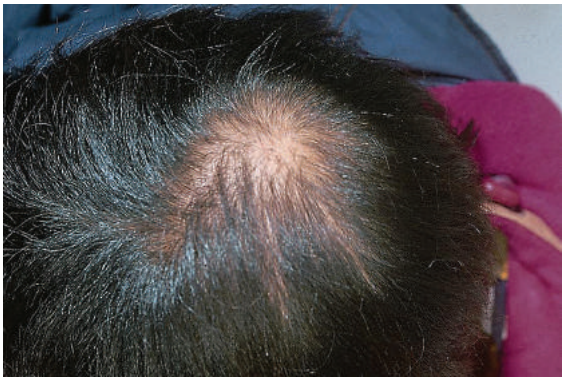

Figure 9.12 **Marie Unna's hereditary hypotrichosis. (Reproduced from Trüeb RM. *Haare Praxis der Trichologie*. Steinkopff, Darmstadt 2003. With permission.)**

diagnosis of Netherton syndrome in the newborn with congenital erythrodermic ichthyosis.

Trichorrhexis nodosa refers to white knots with transverse fractures along the hair shaft. Dermoscopy reveals brush-like hair fracturing. In general, trichorrhexis nodosa is an unspecific finding related to excess stress of hair in relation to its fragility. It can be observed in hair shaft abnormalities with increased fragility, or more frequently as a consequence of hair weathering.

Pili annulati are defined by characteristic alternating light and dark banding in the hair shaft, due to air-filled spaces between the macrofibrillar units of the hair cortex. A priori, not a hair shaft anomaly with increased fragility, the significance of pili annulati lies in that affected hair is more susceptible to weathering, particularly in combination with androgenetic alopecia. With onset of hair thinning due to androgenetic alopecia, progressive reduction of hair shaft diameter may cause increased fragility and trichorrhexis nodosa-like hair shaft racturing.[33]

For treatment, trauma must be minimized, and hair care products and conditioning agents that improve the structural integrity of damaged hair fibers and increase tensile strength are available.

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10 Traumatic alopecia

Pierre Bouhanna

Traumatic alopecia is a hair loss essentially caused by physical trauma. Schematically there are three possible etiologies for these alopecia[1]:

- Trichotillomania is a traction alopecia due to a compulsive disorder. Patients pull on and pluck hairs, often resulting in bizarre patterns of alopecia. In trichotillomania, the patient, with psychological disorders, will manipulate his or her hair, unconsciously or deliberately, but repeatedly, to cause more or less severe baldness.
- Cosmetic alopecia is caused by the excessively strong or aggressive care and handling of hair.
- Traumatic accident alopecia is easy to diagnosis.

We will not deal here with some hair dysplasias (see Chapter 4) that are responsible for brittle hair or occipital alopecia in newborn children, which present at areas of friction (Figure 10.1).

Figure 10.1 **Occipital alopecia of the newborn child.**

TRICHOTILLOMANIA

Psychopathological aspects

Trichotillomania is a tic of hair removal that occurs most often in boys under 6 years and in girls over 6 years (Figure 10.2).[2–4] The most severe cases occur in women between adolescence and 40 years (Figure 10.3); in these instances, the psychological disorders are major.

