Introduction

The production of hair is a characteristic unique to warm-blooded mammals. To our evolutionary ancestors, this outer covering primarily served the purpose of insulation, although secondary roles included protection from injury, camouflage and sexual or social communication. These secondary roles, particularly social and sexual signaling, are the primary uses for hair amongst humans. Hair and its color, texture, length and style are highly variable between individuals. It contributes significantly to the unique appearance of each person and therefore affects both our self-image and the way we are perceived by others. This, in addition to the emphasis that our society places on personal appearance, explains why the loss of hair is so distressing to some individuals and why some may seek methods of hair replacement.

Anatomy and Physiology

A basic understanding of scalp and hair follicle anatomy is essential to the physician managing alopecia. The scalp is made up of five layers that can be remembered using the mnemonic SCALP. The outermost layer is the Skin. The sub-Cutaneous layer contains fat, connective tissue, vessels, lymphatics and nerves. The galea Aponeurotica is a tendon-like structure to which the frontalis muscle inserts anteriorly and the occipitalis muscle inserts posteriorly. Loose connective tissue lies between the galea and the Pericranium or Periosteum of the skull. The scalp has a robust blood supply derived from both the internal and external carotid systems. Anteriorly, the supratrochlear and supraorbital vessels are found. The superficial temporal and retroauricular vessels supply the lateral scalp, and posteriorly are the occipital vessels. Venous drainage follows the arteries, of note is that the emissary veins and the ophthalmic veins drain intracranially and have the potential to allow spread of infection to this space. Many surgical procedures on the scalp may be performed under local anesthesia, therefore it is relevant to understand the innervation of this area. The supratrochlear and supraorbital branches of the ophthalmic division of the trigeminal nerve innervate the forehead and frontal scalp. The maxillary division, via the
zygomaticotemporal branch, supplies the temple region. Sensation of the lateral scalp is
provided by the mandibular division’s auriculotemporal nerve. The cervical plexus
contributes to the great auricular and lesser occipital nerves that innervate the
postauricular area. Finally, the occiput and vertex are innervated by the greater occipital
nerve. Motor innervation of the frontalis, occipitalis and auricular muscles is provided by
the facial nerve (1).

The hair follicles begin to develop between 9 and 12 weeks gestational age and
are derived from the ectodermal and mesodermal layers of the embryo. The ectoderm
gives rise to the hair matrix cells and the melanocytes responsible for the pigmentation of
hair. Two buds form off of this layer, one gives rise to the sebaceous gland and the other
forms the area of attachment for the erector pili muscle. The erector pili muscle itself, the
hair dermal papilla, the fibrous follicular sheath and feeding blood vessels all arise from
the mesoderm. Hair follicle epithelial growth continues down into the mesoderm until
the follicle has reached its full size. At this time, matrix cells begin dividing and pushing
upward, eventually forming a hair shaft. Hair production can typically be seen by 16 to
20 weeks gestation, initially forming fine lanugo hair. Some of the lanugo hair will be
shed around 32 to 36 weeks and after this time more substantial hair may develop on the
scalp, eyebrows and eyelashes.

The outer root sheath and inner root sheath surround the developing hair shaft.
The shaft itself is made up of three layers: an outer cuticle, the bulky middle cortex and
the inner medulla. The cuticle is made of cells containing dense keratin and serves a
protective function. The cortex consists of tightly packed spindle shaped cells containing
keratin and some pigment. The medulla, whose role is unknown, contains cells with
cytoplasmic vacuoles that become air-filled as the cells push upward to the epidermis.

There are an estimated 5 million hair follicles on our bodies. One hundred to one
hundred and fifty thousand of these are located on the scalp. Transverse or horizontal
sections of the scalp show that hair follicles are organized into follicular units. Each unit
contains one to four terminal hairs, one or two vellus hairs, nine sebaceous glands and
erector pili muscle insertions and a perifollicular vascular plexus, neural net and
connective tissue. These units are arranged in a regular mosaic pattern and likely
function as distinct physiologic entities. The density of hair follicles is approximately
1135/cm² at birth and quickly decreases to 795/cm² by 1 year of age. A gradual decrease
is then seen so that 20-30 year olds average 615 follicles/cm², 30-50 year olds average
485/cm² and by age 80-90 average only 435/cm².

