STANDARDIZATION OF TRICHOTILLOMETER

DISSERTATION SUBMITTED IN FULFILLMENT OF THE REGULATIONS FOR THE AWARD OF M.D. DERMATOLOGY, VENEREOLOGY & LEPROSY

GUIDE

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CERTIFICATE

This is to certify that the thesis entitled **STANDARDIZATION OF TRICHOTILLOMETER** is a bonafide work of Dr. V. Shanmuga Sundaram done under my direct guidance and supervision in the department of Dermatology, Venereology & Leprosy, PSG Institute of Medical Sciences & Research, Coimbatore in fulfillment of the regulations of Tamilnadu Dr. MGR Medical University for the award of MD degree in Dermatology, Venereology & Leprosy.

GUIDE & HOD **PRINCIPAL**

DECLARATION

I hereby declare that this dissertation entitled **STANDARDIZATION OF TRICHOTILLOMETER** was prepared by me under the direct guidance and supervision of Professor Dr. C R Srinivas MD, PSG Institute of Medical Sciences & Research, Coimbatore.

The dissertation is submitted to the Tamilnadu Dr. MGR Medical University in fulfillment of the University regulations for the award of MD degree in Dermatology, Venereology & Leprosy. This dissertation has not been submitted for the award of any other Degree or Diploma.

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CONTENTS

Introduction

Introduction

Hair is a defining characteristic of mammals. Hair originally evolved in the mammals as a protective coat. However, it has lost its functional value in humans except in the regions of the scalp where it is thick and coarse. Elsewhere in the body, it is fine and delicate.[20]

The human skin has approximately five million follicles of which only one hundred thousand are on the scalp. Hair loss is a common problem. That may affect both male and female in all age group.

Hair disorders are difficult to treat, example-alopecia. Alopecia can be classified as scaring and non-scarring. The main cause of scaring alopecia are DLE, SLE, LP, for non-scarring alopecia are androgenetic alopecia, diffuse alopecia, alopecia areata.

Among these androgenetic alopecia are most frequently occurring problem. Hair loss can be result from due to systemic and local condition. Diffuse hair loss can further be classified by the type of hair that are shed whether they are anagen or telogen.

Diagnosis and treatment of these disorders are becoming increasingly important.

A detailed history, complete physical examination and relevant lab investigation are indicated to come to the conclusion.

Morphologically hair can be divided into four major categories; straight, spiral, helical, and wavy.

The fetus is covered by soft, fine, highly pigmented hair called languo hair. The fine hair that covers most of the body of adult are termed vellus hair. Long, coarse, pigmented hair is called terminal hair. During ones life time a particular hair follicle may produce different type of hair. A hair follicle in the scalp may initially produce a lanugo hair, later a terminal hair and finally a vellus hair.

There are different types of terminal hair. They are Anagen (growing hair) phase, catagen (involuting hair) phase, and telogen (resting hair) phase.

There are various tests available to investigate the hair loss. They are hair pull test, hair pluck test (trichogram), phototrichogram, digital phototrichogram, unit area trichogram, hair diameter assessment and trichotillometer.

Trichotillometer was first designed and constructed by C L Krumdieck in 1981, to measure the force in grams necessary to epilate individual hairs.[21]

Trichotillometer is not available commercially. We used **Instron universal Tensile Strength Tester 6021** (Instron limited, U K) as trichotillometer. It is a very sensitive machine, used in textile department to test the tensile strength of fibers.

Trichotillometer is used in various studies to measure the amount of force required to pluck the hair in protein energy malnutrition.[21]

Aims & Objectives

Aims and Objectives

To measure the diameter of hair and to determine the force required to pluck the anagen and telogen hair.

Review of Literature

Review of Literature

Hair has no vital function in humans. Yet its psychological functions are extremely important and gained considerable importance.[14]

Functions of hair includes protection of scalp from sun light and trauma, protection of eye from foreign body, sunlight, sweat, social and sexual communication, camouflage, protection against chemical and physical damage.[14]

The knowledge regarding hair follicles and embryology required to understand the hair disorders.

Embryology: One of the complexities of hair anatomy is the changing morphology that occurs as the hair progress through its life cycle. The nucleus of the hair follicle appears first in the region of the eyebrows, upper lips and chins at about 9th week of embryonic development and in other region by the fourth month. By about 22nd week, the full complement of the hair follicle is established.[14]

The hair follicles begin in utero as an epithelial bud projecting down from the fetal epidermis, this primary epithelial germ, or primary hair germ, is guided in its development by the underlying dermal papilla, an accumulation of mesenchymal cells. The hair germ descends anagen to the telogen phase. At

the end of anagen and beginning of catagen phase, the club shaped, keratinized proximal shaft is pushed upwards by a column of epithelial cells. The catagen stage takes 1to3 weeks, which is then followed by resting phase or telogen phase.[23,31,17,24] The telogen phase last for 3to4 months. Just below the column of cells is a group of undifferentiated cells (secondary hair germ cells) from where a new anagen hair develops. The anagen hair of the postnatal life begins as down growth of secondary hair germ cells which envelop and follow the dermal papilla in its descent through the dermis. As the newly forming anagen hair shaft grows up, it pushes the telogen hair out.[31] It is generally accepted that new hair follicles are not formed after birth.[23]

Types of hair

1. Lanugo hair is formed and shed during 7th and 8th month in utero. It consists of fine, soft and non-pigmented hair that has no central medulla.[48]
2. Vellus hair is the fine, unmedullated hair found on glabrous skin that is usually shorter than 2 cm and is non – pigmented.
3. Terminal hair is the course pigmented, long hair found on the scalp, eyebrow and eyelashes prior to puberty and additionally in the pubic, axillary, chest and beard areas of adults.[48] Different types of terminal hair are anagen hair, catagen hair and telogen hair.

Morphology of the normal hair roots of scalp: The structural pattern of the hair roots can be seen well in hairs that are plucked from the scalp, immersed in water and viewed microscopically by transmitted light.[22]

Hair follicle cycle: Although no new hair follicles are made postnatally, each and every hair follicle undergoes three-part cyclical growth pattern in order to produce a new hair. Normally, the hair grows to a maximum length, then hair growth ceases and the hair is shed and replaced. These phases of the hair growth cycle have been described as:

1. Anagen, a long period of growth
2. Catagen, the transitional period from growing to resting lasting 2 to 4 weeks
3. Telogen, a period of inactivity lasting 2-4 months

Although we speak of stages in the development of a hair follicle, it should be made clear that these are actually dynamic, flowing processes and the setting up of stages is purely for our understanding of the human hair follicle cycle.

Anagen: Anagen is the active phase of the hair, and extends from the termination of the inactive phase, telogen, to the beginning of the regressing phase, catagen. This phase involves the complete re-growth or regeneration of the lower, cycling portion of the follicle, i.e., the hair shaft factory. The epidermal cells surrounding the dermal papilla form the germinal matrix or

root of the hair. These cells are constantly dividing, and as new cells are formed they push the older ones upwards and eventually out. During this phase the hair grows about 1 cm every 28 days. Because there is a limit to the time a follicle stays in anagen, there is also a limit to the length of its product, the hair shaft.

The anagen phase is further subdivided into six sub-stages. These are:

- Stage I -growth of the dermal papilla and onset of mitotic activity in the germ-like overlying epithelium
- Stage II -bulb matrix cells envelop the dermal papilla and begin differentiation, evolving bulb begins descent along the fibrous streamer.
- Stage III-bulb matrix cells show differentiation into all follicular components.
- Stage IV-matrix melanocytes reactivate.
- Stage V-hair shaft emerges and dislodges telogen hair.
- Stage VI-new hair shaft emerges from skin surface.

There is generally little variation in the duration of each stage between species, except for anagen VI, which is the period during which the hair is produced at its maximum rate. The developmental processes which must occur before a hair is produced are presumably similar in all mammals.

Histological feature of a mature anagen hair follicle may be divided into two segments.[23]

1. Upper segment: Upper segment is composed of infundibulum that extends from the ostium of the follicle above to the opening of the sebaceous duct below and isthmus that extends from sebaceous duct above to the site of attachment of the muscle of hair errection below.

2. Lower segment: Lower segment is divided into 2 parts – stem and bulb. Stem extends from the base of the isthmus to the summit of the bulb, demarcated by Adamson's fringe where cornification of the inner sheath and hair begins. The bulb is the lowest part of the hair follicle. The bulk of the follicular bulb is constituted of epithelial matrix cells among which melanocytes are interspersed. The follicular bulb encloses a follicular papilla, which is formed by connective tissue sheath and also contains a single capillary and abundant mucin.[23]

The hair follicle from outside inward consists of following layers.[23]

1. **Fibrous sheath-** which surrounds the outer root sheath.

2. **Outer root sheath (ORS)** – composed of pale and clear cells, which cornifies as it ascends up to the shaft.

3. **Inner Root Sheath (IRS)**

a) Henle's layer (one cell thick and the first to cornify)

b) Huxley's layer (two cells thick and filled with trichohyalin granules)

c) Cuticle (one cell thick fully cornified) and interdigitates with the cuticle of the hair

4. Hair shaft

a] Cuticle (one cell thick, fully cornified and interdigitates with the cuticle of the inner sheath)

b] Cortex (constitutes most of the hair)

c] Medulla (present only in terminal hairs and the last part of a follicle to cornify)

The site at which the Huxley's layer loses its trichohyalin granules and begins to cornify defines both Adamson's fringe and the upper boundary of the bulbs. It is at Adamson's fringe that cornification of viable keratinocytes can first be recognized. It is also known as "Keratogenous zone".

The characteristic feature of the anagen hair is the presence of a mitotically active matrix and of a keratogenic zone.[6]

When such a follicle is forcibly plucked, the anatomic features are always altered and sometimes get greatly distorted. Usually dermal papillas and fibrous sheath, remains behind, during the plucking process, in the dermis.[31,17] Thus a normal anagen hair when plucked out contains ORS, IRS and pigmented follicle. Root is usually largest at its base, although it may have an equal diameter through out. The plucked root may show an angle of 20° or more with the shaft, which is presumably artifactual.[13] When the root sheath is

missing from a plucked hair, the proximal shaft shows a characteristic ruffling of the shaft cuticle.[30,31] Often the entire bulb of a hair is missing and the shaft appears to have been cleanly snapped off (like a cut hair) thus leaving the bulb in the scalp. These shafts can be interpreted as belonging to normal anagen hair whose roots are too firmly embedded to be extracted.[30]

Dysplastic Anagen Hair: The matrix is diminished in diameter and often deformed; the root sheath is loose or absent.[13,16] They have a narrow bulb matrix and the inner epithelial sheath often contains less pigment. If these changes progresses, they give rise to dystrophic hair.[49] It is often angulated more than 20°to the shaft.[16]

Dystrophic Anagen Hair: The changes are so severe that the root has broken off at the narrowest level and tapers to a point. Root sheaths are never present.[13,20]

Catagen: Like anagen, catagen is a highly regulated event, in its initiation, development, and termination. The purpose of catagen is to delete the old hair shaft factory and to initiate the stem cells of the bulge and the papilla to set the stage for the formation of a new follicle. Catagen is therefore regarded as the transitional phase in the hair growth cycle, and there are chemical and structural changes that take place in the hair follicle during this phase.

