



# CONN'S CURRENT THERAPY



SAUNDERS  
ELSEVIER



**2006 & 2007 Online Access**

Started on 20 mg of isotretinoin along with 20 mg of prednisone for the first month. The drug also produces dry skin and mucosae, elevated triglycerides in approximately 30% of patients, and occasional muscle or joint aches. Transaminases are occasionally elevated, but investigation usually determines they are muscle derived rather than of hepatic origin. Patients who exercise vigorously are at greater risk for muscle enzyme leakage.

Much public concern focuses on depression caused by isotretinoin, but both personal experience and large studies fail to show a correlation between the drug and mental illness. Unfortunately, disproving a negative can be nearly impossible. Discussing the issue with patients and parents and agreeing to bring up any problems that arise is beneficial.

The major issue with isotretinoin is teratogenicity. The drug produces a tremendous rate of miscarriage and deformed babies, and pregnancy must be rigorously prevented while patients are undergoing treatment. Fortunately, isotretinoin is rapidly eliminated, and patients may conceive safely one full menstrual period after stopping the drug. Perhaps surprisingly, the patient most likely to become pregnant while taking isotretinoin is in her 20s or 30s. All female patients taking the drug must be either surgically sterile or use two means of contraception; one hormonal and one barrier method. A negative pregnancy test must be obtained monthly.

## Rosacea

Although usually considered along with acne, rosacea is a distinct disease. Comedo formation, the hallmark of acne, is absent. Rather, the predisposing factor seems to be vascular hyperreactivity. Patients who blush, especially the fair skinned, often develop some degree of rosacea, although the condition is not limited to the very pale and can be seen in all races if closely observed.

The mildest form of disease is the permanent malar blush. Telangiectasia may follow as may inflammatory papules and nodules. Some patients develop sebaceous overgrowth, particularly on the nose. Termed *rhinophyma*, this process is disfiguring and stigmatizing because people believe it is a sign of alcoholism.

Approximately 50% of rosacea patients also have ocular involvement. Styes, blepharitis, and corneal surface disease may result. The severity of ocular rosacea bears no relation to the severity of facial disease, and all patients should be questioned about symptoms and have their conjunctivae examined.

The pathogenesis of rosacea is a matter of great debate, and few hard facts are available. Vasodilation clearly plays a role, but what it does to promote the process is unclear. The resulting edema fluid is suggested as the cause of



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- Topical retinoids are the cornerstone of acne therapy.
- Mild disease is well treated with topical therapy (e.g., retinoid plus benzoyl peroxide or clindamycin).
- More severe inflammatory acne usually requires oral doxycycline or minocycline.
- Resistant nodular acne may be treated with isotretinoin.
- Ocular rosacea is best treated with doxycycline.

inflammation. Any food or medication that induces blushing worsens rosacea, but probably no one's rosacea has ever been treated effectively by diet alone. *P. acnes* probably plays a role in some patients' inflammatory disease, but drugs that reduce the organism without anti-inflammatory activity are not very effective. *Demodex* mites and gastrointestinal *Helicobacter* were suggested for years as having a role in rosacea, but no convincing studies exist.

No medication adequately treats the vascular phase of rosacea. The temptation to use topical corticosteroids must be avoided; it always makes the condition worse in the long run.

Inflammatory rosacea may be treated topically with azelaic acid or metronidazole creams. Benzoyl peroxide is sometimes helpful. Because vasodilation worsens disease, creams should be used that are not irritating to the patient. Oral therapy with tetracyclines, especially doxycycline and minocycline, is best for refractory, severe, or ocular rosacea. In extreme cases, isotretinoin is an appropriate last resort.

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## DISEASES OF THE HAIR

Method of  
Kimberly May Eickhorst, MD,  
and Eyal Levit, MD

The chief complaint of hair loss or disease is ubiquitous throughout all medical practices. Therefore it is important to have a general understanding of the normal physiologic hair growth cycle and how alterations in this cycle manifest as different hair diseases. Diagnosis and treatment of hair disease can at times be frustrating for both the physician and the patient because of a lack of unequivocal diagnostics and effective treatments. However, a thorough and appropriately directed history armed with a few key diagnostic techniques can help direct care to maximize treatment and patient satisfaction.



