

FAST FACTS

**Indispensable
Guides to
Clinical
Practice**

Disorders of the Hair and Scalp

by Anne Farrell, Rodney Sinclair
and Rodney Dawber

Anatomy and physiology	9
Diffuse hair loss	22
Patchy hair loss	31
Scarring hair loss	37
Ringworm	50
Hirsuties in women	56
Scaly scalp	60
'Funny-looking' hair in children	64
Future trends	84

Indispensable

Guides to

Clinical

Practice

Disorders of the Hair and Scalp

Anne Farrell

Consultant Dermatologist,
Kent and Sussex Hospital,
Tunbridge Wells, UK

Rodney Sinclair

Consultant Dermatologist,
St Vincent Hospital,
Melbourne, Australia

Rodney Dawber

Consultant Dermatologist,
The Oxford Radcliffe Hospital,
Oxford, UK



Oxford

Fast Facts – Disorders of the Hair and Scalp
First published 2000

Text © 2000 Anne Farrell, Rodney Sinclair, Rodney Dawber
© 2000 in this edition Health Press Limited
Elizabeth House, Queen Street, Abingdon, Oxford OX14 3JR, UK

Tel: +44 (0)1235 523233
Fax: +44 (0)1235 523238

Fast Facts is a trade mark of Health Press Limited.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without the express permission of the publisher.

The rights of Anne Farrell, Rodney Sinclair and Rodney Dawber to be identified as the authors of this work have been asserted in accordance with the Copyright, Designs & Patents Act 1988 Sections 77 and 78.

The publisher and the authors have made every effort to ensure the accuracy of this book, but cannot accept responsibility for any errors or omissions.

A CIP catalogue record for this title is available from the British Library.
ISBN 1-899541-03-9

Farrell, A. (Anne)
Fast Facts – Disorders of the Hair and Scalp/
Anne Farrell, Rodney Sinclair, Rodney Dawber

Illustrated by Dee McLean, London, UK.

Printed by Fine Print (Services) Ltd, Oxford, UK.

© 2000 Health Press Ltd. www.fastfacts.com

Glossary 4

Introduction 7

Anatomy and physiology 9

Diffuse hair loss 22

Patchy hair loss 31

Scarring hair loss 37

Ringworm 50

Hirsuties in women 56

Scaly scalp 60

'Funny-looking' hair in children 64

Future trends 84

Key references 85

Index 86

Glossary

5 α -dihydrotestosterone: an androgen converted from testosterone by the action of 5 α -reductase

5 α -reductase: an enzyme catalysing the conversion of testosterone to 5 α -dihydrotestosterone

Alopecia: baldness

Alopecia areata: an autoimmune condition resulting in patchy baldness and exclamation-mark hairs

Alopecia totalis: alopecia areata resulting in complete loss of scalp hair

Anagen: the growth phase of the hair cycle

Arrector pili: involuntary muscles attached to the hair follicles – on contraction, the hairs become erect

Atrichia congenita: a condition resulting in total and permanent absence of scalp hair

Bulla: a large and watery blister

Catagen: the involution (shrinkage) phase of the hair cycle

Cuticle: the outermost part of the hair shaft, comprising between 5 and 10 overlapping cell layers

Ectothrix: a fungal infection in which spores are generated on the hair shaft

Endothrix: a fungal infection in which spores are generated within the cortex of the hair

Exclamation-mark hair: seen in alopecia areata, hair that tapers and is narrowest close to the scalp

FSH: follicle-stimulating hormone, secreted by the anterior pituitary gland

Hair cycle: the repetitive sequence of growth and rest undergone by each hair follicle

Hypertrichosis: excessive hairiness at unusual sites

Ichthyosis: dry and scaly skin

Imbricated: with overlapping edges

Keratin: a structural protein

Kerion: a painful inflammatory abscess-like mass resulting from ringworm infection

Lanugo: soft downy hair that covers a fetus, occasionally seen on neonates, particularly if premature

LE: lupus erythematosus, an autoimmune disease

LH: luteinizing hormone, secreted by the anterior pituitary gland

Marie Unna syndrome: an autosomal dominant disorder in which hair becomes coarse and twisted and is progressively lost from the scalp as the hair follicles are destroyed by scarring

Melanization: the process of imparting the dark pigment melanin

Monilethrix: a rare, inherited defect of the hair follicle characterized by beaded hairs that break easily

Morphoea: localized scleroderma

Netherton's syndrome: a hereditary condition in which an ichthyosiform erythroderma called ichthyosis linearis circumflexa is combined with fragile hair with invaginate nodes (trichorrhexis invaginata)

