31 Hair Transplantation

Agata K. Brys and Daniel E. Rousso

Key Concepts

- Male pattern androgenic alopecia is a hereditary problem due to the effects of dihydrotestosterone on the terminal hairs. The use of 5α-reductase inhibitors to stop testosterone conversion to dihydrotestosterone can reduce hair loss.

- Hair transplantation is a viable option for hair restoration. Best results are seen in men who are over 25 years of age, with stabilized hair loss, coarse, wavy hair, and minimal contrast of skin and hair color. Donor-site density and scalp laxity also influence results.

- Follicular unit transplantation is the gold standard in hair transplant surgery, producing natural, lasting results. Variations of the technique include donor strip harvesting and follicular unit extraction.

■ Introduction

Human concern with hair loss dates back to prehistoric times. Primitive remedies for baldness were quite imaginative, ranging from camel dung to stump water. Ancient Egyptians used castor oil to promote hair growth. Bhringaraj and amla oils were used in Asia. Since then, significant advancements in hair restoration have occurred.

In 1882, autologous hair transplantation was first shown to be feasible in animals by J. Dieffenbach, a German doctoral student. Fifty years later, Okuda described the first hair transplants in humans.[1] He used punch grafts to treat alopecia in burn victims. However it was not until the 1950s, when Dr. Norman Orentreich described the first hair transplant for male-pattern baldness using punch grafting, that attention was given to hair replacement surgery.[2]

This treatment was based on the principle of donor dominance, which states that hairs maintain their innate ability to grow as they would have in their donor site.

Early transplants involved large numbers of hairs in each graft, sometimes referred to as plugs. Unfortunately, this could lead to an apparent "doll's head" appearance. To obtain more natural-appearing results, smaller grafts were developed, called minigrafts, which included three or more hair follicles, and micrografts, which had one to two hair follicles. J.T. Headington is credited with describing the follicular unit as an anatomical entity, and his work gave credence to using smaller grafts. In 1988, Dr. Robert Limmer performed the first pure follicular unit transplantation.[3] Since that time, the procedure has become the gold standard in hair transplant surgery, producing natural, lasting results.

■ Background: Basic Science of Procedure

All hair follicles are formed by gestational week 22. At this time, there are 5 million follicles on the body, of which 1 million are on the head, and 100,000 are on the scalp. As the size of the body increases with age, no new hair follicles are added, and the density of the existing follicles decreases.

The hair follicle is derived from both the ectoderm and the mesoderm. The hair matrix cells and the melanocytes originate from ectoderm, whereas the arrector pili, dermal papilla, follicular sheath, and blood vessels are mesodermal derivatives. The primary hair germ begins as an epithelial bud that protrudes into the dermis and is driven by the dermal papilla. Together, the papilla and the epithelial cells make up the hair bulb. The components of the hair follicle include the sebaceous gland and duct, the apocrine gland and duct, and the attachment point of the arrector pili muscle (**Fig. 31.1**).[4]
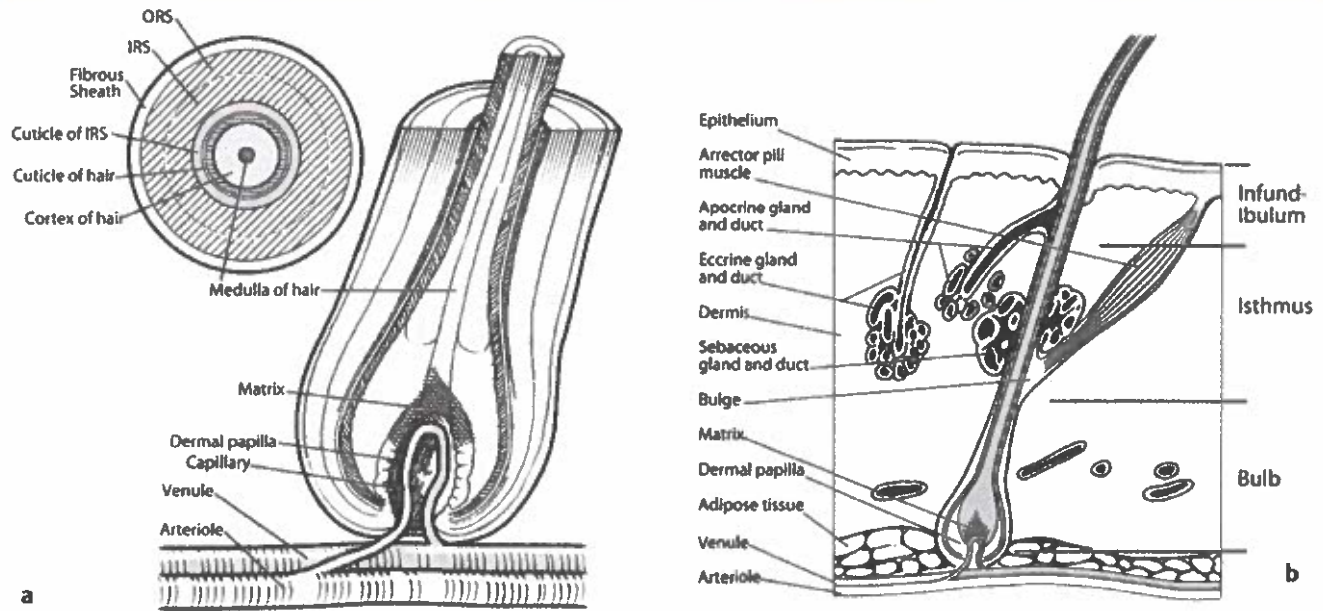
Fig. 31.1 Hair follicle anatomy. Labeled structures include epithelium, dermal papilla, sebaceous gland and duct, apocrine gland and duct, arrector pili muscle, outer root sheath (ORS), inner root sheath (IRS), medulla, matrix, cortex, and dermal papilla. **(a)** Cross-section through hair bulb and hair follicle. **(b)** The hair follicle is divided into three parts: infundibulum—from the entrance of the sebaceous duct to the follicular ostium; isthmus—from the insertion of the arrector pili muscle to the sebaceous duct; bulb— from the base of the follicle to the insertion of the arrector pili muscle.

The hair follicle can be divided into three parts. The superior portion is the infundibulum, which runs from the entrance of the sebaceous duct to the follicular ostium. Apocrine cells may empty into the infundibulum. The middle portion extends from the insertion of the arrector pili muscle to the sebaceous duct and is called the isthmus. The inferior portion is the bulb, which extends from the base of the follicle to the insertion of the arrector pili muscle. It contains melanocytes and the hair matrix with cells responsible for hair growth. The hair bulb surrounds the papilla, which influences the activity within the hair matrix. Keratinization of cells within the hair matrix forms the hair shaft. Vellous hair lacks the innermost portion of the shaft, the medulla. A follicular unit is composed of one to four terminal hairs, with or without several villous follicles, associated sebaceous glands, the arrector pili muscles, and a common vascular and neural plexus, and is surrounded by a connective tissue sheath (**Fig. 31.2**).

The follicular life cycle has three phases: anagen, catagen, and telogen (**Fig. 31.3**). The anagen phase is the phase of active growth, which lasts ~ 3 to 4 years. Most hairs in the scalp are in the anagen phase. The catagen phase is a 2 to 3 week involutional stage in which the inferior portion of the follicle ascends to the level of the attachment of the arrector pili muscle. The telogen phase represents a resting pe-

riod and lasts ~ 3 months. With aging, the number, rate of growth, and diameter of hair shafts decline. In predisposed individuals, the terminal hairs on the adult scalp can undergo involutional miniaturization (become vellus), and the percentage of telogen hairs increases.

