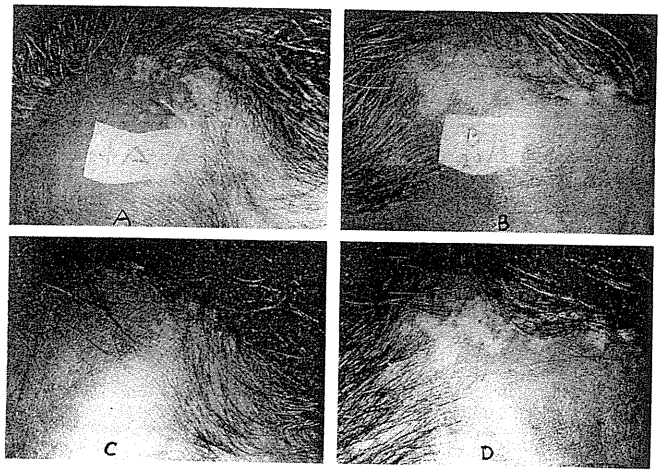

FIG. 2. **A** and **B.** Vitiligo of hair line before treatment, **C** following PUVA and topical minoxidil, **D** following PUVA and placebo.

persistent erythema. A placebo was applied over one side and 2% minoxidil solution over the other side. Repigmentation was relatively more marked over the study site as compared to the placebo site. The woman developed cosmetically acceptable hyperleukotrichosis following topical minoxidil. After 3 months both sides were treated with topical minoxidil and PUVASOL.

Apart from stimulating hair follicles by some unknown mechanism, minoxidil reopens closed luminal vessels and decreases tissue lymphocytes.[4] These factors also may aid the repigmentation in vitiligo. Studies are in progress to assess the therapeutic efficacy of minoxidil with and without PUVA or PUVASOL. At present we are unable to comment on the course of hypertrichosis and its eventual effect on repigmentation once the hair falls on withdrawal of topical minoxidil.

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References

1. Mosher DS, Fitzpatrick TB, Ortonne J-P, et al. Disorder of pigmentation. In: Fitzpatrick TB, Eisen AZ, Wolff K et al, eds. Dermatology in general medicine. 3rd ed. New York: McGraw Hill, 1987:818.
2. Honigmann H, Wolff K, Fitzpatrick TB. Oral photochemon therapy with psoralens and UVA (PUVA): principles and practice. In: Fitzpatrick TB, Eisen HZ, Wolff K, et al, eds. Dermatology in General Medicine. 3rd ed. New York: McGraw Hill, 1987:1549.
3. Rook A, Dawber R. Colour of hair. In: Diseases of hair and scalp. Oxford: Blackwell Scientific, 1982;342–366.
4. Feidler-Weiss VC. Potential mechanisms of minoxidil induced hair growth in alopecia areata. J Am Acad Dermatol. 1987;16:653–656.

Clinical Trial of Prostaglandin E_2 on the Oral Lesions of Pemphigus Vulgaris

To the Editor:

Prostaglandin (PG) was first discovered as a substance inducing contraction of smooth muscle. It has, however, proved to have a meaningful biologic significance through the determination of chemical construction and research for physiologic activity during the past two decades. From 1979 when Robert[1] revealed an antiulcerative effect of PG, it has been widely studied, inducing those in immunological field. It was reported that PGE_2 could inhibit induction activity of Ia antigen through lymphokines[2] and that it might inhibit interleukin 2 production.[3] Furthermore, PGE_2 has been shown to give an induction of suppressor T cells.[4] For these reasons, we used PGE_2 tablets on the oral lesions of pemphigus vulgaris.

A 73-year-old woman had been treated by local doctors from April 1988, because of a painful lesion in her mouth. The pain was present each time she took a meal. Upon the manifestation of bulla formation on her extremities, she visited us on August 9th, 1988. The patient had not had periods of remission before our therapy. There were erosive lesions on the mucous membrane at the hard palate and buccal areas. In addition, several finger tip-sized bulla formations were recognized on the extremities. A skin biopsy was conducted on the erosive lesion of her right thigh. The specimen was not only subjected to H&E stain but also immunofluorescent examination.

The histopathologic findings were as follows: the bulla was in a predominantly suprabasal position. The floor of a bulla showed the basal adherent to the dermis. The cavity contained acantholytic epidermal cells.

There was fluorescence of IgG and C_3 on the intercellular spaces of epidermis.

Based on these findings we diagnosed the patient as having pemphigus vulgaris. We instructed the patient to take a 0.5mg PGE_2 tablet twice a day and let it dissolve in her mouth. It was difficult for her to take a meal because of a painful sensation in oral lesions at the start of PGE_2 therapy. On the 13th day of PGE_2 therapy, painful sensation during the meal was reduced, and the erosive lesions of the oral cavity decreased in degree and size. Both the painful sensation and erosive lesion of the oral cavity almost disappeared on the 4th week of PGE_2 therapy. There was improvement in the skin lesions.

Although the initial trial was encouraging, further work with enhanced numbers of materials and retrieval data on clinical applications of PGE_2 is still needed to fully develop our view in this respect.

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References

1. Robert A. Cytoprotection by prostaglandins. Gastroenterology. 1979;77:761–767.
2. Snyder DS, Lu CY, Unanue ER. Control of macrophage Ia expression in neonatol mice—role of a splenic suppressor cell. J Immunol. 1982;128:1458–1465.

