Lecture (6)
Hair disorders and pigmented disorders of the skin

Objectives: not given.

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Color index: slides, doctor notes, extra explanation.

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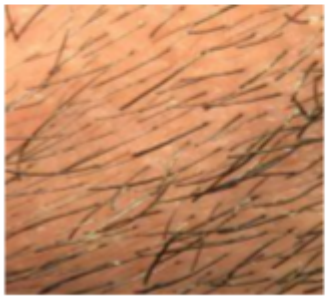
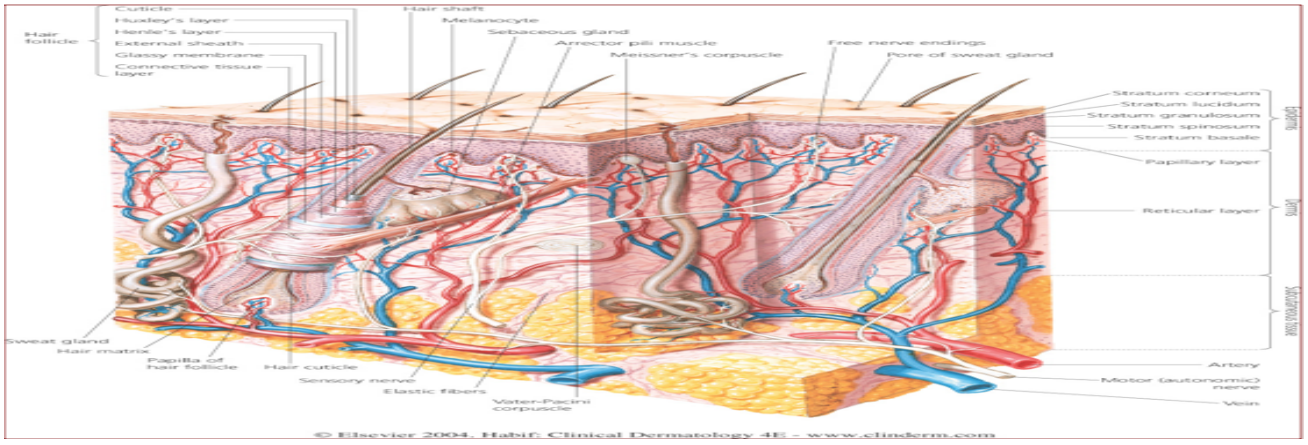
Introduction

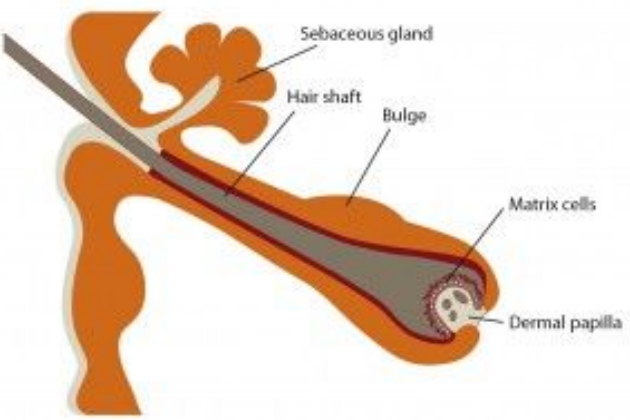
How many hairs are in the body? 5 million hairs; 100,000 in the scalp.
Growth Rate? 0.3mm/day for scalp hair.

Types of Hair		
Terminal	**Villous**	**Lanugu**
Thick, pigmented hair. 90% of scalp hair.	Short, fine, unpigmented hair. 5% of scalp hair. Superficial.	Soft, fine, pigmented hair. Present on fetus. Sheds before/after birth.
		

