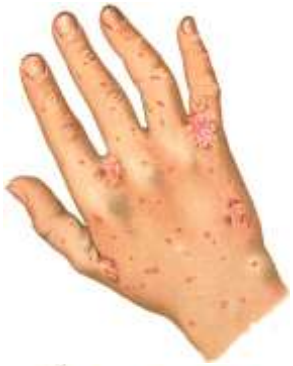
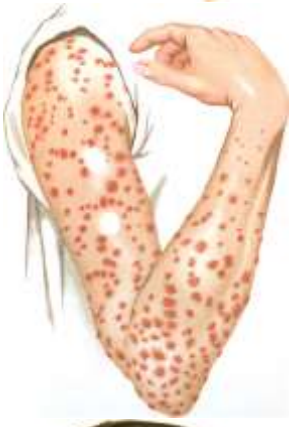
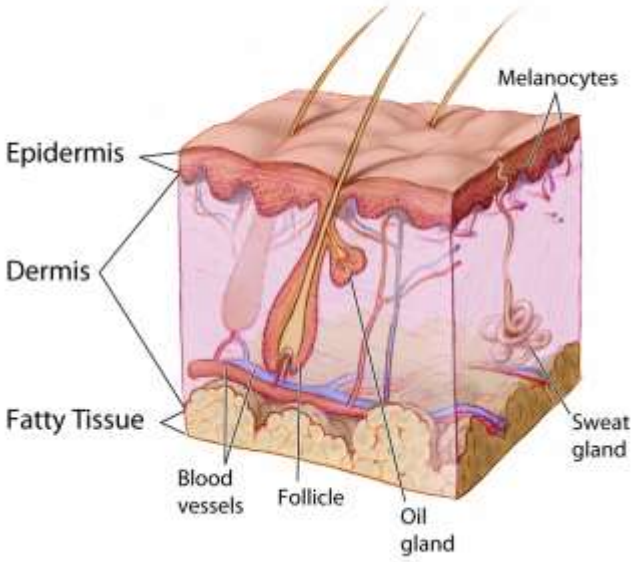
جامعة الملك سعود
King Saud University





432 Teams

Dermatology



Hair disorders

Color Code: Original, Team's note, Important, Doctor's note, Not important, Old teamwork

Done by: Shaikha Aldossari

Reviewer: ***Lama AlTawil***

Team Leader: ***Basil Al Suwaine&Lama Al Tawil***

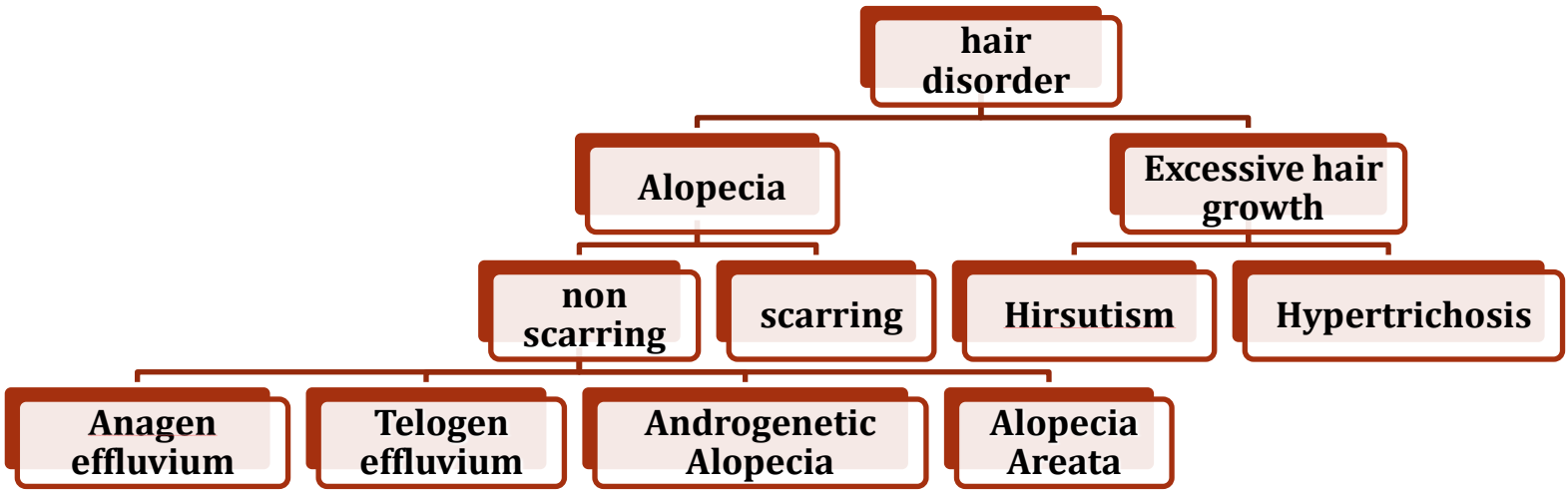
Objectives

1- Normal anatomy of hair follicle and hair cycle.