Alopecia

There are multiple causes of hair loss, including trauma, burns, neoplasms, autoimmune disorders, chemotherapy, and dermatologic conditions. However, the most common form in both men and women is androgenic alopecia. Androgenic alopecia can present as male-pattern androgenic alopecia (MPAA), female-pattern androgenic alopecia (FPAA), or diffuse androgenic alopecia. Other causes of hair loss that can mimic pattern hair loss include acute and chronic telogen effluvium, diffuse or reverse ophiasis alopecia areata, and early cicatricial alopecia.

Telogen effluvium is characterized by massive hair loss as a result of early entry of hairs into the telogen phase,[5] often secondary to emotional or physiological stresses. A variety of causes exist, including eating disorders, fever, childbirth, chronic illness, major surgery, anemia, severe emotional disorders, crash diets, hypothyroidism, and drugs.[6] Hair loss lags the
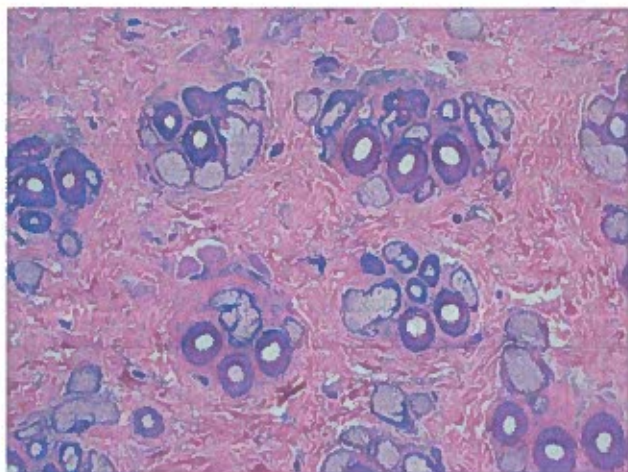
Fig. 31.2 Follicular units composed of one to four terminal hairs, with or without several villous follicles, associated sebaceous glands, the arrector pili muscles, a common vascular and neural plexus, surrounded by a connective tissue sheath. Hematoxylin-eosin, ×25. (From Rousso DE, Presti PM. Follicular unit transplantation. Facial Plast Surg 2008;24:381–388. Used with permission.)

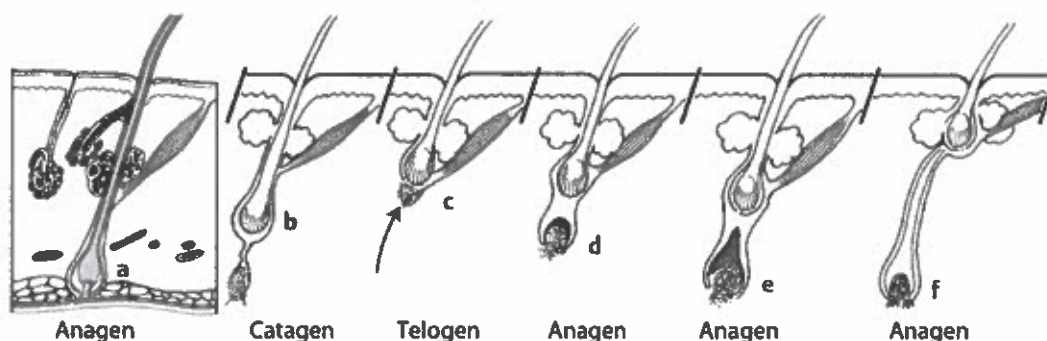Anagen Catagen Telogen Anagen Anagen Anagen

Fig. 31.3 The follicular life cycle is divided into three phases: anagen, catagen, and telogen. Anagen phase (a,d–f) consists of active hair growth. Catagen phase (b) is a stage of involution, in which the inferior portion of the follicle ascends to the level of the attachment of the arrector pili muscle. Telogen (c) represents a resting period.

inciting event by ~ 3 months. Anagen effluvium is the pathological loss of anagen hairs. Classically, it is caused by radiation therapy to the head and systemic chemotherapy, especially with alkylating agents.[7]

Alopecia areata is an immunologically driven condition, often triggered by stress, that causes patchy hair loss, which can range from diffuse thinning to extensive areas of baldness with "islands" of retained hair. On exam, the hair will have an exclamation point appearance, becoming narrower along the length of the strand closer to the base. The diffuse form mimics telogen effluvium and FPAA.

Cicatricial alopecias involve inflammation directed at the upper part of the hair follicle, which leads to destruction of the stem cell and the associated sebaceous gland. The hair follicle is then replaced with scar tissue, and permanent hair loss results. Causes include lichen planopilaris, frontal fibrosing alopecia, central centrifugal alopecia, lupus, pseudopelade (Brocq), folliculitis decalvans, tufted folliculitis, and dissecting cellulitis. There are also many infectious and inflammatory causes of alopecia. Some of the more common infectious causes include dermatophytes and syphilis. These present with a patchy "moth eaten" alopecia.[8] Hair loss in trichotillomania is typically patchy, as compulsive hair pullers tend to concentrate the pulling in selected areas. Often, short stubble hairs are present.

Pathophysiology

MPAA has been recognized as an androgen-dependent hereditary disorder since the 1940s.[9] MPAA was noted to be absent in men castrated before puberty, but developed in 12 castrated men who were treated with testosterone. Men who lack androgen receptor expression also do not develop MPAA.[10] However, it is dihydrotestosterone (DHT) that plays the dominant role in MPAA. DHT causes terminal hairs in the frontal and vertex/crown areas to become vellus, while at the same time causing vellus hairs to become terminal in the beard, mustache, chest, and upper pubic region. The enzyme responsible for converting

free testosterone into DHT is 5α-reductase. Of the two isoforms of 5α-reductase, type II is expressed in androgen-dependent tissues, such as the prostate and hair follicle, and plays a role in MPAA. Men with deficiency of 5α-reductase do not develop MPAA.[11] Men with androgenic alopecia typically have higher levels of 5α-reductase, lower levels of total testosterone, higher levels of unbound/free testosterone, and higher levels of total free androgens, including DHT.[12] Current forms of medical treatment for MPAA aim to inhibit 5α-reductase activity. Men should be warned that anabolic steroids or supplemental androgens may increase hair loss.

MPAA is caused by the genetic sensitivity of hair follicles to DHT, causing the hairs to miniaturize and have a shorter lifespan, therefore preventing the individual from producing hair normally. Approximately 50% of all men are affected by MPAA, and there is a strong hereditary predisposition. The inheritance pattern is most likely polygenic.[13] Sons of men with MPAA have an increased frequency of MPAA, whereas sons of nonbalding men have a decreased risk of MPAA.[14,15] There is a maternal effect on MPAA, but it is less defined.

