Male Pattern Baldness: Classification and Incidence*

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Abstract: The need for a widely accepted, accurate, and reproducible standard of classification for male pattern baldness has increased with the advent and increasing popularity of hair transplant surgery. This report establishes such a classification, and reports its use in determining the incidence of male pattern baldness at various ages in 1,000 white adult male subjects. The action of testosterone as an incitant in male pattern baldness is well known, but this study points out the continued effect of time, even in later years. Since most hair transplant surgery is performed on subjects with male pattern baldness, and because the success of hair transplant surgery is largely dependent on proper patient selection, a complete understanding of male pattern baldness is essential for consistently good results with hair transplantation.

Significant male pattern baldness affects well over one half of the adult male population. It is so common that some degree of hair loss is considered normal in adult males. In spite of its ubiquity, the incidence and patterns of male pattern baldness have escaped critical study and analysis by dermatologists. With the advent and increasing popularity of hair transplant surgery, the need for a widely accepted, uniform, and accurate method of classification of male pattern baldness has become a necessity. Additional and more precise data concerning the incidence as it relates to classification and age are also of increased importance.

The purpose of this report is to establish an acceptable classification of male pattern baldness and determine its incidence at various ages using the classification.

Historical Note

"Eunuchs are not subject to goot nor do they become bald." This observation was made by Hippocrates in the year 400 AC and is contained in the Hippocratic Corpus as a short medical truth or aphorism. Aristotle, himself balding, was interested in the fact that eunuchs did not get bald and were unable to grow hair on their chests. These observations were either forgotten or overlooked for the next twenty-five centuries, and medical science remained baffled by male pattern baldness until James B. Hamilton, an anatomist, in 1949 again made the observation that eunuchs did not become bald. His suggestion that androgens are a prerequisite and incitant in male pattern baldness and his later classification of the patterns and grades of male pattern baldness are a landmark in the study of male pattern baldness. Time and subsequent investigations of hair loss have confirmed the significance of androgens in male pattern baldness, and his classification remains the only acceptable one.

Many of the things we take for granted were proven by Hamilton in some brilliant and remarkably simple experiments and observations. He demonstrated conclusively that the extent and development of male pattern baldness were dependent on the interaction of three factors: androgens, genetic predisposition, and age.

In the course of studying 312 normal men and 104 eunuchs and eunuchoid men, Hamilton noted the following:

(1) Men who failed to develop sexually never devel-
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oped baldness, even if they had a strong family pedigree for baldness. This cannot be explained on the basis of fortuitous lack of genetic predisposition in view of the fact that some degree of baldness occurs in over one half of all adult men.

(2) Certain of these men rapidly developed baldness upon receiving androgen therapy. Progress of the baldness coincided with androgen therapy and stopped immediately with its discontinuance.

(3) The amount of androgen had no relationship to the development and extent of male pattern baldness. Men who were not bald did not differ significantly from men who were bald with regard to urinary ketosteroids.

(4) Androgens can induce baldness in women, as shown by some cases of arthrombomblastic and adrenal virilism. The loss of hair is of the pattern seen in men and ceases when masculinity ends.

(5) The role of genetic predisposition is emphasized by the occurrence of alopecia with androgenic therapy in only those men with familial tendency to baldness, and by the fact that no alopecia appeared on intensive androgen treatment of eunuchs whose pedigree indicated no predisposition to baldness.

(6) Aging is shown to be a factor by the fact that in normal men advancing age is accompanied by an increase in the incidence and extent of baldness. In eunuchs castrated prepubertally and given androgen

FIG 1

Standards for classification of most common types of male pattern baldness.
treatment in the second decade of life, hair was lost slowly over a period of years, resulting in hair loss similar to noncastrated men of the same age. In contrast, the eunuchs who had reached the sixth decade of life before receiving androgens lost hair within a few months after beginning the treatment. Evidently the susceptibility to alopecia increases with age but is not expressed in the absence of inducing agents such as androgens.

In summary then, genetic, endocrine, and aging factors are interdependent. No matter how strong the inherited predisposition, male pattern alopecia will not result if androgens are missing. Neither are the androgens able to induce baldness in individuals not genetically predisposed to baldness. The action of aging is demonstrated by the immediate loss of hair upon exposure to androgens in men in the sixth decade of life, whereas hair in young men exposed to androgens tends to remain much longer.

**Classification**

Hamilton's classification was used as a guide in setting up the standard pictures for this study. Before and during the initial phases of the study, changes and modifications were made in the standards to better conform to observed stages of hair loss. In addition, Hamilton's classification was simplified, and considerable detail was added to all the pictures. The standards were used in studying 1,000 individuals while determining the incidence and degree of male pattern baldness at various ages.

