Degenerative Joint Disease in a Subtropical Population. By J. S. LAWRENCE, W. E. MIAL, J. M. BREMNER and M. MOLYNEUX (*A.R.C. Field Unit*).—Published in full in the *Annals* in July, 1968.

Evaluation of a Simple Articular Index for Joint Tenderness in Rheumatoid Arthritis. By D. A. RITCHIE, J. A. BOYLE, J. M. MCINNES, M. K. JASANI, T. G. DALAKOS, P. GRIEVESON, and W. W. BUCHANAN (*Glasgow*).—An index was described for the numerical measurement of joint tenderness in patients with rheumatoid arthritis. The index is based on the summation of a number of quantitative evaluations of the pain experienced by the patient when the joints were subjected to pressures when exerted over the articular margin or in some instances on movement of the joint.

A statistical analysis in experimentally controlled conditions was undertaken to test the validity and the errors of this simple clinical tool. There was a satisfactory degree of reproducibility when the assessment of joint tenderness using this index was made by one observer on different occasions. However, there was a far from acceptable degree of correlation between the results obtained by different observers.

That the index can record changes in the patient's joint tenderness in the practical clinical situation was shown by the fall in articular score of a group of patients treated in a formal double-blind cross-over study using aspirin, prednisolone and placebo. Clear differences were demonstrated not only for the major analgesic effect of prednisolone (compared with placebo), but also for the minor analgesic effect of aspirin (compared with placebo).

The limitations and advantages of this and other attempts to make a quantitative objective assessment of that most subjective phenomenon, pain, were discussed.

Discussion.—DR. S. GODFREY (*London*): We have studied observer error in pulmonary disease using both the standard deviation index and an experience agreement index which compared the most experienced with the less experienced observers. The results suggested the physical signs lay mid-way between completely random and completely reliable. Could you put your index in these terms?

DR. BUCHANAN. We did not analyse our data in these terms. We were surprised how big the differences were between observers. Indeed, a difference in score of 20 or less between two observers in any one patient was calculated not to be significant. Studies in other fields of medicine have also shown some high degrees of inter-observer error. A clinician using an index or scoring system should assess inter- and intra-observer error as our laboratory colleagues do automatically when they estimate the standard error of a new laboratory technique.

Corticosteroid-induced Hypothalamo-pituitary-adrenal Axis Suppression: Relationship to Regimen of Therapy. By M. K. JASANI, J. A. BOYLE, W. C. DICK, A. K. TAYLOR, J. WILLIAMSON, and W. W. BUCHANAN (*Glasgow*).—Published in full in the *Annals* in July, 1968.

Effect of Corticotrophin Therapy on Pituitary-adrenal Function. By MARY E. CARTER and V. H. T. JAMES (*St. Mary's Hospital, London*).—Patients receiving ACTH therapy were studied to assess the effect of this form of therapy on pituitary-adrenal function, using hypoglycaemia as a test stimulus. The results were compared with those found in steroid-treated patients, and the effect of previous steroid therapy was considered.

Discussion.—DR. A. B. MYLES (*London*): Dr. Jasani—how long after the dose of triamcinolone was the articular index calculated, and how long after the last dose was the plasma cortisol estimated?

DR. JASANI: For the measurement of diurnal rhythm as well as the assessment of the articular index the corticosteroid therapy was left unaltered. However, for determination of the plasma 11-OHCS responses to Synacthen, lysine⁸-vasopressin, and insulin-hypoglycaemia, in keeping with a previous study, corticosteroid therapy was discontinued 12 hours before the test in the patients given triamcinolone 4 mg. nightly.

DR. A. B. MYLES (*London*): In the patients who had the dose at night who had a low plasma cortisol in the morning, you do not know what happened at, for example, 2 p.m., because the night dose of steroids will suppress the early morning peak but the maximum rise may occur later. You also do not know what happened on the steroid-free days. You will get acute suppression by giving steroids at night but it would be surprising if you produced chronic suppression by giving such a small dose of triamcinolone for this period.

DR. JASANI: The schedule for the test procedures was designed to find out if we should obtain HPA axis suppression with such small doses of the corticosteroid given for 1 year. However, you have raised other interesting possibilities that need investigation.

DR. A. B. MYLES (*London*): You have shown some changes in the hypoglycaemia tests but all your results are within the normal range.

DR. JASANI: I agree that, with the exception of response to insulin hypoglycaemia in two out of six patients given triamcinolone 4 mg. nightly, the plasma 11-OHCS levels were not suppressed significantly. These findings probably reflect the use of a relatively smaller daily dose (5 mg. prednisolone equivalent) of triamcinolone. There might of course be a greater HPA axis suppression with administration of the corticosteroid using either regimen for a longer period.

DR. A. B. MYLES (*London*): The cortisol levels would have been influenced by the previous dose if taken on the night before.

DR. JASANI: The diurnal variation would be, and this is why it was not stopped. I accept your comment but I should add that the only compelling reason for adopting this practice was to conform to the design of study employed by Nichols, Nugent, and Taylor (1965). They measured the diurnal rhythm of cortisol during the 24 hours following a dose of dexamethasone at midnight and found it was completely abolished. In our study, the 10 p.m. dose of triamcinolone represents their nightly dose.

DR. A. B. MYLES (*London*): Why do you use triamcinolone which most other people do not use?