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.

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.

References

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 Biochemistry, Oxford)

Fifty-seven normal healthy subjects were studied of whom 30 were female (mean age 27·6 years ± 1·87, SEM, range 18–52 years) and 27 were male (mean age 29·7 years ± 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 obtained 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, intracellular 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 intracellular potassium or to packed cell volume but a significant ($P < 0·001$) relationship to height, weight (whether derived from TBK or Hume-Weyer 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.

### Table Separate regressions of uric acid

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Slope $\beta$</th>
<th>SE ($\beta$)</th>
<th>df</th>
<th>$t$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>0·064</td>
<td>0·016</td>
<td>55</td>
<td>3·93</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>0·047</td>
<td>0·013</td>
<td>55</td>
<td>3·77</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Sex</td>
<td>1·896</td>
<td>0·222</td>
<td>55</td>
<td>8·50</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>LBM</td>
<td>0·113</td>
<td>0·018</td>
<td>55</td>
<td>6·34</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>TBK</td>
<td>0·027</td>
<td>0·005</td>
<td>55</td>
<td>5·93</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Ht, wt, age K (predicted)</td>
<td>0·029</td>
<td>0·005</td>
<td>55</td>
<td>5·76</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Ht, age K (predicted)</td>
<td>0·028</td>
<td>0·005</td>
<td>55</td>
<td>5·22</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Ratio, cell, LBM (Hume-Weyer formula)</td>
<td>0·095</td>
<td>0·021</td>
<td>55</td>
<td>4·17</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Urea</td>
<td>0·087</td>
<td>0·021</td>
<td>55</td>
<td>4·17</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Plasma K</td>
<td>0·372</td>
<td>0·046</td>
<td>55</td>
<td>8·33</td>
<td>&lt; 0·001</td>
</tr>
<tr>
<td>Intracellular K (male)</td>
<td>0·068</td>
<td>0·095</td>
<td>12</td>
<td>0·72</td>
<td>NS</td>
</tr>
<tr>
<td>Intracellular K (female)</td>
<td>0·002</td>
<td>0·061</td>
<td>9</td>
<td>0·04</td>
<td>NS</td>
</tr>
<tr>
<td>Packed cell vol (male)</td>
<td>0·227</td>
<td>0·119</td>
<td>12</td>
<td>1·92</td>
<td>NS</td>
</tr>
<tr>
<td>Packed cell vol (female)</td>
<td>0·054</td>
<td>0·116</td>
<td>9</td>
<td>0·47</td>
<td>NS</td>
</tr>
</tbody>
</table>

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 fulfill 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.
serum C3 level was 83%. All 4 patients who failed to
fulfil the A.R.A. criteria but had a positive ANF and
raised DNA binding capacity were found to have clinically
unnoticed abnormalities on investigation. It is concluded that respiratory function tests and the
EEG and retinal fluorescein angiography are valuable
laboratory aids in assessing the extent of disease involve-
ment in SLE. These tests, taken together with serological
markers, may help to detect a more benign group of SLE
patients. In the 20 patients studied no clear disease patterns
of lupus were shown.

Penicillamine Nephropathy. D. R. SWINSON, E. B. D.
HAMILTON, and F. E. DISCHE (Department of Rheuma-
tology and Pathology, King’s College Hospital, London)
Penicillamine produces benefit in rheumatoid arthritis and is
being increasingly used for this condition. A proportion
of patients develop side effects including proteinuria. In a
series of 106 patients so far treated, nine patients have
developed proteinuria. Four developed proteinuria in
excess of 6 g/day, one with the nephrotic syndrome, and
these patients were biopsied. Duration of treatment was 5
months in one patient and 9–13 months in the other eight.
Creatinine clearance values were normal. The proteinuria
has taken up 10 months to disappear in two of the patients.
By light microscopy, the glomeruli showed minimal capil-
lar thickening and a slight mesangial matrix increase, or
were indistinguishable from normal. Immunofluorescent
examination showed granular deposits of IgG and C3
component of complement in the glomerular capillary walls in all four cases but failed to show the presence of
Clq, in contrast to the presence of this component in four
out of six patients with idiopathic membranous glomeru-
lar nephropathy and one patient with SLE membranous
glomerular nephropathy. Electron microscopy showed
subepithelial electron dense deposits and fusion of epithe-

dial foot cell processes. The findings, therefore, are
similar to those of early idiopathic and SLE membranous
glomerulonephritis except for the relative absence of Clq
and C4. The lesions are presumed to be due to immune
complex localization (Germuth and Rodriguez, 1973)
and the absence of Clq may indicate that complement
activation by the classical sequence has declined in the
interval between the end of treatment with penicillamine
and biopsy.

Reference
GERMUTH, F. G., AND RODRIGUEZ, E. (1973) In ‘Immunopathology
of the Renal Glomerulus’. Little, Brown, Boston

Antibody-Mediated Leucocyte Cytotoxicity to Chang
Human Liver Cells in Rheumatoid Arthritis and Other
Diseases. G. S. PANAYI (Guy’s Arthritis Research Unit
and Department of Medicine, Guy’s Hospital, London)
To be published in full in the Annals.

Notes

Robecchi Prize, 1975

The Robecchi Prize for 1975, founded to commemorate the late Professor Alessandro Robecchi, an outstand-
ing rheumatologist, has this year been awarded equally to a group of research workers at the Westminster
Hospital led by Dr. D. A. Brewerton and Dr. D. C. O. James, and to a group in Los Angeles represented by
Dr. R. Bluestone. The prize is for the original and simultaneous discovery of the association of the histo-
compatibility antigen HL-A 27 and ankylosing spondylitis and the subsequent discoveries which have flowed
from this. The value of the prize is 1500000 lire.

Royal Microscopical Society International Symposia and
Exhibition (Micro ’76)

The symposia and exhibition will be held from 13–17 September 1976 at the Wembley Conference Centre,
Wembley, London, and will include a one-day Symposium on ‘Microscopy in Arthritis’. The emphasis in this
Symposium will be placed both on the technology of microscopy and upon the nature of the pathological
problems studied.

Further information may be obtained from the Secretary, Royal Microscopical Society, 37/38 St. Clements,
Oxford OX4 1AJ, or from Professor D. L. Gardner, Institute of Pathology, The Queen’s University of Belfast,
Grosvenor Road, Belfast BT12 6BA.
Assessment of activity in SLE: a clinical and serological study.

J P Edmonds, C Bruneau and G R Hughes

Ann Rheum Dis 1975 34: 543-544
doi: 10.1136/ard.34.6.543-b

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