OFFICERS and COMMITTEE,

President: DR. A. G. S. HILL, *M.C., F.R.C.P.E.*, Rheumatism Research Centre, Stoke Mandeville Hospital, Aylesbury, Bucks.

President-Elect: DR. L. E. GLYNN, *M.D., F.R.C.P.*, Canadian Red Cross Memorial Hospital, Taplow, Maidenhead, Berks.

Hon. Treasurer: DR. F. DUDLEY HART, *F.R.C.P.*, Westminster Hospital, London, S.W.1.

Senior Hon. Secretary: DR. H. L. F. CURREY, *M.R.C.P.*, The London Hospital, Whitechapel, E.1.

Junior Hon. Secretary: PROF. V. WRIGHT, *M.R.C.P.*, Rheumatism Research Unit, Medical School, Leeds 2.

Hon. Librarian: DR. W. S. C. COPEMAN, *C.B.E., T.D., F.R.C.P.*, 129 Harley Street, W.1.

General Secretary: M. C. G. ANDREWS.

Members of the Executive Committee

DR. J. A. COSH, *F.R.C.P.*, Royal National Hospital for Rheumatic Diseases, Bath.

PROFESSOR K. W. WALTON, *Ph.D., M.D.*, Rheumatism Research Wing, Dept. of Experimental Pathology, The Medical School, Birmingham 15.

DR. A. J. POPERT, *M.R.C.P.*, St. John's Hospital, Droitwich, Worcs.

DR. P. H. N. WOOD, *M.B., B.S., A.R.C.* Field Unit, Clinical Sciences Building, York Place, Manchester 13.

DR. W. W. BUCHANAN, *F.R.C.P.E.*, The Centre for Rheumatic Diseases, Baird Street, Glasgow, C.4.

PROGRAMME FOR 1970

February 27: Clinical Meeting, Stoke Mandeville Hospital, Aylesbury.

May 23 and 24: Combined Meeting of the Society and the French League against Rheumatism, Paris.

September 25: Heberden Round, Royal College of Physicians, Edinburgh.

October 17: B.M.A./Heberden Society, Bath.

November 20 and 21: The Heberden Oration (Dr. J. Ball). Annual General Meeting and Dinner. Royal College of Physicians, London.

Submission of abstracts

The Executive Committee has decided to change the procedure by which abstracts are assessed. In future all abstracts will be considered anonymously, that is without knowledge of the name(s) of the author(s) or the institution of origin.

Members wishing to present original communications to the Society are therefore asked to prepare abstracts which provide sufficient information for assessing the paper on merit—an assessment which amounts to competitive selection between the various abstracts submitted.

In the preparation and submission of abstracts, the following points should be observed:

(1) Abstracts should not exceed 300 words. Each must be headed by a title, authors' names, institution, and address.

(2) An actual summary of the communication is required. This will usually involve:

(i) A brief introduction to the work.

(ii) An outline of the methods used.

(iii) A summary of the results.

(iv) A statement of the main conclusions.

(3) Simple tables may be included. This is an excellent method of summarizing data.

(4) Accepted abstracts may be published as such with the proceedings of the Society in the *Annals*. So, unless they are received in a form suitable for publication, they will be returned to the author(s) for re-writing. In particular, statements such as "The data will be discussed" are entirely unacceptable.

(5) Abstracts should be sent to the Senior Honorary Secretary, The Heberden Society, c/o Arthritis and Rheumatism Council, Faraday House, 8-10 Charing Cross Road, London, W.C.2. Abstracts received at least 2 weeks before each Executive Meeting (normally held on the same day as scientific meetings of the Society) will be assessed at that meeting.

(6) When submitting abstracts, authors must state whether the communication has been or is about to be read at another meeting; or has been or is about to be published.

(7) Authors will be notified (i) when an abstract is received, (ii) when it is either accepted or rejected, (iii) when it is placed on a programme for a particular meeting.

Joint meeting with the Société Française de Rhumatologie

Paris, May 23 to 24, 1970

Hip Involvement in Juvenile Chronic Polyarthritis. By B. M. ANSELL and M. UNLU (*Taplow*):

Involvement of the hip in children suffering from juvenile chronic polyarthritis is relatively common. Thus, of the eighty cases reviewed by Jacqueline, Borjot, and Canet (1961), severe hip involvement was present in 54. To inform ourselves further, those cases we are following up have been appraised 10 years after the onset of the disease. By January 1, 1968, there were 235 such patients, 98 boys and 137 girls. A pelvic x ray had been obtained in all except nine, and these films together with fifty pelvic x rays of young people from a population survey were read on to a specially designed form aimed at including information on the development of the femoral head, neck, and acetabulum, and migration of the femoral head, as well as the usual features of periostitis, erosions, cysts, sclerosis, and narrowing of joint space. Due attention was paid to the state of the sacroiliac joints. Correlations were made with clinical involvement of the hip, the duration of the active disease process, age at onset, serology, and the state of the sacroiliac joints.