3. Baker PE, Fahey JV, Munck A. Prostaglandin inhibition of T-cell proliferation is mediated at two levels. Cell Immunol. 1981;61:52–61.
4. Ceuppens JL, Goodwin JS. Endogenous prostaglandin E_2 enhances polyclonal immunoglobulin production by tonically inhibiting T suppressor cell activity. Cell Immunol. 1982;70:41–54.

Dermatologic Radiation Therapy: Pyrex versus Beryllium Window Units

To the Editor:

With the current revival of interest among dermatologists in the use of x-ray therapy for select cases of skin cancer, much has been recently written about the merits of this modality.[1-3] Many of the studies are based upon data using the newer beryllium window units, having half-value layers (HVL) of 0.1–2 mm Al with a half-depth dose (D½) of 1 to 20 mm of tissue.[4] A drawback to a beryllium window unit is the actual expense of the unit itself. Most of these machines cost in excess of $50,000, which would be quite intimidating and not cost effective for the young dermatologist or the third-year resident who is purchasing his equipment.

We wish to bring out the point that the older Pyrex window units, which have a HVL of 0.7–3 mm Al, with a D½ of 7–10 mm of tissue, are more than adequate for the treatment of the majority of skin cancers seen in an office setting (H. Goldschmidt, personal communication, 1982). These units can usually be purchased, tuned, set-up, and calibrated for under $1,000. Although these units are no longer manufactured, all parts including tubes and transformers are readily available at modest prices. These units are often available in the classified sections of various dermatologic journals. These older units were constructed well, are durable, and were built to last.

Those dermatologists interested in using superficial x-ray therapy and thereby assurring this modality its appropriate place in the dermatologic armamentarium may do so and at a minimal cost.

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References

1. Polano M. Radiation therapy in dermatologic training centers. J Am Acad Dermatol. 1987;16:1267–1268.
2. Goldschmidt H. Dermatologic radiotherapy: risk benefit ratio. Arch Dermatol. 1986;122:1385–1388.
3. Kingery F. Radiation therapy in dermatologic training centers. J Am Acad Dermatol. 1987;14:1108–1110.
4. Goldschmidt H. Physical modalities in dermatologic therapy. New York: Springer-Verlag, 1978:72–73.

Trophic Leprosy Ulcers: Treatment with Topical and Systemic Phenytoin

To the Editor:

Over the years the treatment of the trophic ulcers associated with leprosy has proved to be difficult. Frequently occurring in poorly vascularized areas such as the lower leg, the malleolus, the dorsal and plantar surfaces of the foot, and the tibial crest, these ulcers are difficult to treat because of a combination of factors including loss of innervation, frequent mechanical trauma, poor circulation, and chronic infection. The deep, penetrating plantar ulcer is the most serious and difficult of all, in large part because of its location and the trauma of weight bearing.

Given the need for more effective treatment for these lesions, and because of the reports that phenytoin (PHT) promotes wound healing,[1-6] we have treated a group of leprosy patients afflicted with trophic ulcers of the leg and plantar area during the past 18 months with topical and systemic PHT with good results.

Materials and Methods

Twenty-seven patients (age, 47–77) with 40 ulcers of 12-month to 20-year duration that had proved resistant to all standard therapies (including rest) were chosen for this study. Seventeen of the ulcers were plantar, and 23 were located on the leg and malleolus. Five of the ulcers were between 2 and 4 cm in diameter, 20 ulcers were 5–10 cm, and 15 were 11–30 cm. Twenty patients had the lepromatous type of leprosy; 4, tuberculoid; 1, dimorphic; and 2, indeterminate. All the patients were being treated with multiple-drug antileprosy therapy. All presented negative smears on at least two occasions, and all were without active lesions.

Twenty patients with 33 ulcers were treated with topical PHT and seven patients with seven ulcers were treated with both topical and systemic PHT, 100 mg daily. Treatment duration was a minimum of 3 weeks and a maximum of 6 weeks, depending on the progress of the healing. Seven patients were hospitalized and 20 received treatment as ambulatory outpatients.

Before starting PHT treatment, the ulcers were meticulously debrided of necrotic tissue. Thereafter, they were washed with soap and antiseptic and the PHT powder (as a thin dusting) and a dry-gauze dressing were applied. The hospitalized patients had their dressings changed by nurses. The ambulatory patients were given simple instructions for wound care at home. The doctor examined the patients' ulcers weekly. Serial photographs of the lesions were taken before the initiation of treatment and every 2 weeks until 6 weeks of observation were completed.

Results

At 1 week, all ulcers showed diminished exudate and abundant new granulation tissue. By 3–6 weeks, 20 patients had major improvement—complete healing of small lesions (2–4 cm diameter) to markedly enhanced granulation tissue formation in larger lesions (>4 cm). Seven patients had moderate improvement. None worsened. Consistent with a healthy granulation base, there was no clinical evidence of infection. Although healing rates did not differ, patients receiving oral plus topical PHT were calmer and more cooperative.

The only adverse reaction with topical PHT treatment was a burning sensation in some patients when the powder was first applied.

Comments

Our data indicate that phenytoin is a valuable agent in the treatment of trophic ulcers associated with leprosy. Its effec-