The hair follicles go through a highly controlled process of involution, which is a process of progressive decline or degeneration. The involution process largely

brings about a burst of programmed cell death (apoptosis) in the majority of follicular keratinocytes. Follicular melanogenesis (formation of melanin) also ceases during this stage, and some follicular melanocytes undergo apoptosis as well. Towards the end of the catagen stage, the dermal papilla condenses and moves upward, coming to rest beneath the hair-follicle bulge.

Telogen: Telogen is the resting stage of the hair cycle. During this, the coloumn of epithelial cells that constitutes a club follicle shrinks and moves steadily upwards followed closely by the papilla. The club follicle ascends to a site where the muscle of hair erection inserts in to the follicle and the column of epithelium comes to rest as a rectangle of undifferentiated cells at the base of the club follicle.[23] The keratinized club of the new telogen hair has an epithelial coating or sac derived from the ORS.[30,31] A telogen hair if plucked (or vigorously brushed or pulled) during the early part of the telogen phase, will be extracted with a portion of its epithelial sac intact. Near the end of telogen phase, gentle pulling or spontaneous shedding produces the classic “club hair” with a rough, bulbus, keratinized root.[31,30] Telogen hair comprises of about less than 25% of the total hair on the scalp.[31,17] Duration of the telogen hair on the scalp is about 3-4 months.

Alopecia:

Hair has an extraordinary and inexplicable psychological importance. Hair loss, a common problem, is often a source of distress for patients. Alopecia can be classified as either scarring or non-scarring. The most common causes of non

scarring alopecia are androgenic alopecia (AGA), alopecia areata, and telogen effluvium.

Causes of Alopecia

Non-scarring	Scarring
Androgenitic alopecia	Discoid lupus erythematosus
Telogen effluvium	Lichen planus
Alopecia areata	Severe fungal, viral or bacterial infection
Traction alopecia	Injury or burn
Tinea capitis	

Non scarring alopecia

Alopecia areata: Alopecia areata is a chronic inflammatory disorder of scalp. It is a common problem in primary care and affects the hair follicles and some times the nails. The etiology of alopecia areata remains unknown, the hair loss is thought to have an autoimmune basis and typically presents as patchy areas of hairlessness on various regions of the body. The onset of hair loss is sudden and often random and frequently recurrent.[4] Alopecia areata can be of varying severity, like,[4]

Localized alopecia areata, which accounts for most cases,

Total loss of scalp hair (Alopecia totalis)

Loss of whole body hair (alopecia universalis)

Though other autoimmune disorder at times occurs with alopecia areata, most patients are healthy. Autoimmune thyroid disease and vitiligo generally are found in <10% of patients with alopecia.[4]

Ikeda classified alopecia areata in to 4 types (i) Atopic type (ii) Autoimmune type (iii) Prehypertensive type (iv) Common type.[1]

Pathogenesis: Alopecia areata is known to cause an alteration in the normal dynamics of the hair cycle. Dense peribulbar and intrafollicular lymphocytic infiltrates are seen. The perifollicular inflammation weakness the anagen hair at the keratogenous zones of the developing hair shaft and at the same time precipitates premature entry of the follicle into catagen. The weakened hair breaks when the keratogenous zone reaches the surface of the skin, producing a rapid alopecia. Having entered telogen these fractured hairs are then eventually extruded out as exclamation mark hair, which are about 3 mm long. Light microscopy reveals a broken tip, below which the hair tapers towards a small but other wise normal club hair. As these hair are telogen hair, they are easily extracted from the scalp.[46]

Hair in anagen III are preferentially affected and are lost first. As soon as a new anagen hair reaches anagen III stage, it is damaged by the inflammatory infiltrate and sent straight back into catagen.[46] Anagen III is a stage during which hair bulb melanin is transferred to cortical keratinocytes. This is supported by the fact that-firstly-the process spares non-pigmented hair in the initial phase, secondly, the dystrophic anagen hair that are also seen, shows

variable pigmentation and thirdly regrowth after resolution of a patch of alopecia areata occurs initially with gray hair.[46]

Biopsy taken early in the course of the disease showed that the majority of follicles were in telogen or late catagen phase.[37] Anagen hair were normal or either small in size due to disproportionate reduction in the size of epithelial matrix.[37] In established lesions there was no reduction in over all follicle numbers. Anagen and telogen ration varied considerably and the anagen follicles were small and showed a conical root sheath with evidence of early cortical differentiation but no signs of cortical keratinization.[37] In case of early regrowth phase, both anagen and telogen follicles were larger than those in established lesion.[37]

The response of the anagen hair depends on the severity of the initial insult. A severe insult causes a focal weakness in the hair which would break at that point when it reaches the skin surface,[37] at same time follicles are precipitated to telogen hair.[37] This leads to formation of exclamation hair. Exclamation mark indicates the activity of the disease.[19] In less severe insult, the follicles enter into telogen hair prematurely.[37] In least severe injury the affected follicles continue in anagen hair but produce a dystrophic hair.[37] The near normal proportion of anagen follicles indicates that re-entry into the anagen stage does occur after a premature telogen.[63]

Clinical features: Alopecia areata presents as a round or oval, totally bald, smooth patch involving the scalp or any hair bearing area of the body. The most characteristic feature of alopecia areata is presence of exclamation mark which are nothing but a broken, short hair that tapers proximally.[30,26] Occasionally total loss of scalp hair (alopecia totalis) or total loss of all scalp and body hair (alopecia universals) can occur. The initial regrowth in alopecia areata is often white followed by repigmentation.[1]

The symptoms that can aid in the diagnosis of AA are[44]

Positive gentle traction test

- The hair is plucked easily by gentle traction, from the periphery of the plaques in localized forms and from several areas of the scalp in diffuse forms, in the acute phases of the disease.
- In chronic phases, this test is negative, as the hair is not plucked as easily as in the acute phases.

Presence of cadaverous hair - in these hairs fracture of shaft occurs inside the pilar follicle, producing blackened points inside the follicular ostia that resemble the comedones;

- Development of white fluff about half a centimeter in length along the alopecia area.

In chronic phases these signs are no longer present and a mild follicular hyperkeratosis can appear in the alopecia area. The surface of the alopecia areas finally becomes slightly atrophic, but it is never associated with

cicatrisation. Based on the number of lesions, extension of involvement and topography of hair loss, alopecia areata is clinically classified into classic and atypical forms.[44]

1. Classic forms

- **Single or unifocal plaque**: In this type there is a single, round or oval, smooth alopecia plaque. The skin color is normal with easily pluckable hair of normal appearance in the periphery of the plaque and typical exclamation mark hair can be present.
- **Multiple or multifocal plaques**: Typical alopecia plaques occur that affect the scalp or other pilar areas in this form.
- **Ophiasic alopecia areata**: Hair loss occurs along the line of temporo-occipital implantation, giving rise to an extensive alopecia area, in a band that reaches the inferior margins of the scalp in this type of AA.
- **Alopecia totalis**: There is total loss of terminal hair of the scalp without affecting hair on other parts of the body, it can be associated with ungula involvement.
- **Alopecia universalis**: There is total loss of body hair, involving the scalp, eyelashes, eyebrows, beard and mustaches, armpits and genital areas. In general, it occurs in association with a variety of ungula lesions.

2. Atypical forms

- **SiSaihPoo type alopecia areata**: The hair loss involves the entire scalp except for the lower margins, along the line of temporo-

occipital implantation. It is the inverse clinical image of the ophiasis form.[44]

- **Reticular alopecia areata**: Multiple alopecia plaques occur separated by narrow bands of preserved hair, conferring a reticulated pattern.
- **Diffuse alopecia areata**: The hair loss is acute and widespread and most of these cases develop into alopecia totalis or universalis forms. It is the most difficult form to diagnose, which needs to be differentiated from acute telogen effluvium, androgenetic alopecia, and also alopecia syphilitica.

Extrafollicular involvement in alopecia areata: Extrafollicular involvement in alopecia areata occurs particularly in its more severe forms. It usually comprises of ungual alterations, ocular alterations, and reports of a possible relationship with salmon patch on nape of the neck.

Hair pull test: Is positive at edge of the expanding circumscribed patch or diffusely in wide spread disease. Some dystrophic as well as telogen hair will be found.[30]

Hair pluck test: The more active the disease the higher the telogen count will be and the more likely that dystrophic anagen hair will be present.[30]

When the first bald patch appears, it is virtually impossible to establish a prognosis. In a pilot study it was shown that the trichogram of a control area of

the scalp at an early stage can be a prognostic tool in alopecia areata.[19] If trichogram of the lesional area is normal, it indicates a good prognosis. But an abnormal trichogram in the normal control region leads to a variable course or to alopecia totalis or universalis.[19] It is also advisable to focus special attention on the presence or absence of angulations of >20° and a deformation of hair shaft and/or hair root, as, such patients can go in for alopecia totalis.[63] The findings of exclamation mark at the margins of this lesion indicates activity of the disease.

Telogen effluvium: Telogen effluvium or shedding is one of the most common types of hair loss. It was first described by Kligman in 1961. Telogen hairs are resting hairs that have a nonpigmented club tip at the proximal root and can be pulled easily from the scalp. In this condition, hair follicles prematurely convert from the growth phase to the resting phase or shedding (telogen) phase. Due to this disproportionate shedding there occurs a decrease in total number of hairs. Besides scalp hairs, axillary and pubic areas are often involved.

Causes of telogen effluvium

Physiologic

Newborn	Postpartum
Early stages of androgenetic alopecia	Injury or stress
High or prolonged fever (eg. Malaria)	Severe infection
Hypothyroidism and other endocrinopathies	Severe chronic illness

Severe dieting or malnutrition	Major surgery
Severe psychologic stress (life-threatening situations)	

Drugs and toxins

Antikeratinizing agents (eg. Etretinate)	Antithyroid agents
Anticoagulants (especially heparin)	Alkylating agents
Anticonvulsants	Hormones

Acute telogen effluvium: Acute telogen effluvium is a self-limiting, non-scarring diffuse hair loss from scalp that occurs around 3 months after a triggering factor. It lasts up to 6 months and is usually followed by complete recovery.[10,49]

Various triggering factors like sever febrile illness, pregnancy, chronic systemic illness, a change of medication, a large hemorrhage, crash diet or sudden starvation, accidental trauma, surgical procedure or severe emotional stress can be responsible for acute telogen effluvium.[5]

Chronic telogen effluvium: Chronic telogen effluvium is defined as a chronic diffuse telogen hair loss that persists beyond 6 months.[49] It is much less common than acute telogen effluvium and it may follow an acute telogen effluvium. It is a diagnosis of exclusion, and more commonly no trigger is evident.[5]

Chronic telogen effluvium is much more common in male than in female.

