correlation has been observed between penetration of topically applied substances and sebum production as well as growth activity of the hair follicle. This observation provided the rationale for the development of particular drug formulations and carrier systems to specifically target hair follicles. Therefore, these findings on penetration in and through the hair follicle depending on localization, gender, and growing activity are of high practical relevance and provide implications for the dermatological external therapy and for the development of transcutaneous application systems.

CLINICAL RELEVANCE

The development of new strategies to control the hair follicle cycle is currently in the spotlight of hair research, and a wide range of novel molecules and delivery systems are currently being developed. The investigative challenges in alopecia treatment involve understanding and controlling signal transduction events and their regulatory genes in order to induce and/or prolong anagen and to shorten telogen. A profound knowledge of hair follicle biology could facilitate the targeted control/selective influence of the local regulation systems and the development of novel hair loss therapeutic approaches based on molecular evidence rather than pure empirical evidence.

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2 Hair and scalp investigations

Pierre Bouhanna

INTRODUCTION

For the diagnosis of hair loss or alopecia, it is important to record a complete clinical history to guide the clinical examination. There are additional investigations to quantify the hair loss and confirm its pathological character, to clarify its etiology, to assess the extent of the possible associated alopecia, and finally to objectively measure the effectiveness of specific treatments (Table 2.1).

HISTORY

Diagnosis of alopecia usually proceeds from a good history-taking that analyzes the personal history, the family history, and the history of the disease.[1–3]

Personal history

Many kinds of alopecia are due to underlying or concomitant diseases. Those kinds differ from both scarring alopecia, which results in concomitant follicular and irreversible skin damage, and other nonscarring types of alopecia, which cause reversible alteration of the follicle and skin without final alteration. Nonscarring alopecia (androgenetic alopecia, alopecia areata, trichotillomania, etc.) is an increasingly common reason for consulting a physician (Figure 2.1). In the case of abrupt hair loss (effluvium), we must take careful note of the patient's medications, such as cytostatics, anticoagulants, lithium, antithyroid, vitamin A, etc. Various alopecias are encountered due to endocrinopathies (hypo- and hyperthyroidism, androgen syndrome, Cushing syndrome, etc.), iron deficiency, etc. (see Chapters 5 through 8).

Family history

Family history should be taken systematically, particularly in male and female androgenetic alopecia. The importance of a family history is greater in the diagnosis of certain abnormalities such as congenital or hereditary hypotrichoses (e.g., Marie Unna syndrome), genodermatosis such as anagen loose hair syndrome, or pilar dysplasia (monilethrix, woolly hair, etc.) (see Chapter 4).

Table 2.1 Management of a Hair Loss

History

- Personal history:
  - Initial aspect
  - Concomitant symptoms
  - Evolution
  - Concomitant diseases
- Medications such as:
  - Cytostatics, anticoagulants, lithium, antithyroid, etc.
- Family history about hair and alopecia disease

Methods of clinical exploration

- Pull-test
- Global photography
- Trichoscopy
- Trichogram
- Digital phototrichogram
- Multifactorial classifi ation parameters

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Diagnosis of the hairloss

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Schema of treatment

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Precise follow-up

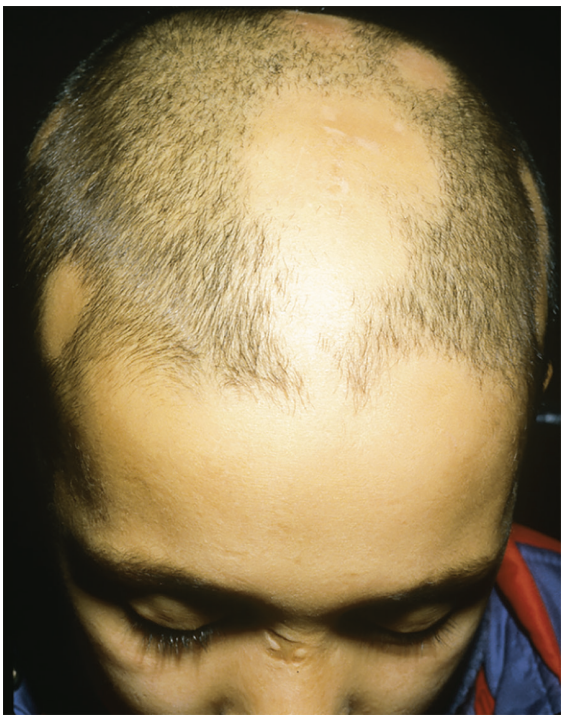


Figure 2.1 **Patches of alopecia areata.**

History of the disease

The clinician will need to check on the aspects the problem displayed initially, the associated clinical signs, and the disease's evolution (Table 2.2).