Clinical hair loss in classic male-pattern baldness follows a well-defined pattern, consisting of hair recession in the frontotemporal region and loss of hair at the vertex. These areas of loss gradually enlarge and coalesce until the entire front, top, and vertex of the scalp are bald. The stages of hair loss were originally described by Hamilton[16] and then modified by Norwood (**Fig. 31.4**).[17] The Norwood classification is as follows: Class I represents an adolescent or juvenile hairline resting on the upper brow crease. Class II hairline is 1.5 cm above the upper brow crease with some temporal recession. This is a mature hair line. Class III is the earliest stage of male hair loss and is characterized by a deepening temporal recession. Class III vertex represents early hair loss in the vertex. Class IV is continued hair loss in the frontal and vertex regions, but with a solid band of hair across the top separating the front and vertex. Class V is continued balding in the frontal and vertex regions with thinning of the bridge of hair separating the two areas. Class VI is the complete loss of the connecting bridge of hair, with a resulting single large bald area on the front and top of the scalp. The hair on the sides of the scalp remains relatively high. Class VII patients have extensive hair loss with only a rim of hair around the sides and rear. The Norwood Class A patterns are characterized by a front-to-back progression of hair loss, without the connecting bridge across the top of the scalp and a more limited hair loss on the vertex, even when advanced.

Usually, women do not suffer classic male-pattern baldness, but 30 to 40% of women are affected by hair loss. Women more commonly have diffuse central thinning[18] or frontal accentuation,[19] otherwise

known as the "Christmas tree" pattern. Although the entire scalp is at risk of alopecia, recession at the temples is less likely than in men, and women tend to maintain the position of their hairlines. Female-pattern alopecia was classified by Ludwig in 1977 into three stages: mild, moderate, and extensive (**Fig. 31.5**).[18] Stage I is mild, with thinning in the crown but preservation of the frontal hairline. Stage II patients have significant widening of the midline part and noticeably decreased volume. Type III patients have diffuse thinning with a see-through look on the top of the scalp.

The role of androgens in FPAA is less straightforward. As already described, FPAA differs in distribution from MPAA. This is due to the fact that aromatase breaks down testosterone to estrogen and prevents the effects of DHT. The affected women typically do not have signs of hyperandrogenism and do not respond to treatment with 5α-reductase inhibitors.[20]
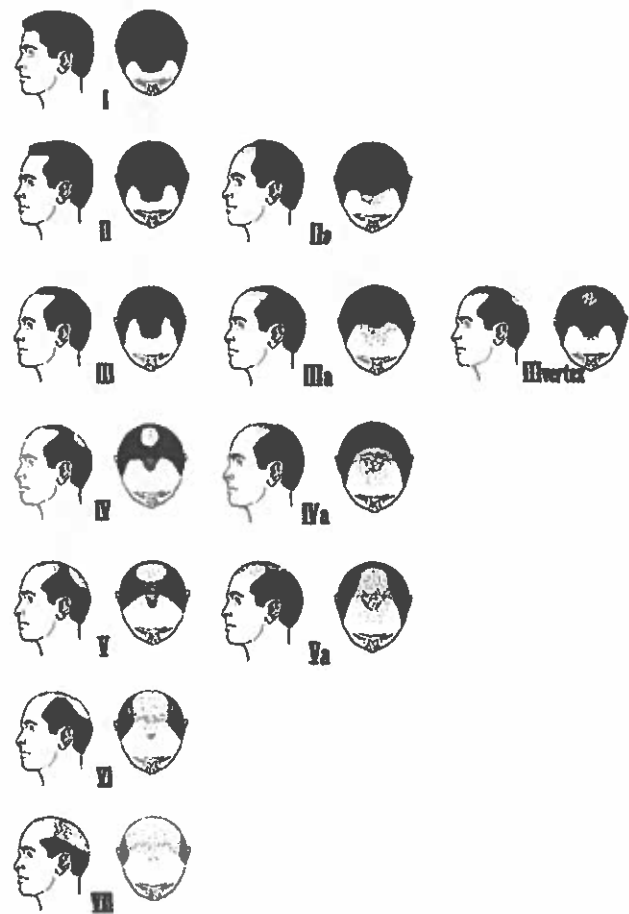

Fig. 31.4 Norwood classification of male-pattern baldness. (From Norwood OT. Male-pattern baldness: classification and incidence. South Med J 1975;68(11):1359–1365. Used with permission.)
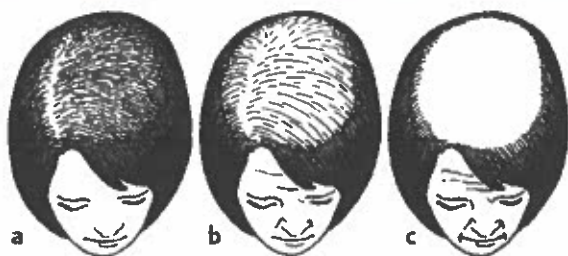
Fig. 31.5 Ludwig classification of female pattern baldness. (a) Stage I, perceptible hair loss with thinning in the crown, but preservation of the frontal hairline. (b) Stage II, significant widening of the midline part and noticeably decreased volume. (c) Stage III, diffuse thinning. (From Ludwig E. Classification of the types of androgenetic alopecia (common baldness) occurring in the female sex. Br J Dermatol 1977;97:247–254. Used with permission.)

There is a smaller subset of women who present with a more typical male pattern of hair loss. These women may be suffering from hyperandrogenism and need to be evaluated for other signs or symptoms of hyperandrogenism, including hirsutism, moderate to severe or treatment-refractory acne, irregular menses, infertility, and galactorrhea. Women with male-pattern hair loss in the presence of signs of hyperandrogenism may respond to treatment with finasteride or cyproterone acetate.[21,22] In addition, a family history should be obtained from the patient, although it may not be as clear as with males.

Medical Treatment

Before therapy is offered, a correct diagnosis must be made. Infections and autoimmune conditions must be identified and treated. Laboratory evaluation in men with androgenic alopecia is minimal. One may consider thyroid testing if the hair loss is diffuse and not following the typical MPAA pattern. Men on a strict vegetarian diet may be deficient in iron, and an iron evaluation may be warranted.

Women should have their thyroid-stimulating hormone (TSH) and serum ferritin levels tested. Deficiencies in either can cause telogen effluvium.[23,24] Iron deficiency can interfere with medical treatment of FPAA.[25] Women with clinical signs of hyperandrogenemia should undergo serological studies. These tests should include free and total testosterone and dehydroepiandrosterone sulfate. If galactorrhea is present, prolactin levels should be checked.

A scalp biopsy is indicated in men with female-pattern hair loss, diffuse hair loss, or scalp changes consistent with cicatricial alopecia. Women benefit from a scalp biopsy to exclude chronic telogen effluvium, diffuse alopecia areata, or cicatricial alopecia.

The topical and oral treatments for androgenic alopecia are numerous. Of these, topical minoxidil (Rogaine, McNeil-PPC, Lititz, PA) and oral androgen modifiers have been found to be effective. Minoxidil, a piperidinopyridine derivative initially used as a vasodilator to treat hypertension, was noted to increase hair growth in 70% of the patients taking the medication for hypertension. The action of minoxidil is not fully understood, but it might increase blood flow to the scalp or stimulate hair follicle growth through growth factor modification. Topical minoxidil applied every night in a 2 or 5% solution has been shown to be effective over 4 to 6 months.[26] Discontinuation of treatment leads to loss of gained hair over 3 to 4 months.[27] Initially, patients may observe an increase in hair loss because hairs are induced into anagen and telogen hairs are shed. Minoxidil is safe for use by both men and women, although the 5% solution may cause unwanted facial hypertrichosis in females. Therefore, it is recommended for women to use the 2% solution. Minoxidil used within days of hair transplantation can delay the typical shedding seen after hair transplantation and may shorten the period needed for hair grafts to regrow. There are minimal side effects associated with minoxidil use, and they are typically dermatologic in nature. The 5% solution is more likely than the 2% solution to cause scalp irritation, dryness, scaling, itching, or redness.

