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-

positive rheumatoid disease. So it may be worth while considering this as an atypical mode of presentation of rheumatoid arthritis.

DR. COSH: I did not realize that quite so many examples had been recorded of rheumatoid arthritis presenting with constrictive pericarditis. One cannot help wondering whether the cardiologists concerned were aware of joint involvement in its earliest stages.

DR. T. BITTER (*Los Angeles*): You did not mention one condition which can mimic pericarditis very closely and is seen much more frequently in patients with rheumatoid arthritis more frequently than rheumatoid nodules in the heart or clinically evident pericarditis, that is amyloidosis. As there is secondary amyloidosis in close to one such patient out of eight, have you done rectal biopsies in the patients of your series, to exclude this possibility? Other authors have emphasized the incidence of rheumatic fever in rheumatoid arthritis and I wondered about the possibility of rheumatic fever in this series.

DR. COSH: My impression is that rheumatic fever and rheumatic heart disease are no commoner in rheumatoid patients than in the non-rheumatoid population. Prof. Bywaters came to this conclusion when he reviewed the subject in 1950.

As for amyloid, we did not do rectal biopsies, and we accepted that the pericarditis was due to the rheumatoid process, particularly as there was so often pleurisy as well, so that it did not seem necessary to look further. Thank you, however, for pointing this out.

PROF. E. G. L. BYWATERS (*Taplow*): Surely rheumatoid patients with amyloid very seldom show heart lesions (apart from senile amyloid) and it is in primary amyloidosis alone that the heart is involved and constricted. We see a good many rheumatoid patients with amyloid and they have not been troubled by cardiac deposits.

I also want to comment on the very interesting case of the woman in whom you actually saw the development of constrictive pericarditis. I have seen seven cases (described in my Croonian Lecture this year), but we were not able to time the development. Could you tell us more about this; how does it develop and how long does it take?

DR. COSH: I did not summarize the course of that particular patient on this occasion, but it is described in the *Quarterly Journal of Medicine* (Kirk and Cosh 1969). The patient was under observation for 7 years, coming to the clinic a few months after the onset at age 53. The arthritis was sero-positive and became generalized and severe, and she was started on steroids a year after the onset. After 5 years on steroids she developed a gastric ulcer, and later had a haematemesis. While in hospital recovering from this, she developed pericarditis with effusion and a right-sided pleural effusion which was aspirated. Later a small left pleural effusion formed. Pericardial friction and effusion persisted, but the pericardium was not aspirated. She improved, but within 7 months of the discovery of pericarditis she developed leg oedema. When this was followed by hepatomegaly and jugular venous engorgement, constrictive pericarditis was recognized, and this was confirmed by cardiac catheterization. A surgical resection of the thickened pericardium was performed, but the patient unfortunately died the day after operation.

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GAIRDNER FOUNDATION AWARDS, 1969

Three Canadian and five United States medical scientists will share in this year's Gairdner Foundation awards.

They include Dr. R. B. Salter of Toronto, an

orthopaedic surgeon, for his work on congenital hip disease and the effect of various forces on growing joints. This is the first Gairdner Award in orthopaedic surgery.

RHEUMATOLOGY—REHABILITATION FELLOWSHIP

A new Rheumatology-Rehabilitation Fellowship programme has begun at Montefiore Hospital, New York City. Clinical training is provided through the direct management of in-patients and out-patients with arthritis and other rheumatic diseases under the supervision of specialists in Rheumatology and Rehabilitation Medicine. Practical experience is provided in the treatment and preventive management of both acute and chronic states with opportunities to become proficient in the techniques of splinting, bracing, and the use of physical

modalities. One year of training in Internal Medicine or Physical Medicine and Rehabilitation is required. The programme is equivalent to one year of medical residency training (American Board of Internal Medicine). Fellowships may be taken up in January or July, 1970.

Full particulars may be obtained from Mr. Nathan Zamoff, Education Co-ordinator, Department of Rehabilitation Medicine, Montefiore Hospital and Medical Center, 111 East 210th Street, Bronx, New York 10467, U.S.A.

ROYAL COLLEGE OF PHYSICIANS

At a comitia held on October 30, 1969, the Editor of the *Annals of the Rheumatic Diseases*, Dr. W. S. C. Copeman, was elected Vice-President of the Royal College of Physicians of London.