this work was virtually ignored for 40 years, I think at least he deserves the courtesy of a reference.

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3 Clarke, J. K. British Journal of Experimental Pathology, 1921, 5, 141.

Oculocutaneous reactions to beta-blocking drugs

SIR.—The "alternative explanation" produced by Drs P M Gaylard and I Sarkany (16 August, p 435), while plausible, does not in fact stand up to critical evaluation. In the first place there have been little, if any, reports of the oculocutaneous reaction in patients on any of the beta-blocking agents apart from pranolol, and most of those in whom oxprenolol or propranolol has been incriminated had also received pranolol in the past. Ten of the 21 patients reported by Felix et al subsequently received oxprenol and four were given propranolol and suffered no adverse effect, although 12 of the group were challenged with pranolol and developed reactions.

The clinical features of the oculocutaneous syndrome, the development of a positive antinuclear factor test, and the irregularities in the occurrence of this condition are very much in favour of an immunological rather than a pharmacological mechanism being involved. Not that they are mutually exclusive. However, the evidence adduced in favour of the possible pharmacological mechanism is faulty.

It is unfortunate that the term "psoriasiform" has stuck to the cutaneous component of this syndrome. It is also erythematous, and eczematous at times. Histologically, the lesion shows evidence of intraepidermal cell death and there is not the typical hypertrophy of psoriasis. One cannot therefore assume that increased epidermopoiesis is playing an important role in the pathogenesis of the epidermal disorder. While the importance of the cyclic nucleotides as regulators of cell division is now accepted, some systems cannot be denied, their role in the control of epidermopoiesis has not as yet been firmly established. Our own experience with phosphodiesterase inhibitors and that of others with other compounds active in this cyclic-cyclic nucleotide system as depressants of epidermopoiesis and as clinically effective agents in psoriasis has been disappointing.

There would appear to be good evidence for the involvement of immune mechanisms in the pathogenesis of the oculocutaneous syndrome and very little in favour of a pharmacological mechanism.

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SIR.—We read with great interest the case report by Mr R B Cubey and Dr S H Taylor (8 November, p 327) of an oculocutaneous reaction to propranolol and its subsequent resolution on continued treatment with a different beta-blocker. We have held for four years a special clinic for patients with coronary arterial disease and have seen no oculocutaneous reactions to propranolol, nor indeed to timolol maleate (Blocadren). Routine ophthalmic examinations have been carried out in the clinic for the four years, and in no case of any suspicion of an adverse reaction the patient is then screened by the ophthalmologists in the normal manner.

There was no reference in the case report to the autoimmune status of the patient at the time that the ocular reaction was found or subsequently. We believe that determination of antinuclear factor may detect early adverse reactions, and other workers have found the antinuclear factor (ANF) titre to be elevated.1 There may well be a place for screening patients before starting treatment with the newer beta-blockers and indeed, since Mr Cubey and Dr Taylor's report, others have also regarded on long-term propranolol therapy.

We have found no significant ANF titre in 12 anginal patients who have been on timolol continuously for four years. Similarly, they have had no oculocutaneous reactions.

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1 Wright. P. British Medical Journal, 1975, 1, 595.