Contents

	knowledgments ntributors	vii xi
1	How to Diagnose the Patient with Hair Loss Michael G. Buontempo and Jerry Shapiro	1
2	Patterned Hair Loss: P. crogenesis, Clinical Features, Diagnosis, and Management Michael G. Buontempo ard Jerry Shapiro	30
3	Telogen Effluvium: Pathogenesis, Clinical Features, Diagnosis, and Management Hsiao-Han Tuan and Jerry Surgiro	75
4	Hair Loss from Drugs and Raciation Kristen Lo Sicco and Lina Alhansha	88
5	Alopecia Areata: Pathogenesis, Cinncal Features, Diagnosis, and Management Michael G. Buontempo and Jerry Sl apiro	114
6	Cicatricial Alopecia and Inflammatory کوری کا Disorders Donna Cummins and Jerry Shapiro	167
7	Hair Restoration Surgery Nina Otberg	201
8	Non-Medical Approach to Hair Loss Nina Otberg, Kristen Lo Sicco and Lina Alhanshali	235
9	Religious and Cultural Headwear Lina Alhanshali and Nina Otberg	248
Ind	lex (5	253

TABLE 1.2 (Continued) Basic Terminology

TERM	DEFINITION	PARENT LOCATIONI AREA
Dermal papilla fibroblasts	Fibroblasts that reside in the dermal papilla and are involved in hair growth and regeneration.	Dermal papilla
Melanin	A pigment that gives color to hair and skin.	Hair matrix
Eumelanin	Brown or black melanin pigment.	Hair matrix
Pheomelanin	Yellow or red melanin pigment resulting from the incorporation of cysteine.	Hair matrix
Keratin fibrils	Long, slender filaments made up of the protein keratin that form the hair shaft.	Hair matrix
Trichilemmal keratinization	The process of keratinization that occurs in the hair shaft, resulting in the formation of the cuticle and cortex layers.	Hair shaft



FIGURE 1.1 Diagrammatic representation of hair anatomy: The hair follicle is divided into four parts: bulb, suprabulbar area, isthmus, and infundibulum.



FIGURE 1.2 (a) Histology of the hair follicle on longit: di al section showing dermal papilla (DP), matrix (M), inner root sheath (IRS), outer root sheath (ORS), and ibrous root sheath (FRS). (b) Two anagen follicles side by side at the level of fat. Note the melanocytes within the matrix providing pigment to the hair. (Courtesy of Dr. Magdalena Martinka and Dr. David Shum.)

	egments		
HAIR FOLLICLE SEGMENT	D.FSC."", ION		
Infundibulum	Within the upper portion of the follicie, the infundibulum extends from the follicular orifice to the sebaceous gland.		
lsthmus	Within the middle portion of the follicle, the istbous extends from the sebaceous gland to the insertion of the arrector pili muscle.		
Suprabulbar area	Within the lower portion of the follicle, the suprabulbar area extends from the insertion of the arrector pili muscle to the matrix.		
Bulb (Figure 1.3)	Within the lower portion of the follicle, the bulb contains the dermal papilla, the matrix which is interspersed with melanocytes.		

TABLE 1.3	Hair Follicle	Segments
-----------	---------------	----------

blue and metachromatically for toluidine blue. The ground substance consists of not only non-sulfated polysaccharides such as hyaluronic acid but also sulfated mucopolysaccharides such as chondroitin sulfate. Increased activity of alkaline phosphatase can be found in the anagen phase.

The **hair matrix**, located above the dermal papilla, is responsible for the formation and growth of the hair shaft, and is highly sensitive to hormonal and environmental changes.



FIGURE 1.3 (a) Close-up of logitudinal section of dermal papilla (DP), which is an invagination of the dermis into the matrix (M). The DP allows capillaries to gain entrance to the cells of the matrix. The signal transduction and communication between the *p*⁻ and the matrix determines how long a hair will grow and how thick a shaft will be produced. Melanocytes for the matrix and produce the pigment of the hair. (b) Cross-section of the follicle at the level of the dermal *p*-pilla. (Courtesy of Dr. Magdalena Martinka.)