Five different functional types of telogen effluvium are proposed based on the changes in different phase and follicle cycle. These are immediate anagen

release, delayed anagen release, short anagen cycle, immediate telogen release and delayed telogen release. [10,27]

It is important to exclude thyroid deficiency, iron deficiency, a protein deficient diet, androgenetic alopecia and also lupus, syphilis and drug induced hair loss.[49,10]

Chronic Diffuse Telogen Hair Loss: Diffuse shedding of telogen hair that persists beyond 3 months may be due to primary chronic telogen effluvium or a may be secondary to a cause.[49] The most common causes are iron deficiency anemia, hypo and hyperthyroidism, 2° syphilis, SLE and other nutritional and metabolic disorders (CRF, chronic hepatic failure) and drug-induced alopecia.

Anagen effluvium: Anagen hairs are in a growing phase, during which the matrix cells of the hair follicle undergo vigorous mitotic activity. These hairs have long, indented roots covered with intact inner and outer root sheaths, and they are fully pigmented.[57]

Anagen effluvium follows any insult to the hair follicle, which cuts down mitosis or weakens the hair shaft. This is the usual way in which drugs cause alopecia.

Anagen effluvium causes diffuse hair loss from follicles in the anagen growth phase rapidly from 1 to 4 weeks after the initial trigger. The characteristic finding in anagen effluvium is the tapered fracture of the hair shafts. The hair shaft narrows as a result of damage to the matrix. Eventually, the shaft fractures at the site of narrowing.

Anagen effluvium is caused as a result of an exposure to chemotherapeutic agents such as antimetabolites and alkylating agents. As approximately 90% of the hair follicles on the scalp are in the anagen phase, follicular damage caused by these drugs causes the anagen hairs to fall out, producing almost completes baldness.[69]

Drugs causing anagen effluvium

Cimetidine	Allopurinol
Colchicine	Haloperidol
Trimethadione	Carbamazepine
Methotrexate	Bromocriptine
Cyclophosphamide	Doxorubicin
Dactinomycin	Bleomycin
Daunorubicin	Vindesine
Mechlorethamine	Ifosfamide
Fluorouracil	Paclitaxel
Thiotepa	Etoposide
Hydroxyurea	Vincristine

Traction Alopecia: Traction alopecia involves unintentional hair loss secondary to excessive stretching of hair shafts by hair-styling practices. It often occurs in persons who wear tight braids that lead to high tension and breakage in the outermost hairs. It also occurs commonly in women who pull their hair tightly in ponytails.

Frontal and temporal areas are the most common sites of traction alopecia; hair loss is focal depending on the way the hair is being pulled. Prolonged traction alopecia can scar the new hair follicle and cause permanent hair loss.

Neonatal Occipital alopecia: There are two consecutive waves of hair loss, first, which occurs over the frontal region when patient is in uterus; and second, over the occipital region where hair enter telogen stage at about 8-12 weeks after birth and falls off.[48] This is described as occipital alopecia of newborn and the only contribution of rubbing the head on the pillow has been blamed to facilitate release of telogen hair.[48,5]

Pressure Alopecia: Continuous pressure on circumscribed area of hairy skin can cause odema, oozing, exudates, crust formation and temporary alopecia. This has been described in infants due to perinatal trauma, in patients following operation and in women after prolonged gynecological

operation. It has also been noted over the temples of nurse who wear stiff starched caps.[30,5] Hair pull and pluck test – no data's available.

Massage Alopecia: The too frequent use of combs or of hard-bristled or wire brushes can lead to a diffuse form of alopecia. Circumscribed patches of alopecia with short, dull broken hairs can also be seen in few.[5]

Marginal alopecia: It is caused by tight hair curlers or it may be the result of artificial straightening of the hair in Negroes. A "pony-tail" hair style may cause alopecia over the temples and forehead folliculitis. Scarring can also occur over the site.[5,18] Frontal and parietal traction alopecia may occur in young Sikh boys.

Trichotillomania: This is defined as a compulsive habit that induces an individual to pluck hair repeatedly.[18] It is more common in females than in males. It is seven times more frequent in children than adults. Various psychiatric studies suggest an emotional deprivation in maternal relationship.[18,5] Such patients are psychologically disturbed.

Clinical features: Patient with trichotillomania is almost adolescent girls. In younger patients, hair pulling habitis develop gradually and unconsciously but is not usually denied by the patient. Hair is plucked more frequently from fronto-parietal region. In more severe form, the patient usually consistently denies touching his or her hair. Majority of the patient suffer from some form of

emotional stress, while same may be frankly psychotic. Short terminal hairs of various lengths are scattered through out the lesions. One or few well circumscribed areas and hair loss are present, often with geometric or bizarre shape.[30] Mostly the entire scalp is involved except the margins and hence the term “tonsure alopecia”.[18]

Hair pull / pluck test: Normal in unaffected areas and not helpful in affected area.[30]

Loose Anagen Syndrome: Loose anagen syndrome is a disorder of anagen hair anchorage to the hair, characterized by ability to easily and painlessly pull out large number of anagen hair from the scalp.[47] This condition is frequently familial and follows an autosomal dominant pattern.[47] The typical patient is a child, usually 2 to 5 years of age. Girls are more commonly affected than boys. The child’s hair requires infrequent cutting and appear thin, matted, lusterless, dull or dry.[65]

Hair Pull Test: Numerous hair can be easily and painlessly pulled out form the scalp. The bulb lacks inner and outer root sheath and shows ruffling of the cuticle.[30,65,47]

Hair Pluck Test: All extracted hair can be expected to lack root sheaths.[30,65,47] A characteristic “mouse tail” hair root has been described as

seen in trichogram.[58] In adults the loose anagen syndrome may present with sudden diffuse hair loss and resembles telogen effluvium. A trichogram will differentiate between this two conditions.[47]

Androgenetic Alopecia

Androgenetic alopecia is the most common form of hair loss. It causes considerable discomfort to patient and has been proven to impair their quality of life.[2,55] It affects at least 50% of men by the age of 50 yrs and 50% of women in the age group of above 50 yrs.[11]

Etiology and Pathogenesis: Both androgenic hormones and genetic factors are necessary for the development of androgenetic alopecia. The disease does not affect men castrated before puberty. The main event in androgenetic alopecia is the conversion of terminal hair to vellus hair. This process can occur only through androgenetic stimulation of susceptible follicules.[2]

Follicles sensitive to androgens, transform testosterone into its more active metabolite dihydrotestosterone. This reaction is mediated by enzyme type-II 5 $\propto$ reductase. Level of this Isoenzyme are higher in men than in women and higher in the areas affected by androgenetic alopecia (frontal and vertex) than in the occipital region, which is always spared by the disease.[2] This 5-DHT acts on the nuclear DNA of the matrix cells so as to result in miniaturization of hair follicles or production of Vellus hair. Miniaturized follicles have a shorter

anagen phase, as a consequence higher number of hairs are in telogen phase in the scalp region in androgenetic alopecia.[2] In androgenetic alopecia miniaturization only occurs in the franto-temporal and vertex areas in men and the crown region in women.[2]

Clinical Features: Androgenetic alopecia can be graded according to Hamilton and Ludwig scales for males and females respectively. Hamilton was first to systematically describe the pattern of androgenetic alopecia; this scale was later modified by norwood.[28] It is graded from I to VIII. Alteration of the frontal hairline with bitemporal recession occurs first and is followed by balding of the vertex. Eventually a more uniform frontal recession joins the bald area and the entire fronto-parietal region bears only fine secondary vellus hair.[45] Sometime men show androgenetic alopecia with female pattern. [2,41]

The female pattern of baldness has been described by Ludwig, which is staged as I, II & III. Grade I manifesting as thinning of hair on the crown, while in grade III, there is near complete baldness of the crown. The characteristic feature in this is preservation of frontal hairline. Hamilton(male pattern) type of balding can also occur in females. Hamilton suggested that 79% of women develop Hamilton–II alopecia after puberty and 25% of women develop Hamilton V by the age of 50.[45] Ludwig pattern I-III occurred in 87% of premenopausal women white Hamilton stage II-IV occurred in 13%. Among postmenopausal women, Ludwig I-III occurred in 63%, white Hamilton II-V occurred in 37%.[45]

Another pattern of androgenetic alopecia seen in female is “Christmas Tree” pattern of hair loss in which there is increasing hair loss towards the front of the scalp.

Investigations and Diagnosis: Diagnosis of androgenetic alopecia is a clinical one and is based on recognizing the pattern of hair loss. But most often, the confusion arises in distinguishing androgenetic alopecia from telogen effluvium, as in early androgenetic alopecia in females, hair loss may be diffuse.[45]

Laboratory investigations for hormonal assessment are usually within normal range, except for in female patients with signs of virilization or hyper androgenism.[2] A scalp biopsy can differentiate androgenetic alopecia from chronic telogen effluvium and alopecia areata.