Initial aspects include the following: hair loss can be acute and diffuse (effluvium in anagen and telogen), acute and focal (in traumatic alopecia, Figure 2.1), chronic and diffuse (androgenetic alopecia), or chronic and focal (pseudopelade, alopecia areata, trichotillomania). It is important to note the age at onset of the first signs: early (pilar dysplasia, congenital hypotrichoses), postpubertal (androgenetic alopecia), or late (acquired diseases) (see Chapter 5).

Associated signs include:

- Seborrhea, acne, hirsutism, and alopecia (SAHA) syndrome for androgenetic alopecia and alopecia.
- Localized inflammation for decalvans folliculitis or ringworm.
- Alterations in the nails, bones, eyes, or teeth for follicular hyperkeratosis (monilethrix, keratosis pilaris decalvans) or elsewhere in different types of ectodermal hair dysplasias.

Table 2.2 Hair Loss Description

Early form	Diffuse and acute	Telogen or anagen effluviums Alopecia areata
	Diffuse and chronic	Androgenetic alopecia
	Focal and acute	Alopecia areata Traumatic alopecia
	Focal and chronic	Pseudopeladic alopecia of Brocq Trichotillomania
Associated symptoms	Seborrhea	Androgenetic alopecia Female constutional hyperandrogenism (SAHA)
	Inflammation	Folliculitis decalvans Tuft olliculitis Tinea capitis Lichen planopilaris Lupus erythematosus
	Follicular hyperkeratosis	Follicular keratosis decalvans Monilethrix
	Ectodermic alterations	Ectodermic dysplasia
Evolution	Progressive	Androgenetic alopecia
	Successive eruptions	Alopecia areata
	Irreversible	Cicatricial alopecia
	Autoinvolutive	Anagen or telogen effluviums Patchy alopecia areata

- Progression that is slow for androgenetic alopecia, advances in pushes for alopecia, is irreversible for scarring alopecia, or is idiosyncratic for telogen effluvium and alopecia areata.[2]

CLINICAL EXPLORATION

Clinical examination of the hair and scalp provides valuable information about alopecia, indicating the morphological appearance of the hair and scalp as well as the form and extent of alopecia.[1–4]

Morphology of the hair

The morphology of the hair is very important to recognize, as it can provide information not only on the type of alopecia in question but also on the mode of progression. Thus, the presence of hair with normal appearance and texture does not have the same meaning as dry and dull hair (pilar dysplasia, hypotrichoses, malnutrition, deficiency diseases). It is very important to observe if there are hairs of smaller thickness or that are shorter and thinner (intermediate hairs) on the frontoparietal recession or the tonsure. These intermediate hairs correspond to miniaturized hair, becoming thinner and thinner through successive hair cycles under androgenetic alopecia (Figure 2.2). In alopecia areata, "exclamation mark" hairs are found, under the microscope, at the edge of the bald patches (Figure 2.3).

Distribution of alopecia

Alopecia can be diffuse (hypotrichoses, androgenetic alopecia, effluvium) or in patches (alopecia areata, pseudopelade, trichotillomania).

Skin appearance

Examination of the skin surface of the scalp allows a physician, in most cases, to recognize scarring alopecia (irregular bald patches where there remain hairs of normal appearance, with skin changes involving erythema, fibrosis, and atrophy) or nonscarring alopecia (round and

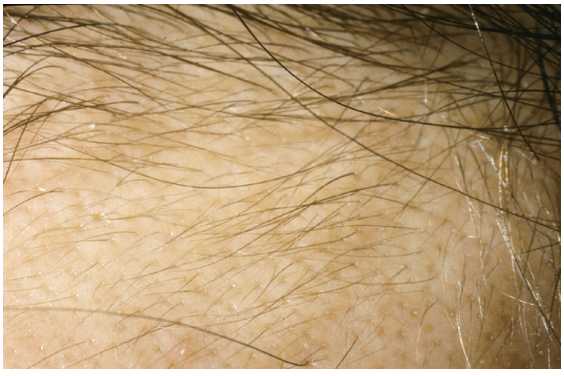


Figure 2.2 **Miniaturized hairs (vellus and intermediate) on the frontal hairline pathognomonic of the androgenetic alopecia.**