Hair growth follows a cyclic pattern consisting of three phases. Anagen is the
active growth phase and its duration varies by anatomic location. In the scalp, anagen
generally lasts between 2 and 8 years. Typically, about 90% of hair follicles in an adult
are in the anagen phase at any given time. The catagen phase, which lasts between 2 and
4 weeks, is characterized by separation of the hair shaft from the dermal papilla and
migration toward the epidermis. During the telogen or resting phase, hair growth ceases
and the follicle attachment weakens until the hair eventually sheds. About 10% of adult
hair follicles are in this phase, which lasts 2 to 4 months.
Scalp hair growth occurs at a rate of approximately .37-.44mm/day and normal scalp hair loss is 50-100 hairs/day. Alopecia results when there is an imbalance between the phases of the hair growth cycle such that loss exceeds growth (2,3).

**Androgenetic Alopecia**

Hair loss is a common problem for both men and women and is associated with a wide variety of conditions. The most common form of hair loss is androgenetic alopecia (AGA) or male pattern balding (MPB). AGA is characterized by a patterned loss of hair that typically affects the bitemporal and frontal hairline first, followed by diffuse thinning of hair over the vertex. As this process progresses, the bald patch over the vertex gradually joins the receding frontal hairline, eventually leaving behind a horseshoe-shaped band of hair on the parietal and occipital scalp. AGA is caused by a gradual miniaturization of hair follicles with each successive hair cycle. The dermal papilla is most likely the target for these androgen-induced changes. As the follicle becomes smaller, hair becomes finer and less pigmented. Also, the time of each cycle spent in anagen becomes shorter while the telogen phase becomes longer. The ratio of time spent in anagen compared to telogen reduces from 12:1 to 5:1. Time in anagen is the primary determinate of hair length, so as this time shortens, so does the length of hair. In addition, the latency between shedding and the next anagen phase becomes longer, reducing the number of hairs present on the scalp at any given time.

AGA occurs in genetically predisposed individuals as long as they have adequate levels of circulating androgens. Although this condition is often regarded as a problem for men, actually equal numbers of men and women are affected by AGA. It is transmitted in an autosomal dominant pattern but has variable penetrance. Approximately 33% of people with a positive family history of AGA will be affected. Thirty percent of white men have AGA by age 30, 50% are affected by age 50.

Circulating androgens have different affects on hair depending on location. Vellus prepubertal pubic, axillary, chest and beard hair follicles are stimulated to grow into terminal hairs. The same hormones have the opposite effect on hair follicles in the scalp. The reason for this site-specific action is not clear. The main circulating androgen in men is testosterone, while in women dehydroepiandrosterone sulfate, androstenediol sulfate and 4-androstenedione are most abundant. The enzyme 5-alpha reductase, which has two forms—type 1 and 2—present in the scalp, converts testosterone or the adrenal androgens to dihydrotestosterone. This metabolite binds to androgen receptors five times more effectively than the parent molecules. Studies have shown that patients with androgen insensitivity syndromes and 5-alpha reductase type 2 deficiencies do not develop AGA. Therefore, we know that the presence of normal androgen receptors and adequate levels of dihydrotestosterone are prerequisites for the production of AGA. However, we do not clearly understand whether individual follicle susceptibility to androgen induced involution is related to increased numbers of androgen receptors, increased local production of dihydrotestosterone or increased levels of circulating androgens (2,4).
Classification

Several different clinical classification systems that outline the progressive and predictable pattern of hair loss in androgenetic alopecia have been devised. Hamilton, in 1951, was the first to publish such a schematic but his system was later revised by Norwood in 1973. Today, the Norwood system is most commonly used to classify male-pattern balding. Female androgenetic alopecia follows the Ludwig classification system that was introduced in 1977 (2).