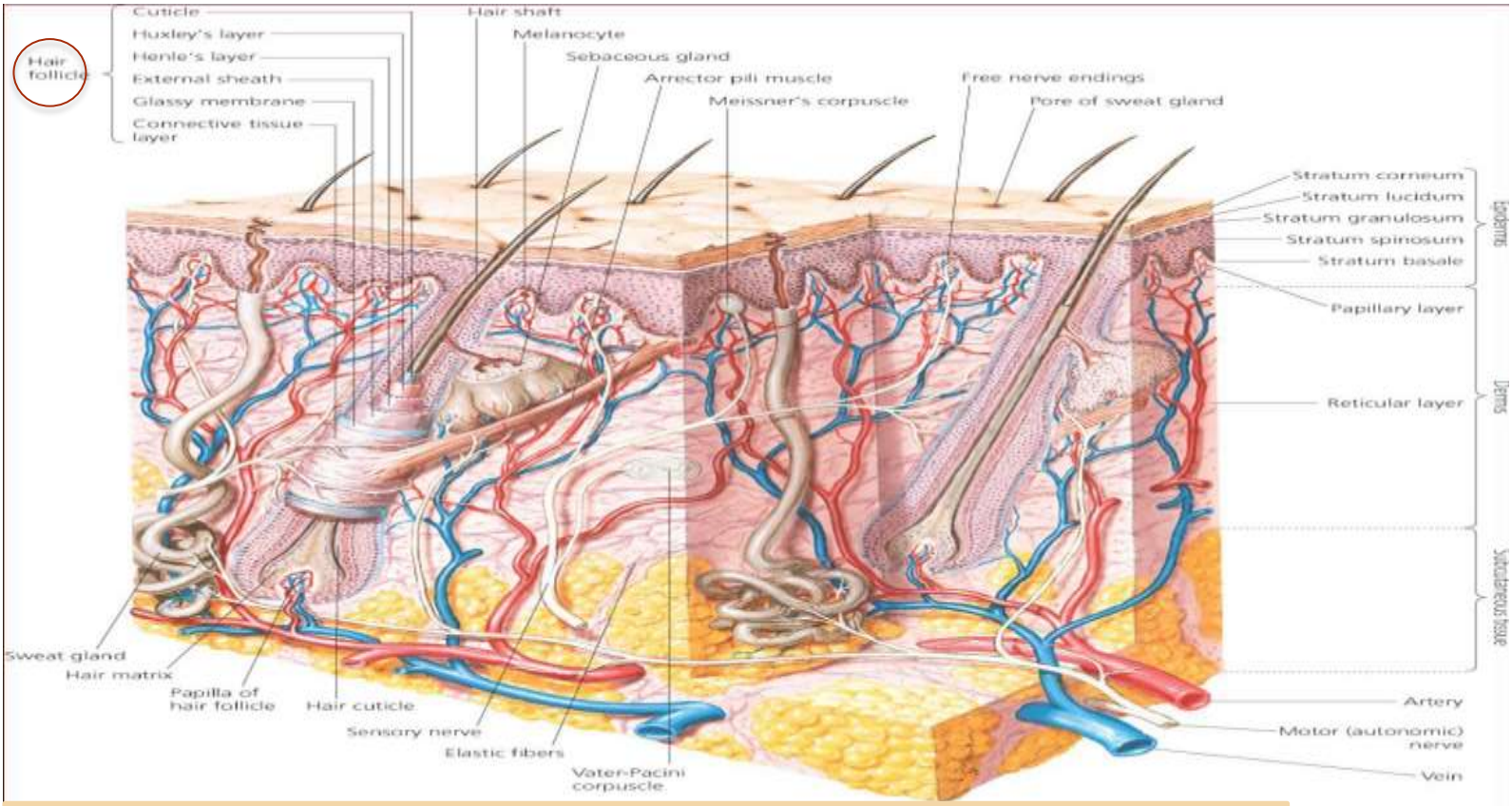
2- Causes, features and management of non scarring alopecia, particularly:
- Alopecia areata
- Androgenetic alopecia
- Telogen effluvium
- Anagen effluvium

3- Causes and features of scarring alopecia.

4- Causes and features of Excessive hair growth.



