time when there was virtually no specific treatment. Folk remedies have of course existed from time immemorial. The natives of Tonga used to apply an extract of the root of the breadfruit tree (*Artocarpus communis*), but hydnocarpus oil (chaulmoogra oil), derived from the plant illustrated on the 1938 Congress stamp, was the only official remedy until the era of modern chemotherapy. This, stemming from the brilliant pioneer work of Nobel prize-winner Paul Ehrlich, resulted from the development of the synthetic dyestuffs industry in Germany and the discovery of the antibacterial properties of the sulphonamides in 1935.

Many tropical countries today are trying to emulate the thoroughness and efficiency with which the Norwegian authorities fought this disease 100 years ago. Funds are contributed by international organizations such as Lions International, which are the modern counterparts of the old religious Orders, and charity stamps, bearing the Cross of the Sovereign Military Order of Malta, are issued on World Leprosy Day every few years. In Africa and elsewhere missionary doctors are still dedicating their lives to leprosy and other tropical diseases and there is a modern organization for research, treatment and especially the rehabilitation of lepers (Fig 2). With a better understanding of the disease the old policy of rigid segregation is being displaced by BCG vaccination of infants at risk, based on the cross immunity between tuberculosis and leprosy; mobile clinics; domiciliary treatment with modern drugs; and plastic surgery of deformed limbs. In some small places the disease is now well controlled by these methods and only three years ago Fiji issued a stamp to celebrate the closure of the old leper hospital there; the only stamp, incidentally, directly connected with leprosy, to be issued by any British administration up to the present time. Nevertheless, in many underdeveloped countries today the disease remains an increasing challenge as more hitherto undetected cases are brought to light, and the final chapters of this remarkable story have yet to be written.

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Cases

Pustular Psoriasis with Adrenal Suppression Following Topical Corticosteroids

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(for P D Samman MD FRCP)
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G C, woman aged 61. Housewife

History: Psoriasis since the age of 3. Previous treatment included: dental clearance, intravenous protein shock therapy (TAB vaccine ×5) and, in 1963, a six-week course of systemic steroids and a right nephrectomy for staghorn calculus and pyonephrosis. For the last nine years the condition has been well controlled with topical fluorinated corticosteroids usually prescribed diluted, sometimes aided by polythene occlusion. The psoriasis flared up in January 1973. It did not respond to dilute betamethasone -17- valerate (Betnovate). In July dithranol was prescribed after suitable test doses. Despite this a severe reaction occurred to the 0.1% preparation. This was treated with clobetasol propionate (Dermovate); four weeks later the skin had virtually cleared. Superficial pustules appeared one week after this, followed by successive waves of pustules spreading outwards from the flexures and associated with erythroderma, intermittent fever, rigors and gross local œdema.

On examination: Widespread erythroderma; superficial pustules around the axillæ, groins and neck; gross œdema of legs and face; severe nail dystrophy; diffuse alopecia; normal blood pressure; no buccal pigmentation.

Investigations: Skin histopathology (during reaction to dithranol) – subacute dermatitis. Synacthen stimulation test: plasma cortisol – before 5 μg/100 ml, after 6 μg/100 ml. WBC (during pustular phase) 17 600/mm^3 (90% polys.). Uric acid 7.6–13.1 mg/100 ml; creatinine clearance 54 ml/min. MSU: culture showed proteus and pseudomonas. The following relevant tests were negative or normal: serum calcium, phosphorus, electrolytes, urea, protein electrophoresis, liver function tests; antistreptolysin-0; Wassermann reaction; autoantibodies to thyroid, gastric parietal cell, antinuclear factor; urine protein, sugar, calcium; blood cultures; swabs from pustules and throat; ureteric specimen for acid-fast bacillus culture; Schilling; thyroid function.

X-rays of abdomen showed no adrenal or pancreatic calcification.