Medical Therapy

Many patients, particularly those in the early stages of hair loss, will prefer to try medical therapy prior to consideration of any surgical intervention. The aim of such medical treatment is twofold: first, to increase hair coverage of the scalp and second, to prevent further hair thinning and/or loss. Currently there are two FDA approved drugs available in the United States for the treatment of AGA.

Minoxidil was initially introduced as an antihypertensive whose mechanism of action is opening of potassium channels and vasodilatation. The occurrence of hypertrichosis as a side effect of this medication led to investigation for its use in AGA. Although the mechanism of action is not known, topical minoxidil has been demonstrated to both stop the progression of balding and reverse some of the changes in the hair follicle induced by AGA. Specifically, vellus hairs develop into terminal hairs, miniaturized hair follicles revert to their normal morphology and the number of hair follicles in the anagen phase increases. Topical minoxidil, available in 2% and 5% formulations, is safe for use in men and women with AGA. The efficacy of both of these solutions has been demonstrated in several large, placebo-controlled studies although individual response to the drug is variable. Results of minoxidil use are noticeable only after several weeks of use and application must be continued in order to retain the effects. Side effects are minimal and primarily include local effects such as scalp dryness, itching or scaling. Women using topical minoxidil have a higher incidence of hypertrichosis that typically occurs above the upper lip, on the chin, between the eyebrows or over the cheeks (5,8).

Finasteride specifically inhibits the action of the enzyme 5-alpha reductase type 2, thereby blocking the conversion of testosterone to dihydrotestosterone and reducing circulating levels of dihydrotestosterone by as much as 60%. Lowering hormone levels likely slows or stops the process of androgen-induced miniaturization of hair follicles. Finasteride, in doses of 1mg/day, has been shown to effectively treat AGA in three blinded, placebo-controlled, randomized studies. Improvement in scalp hair counts and on self and expert assessments of hair growth has been demonstrated. Like minoxidil, results are seen only after several months of therapy and are rapidly reversed upon discontinuing the drug. Finasteride does not act directly on the androgen receptor and therefore does not interfere with the normal activity of testosterone. Side effects are similar to those seen with placebo except a 1.8% reported incidence of decreased libido. Finasteride therapy cannot be safely used in women of reproductive age since 5-alpha reductase inhibition during pregnancy may lead to genital abnormalities in male fetuses.
Additionally, a study of its use in postmenopausal women with AGA failed to demonstrate any efficacy in slowing or reversing hair loss (8).

**Patient Evaluation for Surgery**

It has been well established by several studies that hair loss can have a profound negative psychosocial impact on individuals suffering from the condition. Balding men not only report feeling less attractive and an overall dissatisfaction with their body image, but also, experiencing significant distress about their hair loss. This negative self-image considerably effects quality of life. Also negative social stereotypes are applied to individuals with hair loss. They tend to be perceived as older, weaker and less attractive than individuals without hair loss (5). Given these negative effects of hair loss, it is understandable why so many people seek treatment for the condition.

Hair replacement therapies are cosmetic procedures, and as with any cosmetic surgery, appropriate patient selection for these treatments is key to a successful outcome. These patients must have realistic expectations for improvement of their hairline, therefore adequate counseling regarding what can and cannot be accomplished for each individual is imperative. While an improvement in self-image and self-confidence is expected following hair replacement, a patient with the belief that it will resolve all of life’s problems is doomed for disappointment. It is best if the patient is self-motivated in seeking hair replacement rather than acting on outside influences from family or friends. Self-motivated patients are most likely to complete the full course of replacement therapy, which may include two or more surgeries. Because the course may be long before the end result is achieved, patients must be willing to live through some inconvenience (6).

