Dr Ellison et al comment:

Dr Piette and colleagues make some valuable suggestions in their letter about our article. We were also keen to compare the presence of intramural platelet deposition and titres of antiphospholipid antibodies in our series of patients. Three of the six had died before antiphospholipid antibodies were assessed; however, and we could find no record of these tests in the case-notes of the other three. We were unable to trace any stored serum.

We would agree that a study of other vascular areas in the antiphospholipid syndrome would be interesting. Though difficult to substantiate or to quantify, our impression was that intramural platelet deposition was more readily found in the cerebral vasculature of patients with the longest histories of neuropsychiatric symptoms and the most deformed, thickened, small vessels.

Carcinoid pattern in adrenal phaeochromocytomas

In response to the paper by Harach and Bergholm, I would like to comment on a similar phenomenon that I have encountered in two adrenal phaeochromocytomas. One case was sporadic and the other was associated with multiple endocrine neoplasia type IIa (MEN IIa). The carcinoid areas seen microscopically were reminiscent of the classic mitigird pattern with packets of uniform cells. The tumour cells were smaller and less pleomorphic than the typical pleomorphic, polygonal chief cells of the usual phaeochromocytoma. These carcinoid foci were, however, minor histological components and both tumours had adjacent areas of typical phaeochromocytoma. The medullary carcinoma of the patient with MEN IIa, interestingly, did not share this carcinoid phenotype. The question of metastatic syncytial spread was entertained because of obvious areas of phaeochromocytoma and the characteristic clinical scenario. At the same time, it must be remembered that metastatic medullary thyroid carcinoma within an adrenal phaeochromocytoma has been described.1

Metastases aside, if one believes in the dispersed (diffuse) neuroendocrine system, it is not unexpected that overlaps in histological pattern will occur.2

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Relative friendly Death Certificates

I read Dr Slater's description of his audit of the wording of Death Certificates with interest, and I agree that many of the inaccuracies he identifies are reprehensible. I take a far less hawkish view than he does about the commonest inaccuracy, however, which is to quote the mode of dying qualified by an underlying cause; an unqualified mode of death, on the other hand, is quite obviously silly. General practitioners may have to counsel a bereaved family when the only information they have about the death of their loved one is a Death Certificate, and I do not hesitate to include a mode of dying if I think that it will help with this counselling by clarifying the sequence of events. Why should we believe that patients with “cardiac failure due to coronary atheroma” be deemed wrong when “myocardial infarction due to coronary atheroma” can be accepted? When I carry out a necropsy, I like to think that I can derive the greatest benefit for all concerned, including relatives, clinicians, and epidemiologists. I don't think the Office of Population Censuses and Surveys has any particular difficulty with a Death Certificate if I put in an extra line at the beginning which clarifies the mode of death, because it is the underlying cause of death which is selected.3 Excluding modes of death from Death Certificates is one counsel of perfection which I also choose to ignore.

While on the subject of counsels of perfection, Dr Slater might like to know that the literature contains many references4 about the poor correlation between the clinical and pathological diagnosis of terminal malignancy and necropsy findings. Most are much more informative than the one he cites.5

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Dr Slater comments:

I appreciate Dr Benbow's interest in my audit of wording inaccuracies in relation to death certification. I fully support Dr Benbow's view that histopathologists should be asked to look on all necropsies and, if possible, to achieve this by personal communication with general practitioners and, when appropriate, by spending time with relatives of the deceased. We find this is preferable to the necessary distillation of information from gleaning information from a somewhat "stark" and impersonal Death Certificate. I agree that the inclusion of a "mode of dying" in expert hands (such as Dr Benbow) does little harm. I am sure, however, that if such a policy was adopted by inexperienced doctors then mode of dying would quickly become acknowledged as a definitive cause of death. Perhaps we should also not forget that in mode and not mode of death that we are certifying.

I am also appreciative of Dr Benbow's comprehensive list of references relating to the poor correlation between the clinical diagnosis of terminal malignancy and necropsy findings. This in itself proved an interesting audit and I was relieved that my own references were only 10% deficient. I was saddened to see that Dr Benbow expressed no personal opinion on the term carcinomatosis.

Further to Dr Slater's informative paper on audit of death certification I would like to add our experience in this field. Since 1990 we have audited the accuracy of death certification in this hospital by comparing the cause of death as found at post mortem (COD) with the presumed cause of death as written on the death certificate (PCOD). A post mortem examination is requested on all hospital deaths in this institution; the overall rate in three years is 24.2%, excluding coroner's cases, and the PCOD is normally selected for post mortem examination. Accuracy of certification is scored 1-4: 1 = completely accurate; 2 = relatively accurate, the PCOD and COD match, but secondary causes are inaccurately assessed; by attempting to glean information from a somewhat not suitable and inaccurate where the PCOD may be mistaken for the COD, and 4 = completely inaccurate. The results are shown in the table.

<table>
<thead>
<tr>
<th>Score</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>27 (28%)</td>
<td>43 (43%)</td>
<td>7 (7%)</td>
<td>22 (22%)</td>
<td>99</td>
</tr>
<tr>
<td>1991</td>
<td>23 (50%)</td>
<td>29 (38%)</td>
<td>9 (12%)</td>
<td>15 (20%)</td>
<td>76</td>
</tr>
<tr>
<td>1992</td>
<td>18 (29%)</td>
<td>27 (43%)</td>
<td>6 (10%)</td>
<td>11 (18%)</td>
<td>62</td>
</tr>
</tbody>
</table>
The rates have remained relatively constant over the three year period. Results of post mortem examinations are presented at clinicopathology audit meetings, which are held at two weekly intervals. As the junior medical staff turnover averages six months, however, it may be that the benefits are felt in other hospitals. Unlike Dr Slater, we have not found cases which should have been referred to the Coroner.