The accuracy of any method of classification based on inspection is open to the criticism accorded any procedure based on subjective judgment. To check accuracy, Hamilton classified 200 scalps, repeating the process three months later without reference to previous notations. The results of the two classifications were identical in 199 of 200 subjects. After examining 1,000 individuals, I am certain that if the same subjects were reexamined the percentage of identical classifications would be not nearly as high as that obtained by Hamilton. Nevertheless, classification of individuals, even if only approximate, is essential to an organized and systematic study of the subject.

Classification should provide a useful standard for screening candidates for hair transplant surgery, recording results of examination, discussing patients with colleagues, and documenting case reports.
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The standards depicted in Figures 1 and 2 categorize typical sequences in the development of male pattern baldness. There are varied patterns of male pattern baldness and no single sequence is followed uniformly by all subjects. Some individuals will fall between sequences at the time of examination, but most subjects can be classified quite closely.

Description of Standards for Classification of Male Pattern Baldness

Type I. The essential feature of type I is no recession or very minimal recession along the anterior border of the hairline in the frontotemporal region.

Type II. The anterior border of the hair in the frontotemporal region has triangular areas of recession which tend to be symmetrical. These areas of denudation extend no further posteriorly than approximately 2 cm anterior to a line drawn in a coronal plane between the external auditory meatus. Hair also is lost, or sparse, along the midfrontal border of the scalp, but the depth of the affected area is much less than in the frontotemporal region.

Type III. This represents the minimal extent of hair loss considered sufficient to represent baldness. Most type III scalps have deep frontotemporal recessions which are usually symmetrical and are either bare or very sparsely covered by hair. These recessions extend further posteriorly than a point which lies approximately 2 cm anterior to a coronal line drawn between the external auditory meatus.

Type III Vertex. In this type, the hair is lost chiefly in the vertex. There may be some frontonal recession, but it must not exceed that seen in type III. This type of baldness is most common with advancing age.

Type IV. The frontal and frontotemporal recession is more severe than in type III. Also, there is a sparseness or absence of hair on the vertex area. These areas are extensive but separated from each other by a band of moderately dense hair that extends across the top. This band joins the fully haired fringe on each side of the head. Type IV should not be confused with type III Vertex in which the loss is primarily on the vertex.

Type V. The vertex region of alopecia remains separated from the frontotemporal region of alopecia. The separation is now not as distinct, because the band of hair across the crown has become narrower and sparser. Both the vertex and frontotemporal areas of alopecia have become larger. Viewed from above, types V, VI, and VII are all characterized by areas of alopecia that are outlined by hair on the sides and back of the scalp, forming the shape of a horseshoe.

Type VI. The bridge of hair that crossed the crown in the previous type is now gone. The frontotemporal and vertex regions of alopecia have become confluent, and in addition the entire area of alopecia has increased laterally and posteriorly.

Type VII. This is the most severe form of male pattern baldness. All that remains is a narrow horseshoe-shaped band of hair which begins laterally just anterior to the ear and extends posteriorly on the sides and quite low on the occiput. This hair is usually not dense and frequently is fine. The hair is also extremely sparse on the nape of the neck and in a semicircle over both ears. It should be noted that the anterior border of this band on each side of the head has receded posteriorly to just in front of the ears.

Description of Standards for Classification of Type A Variant

Type A Variant of male pattern baldness constituted approximately 9% of all individual studies. It is distinguished by two major features and two minor features. The major features must be present to make the Type A designation. The minor features are not necessary but frequently are present.

Major Features

1. The entire anterior border of the hairline progresses posteriorly without leaving the usual island or peninsula of hair in the midfrontal region.

2. There is no simultaneous development of a bald area on the vertex. Instead, the anterior recession just keeps advancing posteriorly to the vertex.

Minor Features

1. Scattered sparse hairs frequently persist in the entire area of denudation.

2. The horseshoe-shaped fringe of hair that remains on the sides and back tends to be wider and reach higher on the head.
**Type II A.** The entire anterior border of the hairline lies high on the forehead. The usual midfrontal peninsula or island of hair is represented only by a few sparse hairs. The area of denudation extends no further than 2 cm from the midfrontal line.

**Type III A.** The area of denudation is almost to or may actually reach the midcoronal line.

**Type IV A.** The area of alopecia is now past the midcoronal line. There may be a considerable amount of thinning posterior to the actual hairline.

