similar 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 par-articular 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. Swettenham 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. SWANNELL: 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 peri-articular 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.

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Joint Irrigation 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

factor, LDH cells, and protein were investigated. The clinical evaluation and temperature measurements were performed before treatment and on five occasions during the 6-week follow-up, laboratory estimations being done at the beginning and end of the trial.

From the above investigations we concluded that there was no difference in the temperature curves in either group and this was confirmed statistically. Pain improved to a statistically significant degree in both groups. Joint mobility—measured in terms of knee flexions per 30 sec.—showed a statistically significant improvement at all follow-up stages in the lavage patients but not in the controls; testing between the two groups showed no significant difference. Similarly, as regards the range of movement, there was no difference between the two groups. The laboratory investigations were also inconclusive. Viscosity increased in both groups but not significantly more in the lavage patients.

Although there is benefit from joint irrigation, the statistical difference between the treated and control groups is such that there would appear to be no indication to adopt joint lavage as a routine out-patient procedure.

Discussion.—**DR. A. J. POPERT (Droitwich):** In the last 5 years I have carried out lavage in about sixty patients and have formed some impression of the usefulness of this procedure. It has always seemed to me that the crux of treatment in rheumatoid arthritis in a patient whose joints are radiologically normal, should be to restore the joint to normality and keep it so. I think it is impossible for a joint which contains "rice grains" ever to return to normal; such a joint is likely steadily to degenerate. If you have a patient whose joint cartilage is radiologically normal but whose disease is active and you use lavage as part of a comprehensive regime aiming at inducing a remission of the disease, then to clear the joint of this debris must, it seems to me, help to protect that joint from future degeneration.

DR. F. M. ANDREWS (Reading): I have been using joint lavage procedures somewhat similar to Dr. Popert's. I have been impressed by the extraordinary quantity of fibrinous material that one could in fact obtain from joints by such techniques, although radiographically they may well be normal. I have recently obtained 200 g. from one particular knee that was chronically swollen and similar quantities from three others. I cannot but agree with Dr. Popert that it must in fact be a good thing to be rid of these deposits even if the particular trial described does not reveal by the techniques used, any particular advantage to the lavage treated group.

DR. LINDSAY: May I ask Dr. Andrews and Dr. Popert what size needles they were using? Clearly one cannot extract a large amount of fibrinous material through a small bore needle. I should like to know if their patients were aspirated as out-patients or under theatre conditions.

DR. A. J. POPERT (Droitwich): These were all hospital in-patients. The size of the needle varies according to the size of the debris that one seems to be dealing with. If you think there is obviously debris in the joint and aspiration yields none, you use a larger needle up to a certain limit to see if you can obtain debris.

DR. F. M. ANDREWS (Reading): These procedures were carried out on in-patients. It so happens that there is a

surgeon who shares the ward with me who does a tremendous amount of tapping hydroceles and I used his rather horrid-looking apparatus. Dr. Popert's grains of rice would flow through these wide-bore cannulae with the greatest of ease. I am sure this is why I can get such large quantities. I have done this lavage over a period of time in a closed drainage system using the pre-sterilized drainage bags which are now freely available.

PROF. J. J. R. DUTHIE (Edinburgh): I am against the idea that just because a joint has fluid or debris in it one must clean it out. I have on several occasions seen joints, which might appear suitable for drainage, settle down spontaneously.

I think that the trial reported here shows lavage to be of no benefit.

Rheumatoid Heart Disease. By JULIAN KIRK and JOHN COSH (*Royal National Hospital for Rheumatic Diseases, Bath*): Heart disease in a patient with rheumatoid arthritis is usually due to coincidental and unrelated pathology. However, true rheumatoid heart disease exists in two main forms, although its identification may be uncertain without knowledge of the morbid anatomy.

The main specific form is a granuloma, having some similarities to the classical subcutaneous nodule, which may develop in valves, myocardium, or epicardium. Aortic or mitral valve damage or impairment of conduction may result. Such lesions are found in patients with chronic sero-positive rheumatoid disease of some years' duration.

The other main form, pericarditis, is commoner, but of no specific histology. Although described in 30 per cent. of rheumatoid patients coming to autopsy, pericarditis is not often noted clinically as its manifestations are slight. It may arise at any stage of rheumatoid disease, is often symptomless, and may accompany rheumatoid pleurisy. If an effusion is formed, it is usually not big, and *tamponnade* is rare. It results in partial or even total obliteration of the pericardial sac by light fibrous adhesions, and occasionally leads to frank constrictive pericarditis requiring surgical relief; examples of rheumatoid pericarditis have been studied, ten of them found during a careful review of 100 consecutively admitted patients with chronic rheumatoid arthritis.

Discussion.—**DR. J. H. GLYN (London):** I had a very interesting case which was written up in the *British Medical Journal* (1963) of a man who came into the chest wards of our hospital with a spontaneous pneumothorax and who subsequently developed constrictive pericarditis which needed surgical resection. He came under my care because very shortly afterwards he developed signs of rheumatoid arthritis, and his latex-fixation test was strongly positive from the earliest stage. I thought it was a unique sequence of events, but another letter came in from Australia recording a similar case. Recently I was asked to write an annotation in the *British Medical Journal*, and in the last 10 years there have been reported twenty or thirty cases of rheumatoid arthritis, presenting identically as constrictive pericarditis, followed by sero-