It is important to emphasize that the hair replacement techniques available today do not result in new hair growth, but instead involve redistribution of remaining hair. Therefore, in order to be a candidate for these procedures, a patient must have adequate donor hair available. Also, it must be determined that the patient has a type of hair loss associated with “donor dominance”—meaning that the relocated hair retains the characteristics of the donor scalp rather than adopting characteristics of the recipient scalp. The donor, hair-bearing scalp is measured and assessed in terms of density and shaft diameter. Type I donor hair is dense, 20 hairs/4mm², and coarse. Type II donor hair demonstrates 14 hairs/4mm² and is thick and strong. Types I and II are most ideal for hair replacement surgery. Type III is associated with fine, weak hair with a density of 10/4mm². Finally, type IV hair is very thin and has only 6 hairs/4mm². Type III donor hair can still be used for hair replacement, but the outcome will not be as good. Patients with type IV hair are generally not considered candidates for these procedures (2,7).

Age is not a contraindication for hair replacement surgery. In fact, the ideal patient may well be older individuals, in whom a well-established pattern of hair loss is present. Also in older patients, gray or salt-and-pepper hair may actually provide better coverage of the balding scalp. Young patients seeking surgery present the problem of trying to predict future hair loss, particularly in those under 25 years of age. In this group, a conservative approach is prudent (6).
Surgical Therapy

The types of surgery for alopecia fall into three basic categories—scalp reduction aimed at reducing balding scalp and expanding hair-bearing scalp, scalp rotation flaps aimed at redistributing hair-bearing scalp, and hair grafting methods aimed at transferring follicular units from the hair-bearing scalp to balding areas.

Scalp Reduction

Reduction of the balding area of the scalp can be accomplished in several ways, the first of which is serial excision of non-hair-bearing scalp. This process was originally described in 1978 by Unger and Unger. They outlined six different patterns of scalp reduction including the sagittal midline ellipse, “Y” pattern, lateral patterns (including “S”, “I” and “C” shape excisions), “U” pattern and miscellaneous patterns including the “T”, “I”, transverse ellipse and crescent ellipse. Of course, the pattern of excision must be individualized to each patient taking into account the shape of the bald area and the availability of donor scalp. This technique has enjoyed widespread use, primarily because of ease of performance and reliability of results. Essentially after the pattern of excision has been determined, the bald scalp is excised down to but not through the pericranium, followed by wide undermining of remaining scalp and primary closure of the wound in two layers—the galea and skin (9).

A potential pitfall of this technique is excessive scalp excision, with resultant excessive tension on wound closure. It is well known that excessive tension has negative effects on wound healing including necrosis of tissue and widening of scars. The challenge is finding just the right amount of tension to enhance stretching and expansion of scalp flaps without toppling over into the realm of excessive tension and increased complications. Using a dynamometer, Raposio and Nordstrom found that the amount of tension that afforded the best cost/benefit ratio in the performance of scalp reductions to be within the range of 500 to 1,500 gr., or 4-10 times the natural tension of a scalp flap. Clinical application of these numbers is somewhat difficult to determine (10). Relaxing incisions in the galea aponeurotica, galeotomies, have been utilized to reduce wound tension after scalp reduction procedures. In an attempt to quantitate the gain in scalp flap advancement and the reduction in closing tension, Raposio, et al performed galeotomies in ten patients undergoing scalp reduction. They created three full thickness incisions in the galea, equal in length to the scalp incision, on the deep surface of the scalp flap, the first 2cm from the wound margin and the subsequent two 1cm away from the preceding. The result was an average gain in flap length of 1.67mm/galeotomy and a 40% reduction in wound closing tension (11).

