TABLE 72-4 CAUSES OF ALOPECIA

I. Nonscarring alopecia
- A. Primary cutaneous disorders
  1. Androgenetic alopecia
  2. Telogen effluvium
  3. Alopecia areata
  4. Tinea capitis
  5. Traumatic alopecia[a]
- B. Drugs
- C. Systemic diseases
  1. Systemic lupus erythematosus
  2. Secondary syphilis
  3. Hypothyroidism
  4. Hyperthyroidism
  5. Hypopituitarism
  6. Deficiencies of protein, biotin, zinc, and perhaps iron

II. Scarring alopecia
- A. Primary cutaneous disorders
  1. Cutaneous lupus (chronic discoid lesions)[b]
  2. Lichen planus
  3. Central centrifugal cicatricial alopecia
  4. Folliculitis decalvans
  5. Linear scleroderma (morphea)[c]
- B. Systemic diseases
  1. Discoid lesions in the setting of systemic lupus erythematosus[b]
  2. Sarcoidosis
  3. Cutaneous metastases

[a] Most patients with trichotillomania, pressure-induced alopecia, or early stages of traction alopecia. [b] While the majority of patients with discoid lesions have only cutaneous disease, these lesions do represent one of the 11 American College of Rheumatology criteria (1982) for systemic lupus erythematosus. [c] Can involve underlying muscles and osseous structures.

of the secondary stage of syphilis. Diffuse thinning of the hair is also associated with hypothyroidism and hyperthyroidism (Table 72-4).

Scarring alopecia is more frequently the result of a primary cutaneous disorder such as *lichen planus*, *folliculitis decalvans*, *chronic cutaneous (discoid) lupus*, or *linear scleroderma* (*morphea*) than it is a sign of systemic disease. Although the scarring lesions of *discoid lupus* can be seen in patients with systemic lupus, in the majority of patients, the disease process is limited to the skin. Less common causes of scarring alopecia include *sarcoidosis* (see "Papulonodular Skin Lesions," below) and *cutaneous metastases*.

In the early phases of discoid lupus, lichen planus, and folliculitis decalvans, there are circumscribed areas of alopecia. Fibrosis and subsequent loss of hair follicles are observed primarily in the center of these alopecic patches, whereas the inflammatory process is most prominent at the periphery. The areas of active inflammation in discoid lupus are erythematous with scale, whereas the areas of previous inflammation are often hypopigmented with a rim of hyperpigmentation. In lichen planus, perifollicular macules at the periphery are usually violet-colored. A complete examination of the skin and oral mucosa combined with a biopsy and direct immunofluorescence microscopy of inflamed skin will aid in distinguishing these two entities. The peripheral active lesions in folliculitis decalvans are follicular pustules; these patients can develop a reactive arthritis.

FIGURATE SKIN LESIONS

(Table 72-6) In *figurate eruptions*, the lesions form rings and arcs that are usually erythematous but can be skin-colored to brown. Most commonly, they are due to primary cutaneous diseases such as *tinea*, *urticaria*, *granuloma annulare*, and *erythema annulare centrifugum* **(Chaps. 71 and 73)**. An underlying systemic illness is found in a second, less common group of migratory annular erythemas. It includes *erythema migrans*, *erythema gyratum repens*, *erythema marginatum*, and *necrolytic migratory erythema*.

In erythema gyratum repens, one sees numerous mobile concentric arcs and wavefronts that resemble the grain in wood. A search for an

TABLE 72-5 NONSCARRING ALOPECIA (PRIMARY CUTANEOUS DISORDERS)

	Clinical Characteristics	Pathogenesis	Treatment
Telogen effluvium	Diffuse shedding of normal hairs Follows major stress (high fever, severe infection) or change in hormone levels (postpartum) Reversible without treatment	Stress causes more of the asynchronous growth cycles of individual hairs to become synchronous; therefore, larger numbers of growing (anagen) hairs simultaneously enter the dying (telogen) phase	Observation; discontinue any drugs that have alopecia as a side effect; must exclude underlying metabolic causes, e.g., hypothyroidism, hyperthyroidism
Androgenetic alopecia (male pattern; female pattern)	Miniaturization of hairs along the midline of the scalp Recession of the anterior scalp line in men and some women	Increased sensitivity of affected hairs to the effects of androgens Increased levels of circulating androgens (ovarian or adrenal source in women)	If no evidence of hyperandrogenemia, then topical minoxidil; finasteride[a]; spironolactone (women); hair transplant
Alopecia areata	Well-circumscribed, circular areas of hair loss, 2–5 cm in diameter In extensive cases, coalescence of lesions and/or involvement of other hair-bearing surfaces of the body Pitting or sandpapered appearance of the nails	The germinative zones of the hair follicles are surrounded by T lymphocytes Occasional associated diseases: hyperthyroidism, hypothyroidism, vitiligo, Down syndrome	Topical anthralin or tazarotene; intralesional glucocorticoids; topical contact sensitizers
Tinea capitis	Varies from scaling with minimal hair loss to discrete patches with "black dots" (broken infected hairs) to boggy plaque with pustules (kerion)[b]	Invasion of hairs by dermatophytes, most commonly *Trichophyton tonsurans*	Oral griseofulvin or terbinafine plus 2.5% selenium sulfide or ketoconazole shampoo; examine family members
Traumatic alopecia[c]	Broken hairs, often of varying lengths Irregular outline	Traction with curlers, rubber bands, braiding Exposure to heat or chemicals (e.g., hair straighteners) Mechanical pulling (trichotillomania)	Discontinuation of offending hair style or chemical treatments; diagnosis of trichotillomania may require observation of shaved hairs (for growth) or biopsy, possibly followed by psychotherapy

[a] To date, Food and Drug Administration–approved for men. [b] Scarring alopecia can occur at sites of kerions. [c] May also be scarring, especially late-stage traction alopecia.