**Type V A.** This is the most advanced degree of alopecia described with this variant. If it becomes more extensive, it cannot be distinguished from the usual type V and VI. The area of alopecia has not reached the vertex.

**Other Types of Androgenic Alopecia**

The possible variations of androgenic alopecia are infinite. Designation of all minor types would not only be impossible but would reduce the usefulness of the classification. There are, however, several reasonably well-defined variations that deserve special mention. The common feature of all these types of alopecia is that, instead of the areas involved becoming totally bald, they seem to reach a certain point of decreased density after which further progress is barely perceptible. Generally, to qualify for these designations, the hair should be sparse enough that the scalp is plainly visible upon casual inspection.

**Diffuse, Unpatterned Alopecia.** In this type there is a general decrease in the density of hair without any definite pattern, although it is usually more marked over the top and front. This type is common in women.

**Diffuse, Patterned Alopecia.** The patterns in this type of hair loss are essentially the same as in more common male pattern baldness, but the areas involved do not become totally bald; the hair only decreases in density. This also occurs in women.

**Male Pattern Baldness With Persistent Midfrontal Forelock.** This variant can be of any degree of severity and is essentially like male pattern baldness, except that there is a persistence of the midfrontal forelock.

**Senile Alopecia.** This occurs in all scalps, male and female, with age. The role of androgens is uncertain. The decrease in density involves not just the top and sides, but the entire scalp.

**Material and Methods**

One thousand white men were divided into age groups, examined, and classified according to the previously described classification. Subjects were inpatients and outpatients at the VA Hospital, Oklahoma City, and patients from my general dermatology clinic. To avoid selectivity, all patients in the hospital and visiting the clinic with complaints directly or indirectly relating to hair loss were deleted from the study.

**Results**

Figure 3 shows that the incidence of type III, IV, V, VI, and VII, which represent a significant cosmetic hair loss, increased steadily with advancing age. Incidence of more advanced degrees of alopecia (types V, VI, and VII) remained relatively low until the seventh, eighth, and ninth decades of life. Explanation for this late increase in hair loss despite a steadily decreasing level of testosterone lies in the complex interplay of endocrine, aging, and genetic factors.

The figures in parentheses beside type III in the Table represent type III Vertex individuals. As shown, this type of baldness is more common in older subjects. There were no type III Vertex subjects in the groups aged 18 to 29, and only three (1%) in the 30- to 39-year-old group. There were 15 (40%), how-

**FIG 3**

The incidence of cosmetically significant male pattern baldness (types III, IV, V, VI, and VII) increases steadily with age and is represented by a solid line. The incidence of baldness characterized by only a remaining horseshoe fringe of hair (types V, VI, and VII) is depicted by the dotted line. It remains relatively low until the seventh decade.
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INCEPTION OF MALE PATTERN BALDNESS IN 1,000 MEN BY TYPE AND AGE

<table>
<thead>
<tr>
<th>Type</th>
<th>Age (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>18-29</td>
</tr>
<tr>
<td>Type I</td>
<td>110(60%)</td>
</tr>
<tr>
<td>Type II</td>
<td>52(26%)</td>
</tr>
<tr>
<td>Type III</td>
<td>14(6%)</td>
</tr>
<tr>
<td>Type IV</td>
<td>4(2%)</td>
</tr>
<tr>
<td>Type V</td>
<td>5(2%)</td>
</tr>
<tr>
<td>Type VI</td>
<td>7(3%)</td>
</tr>
<tr>
<td>Type VII</td>
<td>6(3%)</td>
</tr>
<tr>
<td>Total</td>
<td>165(100%)</td>
</tr>
</tbody>
</table>

*Numbers in parentheses under type III represent type III Vertex individuals.

ever, in the 40- to 49-year-old group, and incidence reached and remained about 50% of all type III alopecia in subjects over 50 years of age.

Out of the 1,000 individuals examined, 30 (5%) were type A Variant. Their distribution in the age groups was similar to the more common types.

Subjects with more uncommon variants of androgenic alopecia, i.e., diffuse unpattered alopecia and diffuse patterned alopecia, were not specified as such during the study but were placed in the patterned category they came closest to fitting.

Discussion

Previous studies on male pattern baldness, except for Hamilton's work, were not correlated with age, pattern, and degree of alopecia. Results are more meaningful when these factors are considered. Hamilton reported the incidence and type of male pattern baldness in 812 individuals. One of the factors prompting this study was Hamilton's high reported incidence of alopecia in all age groups, particularly those over 60 years of age. His results are compared to those of this study in Figure 4. The graphs are remarkably similar except Hamilton's figures range from 20% to 50% higher.