One of the major problems with scalp reduction is the phenomenon of “stretch-back” which refers to the tendency for the bald scalp to expand after each reduction. The amount of stretch-back is dependent upon the elastic properties of the scalp and varies between 10 and 50% of the reduction. Most of this re-expansion occurs within two months of surgery (12). Scalp extenders, anchoring galeal flaps and the “Nordstrom suture” have been developed to reduce stretch-back. Frechet introduced scalp extenders in 1993. The extender itself consists of a 1mm thick sheet of silastic with two opposing
rows of titanium hooks. It has the ability to stretch up to 200% and demonstrates memory with a tendency to return to its natural shape. After scalp reduction is performed, this apparatus is attached to the deep surface of the galea parallel to and 1-2cm lateral to the wound margin on one scalp flap. It is then stretched and attached to the opposite flap at a predetermined distance from the wound margin. The wound is closed in layers and the extender left in place for 4-6 weeks. The tendency of the extender to return to its original size places continuous stretch upon the lateral scalp toward the incision line—effectively producing a negative stretch-back or shrinking of the bald area. When the extender is removed at a second stage, more lateral hair-bearing scalp is available so the further scalp reduction can be performed. This technique asserts the benefit of maximizing the area of scalp that can be excised while minimizing the number of procedures needed and the time to achieve results (13). Raposio, et al described the use of anchoring galeal flaps in 1998. In this technique, scalp excision is performed routinely except that on one side of the incision the galea is not excised. Instead, three rectangular 2x3cm segments of galea are left in continuity with the scalp flap. These are then sutured to the undersurface of the opposing flap, drawing the wound together. The incision line is then closed in the usual fashion of two layers. The benefit of the use of galeal flaps was found to be a reduction in stretch-back of 80-88% at one month after surgery (14). The “Nordstrom suture” is the most recently described technique to reduce stretch-back. It is a suture of silicone polymer, 2mm in diameter and attached to a heavy cutting needle, that is capable of stretching to 400% of its original length. After scalp reduction is performed, the suture is introduced through the galea and tied on itself. The galea is then reapproximated using a running, buried, mattress suture, taking 1-2cm bites of galea. The Nordstrom suture induces a shrinking of the remaining bald area that has been shown to be three times greater than that seen with scalp extenders. It also has the advantage of being capable of placing stretch in different directions simultaneously. Another benefit is the ability to remove the suture without a second operation (15).

Tissue expanders are quite useful in the treatment of alopecia. Placement of these devices actually increases the total surface area of hair-bearing scalp available to cover balding areas. The number of hair follicles is not increased, rather the skin between follicles is expanded, providing a more even distribution hair density. The expander is placed in the avascular layer between the galea and the pericranium. The overlying galea protects the feeding vessels of the scalp that run in the subcutaneous layer, a characteristic that allows higher filling pressures than are tolerated elsewhere. Various sizes and shapes of expanders are available and are selected depending upon the area of alopecia to be covered and the donor scalp available. Most devices utilize a remote injection port. The location of incisions for expander placement warrants some consideration so that the end result yields hidden scars. One option is to place the incision in an area planned for future excision. Another option would be to place the incision along one side of a planned scalp flap. Incisions must be distant enough from the expander to minimize the risk of extrusion but close enough to facilitate accurate placement of the device. Typically, at the time of placement, the expander is injected with sufficient saline to obliterate the dead space created by scalp elevation. Most authors recommend waiting 1-2 weeks to begin filling the device. Injections are then performed once or twice a week in the office, the amount tolerated will vary between
individuals but is limited by tissue blanching that would indicate compromise of blood supply. This process typically takes 6-10 weeks before adequate expansion is achieved. The advantages of tissue expansion are, as mentioned previously, the increased area of hair-bearing scalp available for coverage so that the end result can often be accomplished more rapidly and with fewer operative procedures than with other methods. Disadvantages include the cosmetic deformity imparted by the inflated expander, frequent office visits for injection and discomfort associated with inflation. Even if results are achieved more quickly that with other methods, patients must be highly motivated and understand the significant time commitment of this process (16).

Scalp rotation flaps

The use of scalp advancement or rotation flaps has the advantage providing immediate coverage of alopecic areas with dense hair-bearing tissue. Types of scalp flaps include the lateral scalp flap, the temporoparietal occipital (TPO) or Juri flap, the preauricular flap and free scalp flaps. Of these, the most widely utilized is the TPO flap, which was initially described by Juri in 1975.