Taking the group with Still's disease as a whole, 40 per cent. of the 93 male films and 48 per cent. of the 133 female films showed one or more abnormalities. Those cases which were still active 10 years after the onset of disease showed more radiological change, this being present in 65 per cent. of the males and 68 per cent. of the females, compared with 24 per cent. in inactive males

and 30 per cent. in inactive females. Growth anomalies were particularly common in those whose disease had commenced when they were less than 5 years of age, and even in the absence of overt clinical involvement minor growth changes had occurred. As these young people form part of a large prospective study, serial films taken both before and after the 10-year follow-up have since been examined in an attempt to elucidate the mode of progression of hip changes in juvenile arthritis.

JACQUELINE, F., BORJOT, A., and CANET, L. (1961) *Arthr. and Rheum.*, 4, 500.

Anatomo-clinical forms and Haemodynamics of Chronic Ischaemia at the Upper End of the Femur. By J. ARLET and P. FICAT (*Toulouse*).

Arthrosis, arthritis, and ischaemia are the three known causes of apparently simple pathological conditions of the hip; they may coexist and their pathological effects may be cumulative.

Osseous ischaemia does not initially present a specific radiological appearance.

A histological study of the bone and of the synovium or cartilage was carried out by combined biopsy.

A haemodynamic study of the epiphysometaphysal region was made by measuring the intramedullary pressure before and after serum injection and by petrochanteric phlebography.

The experimental material consisted of 300 cases subjected to at least two of these investigations, and fifty underwent a combined biopsy.

We found histological proof of ischaemia in more than 100 cases and especially in forty cases where the hip was radiologically normal. There was a variety of chronic ischaemic lesions (early aseptic ischaemic necrosis) of the upper end of the femur. These are illustrated by the following examples:

- (1) Chronic ischaemia due to medullary overtaxation (Gaucher's disease).
- (2) Chronic ischaemia due to intramedullary haemorrhage occurring in the course of haemorrhagic disease or after osseous trauma (traumatic haematoma).
- (3) Chronic ischaemia due to thrombosis of the large arterial iliac trunks.
- (4) Chronic ischaemia due to impeded venous drainage (post-phlebotic necrosis).
- (5) Chronic ischaemia due to a reflex circulatory disorder: complication of the dystrophic sympathetic reflex.
- (6) Ischaemic necrosis associated with inflammatory synovitis (disseminated lupus erythematosus, rheumatoid arthritis, inflammatory monoarthritis).
- (7) Ischaemic necrosis of hyperuricaemia with or without hyperlipidaemia.
- (8) Necrosis resulting from a dysplastic hip.

Felty's Syndrome: A Clinical and Pathological Survey of 21 Patients. By C. G. BARNES, A. L. TURNBULL, and B. V. ROBERTS (*The London Hospital*).

Six men and fifteen women were diagnosed between 1957 and 1969. The men were significantly older than

the women at the onset of arthritis but developed the syndrome sooner. Many non-articular rheumatoid features were present, Sjögren's syndrome being found in eleven of sixteen patients tested.

The sera of all but two contained rheumatoid factor, usually in high titre, and L.E.-cell preparations and/or antinuclear factor tests were positive in fourteen patients. Absolute neutropenia was characteristic. Red cell survival (^{51}Cr) was reduced in six of thirteen patients studied, with excessive splenic destruction.

Partial haematological remission occurred spontaneously in one patient and in one of two who were receiving a small constant dose of prednisone (8 mg./day). Of eight patients given 20 mg. prednisone/day or more, four had a transient partial improvement and one an apparent clinical cure (5 years).

Ten patients underwent splenectomy, and two of these appear to have been cured. One other patient eventually achieved a normal blood count after an early relapse.

Seven patients have died, three despite steroids and splenectomy, three treated with steroids only, and one who received neither of these forms of therapy but suffered a cerebrovascular accident.

The following features were found in all thirteen spleens examined microscopically:

- (1) Sinus cell hyperplasia and erythrophagocytosis;
- (2) plasma cell hyperplasia and extramedullary haematopoiesis.
- (3) Hyaline change and endothelial hyperplasia of the follicular arteries.

Additional pathological features were observed in some of the spleens but were not present in all cases. Amyloid was not seen in any of the specimens.

Tendinous Ruptures in Rheumatoid Arthritis. By H. SERRE, L. SIMON, J. VIDAL, J. SANY, and Y. ALLIEU (*Montpellier*).

Tendinous ruptures in cases of rheumatoid arthritis are of various and usually associated mechanisms. Once the condition is recognized, effective prophylaxis can be applied. These ruptures may be related to mechanical factors causing tendinous attrition, resulting from rubbing against a bony protrusion. They may also be caused by tenosynovitis, the inflamed tissue often altering the structure of the tendon by means of vascular lesions.

The authors report their experience with such cases, emphasizing the frequency of tendinous ruptures of the extensors of the fingers. Generally located in the wrists, they affect especially the tendons of the third finger and the little finger touching the ulnar head dislocated backwards. Tenography, a new investigative technique, reveals interesting and exact information. Rupture of the extensor pollicis longus is often associated with erosion of the radial styloid process.

Less frequently, tendinous ruptures of the flexors occur in the carpal canal and affect especially the flexor pollicis longus and the flexors of the index finger.

Such lesions must be looked for systematically and identified precisely. They cause serious functional disability that can be treated by surgery. Intertendinous anastomoses and transplants are the best surgical techniques. Surgery is largely preventive. Tenosynovectomy and levelling of bony protruberances (especially resection of the ulnar head) may prevent the occurrence of ruptures.