Pili torti: the presence of fragile hairs that are flattened and twisted through 180° at irregular intervals along the shaft

Progeria: a genetic disorder characterized by premature ageing

Scutula: a yellowish, cup-shaped crust seen in tinea capitis, which is caused by *Trichophyton schoenleinii*

SHBG: sex hormone-binding globulin, a protein that binds circulating testosterone

Spangled hair: appearance of twisted hair that reflects the light at various angles; hair with alternating light and dark bands also has this appearance

Telogen: the resting phase of the hair cycle

Terminal hair: hair that is longer and coarser than vellus hair

Tinea capitis: ringworm of the scalp in which the basic feature is invasion of hair shafts by a dermatophyte fungus

Trichotillomania: artefactual damage resulting from hair pulling

Uncombable hair: an autosomal, dominantly inherited genodermatosis, characterized by triangular hairs

Vellus hair: the short, downy hair that replaces lanugo before or shortly after birth on hair-bearing skin apart from the scalp

Weathering: the process that results in hair becoming jagged and progressively breaking off, caused by chemical and physical insults

Introduction

Warm-blooded mammals owe much of their evolutionary success to the insulation provided by their hairy covering. Paradoxically, the movement of humans from their ancestral forest home to populate the globe was linked with a reversion to relative nudity. Among mammals, hair serves many purposes. In particular, it is concerned with sexual and social communication, for example, the mane of the lion, the beard of the human male or the magnificent display of scalp hair paraded by many humans, albeit with cosmetic assistance. It also plays a role in assisting the dispersal of scents secreted by adjacent sebaceous or apocrine glands.

Although, in humans, none of the functions of hair is vital, its psychological importance is immense and diseases resulting in cosmetic alterations can be a source of significant psychological distress. The aim of this book is to deal with hair problems as they usually present in clinical practice. We hope that this book will provide healthcare workers with a practical guide to diagnose and manage these conditions effectively, and so bring comfort to the thousands of individuals with hair and scalp disorders.

CHAPTER 1

Anatomy and physiology

Embryology

The sequence of events by which the hair follicle is formed in the fetus is partly recapitulated in each adult cycle. The first sign of hair development occurs between about 9 and 12 weeks of gestational age. Crowding of cells in the basal layer of the fetal epidermis is associated with aggregation of mesenchymal cells just below it (Figure 1.1).

The epidermal cells grow downwards to form a solid column of cells called the 'hair peg', the broad tip of which becomes slightly concave. It carries before it the aggregation of mesenchymal cells, which will form the dermal papilla and dermal sheath. As the hair peg grows down, the

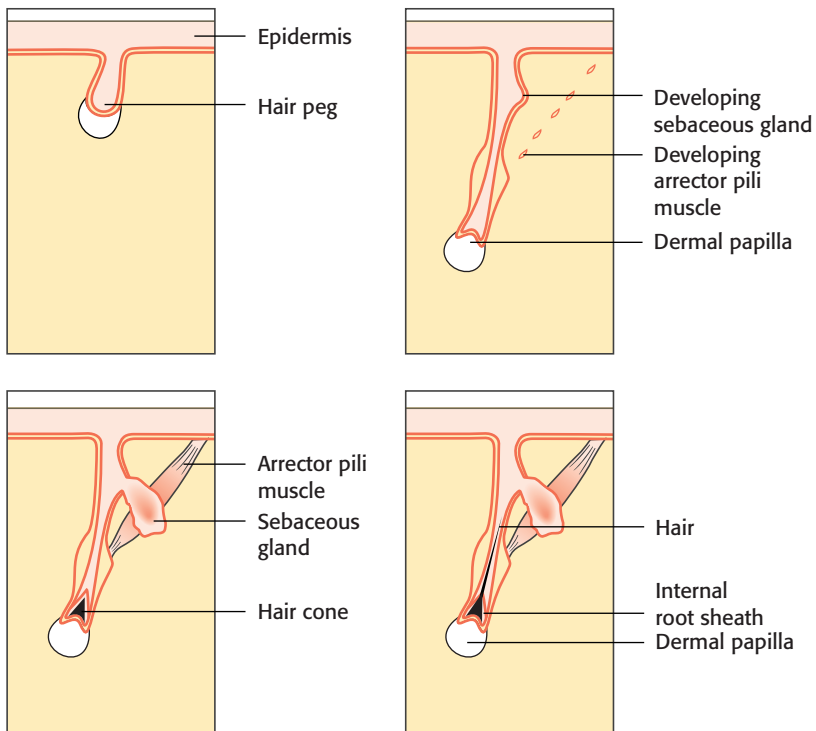


Figure 1.1 The embryological development of hair.

