size and cost. The tunable dye laser with its adjustable wavelength range may soon move from the optical bench to the clinical setting where the operator will be able to select the optimal laser beam for the target tissue. Likewise, new units combining several types of lasers in a single handpiece are arriving from Japan.

Thus, it seems unlikely that the laser will be consigned to the role of an esoteric plaything. More likely, it will become a valuable and unique dermatologic therapeutic tool.

PHILIP L. BAILIN, M.D.

Chairman, Department of Dermatology
The Cleveland Clinic Foundation

References

1. Bailin PL, Ratz JL, Levine HL. Removal of tattoos by CO_2 laser. J Dermatol Surg Oncol 1980; **6**:997–1001.
2. Bailin PL, Ratz JL, Lutz-Nagey L. CO_2 laser modification of Mohs' surgery. J Dermatol Surg Oncol 1981; **7**:621–623.
3. Levine HL, Bailin PL. Carbon dioxide laser treatment of cutaneous hemangiomas and tattoos. Arch Otolaryngol 1982; **108**:236–238.
4. Bailin PL. Treatment of port wine stains with the CO_2 laser; early results. [In] Arndt K, ed. Lasers in Dermatology. New York: John Wiley & Sons (in press).
5. Ratz JL, Bailin PL, Levine HL. CO_2 laser treatment of port-wine stains; a preliminary report. J Dermatol Surg Oncol 1982; **8**:1039–1044.

Androgens and the Pilosebaceous Follicle in Women: Technology Catches Up with the Clinician

In this issue of the *Quarterly*, Kasick et al (page 111) report the promising results of their investigation of 19 white women with female androgenic alopecia. Their data confirm what clinicians have long assumed. The mechanistic link between androgens and female androgenic alopecia, "idiopathic hirsutism," and acne vulgaris seems obvious; however, the link has been difficult to prove. Pioneering clinical studies in the early 1940s established the effect of testosterone on the pilosebaceous follicle and the androgen sensitivity of acne vulgaris.[1] Progress in defining the role of androgens lagged during the next three decades because the only readily available test for studying circulating androgens in women was the relatively imprecise assessment of metabolites of androgens in the urine by means of the urinary 17-kestosteroid test (17 KS). A technologic breakthrough occurred in the 1970s when radioimmunoassays permitted accurate direct measurements of circulating androgens.

Experience in the last decade with the direct measurement of serum steroids has delineated the ovarian and adrenal pathways of androgenic steroids. Catheterization studies of adrenal and ovarian veins show that approximately 25% of circulating testosterone is derived directly from the adrenal gland and 25% from the ovaries. Approximately 50% of circulating testosterone arises from peripheral conversion in the skin, liver, and fat of the prehormones, androstenediol (A) and dehydroepiandosterone (DHEA) and its sulfate (DHEAS). The adrenal cortex contributes 80% of the circulating DHEA and 90% of the DHEAS, making DHEAS a useful marker for adrenal androgen secretion.

The major portion of circulating sex hormones is bound to testosterone-estradiol binding globulin (TeBG). Extensive data suggest that bound steroids are inactive, and that only unbound free testosterone (free T) is biologically active. Levels of free T can be greatly increased while circulating total testosterone (total T) levels are normal or only mildly increased.

In hirsute women, free T levels are increased; however, more than 50% of the patients studied have normal total T levels.[3] The ovaries are the predominant source of androgen in these women.[4] No universal correlation between acne in women and elevated free T, total T, or DHEAS has been demonstrated.[5] It has been postulated that increased enzyme activity within sebaceous cells may amplify the effects of normal levels of circulating androgens, but the androgen connection in acne remains to be solved.

Studies have failed to show a universal correlation between free T and total T in women with androgenic alopecia. Kasick's study, demonstrating increased DHEAS levels in his patients, gives credence to the pivotal role of the adrenal glands in androgenic alopecia. Their data show increased serum prolactin levels in 2 patients. In this small subset of patients a pituitary adenoma appears to be the cause of the adrenal hyperfunction. The basic adrenal mechanism in the majority of their patients awaits further study.

The appropriate laboratory investigation of a premenopausal woman with androgenic alopecia should include determinations of serum DHEAS, serum prolactin, and serum total and free testosterone. The tests are readily available and sensitive. The results provide reliable information

regarding the adrenal, pituitary, or ovarian source of increased androgen production. We await more detailed information on Kasick's promising therapeutic studies with spirinolactone and other antiandrogens.