A hair pull test may demonstrate loss of an increased number of telogen hairs from the franto parietal region, but not from the occipital region.[45] Trichogram may also show an altered ratio over androgenetic dependent areas, while a normal ratio over occipital and parietal area.[45,5] Only in early androgenetic alopecia in females, the hair loss may be diffuse and a hair pull test and pluck test may reveal an increased telogen count all round the scalp.[30, 2] Trichogram reveals an increased telogen count from the androgenetic dependent sites. Dystrophic changes in the hair root can also be seen, but is less frequent.[5] Thus a mixed dystrophic–telogen trichogram is most frequent finding in

androgenetic alopecia.[30] An increase in the number of club hair in the marginal zones of alopecia is a sign of progression.[5]

Occasionally, a diffuse alopecia can coexist with androgenetic alopecia and will be suggested by a history of rapid deterioration and a positive hair pull test from all over the scalp. In such cases a drug history, thyroid function test and a serum ferritin measurements are indicated. [45]

Tinea Capitis

Tinea capitis is due to fungal (Dermatophyte) infection of hair follicle and can be inflammatory or non-inflammatory. The causative organisms varies depending on geographic location and environmental exposure to animals.[54]

It may be present with follicular pustules, nodules, and hair loss. If the diagnosis of hair loss is uncertain, tinea capitis should always be excluded before starting the treatment. As compared to trichophyton infection, infections with microsporum species are not associated with strong inflammatory reaction, if there is no bacterial superinfection. Characteristically in tinea capitis, the hair breaks off above the follicle openings and the scalp is covered by fine scaly material resembling flour. Mycotic infections are most commonly observed in children, favus being the exception, which affects adults and occurs under poor hygienic circumstances.[67]

Diagnosis of tinea capitis is made by potassium hydroxide scraping, mycologic culture, and Wood's light examination. Culture of hair detects trichophyton species in most cases.[54]

Lichen Planopilaris

Lichen planopilaris usually begins with small spotted hairless areas in the vertex region that may confluence to broad patches. It is associated with perifollicular erythema and sometimes hyperkeratotic follicular papules. At times, a violaceous discoloration of the scalp may be detected. The patches of alopecia do not show any noticeable pathology apart from missing follicular orifices. Middle-aged women are affected most commonly.[67]

Examination of the nails, mucous membranes, and the whole integument may give additional evidence of lichen planus. In a patient with only alopecia, it may be difficult to distinguish between cutaneous lupus erythematosus and lichen planopilaris. The characteristic histopathological findings of lichen planopilaris are vacuolar degeneration of the basal cell layer and lichenoid infiltrates along the epidermis and around the follicular epithelium. Burnt-out interfollicular fibrotic strands are the main characteristic in residual lesions.[67]

Diffuse hair loss

The normal hair cycle results in the replacement of every hair on the scalp every 3-5 yrs. Hair growth on the adult human scalp is asynchronous, but continuous. Around 50-150 telogen hair are shed each day normally. Diffuse

hair loss can be classified according to the type of hair lost i.e. anagen or telogen.

Iron Deficiency

Diffuse alopecia can also develop in iron deficiency, even when anemia is not yet manifested.[5] Administration of iron in such cases regress the shedding. As iron is an essential cofactor for ribonuclease reductase, which is involved in DNA synthesis, it has been proposed that iron deficiency reduces proliferation of matrix cells.[49] Telogen effluvium results from the arrest of matrix proliferation.

Protein / Calorie Malnutrition

Malnutrition affects both the hair cycle and the hair structure and sometimes even changes hair color. Short-term protein deficiency causes atrophy of the bulb and loss of the inner and outer root sheath, but there is usually no change in ratio of anagen and telogen hairs.[5]

Easy pluckability of hair has been reported in protein energy malnutrition by using trichotillometer.[21]

In marasmics, the hair becomes fine, dry, lifeless, can be easily plucked, diameter of the bulb is reduced to 1/3rd and almost all the follicles are send into telogen phase.[49,5]

Kwashiorkor results in periods of interrupted hair growth, that either sends the hair into telogen phase of if less sever, affects the caliber of the hair.[49] Flag signs is commonly seen in kwashiorkor. So the hair changes are similar to marasmas, but there are more follicles in anagen phase, although they are mostly atrophied.[5]

Essential fatty acid

Essential fatty acid deficiency has been reported both in adults and infants. Diffuses scalp and eyebrow alopecia, inform of telogen effluvium, with a lightening of the remaining hair is seen.[32,49]

Zinc Deficiency

Zinc is an essential cofactor for various metalloenzymes, including carboxypeptide, carbonic anhydrase, alkaline phosphatase and alcohol dehydrogenase.[32] Zinc deficiency, both hereditary and acquired, leads to sparse, dry little hair and the hair loss is an important clue to the diagnosis.[49] Diagnosis is made clinically, but confirmed by low zinc levels in plasma and hair.[32]

Biotin Deficiency

Protein is an essential intermediate carrier of carbon dioxide for severel co-carboxylase enzymes in the metabolism of acetylCoA and certain amino acids.[32]

Cutaneous findings include a diffuse loss in scalp and body hair and a periorifacial or generalized scaly erythematous rashes. [32,33]

Although biotin deficiency can lead to alopecia, deficiency has not been demonstrated in healthy humans eating a mixed diet. No studies have documented the effects of biotin supplementation in patients with AGA. It is a water soluble vitamin and excess vitamin is excreted in urine. No side effects are seen on supplementation.[3]

Diffuse alopecia of endocrine origin

The hair follicle appears to be responsive to a variety of hormonal factors, although the exact mechanism remains unclear.[32]

Hypothyroidism

Hypothyroidism is frequently associated with diffuse loss of scalp and body hair, with a characteristic loss of the lateral aspect of eyebrow.[5] Both in spontaneous and iatrogenic hypothyroidism there is increased shedding of telogen hair with no significant effect on anagen hair.[52] An increase in telogen count was suspected due to –

i) Abnormal retention of club hair in actively growing follicles

ii] Delay in the resumption or initiation of growth of anagen hair

iii] Premature arrest of growth in a large proportion of anagen hair

Hair loss is also expected following administration of thyroid hormones, as the anagen in resting follicles is stimulated, which then displaces the club hair

out.[52] Failure or delay in the resumption of anagen undoubtedly occurs in hypothyroidism, since there is loss of hair without replacement. Such an effect could contribute an increase in telogen hair count, as increasing number of club hair accumulate in dormant follicles before they are gradually shed.[52] The rapid rates at which telogen hairs are shed suggest that premature arrest also contributes to the changing ration in hypothyroidism.[52]

Normal telogen-anagen ration is restored after thyroid hormone replacement. Hypothyroidism inhibits cell division both in the epidermis and in the skin appendages. This inhibition of mitosis induces catagen formation and delays entry of telogen hair into angen.[49]

Hyperthyroidism

Diffuse alopecia is described in about half of the cases, but it is seldom severe and has a tendency to remission.[5] The mechanism of hair loss unknown. Both trichogram and the hair microscopy reveal an increase proportion of telogen hairs. The histopathology is that of telogen effluvium.[49]

Hypopituitarism

Hypopituitarism commencing after puberty (eg. Sheehan's Syndrome), the hair is very fine and there is loss of scalp, pubic and axillary hair. The skin becomes dry, yellowish and lack in turgor. [5, 32]

Hypoparathyroidism

The scalp hair in hypoparathyroidism is course, thin and dry. Minor injuries may cause it to fall out, and thus, irregular focal or patchy alopecia develops.[5,32]

Other Metabolic Disorders

Chronic renal failure and dialysis is associated with dry, brittle, spare scalp hair and loss of body hair, inducing pubic and axillary hair.[49] Chronic liver failure can produce diffuse alopecia.

Malignancies (Hodgkin's disease and Leukemia), pancreatic disease and upper gastrointestinal disorders, may cause diffuse hair loss.

Infections

Hair loss does not occur in 1°syphilis, except in association with at 1° chancre of the scalp.[50] Hair loss in 2° and 3° syphilis causes a characteristic "moth eaten appearance", occasionally with additional involvement of lateral aspect of eye brows as well as other body hair. It is generally reversible with adequate treatment.[32] The 2 types of 2° syphilitic alopecia are

i) Symptomatic type, which is associated with actual 2° lesions on the scalp.

ii) "Essential" syphilitic alopecia, in which hair loss present without visible syphilitic lesions.[32,30] Essential syphilitic alopecia has been divided into 3 types:–

a) The classic, patchy “moth eaten” type

b) A generalized thinning of the hair and

c) A “moth eaten” type combined with generalized thinning of the scalp hair

Of these the patch moth eaten appearance is the most frequent one. [34,30]

Telogen effluvium follows 2-3 months after a primary infection or after successful treatment as a feature of the Jarisch Herzheimer reaction.[49]

Histologically hair loss in essential syphilitic alopecia shows two patterns. The first is that of a non-inflammatory alopecia, with finding same as in telogen effluvium[29] and second pattern is that of an inflammatory, non scanning alopecia mimicking alopecia areata.[30,34,29]

Hair pull is expected to be positive for an increased telogen hair. Hair pluck test is expected to be positive for increased telogen count (100% in some cases).[30]

Collagen vascular disease

The scalp can be affected in chronic cutaneous lupus erythematosus (CCLE). The lesions start as coin-shaped, scaly, and erythematous patch. As the scales are anchored by hyperkeratotic plaques within the follicle openings, removal of the scales requires force. In approximately 30% to 50% of cases, CCLE leads to atrophic hypopigmented hairless patches.[41]

The diagnosis can be established by serological findings like leucopenia, anemia, antinuclear antibodies, and other tests for SLE. In some patients, the serology can be completely normal. Examination of the biopsy specimen shows vacuolar degeneration of the basal cell layer, compact hyperkeratosis, hyperkeratotic plugs of the hair follicles, and patchy inflammatory infiltrates around adnexum and vessels. Granular deposits of IgG and C3 at the dermo – epidermal junction and follicular epithelium are seen on direct immunofluorescence.[41]

Diffuse hair loss due to chemicals

Chemicals alopecia is rare and is mainly because of accidental inoculation.[5] Various chemicals which can cause alopecia are as follows:

Thallium Salts

Hair loss due to thallium has been well known, since the last century when it was used for night sweats in tuberculosis and later was used for epilation in cases of ring worm.[5,38] Today they are employed more as pesticides and rat poisons. Cases of poisoning due to consumption of contaminated grains and foodstuffs are known.[5] Symptoms of thallium poisoning are primarily neurological. Hair loss occurs after long term poisoning, as thallium accumulates in anagen follicles and impairs keratinization, so that the hair snaps while still in the follicle. Their structure in the dark keratogenic zone is irregular and in the shaft, near the pointed end, there may be air bubbles.[5] Other follicles prematurely enter catagen. Alopecia begins after about 1 week as

in anagen effluvium.[5] Small doses may be followed by gradual loss of club hair over a period of 3-4months. Demonstration of thallium in the urine and stool is needed for proposed diagnosis.[5]

Boric acid and other chemicals

Sodium borate can cause diffuse alopecia in various occupation. While boric acid can cause it as a component of antiseptic solution.[5] The serum boric acid level is elevated. There is report of alopecia of scalp, axilla and pubic hair from swallowing borate containing Listerine and chloraseptic mouth wash.[38,35] It is thought that boric acids gets accumulated in the hair follicles which results in toxic effect on the susceptible hair bulb.[35] Hair loss is reversible after discontinuing the offending agent. Other chemicals which can occasionally cause alopecia are arsenic, bismuth, lead, salt of gold, quinine, nicotinic acid, chloroprene diamer used in production of synthetic rubber industry, DDT and hexachlorocyclohexane, squalene, linoleic acid etc.[5]

Chronic Diffuse Alopecia of Unknown Origin

If we exclude androgenetic alopecia, endocrine factors, telogen effluvium, nutritional and metabolically determined alopecia, chemical factors, nervous and severe systemic disease; we are still left with a great many cases of chronic diffuse alopecia whose cause cannot be ascertained.[5] Mostly they occur in women between the age of 30 and 50 years.

