rarely in linear morphea of the frontal scalp (*en coup de sabre*), there is involvement of the meninges and brain.

The most common causes of nonscarring alopecia include *androgenetic alopecia, telogen effluvium, alopecia areata, tinea capitis*, and the early phase of *traumatic alopecia* **(Table 58-5)**. In women with androgenetic alopecia, an elevation in circulating levels of androgens may be seen as a result of ovarian or adrenal gland dysfunction or neoplasm. When there are signs of virilization, such as a deepened voice and/or enlarged clitoris, the possibility of an ovarian or adrenal gland tumor should be considered.

TABLE 58-5 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—most common Increased levels of circulating androgens (ovarian or adrenal source in women)—less common	If no evidence of hyperandrogenemia, then topical minoxidil; finasteride[a]; spironolactone (women); hair transplant; low-dose oral minoxidil
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; JAK inhibitors
Tinea capitis	Varies from scaling with minimal hair loss to discrete patches with "black dots" (sites of 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 in trichotillomania and traction alopecia Fringe sign in traction alopecia	Traction with curlers, rubber bands, tight 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.

Exposure to various drugs can also cause diffuse hair loss, usually by inducing a telogen effluvium. An exception is the anagen effluvium observed with chemotherapeutic agents such as daunorubicin. Alopecia is a side effect of the following drugs: warfarin, heparin, propylthiouracil, carbimazole, isotretinoin, acitretin, lithium, beta blockers, interferons, colchicine, and amphetamines. Fortunately, spontaneous regrowth usually follows discontinuation of the offending agent.