plex infection, Behcet's disease, viral stomatitis, and erythema multiforme. There is no diagnostic clinical test.

Aphthae begin with burning pain, followed by ulceration, edema, and inflammation. One to thirty lesions may occur. Minor aphthae are small, few, and heal within 4 to 14 days. Major aphthae are larger than 10 mm, numerous, very painful and slow to heal. They may be accompanied by severe malaise and fatigue and may cause mucosal scarring. Behoet's disease has additional symptoms of arthritis, conjunctivitis, uveitis, and genital ulcerations. In both diseases, ulcerations histologically show criteria for delayed hypersensitivity, and in aphthae the presence of Streptococcus sanguis is postulated to cause hypersensitivity. Trauma, foods, chemical irritants, stress and decreased premenstrual estrogen production are other possible factors.

Treatment with corticosteroids seems most effective. Topical triamcinolone in emollient dental paste (Kenalog® in Orabase®), intralesional triamcinolone, and systemic steroids may all be helpful. Topical tetracycline, either as oral suspension or 250 mg dissolved in 30 cc of water may also help. Cyclophosphamide (Cytoxan®) stopped severe destructive lesions in one case. Vitamins A, B, and C are generally not helpful, and smallpox vaccination is not recommended.

ROBERT J. ROTH, D.D.S., M.D.

REFERENCES

Lehner T: Immunologic aspects of recurrent oral ulcers. Oral Surg 33:80-85, Jan 1972

Graykowski EA, Barile MJ, Lee WB, et al: The clinical, therapeutic and histopathologic aspects of aphthous stomatitis. JAMA 195: 637-644, Feb 21, 1966

Lehner T: Pathology of recurrent oral ulcerations in behcet's syndrome: Light, electron and fluorescence microscopy. J Pathol 97:451,

Stanley HR, Graykowski EA, Barile MJ: The occurrence of microorganisms in microscopic sections of aphthous and nonaphthous lesions and other oral tissues. Oral Surg 18:335-341, Sep 1964

Cooke BE: Recurrent oral ulceration. Br J Dermatol 81:159-161, Feb 1969

Darsey C: More observations on relief of aphthous stomatitis on resumption of cigarette smoking—A report of three cases. Calif Med 101:377-378, Nov 1964

Schlappnez OLA, Shelley WB: Telangiectasis, aphthous stomatitis and hypersplenism. Arch Dermatol 104:668-670, 1971

Topical Vitamin A Acid

Topical administration of 0.05 percent to 3.0 percent Vitamin A acid (retinoic acid) in ethanol, propylene glycol, cream or ointment vehicles

can cause clinical or microscopic desquamation of the horny layer and favorably alter several hyperkeratotic and ichthyotic dermatoses. Following treatment with Vitamin A acid, clinical improvement has occurred in many, but not all cases of lamellar ichthyosis (nonbullous congenital ichthyosiform erythroderma), psoriasis, keratoderma of the palms and soles, flat warts and Darier's disease (keratosis follicularis). Consistent therapeutic benefits have been seen in the treatment of comedones of acne vulgaris, chloracne, and aging skin. The follicular hyperkeratosis associated with keratosis pilaris is also helped.

Topical Vitamin A acid is currently available as Retin-A® (Tretinoin), a solution of 0.05 percent retinoic acid in alcohol and propylene glycol, which is approved for use in treatment of acne vulgaris.

DAVID R. HARRIS, M.D.

REFERENCES

Hesbacher E: Zosteriform keratosis follicularis treated topically with Tretinoin. Arch Dermatol 102:209-212, 1970

Kligman AM, Fulton JE, Plewig G: Topical vitamin A acid in acne vulgaris. Arch Dermatol 99:469-476, 1969

Mirrer E, McGuire J: Lamellar ichthyosis—Response to retinoic acid (Tretinoin). Arch Dermatol 102:548-551, 1970

Frost P, Weinstein GD: Topical administration of vitamin A acid for ichthyosiform dermatoses and psoriasis. JAMA 207:1863-1868, 1969

Pedace FJ, Stoughton R: Topical retinoic acid in acne vulgaris. Br J Dermatol 84:465-469, 1971

Photodynamic Inactivation of Herpes Simplex Infections

TREATMENT OF RECURRENT Herpes simplex infections is generally frustrating and disappointing with symptomatic measures designed to dry up lesions and relieve pain. Treatment with repeated smallpox vaccinations and Herpes simplex vaccines have given equivocal results.