Drugs that interfere with type II 5α-reductase, such as finasteride, have been approved by the U.S. Food and Drug Administration (FDA) to treat hair loss in men. These medications, such as Proscar (Merck, Whitehouse Station, NJ), were originally used to treat prostate hypertrophy and were incidentally noted to improve hair loss. They work by reducing production of DHT, thus limiting the action of DHT on scalp hair follicles, but having no intrinsic steroid activity. Recommended dosage of finasteride (Propecia, Merck) is 1 mg daily.[28] Increases in hair counts are seen within the first year of use; following this, there is a plateau of hair growth, but there is also a continued decrease in hair loss. The decreased DHT levels also reduce prostate-specific antigen (PSA), and it is recommended that any PSA value should be doubled for men taking finasteride.[29] In men, side effects are limited to decreased libido, erectile dysfunction, and decreased ejaculate volume. These were noted in 1.8% of men ages 18 to 41 versus 1.1% in those on placebo and were reversible with cessation of medication.[30]

Because DHT is necessary for embryonic sexual differentiation and virilization of the male embryo, finasteride is contraindicated in women of childbearing age. No risk is seen to the fetus or the mother from semen of men taking finasteride.[31] A 1 year, double-blind, placebo-controlled, randomized, multicenter trial of finasteride in postmenopausal women did not show increase in hair growth or decrease

in the progression of hair thinning.[20] Better results are seen in women with a male pattern of hair loss and evidence of hyperandrogenism.[32] All women of childbearing age using 5α-reductase inhibitors or antiandrogens need to use effective birth control. Oral contraceptives have the additional benefit of lowering serum androgens.

Spironolactone is an aldosterone antagonist employed in clinical practice as a potassium-sparing diuretic used to treat high blood pressure. Spironolactone is used off-label to treat alopecia and hirsutism in females. It decreases production and blocks the effect of androgens at the cellular level. Birth defects are possible with this medication, and a form of birth control is recommended when taking this medication. Small, uncontrolled studies with spironolactone show efficacy of spironolactone in women who have hyperandrogenism.[33] The minimum effective dose to treat hair loss is 100 mg daily. Doses above 100 mg a day are associated with uterine bleeding and a possible risk of developing breast cancer.[34]

In Europe, cyproterone acetate is approved for women with alopecia, high ferritin levels, or clinical evidence of hyperandrogenism. It is an androgen receptor-binding molecule that competes with DHT. Combining 100 mg cyproterone acetate with 50 µg ethinyl estradiol on days 5 to 25 of the menstrual cycle helps decrease side effects. Combination products of oral contraceptives and a small amount of cyproterone acetate exist but are not available in the United States.

Grooming

No patient undergoing hair restoration surgery should ignore the benefits of hair styling. A consultation with a hair stylist can maximize the effect of the transplantations, and most patients benefit from the use of a hairstyling blow dryer. Those with thin, straight hair can benefit from a permanent body treatment. The use of topical scalp concealers such as COUVRé or Toppik hair-building fibers (Spencer Forrest, Los Angeles, CA) can give additional benefit by deflecting light away from thin areas.

Surgical Treatment

The basic principle in hair restoration surgery is to redistribute the existing hairs to give the scalp the appearance of increased hair density. This can be achieved through transplantation, flaps, or scalp reductions. Hair transplantation has become the gold standard of hair restoration surgery. Initial methods involved the use of punches to remove large areas of donor hair and to place them in the areas of alopecia. Currently, smaller transplants are used to provide a more natural result.

◼ Patient Selection

Although almost every patient is a candidate for some form of hair replacement procedure, there are certain factors that make some patients better surgical candidates.[35] These factors include age, pattern of alopecia, heredity, hair density in donor area/fringe, hair and skin color, hair texture, and scalp laxity. Very few absolute contraindications to surgery exist, especially if one does not count lidocaine allergy, active autoimmune, inflammatory or infectious disease, and inadequate health status to undergo this type of surgical procedure.

Age

No minimum age limit exists for hair replacement, but experience has demonstrated that patients younger than their early twenties may be less content with the results gained. Waiting for the patient to mature tends to dissipate unrealistic expectations of hair density and allows the patient to accept a more mature hairline. It also allows the surgeon to better evaluate the progressive nature of the patient's alopecia and to determine the final pattern of hair loss. Younger patients with premature alopecia also tend to have progressive hair loss and less impressive gains from restoration surgery.

Pattern of Alopecia

Androgenic alopecia is progressive; therefore the pattern of alopecia at the time of presentation is only a snapshot in time. The rate of alopecia progression is unknown. The Hamilton-Norwood and Ludwig classifications are helpful for communication purposes but do not predict the pattern of hair loss for the patient. It is important to determine the rate of progressive hair loss and to evaluate the patient's family history to better predict the patient's possible future hair loss.

Heredity

Because androgenic alopecia has both maternal and paternal hereditary influences, information about the pattern of hair loss on both the maternal and the paternal sides of the family tree can help in estimating the patient's future hair loss. For example, a patient with hair loss presenting in the early twenties and a family history of extensive alopecia on both the maternal and paternal sides will likely continue to lose hair and may be limited in reconstructive options.

Hair Density in Donor Area/Fringe

Occipital hair density will determine the number of grafts that can be harvested per each session. Patients with advanced alopecia but with excellent density in the donor area can be candidates for hair transplantation with good results. Patients with poor density may only be candidates for restoration of an isolated frontal forelock. One cannot expect to obtain or even attempt to achieve full coverage in patients with type VII alopecia.

Hair and Skin Characteristics

The apparent density of hair following hair transplantation is affected by the characteristics of the hair and scalp. Coarse, wavy hair appears denser than limp, fine hair (**Fig. 31.6**). A strong contrast between the hair and skin color decreases the apparent density of hair. Light hair against a background of light skin appears denser than does dark hair against light skin. A light-skinned individual with dark hair will have a harder time camouflaging the hair transplants and achieving a natural result unless a greater number of grafts is used. On the other hand, a light-skinned patient with curly gray or blond hair will have an apparently better result.

Scalp Laxity

Evaluation of occipital scalp laxity will determine whether graft harvest is reasonable, especially in patients who have undergone prior transplantation.

Patients with significant laxity may be candidates for alopecia reduction. Those with very limited laxity could benefit from scalp expansion or extension prior to undergoing any surgery. In our observation, intraoperative use of hyaluronidase can decrease closure tension in patients with poor scalp laxity (pers. comm., Carlos Puig).

Patient Counseling

It is imperative that hair restoration physicians develop open communication with their patients and counsel them about the appropriateness of hair restoration surgery. Based on the patient's expectations and the evaluation of the patient's age, pattern of alopecia, hereditary history, hair density in donor area/fringe, hair and skin color, hair texture, and scalp laxity, realistic goals can be developed. Each patient needs to be advised that significant changes in density will often require more than one or two transplant sessions. A thorough discussion of the risks and benefits of surgery should also occur at the preoperative visit.