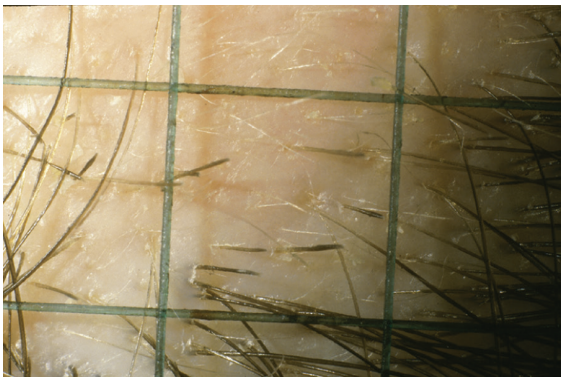


Figure 2.3 **Characteristic dystrophic anagen aspect of "exclamation mark hair," pathognomonic of alopecia areata visible on trichoscopy.**

regular patches with a normal-looking skin surface where all the hair has disappeared).

Types of alopecia

Abnormal hair loss can be acute or chronic, diffuse or localized.[2–4]

Four types of alopecia can be identified schematically (see Chapter 5):

1. Alopecia at the frontotemporal recession or of the crown is characteristic of androgenetic alopecia.
2. Diffuse alopecia is of uniform distribution over the entire scalp and includes effluvium in anagen and telogen phases, and female androgenetic alopecia (especially if it is a lower form with localization to the temporooccipital region). It is not unusual for two types of alopecia to coexist in this group.
3. Alopecia in a band along the edge of the scalp is especially visible in ophiasis alopecia areata, traction alopecia, and frontal fibrosing alopecia.
4. Alopecia in patches is characterized by one or more patches of different shapes and sizes. Several clinical features are present; the most significant are alopecia areata, ringworm, follicular mucinosis, trichotillomania, and pseudopelade.

INVESTIGATIONS

More specific types of investigation to carry out can be divided into two categories: additional clinical tests and laboratory tests aimed at individual cases.

Laboratory tests when hair loss has been observed

A specific blood test is required to detect hematological and biochemical changes.[1–3] It is possible to identify a lack or deficiency of metabolic components of the hair follicle or even whether there is a disease responsible for the androgenetic alopecia or hyperandrogenism.

In women, it is important to carry out first-line investigations for any hair loss, such as a complete blood count (CBC), erythrocyte sedimentation rate (ESR), and serum ferritin and/or iron saturation (total iron-binding capacity, TIBC).

We will suspect a hormonal imbalance if there is facial hirsutism or excess hair (with or without dysmenorrhea or amenorrhea). There should therefore be a prescribed dose blood tests of male hormones such as testosterone (T), dehydroepiandrosterone sulfate (DHEA-S), the Δ4-androstenedione or female hormones such as prolactin (PRL), follicle-stimulating hormone (FSH), and prolactin dehydrogenase (LDH).

It is also advisable to look into a possible thyroid origin, checking FT4 and thyroid-stimulating hormone (TSH).

In men, however, it is not necessary to perform first-line blood tests in male pattern androgenetic alopecia, except in cases of diffuse alopecia of the female type; however, check CBC, ESR, and TIBC in patients with a history or suspicion of anemia.

Clinical tests in routine practice

Critical examinations performed when confronted by any hair loss are the pull-test, standardized global photography, a trichogram, a dermatoscopy or trichoscopy, a digital phototrichogram,[5] and the possible measurement and recording of all parameters in the multifactorial classification[6] in the case of male or female androgenetic alopecia (see Chapter 5).

The pull-test

The pull-test allows for confirmation of hair loss (Table 2.3).[1] It consists of a gentle traction between the thumb and forefinger on a lock of 50–60 hairs on a scalp that had not been washed for 48 hours at three different locations on the scalp. The result is normal if less than six hairs are removed. If the number of hairs that are removed on traction is greater than six hairs per sampling point, we can consider that the patient has abnormal hair loss, as in effluvium in the anagen or telogen phase or alopecia areata (Figure 2.4). The value of this exam is relative to the individual patient and the same examiner.

The standardized global photography

Standardized global photography consists of global photographs of the vertex,[4] frontal, and temporal regions performed in a standardized manner (Figure 2.5). The patient places his or her chin on a stereotactic positioning device. The camera is mounted on a metal rod and fixed at a known distance to allow taking photographs of the vertex, frontal, and temporal regions. This system, developed by Canfield Scientific (Fairfield, New Jersey), allows for an accurate monitoring of alopecia areas and hairy areas.

