

Group	No. of Patients	Mean Creatinine Clearance (ml/min)		Mean Blood Pressure (Lying) (mm Hg)	
		Initial	Final	Initial	Final
I.C.C. <30 ml/min	18	15.2	21.3	187/119	155/90
I.C.C. 31-60 ml/min	14	45.7	47.6	194/122	157/93
I.C.C. >60 ml/min	37	102.5	93.8	180/116	140/86
All patients	69	72.3	69.2	185/118	148/88

intrinsically harmful effect of beta-blockade on renal function can be inferred. It is the general experience of any unit treating hypertensive patients with renal functional impairment that transient reduction in renal function may occur during the early stages of effective blood pressure control. This usually returns to the same level or possibly increases above the initial value. The increased peripheral resistance that can occur with beta-blockade might be shared by the renal vasculature. Should reduction in renal function occur after the institution of beta-blockade this may be an indication for the addition of a peripheral dilator such as hydralazine.

We intend to describe fully in future publications our experience of the treatment of hypertension in the presence of renal disease using propranolol. This will substantiate our earlier findings¹ that there is no indication for a reduction of propranolol dosage with reduced renal function.—We are, etc.,

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¹ Thompson, F. D., Joekes, A. M., and Foulkes, D. M., *British Medical Journal*, 1972, 2, 434.

Autoimmune Haemolytic Anaemia in Ulcerative Colitis

SIR,—We would like to report an interesting case of autoimmune haemolytic anaemia with an antibody showing rhesus specificity in a patient with relapsing ulcerative colitis. The anaemia apparently responded to steroid treatment of the colitis.

The patient, a 23-year-old nulliparous woman known to have colitis, was admitted on the present occasion with an exacerbation. Her haemoglobin concentration was 9.8 g/100 ml, mean corpuscular volume 119 pg, and reticulocyte count 7%. The blood film showed many spherocytes and the direct antiglobulin (Coombs) test was strongly positive. Serum folate (1.6 ng/ml) and red cell folate (131 ng/ml) levels were both low. She was treated with prednisone and folic acid.

The positive antiglobulin test showed IgG specificity and her serum contained antibody of mixed specificity, but an eluate of her red cells contained an antibody with anti- \bar{e} specificity. On treatment the haemoglobin rose to normal, the reticulocyte count fell to 3%, and the antiglobulin test became negative. The serum, however, still contained antibody now appearing to have some anti- \bar{e} specificity. Her probable rhesus genotype was cde/cde and this is consistent with the antibody being an auto-antibody.

There are several reports in the literature¹⁻⁵ describing the presence of positive direct antiglobulin tests in a total of eight patients with ulcerative colitis. Only a few

of these showed active haemolysis and only two of them showed any degree of rhesus specificity (in one patient an anti- \bar{e} plus f from a red cell eluate and in another patient an anti- \bar{e} in the serum). It is important to report such cases, which may help to elucidate the "aetiology and pathogenesis of this autoimmune haemolytic anaemia which does not have to be classified as idiopathic."⁶

We would like to thank Dr. Sheila Worledge for helpful discussion about this patient and Dr. T. M. Chalmers for permission to report the case.

—We are, etc.,

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¹ Lorber, M., Schwartz, L. I., and Wasserman, L. R., *American Journal of Medicine*, 1955, 19, 887.

² Balint, J. A., Hammack, W. J., and Patton, T. B., *Gastroenterology*, 1962, 42, 740.

³ Fong, S., Fudenberg, H., and Perlmann, P., *Vox Sanguinis*, 1963, 8, 668.

⁴ Gastaldi, F., et al., *Rivista di Emoterapia ed Immunoematologia*, 1963, 10, 193.

⁵ Molaro, G. L., and Barillari, B., *Rivista di Emoterapia ed Immunoematologia*, 1964, 11, 43.

⁶ Dacie, J. V., *The Haemolytic Anaemias*, Part III, p. 826. London, Churchill, 1967.

