for staining other components of thick tissue sections.  

Lateral in-depth assessment of microcalcification and the changes in macrocalcification may also facilitate appropriate analysis of mammographic lesions and the diagnostic histological dilemmas resulting from reduction mammoplasty for macromastia.

Prospective comparative study of computer programs used for management of warfarin

Poller et al refer to their experience with, among others, the Hillingdon system for computer-assisted warfarin maintenance.  Their numbers are small, and we should like to record our current experience with a larger series. Nearly all of our patients are given a target INR of 2-8. To compare these as nearly as possible with those of the above authors, who used a range of 2-0 to 3-0, we have used 2-3 to 3-3. The ranges used by these authors are narrow, and divergence from them does not necessarily mean that anticoagulation is at a level which is either ineffective or dangerous. We followed their division into the first 26 weeks of treatment and the period, if any, after that point. The results are shown in the table.

With the important exception of the interval between visits, our experience is not significantly different from that of Poller et al as shown in their table 4. Our mean intervals, both for early and later periods of treatment, are longer than theirs: this may be partly attributable to the maximum permissible interval having been increased from eight to 10 weeks during the period under consideration. We can add that the average interval at the latest visit was 7-04 weeks.

We have only six patients (187 visits) to compare with those who had a higher target INR. Having so few, we would only say tentatively that the average intervals before and after 26 weeks were 2-24 and 3-36 weeks, respectively, and that the interval at the latest visit averaged 3-83 weeks.

The mean interval is important, both for the convenience of patients and economy in the use of hospital resources. Our data support the inherently probable propositions that intervals become longer as treatment proceeds, and are much shorter with higher INR targets. Because they require more frequent attendances, and also because they are more difficult to achieve, high targets require much justification on grounds of clinical necessity.

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Vasculopathy and antiphospholipid antibodies in systemic lupus

We read with great interest the article by Ellison and colleagues showing intramural deposition of platelet derived material in small cerebral vessels in four patients with systemic lupus erythematosus (SLE). The authors recall the strong association between the presence of antiphospholipid antibodies (aPL) and the occurrence of ischaemic cerebral events in SLE. The results of tests for aPL (lupus anticoagulant or anticardiolipin antibodies), however, are not mentioned in their patients with SLE. Such information could probably be obtained from the patients’ charts or from stored serum or plasma. At least the full description of extra-neurological aPL related events—arterial or venous thrombosis, recurrent fetal loss, or thrombocytopenia—would suggest the presence of this peculiar family of antibodies. Such data could allow the hypothesis that intramural deposition of platelet derived material is a feature of a aPL-associated non-inflammatory “vasculopathy” to be tested. The absence of such material in the two patients with SLE and active vasculitis is consistent with this hypothesis, due to the lack of an association between aPL and vasculitis in SLE.

Furthermore, the search for intramural deposition of platelet derived material should be performed in other forms of vascular lesions encountered in patients with antiphospholipid syndrome, either “primary” or secondary to SLE, such as non-inflammatory non-atheromatous large artery lesions and heart valve thickening, the latter being mainly present in patients with SLE, with long-lasting disease. The pathogenesis of these lesions remains unknown: it could involve a complex aPL mediated interaction between platelets and endothelial cells, resulting in platelet derived material incorporation into vessel or heart valve wall, which would explain the “mysterious” thickening frequently observed. Similar remarks could also apply to Sneldon’s syndrome, a condition closely related to aPL, the pathological basis of which has been recently detailed, but the pathophysiology remains obscure.


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 measured, 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 components 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 circulation of patients with the longest histories of neuropsychiatric symptoms and the most deformed, thickened, small vessels.

Carcinoid pattern in adrenal pheochromocytomas

In response to the paper by Harach and Bergholm, 1 I would like to comment on a similar phenomenon that I have encountered in two adrenal pheochromocytomas. One case was sporadic and the other associated with multiple endocrine neoplasia type IIa (MEN IIa). The carcinoid areas seen microscopically were reminiscent of the classic midgut pattern with packets of uniform cells. The tumour cells were smaller and less pleomorphic than the typical pheo-morphic, polygonal chief cells of the usual pheochromocytoma. These carcinoid foci were, however, minor histological components and both tumours had adjacent areas of typical pheochromocytoma. The medullary carcinoma of the patient with MEN IIa, interestingly, did not share this carcinoid phenotype. The question of metastases is, however, not raised because of obvious areas of pheochromocytoma and the characteristic clinical scenario. At the same time, it must be remembered that metastatic medullary thyroid carcinoma within an adrenal pheochromocytoma has been described. 2

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

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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, 1 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 there be difficulty in the use of coroner's "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 possible 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. 2 Excluding modes of death from Death Certificates is one counsel of perfection which I also do not ignore.

While on the subject of counsels of perfection, Dr Slater might like to know that the literature contains many references 3 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. 4

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Vasculopathy and antiphospholipid antibodies in systemic lupus.

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