A promising new treatment involves application of 0.1 percent neutral red or .01 percent proflavine dye to the abraded base of acute vesicular lesions, followed by 15 minute irradiation with an ordinary fluorescent lamp placed four to six inches from the lesions. This treatment is repeated once within four hours. Preliminary reports indicate a marked reduction in both intensity and duration of discomfort, and an apparent decrease in frequency of recurrent attacks. Best results have occurred with Herpes progenitalis. The mechanism of action probably involves combination of dye with guanine bases of viral DNA, followed by breakage of single guanine strands produced by irradiation.

The relative simplicity and efficiency of this technique make it a welcome addition to the treatment armamentarium for recurrent Herpes simplex infections.

ROBERT M. MELNIKOFF, M.D.

REFERENCE

Felber T: Report to the AMA Section of Dermatology, June 21, 1971 (To be published).

Psoralens and Cutaneous Photosensitization

NATURAL PSORALENS, FROM SEEDS and fruit, have been used since 1400 B.C. for the repigmentation of vitiligo. Two psoralens, 8 methoxypsoralen (methoxsalen) and 4, 5, 8-trimethyl psoralen

(trioxsalen), are used clinically to treat vitiligo and to increase cutaneous tolerance to solar radiation.

Psoralens stimulate pigmentation by inducing photosensitization in the presence of long-wave ultraviolet light (320-400 nm). Photoaddition of psoralen with epidermal DNA appears to be responsible for the photosensitization. A heightened but delayed sunburn reaction occurs 20 hours after exposure to appropriate wavelengths of ultraviolet light. Pigmentation subsequent to psoralen photosensitization involves an increased production of the suntan pigment, melanin, and an increase in the number of melanin-producing melanocytes in the skin.

There are no reported adverse systemic reactions. Care must be taken to avoid overexposure and severe sunburn.

FAYE D. ARUNDEL, M.D., F.R.C.P. (C)

REFERENCES

Psoralens and Radiant Energy; Proceedings of a Symposium published as a supplement to the J Invest Dermatol 32:132-391, 1959 El Mofty AM: In Vitiligo & Psoralens. Oxford, Pergamon Press, 1968

Pathak MD: Mechanism of psoralen photosensitization and in vivo biological action spectrum of 8-methoxypsoralen. J Invest Dermatol 37:397-407, 1961

Musajo L, Rodighiero G: Studies on the photo-C4-cyclo-addition reactions between skin-photosensitizing furocommarins and nucleic acids. Photochem Photobiol 11:27-35, 1970

Toda K: Nongenetic factors affecting melanosome complexes, J Invest Dermatol 56:255, 1971

ADVISORY PANEL TO THE SECTION ON DERMATOLOGY

ROBERT I. FREEDMAN, M.D., Chairman Downey

MEYER BERKE, M.D. Metropolitan Dermatological Society Lakewood

Molleurus Couperus, m.d. Loma Linda University

W. L. EPSTEIN, M.D. University of California, San Francisco

EUGENE M. FARBER, M.D. Stanford University

WILLIAM M. GOULD, M.D. CMA Section on Dermatology, Secretary Palo Alto

JAMES GRAHAM, M.D. University of California, Irvine California College of Medicine

EDWIN KOMISARUK, M.D. Sacramento Dermatology Society Sacramento

NORMAN E. LEVAN, M.D. University of Southern California Los Angeles

HOWARD I. MAIBACH, M.D. San Francisco Dermatological Society CMA Scientific Board San Francisco

T. RICHARD MIHAN, M.D. CMA Section on Dermatology, Assistant Secretary Los Angeles

CORTLAND MYERS, M.D. Orange County Dermatology Society Santa Ana

WALTER NICKEL, M.D. University of California, San Diego San Diego Dermatology Society

RONALD REISNER, M.D. Los Angeles Dermatological Society Torrance

DONALD SEIDMAN, M.D. San Fernando Valley Dermatology Society, President Reseda

THOMAS STERNBERG, M.D. University of California, Los Angeles