Case No.	Age (Years)	Sex	Day of Illness	Presenting Features	Additional Observations	*Immunoglobulin Levels (% M.N.A.)		
						IgG	IgA	IgM
1	17	F.	14	Sore throat, cervical lymphadenopathy	Persistent H.S.V. excretion in urine. History of recurrent herpes.	155	220	210
2	19	F.	5	Genital herpes (persisting for 16 days)	H.S.V. isolated from genital lesions. Fourfold C.F. antibody rise to H.S.V.	66	34	62
3	5	M.	20	Erythema multiforme	Primary H.S.V. infection. Sibling with herpes labialis.	72	38	100
4	68	F.	N.K.	Atypical purpuric varicelliform rash	Carcinoma of cervix. Treated with cyclophosphamide 2 months previously; on steroids. >4-fold C.F. antibody rise to H.S.V.	68	24	20
5	14	M.	26	Probable herpes encephalitis	C.F. titre to H.S.V.: serum, 80/320 (acute/conv.); C.S.F., 12.	62	125	115

*Normal limits: IgG, 54-170% mean normal adult value (M.N.A.); IgA, 45-172%; IgM, 50-180%.
H.S.V. = Herpes simplex virus. C.F. = Complement fixation. N.K. = Not known.

infection showing a poor response to treatment with local idoxuridine. The occurrence of low serum IgA levels in recurrent herpes simplex infections has been previously reported by Tokumaru.[2] He also speculated on the association of complications with a deficiency of specific IgA. No correlation between the presence or absence of secretory IgA and recurrent herpes infections was, however, noted by other workers.[3,4] Our own finding and these reports prompted us to examine IgG, IgM, and IgA levels in a few more sera from patients with evidence (serological or by virus isolation) of current herpes infection.

In the few cases we have studied by a standard technique using Hyland Immunoplate III (see table) three out of five patients had considerably reduced serum IgA levels, one having in addition a low IgM level (Case 4). Interestingly, two of the three patients with low total IgA levels had generalized skin manifestations (erythema multiforme (Case 3) and an atypical varicelliform rash (Case 4)). The findings in the child with erythema multiforme might, however, reflect an age-associated immaturity of the IgA system.

This is a very small series of cases from which no definite conclusions can be drawn. We would, nevertheless, be very interested to hear whether other workers have made similar observations.—We are, etc.,

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1 Zanussi, C., and Medina, F., in *Gammapathies, Infections, Cancer, and Immunity*, ed. V. Chini, L. Bonomo, and C. Sirtori. Milan, Carlo Erba, 1968.
2 Tokumaru, T., *Journal of Immunology*, 1966, **97**, 248.
3 Douglas, R. G. jun., and Couch, R. B., *Journal of Immunology*, 1970, **104**, 289.
4 Centifanto, Y. M., Little, J. M., and Kaufman, H. E., *Annals of the New York Academy of Science*, 1970, **173**, 649.

Potentiation of Tardive Dyskinesia: Possible Drug Interaction

SIR,—Like others who use depot phenothiazines, I am concerned about the side effects—for example, tardive dyskinesias. Our Modecate (fluphenazine) clinic,[1] has 380 outpatients and there are 20 with this type of dyskinesia. The suggestion of Dr. P. M. O'Flanagan (1 February, p. 269) to use clonazepam will be gratefully evaluated.

Recently three patients who have been on fluphenazine decanoate developed extrapyramidal side effects (two with Parkinsonism and one with akathisia), which responded to benztropine. Some months later all three developed the classical features of tardive dyskinesia. Treatment of such dyskinesias is difficult; often it is worsened by anticholinergic anti-Parkinsonian drugs. One explanation may be that benztropine lowers the threshold for the appearance of these dyskinesias which, combined with the increased responsiveness of dopamine receptor sites due to the induction by the phenothiazine of denervation hypersensitivity,[2] produces this syndrome. There is, of course, a need for critical assessment of this possible interaction between benztropine and fluphenazine.

I would welcome correspondence with other research groups who have had similar experiences.—I am, etc.,

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1 Marriott, P. F., *et al.*, *Medical Journal of Australia*, 1973, **2**, 957.
2 Klawans, H., *American Journal of Psychiatry*, 1973, **130**, 82.

Exfoliative Dermatitis during Treatment with Pheneturide

SIR,—We wish to report a case of exfoliative dermatitis apparently due to pheneturide (Benuride).

A man aged 23 years, a long-standing epileptic, was maintained on phenobarbitone 60 mg three times a day and phenytoin 100 mg twice daily. Phenytoin was discontinued because the patient developed gum hypertrophy and was replaced by pheneturide 200 mg three times a day in addition to the phenobarbitone. Three weeks later he developed generalized pruritus and a vesicular eruption followed within a few days by exfoliation, lymphadenopathy, and enlargement of the spleen to 3 cm below the costal margin. The white cell count was $7{\cdot}2\times10^9$/l (7200/mm^3), 50% of which were eosinophils ($3{\cdot}6\times10^9$/l (3600/mm^3)).

The pheneturide was discontinued and he was put on prednisolone 40 mg daily. Within one week of starting prednisolone treatment the lymphadenopathy and pyrexia had disappeared, the spleen was impalpable, the rash had virtually resolved, and the eosinophil count had fallen to $2{\cdot}0\times10^9$/l (2000/mm^3).

Exfoliative dermatitis has not previously been described in association with pheneturide. Because this drug has been recently introduced it is important to be aware of this possible complication.—We are, etc.,

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Undiagnosed Haematuria

SIR,—Your leading article (22 March, p. 647) is reasonably satisfactory, though in the group of patients in which early positive diagnosis is not obtained the point that further reinvestigation is essential was not adequately made.

I am writing, however, to point out that the statement that "the accuracy of cystoscopic diagnosis and biopsy has been transformed by the introduction of fibreglass illumination" is totally illogical. Fibrelit illumination is no better than conventional illumination, but many people consider it to be more convenient, though I personally would not agree; and it is seriously misleading to suggest that fibrelit endoscopic equipment is essential for modern urology. If there has been any improvement it is because the eye at the other end of the cystoscope is now better trained than it used to be.—I am, etc.,

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Medicine in the Sun

SIR,—Dr. C. H. C. Thomas describes in his Personal View (22 March, p. 678) how a hospital's refusal to admit an unconscious patient finally drove him to emigrate "to sunnier climes." The land he chose to settle in is one where for 70% of the population the infant mortality is 140 per 1000. In a survey of 3000 African children in the Transvaal Richardson[1] reported kwashiorkor or marasmus in 6%. In the Cape Town townships of Zanga, Nyanga, and Gaguletu one doctor serves 108 379 residents.—I am, etc.,

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1 Richardson, B. D., *South African Medical Journal*, 1973, **47**, 688.