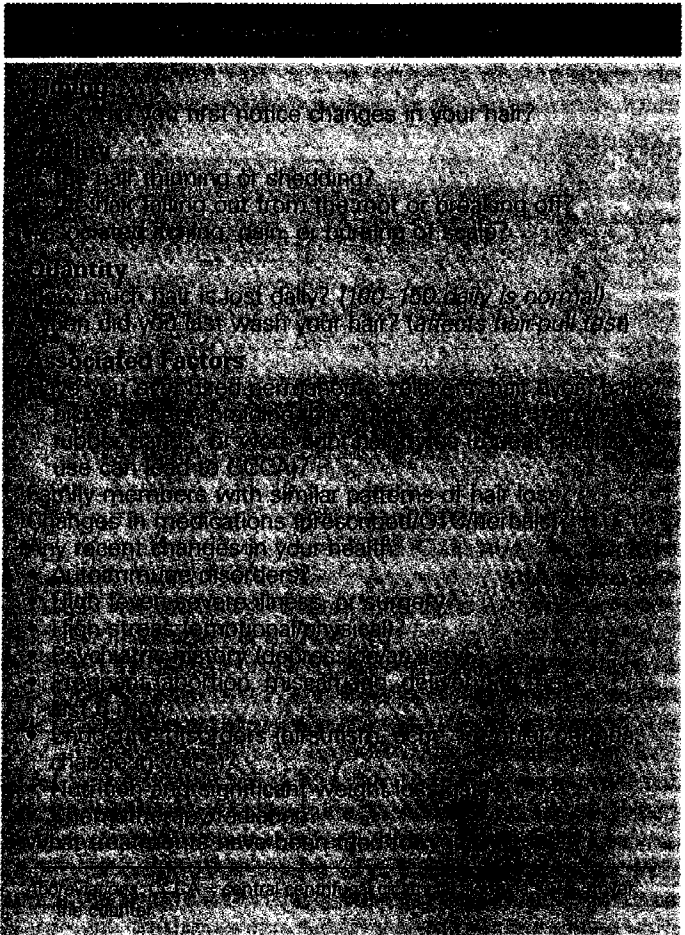
## CURRENT DIAGNOSIS

- Determine severity of acne: comedonal, papular, nodular.
- Evaluate possibility of endocrinopathy.
- Evaluate rosacea patients for ocular involvement.

A hair cycles through three stages: anagen, catagen, and telogen. The duration of each cycle varies from one body area to the other. Within each body area, each follicle cycles at a different periodicity but maintains the same growth control characteristics. However, in this chapter we concentrate on the scalp. During the first stage (anagen), the bulb or hair root is located in the subcutaneous or dermal portion of the skin and actively grows for a period of approximately 2 to 5 years. As the cycle continues and the hair matures, the hair bulb begins to progress toward the scalp surface. After transiently passing through the catagen or resting phase (2 to 4 weeks), the hair enters telogen (3 to 4 months). It is in this final stage that the hair is ultimately dislodged from the follicle and shed. At any given moment, the telogen-to-anagen ratio is 85% to 15% in females and 90% to 10% in males. Hair grows at a rate of approximately 1 cm per month. An average scalp contains approximately 100,000 hairs, with no known racial or sexual differences; this translates into a completely normal hair loss of approximately 100 to 150 hairs per day, in any given individual. Alterations in this physiologic cycle often result in different types of hair loss; inherent metabolic insults or abnormalities can result in other hair disorders.

History is critical to guiding the physician toward a correct diagnosis. Table 1 lists several questions to pose to your patient when formulating a differential diagnosis. Box 1 lists some specific diagnostic techniques.

Additionally, examine all hair-bearing areas, making note of hair quality (fine, terminal, or vellus; brittle, dry,



- **Hair-pull test:** Gather approximately 40 hairs between the fingers, and then while holding the hairs up away from the scalp under tension, slowly pull along the hair shafts until the distal ends are reached. This technique should be repeated approximately seven times in different areas of the scalp. If more than 4–6 hairs are shed during any one of the eight pulls, the test is considered positive and indicative of an effluvium.
- **Hair parting:** Make a coronal part at the vertex. Measure the width of the part. Proceed to part other areas of the scalp and compare widths among different scalp locations. A widened coronal part with retention of the frontal hairline is seen in female androgenetic alopecia.
- **Laboratory data and scalp biopsy** (Table 3):
  - **Light microscope examination of hair bulb:** aids in determining stage of hair cycle during which alopecia is occurring (Figures 1A and B).

frayed, or sharp distal ends), density, distribution, and associated skin changes (erythema/inflammation/scale/scalp follicular plugging). Remember, with the exception of the palms, soles, glans, and prepuce, hair grows on all skin surfaces. A magnifying glass and side lighting can be of great assistance. Additionally, the nails, oral mucosa, and thyroid should be closely evaluated. Certain types of hair loss are associated with distinct nail findings (i.e., alopecia areata, lichen planus and lupus can have oral lesions, and thyromegaly can indicate a thyroid disorder).

If hair loss is the complaint, a hair-pull test can be extremely instrumental. This maneuver serves to estimate the number of hairs in telogen. The test is performed by gathering approximately 40 hairs between the fingers and then while holding the hairs up away from the scalp under tension, slowly pulling along the hair shafts until the distal ends are reached. This technique should be repeated approximately seven times in different areas of the scalp and should be mildly uncomfortable to the patient if done correctly. More than six to eight hairs dislodged on any one pull indicates an increased percentage of hairs in the telogen phase and a positive test. The classic telogen hair has a clublike root. A caveat: The hair-pull test is highly subjective and strongly influenced by its relation to the shampoo/combing.

If a patient has vigorously brushed or shampooed hair prior to the visit, a large amount of telogen hairs may have already been dislodged, thus confounding the hair-pull test with increased false-negative results. If factors exist that prohibit a valid hair-pull test, the patient can be instructed to collect *all* hairs lost over a 1-day period and store them in a small sealable plastic bag. Over the course of 7 full days, each day's worth of lost hair should be counted by the patient, stored in an individual bag, labeled with the date and hair number, and brought to the physician's office. This collection should include hair lost on the pillow, in the shower, and on combs/brushes.