FAYE D. ARUNDELL, M.D.

Clinical Associate Professor of Dermatology
Stanford University Medical School

References

1. Hamilton JB. Male hormone substance. A prime factor in acne. J Clin Endocrinol Metabol 1941; **1**:570–592.
2. Lobo RA, Paul WL, Goebelsmann U. Dehydroepiandrosterone-sulfate as an indicator of adrenal androgen function. Obstet Gynecol 1981; **57**:69–73.
3. Paulson JD, Keller DW, Wiest WG, Warren JC. Free testosterone concentration in serum: Elevation is the hallmark of hirsutism. Am J Obstet Gynecol 1977; **128**:851–857.
4. Kirschner MA, Zucker IR, Jepperson D. Idiopathic hirsutism—an ovarian abnormality. N Engl J Med 1976; **294**:637–640.
5. Pochi PE, Strauss JS. Endocrinologic control of the development and activity of the human sebaceous gland. Invest Dermatol 1974; **62**:191–201.

The Value of Persistence and Thoroughness in Searching for the Allergen in Contact Dermatitis

The description of 3 cases of dermatitis in this issue by Taylor et al (page 123), "Contact Dermatitis to Knee Patch Adhesive in Certain Types of Boys' Jeans," emphasizes the value of thoroughness, insight, and persistence in pursuing the identity of an allergen. On a superficial level the authors identified epoxy resin as the cause of chronic dermatitis on both knees of three boys. On closer examination, there are important lessons to be learned regarding moral responsibilities and vigilance.

A rare or common allergen? Three patients were identified as having epoxy clothing dermatitis in a limited geographic area. It is highly improbable that these were the only children who developed such an allergy from these jeans. The investigative work of the authors identified the etiology in spite of the difficulty in obtaining information. Most physicians confronted with this dermatitis presumably muddled on, allowing children to continue with chronic dermatitis until the jeans were discarded or until repeated washing decreased the allergen level. Often, identification of an allergen in a few cases proves the problem instead to have been widespread. The ethylenediamine in nystatin-neomycin sulfate-gramicidin-triamcinolone acetonide (Mycolog) cream and its subsequent demonstration as a common allergen is possibly analogous to this epoxy episode.[1]

Exogenous versus endogenous dermatitis. When examining patients with dermatitis, we define the cause by appropriate history, morphology, distribution, and, when indicated, by patch testing. Many of us undoubtedly saw similar patients, and assumed that the process was endogenous in origin. The diagnosis of atopic dermatitis is all too often applied to children with eczema of unknown origin. However, these authors looked further, identifying the allergen by appropriate patch testing, thus immediately stopping a chronic dermatitis. The value to the public surely is greater. When the allergen was identified, the manufacturers altered the product.

An adequate corporate scientific staff or an inefficient system? It would be easy in retrospect to fault the clothing manufacturer for this dermatitis. Unfortunately, this is not an unusual event. Manufacturers and scientists often learn after the fact to be more careful and thorough in their dermatotoxicologic approach to product safety. In this case the manufacturer first claimed ignorance on the basis of inadequate information from his supplier. Proprietary data are often used as an excuse. Until more adequate legislation mandates away such secrecy, manufacturers must be demanding and persistent in obtaining the chemical composition from suppliers.

Because few manufacturers can justify the services of a full-time dermatotoxicologist, even if adequate numbers were trained, they must learn to seek part-time consultation in making risk assessments. An ever-expanding library of reference works simplifies the task significantly.[2,3]

Credibility of the manufacturer. When the authors first contacted the manufacturer, the presence of epoxy was denied. This all too often is the case. A classic example is that of Wilkinson,[4] who, with the aid of a black light, identified a fluorescent compound, a biocide in soap, as the cause of an epidemic of photoallergy in England.

We in no way wish to attack the credibility of manufacturers. We believe that it is the responsibility of manufacturers to know what they are selling. It is only recently that some corporations have insisted on knowing the compositions of fragrances they utilize. There is much room for improvement in defining this aspect of the chemical environment.

Allergen alternatives. Often manufacturers claim that they must use an allergen because no alternatives exist. Fortunately, with ingenuity and