Causes of thinning of hair

There are various conditions where hair loss can be associated with hair thinning. At times hair loss may not be noticeable enough and it's the thinning of hair which makes the scalp more visible.[30] Conditions which are associated with progressive thinning of hair are senescent alopecia, androgenetic alopecia, infections like syphilis, nutritional deficiency. [30, 34]

Barmen et all in 1964 first tried to measure the hair diameter by using a hair microscope with an ocular micrometer.[16] Anagen hairs were measured 0.7 and 1.4 cm from the root of the hair in various studies.[12] Some authorities suggest measurement of hair index i.e. the ratio of the least to the greatest diameter of the hair, and the area of the cuticle, expressed as a percentage of the area of cross section.[12]

Hair shaft dimensions shows variation between races too. Mongoloids hair is circular in cross – section and is large with a mean diameter of 120 μm. While Caucasoids hair is elliptical and small. It has a mean diameter between 50 and 150 μm. Negroid hair is flattened, curled and oval in cross – section.[48] a study on unit area trichogram for assessment of androgenetic alopecia showed that hairs greater than 40 μm in diameter per cm^2 grows more than 80 mm and thus provide meaningful hair in the cosmetic sense. [13,51,60]

Hair shaft diameter can also be assessed in a transverse section of scalp biopsy. Terminal hair measure >0.06 mm and a vellus hair >0.03mm.[30]

Evaluation of hair loss

History and physical examination are essential for the diagnosis of alopecia. Investigations may be needed to exclude systemic diseases. Numerous methods have been devised to assess the hair growth. Not only do these aid diagnosis, these are useful in measuring response to therapy.[8] The parameters measured for hair growth include:

- Rate of linear growth
- Hair shaft diameter
- Hair density
- Hair-cycle status (anagen: telogen ratio or percentage of anagen-VI hairs)
- Pigment content[8]

History and Examination: The presentation of male and female patients varies. In men with androgenic alopecia, a family history is usually present and the patient presents with characteristic thinning involving the frontal and the vertex areas of the scalp. Female patients may present with diffuse hair loss with complaints of "thinning of hair" (reduced density), increased shedding of hair or a reduction in growth rate.[8] In the history the important questions to be recorded are: [68]

- Time period of hair loss (congenital, acquired)
- Progression of hair loss, remissions if any (alopecia areata may show remission)

- Any positive family history for hair loss
- History of gastrointestinal dysfunction, dysfunction of the thyroid gland, or psychological disorders
- History of recent surgical intervention, blood loss, or chronic illness
- All medications
- In females, the menstrual and obstetric history
- Hair care routine and hair products applied.[68]

Drugs, particularly anticancer agents, anticoagulants, anticonvulsants, thyroid drugs. β-blockers, and tricyclic antidepressants, can cause diffuse thinning.[43]

Physical Examination: The most important feature is the pattern of hair loss. Hair loss can be diffuse involving whole of the scalp or patterned involving one or several portion of the scalp.[30] Hair shaft should be looked for its fragility and nodes. Scalp should be examined for any erythema, pustules, follicular papules etc. Wide spacing between follicular clusters of shaft exiting single follicular opening are the signs of scarring alopecia.[30] A magnifying glass can be used to look for exclamation mark in case of alopecia areata.

Blood investigations: Blood investigation may be done to exclude any systemic disorder. The investigations include.[56,59]

- Complete blood count to rule out systemic infection
- VDRL for syphilis
- Serum iron, serum ferritin, total iron binding capacity
- Thyroid function test

- Antinuclear antibody
- Hormone levels

Investigative Techniques: The history and physical examination will guide the physician in selecting the appropriate tests. Hair is examined four days after the last hair wash. The laboratory investigations can be divided into invasive, semi-invasive and non-invasive techniques.

Hair pull test: Normally only telogen hair can be extracted. Two or fewer hair can be extracted with a single pull. Any thing above 2 hairs indicates active shedding.[30] if the proximal ends tapers to a point, shows a fracture, or in anagen phase, all indicates disease process.[30] In case of loose anagen syndrome, protein calorie malnutrition, anagen effluvium, occult poisoning and occasionally early alopecia areata, dysplastic or dystrophic anagen hair are obtained.[48] Hair pull test is an easy method of confirming whether an abnormal hair loss is occurring or not and also its distribution.[48]

The drawbacks of this test are:

1. Washing hair before the test may give a falsely low number of telogen hairs.
2. The frequency of telogen shedding varies from day to day, thereby increasing the risk of a false negative test. Ideally hair pull test should be done daily for at least 1 week.

3. There is a seasonal variation in shedding of hair with increased hair loss in spring and autumn.
4. More telogen hair may be present in the frontal and the vertex as compared to the occipital region. Hence, this test should be done over different region of the scalp.
5. Hair pull test needs to be evaluated along with a measure of hair growth to get an idea about the net hair loss.
6. Alopecia may be due to failure of development of new anagen hair rather than increased telogen hair ratio. In these patients, although hair pull test may be normal, progressive thinning of hair may be seen.

If a more exact ratio of anagen to telogen hairs is needed, a hair pluck test or the trichogram is useful. [68,40,42]

Hair Feathering Test: It is useful in patients with complaints of decreased hair growth or easily broken hair as it can help in detecting abnormal hair fragility and hair shaft breakage. In this test, the distal 2 to 3 cm of the hairs in the involved areas are grasped between the thumb and index finger and pulled. The hair shafts held between the thumb and index finger are then checked for short broken fragments. Microscopic examination of these hairs may confirm the nature of the hair shaft defect and the type of fracture. Any of the extrinsic or intrinsic hair shaft abnormalities may result in abnormal breakage.[40]

Trichogram: It mainly involves the study of the hair cycle. In a trichogram about 50 scalp hairs are evaluated. Hair is cut 0.5cm above the surface of

scalp. They are pulled out with epilating forceps/artery forceps in groups of 5 to 10 hairs. The pulling should be rapid and in the direction of hair growth to avoid getting a large number of dystrophic hairs. The roots of the pulled hair are examined and they are classified as anagen telogen, catagen or dystrophic. The ratio of anagen to telogen hair (A/T ratio) is calculated on the basis of microscopic examination of the hair bulb. [36,68,66]

The various types of hair obtained include:

- **Anagen hair**: About 80% to 95% of the adult hair should be of anagen phase. It has thick, dark base with preserved inner and outer sheaths. The bulb is at an angle of 20° with shaft.
- **Telogen hair**: It constitutes 10% to 20% of the plucked hair in normal person. The proportion of telogen hair are highest in the fronto-vertical region, even in non-balding person. It is thin, club shaped with a smooth contour. The shaft is straight and tapers slightly into a nonpigmented bulb covered by loose outer sheath, which may even be absent. Telogen counts over 25% are considered abnormal.
- **Catagen Hair**: It is rare with only 1% to 2% of hair obtained being of this variety. The hair is similar to telogen hair except that the bulb is rough and covered by loose and thick outer and inner sheath.
- **Dystrophic/dysplastic hair**: It is also known as traumatized anagen hair. It lacks both outer and inner sheath and has an angulated bulb.

It is mainly used to monitor the response to therapy for female androgenic alopecia. Transverse section examination of the collected hair shafts can provide accurate measurement of the hair diameter. The average diameter of a healthy hair fiber is ≥80 μm. Hair with diameter <40 μm are too thin for adequate scalp coverage.

Unit Area Trichogram: In unit area trichogram, the proportion of various type of hair is counted after epilating all the hair in a small marked out area.[9] It is more sensitive than the trichogram although it is more time consuming.
In addition to the A/T ratio, it also allows measurement of the density and the shaft diameters.[36]

Timed Shed Hair Count: In this patient is instructed to collect every shed hair from the skin and tub drains, pillowcases, combs and brushes, clothing and so on, and to count the number of hair collected over a 24 hrs period.[30] This is done for 7 days and the counts are averaged. 50 to 100 hairs are considered as normal. The method is tedious and time consuming.[30] Kligmann proposed a method in which patients hair is brushed for exactly 1 min and the number of shed hair are counted. On a normal scalp, the average count is 50.[30]

Hair Growth window: This is a useful and simple test for assessing the rate of hair growth, especially in patients who claim that their hair does not grow at all.[30]

Measurement of Hair Growth Rate: The rate of hair growth varies depending upon the species, age, sex and from region to region in the same species. This can be done by using calibrated capillary tube, macrophotography or by autoradiography using a radioactive tracer.[13]

Scanning electron Microscopy (SEM): This is a specialized technique for examining hair anatomy. It is required only in few clinical situations especially for studying hair shaft anomalies such as "uncombable hair syndrome" and the "loose anagen syndrome".

Scalp scores: These include validated questionnaires which address patient perception of hair growth and satisfaction with hair appearance. Global photographs (GPs) are head shots taken a short distance away from the patient who is seated in front of a plain color cloth or other non-reflective surface on a wall. Standard global views include-vertex, midline, frontal and temporal. GPs taken before treatment and at various stages of treatment are compared and rating is done on a seven point scale (from greatly decreased -3 to greatly increased +3).[61]

As intra-individual comparisons of hair density are made over time, the target area must be clearly identifiable so that exactly the same target area may be examined at each visit. Physical landmarks or tattoos can be used for that

purpose. Increasing the size of the target area to a circle of 3 cm in diameter allows for greater ability to show small differences in hair density.[40]

Phototrichography: First introduced by Saitoh in 1970,[53] the phototrichogram (photographic trichogram) is a noninvasive technique that is simpler and more reproducible and sensitive than a trichogram. It allows the in vivo study of the hair growth cycle. It can be used to find the rate of hair growth, size of hair fibers and frequency of telogen hair follicles and to quantify shed hair. Several variants of the phototrichogram have become popular for evaluating hair in the clinic and in clinical research trials.

Saitoh et al.[53] Developed a method for measuring the ratio of anagen, catagen and telogen to the full cycle of growth in human scalp. All hairs in a 2sq cm area are trimmed 1 mm from the skin surface and a baseline photograph is taken. After a week, the same region is photographed and the hairs are trimmed again. This process is repeated until enough pictures are available for comparison. By comparing with the baseline picture, one can observe as to which hair fibers have grown (follicles in anagen) and which have not (follicles in telogen), the rate of hair growth (the length of hair in 7 days), the density of the hair (the number of hairs in the photograph) and which hair fibers are missing 5 days later (an indication of the rate of hair shedding).