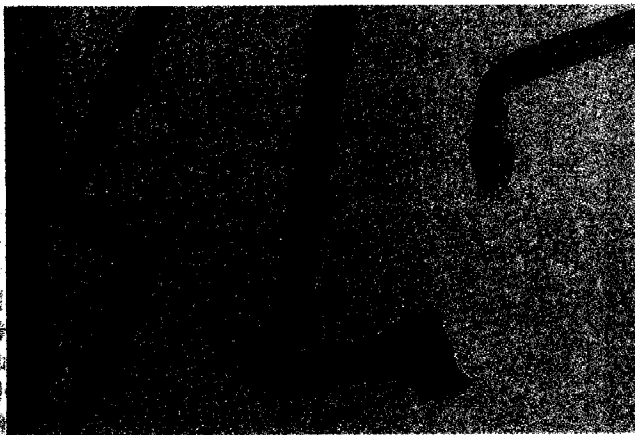
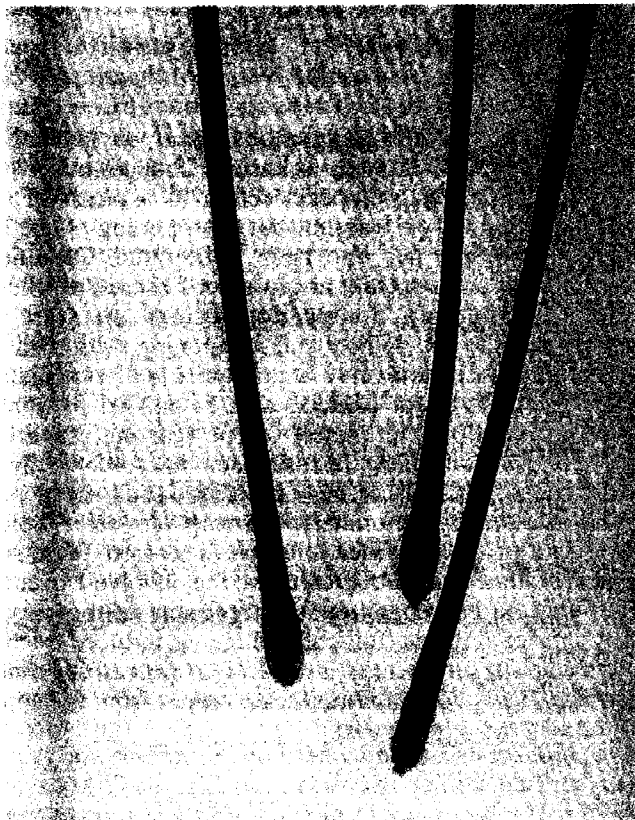
The hairs harvested from the hair-pull test can also be examined under the light microscope. The proximal ends of the hairs can be placed under a coverslip with potassium hydroxide (KOH) as background media

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FIGURE  
Hair under  
from Bol  
Alopecias.

Simply sandwiched between two glass slides and viewed under low power. The hairs can then be evaluated simultaneously for stage of cycling (anagen/telogen) and the presence of fungus. An anagen has retention of pigment at its proximal "root" as well as some remnants of root sheath, creating an irregularly shaped and glistening bulb. In contrast, a telogen hair has a more swollen, white, rounded, and cornified bulb likened to a cotton applicator tip (Figure 1). Fungus presents as hyphae or spores within or lining the hair shaft and may clinically be associated with cervical lymphadenopathy or "black dot alopecia," in which stubbles of darker hair are seen at the follicular orifices.



**FIGURE 1.** What to expect when examining a hair-pull test under the light microscope: telogen hair (A); anagen hair (B). (From Bologna JL, Jorizzo JL, Papini RP: *Dermatology*, vol 1, 1st ed. Philadelphia, PA: Saunders, 2003, p. 1035.)

The scalp should also be parted in several different locations to compare the width of the parts. Parting not only helps define and compare hair density throughout the scalp but can also be a diagnostic tool. A midline widened coronal vertex part that resembles a "Christmas tree" pattern and displays central thinning while maintaining the frontal hairline is characteristic of female androgenetic alopecia.

Scalp biopsy is usually reserved as a later step in the hair disease workup when alopecia is refractory, when suspicion is high for a scarring component, or when the patient simply desires a definitive reason, specifically proof, for his or her hair disease. Scalp biopsy entails infiltrating an area of the scalp with local anesthesia and then using a 4- to 6-mm punch biopsy to obtain a full-thickness skin specimen down to the fat, where many of the hair bulbs reside. A 6-mm biopsy or two 4-mm punches are recommended over a single smaller diameter punch biopsy. It is also suggested that two biopsies from different involved scalp sites be harvested. The specimen is then sent for both vertical and horizontal sectioning. If scarring is suspected, direct immunofluorescence testing should also be considered (Table 2). Overall, a scalp biopsy is extremely useful in definitively identifying scarring versus nonscarring alopecia and the presence and type of inflammation.

Certain laboratory data (see Table 2) can also help unravel the mystery of troubling hair disease. Equipped with a thorough history and exam, one can begin to narrow the differential diagnosis of hair disease and to test and treat the suspected malady. Following is a brief discussion of some of the more commonly encountered hair disorders, as well as suggested diagnostics and treatment.