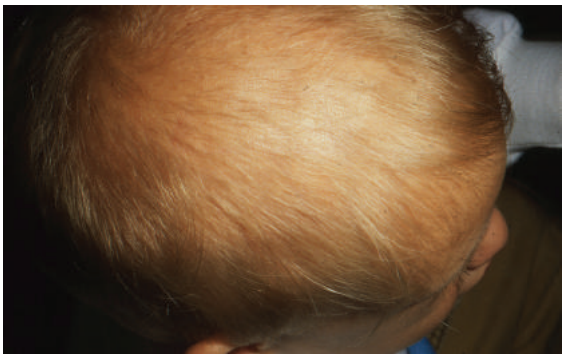


Figure 10.2 **Trichotillomania in a 2-year-old child.**

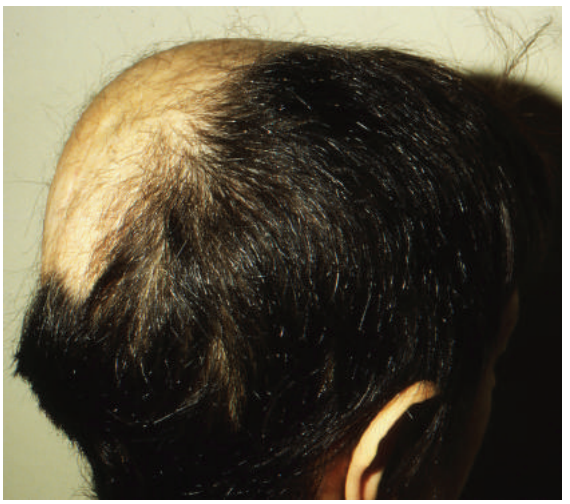


Figure 10.3 **Circumscribed alopecia caused by trichotillomania located at the left half of the scalp in a young woman. (Note the angle and the rectilinear border.)**

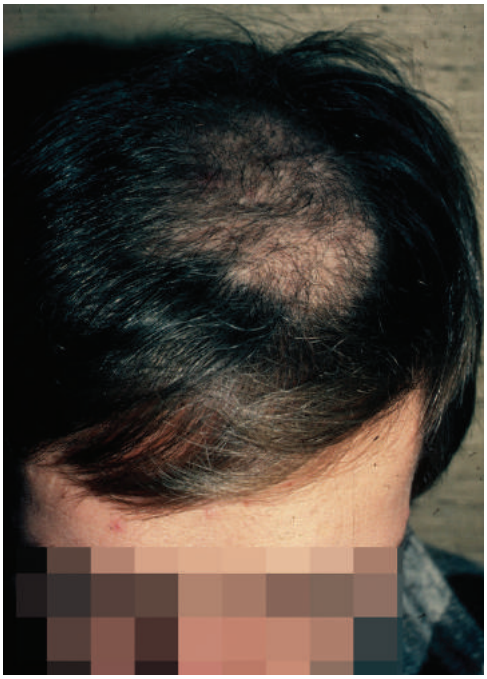


Figure 10.4 **Circumscribed alopecia caused by trichotillomania located at the vertex in a young adult. (Note the angle and the rectilinear border.)**

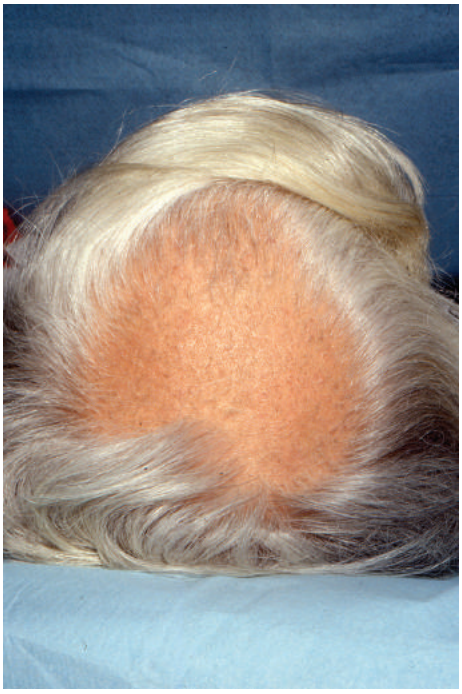


Figure 10.5 **Circumscribed alopecia caused by trichotillomania located at the vertex in an elderly woman. (Note the geometric contours.)**

Clinical aspects

Alopecia appears as an area with a clear boundary (Figure 10.4). The skin surface is normal and nonsquamous. The manipulated hairs are broken at different lengths. The parietal region is the most frequently affected.

In adults, the area of alopecia is often extensive, with short broken hairs. It can be localized to the vertex (Figure 10.5), appear unilaterally in the fronto-parietal–occipital region (Figures 10.3 and 10.6) or, exceptionally, be seen on the entire scalp.

Tics of removing eyelashes, eyebrows, or other body hair (pubic or perianal) are exceptional.

