TREATMENT OF A COMMON OCULAR COMPLAINT

To the Editor: Dr W. Rakusin[1] makes reference to the comparative efficacy of two ophthalmic solutions containing different concentrations of zinc sulphate.

Having experimented quite extensively with eyedrops in animals, I should like to say that unless Dr Rakusin can assure us that the solutions he compared were in all respects identical, except for the differences in the zinc concentrations, his conclusions, as expressed, are not acceptable.

Differences in excipients such as buffers, thickeners, preservatives, stabilizers, etc., can all alter the efficacy. Most marketed eyedrops containing zinc sulphate are formulated with one or more other active ingredients such as naphazoline, tetrahydrozoline, phenylephrine, antazoline, etc. If other active agents were also present in the solutions used, were they the same, and were they present in identical concentrations?

Many 'clinical trials' compare the efficacy of different marketed products that are intended for the treatment of the same condition/s. If this is what Dr Rakusin did, then his comments are of value, but one must know which of the available options he chose to follow before his conclusions can be taken at all seriously.

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1. Rakusin, W. (1979): S. Afr. med. J., **55**, 3.

Dr W. Rakusin comments: I agree with Dr Straughan's comments that buffers and preservatives, etc., alter the efficacy of the drops. The two drops used in the trial were Ocufort, which contains zinc sulphate 0,1%, phenylephrine hydrochloride 0,12% and tetrahydrozoline 0,4%, herbs and aromatic water in an isotonic sterile eyedrop solution, and Oculosan, which contains zinc sulphate 0,02%, naphazoline 0,005%, herbs and aromatic water in an isotonic sterile eyedrop solution. The vehicle in each drop was obviously not identical.

However, the aims of the trial were (*i*) to ascertain the effectivity of Ocufort as an astringent and decongestant compared with a known astringent, Oculosan; (*ii*) to determine the safety of Ocufort and its lack of side-effects; and (*iii*) to determine patient tolerance of Ocufort.

HEARTFELT THANKS

To the Editor: I wish, through the courtesy of your *Journal*, to record my appreciation of and gratitude to my colleagues in this town.

During 1972 I developed a postinfective myelopathy, with resulting paraplegia, and could not attend to my duties for a long time. My colleagues came to my rescue by attending to my practice, and even lent financial aid.

Since I have returned to active practice, they have helped me by doing visits on my behalf, as well as performing operations for me.

I am also most grateful to several specialist neurologists, neurosurgeons and urologists who have attended me on various occasions, in Cape Town, Johannesburg and Port Elizabeth.

Money could never express the gratitude I owe to all these people.

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OBSERVATIONS ON HYPERURICAEMIA

To the Editor: A White man aged 44 years presented with a blood pressure of 160/110 mmHg. He was free of symptoms and otherwise healthy. His wife, a qualified nursing sister, had been taking his blood pressure for some time previously, and had found it to be elevated.

The only abnormality found on routine investigation was a serum uric acid level of 9,1 mg/100 ml. It was decided to defer treatment of his hypertension until his uric acid level, through medication, returned to normal. Within 6 weeks the uric acid level had decreased to 5,7 mg/100 ml, and the blood pressure was 120/80 mmHg, and has remained so.

The patient's son, aged 17 years, presented shortly afterwards with pain in his right knee; he had suffered pain and swelling of the joints since childhood. Periodic investigations (which had never included estimation of the serum uric acid level) were negative, and he was said to be 'rheumatoid'. An estimation of serum uric acid revealed a level of 9,1 mg/100 ml. His two siblings, aged 15 and 12 years, were then investigated, and were found to have uric acid levels of 10,4 mg/100 ml and 7,9 mg/100 ml respectively.

Hyperuricaemia is transmitted as a dominant autosomal gene with different degrees of penetration in the two sexes,[1] and 90% of patients are male.

I now do the following as a routine:

In the case of every new hypertensive patient with a raised serum uric acid level, I lower the uric acid level to normal before treating the hypertension.

In every confirmed case of hyperuricaemia, I screen the whole family, as is done in cases of hypercholesterolaemia, diabetes, etc.

For every child with a musculoskeletal complaint, I include a uric acid determination in the differential diagnosis.

There are certain matters which are still not clear concerning children, viz. (*i*) at what stage should the serum uric acid level be considered to be sufficiently high for treatment to be instituted? (*ii*) at what age should a child be treated? (*iii*) should the child be treated if he has no symptoms? and (*iv*) having regard for the sequelae, should a child be subjected to treatment for life?

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1. Vaughan, V. C. and McKay, R. J., eds (1975): *Nelson's Textbook of Pediatrics*, 10th ed., p. 448. Philadelphia: W. B. Saunders.

SMOKING AND INSURANCE POLICIES

To the Editor: With regard to the correspondence[1,2] about smoking and life insurance, I should like to ask whether the tobacco companies invest large sums of money with life insurance companies?

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1. Muller, H. (1978): S. Afr. med. J., **54**, 594.
2. Haifer, M. (1978): *Ibid.*, **54**, 1043.

ERRATA

In the book review entitled 'Achalasia of the cardia', by W. Silber, which appeared on page 229 of the *SAMJ* of 10 February 1979, there was an error in the fifth sentence of the first paragraph, which should have read as follows:

'The term cardiospasm should be excluded. It should not be used synonymously with achalasia, which is neither a disease of the cardia nor a spasm.'

* * *

In the article 'A possible role of prolactin in preventing heartburn during pregnancy', which appeared on page 127 of the *SAMJ* of 27 January 1979, the degrees of one of the co-authors, Dr E. J. Robertson, should have been M.B. B.Ch., M.D.