higher water content and galactosamine-glucosamine molar ratio than the control tissue. Periarticular osteophyte formation was also evident very early in the disease process.

Focal sites of tibial cartilage were extracted sequentially with (a) 0.15 mol/l sodium acetate, (b) mol/l calcium chloride, and (c) 4 mol/l guanidinium chloride. A larger proportion of the total proteoglycans was extracted from the cartilage of operated joints than from the corresponding controls and the proportion of the total proteoglycan in each extract changed in such a way as to suggest that the association of proteoglycans with collagen was reduced; thus (a) contained more and (c) less than controls (Table I). These changes, which preceded the appearance of fibrillation as detected by indian ink staining, were confined to area A of the tibial cartilage in the dog killed 3 weeks after surgery, but was evident throughout the whole tibial plateau of the operated joint of the dog killed 48 weeks after surgery.

The purified proteoglycans extracted from osteoarthrotic cartilage had higher galactosamine-glucosamine molar ratios than those from control cartilage. In the cartilage residue this difference was less marked (Table II). Thus, proteoglycans extracted from osteoarthrotic compared with control cartilage contained more chondroitin sulphate. The results suggest that in response to mechanical stress due to the instability of the operated joint, the chondrocytes synthesize a different proteoglycan, relatively rich in chondroitin sulphate, which is less firmly associated with collagen than normal proteoglycans. This might account for the increased hydration of the tissue, because it would increase the swelling pressure of the proteoglycans in the tissue, since at this stage in the disease process the total amounts were unchanged. That the proteoglycans are important in stabilizing collagen fibres is consistent with the report that the shrinkage temperature of human articular cartilage collagen was closely correlated with the hexosamine content of the tissue (Herbage and others, 1972).

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- Graphic Illustration in Handbooks for Patients. J. M. H. MOLL, V. WRIGHT, M. R. JEFFREY, J. D. GOODE, AND P.M. HUMBERSTONE (Sheffield Centre for the Investigation and Treatment of Rheumatic Diseases, and the Rheumatism Research Unit, University Department of Medicine, Leeds)
- In a previous study of the standard Arthritis and Rheumatism Council 'Handbook on Gout', we found the 9-cartoon booklet to be a satisfactory doctor-patient communication aid (Moll and Wright, 1972). An extension of this study has been undertaken to examine the communication value of increasing the number of illustrations

in such booklets. Two variants of the standard 'Handbook on Gout' were printed specifically for the study. One contained text with 89 cartoons, and the other was purely textual. The text in each booklet was the same as that of the standard handbook.

Fifty patients with gout were studied. On a random basis, 28 were given the booklet with 89 cartoons and 22 the booklet without cartoons. At routine follow-up each patient completed a multichoice questionnaire based on the main points in the handbook. No prewarning of the test was given. The mean overall scores obtained by patients were compared with the mean score of patients tested in the previous study using the standard booklet. Text with 89 cartoons 65.5%; text without cartoons 67.1%; text with 9 cartoons (standard booklet) 70%.

Comparing results obtained in response to individual questions (as opposed to the overall test score), in only one out of 14 questions (that based on 'Lifelong and Preventive Treatment') was there a statistical difference ($P < 0.05$) between the two groups. Text with cartoon 78.7%; text without cartoon 57.1%.

The failure of the illustrated booklet to show better overall test results is not yet understood, but may arise from a 'knowledge saturation effect' inevitable in patients reading material devoted to their own disease. Technical problems to do with presentation of data may also be important.

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Osteoporosis in Patients with Rheumatoid Arthritis. A. C. KENNEDY, D. A. SMITH, G. GREY, M. K. JASANI, and W. W. BUCHANAN (*The Centre for Rheumatic Diseases, University Department of Medicine, Royal Infirmary, Glasgow; the Bone Metabolism Research Unit, University Department of Medicine, Western Infirmary, Glasgow*)

361 patients with 'definite' or 'classical' rheumatoid arthritis were studied, of whom 201 were female and 150 male. The metacarpal indices and femoral indices (Barnett and Nordin, 1960) from these patients were compared with those derived from 119 female and 76 male controls. The source and characteristics of these control subjects have been reported elsewhere (Smith, 1971).

The results of this study show that both male and female patients with rheumatoid arthritis show a significant bone loss in the femur compared with the loss of bone seen in normal subjects with increasing age. There was no evidence to suggest that corticosteroid therapy caused a greater loss of bone from the femur than would have occurred as a result of the rheumatoid arthritis alone. There was a statistically significant relation between the duration of the rheumatoid arthritis and femoral bone loss in women over the age of 45 years ($P < 0.001$) whether or not they had received corticosteroid therapy. There was also a significant relationship between femoral bone loss and the duration of corticosteroid therapy in both male and female patients over the age of 45 years. However, when a correlation excluding disease duration was conducted there was no residual effect of corticosteroid therapy. The metacarpal and femoral indices of the patients were significantly related in both male and female groups ($P < 0.001$).