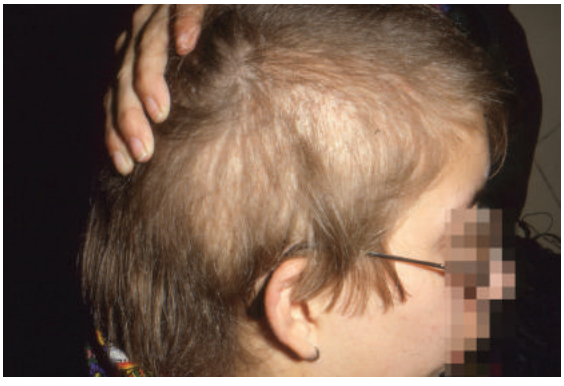


Figure 10.6 **Trichotillomania located in the parietal-occipital region in a 14-year-old boy. (Note the rectilinear or angular and geometric contours.)**

Diagnosis

Histological aspects: The most constant element is that many hair orifices are empty. In addition, telogen hairs are absent or very few. Biopsy specimens from acute lesions of trichotillomania may demonstrate perifollicular hemorrhage and fractured fibers. Later stages of trichotillomania characteristically demonstrate the presence of normal follicles surrounded by empty follicles in a noninflammatory dermis. Numerous catagen follicles (characterized by numerous apoptotic cells and a wavy surrounding vitreous membrane) and pigment casts within the upper segments of the follicle may be noted.

Trichoscopy[5,6] (Figure 10.7) is useful to confirm the evidence of pulling. The scalp presents short, coiled, fractured hairs. The broken hair shafts show longitudinal splitting (see Chapter 3).

Trichograms (1) show the almost exclusive presence of normal anagen hair (see Chapter 2).

Differential diagnosis

In children, a patch caused in trichotillomania may be confused with ringworm or alopecia areata.

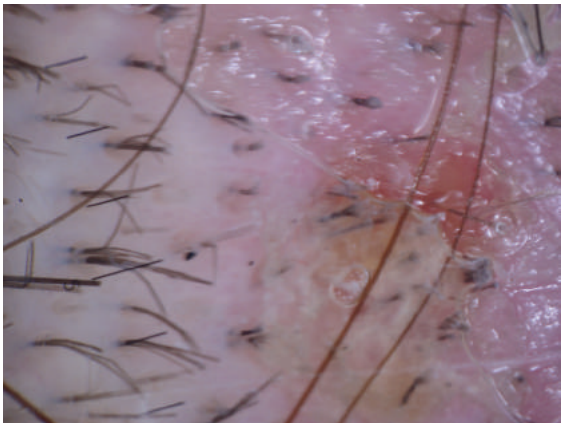


Figure 10.7 **Trichotillomania with erosion, black spots, hairs in "tulip" and hairs in "V." (Courtesy of Dr. Y. Bourezane.)**

With ringworm, the skin surface is rough. Infected hair is recognized on examination by Wood lamp and by microscopic examination.

In the case of alopecia areata, the presence of alopecia hair in "exclamation marks" is particularly evocative. Hair regrowth is fluffy and often clear or white, at first. In addition, abnormalities deriving from dystrophy of hair bulbs are visible on a trichogram.

The major forms of trichotillomania in adults should be differentiated with a careful clinical examination and a trichogram from androgenetic alopecia localized to the vertex and which can be associated (Figure 10.8).

Prognosis and treatment

In children, the tic hair removal is almost always favorably resolved after raising the issues in the presence of the parents.

In long-term cases of trichotillomania, permanent alopecia may occur.

The prognosis is more difficult in the extended forms found in the adult (Figures 10.3 and 10.5), in that they deny they are responsible for the act and often refuse psychotherapy as a solution.

COSMETIC ALOPECIA

The requirements attributed to ethnic or religious customs or the social pressure exercised by fashion cause a variety of hair damage.[7,8] Their diversity is directly proportional to the imaginative intensity of each individual (Figures 10.9a and b, 10.10a and b).