Digital phototrichogram: At the beginning, phototrichogram analysis was done manually; the photographs on day 0 and day 5 would be placed side by side for comparison. The length and diameter of hair fibers would be measured

with a ruler and then the average hair growth rate calculated. This technique has been improved by image analysis and later, by the use of immersion oil (scalp immersion proxigraphy photographic method)[62] and digital contrast enhancement.[63] The scalp immersion proxigraphy photographic method involves using close-up photography, where the scalp is viewed under a glass slide with a drop of immersion oil. This increases the resolution of the image and gives more clarity to the image under similar magnification conditions. The remaining procedure is similar to that in a classical phototrichogram. The contrast-enhanced phototrichogram procedure involves coloring hair with black-colored dye immediately before the procedure. These temporarily colored hairs give a better contrast against the white scalp, making this method more sensitive for less pigmented and thin hairs.

However, all these methods are tedious and more time consuming; consequently, a phototrichogram is not commonly used in the general dermatology clinic.

Digital Epiluminescence Microscopy: Also known as TrichoScan,[25] it is based on the principle of epiluminescence microscopy of a defined area of scalp hair analyzed by digital image analysis. Special software is used to analyze the biologic parameters of hair growth, which are:

1. Hair density (number of hair/cm^2)
2. Hair diameter (in μm)
3. Hair growth rate (mm per day)

4. Anagen / telogen ratio

Global Photographs in Phototrichogram: The phototrichogram is often used along with global photographic assessment to determine the overall clinical changes in the patient over time in a standardized manner. Global photographs are head shots taken a short distance away from the patient. They need not be highly accurate as the idea is to monitor the response of the patient to the treatment given. The patient is usually photographed from the front with their hair down to show the frontal hair line and crown and also from the back to show the vertex. Other features such as patchy hair loss in the sides of the scalp, if present, are also photographed. [7,15]

Invasive Methods: Matrix cell kinetics is the most sensitive methods of evaluating hair growth. Two main indices are measured. The mitotic index is the count of the number of actively dividing cells at a given point in time, as measured on biopsy. The labeling index is a count of the number of cells entering mitosis during a given period. They are invasive, tedious, and provide a static appreciation of a dynamic process.[8]

Scalp Biopsy: Scalp biopsy is done if the diagnosis of AGA remains questionable. It helps in distinguishing AGA from telogen effluvium, alopecia areata and a concomitant primary scarring alopecia. Earlier only vertical sectioning was done, but now, horizontal sectioning is also recommended. Vertical sectioning delineates the distribution of the inflammatory infiltrate and

fibrosis. Horizontal sectioning is particularly helpful in conditions with miniaturization of follicles or in scarring alopecia with sparse follicles. It also helps in quantitative assessment of hair cycle status (identification of telogen, anagen, and catagen phase) and in measurement of hair diameter (designation as vellus or terminal). Stains used include hematoxylin and eosin, though special stains may be occasionally used. These include PAS stain for fungal infection and mucin stain (Alcian blue) for follicular mucinosis.

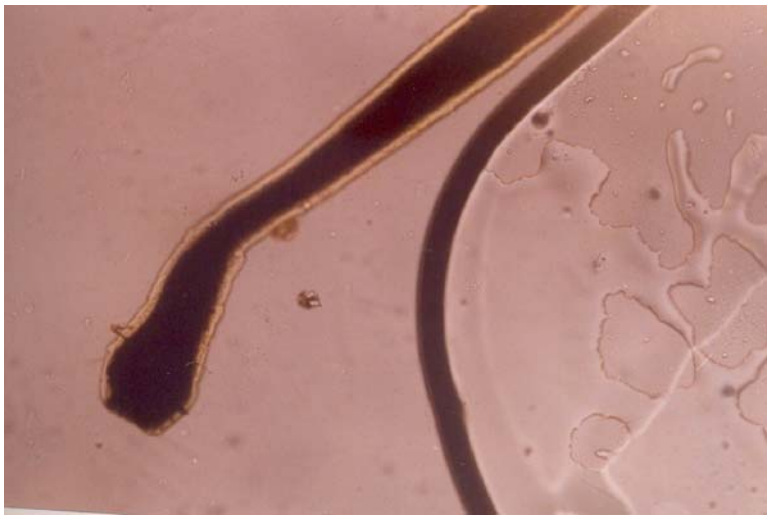
Trichotillometer: The trichotillometer is a hand held spring dynamometer designed and constructed for specifically to measure the force in grams necessary to epilate individual hairs.[21]

Instron Universal Tensile Strength Tester

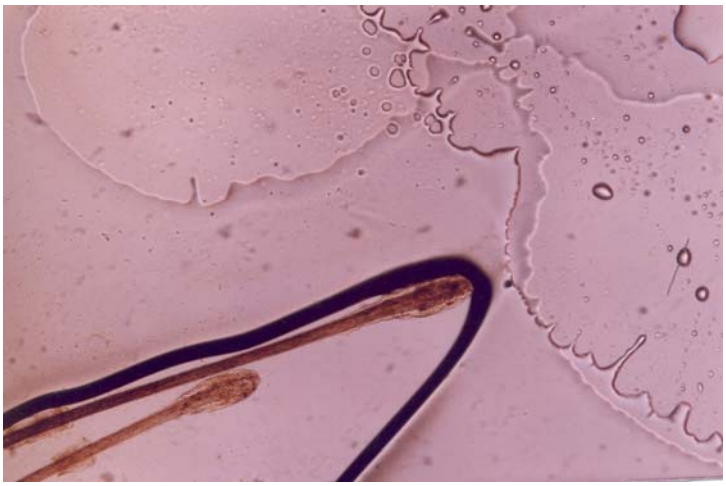
Volunteer with Instron

Image Analyzer (Video Camera Module)

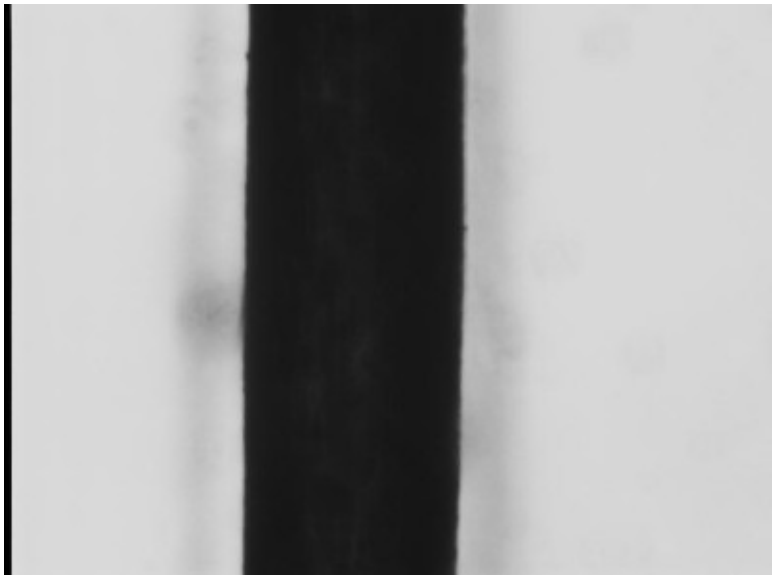
Anagen Hair



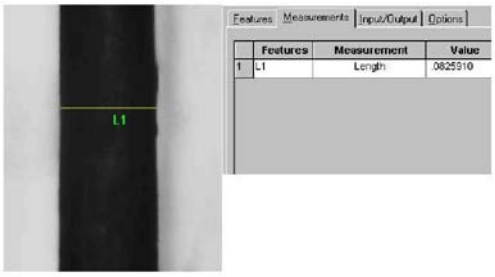
Telogen Hair



Hair Under Image Analyzer



Measuring Diameter



Materials & Methods

Materials and Methods

The Study was carried out from Oct 2007 to March 2008 in the department of Dermatology, Venereology and Leprology, PSG Hospitals, Coimbatore in collaboration with SITRA (South Indian Textile Research Association). Ten healthy volunteers (5 males and 5 females) were selected and included in the study.

The volunteers were subjected to test by using Instron Universal Tensile Strength Tester and the hair was plucked from four regions of the scalp (frontal, vertex, occipital and parietal) and the force required to pluck the hair was recorded. The plucked hair was examined under microscope to determine whether the hair was in anagen or telogen phases.

The diameter of each hair was measured using image analyzer. Data were analyzed using SPSS PC (11.5version). Mean and standard deviation (SD) were calculated. Z test was done to test the statistical significance.

Examination of hair was carried out at standard time (atleast 3 days) after the last washing of the hair.

Inclusion criteria

Volunteer who was not on any medication.

Not suffering from chronic disease.

Exclusion criteria

Those who are on medications.

Individuals with diffuse hair loss.

Trichotillometry: Trichotillometer is a hand held spring dynamometer designed and constructed to specifically measure the force in grams necessary to epilate individual hair,[21] this instrument is not commercially available, so we used Instron Universal Tensile Strength Tester. Which is very sensitive machine used in textile physics to determine the tensile characteristics of textile materials.

Then the plucked hair was examined under microscope to determine whether the hair anagen or telogen phase.

Hair diameter assessment: The plucked hair was mounted and marked at 1 cm above the hair root and examined using image analyzer with 200 X magnification (video camera module) and the hair diameter was calculated and recorded.

Results & Analysis

Results and Analysis

10 healthy volunteers, 5 males and 5 females were selected and subjected to the test. The hair was plucked from four regions of scalp (frontal, vertex, occipital and parietal) using Instron universal tensile strength tester and the plucked hair was examined under microscope to determine whether the hair was in anagen or telogen phase. Subsequently hair diameter was calculated by using image analyzer.

Total number of volunteers - 10

Male volunteer - 05

Female volunteer - 05

Total number of hair examined- 400

The force required to pluck the each hair is shown in the master chart. The **Table-1** shows the mean force required to pluck the

Anagen hair - 63.10 gms

Telogen hair - 39.86gms

P-value - 0.000

The **Table-1** also shows the mean force required to pluck the hair from individual regions. That is frontal, vertex, occipital and parietal.

In frontal area the mean force required to pluck the

Anagen hair - 69.62gms

Telogen hair - 53.22gms

P-value - 0.128

In vertex area the mean force required to pluck the

Anagen hair - 68.30gms

Telogen hair - 38.29gms

P-value - 0.000

In occipital area the mean force required to pluck the

Anagen hair - 57.72gms

Telogen hair - 26.78gms

P-value - 0.000

In parietal area the mean force required to pluck the

Anagen hair - 59.31gms

Telogen hair - 26.92gms

P-value - 0.034

The **Table-II** shows the mean force required to pluck the anagen hair and telogen hair in both sex separately.

In male the mean force required to pluck the hair

Anagen hair - 73.98gms

Telogen hair - 46.87gms

P-value - 0.037

In female the mean force required to pluck the hair

Anagen hair - 52.16gms

Telogen hair - 33.39gms

P-value - 0.000

The **Table-III** shows the mean force required to pluck the anagen hair and telogen hair in men in different regions of the scalp.