Anatomy of hair follicle:



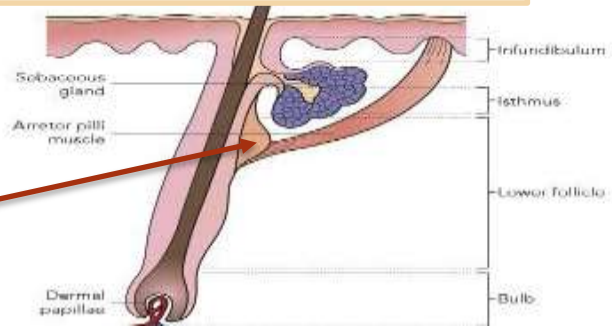
The Arrector pili→Responsible for piloerection (goose bumps) that happens when one is cold (produces energy and therefor warmth) . hair follicle becomes vertical instead of oblique
Cuticle →is the last layer here . what we can see outside . it has 7 layers of keratinocytes

- **How many hairs in the body?**

5 millions hairs in the body, 100,000 in the scalp.

- **Growth rate:**

0.3mm/day for scalp hair i.e.1cm/month



Hair follicle bulge:

-Very important part since it has stem cells .its the inertion of the arrector pili
-So any pathological process affecting any part **other than this**, hair would still be able to regrow.
-If we want to destroy a hair follicle, we'd target the bulge. Which is what happens when you go for laser hair removal the inflammation will attack and destroy it
-The hair bulb forms the base of the hair follicle
-Thick hair = thick hair follicle.
The insertion of the hair follicle is at 45 degrees and its creation is at the hair bulb

Hair follicle on vertical section:

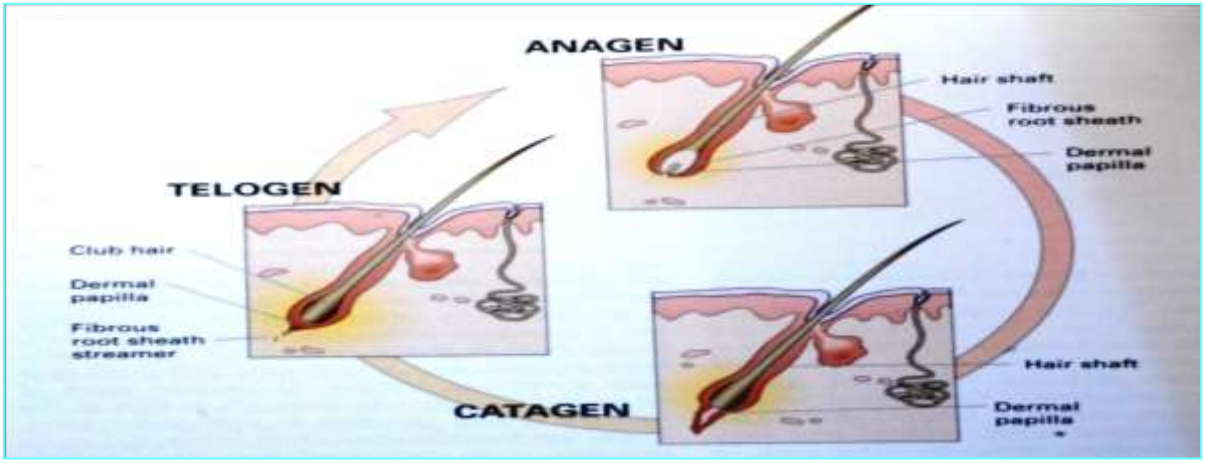
Initially the shaft and the follicle are one organ then when you reach 1/3 the follicle the shaft separates
Vertically the hair is divided into
1-infndibulum →from the follicle opening till the sebaceous gland opening
2-isthimus →opening to the sebaceous gland till the arrector pili (here you find the bulge)
3-lower follicle
4-bulb

- ***Hair type:***

1. Lanugo: covering fetus and newborn baby.
2. Vellous: thin, light, fine ,present all over the body.
3. Terminal: thick and dark-colored, seen for example, on scalp, eyebrow or axilla.
4. Androgenic hair: thick but androgen dependent area crown area (Grow during & after puberty in males & females (e.g. axilla, pubic area). So in case of an androgen problem you may develop androgenic alopecia

- ***Hair Cycle:*** "ACT"

