(1) Serum calcium, phosphate, and albumin concentrations were not given. This is an omission on our part which we failed to note on proof reading before submitting the letter to you. Please accept our apologies. The results were as follows: serum calcium (uncorrected) 4.07 mmol/l; phosphate 1.36 mmol/l; albumin 22.0 g/l.

(2) The red blood cell count was correctly reported as 4.1 x 10^{11}/l and not 4.1 x 10^{10}/l. The white cell count was not included in the script, but was 10.9 x 10^{9}/l.

(3) Our paragraph 4 states the diagnosis to be non-Hodgkin’s malignant lymphoma. The confusion over diagnosis arises because the title of the original letter by Mayne and Burch was “Hodgkin’s disease presenting with hypercalcaemia”, which was, therefore, logically used by the Journal as the heading for our letter.

experience the dichotomised prognostic index did not improve correct predictions, compared with predictions derived from lymph node status alone. A comparison of the effect of lymph node status alone, to Baak’s prognostic index, in a year survival curves, showed both virtually to overlap with each other.

There are two possible explanations for our inability to reproduce Baak’s results. On the one hand, the observed improvement by the dichotomised prognostic index in the prospective study is minor (an increase of 6.7% in accuracy) and therefore difficult to detect with a study of only 75 patients. The other possible explanation is that additional information has been introduced by our lack of experience with Baak’s techniques, a different population, different treatment protocols, and different follow up periods. Both possibilities suggest that the use of Baak’s prognostic index is not likely to have much impact in the ability to differentiate patients at greater risk.

**Prognostic index for breast carcinoma: a 22% improvement in the prediction of outcome?**

There is a significant arithmetical error in Baak et al’s data, leading them to conclude that, compared with using the presence of lymph node metastases as a classifying variable, their method performed more effectively in 22% of instances to predict the actual prognosis of their patients. The actual difference is 6.7%.

Baak’s prognostic index predicts accurately the outcome in 139 out of 195 patients (71.3%) of cases; lymph node status alone predicts accurately in 126 out of 195 (64.6%) of cases. The difference between the effectiveness of the two methods is thus 6.7%. The discrepancy of 6.7% vs 22% is explained as follows:

<table>
<thead>
<tr>
<th>Data from original paper</th>
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<tbody>
<tr>
<td>Baak’s prognostic index</td>
</tr>
<tr>
<td>-------------------------</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>&lt; 0.6</td>
</tr>
<tr>
<td>&gt; 0.6</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

21 + 7 = 28 cases where prediction by prognostic index is confirmed and prediction by lymph node status is contradicted.

2 + 13 = 15 cases where lymph node status correctly predicts outcome and the prediction by prognostic index is contradicted.

The number of cases where prognostic index and lymph node status indicate a different prognosis is indeed 23 + 20 = 43 cases (or 22%). The purpose of the comparison, however, is to prove the superiority of the prognostic index over the prediction by lymph node status alone: only an added 13 cases (28 - 15) or an added 6.7% are correctly predicted by the prognostic index, not 22%.

We applied Baak’s prognostic index to our own series of 75 patients with breast cancer known 10 year follow up. We have been particularly careful to count mitoses per 10 high power fields (400 x magnification), as recommended by the authors. In our

**BOOK REVIEWS**


This is the first in a series of books, the stated aims of which are to cover the biology of extracellular matrix components, their effects on gene expression, and their control by cytokines, and to bridge the gap between these basic biological aspects and their implications for disease processes. This first volume contains five chapters, devoted to immunomodulation, connective tissue turnover, animal models of connective tissue disease, non-invasive assays of organ fibrosis, evaluation of hepatic fibrosis by molecular hybridisation, and clinicopathological analysis of pulmonary fibrosis. As is evident, these form a pretty varied group and are unlikely to be of equal interest to the different possible audiences. The text is generally well written (with few typographical errors) and the illustrations are of reasonable quality. Given the rapidly changing nature of this field, it is disappointing that references rarely go beyond mid 1987. Because of its heterogeneous content, this book is unlikely to appeal to anyone but the collagen “fetishist”.

Perhaps the editor should consider devoting future volumes to single subject areas, such as normal biology, fibrosis, desmoplasia, neoplasia, and so on.

CDM FLETCHER


The book is divided into three: non-neoplastic conditions in seven chapters; thyroid tumours in nine; and special diagnostic techniques in three. In addition to detailed discussion of papillary and follicular lesions, medullary and anaplastic carcinoma and lymphomas, the material is specially organised for ease of reference. The chapter headings therefore include granulomas in the thyroid, lymphocytes in the thyroid, fibrosis,