Pathogenesis

The pathogenesis of alopecia cosmetics may be attributed to

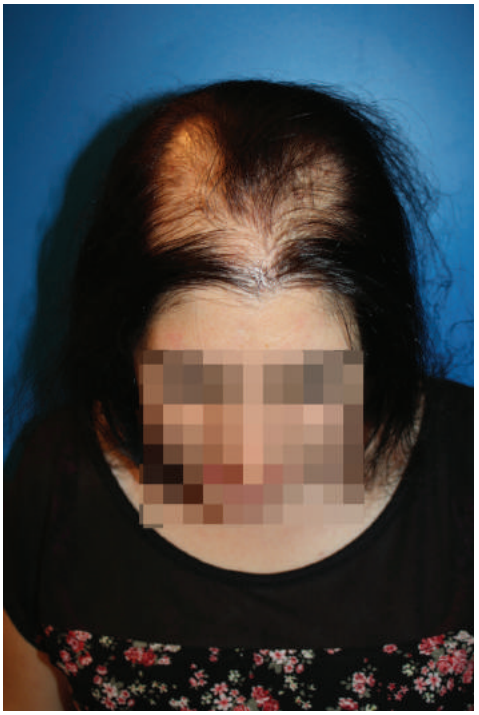


Figure 10.8 **Female androgenetic alopecia and traction alopecia. (Note the linear straight border.)**

- Breakage of hair secondary to manipulation of hair shafts, which have sometimes been weakened by chemical applications.
- Repeated traction at the shaft o stiffen it.
- Scarring alopecia induced by repeated pulling with preexisting inflammatory follicular lesions.[9]

Clinical aspects

The essential elements of frontal traction alopecia are the presence of short broken hair, folliculitis lesions, and some small scar patches located on the frontotemporal edge (Figure 10.11). This clinical form often affects women making a tightly pulled "bun," a "ponytail," or even one or two big braids (Figure 10.9a and b). Note also that parietal bald patches are often observed in nurses due to traction on the hair by clips that keep their cap in place (Figure 10.12).

The differential diagnosis is postmenopausal frontal fibrosing alopecia.

- Alopecia due to the rollers. This process, used to "curl" hair deemed too stiff, causes bald patches surrounded by broken hair and a rash if used regularly or too frequently.
- Alopecia due to wearing wigs braided to the remaining hair or attached by clips (Figure 10.13a

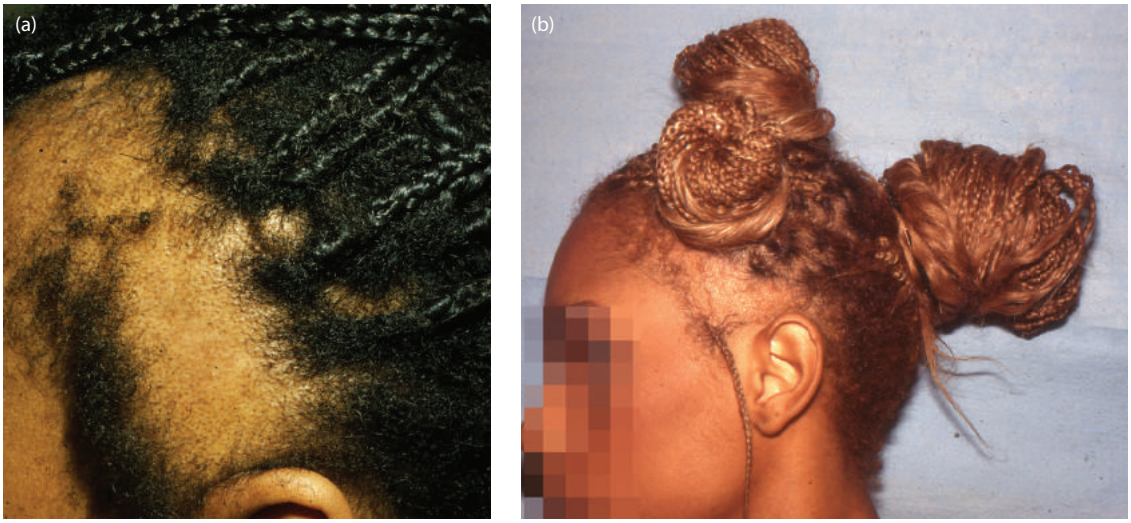


Figure 10.9 **(a,b) Alopecia from frontotemporal traction occurring in women who regularly braid their hair.**

and b). The clinical aspect of alopecia secondary to wearing a hairpiece is similar to that described above.

