local problems on the scalp such as irritation and dryness.

**Finasteride (Propecia, Proscar)**

Finasteride is an inhibitor of 5-alpha reductase that converts testosterone into dihydrotestosterone (DHT). This is a prescription only oral medication to treat male pattern baldness and is only approved for male patients with androgenetic alopecia. It is given at a once daily dose of 1mg. Finasteride should not be used in women of childbearing age, because 5-alpha reductase inhibitors may cause abnormalities of the external genitalia in the male foetus. In randomised studies, finasteride has not been useful in post-menopausal women with androgenetic pattern hair loss.

Further hair loss is prevented in most patients treated with finasteride. About 66% had regrowth and 83% showed stabilisation after a 2-year follow up in a doubled-blind, placebo-controlled clinical trial involving 1553 men. Other studies done so far showed that the therapeutic benefit is greatest for hair loss at the vertex. However there is some statistically significant improvement of hair counts in the fronto-temporal scalp area.

Regrowth of hair occurs after 6 to 12 months of treatment and benefits are maintained as long as treatment is continued. The most common side effects are diminished sex drive, erectile dysfunction and decreased ejaculate volume. These effects are reversible and tend to be less of a problem over time if the patient continues taking the drug. Like Minoxidil,
the benefits of hair growth stop with cessation of the drug. Intended effects are seen after about four months of therapy.

New medications/therapy to treat androgenetic alopecia include smaller and more flexible grafts, laser techniques and new surgical techniques. The following describes some of the established surgical techniques to restore hair loss.

**Non-Pharmacological Methods**

The lay-person is presented with a bewildering variety of commercialized hair restoration programs. However, many of these programs/treatment centers falsely justify their claims. The doctor should caution patients about such treatments as it may be ineffective and costly. Although many of these programs use methods that are generally safe for external use, they have not been shown to conclusively promote hair growth or prevent hair loss. These programs employ ineffective methods such as scalp massage, dietary modification, medicated shampoos, Chinese herbs and extracts, and electrical stimulation.

**Surgical Methods**

With advances in dermatological surgery, newer, better materials and techniques have been developed. Examples include smaller and more flexible grafts, laser techniques, and new surgical techniques. The following describes some of the established surgical techniques to restore hair loss.

i) Hair transplants

Parts of the hair-bearing scalp (usually the back and the sides of the head) are removed surgically. They are divided into small sections called minimicrografts (section of scalp with 1-3 complete hair follicles, removed under stereomicroscope) and are carefully inserted into small holes or slits that are made in the balding scalp. These minimicrografts or follicular implants have been greatly improved over the past decade and are now widely used. When done well, hair transplants often give a good natural look.

ii) Skin Flaps

A "flap" of hair-bearing scalp area is created surgically and then rotated onto the bald areas. This is limited by the arrangement and distribution of the areas with and without hair.

iii) Scalp reduction

The bald areas are excised and the surrounding areas are brought together and attached surgically to reduce the areas of balding.

iv) Wigs and hair styling/hair weaving

For those who do not fancy the idea of going under the blade, wigs are a viable option. The use of wigs can be versatile and customised. It can be worn on top of the scalp or interwoven onto existing hair. Certain ways for hair styling can also give the illusion of a greater body of hair. Artificial hair can be weaved or glued onto existing hair to increase hair mass.

**Telogen Effluvium**

Telogen effluvium is diffuse hair loss that results from an abrupt shift of hair follicles from normal anagen phase to telogen. Excessive generalised shedding of normal telogen hair can be induced by several stresses (Table 4). The hair loss does not occur 2 to 4 months after the initial stressor as the hair is shed about 2 to 4 months after entering telogen phase. Acute telogen results from such triggers and will usually remit within 12 months in 95% of cases. In most cases, it is important to explain to the patient the natural history of the condition to allay anxiety and to try to identify and treat the triggering factor if possible.

In chronic telogen effluvium of unknown origin, the hair loss lasts from 6 months to several years and may wax and wane.

**Table 4. Causes of Telogen Effluvium**

<table>
<thead>
<tr>
<th>Physiologic conditions</th>
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<tr>
<td>Early stages of androgenetic alopecia</td>
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<td>Physiologic effluvium of the newborn</td>
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<td>Postpartum effluvium</td>
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<th>Injury or stress</th>
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<td>Crash or liquid protein diets</td>
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<td>High fever (e.g., malaria)</td>
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<tr>
<td>Hypothyroidism and other endocrinopathies</td>
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<tr>
<td>Major surgery</td>
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<td>Severe chronic illness</td>
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<tr>
<td>Severe infection</td>
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<td>Severe psychological stress (e.g., life-threatening situations)</td>
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<tr>
<th>Drugs and other substances</th>
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<tr>
<td>Anticoagulants (especially heparin)</td>
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<td>Anticonvulsants</td>
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<td>Antikeratinizing agents (e.g., etretinate [Tegison])</td>
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<td>Antithyroid agents</td>
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<td>Heavy metals</td>
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<td>Hormones</td>
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**Alopecia Areata**

This is the result of an autoimmune process and is usually self-limiting. It has no gender predominance and is commonly seen under the age of 30.