For better control of the treatment's efficacity on androgenetic alopecia, we suggest that the global photographs

Table 2.3 Hair-Pull Test for Various Hair Diseases

Disease	Hair-Pull Test
Normal	0–5 hairs can be pulled; a test with ≥6 hairs is positive
Alopecia areata	Positive ≥6 hairs on light microscopy show dystrophic anagen and telogen stage
Androgenetic alopecia	Mostly normal; in active AGA, positive on the top of the scalp, negative in occipital area
Acute anagen or telogen effluvium	Positive in active phases with increased numbers of anagen or anagen dysphastic hairs or normal telogen hairs
Chronic telogen effluvium (CTE)	Positive only in active phase of CTE always with 6–8 telogen roots when examined with light microscopy
Trichotillomania	Negative with no pluckable hairs
Loose anagen syndrome	Highly positive; up to 100% when hairs are examined under the microscope; anagen hair root mostly lacking the hair sheath (anagen dysplastic)

Source: With kind permission from Springer Science+Business Media: *Hair Growth and Disorders*: *With 85 Tables*, 2008, Berlin Heidelberg: Springer, Blume-Peytavi U.

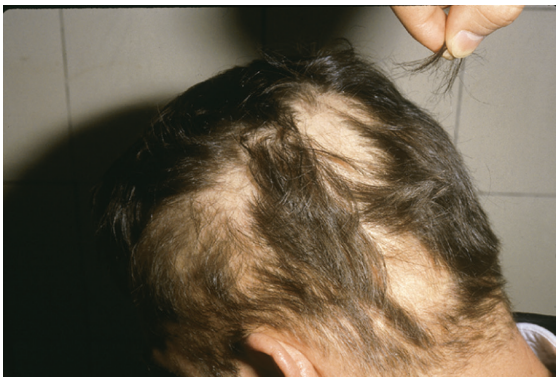


Figure 2.4 **Positive plucking test (+++) performed on a patient with very extensive alopecia areata progressing to total alopecia.**

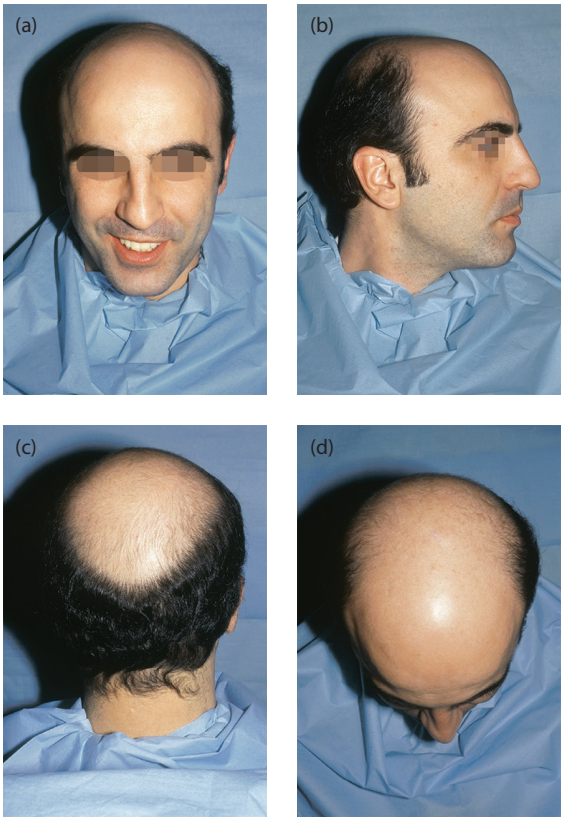


Figure 2.5 **Global photographs: (a) front view, (b) side view, (c) back view, and (d) vertex view of a patient with androgenetic alopecia.**

be taken with wet hair combed forward to eliminate the artifactual effect (Figure 2.6).

The trichogram

This type of investigation allows us to determine the hair formula—that is, the distribution of hair as a percentage,[5,7] in each hair phase.

In adulthood, the number of hairs on a normal scalp varies from 100,000 to 150,000. They grow on average from 0.35 to 0.44 mm/day, and growth is cyclical.

The hair cycle consists of three phases (Figure 2.7): a growth or anagen phase, a transition or catagen phase, and a falling or telogen phase. Each telogen hair, once fallen, is replaced by a new anagen hair, and the balance of anagen/telogen hair remains constant.

Schematically, the anagen phase lasts an average of 3 years (2–4 years in males and 4–6 years for women), the catagen phase 3 weeks, and the telogen phase 3 months.

This explains that at every moment of the hair cycle, 80%–85% of hair is in the anagen phase, 1%–2% in the catagen phase, and 15%–20% in the telogen phase.

The percentage of anagen hair is more important in women (85%–90%) with 10%–15% of hair in the telogen phase. Normal drop is on average 25–60 hairs a day.

