Deposition of Urate Crystals in Man. By W. A. KATZ and G. E. EHRLICH (*Albert Einstein Medical Centre, New York*): For more than 60 years the mainstream of investigation into the pathogenesis of gout centred in the mechanism of hyperuricaemia while recently urate crystal-induced arthritis had rekindled interest. Little attention, however, had been drawn to the deposition of these urate crystals in body tissues. It was the purpose of this experimentation to substantiate at a biochemical level heretofore anatomical observations of the predilection of urate crystals to deposit in the connective tissues such as cartilage and synovia. To this end it was demonstrated that an important ground substance component, PPL, a protein-polysaccharide containing chondroitin sulphate, was capable of augmenting urate solubility and inhibiting urate crystallization at 4°C.

A similar substance had been identified in the sera of both normal subjects and those with gout. It travelled electrophoretically with the alpha and beta globulins and was primarily responsible for the ability of serum to hold more urate in solution than buffers of identical molarity and hydrogen ion concentration. Alteration of the urate-bound PPL molecule resulted in precipitation of urate crystals in a manner proposed to occur *in vivo*. Utilizing this material, a schema was evolved which provided a new concept for the development of gout in man.

Discussion.—DR. J. T. SCOTT (*London*) asked if this hypothesis explained the predilection of urates to deposit in the great toe.

DR. KATZ felt that it would be interesting to analyse the nature of the polysaccharide in cartilage from various sites.

Centenary: Charcot's Other Joints. By J. T. SCOTT (*London*): It was exactly 100 years since Charcot had written his original description of neuropathic joints. This account, however, formed only the first part of a double paper dealing with "Arthropathies which appear to depend on a lesion of the brain or spinal cord". The second part described a form of arthropathy complicating cerebral thrombosis and in fact seemed to be a good description of what was now called the "shoulder-hand syndrome". In Steinbrocker's original account of this syndrome in 1947, no mention was made of Charcot's contribution and in the Centennial year it might be thought interesting to bring this to the notice of Heberden members.

Double Charcot's Disease. By F. E. BRUCKNER (*Middlesex Hospital*): Neuroarthropathy was a well-known complication of tabes dorsalis, diabetic neuropathy, syringomyelia, and in children, of meningomyelocoele. Rarer causes were congenital insensitivity to pain, familial dysautonomia, hereditary sensory neuropathy, peripheral nerve injury, the neuropathy of leprosy, and spinal cord injury. A similar radiological picture was described by Alarcon-Segovia and others (1965) after intra-articular corticosteroid injection.

Five cases of Charcot-Marie-Tooth disease were described with arthropathic changes, varying from minor deformity and bony hypertrophy to the full-blown picture of Charcot's arthropathy.

Some Rum and Odd Arthropathies. By DR. DUDLEY HART (*London*): Prof. Bywaters had suggested in the past that at the Annual General Meeting of the Heberden Society, or the Yuletide orgy, there might be one paper a little less serious, while still containing some items of general interest; a break in the academic austerity of the rest of the meeting, a little divertissement between the more serious movements, as it were. This paper described Hart's Retropulsive Arthropathy (H.R.A.), Septic Spinal Polymyalgia (S.S.P.), various arthropathic drug disorders (V.A.D.D.), and some odd bits and pieces (O.B.P.). The whole thing might be called an Arthropathic Hors d'oeuvre (A.H.D.). Avanti!

Effect of Glyvenol (Ciba) on Pain and Capillary Resistance in Rheumatoid Arthritis. By W. C. DICK, G. M. CUNNINGHAM, M. K. JASANI, W. W. DOWNIE, and K. WHALEY (*Glasgow*): This paper was published in the *Annals* (1969), 28, 187.

Azathioprine: a Controlled, Double-blind Trial in Rheumatoid Arthritis. By C. G. BARNES, H. L. F. CURREY, J. F. DUNNE, B. HAZLEMAN, R. M. MASON, I. D. STRICKLAND (*London Hospital*): The ability of azathioprine to lower the corticosteroid requirements of rheumatoid patients was tested under double-blind conditions against placebo.

49 patients (mean age 53 years) had "definite" rheumatoid arthritis with positive serological tests for rheumatoid factor. All had been taking corticosteroids for at least 6 months and the minimum prednisolone requirement of each patient had been established and maintained for at least 2 months. This dose ranged from 5 to 20 mg. prednisolone per day (mean: 11.1 mg.).

Patients then received either azathioprine 2.5 mg./kg. daily or matching placebo tablets. Treatments were allocated randomly; assessments were made under double-blind conditions, and the results were analysed sequentially. The mean steroid requirements after one year of continuous treatment had fallen in the azathioprine group by 4.2 mg./day and in the placebo group by 0.7 mg./day. This difference is significant at the 5 per cent. level.

Of fourteen patients withdrawn from the trial, five were taking azathioprine. In one the white cell count dropped below 3,500 cells/c.mm.; one developed a macrocytic blood picture; another showed both these abnormalities and a fourth developed a rash. These abnormalities reversed rapidly when the drug was withdrawn. The fifth patient had an exacerbation of rheumatoid arthritis two months after starting azathioprine.

Analysis of monthly blood counts among the patients taking azathioprine showed a mean fall in the total white cell count of 2,500 cells/c.mm. (S.D. $\pm$ 2,600) at one year involving both the neutrophil and lymphocyte series.