We would agree that the accurate wording of death certificates is of paramount importance for stastical reasons and the future provision of health care services. Education is certainly necessary—we cannot say, in our experience, to have definitely made inroads into this problem through audit, but will continue to emphasise the importance of this subject to the medical staff responsible for writing certificates. As the medical students also attend the meetings perhaps we shall see improvements in future years.

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Post mortem sampling for biochemistry and toxicology

Dr Forrest is to be congratulated on his ACF broadsheet concerning the usefulness of post mortem sampling for biochemistry and toxicology,1 a much neglected subject. There is one assay not mentioned among the generally useless enzyme determinations and that is the gamma glutamyl transpeptidase (gGT). Over many years I have found it to be a reliable additional investigation in those dying with indications of alcohol misuse. Where there is no active liver disease, a raised gGT result from a peripheral blood sample gives added confidence for chronic alcoholism to be included in the cause of death.

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Multiple Choice Questions in Pathology.


This book aims to provide practice in pathology multiple choice questions (MCQs) for medical students.

An attractive feature is the good mixture of question styles. This helps relieve the monotony of reading through MCQs and gives excellent practice in exam technique. There is a good breadth of subjects, neatly divided into 25 sections. I found that the explanatory comments were written concisely and in an appropriate amount of detail. The answers to the MCQs, however, are printed in bold type in a column down the middle of the page between the questions and the explanations. It is frustrating to see the MCQ answer boldly in front of you before you have a chance to cover it up. Perhaps in subsequent editions the answers could be on the following page.

This edition has occasional ambiguities, but they are outweighed. The format of the MRCPath primary exam has changed and does not include MCQs, so it seems curious that the back cover of this edition mentions its use in preparation for this exam.

This book is excellent value and I would recommend it for medical students who are preparing for pathology exams and who require practice in MCQ technique.

TF MILLARD

Measuring Alcohol Consumption.

Psychosocial and Biochemical Methods.


Measuring Alcohol Consumption is an excellent resource for all those interested, at either a research or clinical level, in alcohol use and misuse. Accurate assessments of alcohol use are vital in monitoring alcoholism treatment and prevention programmes and investigating the links between alcohol consumption and other medical problems. Ray Litten and John Allen have edited a multiauthored volume which is highly organised, cohesive, integrated and practical. It is divided into two main sections: the first dealing with psychosocial measures; and the second with biochemical measures of alcohol consumption.

The first chapter provides a good overview of self-report methods, and emphasises that verbal reports are neither valid nor invalid, but that the important issue is that certain conditions and procedures are more conducive to response accuracy and validity. The second chapter provides an excellent review of "computerized approaches to alcohol assessment", and the finding that the results of computerised testing are generally similar to those of personal or pencil-and-paper interviews.

Timeline Follow-Back (TLFB) is the best psychometrically evaluated and field-tested self-reported alcohol consumption instrument to date. Chapter 3 provides a description of the methods and a thorough discussion of its validity, and appropriate applications of this and other self-report measures in various research and clinical situations. A useful appendix provides instructions for administering TLFB which can be modified for different target groups or research projects. The final chapter of the section on psychosocial measures discusses the accuracy of subjective and collateral accounts of drinking behaviour.

The second section reviews many new and complex biochemical indicators of alcohol consumption. An overview divides biological markers into several types: markers of predisposition to alcoholism (trait markers); markers of chronic or acute consumption (state markers); and markers of damage. Blood and body fluids are considered in the estimation of alcohol consumption, two new markers of high alcohol consumption (carbohydrate-deficient transferrin and 5-hydroxytryptophol), and the usefulness of protein acetaldehyde adducts as state markers of consumption are all discussed. The last two chapters describe non-invasive methods for the measurement of transfernal ethanol as an assessment of ethanol consumption. The use of breathalysers is easy to use in an outpatient setting where patients are seen on a weekly basis and has a high degree of sensitivity and specificity. A wearable, electronic ethanol sensor/ recorder, albeit a resource consuming chemical detection technology used in breathalysers, is also discussed. Unlike the dosimeter, it provides real-time rather than cumulative monitoring of alcohol use, and therefore gives accurate quantitative and temporal tracking of ethanol consumption over extended periods.

This superbly organised, thorough, and readable book is highly recommended for all those who need to assess alcohol intake.

CAROLINE C HORWATH


The authors stated in the Preface that this volume was intended to be a single guide to the clinical diagnosis of non-neoplastic diseases encountered in diagnostic human ultrastructural pathology. It is a companion volume to the book Ultrastuctural Appearances of Tumours prepared by the same authors.

This is a multiauthor work with uniformly high standards throughout, although the chapters range in the extent to which they cover aspects of the subject. As a whole the volume is best regarded as an atlas of high quality photomicrographs with a relatively brief, but extensively referenced, textual introduction to each chapter. The photomicrographs cover most of the commonly encountered entities and there is a generous selection of illustrations of the infrequent or rare lesions, but this cannot be regarded as comprehensive, given that the authors intended to cover the range of non-neoplastic diseases where electron microscopy can contribute to the diagnosis. There is a wide enough coverage, however, for the book to act as a valuable aide memoire for an ultrastructural pathologist while pondering over a difficult specimen. This approach will be of little value to the histopathologist with occasional exposure to electron microscopy.