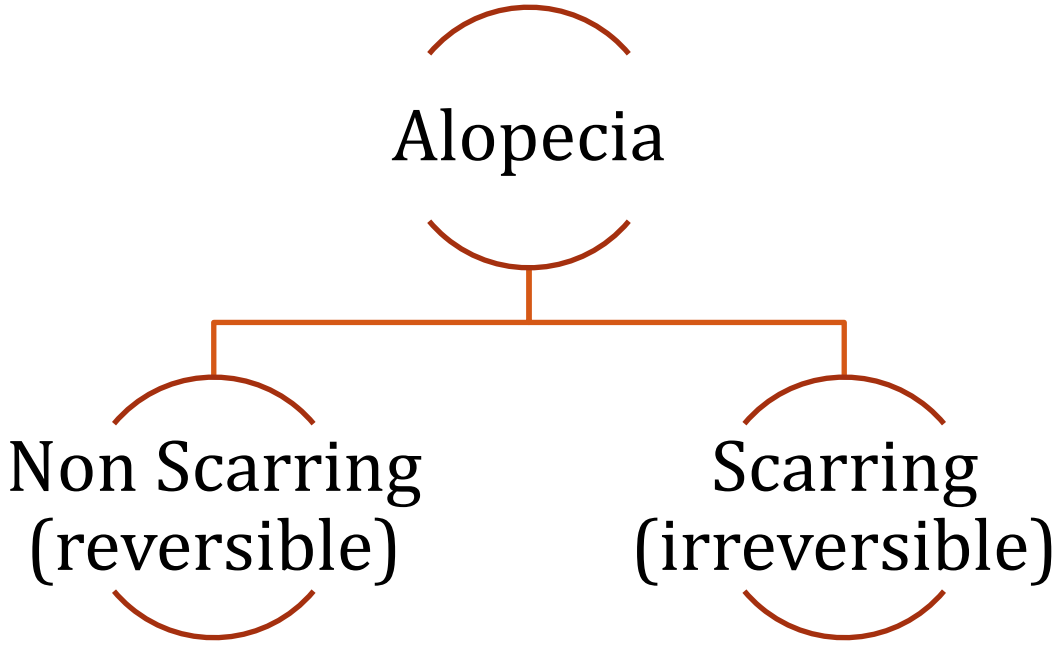
Phase	Region	Time	Description
Anagen	Scalp	2-5 years	**Growing** of hair. The length of this phase determines the length of the hair. **85-90%** of hair is in this phase. Longer Anagen phase = Longer hair. (this is part of the reason why not all hair products and oils work for lengthening) it's a genetically determined phase.
Catogen	Scalp	2 weeks	A short **transition phase** of conversion from active growth to the resting phase with degradation of hair follicles. <5% of hair is in this phase.
Telogen	Scalp	2-3 months	A **resting phase** at the end of which the hair is shed and new hair grow. 10-15% of hair is in this phase. It has a bulb or **club-shaped hair and a fibrous root sheath streamer**



Hair growth is very dynamic

Notes:

- Average hair loss per day is **100-150**. (in telogen phase)
- The phases are equally distributed and intermingled together evenly
-Hair fall during Anagen phase or if the cycle is too rapid = Strong pathologic process.
-The closer the damage to the roots, the worse the outcomes. However split ends are due to environmental factors and needs to be trimmed as it's the only treatments (weathering effect)
-Could shampoos lengthen hair? No.
-Could conditioners cause dandruff? No. on the contrary in seals the gaps and makes the hair shiny
-Frizzy (fly away) hair is a sign of a potential hair damage.
-Hair highlights disturb keratinocytes layered organization in the cuticle →no longer parallel due to the holes→ light won't reflect evenly → hair gets less shinier (first sign of hair damage). Oils are temporary moisturizers
-A scar means loss of hair follicle opening and therefore loss of its ability to regrow. It needs to be treated to prevent progression. However the original damage is irreversible and since it's fibrotic it wont respond to grafting
- Keratin hair treatment:
1. It breaks keratin bond so it weakens (straightens) hair, so it was basically designed to suit african american hair.
2. It's carcinogenic! Especially Brazilian one. Because it contains > than 1% formaldehyde . new types don't have the li relaxa
- **Hair botox** is the least damaging option for hair . Good for dyed hair that's frizzy (it's temporary)
-protein has no formaldehyde but when it interacts with hair its produces formaldehyde
- It's better to put a 6-week gap between hair treatment sessions rather doing everything at once. (hair dye, straightening…etc)
All the above treatments cause an acquired hair shaft damage . and any hair damage is usually cumulative



Nonscarring alopecia	Scarring alopecia
Telogen effluvium *most common	Developmental defects (e.g., Aplasia cutis)
Anagen effluvium	Infections (bacterial, viral, fungal)
Alopecia areata	Trauma (irradiation, thermal or caustic burns) 3rd degree burns are scarring
Androgenetic alopecia	Neoplastic disorders
Hair shaft abnormalities	Lichen planus (lichen planopilaris), lupus erythematosus, morphea, scleroderma, sarcoidosis Esp. discoid lupus
Trauma (e.g., traction)	Keratosis pilaris atrophicans
Infectious disorders (e.g., dermatophyte, syphilis)	Folliculitis decalvans
Systemic diseases (e.g., thyroid, systemic lupus erythematosus, iron-deficiency anemia)	Dissecting cellulitis of the scalp
Intoxications (e.g., vitamin A, Bismuth)	Acne keloidals
Nutritional deficiencies (e.g., zinc, biotin)	Pseudopelade
Medications	Alopecia mucinosa