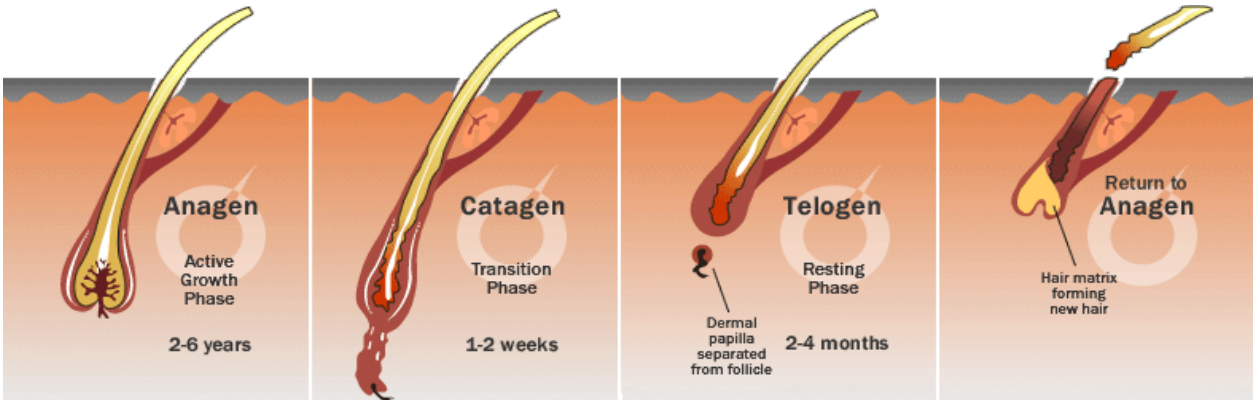
Structure of Hair Follicle



- **Dermal Papilla:** contains a rich supply of capillaries that provide nutrients and oxygen for the hair follicle.
- **Arrector pili muscle**: ONE muscle attaches to multiple follicles. Contractions form "**goose bumps**"
- **Bulge:** reservoir for stem cells
- **Sebaceous gland:** Produce sebum (moisturization)



Hair Cycle



Anagen: Hair growth phase (2-6 yrs)

Catagen: Transition phase (2 weeks) Dying phase

Telogen: Resting phase (2,3 months)

Anagen Phase:

- **Hair grows** during Anagen phase.
- Duration: **(2-6 years).**
- About **85-99%** of hairs will be in Anagen phase.
- With age, the Anagen phase may shorten.

Side note:

Different hairs have different different synchronizations. Which explains why our hair doesn't shed at once.

Catagen Phase:

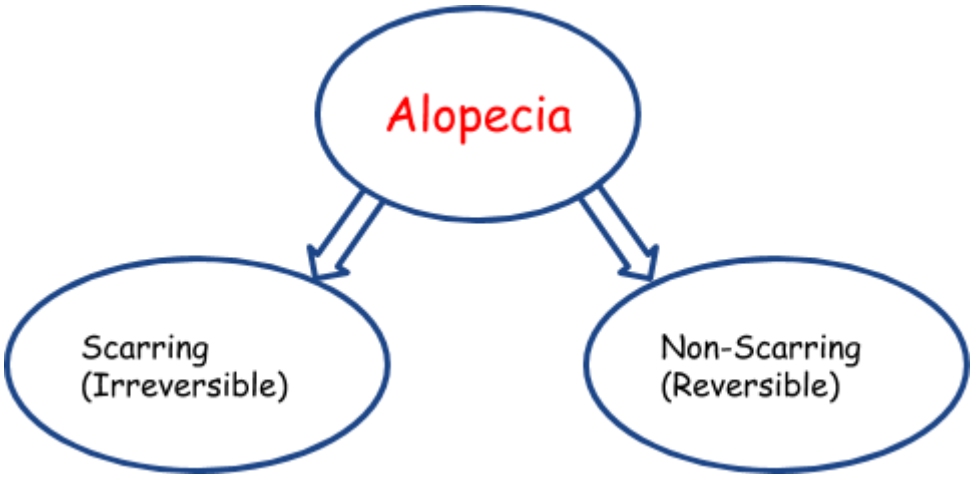
- Apoptosis-driven phase **(dying phase)**[1]**.**
- Duration: **(2 weeks).**
- About **1%** of hairs are seen in this phase.

Telogen Phase:

- "Resting phase".
- **Normal hair loss (shedding)** during this phase.
- Telogen hairs are **CLUB HAIRS** with depigmented proximal ends.
- Duration: **(2,3 months).**
- About **10%** of hairs are in this phase.

[1] Doctor described this phase as dying phase. Other resources have described it as **transition phase** and **degenerative stage.**

Hair Disorders



Hair fall can be diffused or localized, but most patients present with both.

Androgenetic Hair Fall usually spares occipital and temporal hair.