Hairline design should be decided preoperatively with the patient's input. Low hairlines and blunted frontotemporal gulfs are to be avoided because these will result in an unnatural appearance. The anterior hairline should be set 7.5 to 9 cm above the glabella (**Fig. 31.7**). The contour of the hairline can be flared, rounded hemioval, flat, or slightly rounded. In addition, the hairline can be drawn with an irregular in-and-out microcontour. Feathering of smaller, single-hair follicular unit grafts anterior to the hairline allows for further blending of the hairline.

Fig. 31.6 Preoperative (a,c) and postoperative (b,d) views of hair transplantations. Each patient had one transplant session with 1,500 follicular unit transplants. The patient in (a) and (b) has finer hair. The patient in (c) and (d) has coarser hair, giving the appearance of a denser transplant result.

Fig. 31.7 The anterior hairline should be set 7.5 to 9 cm above the glabella. This may be more easily examined if the surgeon knows the width of his/her own palm. The contour of the hairline is rounded, but can also be flared, rounded hemioval, or flat, and drawn with an irregular in-and-out microcontour.

■ Technical Aspects of Procedure

Punch Grafts

In the 1950s, the work of Norman Orentreich popularized the use of punch grafts for hair restoration. The use of a 4 mm round punch graft, with 12 hairs per graft, was the standard treatment through the 1980s. Excellent density could be achieved with repeat sessions, but the effect often resulted in a tufted, unnatural appearance. The procedure involved punching out donor hair from the fringe area using hand- or power-driven punches. The use of power tools greatly hastened the process. The grafts were taken ~ 1 mm apart, and the donor sites were allowed to heal by secondary intention. Care had to be taken to align the punch with the angle of the hair shaft to minimize follicle transection. Next, punches were performed in the recipient area to make room for the harvested hair-bearing donor plugs (**Fig. 31.8**). These were typically made slightly smaller than the hair-bearing donor plugs and were spaced apart to allow adequate blood flow to the hair graft. Complete hair restoration using punch grafts required a minimum of three sessions to evenly fill out the recipient area. With these repeat sessions, excellent density could be achieved, though the result was often an unnatural "doll's head" appearance, with significant scarring in the donor area (**Fig. 31.9**).

The current treatment for patients presenting with the unsightly appearance of plugs is to partially punch out the plugs and redistribute the removed

Fig. 31.8 Intraoperative view of recipient sites created for punch grafts. These are staggered and require repeat procedures to fill in the ungrafted area.

Fig. 31.9 Postoperative result following four punch grafting sessions. This patient achieved excellent density, and with proper hairstyling can achieve a very natural result. However, certain situations, such as wet hair, give a "doll's head" appearance.

hair anterior and posterior to the plug reduction sites.[36] Scar revision can be performed to the donor site if adequate scalp laxity is present. In addition, the patients may be candidates for follicular unit grafting to help camouflage the plugs.

Follicular Unit Transplantation

The alternative to the 4 mm punch graft was to use higher numbers of much smaller grafts. Initially, mini-grafts containing 3 to 12 hairs and micrografts containing 1 to 2 hairs were used (**Fig. 31.10**). Currently, individual follicular units containing 1 to 4 hairs are being transplanted ▶ Video 31.1 (**Fig. 31.11**). The transplant of individual follicular units minimizes the transfer of non-hair-bearing tissue and therefore allows for maximal density of hair distribution. There is also a decrease in trauma to the recipient area because the need to create large recipient sites is obviated.
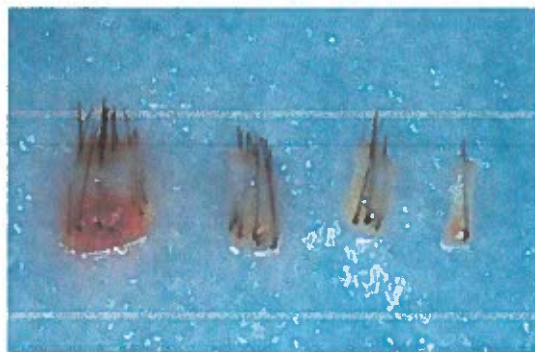
Fig. 31.10 View of various-sized grafts prior to placement. 4 mm square graft (*far left*), minigrafts (*center left and right*) containing between 3 and 12 hairs, and a micrograft (*far right*) containing fewer than 3 hairs.

Fig. 31.11 Close-up of a follicular unit transplant. (From Rousso DE, Presti PM. Follicular unit transplantation. Facial Plast Surg 2008;24:381–388. Used with permission.)

Anesthesia

Follicular unit transplantation can be performed under strictly local anesthesia or local with mild sedation. Thirty minutes prior to injection of local anesthesia, the patient is typically given 10 to 20 mg of oral diazepam, depending on the patient's habitus and experience with benzodiazepines. Supraorbital, occipital, and postauricular nerve blocks are performed with 2% lidocaine with 1:100,000 epinephrine. This is followed by a ring block using 1% lidocaine with 1:100,000 epinephrine. The scalp is then infused subdermally with 1% lidocaine with 1:100,000 epinephrine.

Strip versus Follicular Unit Extraction

The traditional method of harvesting donor hair is by harvesting a strip of tissue from the occipital scalp. The density of the occipital scalp will determine the number of grafts that can be obtained. The average density in Caucasians is 1 FU/mm² (~ 175 to 275 hairs/cm²).[37] The densest area of the occipital scalp and possibly the parietal scalp is chosen, and the proposed donor site is trimmed with scissors, leaving a 2 mm stub of hair to allow easier determination of the angle that the hair exits the scalp (**Fig. 31.12**). This aids in preventing transection of the follicles during harvesting. Harvesting of the strip in a block fashion can be performed with a no.10 blade (**Fig. 31.13**). The superior incision is beveled parallel to the direction of hair growth. The inferior incision is overbeveled, leaving deepithelialized follicles in the inferior portion of the scalp. This creates a trichophytic incision and allows for hair growth through the resulting scar (**Fig. 31.14**). Alternatively, a multibladed knife can be used for strip harvesting (**Fig. 31.15**). This method places multiple incisions paral-

Fig. 31.12 The donor area consists of the densest hair-bearing areas of the occipital scalp and possibly the parietal scalp. The hair is trimmed with scissors, leaving a 2 mm stub of hair to allow easier determination of the angle at which the hair exits the scalp. (From Rousso DE, Presti PM. Follicular unit transplantation. Facial Plast Surg 2008;24:381–388. Used with permission.)

