Renal impairment associated with non-steroidal anti-inflammatory drugs

Sir, The study by Unsworth et al.\(^1\) would appear to have methodological deficiencies which limit both its conclusions and its applicability to rheumatic patients taking non-steroidal anti-inflammatory drugs (NSAIDs). These include the reason for choosing to discontinue the medication in a particular individual; the lack of information about the reason for admission of the patients, and particularly their state of hydration, and stability of renal function compared with previous estimations; the variable time before discharge or reintroduction of NSAIDs, which results in distortion of the slopes in Fig. 2, in which the serum creatinine and urea graphs include results of patients 23–27 who are not referred to anywhere else in the article; the reliance on creatinine clearance as a measure of renal function, with its inherent inaccuracies (two validations with CrEDTA out of 22 estimations are not sufficient reassurance); and the lack of randomisation.

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Reference

Sir. Perhaps we did not clearly explain some of the points that Dr Gow raises in his letter: NSAID ingestion is increasingly recognised as a cause of renal failure; our patients show reversible renal suppression. We feel that this observation is applicable to all patients taking these drugs. Our patients were admitted sequentially from a waiting list for treatment or assessment of their rheumatological condition(s), and this is of no relevance to the ensuing discussion. They volunteered to stop NSAIDs to assess the effect of withdrawal of these drugs on renal function. That their length of stay and tolerance to NSAID removal would be variable is obvious. We clearly state that, unless described in the text, all recognised pre-dispositions to NSAID nephrotoxicity had been excluded, and dehydration is referenced in this context. The inclusion of patients 23–27 is an illustrative error and has no bearing on this or previous discussions; they form part of a different study and our statistics do not include these data. Creatinine clearances remain the best method commonly available for the measurement of renal function. The fact that they change significantly and consistently is the message that we have tried to get across. Our aim in publishing this small study, which poses more questions than it answers, is to alert NSAID prescribers to possibly common side effects, and we feel that renal monitoring should be considered in all patients receiving these drugs.

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Rheumatoid factor in patients with systemic lupus erythematosus

Sir. In the report by Helin et al.\(^1\) in the Annals\(^1\) the authors suggest that rheumatoid factor (RF) protects against nephropathy in patients with systemic lupus erythematosus (SLE).

We have reviewed our series of 78 patients with SLE fulfilling the criteria of the above report for evaluating nephropathy and assaying RF. The Waaler-Rose sensitised sheep cell agglutination test was used to measure RF, with titres equal or greater than 1/64 considered positive. Both groups with and without renal disease were comparable for age and sex. A renal biopsy was performed in 38 out of 40 patients with nephropathy. The results are shown in Table 1.

Table 1 Rheumatoid factor and nephropathy in SLE*

<table>
<thead>
<tr>
<th>RF (+)</th>
<th>With nephropathy</th>
<th>Without nephropathy</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>5</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>36</td>
<td>33</td>
<td>69</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>38</td>
<td>78</td>
</tr>
<tr>
<td>% RF (+)</td>
<td>10</td>
<td>13.1</td>
<td>11.5</td>
</tr>
</tbody>
</table>

*Numbers of patients are given.

Our results differ from those obtained by Helin et al.\(^1\), Davis and Bollet,\(^2\) and Hill et al.\(^3\) and agree with those obtained by Kantor et al.\(^4\) and Baldwin et al.\(^5\). We found no significant difference between the percentages of positive RF in the groups with or without nephropathy. In addition, in the four cases with renal disease and RF (+) the renal biopsy specimens showed severe morphological lesions (three cases of diffuse proliferative glomerulonephritis and one case of membranous glomerulonephritis).

The discrepancy between these studies does not permit definite conclusions to be reached about the role of RF in the aetio-pathogenesis of SLE nephropathy.

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877

Page 877

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