Further Section

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Letters to the Editor

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The response to vitamin E of dystrophic epidermolysis bullosa as reported by Sehgal et al. in *Dermatologica* 144: 27–34 (1972), confirms previous reports by Price and by Wilson, and should stimulate further use of this therapeutic modality in this otherwise hopeless condition. We also have reported the almost complete control of epidermolysis bullosa simplex in two children in whom the condition was severe enough to interfere with the customary activities of childhood, such as running, athletic games, etc.

We were surprised at the relatively small doses of vitamin E which the authors found to be effective in this serious condition, namely 300 mg daily, roughly equivalent to 300 IU.

Furthermore, they used the 'dI' or synthetic type of tocopherol rather than the 'd' or natural form, which most authorities consider to be more potent. In one of our cases of the simplex type of epidermolysis bullosa, the condition was adequately controlled by a dose of 200 IU daily of δ - α -tocopherylacetate, whereas the other patient required 800 IU daily. In the severe case reported by Wilson, a daily dose of 4,800 IU was required for control.

If the disease is due to a genetic defect in the body's ability to utilize vitamin E for its various protective functions, the therapeutic dose may be many times the so-called average daily requirement. It is important that no iron is administered while taking vitamin E, since iron combines with and inactivates vitamin E. An individual with severe hypertension, a serious heart condition, or a diabetic on insulin should be started on a low dose of approximately 100 IU daily, which can be gradually increased. Under ordinary conditions, a daily dose of 400–1,600 IU, or even 2,000 IU or more, according to age, severity, etc., may be necessary to bring the condition under control. A smaller maintenance dose will probably be required indefinitely.

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Dear Sirs,

In reference to the interesting article 'Some Electron-Microscopic Aspects of Pemphigus erythematosus compared to those of Pemphigus vulgaris' by Drs. van de Staak and Bergers published in *Dermatologica* 144: 294, 1972, we would like to stress that we do not deny a connection between pemphigus erythematosus and pemphigus vulgaris, but had only brought to light some immunological peculiarities of pemphigus erythematosus [Chorzelski et al., 1968]. These peculiarities are important because of the possible coexistence of pemphigus erythematosus with other immunological disorders [Beutner et al., 1968, 1970], and because of a better response of this form of pemphigus to the immuno-suppressants [Jablonska et al., 1970].

In the patient described in our paper, 'Koexistenz von Pemphigus erythematosus und Myasthenia gravis', mentioned by Drs. van de Staak and Bergers, there were some periods when the skin lesions of the pemphigus erythematosus type resembled in the clinical and histological picture more those of pemphigus vulgaris (deeper acantholytic bullae). Thus, we fully agree that both pemphigus vulgaris and pemphigus erythematosus are merely variants of the same basic disease.

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Concerning the transition of pemphigus erythematosus into pemphigus vulgaris and vice versa we are now of the same opinion as the authors that it may occur, and this has been emphasized in our recent paper: 'Im Lichte der immunpathologischen Untersuchungen werden die Übergänge, die man klinisch zwischen den Abarten des Pemphigus beobachtet, bestätigt, insbesondere Übergänge zwischen dem Pemphigus vulgaris und erythematosus, etc' [Chorzelski-Jablonska, *Hautarzt* 23: 477, 1972].

We think that Drs. van de Staak and Beroers are right in bringing out this controversial problem.

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