- Alopecia due to strong and repeated brushing. It will cause, among black patients whose hair is naturally weak (see Chapter 12), a recession of the frontotemporal line.
- Alopecia due to the use of straightening irons. Heat trauma causes a scarring alopecia gradually extending toward the vertex (Figure 10.14).
- Alopecia due to repeated friction or massage. Massage or excessive and repeated friction can cause alopecia, such as repeated rubbing of the arms of glasses (Figure 10.15).

Figure 10.10 **(a) Traumatic alopecia after the break dance practice, (b) the break dance.**

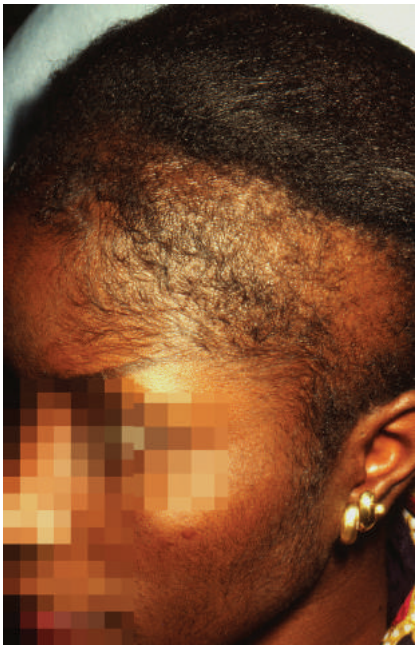


Figure 10.11 **Frontotemporal alopecia after repeated hair straightening in a black patient. (Note the excess growth of vellus above the eyebrow after applying minoxidil lotion 2%.)**

Differential diagnosis

The diagnosis is easily established by the results of a precise clinical examination, a trichoscopy examination, a trichogram evaluation, and eventually a biopsy.

Treatment

The treatment is obviously related to the removal of the cause and prescription of cosmetic products (such as cream

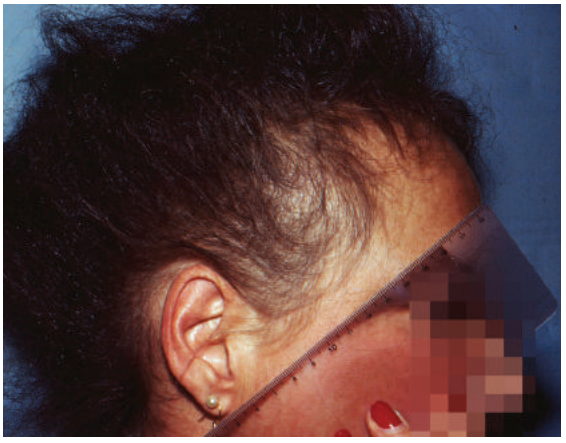


Figure 10.12 **Temporoparietal alopecia in a woman after regularly wearing a cap.**