The hair matrix has large vesicular nuclei and a deeply basophilic cytoplasm, with DOPA-positive melanocytes interspersed between the casel cells of the matrix. *Melanocytes* are dendritic neural crest-derived cells that migrate into the orderwis in the first trimester. *Melanin* is a complex quinone/indole-quinone-derived mixture of biopolyme produced in melanocytes from tyrosine (46). Melanin is incorporated into the future cells of the hair chaft through phagocytosis of the distal portion of the dendritic melanocyte (Figure 1.4). *Melanosome* or the hair follicle are larger than those of the epidermis



FIGURE 1.4 Melanosomes, either eumelanin or pheomelanin, during anagen are transferred from melanocytes to matrical cortex cells via dendritic ends.



The *glassy or vitreous membrane* forms a homogeneous eosin philic zone peripheral to the ORS, is periodic acid–Schiff (PAS) positive, and is diastase-resistant. It mers from the interfollicular basement membrane zone by being much thicker. It is thickest around the lower third of the hair follicle in the

suprabulbar area. The fibrous root sheath is composed of thick collagen bundles.

Hair Cycling

Each hair follicle undergoes a unique recurring cycle, consisting of four distinct phases: **anagen** (growth), **catagen** (transition), **telogen** (resting), and **exogen** (shedding) (Figure 1.6). The structure and contents of the hair follicle are remarkably dynamic and vary depending on which of these stages the hair follicle is in, as will be mentioned later. Clinically, this allows for biopsies of the scalp to determine the exact stage a hair follicle is in within **hair cycling disorders**.

As it changes throughout the hair cycle, the hair follicle itself can be divided into three distinct regions: the lower, middle, and upper portions (Figure 1.7). These portions can be defined as permanent or



FIGURE 1.7 During the hair cycle, the middle and upper portions of the hair follicle are the permanent segments of the hair follicle, while the lower portion is non-permanent. (a) The growing or anagen hairs are anchored deeply within the subcutaneous fat and cannot be pulled out easily. The telogen hairs are located higher up in the dermis and can be pulled out relatively easily. The scalp consists of almost 90% hairs in anagen, 1% in catagen, and 10% in telogen. Anagen may last up to 2–6 years, telogen 3 months, and catagen 3 weeks. This ratio is usually uniformly distributed over the entire scalp. The dermal papilla (DP) is pulled upward with each cycle, and during telogen is closely associated with the stem cells of the bulge area. Communication signals between DP and stem cells of the bulge probably determine the length of anagen and the matrix girth of the next hair cycle. (b) The newly formed anagen hair pushes out the previous telogen hair.

Unlike most fur-bearing animals, where the hair cycle is synchronous, the human scalp has an asynchronous mixture of actively growing (anagen) and resting (telogen) hairs. In a healthy scalp, 80%–90% of hair follicles are in the anagen phase (55). The normal anagen-to-telogen ratio for scalp hair is 9:1, with seasonal variations observed (56, 57). The scalp sheds about 100 telogen hairs per day. Normal scalp skin also contains a variable number of small vellus hair follicles. These follicles are present throughout the body, except for the palms and soles, with different densities in different body sites. The hair shaft of vellus hair is less than 30 μ m thick, lacks color, and is anchored in the *mid-upper dermis* (Figure 1.8) (58).



FIGURE 1.8 Vellus-like hairs are less than 0.03 min in diameter and rarely grow more than 1–2 mm. Terminal hairs are coarse, over 0.06 mm in diameter, and can gro *N* up to 3 feet. A true vellus hair does not have an attached arrector pili muscle. Only miniaturized vellus-lite matrix of androgenetic alopecia have arrector pili muscle.

PATIENT APPRCACH

In dermatology, it is essential to accurately diagnose hair loss in order to provide effective treatment and ensure patient satisfaction. The following five steps are crucial in making the correct diagnosis and developing a comprehensive treatment plan (Figure 1.9):

- 1. Listen
- 2. Look
- 3. Touch
- 4. Magnify
- 5. Sample

Hair loss is a common concern for patients and can be caused by various factors, including genetic predisposition, systemic illness, drugs, endocrine abnormalities, psychological distress, diet, trauma, infections, autoimmunity, and structural hair defects. As hair is one of the fastest-growing tissues in the body, any metabolic or hormonal changes can result in shedding and alopecia. Given the multitude of potential causes of hair loss, obtaining a thorough history is critical in developing an initial differential diagnosis.