induced vasoconstriction and platelet aggregation. Serotonin has been implicated in the pathophysiology of both Raynaud's disease (Halpern *et al.*, 1960) and systemic sclerosis (Winkelmann, Goldyne & Linscheid, 1976). We have studied, therefore, the effect of ketanserin on peripheral blood flow in these two conditions.

Intravenous ketanserin 10 mg was given to six patients (three with Raynaud's disease; three with systemic sclerosis). Changes in blood flow were measured by monitoring skin oxygen tension using a Hellige Oxymonitor and by measuring thermal clearance using calorimetry. A significant and immediate rise in oxygen tension and heat output occurred in all subjects following intravenous administration of the drug.

We also investigated the effect of 20 mg and 40 mg ketanserin given orally. Following a single oral dose of 40 mg, three of five patients showed an increase in blood flow as demonstrated by calorimetry and oxymetry.

In five patients (four with systemic sclerosis and one with Raynaud's disease) who responded to either the oral or the intravenous drug, ketanserin 40 mg b.d. was used as a therapeutic agent. Two patients with systemic sclerosis showed clinical improvement but long-term therapy was discontinued because of side-effects, i.e., dizziness, depression and postural hypotension.

The presentation will include details of the techniques and the results.

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Discussant: N.R.Rowell

The role of inosine pranobex in the treatment of herpes zoster and postherpetic neuralgia

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The severity of herpes zoster worsens with age and postherpetic neuralgia is almost limited to the elderly (Wildenhoff *et al.*, 1979, 1981). Acyclovir speeds healing and lessens pain, but has no effect on postherpetic neuralgia (Peterslund *et al.*, 1981; Bean, Braun & Balfour, 1982). Inosine pranobex (or isoprinosine, inosiplex: trade name Imunovir), an immuno-modulator with low toxicity, is effective in herpes simplex (*Lancet*, 1985). Uncontrolled observations indicate a beneficial effect in herpes zoster (Sternberg & Ruiz, 1972).

We report the first double-blind placebo-controlled clinical trial of inosine pranobex in the treatment of herpes zoster and postherpetic neuralgia in the elderly.

Forty-two consecutive patients (19 males, 23 females) over 60-years old and in the first 96 h of symptoms were stratified by sex, duration of pain (up to and/or over 48 h) and localization (trigeminal/other) and were randomly allocated 6 days of placebo or of inosine pranobex tablets. Five clinical and five photographic variables were monitored daily for 7–9 days (Peterslund *et*

al., 1981). Pain was assessed monthly for a further 3 months. Investigations at entry, 1 and 4 weeks, comprised haematology, biochemistry, viral culture, immunology, including circulating T-cell subsets, and serology, including ELISA and complement fixation tests.

Results, statistically analysed by multiple regression analysis (Peterslund *et al.*, 1981) and life-table techniques, will be presented.

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Discussant: R.Camp.

Deflazacort—a safer systemic steroid for the treatment of chronic dermatoses

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Systemic steroids are widely and effectively used for the management of many chronic skin disorders. However, prolonged administration of glucocorticoids is associated with unwanted effects, including impairment of glucose tolerance, weight gain and acceleration of bone loss. Clinical and animal studies have shown that deflazacort, an oxazoline derivative of prednisolone, may have less deleterious effects on bone and glucose metabolism at doses with equivalent immunosuppressive and anti-inflammatory activity (Cannigia *et al.*, 1977; Gennari *et al.*, 1980; Hahn, Baran & Halstead, 1979). We have studied 15 patients established on long-term treatment with oral prednisolone (> 3 months) for a variety of dermatological conditions and in whom deflazacort was substituted in an equivalent dose (5 mg prednisolone = 6 mg deflazacort). Patients were investigated before, and 3 months after, substitution.

The dose required of deflazacort did not change, nor did the clinical condition deteriorate, suggesting the adequacy of the substitution to control the dermatological disorder. Fasting urine calcium/creatinine, an index of net calcium release from bone, decreased, and this decrease was most marked in adult patients in whom calcium excretion was increased before starting deflazacort ($P < 0{\cdot}005$ at 1 month; $P < 0{\cdot}05$ at 3 months). This was associated with a decrease in hydroxyproline excretion but no change in serum alkaline phosphatase, suggesting that the decrease in skeletal losses was due to a decrease in bone resorption. Patients who had marked weight gain on prednisolone had significant weight loss (mean 5·48 kg) within 3 months of changing to deflazacort. There were no significant changes in glucose metabolism and blood pressure. We conclude that deflazacort is well tolerated, maintains adequate control of steroid-requiring dermatoses, and may have less adverse effects than prednisolone.