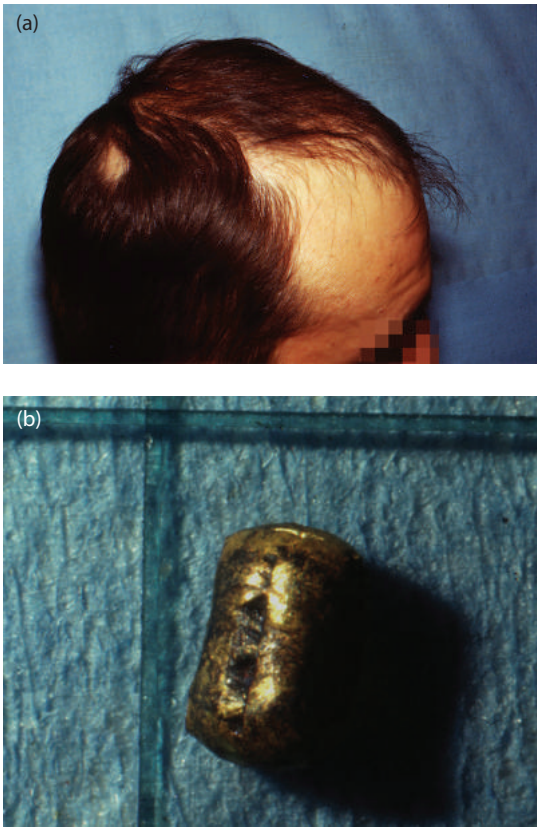


Figure 10.13 **(a) Alopecia caused by traction from clips holding the hair in place. (b) Example of a small metal cylinder used to secure a prosthesis.**

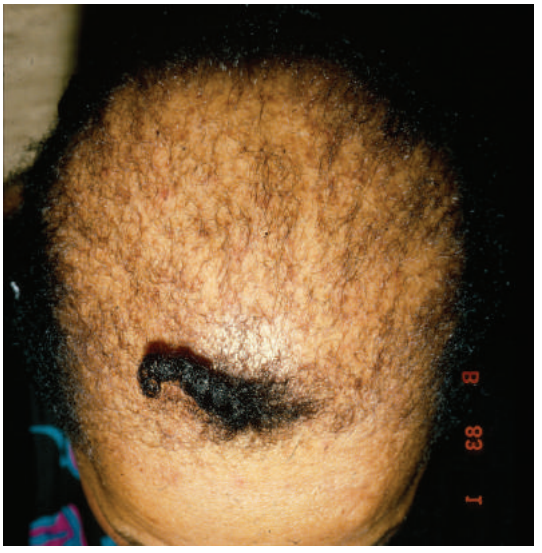


Figure 10.14 **Almost complete scarring alopecia in a black woman who had been using hot hair-straightening irons for many years.**

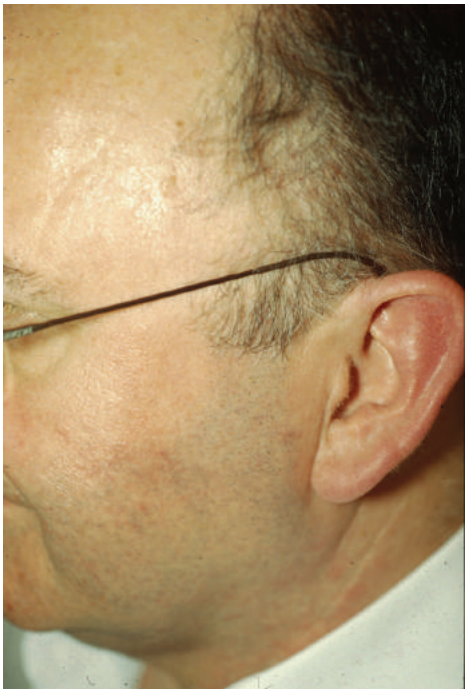


Figure 10.15 **Temporal alopecia due to repeated rubbing and compression of the glasses arms.**