Casual observation alone suggests that an 80% incidence of male pattern baldness of types IV, V, VI, and VII in men over 60 years of age is rather high. Because of the infinite variation of male pattern baldness, a considerable amount of judgment is required in classifying individuals with any standard. Subjects seldom fit a category exactly and usually must be placed in the category they come closest to fitting. Also, changes in the standards and differences in interpretation and application of the standards could possibly account for some difference between the two studies. In addition, Hamilton's sample was smaller and had an uneven distribution of subjects in various age groups. He had 104 individuals in the 20 to 30 age group and only 79 in all three age groups above 60.

Both studies demonstrate dramatically how advancing age is accompanied by an increased incidence and degree of alopecia. Judging from the figures, it can generally be assumed that young men in early stages of baldness will progress steadily in degree of hair loss as they grow older.

In conclusion, this study shows that male pattern baldness is a progressive condition, and much more common than generally believed.

FIG 4

Comparison of types IV, V, VI, and VII of Norwood study with Hamilton study. The progression in incidence in both studies in remarkably similar except that Hamilton's ranges from 20% to 50% higher.
References


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A course entitled "The Alton D. Breshears Postgraduate Course in Head and Neck Anatomy" will be conducted at the Medical College of Virginia, Jan 19-22. For details, contact Hugo R. Seibel, PhD, Department of Anatomy, Medical College of Virginia, PO Box 906, Richmond, Va 23298.

The Annual Meeting of the Florida Society of Pathologists will be held at the Daytona Beach Hotel, Miami Beach, Fla, Jan 23-25. This meeting will be preceded by a symposium sponsored by the Department of Pathology, University of Miami School of Medicine, Jan 20-23. For information on either function, contact the Division of Continuing Medical Education, University of Miami School of Medicine, PO Box 520875 Biscayne Annex, Miami, Fl 33152.

The Retinal Vascular Service of the Wilmer Ophthalmological Institute will present the second annual symposium on Fluorescein Angiography and the Macula, Jan 26-27 at the Wilmer Institute, Baltimore, Md. For details, write Ms. Peggy Rasmussen, secretary to Stuart L. Fine, MD, the Wilmer Institute, The Johns Hopkins Hospital, 601 N Broadway, Baltimore, Md 21205.

The Society of Thoracic Surgeons will meet at the Washington Hilton, Washington, DC, Jan 26-28. For details, contact W. G. Purcell, 111 E Wacker Dr, Chicago, III 60601.

The American Society for Surgery of the Hand will meet at the Fairmont Hotel in New Orleans, Jan 28-30. For further information, contact Ms. G. M. Gorman, 3 Parker Pl, Suite 203, 2600 5 Parker Rd, Aurora, Colo 80022.

The Southern Radiological Conference will meet at the Grand Hotel, Point Clear, Ala, Jan 28-Feb 1. For more information, contact J. W. Maxwell, MD, Box 7544, Mobile, Ala 36601.

The American Orthopaedic Foot Society will hold a meeting in New Orleans, Jan 30. For information, write R. A. Mann, MD, 5495 Fernhoff Rd, Oakland, Calif 94619.

The American Academy of Orthopaedic Surgeons will meet Jan 31-Feb 4 at the Marriott-Rivergate in New Orleans. For details, contact C. V. Heck, MD, 450 N Michigan Ave, Chicago, III 60611.

The Department of Neurology, University of Miami School of Medicine will sponsor its Third Annual Course in Practical Modern Neurology, Feb 26 at the Hotel Fontainebleau, Miami Beach, Fla. For information and registration materials, contact the Division of Continuing Medical Education, University of Miami School of Medicine, PO Box 520875 Biscayne Annex, Miami, Fl 33152.

The University of Texas Health Science Center at Houston, Division of Continuing Education, in cooperation with Baylor College of Medicine and the University of Texas Systems Cancer Center, M. D. Anderson Hospital and Tumor Institute, will present a symposium entitled "Applied Clinical Immunology," Feb 5-7. For further information, write The Office of the Director, The University of Texas Health Science Center at Houston, Division of Continuing Education, PO Box 20307, Houston, Tex 77025.

The Southeastern Surgical Congress will meet at the Marriott in New Orleans, Feb 8-12. For details, contact A. H. Letton, MD, 315 Boulevard NE, Suite 500, Atlanta, Ga 30312.

The Eye Foundation of America will hold a meeting "Symposium on Glaucoma," Feb 13-14 in New Orleans. For details, write George M. Hair, MD, 1542 Tulane Ave, New Orleans, La 70112.