It is concluded that osteoporosis is a generalized phenomenon in rheumatoid arthritis, relating more to the duration of the arthritis than to the presence of treatment with corticosteroid therapy.

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Serum Uric Acid—Its Relationship to Lean Body Mass, Sex, Plasma Urea, Intracellular Potassium and Packed Cell Volume in a Normal Population Group. A. C. KENNEDY, J. BRENNAN, J. ANDERSON, P. BROOKS, W. W. BUCHANAN, AND W. C. DICK (*The Centre for Rheumatic Diseases, the University Department of Medicine, Royal Infirmary, Glasgow, and the Department of Biomathematics, Oxford*)

Fifty-seven normal healthy subjects were studied of whom 30 were female (mean age 27.6 years $\pm$ 1.87, SEM, range 18–52 years) and 27 were male (mean age 29.7 years $\pm$ 1.9 SEM, range 21–57 years). Each subject was receiving a normal solid and fluid diet and none had taken alcohol or any drug within 24 hours of this study. Height, (cm) and weight (kg) were established and a sample of serum obtained for uric acid estimation. Total body potassium (TBK) was measured in each subject using a mobile whole body monitor with a sodium iodide detector and shadow shield protection (Boddy and others, 1971). Lean body mass (LBM) was derived from the formula of Hume and Weyers (1971) and also from the TBK content (Boddy, and others, 1972).

In 14 male and 11 female subjects a further sample of venous blood was obtained for the determination of plasma potassium, intrared cell potassium, packed cell volume, and urea. A regression analysis (Table) of Serum uric acid upon each of these parameters showed no relationship to plasma or intrared cell potassium or to packed cell volume but a significant ($P < 0.001$) relationship to height, weight (whether derived from TBK or Hume-Weyers formula), and plasma urea. The most marked relationship, however, was to sex and the results of a further regression analysis with adjustment for sex gave evidence that the concentration of serum uric acid is influenced predominantly by the sex of the subject but that a series of secondary factors contribute and summate within either the female or male group.

The most striking results from this study which has not to our knowledge been reported previously is the relationship between serum uric acid and plasma urea. A result which invites a great deal of interesting speculation.

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Assessment of Activity in SLE: A Clinical and Serological Study. J. P. EDMONDS, C. BRUNEAU, AND G. R. V. HUGHES (*Department of Medicine, Royal Postgraduate Medical School, London W12*)

The entity of SLE may consist of groups of patients with distinct disease patterns in which involvement is limited to certain systems. To investigate this possibility, 20 patients were admitted for a 48-hour period for full clinical and laboratory assessment, including renal and respiratory function, EEG, and fluorescein retinal angiography. 19 patients were female and the average age was 33 years. Four patients did not fulfil the ARA criteria for the diagnosis of SLE.

The frequency of disease manifestations was similar to that reported by Dubois with the exception of renal involvement which occurred in only 25% of our group. While only one of the 20 patients had over 3.5 g proteinuria, 6 had proteinuria of >0.5 g daily, urinary red cells or a creatinine clearance of <60 ml/min. Five of the 6 patients on whom a renal biopsy was performed showed changes on light microscopy. Nine patients had respiratory symptoms at the time of the study but respiratory function tests were abnormal in 16, the most common abnormality being diffusion and restrictive defects. Three patients had central nervous system symptoms when studied and a further 5 had previously been symptomatic: of these 8 patients, 6 had severe headaches, 5 had an abnormal EEG, and 6 showed leakage of dye on fluorescein angiography; none had an abnormal brain scan. Of 11 patients without apparent CNS involvement, one had headache and 2 had an abnormal EEG and leakage of dye on fluorescein angiography. Twelve patients were considered to have active lupus: their mean DNA binding capacity was 70% (normal 0–30%) with a mean serum C3 level of 67% (normal $>70\%$); of the 8 patients with inactive disease the mean DNA binding capacity was 67% and the mean

Table Separate regressions of uric acid

Independent variable	Slope β	SE (β)	df	t	P
Height (cm)	0.064	0.016	55	3.93	<0.001
Weight (kg)	0.047	0.013	55	3.77	<0.001
Sex	1.898	0.222	55	8.50	<0.001
LBM	0.113	0.018	55	6.34	<0.001
TBK	0.027	0.005	55	5.93	<0.001
Ht, wt, age K (predicted)	0.029	0.005	55	5.78	<0.001
Ht, age K (predicted)	0.028	0.005	55	5.22	<0.001
Ht, wt, LBM (Hume-Weyers formula)	0.095	0.016	55	6.06	<0.001
Urea	0.087	0.021	55	4.17	<0.001
Plasma K	0.372	0.446	55	0.83	NS
Intracellular K (male)	0.068	0.095	12	0.72	NS
Intracellular K (female)	0.002	0.061	9	0.04	NS
Packed cell vol (male)	0.227	0.119	12	1.92	NS
Packed cell vol (female)	0.054	0.116	9	0.47	NS