FIGURE 394-1 Hirsutism scoring scale of Ferriman and Gallwey. The nine body areas that have androgen-sensitive areas are graded from 0 (no terminal hair) to 4 (frankly virile) to obtain a total score. A normal hirsutism score is <8. *(Modified with permission from LJ DeGroot, JL Jameson: Endocrinology, 5th ed. Philadelphia, PA: Saunders; 2006.)*

■ HORMONAL EVALUATION

Androgens are secreted by the ovaries and adrenal glands in response to their respective tropic hormones: luteinizing hormone (LH) and adrenocorticotropic hormone (ACTH). Testosterone is the principal circulating steroid involved in the etiology of hirsutism; other steroids that may contribute to the development of hirsutism include androstenedione and dehydroepiandrosterone (DHEA) and its sulfated form (DHEAS). The ovaries and adrenal glands normally contribute about equally to testosterone production. Approximately half of the total testosterone originates from direct glandular secretion, and the remainder is derived from the peripheral conversion of androstenedione and DHEA **(Chap. 381)**.

Testosterone is the most important circulating androgen, but it is a precursor hormone in mediating hirsutism. Testosterone is converted to dihydrotestosterone (DHT) by the enzyme 5α-reductase, which is located in the PSU. DHT is more potent than testosterone as it has a higher affinity for, and slower dissociation from, the androgen receptor. The local production of DHT allows it to serve as the primary mediator of androgen action at the level of the PSU. There are two isoenzymes of 5α-reductase: type 2 is found in the prostate gland and in hair follicles, and type 1 is found primarily in sebaceous glands.

One approach to the evaluation and treatment of hirsutism is depicted in **Fig. 394-2**. In addition to measuring blood levels of testosterone and DHEAS, it is often important to measure the level