If the basic concepts of treatment of male pattern baldness prove to be incorrect, the causative mechanism will not be revealed and effective treatment will not be developed for this condition.

19.1 Review of Hormonal Mechanisms in Hair Regeneration

Hamilton (1942) was the first to report that male hormones play a very important role in hair regeneration. Testosterone originally shows weak hormonal action, but, when it is converted to 5α-DHT (dihydrotestosterone) by 5α-reductase, this compound shows strong hormonal effects (Fig. 15.1). Testosterone produced in the testes is carried to the hair matrix by plasma circulation and promotes hair formation when it is converted to DHT (Dorfman and Dorfman 1962; Anderson and Fulton 1973) (Fig. 19.1a). It still remains to be seen why DHT inhibits hair growth in the parietal region of the scalp while, on the other hand, it promotes the growth of beard and chest and axillary hair. Adachi and Kano (1970) found that DHT inhibited adenyl cyclase activity and that cell proliferation was inhibited as a result. The apparently contradictory phenomenon of 5α-DHT both promoting the growth of beard and chest and axillary hair and, at the same time, inhibiting the growth of hair in the parietal region has yet to be completely elucidated, and it has generally been accepted that hormonal imbalance plays a vital part in this phenomenon. We (Inaba et al. 1988b) conducted studies on the differences in male and female hormone receptor distribution in various regions of the scalp and at various ages but we found no differences in distribution (Section 15.4). According to Schweikert and Wilson (1974a), more 5α-reductase is present in the hair follicles in bald regions than in those in other regions. Although there have been several such reports, the causative mechanism of male pattern baldness has not yet been clearly elucidated.

19.2 Development of the Sebaceous Gland Hypothesis

The authors developed a radical surgical technique they called the subcutaneous tissue shaving method for the treatment of bromidrosis and hyperhidrosis (Inaba 1976; Inaba et al. 1978a,b). This treatment method has been explained in some detail in Chapter 8, but it is important to note that it is a method by which the sweat glands and hair roots are removed beneath the axillary skin. After the subcutaneous tissue is removed by a shaving technique, the surface skin is then grafted and preserved in place. It was found that when the sebaceous gland appended to the hair follicle was left intact, the hair would grow again, but if the sebaceous gland was removed, the hair would not regrow.

The central portion of the hair follicle was thought to lie at the lower portion of the telogen stage follicle. Clinical and histologic observation led to experimentation and the new hypothesis (sebaceous gland hypothesis) that the central generative site of the hair follicle also lies close to the duct opening of the sebaceous gland, especially in the sebaceous isthmus, in the upper isthmal portion of the hair follicle.

The new young hair is already formed at the lower portion of the germinal layer prior to the formation of a new hair bulb.

19.3 Mechanism of the Development of Androgenetic Alopecia

Can this sebaceous gland hypothesis shed any light on the onset of baldness? Generally speaking, baldness seems contingent on male hormone activity, but to simply say “the cause of baldness is hormone imbalance” falls short of the mark. In brief, the common explanation for the effect of male hormone activity is as follows. As shown in Fig. 19.1, the male hormone testosterone is produced in the testes, and it is then circulated to the dermal papilla in the hair bulb. Testosterone affects the hair matrix cells and interacts with the enzyme 5α-reductase in the cells. Testosterone is thereby converted into an even more powerful hormone, 5α-dihydrotestosterone (5α-DHT).

This particular hormone is said to accelerate cell division activity by anabolism and to encourage the formation of hair. In men, the phenomenon of body hair (such as the beard) appearing during puberty is a good example of the effect.

According to the hormone imbalance supposition, the parietal region of the scalp is controlled by the female hormone, which normally prevents women from experiencing baldness, whereas excessive production of male hormone, when conveyed to the hair follicle, inhibits the generation of new hairs by producing a hormone imbalance.

The testosterone is conveyed both to the hair bulb and to the sebaceous gland. Any explanation of baldness which excludes this fact can be very misleading.

Within the sebaceous gland, 5α-reductase converts testosterone to 5α-DHT. The blood vessels that surround the hair follicle (see Section 5.3) convey this 5α-DHT to the hair matrix, where, in excessive amounts, it acts to inhibit matrix activity by inhibiting the activity of adenyl cyclase.

19.4 Hair Follicles and Sebaceous Glands Relative to 5α-DHT Production

Again, the larger the sebaceous gland becomes, the smaller the hair follicle (Montagna and Parakkal 1974). An abnormally enlarged sebaceous gland produces excess 5α-DHT and this inhibits the hair matrix. The result is that mature terminal hairs become downy (vellus) hairs. The authors believe that this is the principal causative mechanism of male pattern baldness (Inaba 1982a, 1985).

In the healthy hair follicle, the appended sebaceous gland is not large, so that the volume of 5α-reductase within it is normal. The action of this enzyme on testosterone does not cause the formation of large amounts of 5α-DHT. Rather, the formation of 5α-DHT in proper quantities enhances hair growth (Fig. 19.2a).

If, however, the sebaceous gland is enlarged for some reason, such as genetic inheritance, dietary habits, hormone influence, etc., the quantity of 5α-reductase will also increase and more 5α-DHT will be produced. Just as excessive use of fertilizer will stunt rather than enhance the growth of plants, too much 5α-DHT can act to inhibit hair growth. The authors have found that the effect of excessive 5α-DHT on the hair matrix is an inhibition of cell division and gradual atrophy of the hair follicle (Fig. 19.2b). This triggers the onset of baldness. We have said that an atrophied hair follicle is the cause of baldness, but this is not quite the same as loss of the hair root. In fact, the root remains but the hair grows thin and short, as a vellus hair (Fig. 19.2c).

Likewise, baldness does not imply that the sebaceous gland disappears. The hair can regenerate as long as the sebaceous gland exists, as we have already noted in touching on our treatment procedure for the relief of bromidrosis. Our own