Alopecia

• *Non-scarring alopecia: (reversible)*

1- Alopecia Areata: (الثعلبة البقاعية)

- Sudden hair loss (localized or generalized). Involves all stages
- Alopecia Areata affects up to 2%.
- 75% Self recovery with 2-6 months.
- 30% +ve Family history.
- Autoimmune. Mainly lymphocytes

Thinner hair as we move centrally

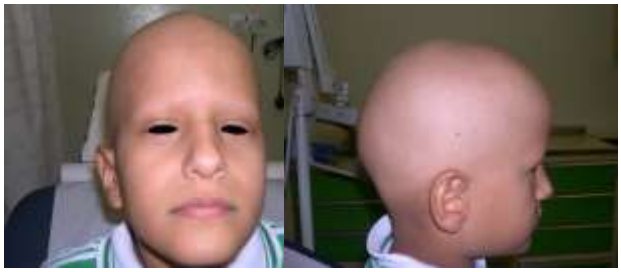
Thick hair as you come closer to the scar thin

○ *Clinical findings:*

- Well demarcated non-scarring hairless patch.
- Exclamation point. (!)
- Normal scalp. you need to rule out inflammatory lesion due to scales , infections
- Nail: pitting, ridges (indicating severe alopecia).

○ *Types of alopecia areata:*

- Localized partial (1-2 patches). Most common
- Localized extensive (more than 2-7).
- Alopecia ophiasis (occipital and parietal area).
- Alopecia totalis (Total hair loss in the sc alp).
- Alopecia universalis (whole body).



Alopecia universalis hint no eyebrow hair

○ *Bad prognostic signs:*

- Young age.
- Atopy.
- Alopecia totalis, universalis, ophiasis.
- Nail changes.

Alopecia ophiasis

○ *Diagnoses:*

- Clinically
- H/E stain : swarm of bees (inflammation around hair follicle)
- Hair pull test from the periphery or the alopecia areata if it falls its an active inflammation

- ***Treatment:***
  - Observation. If it's a baby , late presentation , or –ve hair fall test
  - Intralesional Corticosteroids. (drug of choice) in adult or older children and in the active phase
  - *Skin Sensitizers: immune modulator
    - Anthraline.
    - Diphencyclopropenone (DPCP).
  - Others:
    - Topical steroids (under **occlusion** i.e shower cap to increase potency if intralesional can't be used) & Minoxidil. (just an adjuvant that's a vasodilator of the hair follicle and it prolongs anagen phase with an increase of the growth rate)
    - Systemic Steroids. For severe **active** process.
    - Cytotoxic Rx. methotrexate
    - Phototherapy (PUVA). In case it was extensive and unresponsive.2nd line works as localized immune suppressor
    - **Hair Transplant – (NO!)** because the immune system will attack it.
      -Scalp skin is a thick skin so it needs potent therapy.
      -*Skin sensitizers are used to fool the immune system into thinking that the sensitized area need to be protected so it stops damaging the previous area and redirects into the new sensitized area. In the past they used garlic

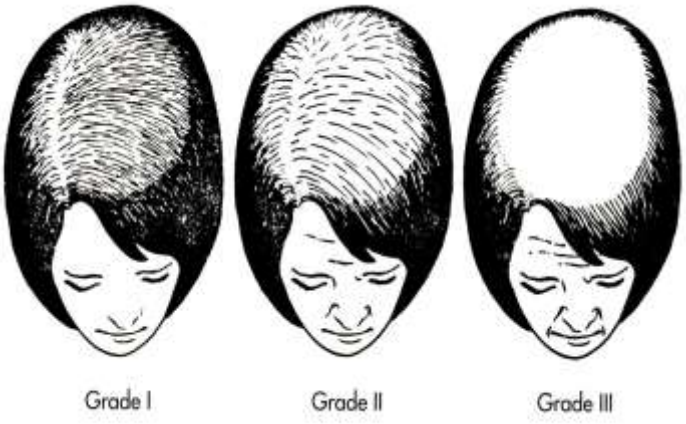
Manegment (from doctor)		
Localized		
	Children	**Adults**
First	Topical steroids	Intralesional Corticosteroids
Second	Skin Sensitizers	Skin Sensitizers
Totalis		
First	Skin Sensitizers	
Second	Systemic Steroids	
Universalis		
Skin Sensitizers + Systemic Steroids		

2-Androgenetic Alopecia(Male and Female Pattern Hair Loss)

- Androgen dependent loss of scalp hair
- Androgenetic Alopecia affects up to 50% of males and 40% of females
- Autosomal dominant with variable penetrance
- 85% : +ve family history especially from the mothers side

Testosterone → (5 Alpha reductase) → **DihydorTestosterone (Active)** → **Accelerate telomerase** → **Miniaturization of Terminal Hairs**
-Terminal to villous hair.
-Hair thinning NOT falling!

