or radiological changes were presented. Two had been previously diagnosed as "neurotic" and one as possible rheumatoid arthritis.

Arthroscopy and synovial biopsy in these subjects was normal. Measurements of the intra-articular pressures in the knee with varying volumes of simulating effusion showed that the elastance or pressure change for unit change in volume was considerably higher in normal subjects.

It is suggested that in this syndrome there is an abnormality of the joint lining, producing a specific physical change of increased stiffness. Use of the joint causes abnormal tensions in the tissue and recurrent pain.

Discussion.—Prof. E. G. L. Bywaters (Taplow): It is very interesting that similar rheumatic symptoms appear to occur both in hypermobile and in hypomobile patients. I do not see why this result should not be obtained from subjects with abnormally small joints. What happens if you take a child's joint and put fluid into it? Would you not get the same type of curve?

Dr. Jayson: We have not examined joints from any children. The youngest person examined was aged 18. The normal controls were of all sizes, shapes, and nationalities, and the smallest of them gave the highest pressures, but these were considerably lower than those recorded in our three patients.

Prof. E. G. L. Bywaters (Taplow): If you have a very small joint space, say a rheumatoid joint, which is fibrosed until there is only a few ml. of space left, would one not get curves like those you have shown?

Dr. Jayson: One would, but in these three patients there was no evidence of objective disease. There may be many causes for this specific physical change in the joint capsule, but the end-result is increased joint elastance. I suggest that this change is responsible for arthralgia in this condition and also in many types of arthritis.

Dr. J. Ball (Manchester): Would variation in permeability affect results? If there was a low permeability in the joint membrane or joint capsule, then the fluid might accumulate, and vice versa.

Dr. Jayson: At the end of the experiment we aspirate all fluid from the joint and expect to obtain all of it back without significant loss.

Dr. J. T. Scott (London): Was there any clinical pattern in the arthralgia which might enable you in the future to suspect its presence from the history?

Dr. Jayson: The pattern was that of arthralgia present mainly in the large but also in the small joints. I think it would be difficult to define a pattern because I suspect that many conditions might lead to the end-result of stiffening of the capsule but with no residual clinical changes in the joint itself. The patient who had this as a result of rheumatoid arthritis might show more of these symptoms in the small joints of the hands than other patients who might show only degenerative changes in the capsule.

Dr. V. Wright (Leeds): Have you had the opportunity to analyse the first part of the curves, which is probably the most meaningful physiologically. Our work with skin suggests that the initial part may well be due to elastin rather than collagen. I was not quite clear from your slide about diseased patients, how you interpreted the different curves? Did you think there were differences in elastin or did you think there were differences in collagen? Finally, for the sake of accuracy, I think we should describe your findings as intracapsular pressure and not intra-articular pressure; the former is 15-20 lb./sq. in. compared with a pressure of 400 lb./sq. in. or more between the articular surfaces.

Dr. Jayson: I accept your point. The interpretation of the curves is rather difficult. It is possibly speculative to suggest that the empty joint is controlled by elastin and the distended joint by collagen, but the shapes of the curves fit in with this hypothesis and it does appear that there may be functional shortening of the unstretched length of collagen fibres. It would be possible to obtain pressure changes of this magnitude, but with a differently shaped pressure/volume curve, so that the unstretched length of the collagen fibres would be similar to that of the controls, so I think that we have demonstrated a real change here.

Dr. R. Harris (Buxton): Have pairs of joints been examined?

Dr. Jayson: Very similar results were obtained in all the normal controls, and repeated measurement in the same knee in two subjects and both knees of another produced similar results.

Extra-articular Calcification mimicking Acute Arthritis.

By Dr. A. J. Swannell and Dr. A. St. J. Dixon (Royal National Hospital for Rheumatic Diseases, Bath): Calcium deposition occurring outside the joint must be distinguished from calcification within the joint cartilage (chondrocalcinosis). Painter (1907) first described such deposits near the shoulder, and Sandström (1938) and Gondos (1957) observed them in tendons surrounding other joints. Sandström emphasized the disappearance or decrease of the deposit after the acute phase had resolved. Pinals and Short (1966) and McCarty and Gatter (1966) stressed the similarity to acute gout and Thompson, Ming Ting, Riggs, Finn, and Denning (1968) identified hydroxyapatite crystals from the calcific deposits—another point of difference from chondrocalcinosis.

We present eighteen patients who had radiologically demonstrable single or multiple calcific deposits, most of whom had normal serum uric acid, calcium, and phosphorus levels. All presented with acute pain and swelling and with limitation of movement of the underlying joint. The shoulder was affected in seventeen attacks in ten patients, and the wrist, fingers, and hips were involved once each in two patients. The great toe was involved once in four patients, with striking resemblance to acute podagra. Recurrence involving the same joint occurred in six out of the eighteen patients. In fifteen patients more than one joint was involved. Some patients with recurrent and typical painful episodes associated with radiologically apparent deposits also had
similarity episodes in which no radiological change could be demonstrated—presumably because the deposit was too small or the soft tissue too thick. Thus patients may exist who show similar syndromes with no x-ray signs.