## Alopecia

Hair loss, or alopecia, is commonly divided into scarring and nonscarring alopecia (Table 3). Variants of hair loss

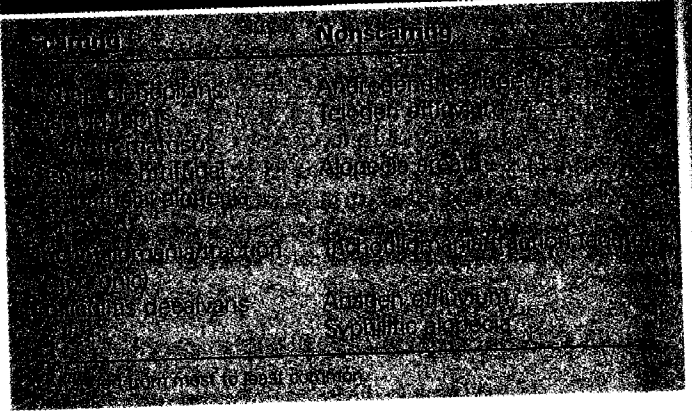
### CURRENT DIAGNOSIS

- Detailed history (Table 1)
- All hair-bearing sites should be examined using specific techniques:
  - Hair quality (dry, brittle, fine, short/long, sharp or frayed distal ends)
  - Scarring versus nonscarring; diffuse versus focal involvement description
  - Hair-pull test
  - Part width measurements and comparisons
- Appropriate laboratory data collected (Table 2)
- Two 4-mm or a single 6-mm punch biopsy of the scalp helps distinguish scarring from nonscarring alopecias:
  - The specimen should be harvested from the involved edge of the scalp where some hair is still present. The specimen should then be sent for *horizontal* and *transverse* sectioning by an experienced dermatopathologist.
  - If connective tissue disease is suspected, another 3-mm biopsy should be sent for direct immunofluorescence (DIF).

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- Start aggressive treatment early. True scarring alopecias are permanent. However, if diagnosed and treated early, scarring can be prevented.
- Regarding topical Minoxidil 5% (Rogaine):
  - Once started, it must be continued indefinitely; if stopped, all the hair gained will shed, usually within the next 4-6 mo.
  - Patients should always wash their hands after scalp treatment application to prevent accidental unwanted hair growth on the face.
  - Warn patients that they may first experience a small increase in hair loss at the very beginning of treatment as the growth of new anagen hairs replaces old telogen hairs out of the hair follicle.
- Physicians should be prepared to refer patients to reputable stylists and shops that can provide alternative natural hair styling and accoutrements.

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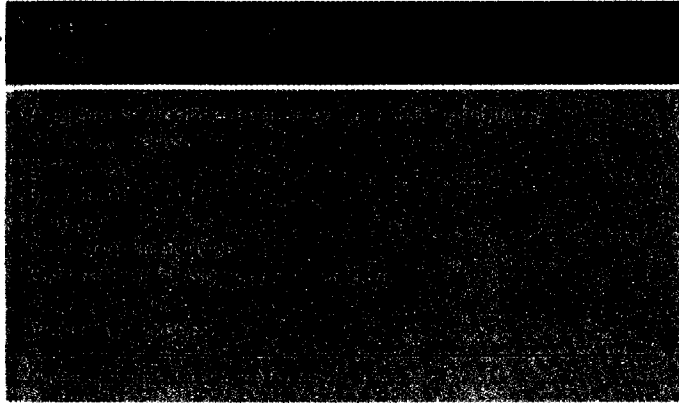


can then be further grouped by focal or generalized involvement and acute versus chronic changes in the hair. Clinically, a scarring alopecia refers to a patch of skin where hair is not only absent but the opening of the hair follicle or orifice has also been obliterated. Oftentimes scarring alopecias present as glossy or fibrosed patches of skin. Contrastingly, nonscarring lesions maintain the integrity of the hair follicle and its opening. Although this dichotomous schema may seem quite self-explanatory, even the most adept clinician can be misled by clinical examination alone; ultimately a true scarring alopecia is defined by hair follicle fibrosis seen microscopically on scalp biopsy. Most scarring alopecias result from prior inflammation. Unfortunately, scarring alopecias hold a very poor prognosis. Once a follicle is scarred, there is no hope for renewed hair growth at the involved location. Therefore early recognition and treatment of the prescarring signs of alopecia (follicular plugging, induced traction, and follicular erythema) is critical in preventing future scarring. If any doubt exists, a scalp biopsy from the appropriate location is warranted.

## Nonscarring Alopecias

### TELOGEN EFFLUVIUM

One of the most common causes of hair loss/shedding and thinning is telogen effluvium. This type of diffuse hair loss has both acute and chronic variants, but both are the result of a greater number of hairs (more than 10% to 20%) prematurely entering the telogen phase of the hair cycle. This shift in the overall number of telogen hairs can be clearly demonstrated by a positive hair-pull test, as described earlier. Telogen effluvium can result from a multitude of medical states including hormonal abnormalities (hypothyroidism, hyperthyroidism, pregnancy), nutritional disorders (anorexia, excessive weight loss, iron/zinc/biotin deficiencies), medications (Table 4) and systemic stress (high fever, surgery, systemic lupus erythematosus, dermatomyositis). In approximately 33% of cases of acute telogen effluvium, no trigger can be identified. Anxiety, depression, and other types of emotional stress are commonly blamed for causing telogen effluvium. However, little scientific



evidence exists to support the belief that everyday life stress is sufficient to induce diffuse hair loss.