Fig. 31.13 Harvesting of the strip in a block fashion can be performed with a no. 10 blade. (From Rousso DE, Presti PM. Follicular unit transplantation. Facial Plast Surg 2008;24:381–388. Used with permission.)
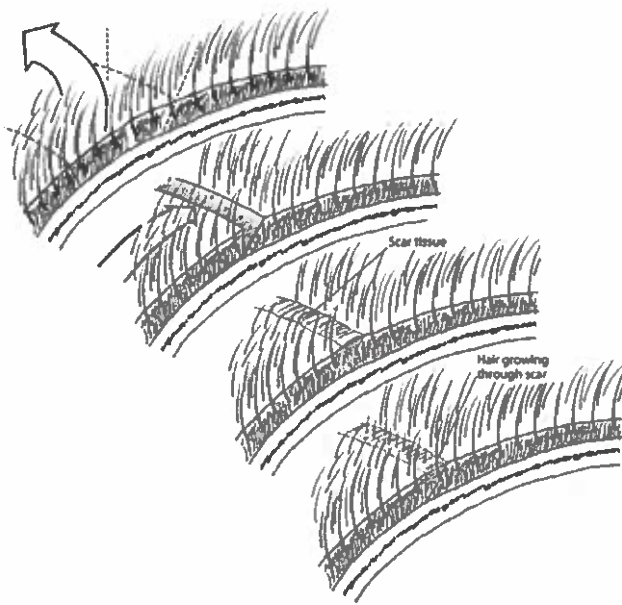
Fig. 31.14 Trichophytic incision. The superior incision is beveled parallel to the direction of hair growth. The inferior incision is overbeveled, leaving deepithelialized follicles in the inferior portion of the scalp. This allows for hair growth through the resulting scar.
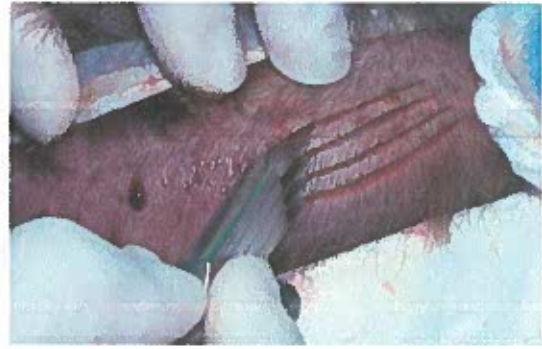

Fig. 31.15 The strip can be harvested either as a block excision or with a multibladed knife, as shown.

Fig. 31.16 Strip elevation is made in the deep subcutaneous plane, above the plane of the occipital arteries and nerves. (From Rousso DE, Presti PM. Follicular unit Transplantation. Facial Plast Surg 2008;24:381–388. Used with permission.)

Fig. 31.17 The harvested strip of hair.

lel to the direction of hair growth. However, its use increases the incidence of transection of the follicles and graft loss. To obtain a trichophytic closure with this method, one needs to return to the area and remove a strip of superficial skin from one side, leaving the hair bulbs in place.

Once the incisions are made, the strip is sharply dissected in the deep subcutaneous plane, above the plane of the occipital arteries and nerves (**Fig. 31.16**). The typical dimensions of the strip are 18 to 22 cm in length by 1 to 1.5 cm in width (**Fig. 31.17**).[38] The harvested strip is passed to the hair technician team, who dissect it into slivers that are one follicular unit thick, which can be more easily dissected into individual follicular unit grafts. The donor site defect is closed with a running locking 2–0 Prolene suture (Ethicon, Somerville, NJ). The suture is placed intradermally to avoid the hair follicle bulbs (**Figs. 31.18** and **31.19**). Stainless steel autostaples can also be used; however, in the authors' opinion, this is more uncomfortable for patients. The beveling performed allows a trichophytic closure that is easily concealed if hair length is maintained at ≥ 1 cm.

Alternatively, an individual follicular unit graft can be extracted from the scalp using a 1 mm punchlike instrument without the need for microscopic dissection. This method is called follicular unit extraction (FUE). The donor area is the entire possible donor zone in the occipital and parietal region, as well

as the low supraauricular region and neck, where smaller-caliber hairs can be obtained.[39] The entire donor area needs to be shaved prior to harvesting to allow better visualization and access. If the patient is concerned about shaving the area, then intervening strips of hair-bearing scalp can be left between the shaved areas, or the hair can be cut short on only the follicular units to be extracted. Both modifications
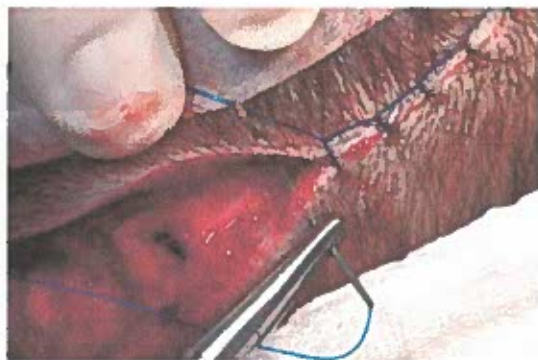
Fig. 31.18 A running locking 2–0 Prolene suture (Ethicon) is placed intradermally to close the donor site defect. (From Rousso DE, Presti PM. Follicular unit Transplantation. Facial Plast Surg 2008;24:381–388. Used with permission.)

Fig. 31.19 Closed donor site scalp incision.

Fig. 31.20 The harvested strip is initially dissected into slivers that are the thickness of a single follicular unit, which can be more easily dissected into individual follicular unit grafts.

Fig. 31.21 The slivers are dissected into follicular unit grafts.

are more time consuming, and care must be taken to ensure a random distribution of extraction sites.

The follicular extractions are performed with either a sharp or a dull dissecting punch of variable sizes, and the graft is removed with a forceps. The key is to align the punch with the angle of the hair shaft and presumably the follicles. Because the alignment of the hair follicle may not mimic the angle of the hair shaft, there is a risk of follicle transection with this method. Depending on the type of punch used, transection rates vary from 1.3% to more than 10%.[39]

The key benefit to follicular unit extractions is the absence of a linear scar in the donor site. No sutures are necessary, and there may be less postoperative discomfort. The disadvantages include an increased risk of follicular damage during the extraction, which can lead to suboptimal growth of the hair transplant.[40] The removed graft also has a decreased amount of surrounding fat and is at risk for desiccation while awaiting placement.

Follicular Unit Preparation

Once the strip is harvested, it is passed to the hair technicians for preparation. Throughout the process until placement, the grafts are kept moist in saline-soaked nonadherent gauze (Telfa, Kendall, Mansfield, MA). The harvested strip is initially dissected into single-follicular-unit-thick slivers under the microscope (**Fig. 31.20**). The slivers are then dissected into follicular unit transplants (**Fig. 31.21**).

Recipient Site Creation

The recipient sites can all be created prior to initiating implantation. Alternatively, they can be made along with placement of grafts, the so-called stick and place technique. The authors prefer to make all

of the recipient sites initially, prior to placing the grafts. This method gives the surgeon complete control over hairline placement, and the positioning and direction of each graft, and it gives time for the hair technicians to start dissecting the grafts.

A lightening knife handle with an SP-90 or SP-91 blade (Swann-Morton, Sheffield, England) or a 1.3 or 1.5 mm Minde knife with a 40 degree angle knife

(Surgical Specialties, San Juan, PR) is used to create precise stab incisions (**Figs. 31.22** and **31.23**). Attention is given to the surrounding hair growth to angle the incisions along the natural pattern of hair growth. The incisions are feathered along the anterior hairline.

Placement

The grafts are meticulously placed into the individual slits with fine jeweler's forceps (**Fig. 31.24**). One set of forceps can be used to open the slit and another to place a graft, but this is not necessary. Attention must be given to prevent already-placed grafts from extruding by avoiding undue tension and preventing bleeding. Hemostasis can be achieved with reinjection of 1% lidocaine with 1:50,000 epinephrine. Graft extrusion can also occur from large graft sizes, shallow recipient sites, and scar tissue at the recipient site (**Fig. 31.25**).

Megasessions

A traditional hair transplant session usually uses 2,000 or fewer grafts. During a megasession, 3,000 to 4,500 grafts may be placed, and a super megasession consists of over 4,500 grafts. Dramatic results can be obtained in one session but require significant operating time and a sufficient number of well-trained staff. One of the disadvantages of megasessions includes decreased graft survival because grafts are not inserted in a reasonable amount of time. Some patients are not candidates for a megasession due to inadequate donor hair density or scalp laxity.

