This contrasted significantly ($P < 0.01$) with a mean rise in the placebo group of 2,000 cells/c.mm. (S.D. $\pm 2,100$). There was no important overall difference in the haemoglobin, the platelets, the E.S.R., the titre of the latex tests, the strength of grip, or the blood pressures between the two groups.

Discussion.—DR. G. D. KERSLEY (*Bath*): reported that he had treated 21 cases of severe rheumatoid arthritis, uncontrolled by a reasonable dose of steroids, with azathioprine. Nine patients improved during the period of treatment, and in four the dose of corticosteroid was lowered. Leucopenia, which was corrected on withdrawal of the drug, occurred in one case. Gastrointestinal symptoms were the most consistent and troublesome side-effects.

DR. J. A. MATTHEWS (*St. Thomas's Hospital*) asked if it were possible from the data to say whether azathioprine was suppressing the disease directly or was potentiating the action of corticosteroids.

DR. DUNNE suggested that on the evidence available azathioprine exerted a direct suppressive effect on the disease.

Natural Antibody to Procainamide and Procainamide-induced Systematic Lupus Erythematosus. By A. S. RUSSELL and M. ZIFF (*Southwestern Medical School, University of Texas, Dallas*): A method was developed for assay of antibodies reactive with procainamide* (PrA) which utilized sheep red blood cells coupled with diazotized PrA. Antibodies of the IgM group were found in 19 to 50 per cent. of populations tested. These included patients with SLE, RA, juvenile RA, miscellaneous diseases, cardiac patients receiving PrA, and normal adults. Only 6 per cent. of patients with PrA-induced SLE had antibody. Antibody was not found in neonatal children nor in rabbits and hamsters but was found in 78 per cent. of normal dogs.

Immunization of rabbits with PrA—bovine serum albumin complex induced anti-PrA antibody in high titre. Antibodies in these rabbits and in man could be removed by adding PrA to the titration system.

74 per cent. of unselected patients receiving PrA for over 2 months were found to have antinuclear antibody in significant titre. It was suggested that the high incidence of antinuclear antibody and SLE-like disease in patients receiving PrA might be related to the wide occurrence of "natural antibody" to PrA.

Discussion.—DR. D. A. PITKEATHLY (*Wigan*) asked why antibody to procainamide should be so common in the population.

DR. RUSSELL suggested that the high incidence of antibody could be explained by cross-reactivity with other substances.

DR. L. E. GLYNN (*Taplow*) asked if procainamide-induced SLE might provide a model for testing drugs.

DR. RUSSELL suggested that this might be so in the

States, but in view of the small numbers taking the drug in Great Britain it might be hard to find suitable patients.

Experiences of Anthroplasty of the Rheumatoid Knee using Macintosh Prostheses. By A. KATES, A. KAY, J. WOJTULEWSKI, T. RILEY, E. N. COOMES (*St. Mary Abbots and St. Stephen's Hospitals*): Macintosh arthroplasties have been introduced into the knees of forty patients with rheumatoid arthritis. In six patients the procedure was carried out in both knees.

The period of follow up is from 6 months to 2½ years. The results, so far, suggest that it is a worthwhile procedure.

Selection of patients and problems of management were discussed.

Discussion.—DR. J. D. JESSOP (*London Hospital*) commented that five of thirty patients in whom the Mackintosh operation was performed developed marked flattening of the femoral condyles about 30 months after operation. This phenomenon was noticed whether or not the patients were on corticosteroids.

MR. KATES said that they had not observed this flattening of the condyles. He considered that it might be caused by early manipulation post-operatively. He felt that cementing of the prosthesis greatly improved the results of the operation.

Aggressive Action of Lymphocytes *in vitro*—Investigation of their Property in relation to Inflammatory Joint Disease. By I. C. M. MACLENNAN (*Taplow*): Lymphocytes from the blood of healthy persons had been shown to damage certain homologous and heterologous cells *in vitro*. Quantitative assessment of this phenomenon had shown this aggression to be similar in patients with chronic inflammatory joint disease and healthy controls. Lymphocytes from the synovial fluid of certain patients with arthritis had been found to be quantitatively more cytotoxic to homologous target cells than were equivalent numbers of peripheral blood lymphocytes. Joint lymphocytes from about one-third of patients with seropositive rheumatoid arthritis showed this increased cytotoxicity, and it was seen with joint lymphocytes from a high proportion of patients with psoriatic arthritis and systematic lupus erythematosus. This high level of cytotoxicity of certain joint lymphocytes was not specific to a particular type of human target cell as it had been shown by Hedberg against diploid human foetal cells of both renal and cutaneous origin and by the speakers against polyploid human liver cells.

A factor in joint fluid and serum from certain patients with chronic inflammatory joint disease was described, which increased the cytotoxicity of normal lymphocytes to a polyploid strain of human lymphocytes. Evidence was presented to support the hypothesis that this was an IgG antibody specific for the target cell. This factor had also been found in non-arthritic patients and its significance had still to be worked out.

* Pronestyl.