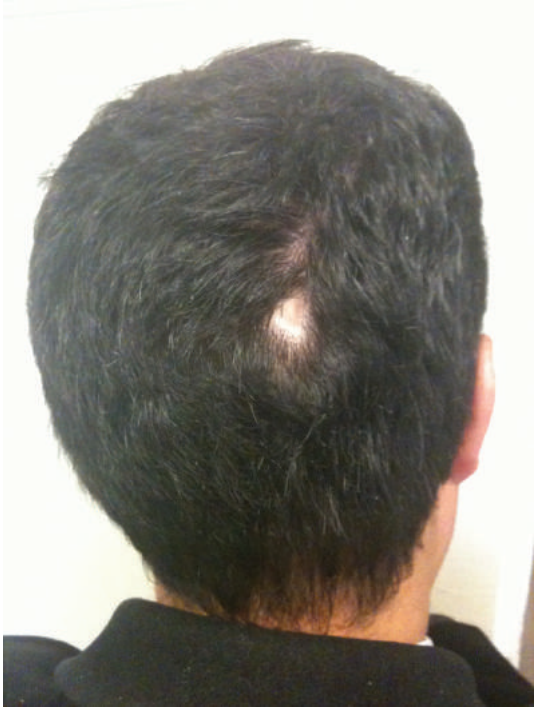
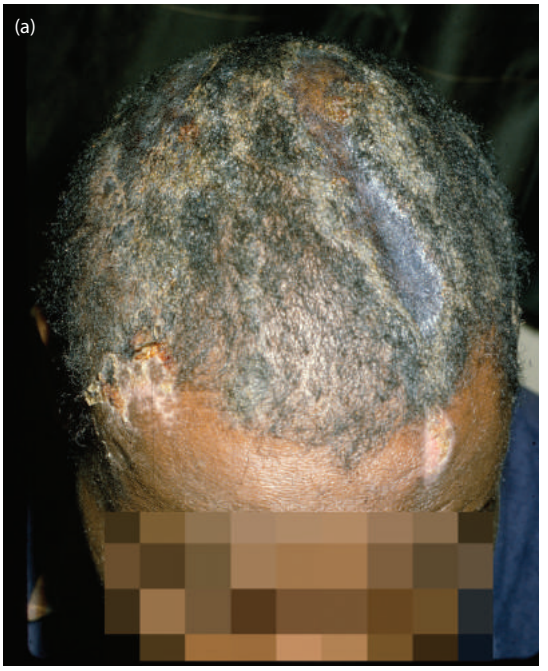


Figure 10.16 **Adult male patient with an occipital alopecia post-forceps.**

with karite) to decrease the possible underlying fragility of the hair shaft. It is not proved that the prescription of topical minoxidil encourages some regrowth because nearly all the hairs are in anagen phase, and it may increase the growth of vellus on the face of the female patient.

TRAUMATIC ACCIDENT ALOPECIA

Some traumatic accidents can cause nonscarring alopecia, for which the diagnosis of cause can be difficult; it is, most often, ischemia caused by prolonged or repeated compression of part of the scalp (Figure 10.15).[7] This can occur, for example, from compression by forceps during childbirth (Figure 10.16) or during compression by repeated and prolonged wearing of a cap or orthodontic headgear (a head cap) by a child.



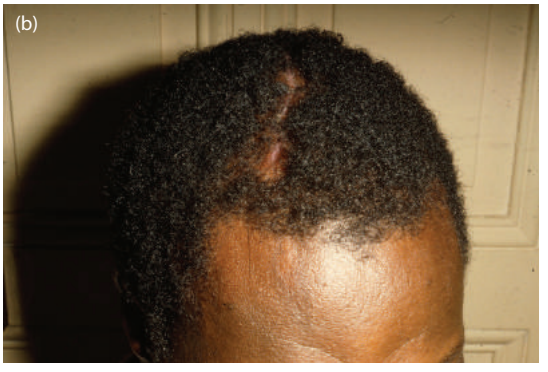


Figure 10.17 **(a) African patient after scalp burning with caustic soda. (b) Spontaneous healing without treatment.**

TRAUMATIC THERMAL, CHEMICAL, AND ISCHEMIC ALOPECIA

A variety of chemical (corrosive) and physical agents can cause permanent alopecia of the scalp. In general, any thermal, physical, or chemical injury sufficient to cause scalp necrosis can produce a permanent alopecia. In the acute stage, necrosis with crusting, an adherent eschar, or ulceration may be present (Figure 10.17a and b). Later, the scalp heals by reparative fibrosis. The result is usually an atrophic alopecic patch. Ischemic necrosis of the scalp produces a similar clinical picture. Ischemic scalp necrosis may be seen after prolonged anesthesia. The necrosis generally involves the occipital scalp and represents a pressure phenomenon. Ischemic necrosis may also occur as a result of vasoconstriction related to the infusion of pharmacologic agents such as vasopressin.