Complications

Infection is a rare entity with follicular unit transplantation but can occur in less than 1% of cases. Perioperative use of antibiotics is routine. Bleeding is most commonly seen in the occipital region. By keeping the strip dissection in the deep subcutaneous plane, the occipital arteries can be avoided. Punch harvesting, such as follicular unit extraction,

Fig. 31.22 A lightening knife handle with an SP-90 (*upper*) or SP-91 (*lower*) blade.
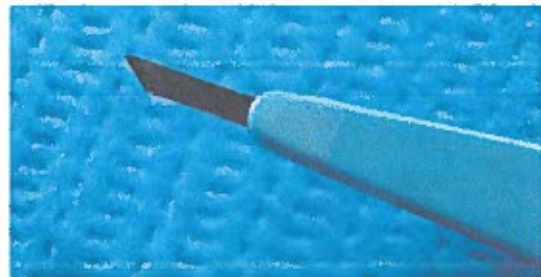

Fig. 31.23 A 1.3 Minde knife with a 40-degree-angle knife.

Fig. 31.24 Placement of a follicular unit transplant using a fine jeweler's forceps. (From Rousso DE, Presti PM. Follicular unit transplantation. Facial Plast Surg 2008;24:381–388. Used with permission.)

Fig. 31.25 View of scalp after placement of all transplants.

can rarely result in an arteriovenous fistulas. These can be encountered in repeat surgery and should be treated with suture ligation. If bleeding occurs after the patient has left the operating room, the patient is advised to apply manual pressure. Bleeding in the recipient spaces can be controlled with pressure or injection of 1% lidocaine with 1:50,000 epinephrine. Patients need to be forewarned about the possibility of postoperative edema, especially with extensive grafting. Edema can be decreased by maintaining 30-degree head elevation at night and with the use of oral prednisone during the first postoperative week.

Graft survival can diminish with prolonged exposure to dry, dehumidified air. All precautions should be taken to keep the grafts constantly bathed with saline prior to implantation. Prolonged time outside of the body also affects graft survival. Grafts kept out of the body for 6 hours have a 92% survival rate, and this rate decreases ~ 1% for each additional hour.[41] Any additional trauma, such as during placement, can also decrease graft survival. Placement of graft below the level of the epithelium can cause epithelial inclusion cysts and ingrown hairs to form.

Scarring is rarely a concern with small follicular unit grafting. In African Americans predisposed to keloids, a waiting period of 3 months after the initial session is adequate time to assess for any possible keloid formation prior to continuing with further transplant sessions.

Two to three months postoperatively, patients may experience a significant number of ingrown hairs as the transplanted hairs are growing. These can simply be uncapped in the office. If folliculitis develops, it may be treated with oral antibiotics, such as tetracycline.

Flaps

With the success and natural results obtained with hair transplants, flaps have become a less popular method of treating alopecia. However, there is a subset of patients who are excellent candidates for flap surgery. Patients with alopecia restricted to the frontal area are most ideal candidates. In addition, there are advantages to flaps over transplants. The results achieved are seen immediately. The blood supply is maintained to the hair follicles, therefore there is no associated hair loss secondary to follicles going into telogen phase. The hairs continue to grow throughout the process. Also, a higher density of hair can be achieved than with transplants.

Axial and Random

Flaps were initially described for scalp reconstruction in 1897 by Tillmanns.[42] They can be divided into axial and random flaps. Axial flaps are based on specific arteries and include the Juri flap, the temporoparietal

occipital (TPO) flap, and the temporoparietal (TP) flap. These are based on the superficial temporal artery.[43] The Juri and TPO flaps are twice delayed to ensure flap viability and, with a length of 25 cm, are designed to span the entire frontal hairline. The Juri flap is 4 cm wide, whereas the TPO flap is 3 cm wide. This modification allows easier turning of the base of the flap with less tendency for a lateral standing cone deformity. It also facilitates closure of the donor area. The Mayer and Fleming modification of the Juri flap makes the anterior hairline irregular, and gives a softer appearance.[44]

The TP flap is also based on the superficial temporal artery but is shorter, at 15 cm. For complete creation of a frontal hairline, it needs to be combined with a flap from the contralateral side.[44] Because the blood supply is primarily axial in nature, no delay procedure is required. Although the TPO flap is preferred, there are certain situations where the TP flap is the procedure of choice.[45] Patients with scarring that could interfere with blood flow to the distal tip of the TPO flap can undergo the shorter flap with less risk of flap compromise. Also, the TP flap can be used as salvage surgery for those with distal necrosis of longer flaps. Patients with poor results from punch grafts can also be salvaged with the TP but not the TPO flap due to the significant scarring in the occipital donor area.

The disadvantage to both the TPO and the TP flap is the posterior superior direction of hair growth, which can complicate styling. To overcome this issue, random blood supply flaps have been proposed. These are based more superiorly and result in hair growth directed anteriorly and inferiorly. Random flap procedures include those by Dardour,[46] Nataf et al,[47] and Frechet.[48] Because the blood supply of random flaps is not as predictable as that of axial flaps, problems with tip necrosis and early telogen are more likely to occur. These flaps are also shorter; therefore bilateral flaps need to be used to span the entire frontal hairline. It is possible to use tissue expanders prior to flap creation to improve results.[49] However, patients seeking cosmetic surgery may not be as tolerant of the deformity that is associated with tissue expanders, especially toward the end of the expansion process.

Alopecia Reduction

Alopecia reduction techniques allow the surgeon to excise the balding scalp and close the defect by advancing the temporal and parietal scalp. Alopecia reduction procedures are limited by the degree of scalp laxity. Excessive wound closure tension is associated with widening of the scar and possible necrosis, as well as the stretch-back phenomenon, which is the tendency of the remaining bald scalp to expand after a reduction. Often, complete excision of the bald area requires a series of reduction procedures, with 3 months between procedures to allow for scalp relax-

ation. The time between procedures can be shortened with the use of tissue expanders or extenders. These devices apply forces that stimulate mechanical creep, an increase in connective tissue between the cells, as well as biological creep, an increase in cell division.

A tissue expander is a Silastic balloon implanted under the skin adjacent to the area of alopecia that is inflated with a series of percutaneous injections of saline into a self-sealing port over a course of 8 to 10 weeks (Model SRE-RD-640s, Specialty Surgical Products, Inc., Victor, MT).[50] Although tissue expanders can allow for complete excision of the bald area with one reduction procedure, the deformity that the patient has to endure limits its use in cosmetic procedures (**Fig. 31.26**). As an alternative, Frechet[51] introduced the scalp extender in 1993. This device is a Silastic band with two series of hooks that are engaged into the undersurface of the scalp at the time of the initial reduction procedure (Frechet extender, France). It is left in place for 4 weeks, allowing for continuous horizontal traction force and tissue stretch with minimal cosmetic deformity. At 4 weeks, the device is removed and a more complete reduction procedure can be performed.

Various patterns of alopecia excisions have been proposed, with the midline sagittal ellipse pattern being the most simple. Placing the elliptical incision in a paramedian position or in a reverse J fashion provides for easier scar camouflage. Other patterns include a Mercedes or a horseshoe (**Fig. 31.27**). These patterns differ in the resulting direction of hair growth and the position of the scar and are individualized to each patient's pattern of hair loss. The incision is made through the galea, and the scalp is undermined to the superior attachments of the auricles and the nuchal line in the subgaleal plane. The flaps are advanced, and excess alopecic scalp is excised, followed by tension-free wound closure in two layers.