Comment

Generalized pustular psoriasis may be provoked by pregnancy, infection, injudicious topical therapy, hypocalcæmia, sunlight and by reduction or withdrawal of systemic steroids (Baker & Ryan 1968). There was no evidence of hypocalcæmia and, although the patient did have a severe reaction to dithranol, the skin had nearly returned to normal before the onset of pustular psoriasis. Chronic pyelonephritis could have been a factor, but probably of greater importance was the use of clobetasol. Approximately 40 g a day of this preparation had been applied to the skin for most of the five weeks preceding the onset of pustular psoriasis. Even more was used afterwards for periodic exacerbations of pustulation and erythroderma. The Synacthen stimulation test was carried out after she had been applying quarter-strength clobetasol for two weeks, having previously used the full strength preparation for eight weeks. The test showed gross adrenal suppression (Wood *et al.* 1965). As there was no evidence of Addison's disease or of any of the conditions sometimes associated with it, significant absorption of the steroid from the skin is likely to have occurred.

Pustular psoriasis following use of topical steroids under polythene occlusion is recognized. It would seem that clobetasol without occlusion, but used in large quantities for many weeks in a patient with extensively involved inflamed skin, is liable to produce the same complication. One wonders how much of this potent topical preparation, when used regularly in less severe cases, is absorbed, and whether patients on this preparation should be treated as though they were taking systemic steroids with all the precautions this entails. In addition, the danger of precipitating a severe complication of psoriasis by using clobetasol cannot be ignored.

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Dr Harvey Baker: I have under my care a middle-aged man with extensive discoid psoriasis who was recently given 200 g per week of clobetasol cream for about five weeks by his general practitioner. Polythene occlusion was not used. When I saw him he was Cushingoid and hypertensive and has gained 20 lb (9 kg) in weight during this period. Synacthen stimulation test, about a week after the clobetasol was stopped, showed adrenal-pituitary suppression.

The patient was treated with dithranol, and later small quantities of diluted topical steroids, and his psoriasis partly settled without any serious complications.

Dr M Feiwel: Dr Harvey Baker himself has recently suggested (*British Medical Journal* 1973, iv, 605) that the potency of clobetasol is near that of the 'forte' preparations. It is available in 25 g or 100 g tubes. Side-effects may be anticipated and in infants it should be applied very sparingly and in diluted form.

Proliferative Cutaneous Arteritis

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P S, woman aged 51

History: Ulcers across the lower abdomen for four years. For two years before this, the patient had transient erythematous small maculopapular eruptions across the lower abdomen which lasted 7–10 days and disappeared spontaneously. The ulcers developed insidiously, were not painful and possessed red indurated borders. The patient received various ointments and powders from her doctor to no effect.

She has also suffered from: (1) Arthritis for six years diagnosed as rheumatoid arthritis, involving the hands, knees, feet and more recently the temporomandibular joints. She was treated with various analgesics, the last being phenylbutazone. (2) Diarrhœa for one year from July 1972 to summer 1973. Her bowels were open 5–6 times daily. The motions were loose and associated with mucus and lower abdominal pain. They contained no blood. (3) Rigors and fevers during a visit to Nairobi last year, and repeated on 4 occasions since. She assures us she took adequate malarial prophylaxis.

On examination: Across the lower abdomen a band of thickened dusky red skin with superficial ulcers covered with black hæmorrhagic crusts and pus with raised slightly indurated edges (Fig 1). There were 2 similar ulcers on the back. Whilst in

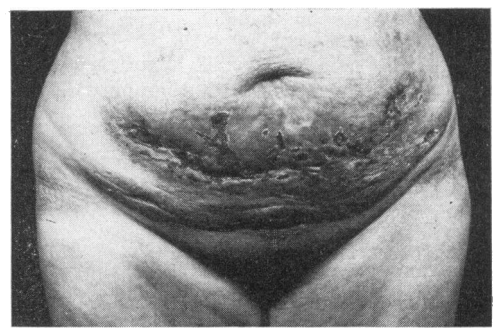

Fig 1 *Ulcers on abdomen, present for four years*