The total anagen hair - mean force required is 73.98ms

The total telogen hair - mean force required is 46.87gms

P–value - 0.037

In frontal region the force required to pluck the hair

Anagen hair - 71.73gms

Telogen hair - 55.54gms

P-value - 0.226

In vertex region the force required to pluck the hair

Anagen hair - 87.48gms

Telogen hair - 40.08gms

P-value - 0.000

In occipital region the force required to pluck the hair

Anagen hair - 69.45gms

Telogen hair - 28.79gms

P-value - 0.340

In parietal region the force required to pluck the hair

Anagen hair - 68.27gms

Telogen hair - Nil

The **Table–IV** shows the mean force required to pluck the anagen hair and telogen hair in female in different regions of the scalp.

The total anagen hair - mean force required is 52.16gms

The total telogen hair - mean force required is 33.39gms

P–value - 0.000

In frontal region the force required to pluck the hair

Anagen hair - 63.86gms

Telogen hair - 46.27gms

P-value - 0.000

In vertex region the force required to pluck the hair

Anagen hair - 49.12gms

Telogen hair - 36.50gms

P-value - 0.214

In occipital region the force required to pluck the hair

Anagen hair - 45.50gms

Telogen hair - 26.11gms

P-value - 0.002

In parietal region the force required to pluck the hair

Anagen hair - 49.79gms

Telogen hair - 26.92gms

P-value - 0.001

The **Table-V** shows the mean diameter of anagen and telogen hairs

Anagen hair diameter - 0.102mm

Telogen hair diameter - 0.101mm

P-value - 0.876

The **Table-V** also shows the mean diameter of hair from individual regions

In The frontal region mean diameter of hair

Anagen hair diameter - 0.098mm

Telogen hair diameter - 0.096mm

P-value - 0.995

In The vertex region mean diameter of hair

Anagen hair diameter - 0.103mm

Telogen hair diameter - 0.109mm

P-value - 0.378

In The occipital region mean diameter of hair

Anagen hair diameter - 0.107mm

Telogen hair diameter - 0.091mm

P-value - 0.686

In The parietal region mean diameter of hair

Anagen hair diameter - 0.100mm

Telogen hair diameter - 0.090mm

P-value - 0.367

The **Table-VI** shows the mean diameter of hair from both sex separately

In male the mean diameter of hair

Anagen hair diameter - 0.105mm

Telogen hair diameter - 0.111mm

P-value - 0.294

In female the mean diameter of hair

Anagen hair diameter - 0.099mm

Telogen hair diameter - 0.091mm

P-value - 0.643

The **Table-VII** shows the mean diameter of hair from different regions of the scalp in male volunteer

Total anagen hair diameter - 0.105mm

Total telogen hair diameter - 0.111mm

P-value - 0.294

In The frontal region mean diameter of hair

Anagen hair diameter - 0.10mm

Telogen hair diameter - 0.10mm

P-value - 0.825

In The vertex region mean diameter of hair

Anagen hair diameter - 0.10mm

Telogen hair diameter - 0.12mm

P-value - 0.133

In The occipital region mean diameter of hair

Anagen hair diameter - 0.107mm

Telogen hair diameter - 0.107mm

P-value - 0.971

In The parietal region mean diameter of hair

Anagen hair diameter - 0.11mm

Telogen hair diameter - nil

The **Table-VIII** shows the mean diameter of hair from different regions of the scalp in female volunteer

Total anagen hair diameter - 0.099mm

Total telogen hair diameter - 0.091mm

P-value - 0.643

In The frontal region mean diameter of hair

Anagen hair diameter - 0.10mm

Telogen hair diameter - 0.08mm

P-value - 0.370

In The vertex region mean diameter of hair

Anagen hair diameter - 0.10mm

Telogen hair diameter - 0.10mm

P-value - 0.844

In The occipital region mean diameter of hair

Anagen hair diameter - 0.11mm

Telogen hair diameter - 0.09mm

P-value - 0.748

In The parietal region mean diameter of hair

Anagen hair diameter - 0.09mm

Telogen hair diameter - 0.09mm

P-value - 0.820

Table-I: Force required in 4 regions

Region	Phase	Number	Mean (SD)	Z-Statistics	p-value
Total	Anagen	375	63.10 (35.48)	5.441	0.000*
	Telogen	25	39.86 (19.29)		
Frontal	Anagen	92	67.62 (25.43)	1.54	0.128
	Telogen	08	53.22 (25.83)		
Vertex	Anagen	90	68.30 (51.56)	4.41	0.000*
	Telogen	10	38.29 (12.92)		
Occipital	Anagen	96	57.72 (32.80)	7.82	0.000*
	Telogen	04	26.78 (04.22)		
Parietal	Anagen	97	59.31 (25.94)	2.15	0.034*
	Telogen	03	26.92 (05.88)		

Table-II: Force required (For Males & Females)

Sex	Phase	Number	Mean (SD)	Z-Statistics	p-value
Male	Anagen	188	73.98 (44.38)	2.095	0.037*
	Telogen	12	46.87 (23.12)		
Female	Anagen	187	52.16 (17.64)	3.766	0.000*
	Telogen	13	33.39 (12.65)		

Table–III: Force required (For males)

Region	Phase	Number	Mean (SD)	Z-Statistics	p-value
Total	Anagen	188	73.98 (44.38)	2.095	0.037*
	Telogen	12	46.87 (23.12)		
Frontal	Anagen	44	71.73 (30.36)	1.226	0.226
	Telogen	06	55.54 (30.13)		
Vertex	Anagen	45	87.48 (64.47)	4.542	0.000*
	Telogen	05	40.08 (9.10)		
Occipital	Anagen	49	69.45 (41.72)	0.965	0.340
	Telogen	01	28.79		
Parietal	Anagen	50	68.27 (32.31)	-	-
	Telogen	0**	-	-	-

Table-IV: Force required (For Females)

Region	Phase	Number	Mean (SD)	Z-Statistics	p-value
Total	Anagen	187	52.16 (17.64)	3.766	0.000*
	Telogen	13	33.39 (12.65)		
Frontal	Anagen	48	63.86 (19.47)	6.181	0.000*
	Telogen	02	46.27 (0.64)		
Vertex	Anagen	45	49.12 (10.34)	1.258	0.214
	Telogen	05	36.50 (16.88)		
Occipital	Anagen	47	45.50 (10.34)	3.202	0.002*
	Telogen	03	26.11 (4.96)		
Parietal	Anagen	47	49.79 (10.62)	3.669	0.001*
	Telogen	03	26.92 (5.88)		

Table-V: Diameter 4 regions

Region	Phase	Number	Mean (SD)	Z-Statistics	p-value
Total	Anagen	375	0.102 (0.041)	0.157	0.876
	Telogen	25	0.101 (0.016)		
Frontal	Anagen	92	0.098 (0.016)	-0.007	0.995
	Telogen	08	0.096 (0.013)		
Vertex	Anagen	90	0.103 (0.023)	-0.885	0.378
	Telogen	10	0.109 (0.017)		
Occipital	Anagen	96	0.107 (0.075)	0.405	0.686
	Telogen	04	0.091 (0.017)		
Parietal	Anagen	97	0.100 (0.019)	0.906	0.367
	Telogen	03	0.090 (0.013)		

Table-VI: Diameter (For Males & Females)

Sex	Phase	Number	Mean (SD)	Z-Statistics	p-value
Male	Anagen	188	0.105 (0.017)	-1.051	0.294
	Telogen	12	0.111 (0.013)		
Female	Anagen	187	0.099 (0.056)	0.464	0.643
	Telogen	13	0.091 (0.014)		

Table-VII: Diameter (For Males)

Region	Phase	Number	Mean (SD)	Z-Statistics	p-value
Total	Anagen	188	0.105 (0.017)	-1.051	0.294
	Telogen	12	0.111 (0.013)		
Frontal	Anagen	44	0.10 (0.01)	-2.22	0.825
	Telogen	06	0.10 (0.01)		
Vertex	Anagen	45	0.10 (0.02)	-1.527	0.133
	Telogen	05	0.12 (0.01)		
Occipital	Anagen	49	0.107 (0.028)	0.036	0.971
	Telogen	01	0.107		
Parietal	Anagen	50	0.11 (0.01)		
	Telogen	0**			

Table–VIII: Diameter (For Females)

Region	Phase	Number	Mean (SD)	Z-Statistics	p-value
Total	Anagen	187	0.099 (0.056)	0.464	0.643
	Telogen	13	0.091 (0.014)		
Frontal	Anagen	48	0.10 (0.02)	0.905	0.370
	Telogen	02	0.08 (0.01)		
Vertex	Anagen	45	0.10 (0.02)	0.198	0.844
	Telogen	05	0.10 (0.02)		
Occipital	Anagen	47	0.11 (0.11)	0.323	0.748
	Telogen	03	0.09 (0.02)		
Parietal	Anagen	47	0.09 (0.02)	0.228	0.820
	Telogen	03	0.09 (0.01)		

***P <0.05**

****.t cannot be computed because at least one of the groups is empty**

Discussion

Discussion

This study was conducted to find out whether the anagen hair is better anchored to the scalp skin than the telogen hair since the anagen phase the hair bulb lies deeper in the dermis than the telogen hair.

Our data showed the mean force required to pluck the anagen hair was 63.10 gms. Whereas the mean force required to pluck the telogen hair was 39.86 gms. Thus there is statistically significant difference in the force required to pluck the anagen hair and telogen hair (p-value=0.000).

We also subsequently analysed the data for each of the 4 regions (frontal, occipital, vertex and parietal). The mean force required to pluck the anagen hair over the frontal region was 67.62 and the mean force required to pluck the telogen hair over the frontal region was 53.22 gms. Although it was observed that more force is required to pluck the anagen hair than compared to the telogen hair over the frontal area. The difference in the force was not statistically significant (p-value=0.128).

We also found that the difference was statistically significant over vertex, occipital and parietal region (p-value= 0.000, 0.000 and 0.034 respectively). We compared the mean force required to pluck the hair among the male and female. The mean force required to pluck the anagen hair in male is 73.98 gms

and the telogen hair is 46.87 gms and it was statistically significant (p-value=0.037), similarly there was statistically significant difference in females (p-value=0.000).

Few studies are available regarding the force required to pluck the hair. However, trichotillometry was performed as a method of nutritional assessment.[21] They assessed the force required to pluck the hair in the patient with malnutrition and compared with healthy individuals.[21] The plucking force correlated significantly and positively with serum albumin, hair shaft diameter, triceps skin fold, arm muscle circumference, weight, hematocrit and beta carotene, it did not correlate with vitamin status and following surgical stress.[21]

The authors used hand held trichotillometer. The present study was performed with Instron Universal Tensile Strength tester, used in textile department to test the tensile strength of the textile fibres, yarns etc, which is very sensitive device and we used to assess the force required to pluck the hair. However, the instrument is large and expensive and used in textile industries.