advancing extremity becomes bulbous ('the hair bulb' or 'bulbous hair peg') and the cavity at the tip deepens to enclose the dermal papilla. The rapidly dividing cells in the bulb surrounding the dermal papilla make up the hair matrix. Two swellings then appear – the upper swelling is the precursor of the sebaceous gland, the lower (the bulge or *Wulst*) will be the site of attachment of the arrector pili muscle. In many follicles, a third bulb appears above the sebaceous gland bud – in some areas of the skin, notably the axilla, groin, genital skin, areola and face, this will form the apocrine gland, while elsewhere it involutes.

Above the hair matrix, a cone of cells differentiates to form hair, organizing into the cuticle and cortex. A second concentric cone differentiates around it to form the inner root sheath. Three components of the inner root sheath develop – the outer of these is Henle's layer, inside this is Huxley's layer and then inside this is the cuticle of the inner root sheath. The invaginated epidermis forms the outer root sheath and surrounding this is a connective tissue layer. These developmental events depend on a complex series of interactive messages between the dermis and epidermis.

The first hair follicles start to appear in the regions of the eyebrows, upper lip and chin at about 9 weeks of embryonic development. Hair follicle development proceeds in a cephalocaudal direction and is complete by 22 weeks. This first coat of fine lanugo hair is shed *in utero* about 1 month before birth. The second coat of lanugo, covering all areas except the scalp where there is longer hair of thicker calibre, is lost almost imperceptibly during the first 3–4 months of life. These first two coats are synchronized in growth and in sequence of shedding. Thereafter, an unsynchronized (or mosaic) pattern of growth occurs.

As the body surface increases, there is a decrease in actual density of follicles. It is generally accepted that, under normal circumstances, new follicles cannot develop in adult skin. The total is estimated to be 5 million, of which about 1 million are on the head and perhaps 100 000 in the scalp. There appear to be no significant sexual or racial differences in follicle number.

Anatomy

The structure of the adult hair follicle is shown in Figure 1.2. As described above, the outer root sheath derives from the epidermis and structures inside

CHAPTER 3

Patchy hair loss

The two most common causes of patchy hair loss are alopecia areata and trichotillomania (artefactual damage as a result of hair pulling).

Alopecia areata

The characteristic presentation of alopecia areata is the appearance of a well-circumscribed, totally bald, smooth patch, often with the presence of exclamation-mark hairs at the border (Figures 3.1–3.3). In the majority of cases, the lesion is asymptomatic and may be noticed by the patient only by chance, though some patients do complain of local irritation or paraesthesia preceding the hair loss. The reason why white hairs appear to be relatively spared compared with pigmented hairs is poorly understood, but this phenomenon may be the explanation behind reports of ‘hair turning white overnight’ in historical figures such as Marie Antoinette.

It has been claimed that the scalp is the first site to be affected in 60% of cases but all sites of the body may be involved. The subsequent progress is varied; the initial patch of hair loss may regrow within a few months, though new hair is often white and finer than hair elsewhere. Alternatively new areas of hair loss may develop in some cases, resulting in total hair loss (Figure 3.4). Several studies have shown varying incidences of recovery, or progression to complete baldness, and the prognosis seems to differ pre- and



Figure 3.1

A typical patch of alopecia areata.

post-puberty. One study from Chicago reported that, of patients developing alopecia areata before puberty, 50% became totally bald and none recovered, while of those developing it after puberty only 25% became totally bald and 5.3% recovered. It also appears that alopecia areata occurring in atopic patients has a worse outcome.

Aetiology. The precise aetiology is uncertain, but a genetic predisposition appears to exist – family history increases the likelihood that a person will have the condition, and there are several reports of its occurrence in twins. Reports that alopecia areata is associated with autoimmune diseases, including thyroid disease, vitiligo and Addison's disease, have been interpreted as evidence for an autoimmune origin. Further evidence is the presence of a lymphocytic infiltrate in and around hair follicles of sufferers. Although antibodies that bind to extracts of human anagen hair follicles have been reported in the serum of patients



Figure 3.3 Moderately extensive alopecia areata.



Figure 3.2 Exclamation-mark hair.

with alopecia areata, similar antibodies are found in normal individuals, albeit at lower titres. However, alopecia areata differs from other recognized autoimmune diseases: it does not result in complete loss of function of the target organ, but rather in a temporary switching off of hair follicle activity, which can return to normal. This suggests that the target in alopecia areata may be a controlling growth factor or its receptor.