Telogen effluvium is usually self-limited but can become chronic if the triggering factors are not removed. Clinically, at least 15% to 25% of scalp hairs must be lost before telogen effluvium can be objectively observed. It is important to reassure patients that although they may suffer temporarily from decreased hair density, they will not go completely bald, despite what might appear to be continued hair loss. The diagnosis of telogen effluvium is usually clinched with a positive hair-pull test and a history describing some recent (within the past 6 weeks to 4 months) physiologic/emotional stress, followed by diffuse scalp hair loss/shedding. In addition to removing any persistent causative factors, topical 2% or 5% minoxidil (Rogaine) applied twice daily to the scalp can encourage new hair growth until the distribution of hairs throughout the hair cycle returns to baseline.

### ANAGEN EFFLUVIUM

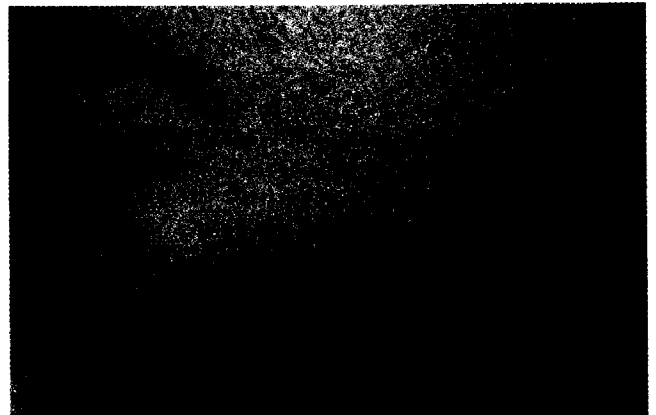
Anagen effluvium results from acute and direct insult to the nearly 90% of hairs in the initial hair growth phase. Chemotherapy, followed by radiation and poisoning (e.g., arsenic), are the more common culprits. During this toxic event, the follicle and stem cells are neither harmed nor converted to a different stage in the hair cycle. However, mitosis is inhibited. The result is a hair that is proximally weakened and narrowed. As a consequence of this proximal hair shaft weakness, the affected hairs usually break off as they approach the scalp. Anagen hair shedding may begin approximately 1 to 2 weeks following the inciting event. But hair loss may be most evident after approximately 1 to 2 months and can be clinically profound. Patients should be assured that this condition is completely reversible and once the insult to the metabolic function of the hair follicle is removed, normal hair production and growth should resume. If chemotherapy is anticipated, a prophylactic approach to anagen effluvium involves applying a pressure cuff around the scalp during chemotherapy.

### ALOPECIA AREATA

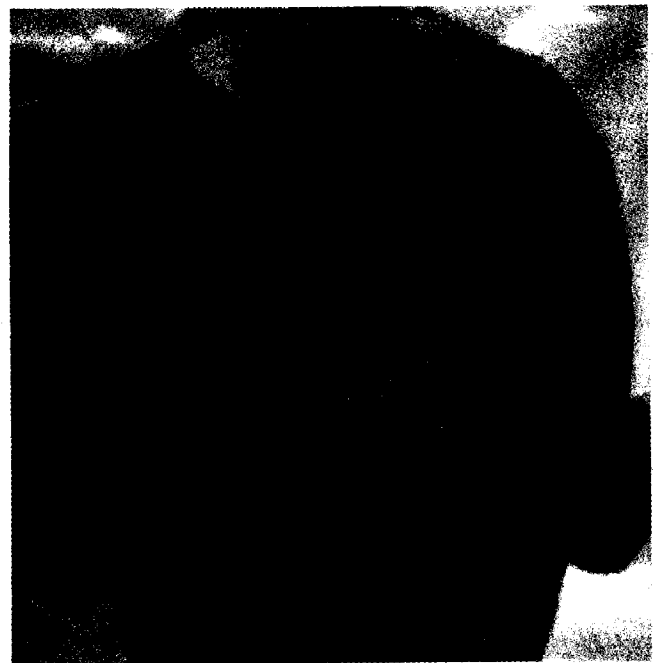
An autoimmune, cell-mediated disorder, alopecia areata may be found in association with vitiligo, thyroid disorders, lupus, atopic dermatitis, or Down's syndrome. There may also be a strong familial predominance. However, it most

commonly presents without any other disease associations. Clinically, asymptomatic or mildly pruritic ovoid patches of hair loss are seen. These patches can be small and focal (Figure 2A) or extend over large areas of skin (Figure 2B). At times there can be such diffuse focal involvement that the scalp begins to look almost "moth eaten." In this instance a rapid plasma reagent (RPR) test may be warranted to rule out syphilitic alopecia, which can take on a similar appearance.