Urethral Prolapse in Children

SIR,—Your leading article (4 May, p. 240) perhaps makes this condition sound more complicated than it really is. From an experience of some 25 patients under the age of 8 may I suggest two points? (1) The diagnosis is usually easily made by passing a urethral catheter—prolapse is the only tumour which symmetrically surrounds the catheter. (2) Treatment is best done as an outpatient, avoiding the psychogenic trauma of separation in a frightened young child. Under general anaesthesia (so rapidly eliminated today) two stitches (Dexon (polyglycolic acid) may be best) are inserted through normal urethra at right angles to one another. The prolapsed tissue distal to the sutures is then excised. The "cross" is then picked up with forceps and converted into four stitches, which can be tied with no fear of retraction of the mucosa.

I believe it is unwise to leave a catheter in the bladder as this only leads to infection. I have not found it necessary to catheterize subsequently for retention and I am not aware of urethral stricture developing subsequently (though my follow-up is not complete).—I am, etc.,

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Photosensitivity to Pyrimethamine?

SIR,—I wish to report an apparent photosensitivity reaction to pyrimethamine (Daraprim). The Committee on Safety of Medicines has had only one similar notification, in a male of unknown age who was given a course of pyrimethamine. He de-

veloped a photosensitivity rash 10 days after taking his first tablet.

My patient was a 9-year-old boy of English parents who arrived by air in Lagos during March 1974. At 23.00 hr that day he was given one 25-mg Daraprim tablet. At 17.00 hr the following day he developed a pink, confluent, macular, non-pruritic rash over his cheeks, forearms, and legs which corresponded exactly with those areas exposed to the sun. There was no malaise. The rash disappeared overnight but recurred on the third day following exposure to the sun. It again disappeared overnight and reappeared on the fourth day following exposure to the sun. On the fifth day there was no rash. He had taken pyrimethamine on many occasions previously, though this tablet was the first of his present course. He had never before had a skin rash and had no known drug or other sensitivities. There was no family history of drug or other sensitivities. He was taking no other drugs.

Wellcome-Lepetit (Nigeria) Ltd. analysed the remaining tablets from the packet and tablets from their quality-control stock and were unable to detect any deviation from their standard product.—I am, etc.,

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Fenticlor, Actinic Reticuloid, and Antihistamines

SIR,—In the article "Diseases of the Skin: Acne Vulgaris" (15 December 1973, p. 667), Dr. W. J. Cunliffe suggests fenticlor as a suitable topical antimicrobial agent in treatment. Fenticlor causes photocontact dermatitis¹ and two cases of chronic dermatitis from persistent photosensitivity² have followed its incorporation into a hair cream in the United Kingdom. Except for the absence of two chlorine molecules, fenticlor is identical with bithionol,³ known to cause distressing persistent photodermatitis which forces the sufferer to live a confined indoor life.⁴ Fenticlor and bithionol cannot be imported into Australia without Commonwealth authority, and the use of bithionol in consumer goods in the U.S.A. and Canada is restricted by government legislation.

The term "actinic reticuloid" describes a chronic dermatosis associated with severe photosensitivity and histological resemblance to lymphoma. Ives *et al.*⁵ in the original report of this condition suggest a relationship with the chronic photodermatitis caused by photoallergens such as bithionol and fenticlor. Wilkinson⁶ described a case of actinic reticuloid which first developed in 1969 but in which sensitivity to tetrachlorosalicylanilide, a halogenated salicylanilide related to fenticlor and bithionol, had been diagnosed in 1960. A direct causal relationship between actinic reticuloid and contact photoallergens has not been established, though such a relationship must be suspected. Sneddon⁷ described a case of actinic reticuloid which may have developed into a true lymphoma.

It has been proposed that persistent light reactions following the use of contact photoallergens are caused by the retention of those photoallergens in the dermal layer of skin for long periods.⁸ This concept might con-