RADIATION

Radiation injury may cause transient nonscarring alopecia (epilating dose). Higher doses[9] produce permanent alopecia with scarring. Histological changes in anagen

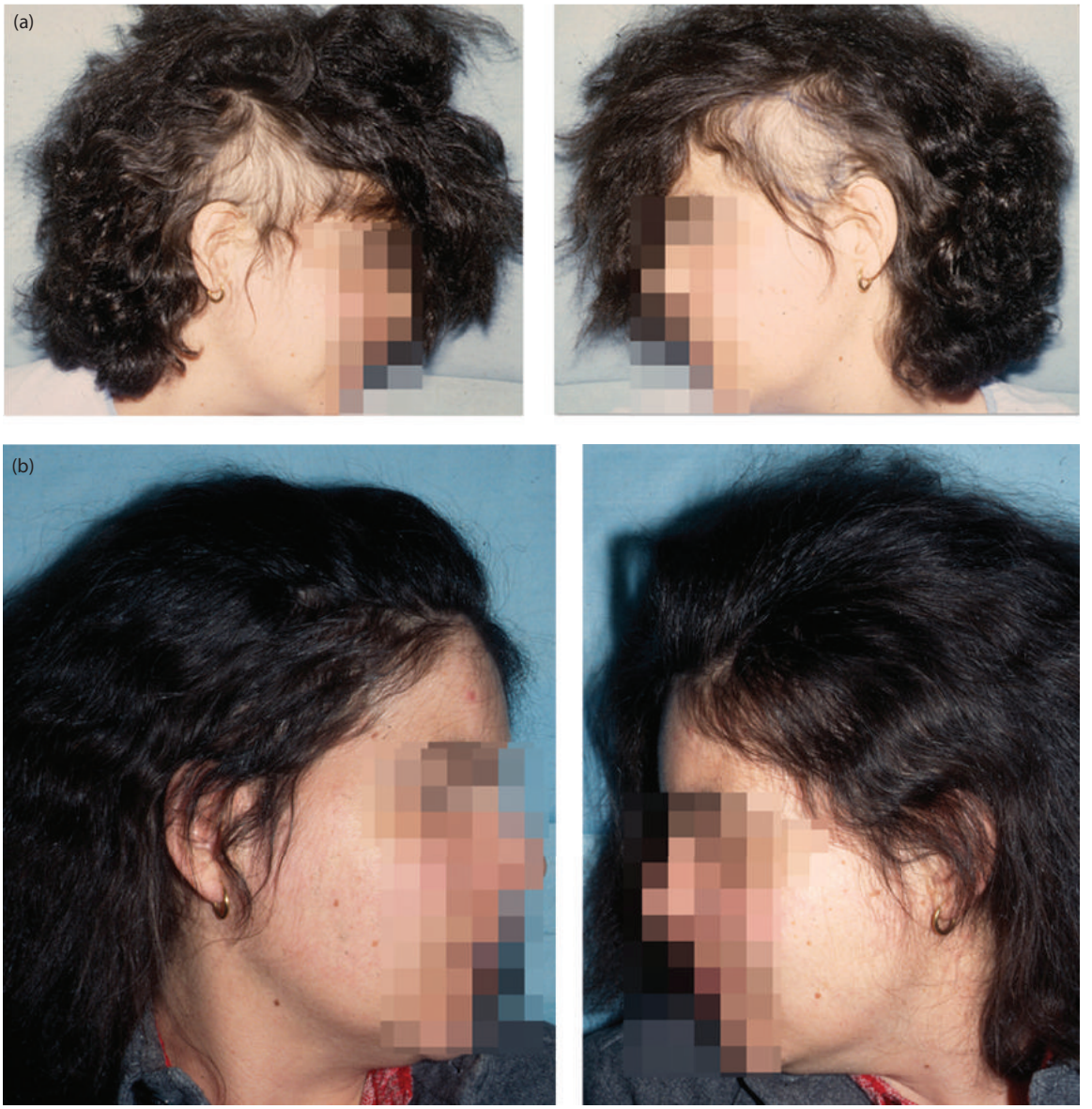


Figure 10.18 **(a) Bitemporal cicatricial alopecia after radiotherapy for brain tumor, and (b) correction after one hair transplant session.**