A review of the literature has shown that no controlled trial of treatment has been performed in this condition and this is reflected by the diversity of recommended treatments. It is doubtful if any treatment greatly affects this condition where spontaneous recovery with no permanent limitation of joint movement is the rule.

Analysis of a resistant calcific deposit removed from the supraspinatus tendon yielded a powdered white amorphous mass that was radio-opaque. No crystals were seen under polarized light microscopy and chemical analysis has shown it to consist mainly of calcium, phosphate, and carbonate.

X-ray diffraction studies by the kind co-operation of Dr. J. F. Underwood from Bath University have shown this compound to be hydroxyapatite.

Whether this material is the result of tissue degeneration and subsequently excites an inflammatory response by a foreign body type of reaction is still debated. Clinically the disease bears a striking resemblance to acute gout and could well be the result of a crystal-induced inflammation.

**Discussion.**—**Dr. J. A. Mathews (London):** It may be relevant to mention a patient whom I saw several years ago at The London Hospital under the care of Dr. Michael Mason. She was a middle-aged lady who had what we called multiple calcific periarthritis, whose symptoms closely resembled those that have just been described. She had pain affecting both hips and shoulders and pararticular calcification, and we wondered whether there was a crystal synovitis. We tried to aspirate fluid from the shoulder joint affected at the time, but unfortunately only obtained a sample by irrigation. Nevertheless we sent the fluid to Mr. K. V. Swettemham at The London Hospital for crystal examination. He reported finding, amongst other things, white cells and crystals with the optical appearance of hydroxyapatite. We felt we could not establish hydroxyapatite crystals as being the cause of this apparent shoulder arthritis as the fluid collected was by irrigation and not direct aspiration, and we do not know what the diluting effect of the saline on the appearances would have been; however we thought at the time that these findings supported the idea that this syndrome was another example of crystal synovitis.

**Dr. H. L. F. Currey (London):** I wonder whether any of your patients had renal failure? We have seen calcific deposits in patients with renal failure, maintained on chronic dialysis, who presented this picture, and Decker (1965) has described this. It is now realized that this is a not uncommon complication in patients who are perhaps inadequately maintained on chronic haemodialysis after their kidneys have failed.

**Dr. Swanell:** Most of our patients were young healthy subjects, with no clinical evidence of renal failure, but we did not go into the renal function studies in detail.

**Prof. E. G. L. Bywaters (Taplow):** Most people would agree that chondrocalcinosis is quite different from the picture presented here. I take it we would not expect to find pyrophosphate crystals in these deposits. You sampled one deposit and there must be many people who have sampled other deposits and they presumably consist of hydroxyapatite. As a rough rule for everyday practice, if you see crystals they are pyrophosphate, whereas the hydroxyapatite deposits do not appear to be birefringent. This is useful clinically. A slide which I used in my talk to the Heberden Society on the subject of tendinous calcifications in 1962 (Annals, 21, 304) shows the multiple areas which are affected in some patients. This patient had nineteen sites affected altogether (elbows, ankles, knees, wrists, hands, shoulders, and so on), and even in the hand itself you can see an enormous number of small deposits presumably somewhere near tendon insertions. Is this how you interpret the radiological pathology? I do not suppose anyone has ever seen the pathological details of where these things occur.

**Dr. J. Ball (Manchester):** We had one case of periarticular calcification, and the calcific foci were in the capsule. I agree with Prof. Bywaters, if one can see a crystal form, it cannot be hydroxyapatite because this is amorphous in the light microscope.

**References**


**Joint Irritation in Rheumatoid Arthritis: A Controlled Trial.**—**By D. J. Lindsay, E. F. J. Ring, P. F. J. Coorey and M. I. V. Jayson (Royal National Hospital for Rheumatic Diseases, Bath):** Uncontrolled observations performed on patients after arthroscopy have suggested that joint irrigation is beneficial to the rheumatoid knee. A double-blind controlled clinical trial was devised to test joint aspiration with lavage against joint aspiration alone in order to assess whether lavage was the more effective form of treatment for out-patients. The trial was carried out on rheumatoid patients with knee effusions, all of them in the definite or classical RA category. Of the 24 patients who began the trial, ten completed in the lavage group and nine in the control group. There were no complications from haemarthrosis or joint infection.

A No. 2 Braun cannula (internal diameter 1.45 mm.) was used to aspirate the joint fluid. Irrigation was performed using 4–3 per cent. dextrose, 1–18 per cent. saline solution, 50-ml. volumes being flushed in and out of the joint to a total of 500 ml. The patients were evaluated clinically for pain, stiffness, and range of movement. Twenty-minute rewarming curves of the patella skin temperature were plotted for each patient. Viscosity and fibrinogen were estimated in plasma and synovial fluid. In addition synovial fluid levels of rheumatoid...
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