The Juri flap is primarily useful in replacing balding frontal scalp. Candidates for this technique must possess adequate lateral and posterior hear-bearing scalp. This flap is based anteriorly on the parietal branch of the superficial temporal artery. The artery is identified by palpation or Doppler and marked on the scalp. The flap is then designed around the artery with a pedicle width of 2-2.5cm. This protects the vessels from excessive torsion while minimizing resistance to the 180-degree transposition necessary. The flap is then widened to 4-6cm and is curved postauricularly into the occipital region. The potential length of this flap is typically between 23 and 25cm giving a length-to-width ratio of approximately 7:1. This ratio necessitates a delay between incising and raising the flap in order to ensure adequate blood supply. Some authors advocate two delay procedures, the first to incise the temporal and parietal margins of the flap and the second to incise the occipital portion. Ten to fourteen days after incision the flap is raised and inset. Prior to insetting, the frontal hairline is designed and marked with the patient in an upright position. It is essential that the newly designed hairline is not excessively low on the forehead and maintains a natural age-appropriate appearance with bilateral frontotemporal triangles. The anterior incision is created and the flap transposed. The donor site is widely undermined and reapproximated in two layers before excising any balding scalp in order to ensure a tension free closure. The redundant frontal scalp is then excised and the flap sutured in place. To soften the appearance of the new frontal hairline, 2mm along the anterior aspect of the flap may be deepithelialized to encourage hair growth through the scar. Disadvantages of the Juri flap include the need for one or more delay procedures, the potential need for revision of a temporal dog-ear and the unnatural posterior direction of frontal hair growth. To avoid some of these drawbacks, Juri and Juri also describe the use of a free TPO flap. The design of the flap is essentially the same as with the rotation flap, with the potential for added length up to 30cm. First, the recipient superficial temporal vessels are isolated, typically at the level of the tragus. The flap is then raised and bleeding along the entire length of the flap can be confirmed, this obviates the need for any delay. The flap vessels are clamped and ligated and the flap transferred. Reanastomosis of the vessels is performed and bleeding
from the flap confirmed. The donor site is closed and the flap inset in the previously described fashion. The reversed orientation of the flap allows for a more natural anteriorly directed pattern of frontal hair growth (17).

**Hair grafting**

The method of using small full-thickness autografts of hair-bearing skin to correct alopecia was first described by Okura, a Japanese dermatologist, in 1939. His work went essentially unrecognized and the credit for introducing this technique is often given to Orentreich who published his experience in 1959. Since then, hair transplantation has become the most common cosmetic procedure performed in men. The three most commonly described types of hair grafting techniques are the punch graft, the strip graft and micro- or minigrafting.

The original technique described by Okura and Orentreich was that of punch grafting. This method utilizes small, typically 4-5mm, sharp, round punch trephines to harvest hair-bearing tissue, most often from the parietal and occipital scalp. This provides grafts that typically contain 10-20 hairs/punch depending on hair density of the donor site. Similar punch trephines are also utilized to create recipient sites in the balding areas. The recipient punch is .25-1.0mm smaller than the donor punch to allow for expected graft shrinkage after harvest. Important aspects of this technique are proper spacing of the punches so as to not compromise blood supply to the remaining scalp, directing the recipient punches to allow for hair growth in a natural direction and appropriate timing of subsequent sessions. It is not unusual for four or more sessions to be necessary to achieve an optimal result. Most surgeons advocate waiting a minimum of 6 weeks between surgeries, others time subsequent procedures based on growth from previous grafting so that hair distribution can be visualized and the remaining spaces filled in with new grafts (18, 19).

Vallis was the first to describe strip grafting in 1964. This technique involves a free composite graft of hair-bearing scalp. It is most commonly utilized to enhance the appearance of a spotty or thin frontal hairline. The length of the graft is limited only by the donor tissue available and the amount of frontal hairline to be covered. The width of the graft is the limiting factor in graft survival. In previously unoperated frontal scalp, a graft up to 8mm wide can take without difficulty. However, a majority of patients undergoing strip grafting will have previously undergone punch grafting. In these cases, a width of 5mm is more likely to survive. The graft is harvested by creating two parallel, horizontal incisions in the donor scalp, down to the level of the galea. It is elevated in this plane and the donor site closed. The recipient site is incised along the anterior border of the existing hairline. In this case, the fascia is incised to allow some relaxation of the recipient site and accommodate the graft. The graft is inset so that the hair follicles are angled anteriorly to mimic the natural direction of hair growth. The graft is then sewn in place. Typically strip grafting is performed in two stages, first reconstructing one side of the frontal hairline and then the other (19, 20).