Sampling is effected with the aid of a curved Kocher clamp with jaws coated with rubber. A row of about fi y hairs is pulled sharply in the direction of their emergence from the skin in three different areas: occipital, frontotemporal and temporoparietal (Figure 2.8). It is important to take the sample on hair that has not been washed for three days, as washing or brushing removes telogen hair and thus leads to an overestimation of the anagen/telogen ratio. Plucking hair in the direction of the emergence

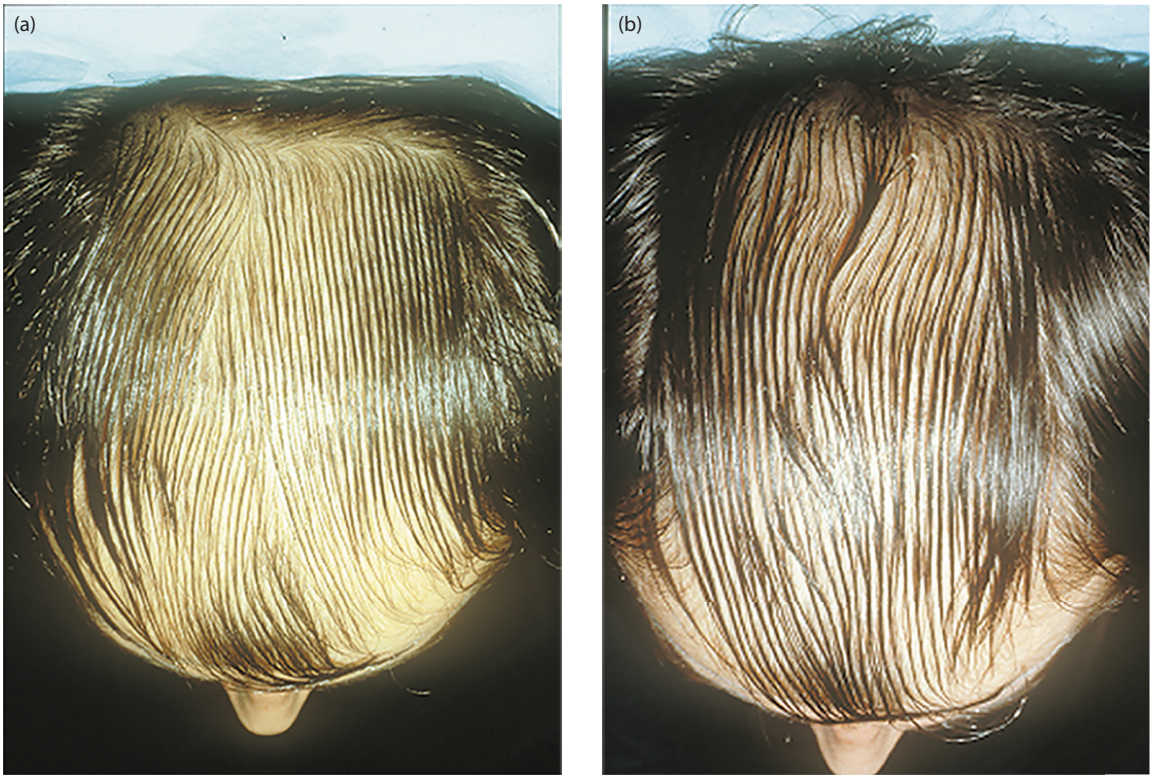


Figure 2.6 **(a) Global photography of an androgenetic alopecia (with hair wet and combed forward to eliminate artifactual effects), (b) the same patient with clinically visible regrowth after 6 months of treatment with 2% minoxidil treatment.**

of hair shafts can minimize the number of broken hairs. These hairs are cut about 1 or 2 cm from the root and placed between a slide and a cover slip (Figure 2.9). The reading is made on a microfi he reader or a microscope at low magnifi ation; the image, magnifi d 20–40 times, allows detailed analysis of the root and part of the hair shaft (Figu es 2.10 and 2.11).