This condition presents with sharply defined non-inflamed patches of hair loss. Exclamation point hairs are seen at the edges of hair loss, which is pathognomonic of this condition. The hair is seen to taper towards the scalp. This is a result of anagen hair, which is rapidly arrested to become telogen hair. Hair on the beard and eyebrows may be affected and there is also an associated nail pitting.

This condition affects about two percent of the population. If the condition affects large areas, occurs before puberty or is associated with atopy, these suggest a poor
prognosis. In some cases, all the scalp hair is lost (alopecia totalis) or all body hair is lost (alopecia universalis). Alopecia areata is also associated with other skin conditions such as vitiligo, atopic dermatitis or in systemic diseases such as thyroid diseases, idiopathic thrombocytopenia and diabetes mellitus.

The initial management of this condition involves also addressing how the condition has impacted the psychosocial wellbeing of the patient. In a recent study of a random sample of 32 patients with alopecia areata (patchy form), there was a high incidence of co-morbid psychiatric conditions (anxiety and mood disorders). These affected patients required further systematic psychiatric evaluation. The authors suggest that psychiatric evaluation of patient with alopecia areata is needed if an overall study of the condition is to be performed.

**Treatment**

The treatment of alopecia areata depends on the age of the patient and also the extent of the condition. If the natural history of the condition is explained properly to patient, patients can be observed for a few months avoiding active treatment in the meantime. If medical treatment is considered, the option depends on the extent of the condition.

- **Intralesional glucocorticoid** (e.g., Triamcinolone) injection is the most common therapy for limited scalp involvement to accelerate hair growth. Brow and beard areas may also be injected.

- **Topical steroids** (moderate to potent steroids such as betamethasone dipropionate) have also been used. Some dermatologists advocate the use of topical steroids in children. Topical steroids may be used in combination with other methods of treatment such as intralesional steroid injections or in combination with other topical treatment such as minoxidil or anthralin (as described below).

- **Topical minoxidil** has been evaluated in some studies involving children and adults with more than 25% hair loss due to alopecia areata. The 5% preparation is more effective. Hair growth is stimulated after 12 weeks of use with maximal response in 1 year. Treatment is usually continued until full remission of the condition.

- **Anthralin**, available as either a cream or an ointment, is a synthetic, tarry substance. It acts to irritate the scalp and is typically used to treat psoriasis. It may stimulate new hair growth for mild cases of alopecia areata. Anthralin is applied and left up to 20-60 minutes. It is then washed off well with shampoo. Because it is generally safe, it is commonly used in children. Hair growth is reported to occur within two to three months after initial usage.

- **Oral corticosteroids** may be used in more extensive conditions as determined by the dermatologist. Doses used are 1mg/kg/day for 1 month and tapered off. However, the systemic side effects of steroids is a consideration limiting its use.

- **Topical immunotherapy** with agents such as squaric acid, diphenylcyclopropenone are also used for more extensive lesions. These treatments are usually undertaken under close supervision by the dermatologists.

- **Photochemotherapy (PUVA)** has also been shown to be useful.

**Loose Anagen Hair Syndrome**

This is characterised by painless extraction of increased numbers of anagen hairs on a pull test. It is seen predominantly in children improving with age. However this condition can also be seen in adults. In affected adults, further questioning may reveal a history of being able to easily pull out tufts of hair since childhood. The hair may not grow long and show uneven ends. The hair texture may also show variation. A doubtful diagnosis of this condition, especially if diffuse alopecia areata is considered warrants further referral and scalp biopsy.

The treatment is expectant and the patient should be counselled regarding the condition.

**Trichotillomania**

In trichotillomania, the affected patient has an obsessive compulsive disorder which results in repeatedly pulling, twisting and breaking his/her own hairs. As a result what is seen is an irregular and often bizarre pattern of hair loss. Hair is broken at different lengths and some patients pull hair from the eyebrows and upper eyelashes too. Often, it is this done in response to a stressful situation. Although this disorder may occur in children and teenagers, the prognosis is worst in adults.

**Treatment**

Trichotillomania is often difficult to treat. Medications including antidepressant have been used. Patients may often need a combination for pharmacological therapy together with some form of behavioural therapy. A double-blind crossover trial comparing clomipramine with desipramine showed that clomipramine decreased compulsion intensity more than desipramine and was more partially more effective in controlling this condition.
dermatologist for further assessment and appropriate management is important. Certain hair treatment such as the use of curling irons, brush rollers can cause physical damage to the hair itself. Frequent use of chemicals can also damage hair follicles and the hair shaft.

