**Book review**


The authors set out to review such clinical trials as have been conducted in the rheumatic diseases. Individual chapters are devoted to trials in osteoarthritis, rheumatoid arthritis, juvenile arthritis, seronegative arthritides, crystal arthritis, systemic lupus erythematosus, and low back pain. In addition, there is a useful preliminary chapter on methodological considerations in clinical trials and a final chapter which includes a proposal for a system by which the design, execution, and analysis of trials may be coded so as to improve reporting and interpretation of trials.

Each chapter includes discussion of methodological problems relevant to the disease and recommendations on methods to be used in future studies. Clinical trials are discussed in general terms in the text, while specific comments on methodology and results are included in tables. This format does not make for particularly easy reading, especially since the abbreviations used in the tables are not always self evident. Despite the length of the book, discussion of individual drugs is often brief; for instance trials of penicillamine in rheumatoid arthritis are dealt with in less than two pages of text and three pages of tables. The authors state that they have made no attempt to provide guidelines for prescribing since they recognise that formalised trials are not the only source of information used by clinicians in therapeutic decision making. Indeed while reading this book one is repeatedly struck by the fact that common beliefs regarding therapy in rheumatic disorders, such as the relative efficacy of different non-steroidal anti-inflammatory drugs, are clearly based on factors other than the results of clinical trials.

This book is an invaluable source of references to clinical trials reported up to 1983, and the associated comments on methodology should be read by anyone planning to undertake a trial in the rheumatic diseases.

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**Correction: Evaluation of an ELISA system for determination of class-specific antibodies to native and denatured DNA in man**

In the paper by G M Halliday, M R Salaman, M H Seifert, K J Johnson, and A D B Malcolm (Ann Rheum Dis 1985; 44: 507-13) we regret that Fig. 8 was incomplete. The figure and explanatory text are reproduced below.

![Fig. 8](http://ard.bmj.com/)

**Fig. 8** Inhibition of ELISA for IgM antibodies to denatured DNA (dDNA) in the presence of our standard preparations of native DNA (nDNA) (●) and dDNA (▲) and of plasmid DNA (nDNA-P) (○). Two experiments were carried out but one did not include the highest level of inhibitor (100 μg/ml). Percentage inhibition is given as mean±SEM. (SI conversion: μg/ml=mg/l).

The source of antibodies was pooled plasma from patients with systemic lupus erythematosus. The figure was previously printed without the triangle point at 100 μg/ml which demonstrates a high level of inhibition by dDNA. The native preparations were less potent inhibitors at this and lower concentrations, and these results may be compared with those obtained in the inhibition of ELISA assays for IgM antibodies to nDNA and for IgG anti-DNA antibodies (Figs 5-7).