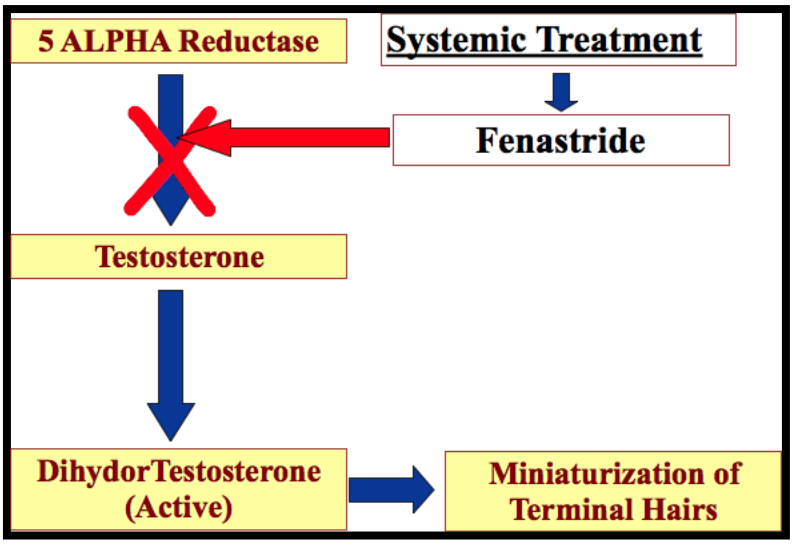
Androgenetic Alopecia (Male/Female pattern baldness) :

- Most common type of hair loss
- Androgenetic Alopecia affects up to 50% of males and 40% of females
- Autosomal dominant with variable penetrance
- 85% : +ve family history

Pathophysiology:

1. **5-alpha reductase** enzyme converts Testosterone → Dihydrotestosterone (DHT).

2. **DHT → Miniaturization**[2] of terminal hair.



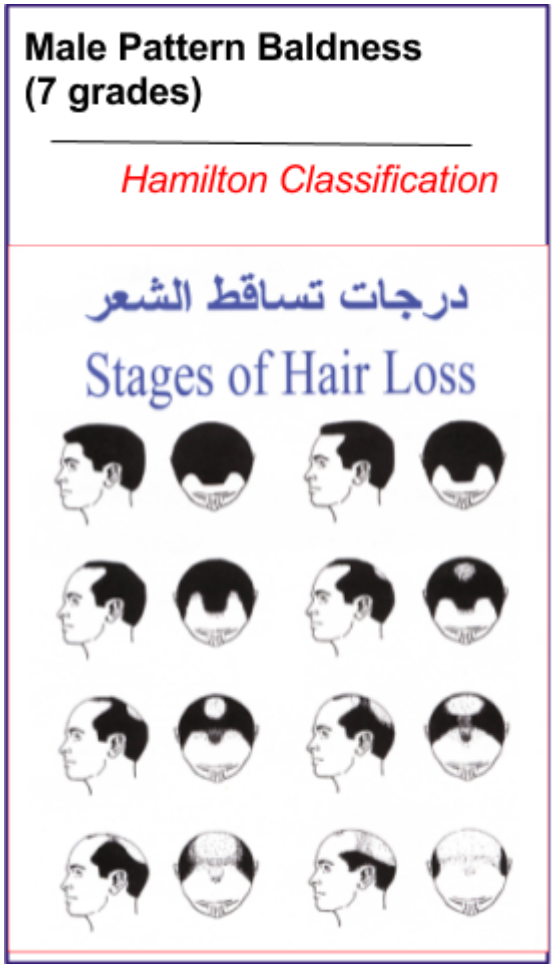
[2] a phenomenon in which strands of hair become thinner, shorter, more brittle, or weak with each successive progression through the hair growth cycle.

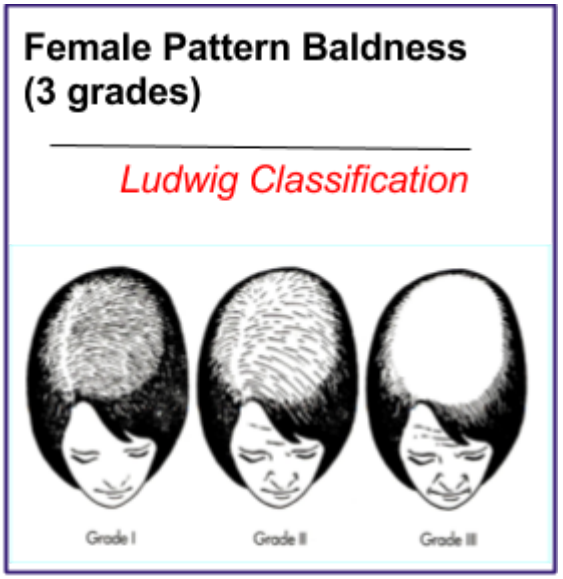
Treatment:

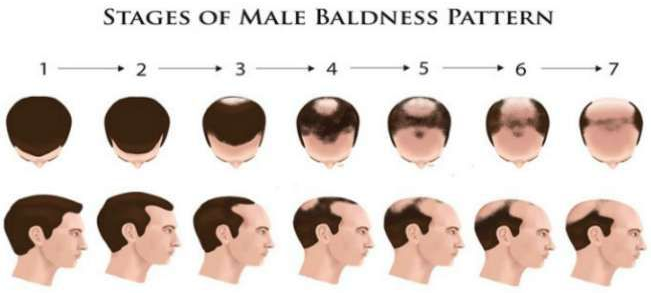
- Topical: Minoxidil 2% - 5% solution.
- Systemic: Finastride (blocks 5-alpha reductase).

Side effects of treatment:

- Initial side effect: *Temporary* hair fall.
- **Hypertrichosis[3] !!!**
- Allergic contact dermatitis.







[3] Hypertrichosis actually refers to excess hair (terminal or vellus) in areas that are not predominantly androgen dependent. Hirsutism is defined as the excessive growth of thick dark hair in locations where hair growth in women usually is minimal or absent

Non-scarring alopecia (reversible)

2-Alopecia Areata الثعلبه

Definition: Alopecia areata is a type of hair loss that occurs when your immune system mistakenly attacks hair follicles , which is where hair growth begins.

Alopecia Areata affects up to 2%.
30%: +ve Family history.
75% : Self recovery.
Role of immune system in the pathogenesis.

Clinical findings:

1-Well demarcated non-scarring hairless patch.
2-Nail: pitting, ridges.

Bad prognostic signs:

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

Types of alopecia areata:

- localized partial (1-2).
- localized extensive (more than 2).
- alopecia totalis (Total hair loss in the scalp).
- alopecia universalis (whole body).

Treatment:

1-Observation. ولا شي ,,
2-Intralesional Corticosteroids. (injection).
3-Skin Sensitizers: – Anthraline anti-proliferative and anti-inflammator can be use for psoriasis – Diphencyclopropenone (DPCP) immunotherapy.
4-Others: Topical steroids & Minoxidil Side effect hypotension!! – Systemic Steroids. – Cytotoxic Rx. – Phototherapy (PUVA)
No Hair Transplant (immune system)

Doctor's notes:

1-Alopecia Areata does not affect the white hair (الشيب).

2- Alopecia Areata it is not inflammatory.

3- for children give topical steroids while adults give Intralesional Corticosteroids as the first line.

3-Anagen effluvium:

Always related cytotoxic chemotherapy.
Acute and severe alopecia.
Mostly reversible but not always.
2-3 week from trigger.

4- Telogen effluvium:

Chronic alopecia.
Anemia with chronic disease.
3-4 months from trigger.
Common and None specific.

Additional cause:
drugs.
Weight loss.
Acute blood loss.
General anasthesia.
Low iron.
Others.
Reversible (but may be become chronic).

Treatment:

Remove or treat the cause. Minoxidil 2%-5% Solution.

Vitiligo: بهاق

Acquired depigmentation (loss of melanocyte) Kobner phenomena": dermatologic disease occur in the site of the trauma could be (vitiligo, psoriasis, eczema)

Causes:
1. Genetic 2. Autoimmune disease (the most prominent). 3. Neural 4. Cytotoxicity.
They have a risk of cancer.

Treatment:

No single effective Rx

Topicals: first line
.Corticosteroids
Tacrolimus (immunosuppressant)
.Immunomodulators
Light Therapy:
1. UVA or UVB The sunlight that reaches us is made up of two types of harmful rays: long wave ultraviolet A (UVA) and short wave ultraviolet B (UVB).
2. UVA + Psoralen = PUVA (Topical and Systemic).
3. UVB = Laser (MCQ).
Systemic Treatment: NOT USED.
Surgical Rx: .
Melanocyte Transplant a Surgical treatment for vitiligo are Grafting of melanocyte-rich tissue (tissue grafting). Melanocyte, cells can produce melanin
.Skin Grafts Transplant
Bleaching Agents: Depigment all skin by Benoquin (Used as final solution when the vitiligo is more than 50% of the skin).

Treatment Pearls:

Tips of fingers and toes
• Lips
• Bony prominence اماكن الاحتكاك (trauma)
• Recurrence
• Rarely 100% repigmentation for large area
• Adverse effect of Rx