When complete loss of scalp hair is seen as a result of alopecia areata, the condition is called *alopecia totalis*. When hair is absent on *all* hair-bearing areas of the body, the term *alopecia universalis* is used. *Ophiasis* describes alopecia areata in a bandlike distribution over the periphery of the temporal and occipital scalp, whereas the term *sisaiapho* describes the inverse, with balding of the superior scalp. At the periphery of many of these balding patches, hair may seem loose. Forcefully dislodging these hairs can



A



B

**FIGURE 2.** A, Small patches of hair loss on the chin and neck. White hairs are seen in some areas and represent signs of early hair regrowth. B, Large patch of alopecia areata on the occipital scalp with patches of regrowth at the edges.

reveal a more tapered proximal end of the hair shaft. Thus, these hairs are called *exclamation hairs*. Gridlike nail pitting is another hint to the presence of alopecia areata.

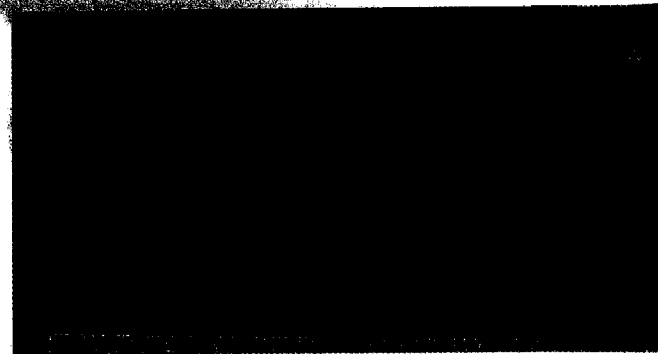
Although alopecia areata may spontaneously remit, treatment is encouraged and decreases disease duration. Intralesional triamcinolone (Kenalog) is used for localized disease, but more diffuse scalp involvement usually lends itself to topical application of high potency class I topical steroids and calcineurin inhibitors like topical pimecrolimus (Elidel) or tacrolimus (Protopic). Steroid treatment demands close follow-up to avoid hypothalamic-pituitary axis (HPA) suppression or skin atrophy signs such as hypopigmentation, thinning, and telangiectasias. If these treatments fail, the use of anthralin or squaric acid dibutyl ester can be cautiously attempted with gradual increase in contact exposure time. Refractory cases may even require PUVA (oral psoralen plus UVA light treatment) or a short course of oral steroids for response. Atopic dermatitis, childhood onset, widespread involvement, ophiasis, duration longer than 5 years, and onychodystrophy tend to predict a poor prognosis.

### ANDROGENETIC ALOPECIA

Androgenetic alopecia can occur in both males and females and is by and large linked to the presence of excess androgens that subsequently cause follicular miniaturization and loss of hair. The reduction in the size of the follicle is accompanied by shortening of the anagen phase and increased telogen shedding. In males there is clinical regression of the frontal-temporal hairline, whereas in women there is retention of the frontal hairline but widening of the coronal part and decreased hair density over the vertex. In both sexes, a similar family history of patterned hair loss is often present. Interestingly, a higher risk of coronary heart disease is associated with male-patterned baldness.

Testosterone is converted to dihydrotestosterone (DHT) by the enzyme 5- $\alpha$  reductase (type II). In males with androgenetic alopecia, 5- $\alpha$  reductase (type II) activity and DHT are increased as opposed to nonbalding scalp skin. Therefore, finasteride (Propecia), a 5- $\alpha$  reductase (type II) inhibitor, at 1 mg orally daily, can halt or slow further hair loss in men. Pregnant women should not so much as touch this drug because pregnant women handling of finasteride (Propecia) risks feminization of the fetus. Female-patterned baldness is most commonly seen in the perimenopausal stages of life, although younger and younger patients of both sexes seem to be presenting with this complaint. Although this process may begin at any age after puberty, it usually becomes clinically apparent in men by 17 years of age and in androgenetically normal women by 25 to 30 years of age.

When younger patients, especially in the face of coexistent hirsutism, present with this classic patterned hair loss, and/or females present with a male-patterned hair loss, laboratories should be drawn to assess testosterone and dehydroepiandrosterone sulfate (DHEAS) levels. Free testosterone represents the ovarian component of hyperandrogenism, whereas DHEAS levels represent androgen contribution from the adrenals. Potential treatments for female-patterned hair loss include oral contraception with relatively higher estrogen levels or antiandrogens like spironolactone (Aldactone) at 50 to 200 mg orally daily. Although many of my female patients have had success



**FIGURE 3.** Traction alopecia. Hairs of varying lengths with a well-defined border.

using Finasteride (Propecia), 1 mg orally every day, a single small study by Merck failed to show statistical benefits. Topical Minoxidil 5% (Rogaine) may also be helpful, but like finasteride, it must be continued indefinitely to maintain its effect.

### TRICHOTILLOMANIA

Trichotillomania refers to the act of forcibly pulling/plucking out one's hair, resulting in patchy or full alopecia of the scalp. The scalp is the most frequent hair-pulling site, followed by the eyebrows, eyelashes, pubic area, trunk, and extremities. This type of chronic alopecia forms with more linear, well-defined borders, which include hairs of varied lengths (Figure 3). The occiput generally tends to be spared. Although patients with obsessive-compulsive disorders and neurotic personality traits are suspect for this variant of alopecia, a scalp biopsy can confirm the diagnosis with evidence of abundant catagen hairs, retained follicular pigment, and hemorrhage. Observed or reported hair-pulling behavior from family and friends may help avoid a biopsy. Such pulling can also be caused from tightly styled hair as with ponytails or braiding. Additionally, if trichotillomania is high on the differential, as a last resort, shaving a 3 x 3 cm area of the scalp may help clinch the diagnosis. Subsequent normal hair growth would support the diagnosis; these new hairs will be too short for the patient to pull out.