Microscopic aspects

- *Normal microscopic aspects*. Three types of hair roots are visible:
  - *Anagen hair* has a large pigmented bulb (Figure 2.12).[3,5] In the early anagen phase, the bulb is at its largest with a pyramid shape; it then becomes quadrangular and narrower, and pigmentation remains strong. When the epithelial ducts are present, they are visible and clear; when these are absent, the anagen hairs are "naked."
  - *Telogen hair* has a rounded bulb, which is very obvious and easily identifiable by the appearance as a "golf club" or "cotton swab" (Figure 2.13). In early telogen phase, epithelial ducts form a transparent bag that covers the bulb. Later, when the telogen phase is concluding, ducts are not visible (or scarcely visible) with a marked depigmentation.
  - *Catagen hair* is in an intermediate phase. It is purely a transitional period of short duration during which the bulb is narrower and has already begun to lose pigmentation. Epithelial ducts are narrower, more irregular, and cover a smaller part of the bulb (Figure 2.14).
- *Modified microscopic aspects*.
  - *Dysplastic (distorted) anagen hair*. Plucking can distort the anagen hair, giving it the appearance of the "handle of a walking stick." The bulb is discolored, and ducts are often absent. These hairs do not have any bad significance.
  - *Broken hair*. This is most often caused by plucking rather than by a break caused by traction on dystrophic anagen hair. A percentage greater than 10% indicates a faulty technique.
- *Abnormal microscopic aspects*.
  - *Dystrophic anagen hair*. These are thin, nongrowing anagen hair, most often secondarily to

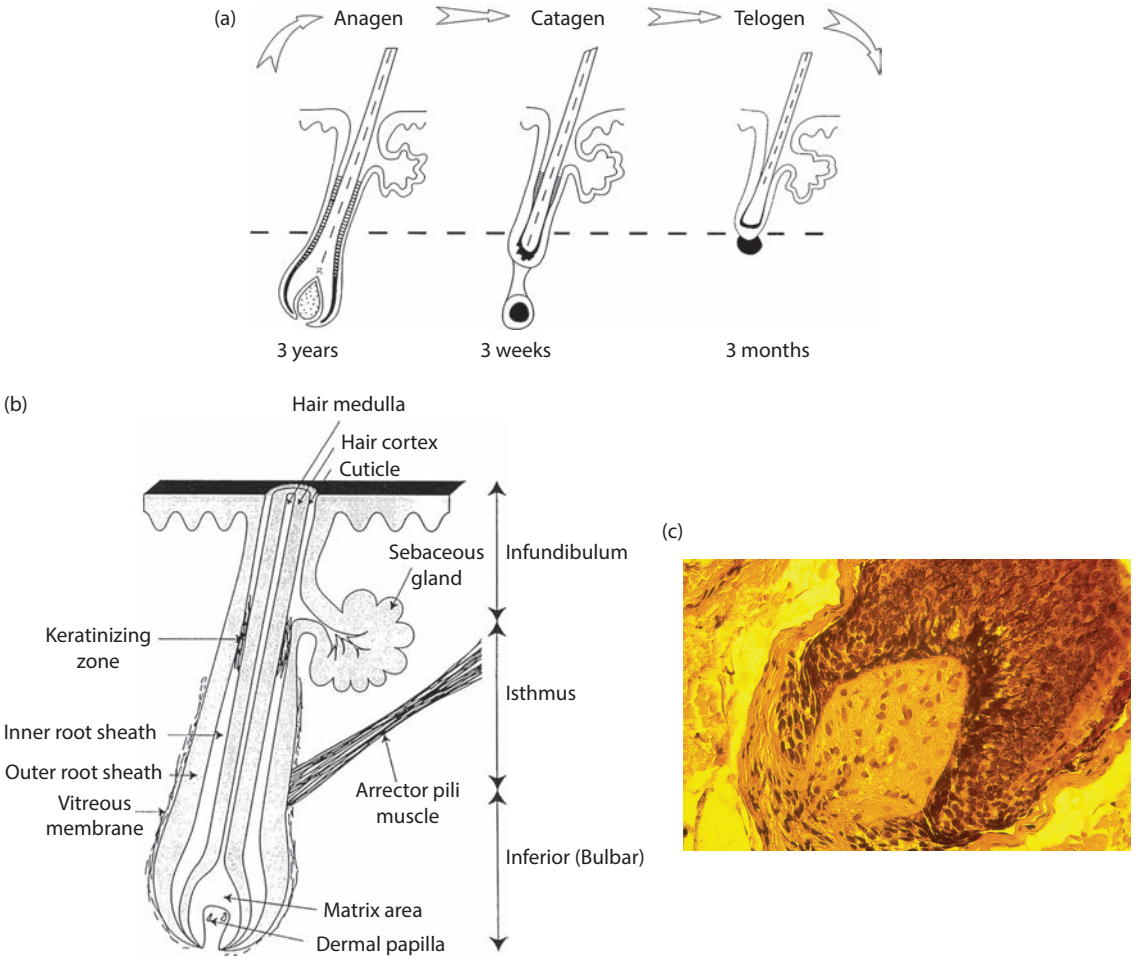


Figure 2.7 **Hair cycle (a) and schematic aspect of the hair follicle (b). (c) Histological view of the lower segment of the hair follicle, with the matrix.**

an assault. This anagen hair has a tapered end, is without a matrix, is without a sheath, and is often discolored. The keratinization is defective. This appearance is rare on a normal hair (Figure 2.15).