Male Pattern Hair Loss (Hamilton stages)
1.Frontoparietal recession (bitemporal rescission)
2. Two foci the second involves the crown
3.Receding of frontal hairline
(note that 1+3 don't occur in females)
4. merging together
Female hairline is always present (never reach baldness) but she gets crown hair thinning

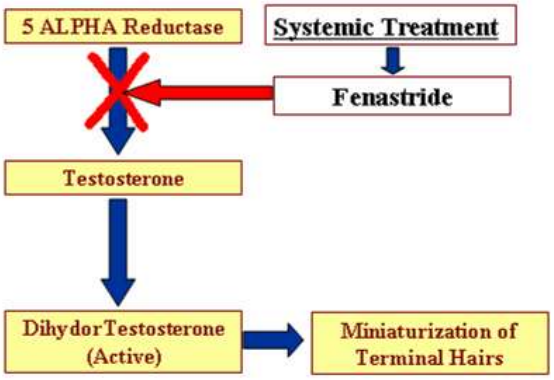


Female Pattern Hair Loss (Ludwig)

Left : male right :female notice no frontal recession in the female

- Male pattern hair loss: It starts with thinning; it is called fronto- parietal recession and then it goes upwards. It usually spares the Temporal and occipital areas.
- Female pattern hair loss: There is no fronto-parietal recession and no frontal recession, so the frontal hairline is preserved. There is never complete baldness, there is thinning only. It is more common in postmenopausal women.

- ***Treatment:*** most are in stage 1 and don't need treatment . and treatment is long life because the hair follicle is sensitive to the normal androgen
  - Topical:
    - *Minoxidil , **Neoxidil** 2% (females)- 5% (males or severe female balding)solution. It's a foam and needs 3 months before effect is evident
  - Systemic:
    - Spironolactone. Anti-androgenic diuretic
    - Fenastride mainly for males
    - OCP. (like diane and yasmin)
  - Hair transplant.



3- Telogen effluvium:

- **Acute** alopecia.
- Reversible (but may be become chronic).
- **3-4 months** from trigger.

Causes:

Physiologic
Physiologic effluvium of the newborn
Postpartum effluvium

Injury or stress
High fever
Severe infection
Severe chronic illness
Major surgery
Hypo- or hyperthyroidism
Crash diets, precipitous decrease of calories or protein (Fig. 11.38)
Iron deficiency
Essential fatty acid deficiency
Biotin deficiency
Drugs (Table 11.8)

*Causes include: **hyper/hypothyroidism and pregnancy**.
-in hyperthyroidism, hair falls in telogen but the **cycle itself is faster than usual.**
-Estrogen retains hair, once a pregnant woman delivers, hair falls.
-It'd regrow 2-4months later.
-Pregnant + Anemic or vit.D deficiency or bleeding → faster rate of hair fall , early and more hair loss and restoration takes longer time. Treat underlying cause (anemia)
-anesthesia and surgery → ask about whether they were exposed to it in the past 3-6 months because the effect is delayed
Period status

Diagonosis :

- Hair pulling test → you take 50 hairs and pull if it was 3 or more its positive
- Then do the hair parting test to see the hairline if its wider in the crown then its female apattern ndrogen hair loss , but if its equally thinning till the occiput and the hair pull test is positive then its telogen effluvium
- Labs → vit.D , ferritin (not iron) , if old woman add TFT

Treatment:

- Remove or treat the cause.
- Minoxidil **2% Solution**.

Note :

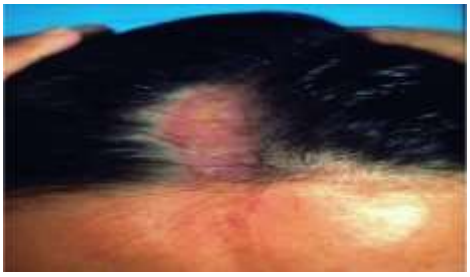
- Hair shedding when combing or showering is in telogen effluvium
- Hair breaking is in acquired shaft problems → here you don't see the clubbing and the length of hair is not equal
- Hair thinning →androgen pathology

4- Anagen effluvium:

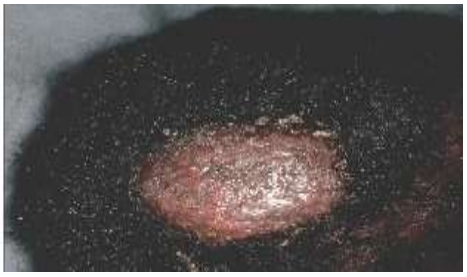
- Always related to **cytotoxic chemotherapy**
- Acute and severe alopecia.
- Mostly reversible but not always.
- **2-3 week** from trigger.
- Can be bald because Anagen represents around 90% of hair, unlike telogen which is 10 to 15%

• *Scarring alopecia: (irreversible)*

- SLE—DLE.
- Lichen Planus.
- Sarcoidosis.
- Leprosy.
- *Kerion. Fungal and inflamed and severe another type is favus
- Trauma.