We measured the diameter to find out if there was significant difference in the diameter between anagen and telogen. The hair diameter was measured using image analyzer (video camera module).

The diameter of the hair the anagen phase and telogen phase as a whole and for each of the four regions of the scalp is shown in the table-V. We did not detect significant difference in the diameter of anagen and telogen hair.

Conclusion

Conclusion

This study was conducted to determine and compare the force required to pluck the anagen and the telogen hair.

Trichogram is commonly done procedure to calculate the percentage and ratio of anagen hair and telogen hair. The hair can be differentiated by simple microscopic examination as discussed earlier.

This procedure is time taking and does not give information regarding the force required to pluck the hair.

The present study measures the force required to pluck the anagen hair and telogen hair.

Trichotillometery can be used calculate or assess the force required to pluck the hair in individuals or in patients with alopecia. Trichotillometery can be subsequently done to find whether the treatment is effective in increasing the growth of hair, prolonging anagen phase and increasing the number of anagen hair.

The equipment used was large and expensive. However a smaller and portable one is being fabricated for rapid use in outpatient department.

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Master Chart

			Frontal			Vertex			Occipital		
S. No.	**Age**	**Sex**	**Force (gms)**	**Diameter (mm)**	**Phase**	**Force (gms)**	**Diameter (mm)**	**Phase**	**Force (gms)**	**Diameter (mm)**	**Phase**
1.	52	M	33.75	0.1207	T	64.45	0.1329	A	415.6	0.1156	A
			58.83	0.1037	A	58.71	0.1026	A	30.47	0.1159	A
			73.75	0.1063	A	72.66	0.1011	A	24.06	0.1273	DA
			70.66	0.1281	A	57.42	0.1230	A	30.98	0.1094	A
			41.21	0.1031	T	58.63	0.1221	A	20.39	0.1194	A
			75.86	0.1319	A	59.73	0.1368	A	28.79	0.1061	T
			72.30	0.0986	A	57.70	0.1101	A	32.58	0.1360	A
			71.33	0.1101	A	45.27	0.1159	A	27.54	0.0804	A
			66.48	0.1044	DA	33.28	0.1228	T	31.80	0.0926	DA
			86.21	0.1101	A	39.06	0.1249	T	36.33	0.1108	A

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2.	24	M	66.55	0.1077	A	68.39	0.0739	A	35.50	0.0714	A
			40.35	0.0842	A	50.56	0.1077	A	28.07	0.0862	A
			72.92	0.0898	A	27.45	0.0624	A	38.12	0.1132	A
			35.15	0.1023	A	54.43	0.0964	A	42.07	0.1053	A
			45.98	0.0891	A	29.68	0.0869	A	49.66	0.901	A
			35.93	0.0988	A	29.37	0.1188	A	78.79	0.1085	A
			52.47	0.0816	A	33.39	0.1068	A	78.56	0.0648	A
			31.28	0.0939	A	18.34	0.0826	DA	32.61	0.1051	A
			16.31	0.0888	T	36.17	0.1036	A	59.55	0.0928	A
			18.18	0.0948	DA	35.58	0.1118	A	63.81	0.0738	A

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3.	28	F	86.49	0.1141	A	48.25	0.0972	A	41.21	0.1087	A
			89.93	0.1085	A	55.80	0.1106	A	45.83	0.0967	A
			63.15	0.0945	A	18.85	0.1345	DA	33.24	0.0844	A
			99.47	0.0887	A	53.92	0.1148	A	47.16	0.0980	A
			88.37	0.0996	A	32.49	0.0817	A	44.46	0.0866	A
			96.97	0.1158	A	40.90	0.0915	A	49.54	0.0796	A

	88.06	0.1044	A	27.53	0.1233	A	63.03	0.0809	A
	74.84	0.0909	A	21.08	0.0826	A	37.73	0.0929	A
	82.19	0.1161	A	36.56	0.1304	A	36.01	0.0801	A
	57.52	0.1095	DA	19.71	0.1085	T	32.26	0.0819	DA

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4.	41	F	86.96	0.1075	A	34.72	0.1400	A	47.74	0.0866	A
			72.26	0.0895	A	43.56	0.1036	A	40.16	0.0985	A
			77.38	0.1015	A	37.30	0.1423	A	35.04	0.0777	DA
			81.41	0.0812	A	38.83	0.1077	A	49.89	0.1034	A
			54.86	0.1070	A	35.00	0.1044	T	42.97	0.8040	A
			76.68	0.0931	A	41.33	0.0964	A	63.50	0.0890	A
			77.03	0.0842	A	45.16	0.0851	A	40.47	0.0842	A
			64.60	0.0826	A	47.08	0.0971	A	40.39	0.1012	A
			50.75	0.0867	A	35.97	0.1054	DA	24.59	0.1059	T
			58.61	0.0876	A	52.71	0.1060	A	42.62	0.0875	A

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5.	33	F	53.26	0.0708	A	51.85	0.0654	A	49.74	0.1036	A
			49.03	0.1126	A	44.93	0.0842	A	33.20	0.0747	DA
			58.65	0.0774	A	31.20	0.0753	A	31.59	0.0769	T
			61.82	0.0836	A	42.93	0.0808	A	40.08	0.0642	A
			67.27	0.0811	A	43.44	0.0999	A	46.30	0.0551	A
			47.55	0.0790	A	35.07	0.0975	A	47.34	0.0757	A
			40.94	0.1155	A	40.39	0.0647	A	48.80	0.0836	A
			46.41	0.1000	A	32.34	0.971	A	46.88	0.0800	A
			32.38	0.0701	A	42.70	0.0748	A	46.06	0.0926	A
			47.86	0.0707	A	32.30	0.0729	A	51.22	0.0766	A

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S. No.	**Age**	**Sex**	**Force (gms)**	**Diameter (mm)**	**Phase**	**Force (gms)**	**Diameter (mm)**	**Phase**	**Force (gms)**	**Diameter (mm)**	**Phase**
6.	34	F	79.69	0.1103	A	72.70	0.1069	A	52.85	0.1355	A
			82.97	0.0782	A	70.23	0.1275	A	67.62	0.1070	A
			74.61	0.9724	A	72.23	0.1387	A	52.23	0.1339	A
			77.30	0.1124	A	93.12	0.0988	A	62.19	0.1033	A
			39.83	0.0799	A	134.70	0.1317	A	65.16	0.0927	A
			87.03	0.1061	A	62.23	0.0981	A	60.31	0.1141	A

	37.42	0.1439	A	61.21	0.0959	A	51.68	0.1309	A
	101.01	0.1304	A	78.83	0.1200	A	60.31	0.1176	A
	50.94	0.0769	A	73.16	0.1477	A	52.03	0.1101	A
	53.05	0.1187	A	91.09	0.1337	A	50.82	0.1008	A

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7.	23	M	56.64	0.1060	A	88.63	0.1829	A	122.8	0.1237	A
			54.49	0.0874	A	127.50	0.1198	A	103	0.0975	A
			48.16	0.0872	A	213.90	0.0756	A	111.6	0.0874	A
			70.82	0.1098	A	183.30	0.1181	A	120.6	0.1184	A
			69.65	0.0963	A	195.30	0.1109	A	124.8	0.0863	A
			38.44	0.1355	A	209.10	0.1290	A	136.9	0.0800	A
			51.64	0.1154	A	231.40	0.0967	A	161.1	0.0998	A
			19.08	0.1028	A	219.10	0.1076	A	135.8	0.1164	A
			18.28	0.1190	A	246.10	0.1108	A	142.2	0.1016	A
			79.18	0.1012	A	77.97	0.1349	A	140.8	0.1222	A

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S. No.	**Age**	**Sex**	**Force (gms)**	**Diameter (mm)**	**Phase**	**Force (gms)**	**Diameter (mm)**	**Phase**	**Force (gms)**	**Diameter (mm)**	**Phase**
8.	24	M	80.71	0.0953	A	51.54	0.0971	A	66.20	0.1183	A
			94.31	0.0987	A	112.7	0.0837	A	77.58	0.1224	A
			80.08	0.1116	A	137.0	0.0914	A	101	0.1131	A
			83.99	0.1039	A	137.6	0.0873	A	81.33	0.1182	A
			58.18	0.1019	A	86.34	0.1270	A	68.62	0.1139	A
			95.25	0.1208	A	94.78	0.0769	A	83.52	0.0793	A
			71.01	0.1009	T	89.11	0.1069	A	92.75	0.0980	A
			74.14	0.0901	A	117.6	0.1039	A	94.78	0.1177	A
			88.53	0.0892	A	122.9	0.0909	A	136.9	0.1206	A
			76.48	0.0939	A	118.2	0.0915	A	132.3	0.0916	A

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9.	23	M	113.7	0.1060	A	27.90	0.1436	DA	36.87	0.1266	A
			122	0.0858	A	22.66	0.0915	A	28.44	0.1239	A
			144.5	0.0658	DA	20.70	0.0793	A	28.75	0.1244	A

	122	0.0995	DA	45.55	0.1064	T	33.75	0.1304	A
	146.2	0.1127	DA	30.08	0.0813	A	14.30	0.0729	A
	98.75	0.0922	A	30.04	0.1141	T	73.12	0.1224	A
	91.56	0.1120	A	15.07	0.0966	A	72.70	0.1239	A
	105	0.1169	DA	56.09	0.0804	A	35.43	0.1135	A
	73.44	0.0963	T	52.46	0.1328	T	32.54	0.1223	A
	97.5	0.1106	T	46.33	0.1028	A	30.74	0.1262	DA

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10.	32	F	46.72	0.0789	T	27.42	0.0706	T	54.77	0.0746	A
			45.82	0.888	A	64.30	0.1074	T	31.64	0.0927	DA
			36.84	0.1249	A	56.52	0.0471	DA	21.99	0.0931	A
			48.09	0.0896	DA	42.11	0.0817	A	39.57	0.0623	A
			59.3	0.0892	A	39.84	0.0681	A	32.85	0.0664	DA
			43.36	0.0840	A	28.52	0.1049	A	35.82	0.0818	A
			32.50	0.0842	A	43.48	0.0906	A	22.15	0.0763	T
			20.94	0.0675	T	59.69	0.1014	A	38.16	0.0931	A
			43.24	0.0736	A	21.76	0.0681	A	29.57	0.0526	DA
			39.41	0.0799	A	36.05	0.1009	T	46.13	0.1026	A