- "*Exclamation mark hair*" *in alopecia*. This is an anagen hair that is approximately 3 cm from the base, fringed fracture above a terminal bulge (Figure 2.15). It is almost visible in evolutive alopecia areata (Figures 2.3 and 2.4).

Whatever their form, dystrophic hair without sheaths and with little pigmentation does not represent more than 5% of normal hair.

Results of a normal trichogram

The anagen phase lasts 3 years, the catagen stage 3 weeks, and the telogen stage 3 months.

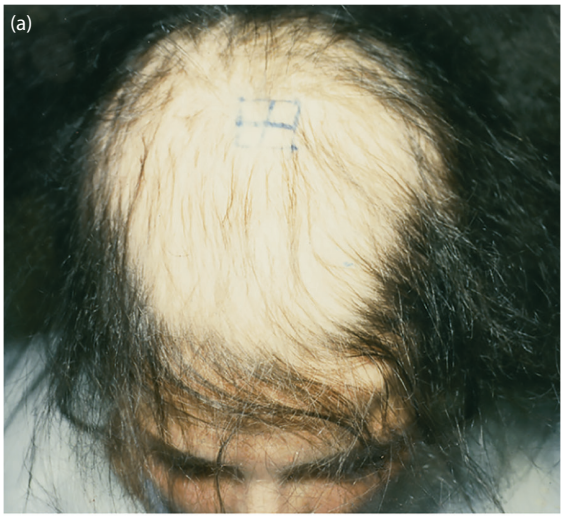
The normal hair formula for young adults is 80%–85% of hair in anagen, 1%–2% in catagen, and 14%–20% in telogen.

There are normally 20% of dysplastic anagen hairs called "naked anagen hair" (without ducts) and less than 2% of dystrophic anagen hair. In children, the number of anagen hairs is at least 90%. The number of telogen hairs is also very high in the newborn and the infant.

Physiological changes on the trichogram

The number of telogen hairs is higher on the frontoparietal regions, and the percentage of telogen hairs is highest for patients between 50 and 60 years.

Note that the formula for hair cycles varies with the seasons: in summer, the anagen stage is longer; falling telogen hair is up in March to April and September to October; while telogen hair falls out least during the months of January and July.



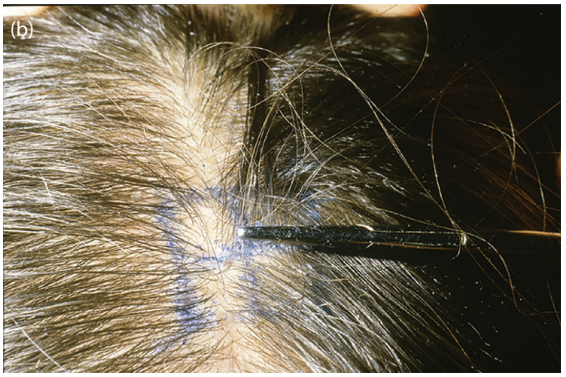


Figure 2.8 **Area of hair to be sampled for a trichogram: (a) choice for sampling in a patient with type III female androgenetic alopecia, (b) the tuft of hair is separated out in an area bounded by tattooing at four points and divided into four quarters.**

Figure 2.9 **Tuft of hair taken before alignment between the slide and cover slip.**

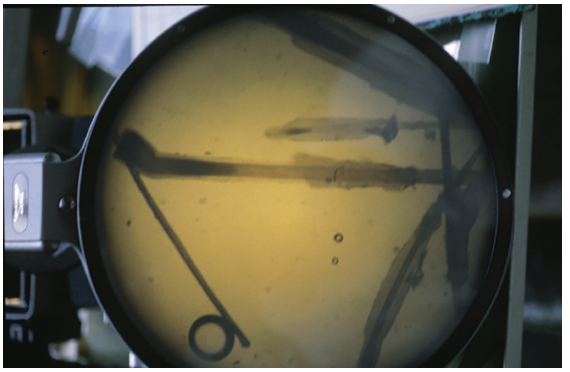


Figure 2.10 **Microprojector view for a trichogram.**

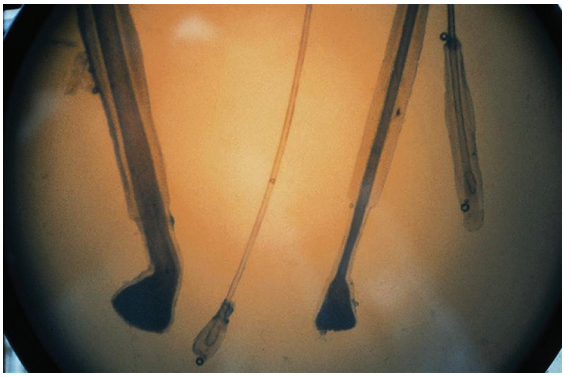


Figure 2.11 **View of the three hair evolution stages (anagen, catagen, and telogen).**