Treatment is difficult, and just breaking the hair-pulling habit is key. Sometimes instructing the patient to apply any salve (e.g., olive oil) to the scalp area each night under a shower cap and wearing it to bed may break the habit. Even if treatment fails, patients are often relieved to find that others pull out hair. Organizations offering educational materials and support contacts can help. Although clomipramine is the only drug that was effective in controlled trials, other selective serotonin reuptake inhibitors (SSRIs) have anecdotally led to improvement. Additionally, behavior therapy, hypnosis, insight-oriented psychotherapy, habit modification, and close, lengthy follow-up should also be treatment considerations.

### Scarring (Cicatricial) Alopecia

Scarring alopecia represents fibrosis of the hair follicle, most commonly secondary to previous inflammation.

Discoid lupus erythematosus, lichen planopilaris, and central, centrifugal scarring alopecia are the most common forms of scarring hair loss. The most helpful methods to differentiate these subtypes are scalp biopsy, early in the process, and bacterial and/or fungal culture if active signs of superficial inflammation such as pustules or crusts are present. Early diagnosis is key. If the insulting inflammatory process can be halted before complete follicle fibrosis, there is still hope for improvement. However, if complete fibrosis occurs, the result is a burnt-out, noninflammatory, end-stage scarring alopecia termed pseudopelade of Brocq that has no recourse.

### Discoid Lupus Erythematosus

Discoid lupus erythematosus (DLE), a cutaneous form of lupus, manifests as sharply demarcated atrophic plaques with adherent scale and follicular plugging. Plaques are often circumscribed by a fine outline of hyperpigmentation (Figure 4). Key areas of involvement, in addition to the scalp, include the ear, perioral, and perinasal regions. Despite the often classic clinical appearance, a scalp biopsy for direct immunofluorescence (DIF) and H&E (hematoxylin & eosin) should be sent (Current Diagnosis box). Although DLE can progress to systemic lupus in approximately 5% of individuals, it is predominantly stable and can be most effectively treated with sun protection and topical and intralesional steroids. Refractory cases may also respond well to antimalarials, systemic retinoids, and methotrexate.

### LICHEN PLANOPILARIS

Four times more common in women, this entity is a follicular-based variant of lichen planus. The scalp, as well as other hair-bearing areas, can be involved (Graham Little syndrome). The hair-pull test is positive for anagen hairs. Clinically this condition begins as perifollicular erythema that then leads to hyperkeratotic and spiny follicles and eventual permanent scarring. If early in the disease process, successful treatment can include potent topical and intralesional steroids as well as antimalarials.



FIGURE 4. Discoid lupus erythematosus (DLE). Scalp showing multiple old burned-out areas and newly inflamed erythematous patches with perifollicular scale, crust, and erosions.

### CENTRAL CENTRIFUGAL CICATRICAL ALOPECIA

Largely an umbrella term for "hot comb alopecia," the "follicular degeneration syndrome," and central centrifugal scarring alopecia. Central centrifugal cicatricial alopecia (CCCA) is defined as premature desquamation of the inner root sheath eventually leading to loss of the follicular epithelium and replacement with fibrosis. Patients may be asymptomatic or complain of sensations of pruritus, pain, or pins and needles. Most commonly found in a subset of African American women, this insidious, non-inflammatory primary scarring alopecia starts in the central midline scalp and spreads centrifugally over the vertex. At times polytrichia, multiple hairs exiting one hair follicle ostia, can be observed. There is little scalp boggingness or tautness, but this type of alopecia has long been associated with the hair care regimens of certain ethnic backgrounds. However, this anecdotal association remains to be scientifically validated. If treated in the early stages, the condition can be improved with both high potency topical steroids and tetracycline (500 mg orally twice a day) and cessation of any traumatic or chemical hair care practices.

### FOLLICULITIS DECALVANS

Folliculitis decalvans, a recurrent, inflammatory process, is defined by well-circumscribed patches, along which follicular papules and pustules line the advancing margins (Figures 5A and B). If progressive, these often boggy