The extensive scalp lift continues the undermining beyond the nuchal line into the nape of the hairline, with ligation of the occipital arteries to maximize mobility.[52] The extensive scalp lift permits much
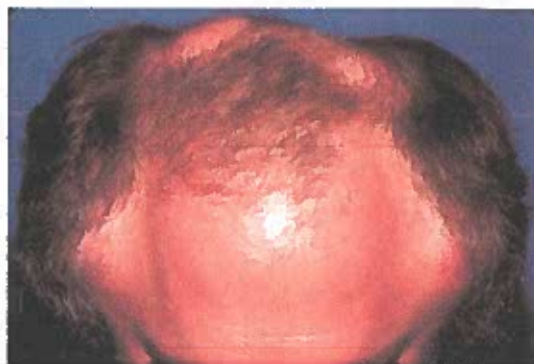

Fig. 31.26   The deformity with use of tissue expanders can prevent its use in cosmetic procedures.

greater mobility of the hair-bearing scalp but is associated with an incidence of occipital hair-bearing scalp necrosis. Mangubat[53] has modified this procedure to preserve the occipital artery and eliminate the incidence of necrosis.

Patients with crown balding are excellent candidates for scalp reduction and obtain results superior to transplantation alone. In general, scalp reduction is usually followed by hair transplantation to complete the restoration and to camouflage the scar area.

■ Postoperative Care

Following hair transplantation, hairline advancement, or scalp reduction, the patient is sent home with a nonadhesive dressing covering the scalp. This is removed the morning after surgery during the first postoperative appointment. The patient is to shower daily with lukewarm water and use baby shampoo starting the second night after surgery. Patients are told to gently pat the scalp with the balls of their
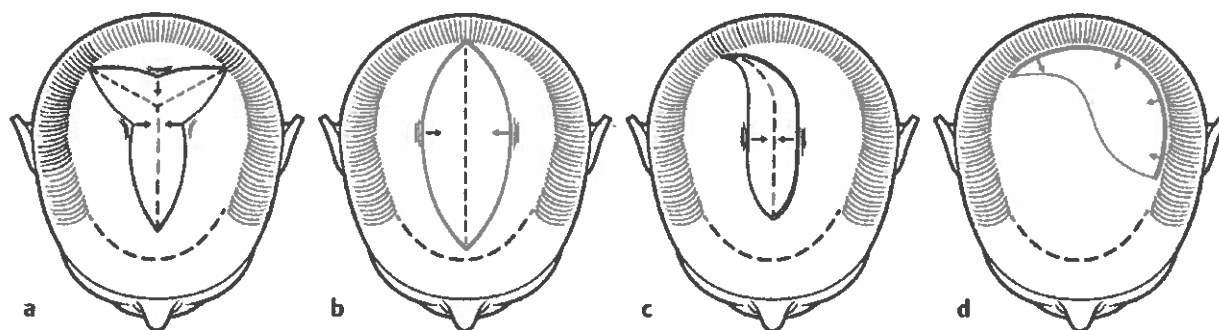

Fig. 31.27   Various patterns of alopecia excisions. (a) Mercedes. (b) Midline sagittal ellipse. (c) Reverse J. (d) Parasagittal.

fingertips during shampooing. Additionally, the patients are to apply witch hazel four to six times a day to the graft and donor sites, or the reduction incision, until all scabs and crusts are gone. Patients can expect some hair to fall out along with the scabs. To help decrease edema, patients are encouraged to sleep with their head elevated 30 to 40 degrees using pillows, a wedge cushion, or a recliner for 2 weeks. Patients are also restricted from heavy exercise for 2 weeks and should not bend over at the waist to pick up objects. Patients may use a hair dryer on a cool setting only and may comb hair with a clean wide-tooth comb, not a brush. Sutures and staples are removed on postoperative day 10.

Patients are told to contact the clinic and report any temperature elevations, sudden swelling or discoloration, excessive bleeding, discoloration from the wound edges or other evidence of infection, and development of any drug reaction.

Patients may expect the transplanted grafts to fall out ~ 4 weeks postoperatively. The graft hair will remain in telogen, the resting phase, for ~ 3 months

and enter anagen to begin regrowth thereafter. Initially, the hair will be very fine, but as the hair grows longer, it will become similar to the hair texture and color in the donor area. Within 5 to 6 months postoperatively, patients can expect to have hair that is long enough to style and comb.

■ Conclusion

No other area in cosmetic surgery has undergone as much transformation in the past 20 years as hair restoration surgery. Today's methods of follicular unit transplantation can provide most patients with excellent, natural-appearing results that are indistinguishable from naturally occurring hair (**Figs. 31.28** and **31.29**). Proper patient selection and counseling are crucial for a successful outcome. Women can also achieve excellent results with follicular unit grafting but need to be evaluated preoperatively for underlying endocrine abnormalities.
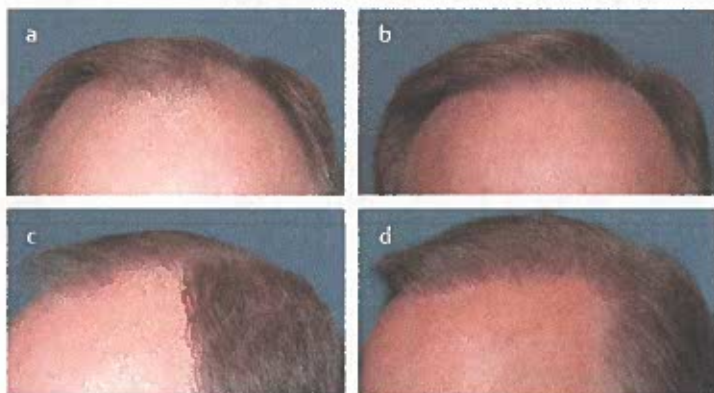

Fig. 31.28 Preoperative (a,c) and postoperative (b,d) views of a 45-year-old man who underwent three hair transplantation sessions, showing the pleasingly natural results that can be achieved.
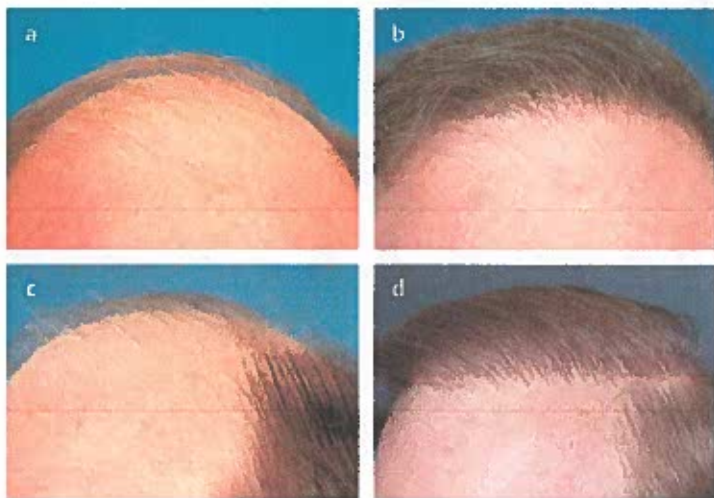

Fig. 31.29 Preoperative (a,c) and postoperative (b,d) views of a 52-year-old man who underwent three hair transplantation sessions, showing the natural results that can be achieved.

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