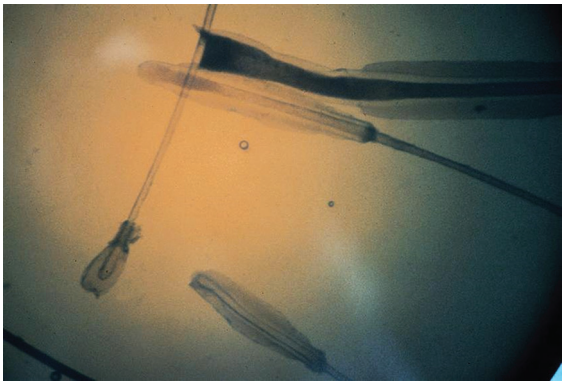


Figure 2.12 **Microscopic aspect of the three types of hair under magnification (×25): two anagen hairs, one catagen hair, and one telogen hair.**

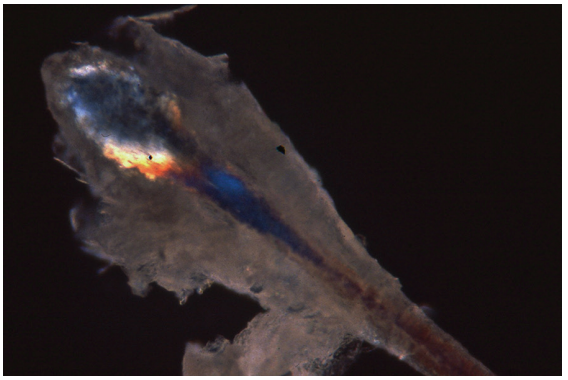


Figure 2.13 **Microscopic view of a telogen hair (in polarizing light).**

Figure 2.14 **Microscopic view of a catagen hair.**

Figure 2.15 **Dystrophic anagen hair visible in alopecia areata.**

Pathological changes on the trichogram

One can observe three types of abnormal hair cycle percentages:

- The telogen type in which the percentage of telogen is increased (>20%–25%); this is telogen effluvium.
- The dystrophic type in which the number of dystrophic anagen hairs is increased at the expense of normal anagen; this is anagen effluvium (e.g., in highly developed alopecia).
- The most common mixed dystrophic telogen type, in which there is an increase in both dystrophic anagen and dystrophic telogen (e.g., in androgenetic alopecia).

The trichogram is used to assess the diameters of the hair shafts and to highlight a decrease in the size of hair follicles, which shows the miniaturization characteristic of the androgenetic alopecia.

The goal of the trichogram

The trichogram has a role that is both diagnostic and prognostic. It is indicated in five situations:

1. In the case of any hair loss for physiological reasons, a normal trichogram can reassure anxious patients.
2. In case of any diffuse hair loss without alopecia, it can decide between telogen effluvium, androgenetic alopecia, and diffuse alopecia.
   a. Telogen effluvium (pregnancy, iron deficiency, acute infection, hemorrhage, accident, general anesthesia, weight loss, emotional distress, etc.) is characterized by the abrupt and synchronized passage of many follicles into the telogen phase, resulting in hair loss 2–3 months later. The trichogram shows at that time a large number of telogen hairs (>20% or 25%) in all regions of the scalp examined.
   b. Anagen effluvium (toxic or drug-induced, chemotherapy alopecia) is due to an abrupt halt in the growth of hair in the anagen phase without passage into the the telogen phase. The loss of hair occurs 4–6 weeks later. Thus, we find in the trichogram a high number of dystrophic anagen hairs. In alopecia areata, there may be a dystrophic form of hair cycle percentages, or a mixed dystrophic telogen type.
   c. In androgenetic alopecia, the formula for hair cycle percentages is normal on the occipital area, while the number of telogen hairs and therefore the anagen/telogen ratio decreases gradually as one moves toward the front area. In addition, the hairs have diameters of different sizes, with the presence of miniaturized hair. The trichogram has a prognostic value in androgenetic