Follicular-unit transplantation is probably the most widely used hair grafting method today. In this technique, large numbers of minigrafts (3-4 hairs/graft) and
micrografts (1-2 hairs/graft) are utilized to cover significant areas of balding scalp. Several different methods of follicular-unit transfer are described by various authors, herein is the technique as outlined by Barrera. Donor hair is harvested from the occipital scalp in one large ellipse, the size of which is determined by hair density, scalp elasticity and the area of alopecia to be covered. After injection of local anesthesia, the donor site is marked and the subcutaneous tissue infiltrated with 30cc of tumescent solution. This simplifies the harvesting process by separating the donor tissue from the underlying fascia and improving hemostasis. It also aids graft dissection by separating the hair follicles from each other. After the donor ellipse is resected, the site is closed in one layer without undermining. Graft dissection proceeds on a separate table under bright lighting and magnification. The donor tissue is first cut into 2mm segments, aligning all incisions in the direction of follicle growth. Then, a #10 blade is used to further dissect the segments into micro- and minigrafts, taking care to preserve natural groupings of hair follicles. The grafts are kept moist and cool while the recipient area is prepared. Again, a tumescent solution is infiltrated into the recipient scalp. Since the frontal hairline is the most critical for achieving a satisfactory result, graft insertion begins here and priority is given to obtaining optimal density in this area prior to proceeding to the crown and vertex regions. Slits are created in the recipient scalp using a #11 blade. As the surgeon partially removes the scalpel, an assistant inserts a graft using jeweler’s forceps. During the first pass, the slits are placed 4-5mm apart, a second and sometimes third pass over the same area may be performed to obtain the desired density. The direction of grafted hair growth can be controlled by changing the angle of the scalpel when creating the slits. This is particularly important along the frontal hairline where the direction of growth should be angled 45-60 degrees anteriorly. If there are residual native hairs in the region being grafted, the slits should be placed parallel to the existing hairs. Barrera advocates placement of a moist, light pressure dressing that is removed after 48 hours. Patients are then instructed to shampoo daily and begin applying minoxidil 2% topical spray twice daily (2).

Pitfalls and Complications

The goal of any hair replacement technique is to restore a natural appearance of the hair to the individual patient’s satisfaction. Poor outcomes or patient dissatisfaction often occur as a result of an unnatural or “operated on” appearance. The good news is that many corrective techniques have been developed to improve upon undesirable outcomes. For the patient who wishes complete reversal of hair transplantation, graft removal can be performed. The grafts are excised with a punch trephine and the defects closed primarily. Often, this needs to be done over several stages so that excessive tension is avoided on the punch closures. To reposition poorly placed grafts, the same method of punch removal of grafts can be utilized, but the excised hair is then retransplanted in the appropriate location. Correction of rows of large punch grafts—the cornrow plugs—can be done by reducing the size of the original grafts and placing smaller grafts in the intervening spaces. Excessively thin transplanted hair or an unnaturally abrupt thick frontal hairline can be improved with additional grafting. This will be dependent upon adequate remaining donor hair. To soften the frontal hairline, placement of several layers of irregularly spaced mini- or micrografts can be very effective. Unsightly donor site or scalp reduction scars may be improved with scar
revision techniques. For any revision procedure, appropriate preoperative counseling cannot be overemphasized. A good outcome and a satisfied patient will be dependent not only on choosing the right method of correction and meticulous performance of surgery, but also on honest communication with the patient about what he/she can expect from the surgery. As Epstein said, “an uninformed patient is a disappointed patient” (21,22).

Conclusion

As detailed in this discussion, there are a wide variety of methods available for the correction of alopecia. No one procedure is necessarily better than the others and the application of these techniques must be individualized to each patient. As more experience is gained by physicians performing these procedures, it is likely that patient satisfaction will increase, potentially also increasing the number of patients seeking these therapies.

BIBLIOGRAPHY