Systemic sclerosis (en coup de sabre)



*kerion: inflamed, well demarcated and boggy if it wasn't inflamed and there is a hair follicle the ddx would be alopecia areata

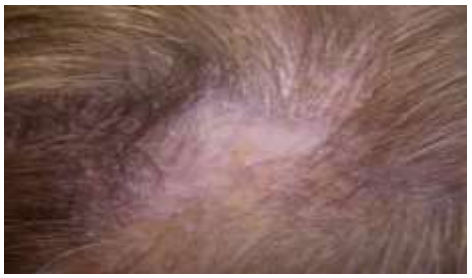


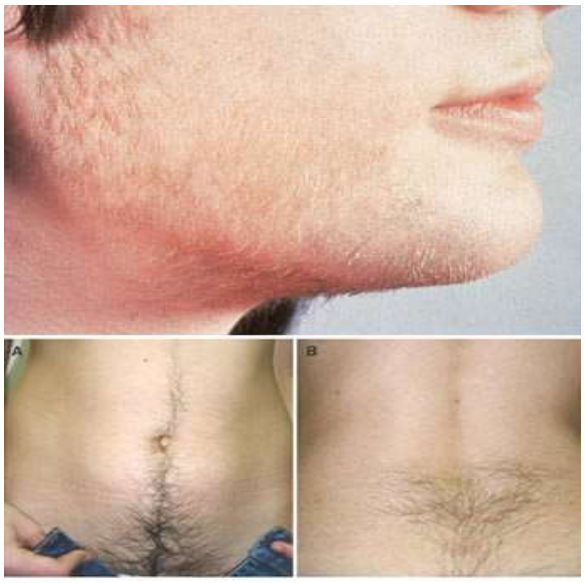
Figure 31-17. Scarring alopecia of scalp: lichen planopilaris (LPP) The frontal hairline has gradually receded; the area of alopecia lacks the pigmentation of forehead skin, which has had lifelong sun exposure. Both eyebrows have no hair; the eyebrow on the right is penciled in. The eyelashes appear normal. No other clinical findings of LP were detected. This clinical variant of LPP is called frontal fibrosing alopecia.

Perifollicular white ivory scale with inflamation and loss of follicles = Lichen planopilaris

Lichen Planopilaris (LPP):

a rare inflammatory condition that results in patchy progressive permanent hair loss mainly on the scalp unlike discoid which will have depigmentation

Excessive hair growth (wasn't in the female slides)

Type	*Hirsutism*	*Hypertrichosisause*
Defination	Excess growth of androgen-dependent hair in a male pattern affecting Female.	Excess growth of hair in a non-androgenic pattern affecting both sex.
Cause	Idiopathic (the commonest). Adrenal, pituitary. Ovarian (PCO). Turner syn. iatrogenic (drug).	Congenital. Acquired: drug, porphyria, endocrine (thyroid , anorexianervosa).
Tretment	Underline cause + laser	
Pictures		

From Fitzpatrick's:

(some of these conditions were briefly mentioned by dr. Ghada Bin Saif)

Alopecia Mucinosa (Follicular Mucinosis)

• Erythematous lesions (papules, plaques, or flat patches) of alopecia, occurring mainly on scalp and/or face.
• Dermatopathology: prominent follicular, epithelial/sebaceous gland mucin, perifollicular lymphohistiocytic infiltrate without concentric lamellar fibrosis.
• May be symptom of cutaneous **T-cell lymphoma**

Follicultis Keloidalis Nuchae

• Synonym: acne keloidalis (nuchae).
• Occurs most commonly in black men.
• Usually occurs on the occipital scalp and nape of the neck, starting with a chronic papular or pustular eruption (Fig. 31-21).
• Keloidal scar formation may occur.
• Distribution: nape of the neck, occipital scalp.
• Early mild involvement may respond to **intralesional triamcinolone**. If S. aureus is isolated on culture, treat with appropriate antimicrobial agent.