**Infections**

Infections occasionally also cause scarring alopecia. Tinea capitis is caused by an animal fungus such as Microsporum canis. This is usually caught from a pet cat or dog. Tinea infections are characterised by irregular or well-demarcated alopecia and scaling. “Black dot” alopecia is seen when swollen hairs fracture a few millimeters from the scalp. In some patients, a cell-mediated immune response results in a boggy, inflammatory scalp mass, which is called a “kerion” In children with tinea capitis, cervical and occipital lymphadenopathy may be prominent.

Tinea capitis can be identified by the presence of branching hyphae and spores on KOH microscopy. Wood’s lamp examination can be performed. Some species of fungus causing tinea capitis fluoresces bright blue-green. However, there are other species that do not exhibit fluorescence. Fungal culture may be indicated in highly suspect cases when KOH microscopy and Wood’s lamp examinations are negative.

Differentials to consider include seborrhoeic dermatitis (no hair loss), atopic dermatitis (rashes in limbs, neck), psoriasis (nail lesions and erythematous plaques with silvery scales elsewhere), alopecia areata, trichotillomania.

**Treatment**

Tinea capitis does not respond well to topical treatment. Systemic antifungal therapy is required to penetrate the hair follicles. Oral antifungals such as Griseofulvin are effective. The recommended doses are 250 to 500mg/day for 4 to 6 weeks.

**CONCLUSION**

With a thorough history and directed physical examination, most common causes of hair loss can be identified. Hair practices that may potentially damage the hair should be discouraged.

The family doctor should also be able to diagnose and manage common causes of hair loss. If the diagnosis is in doubt, or if initial therapy fails, then a dermatological consult should be considered for further evaluation and management.

An important aspect of management also includes proper communication of medical information to the patient on the causes of his/her hair loss and the natural history. In treating conditions like androgenetic alopecia and alopecia areata, there are various treatment modalities. The doctor needs to explain the indications and limits of each treatment mode and together with the patient come up with a management plan. Unrealistic expectations on the patient’s part may have to be addressed to ensure compliance to treatment and to establish therapeutic alliance. It is useful to re-iterate that the patient’s ideas, concerns and expectations about the hair loss should be properly addressed.

**REFERENCES**

1. Hair growth on the scalp is characterized by 3 phases.
   a) The resting phase of hair growth is also known as telogen.
   b) Up to 90% of hair at any time is in telogen phase.
   c) Telogen effluvium results from a shift of follicles from telogen to anagen phase.
   d) Hair is shed at the end of anagen phase.
   e) During catagen phase, there are short periods of growth of hair follicles.

2. The following are causes of scarring alopecia
   a) Androgenetic alopecia.
   b) Trichotillomania.
   c) Alopecia areata.
   d) Secondary syphilis.
   e) Infections.

3. Androgenetic alopecia is the commonest cause of balding in the male.
   a) It will not occur in a 40-year-old male who has not lost significant amounts of hair yet.
   b) Androgenetic alopecia only affects males.
   c) It is caused by increased sensitivity to dihydrotestosterone.
   d) The pattern of hair loss usually begins with thinning at the vertex.
   e) The Hamilton scale of 1 to 7 is used to describe the degree of baldness.

4. Minoxidil and finasteride are used in the treatment of androgenetic alopecia.
   a) Topical minoxidil is indicated for the treatment of androgenetic alopecia and alopecia areata.
   b) Topical minoxidil should not be used in females who have alopecia areata.
   c) Finasteride can be given to all patients who have androgenetic alopecia.
   d) The therapeutic effect of finasteride is greatest for patients who have frontal hair loss.
   e) The benefits of hair growth is lost when finasteride is stopped after 4 months of therapy.

5. Alopecia areata is an immune mediated condition causing hair loss.
   a) It occurs usually in females.
   b) Exclamation point hairs indicate another cause of hair loss other than alopecia areata.
   c) It can be associated with nail pitting.
   d) Oral corticosteroids can be used as initial therapy in mild cases.
   e) Intralesional glucocorticosteroids can be given for lesions with limited scalp involvement.

ANSWERS: 1a - True; 1b - False; 1c - False; 1d - False; 1e - True
         2a - False; 2b - False; 2c - False; 2d - False; 2e - True
         3a - False; 3b - False; 3c - True; 3d - False; 3e - True
         4a - True; 4b - False; 4c - False; 4d - False; 4e - True
         5a - False; 5b - False; 5c - True; 5d - False; 5e - True