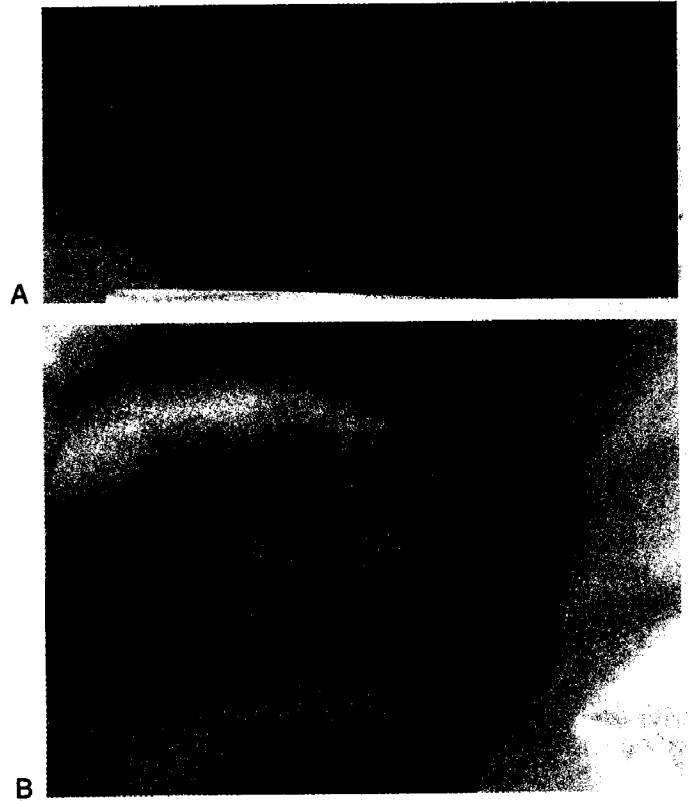


FIGURE 5. A, Folliculitis decalvans. Well-defined carbuncle on the scalp with overlying hair loss. B, Folliculitis decalvans. Well-circumscribed patches of boggy scarred scalp with few areas showing "tufted" folliculitis.

scalp areas eventually become scarred. Variants include so-called tufted folliculitis in which multiple hairs emerge from erythematous and crusted follicles resembling doll-like hair. During active disease, there is an abundance of gram-positive organisms. Hypotheses exist affirming *Staphylococcus aureus* to be the causative agent. However, the true etiology is unknown. Fungal and bacterial cultures are warranted as well as screening for potential immune deficiency. Long-term oral and topical antibiotics and/or retinoids are the mainstay of therapy.

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Method of

**Richard F. Wagner, Jr., MD**

Ultraviolet light (UVL) skin damage plays an important etiologic role in most basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). UVL interacts with a variety of factors, including host genotype, Fitzpatrick skin type, and immunocompetence, that determine whether acute (sunburn) and chronic cumulative UVL injury will cause skin cancer. Less frequent causes of skin cancer in the United States are ionizing radiation exposure, arsenic ingestion, traumatic skin injury, tobacco (SCC of lip), and chemical carcinogen. Many genetic disorders predispose patients to skin cancer through a variety of mechanisms, such as albinism, basal cell nevus syndrome, xeroderma pigmentosum, and epidermolysis bullosa. Genital SCC is almost always associated with human papilloma virus (HPV), often identified as HPV-16 or -18. HPV interacts with sunlight to result in life-threatening SCC in a rare skin disease, epidermodysplasia verruciformis.

## Clinical Features

Nodular BCC is the most common subtype of BCC (80%), and it classically manifests as a shiny papule with

visible telangiectasia. Nodular BCC may be pigmented and can be confused with nodular melanoma. Superficial BCC (15%) usually appears as an erythematous thin plaque on the trunk, with a subtle threadlike raised border. The most difficult type of primary BCC to recognize clinically is the morpheaform or sclerotic type, appearing much like a scar. Neglected BCC, although usually painless, may ulcerate ("rodent ulcer") and bleed, have a foul odor, and reach enormous size.

SCC may be difficult to distinguish clinically from BCC. The presence of keratotic scale and the absence of classic BCC morphology are the best clinical indicators for SCC. Organ transplant patients on immunosuppressive therapy are more likely to have SCC than BCC. SCC remains the most common malignancy of the mucosal lip. Bowen's disease, or SCC in situ (SCCIS), may resemble superficial BCC. When SCCIS arises on the uncircumcised mucosal penis (erythroplasia of Queyrat), it is typically moist and bright red with sharp clinical margins. SCCIS may develop into invasive SCC, and invasive SCC may also arise from actinic keratoses. Invasive SCC classically manifests as a red papule or nodule with scale. The keratoacanthoma is regarded by many dermatopathologists as a subtype of well-differentiated invasive SCC, often having a history of sudden rapid growth and a central keratotic crater. Large (2-cm diameter or greater) invasive SCCs are more likely to have perineural invasion, especially if the tumor is recurrent.

Regional lymph nodes should be examined for patients with BCC and SCCs, and a bimanual examination of the mouth is recommended for patients with SCC of the mucosal lip. Pathologic lymph nodes should be referred to a specialist for fine-needle aspiration (FNA) and imaging studies to exclude metastatic skin cancer to lymph nodes, the most frequent site of metastasis.

## Treatment Cryosurgery

Cryosurgery with liquid nitrogen, an effective treatment for BCC and SCC, relies on the destructive effect of the freeze-thaw cycle on living cells. It should be performed under the guidance of a thermocouple to ensure an adequate depth of freeze. Cold sensitivity disorders such as cryoglobulinemia are contraindications.

## Electrodesiccation and Curettage

Electrodesiccation and curettage (EDC) remains the most frequently used treatment modality for BCC and SCC in the United States. It is highly effective in selected tumors, and although variability is reported among physicians using the technique, 95% cure rates are cited for small primary BCC and SCC. It is often the fastest and least expensive treatment, and it is very effective for small BCC and SCC (5 mm or less) that arise on areas of skin that are not high tumor recurrence zones, such as on the arms, legs, and trunk. It can also be effective for superficial BCC and SCCIS, as long as the tumor does not extend down the hair follicle. EDC treatment sites heal by second intention and are usually flat and hypopigmented. In one private dermatology practice setting, there was no statistical difference in recurrence rates for primary BCC and SCC treated with



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