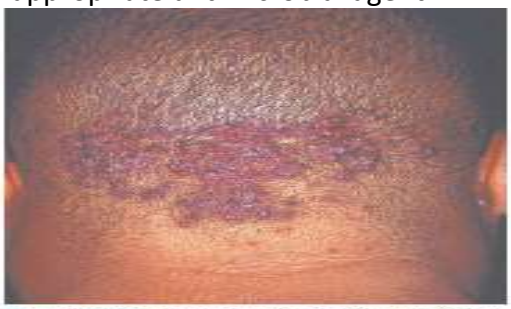


Figure 31-21. Scarring alopecia of scalp: follicu-litis keloidalis. A 31-year-old black male with papular scars of 3 years' duration, and follicular pustules becoming confluent on the occipital scalp and neck.

Pseudofolliculitis Barbae

- *Synonyms:* "razor bumps."
- Occurs commonly in black men who shave.
- Related to curved hair follicles. Cut hair retracts beneath skin surface, grows, and penetrates follicular wall (transfollicular type) or surrounding skin (extrafollicular type), causing a foreign-body reaction.
- Distribution: any shaved area, i.e., beard (Fig. 31-22), scalp, pubic.
- Keloidal scarring in varying degrees occurs at involved sites.
- *S. aureus* secondary infection is common.

Acne Necrotica

- Pruritic or painful erythematous follicular-based papule with central necrosis, crusting, and healing with depressed scar.
- Lesions occur on anterior scalp, forehead, nose; at times, the trunk.
- Dermatopathology: lymphocytic necrotizing folliculitis.

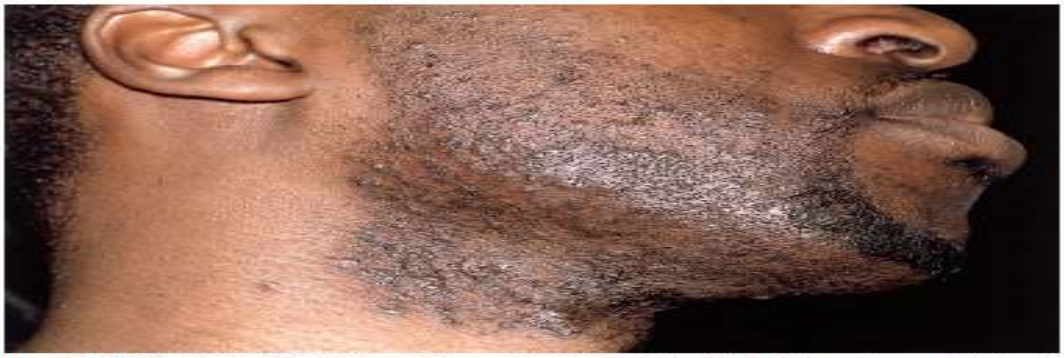


Figure 31-22. Pseudofolliculitis barbae. A 29-year-old black male with multiple follicular papular scars in the beard; the presence of follicular pustules usually indicates secondary *Staphylococcus aureus* folliculitis. Folliculitis keloidalis is often seen on the occipital scalp and neck (see Fig. 31-21).

Summary (431 team)			
Disease	***feature***	***Clinical Findings***	***Treatment***
Alopecia Areata	Reversible. 30% of Down syndrome. 75% self recovery.	Well demarcated. Exclamation point. Normal scalp.	▪ Adults: Localized: Intralesional steroids. Generalized: sensitizers. ▪ Children: topical steroids.
Androgenetic Alopecia	50% of males. 40% of females. Autosomal dominan.	▪ Males: fronto- parietal recession. ▪ Females: After menopause. No baldness.	Minoxidil 2%-5% solution. Fenastride.
Telogen Effluvium	From any chronic disease.	Chronic.	Treat the cause + Minoxidil.
Anagen Effluvium	From chemotherapy.	Acute complete hair loss, but reversible.	

MCQs

1- A 40 year old lady with lymphoma on chemotherapy. Soon after starting chemotherapy she lost all of her hair. In what phase of hair cycle, the most likely the defect in her hair?
A. Anagen.
B. Catagen.
C. Telogen.
D. Unknown.

2- A 31-year-old obese male patient who did diet and lost 35Kg of his weight over 4 months presented with diffuse hair fall.
What is the most likely diagnosis ?
a. Androgenetic alopecia.
b. Telogen effluvium.
c. Anageneffluvium.
d. Alopecia areata.

3- 32 years old male presented to the dermatologist complaining of hair loss. On Examination, there were multiple well-defined smooth patches over the vertex area of his scalp.
Which of the following could be a bad prognostic sign for the condition he is suffering from?
A- History of atopy.
B- Mucus membranous involvement.
C- Diabetes mellitus.
D- Rapid progression of the disease.

Answers:
1-A.
2-B.
3-A