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.

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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.