3 Piette JC, Frances C. Quelle est la nature des lésions vasculaires du syndrome des antiphospholipides? *Rev Med Interne* (in press).
4 Galve E, Candell-Riera J, Pigrau C, *et al.* Prevalence, morphologic types, and evolution of cardiac valvular disease in systemic lupus erythematosus. *N Engl J Med* 1988; **319**:817–23.
5 Galve E, Ordi J, Barquinero J, *et al.* Valvular heart disease in the primary antiphospholipid syndrome. *Ann Int Med* 1992;**116**: 293–8.
6 Kalashnikova LA, Nasonov EL, Borisenko VV, *et al.* Sneddon's syndrome: cardiac pathology and antiphospholipid antibodies. *Clin Exp Rheumatol* 1991;**9**:357–61.
7 Zelger B, Sepp N, Schmid KW, *et al.* Life history of cutaneous vascular lesions in Sneddon's syndrome. *Hum Pathol* 1992;**23**: 668–75.

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 regularly 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 lesions 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,[1] I would like to comment on a similar phenomenon that I have encountered in two adrenal phaeochromocytomas.

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 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 midgut carcinoids was not 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.[2]

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

RUNJAN CHETTY
University of Oxford,
Nuffield Department of Pathology,
John Radcliffe Hospital,
Headington, Oxford OX3 9DU

1 Harach HR, Bergholm U. Medullary carcinoma of the thyroid with carcinoid-like features. *J Clin Pathol* 1993;**46**:113–7.
2 Linnoila RI, Keiser HR, Steinberg SM, Lack EE. Histopathology of benign versus malignant sympathoadrenal paragangliomas: Clinicopathologic study of 120 cases including unusual histologic features. *Hum Pathol* 1990;**21**:1168–80.

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 "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 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 shall happily ignore.

While on the subject of counsels of perfection, Dr Slater might like to know that the literature contains many references[3–11] 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.[12]

EW BENBOW
Department of Pathological Sciences
University of Manchester
Oxford Road
Manchester M13 9PT

1 Slater DN. Certifying the cause of death: an audit of wording inaccuracies. *J Clin Pathol* 1993;**46**:232–4.
2 Office of Population Censuses and Surveys. *1990 Mortality Statistics—Cause.* London: HMSO, 1991.
3 Gobbato F, Vecchiet F, Barbierato D, Melato M, Manconi R. Inaccuracy of death certificate diagnoses in malignancy: an analysis of 1,405 autopsied cases. *Hum Pathol* 1982;**13**: 1036–8.
4 McFarlane MJ, Feinstein AR, Wells CK, Chan CK. The "epidemiologic necropsy". Unexpected detections, demographic selections, and changing rates of lung cancer. *JAMA* 1987;**258**:331–8.
5 Anderson RE, Hill RB, Key CR. The sensitivity and specificity of clinical diagnostics during five decades. Toward an understanding of necessary fallibility. *JAMA* 1989;**261**: 1610–7.
6 Bloor MJ, Robertson C, Samphier ML. Occupational status variations in disagreements on the diagnosis of cause of death. *Hum Pathol* 1989;**20**:144–8.
7 Chan CK, Wells CK, McFarlane MJ, Feinstein AR. More lung cancer but better survival. Implication of secular trends in "necropsy surprise" rates. *Chest* 1989;**96**: 291–6.
8 Nemetz PN, Tangalos EG, Kurland LT. The autopsy and epidemiology—Olmsted County, Minnesota and Malmo, Sweden. *APMIS* 1990;**98**:765–85.
9 Di Furia L, Piga A, Marmili S, *et al.* The value of necropsy in oncology. *Eur J Cancer* 1991;**27**:559–61.
10 Papadakis MA, Mangione CM, Lee KK, Kristof M. Treatable abdominal pathologic conditions and unsuspected malignant neoplasms at autopsy in veterans who received mechanical ventilation. *JAMA* 1991;**265**: 885–7.
11 Hasle H, Mellemgaard A. Hodgkin's disease diagnosed post mortem: a population based study. *Br J Cancer* 1993;**67**:185–9.
12 Slater D. "Patients with terminal cancer" who have neither terminal illness nor cancer. *Br Med J* 1987;**295**:669–70.

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 "relative friendly". Locally, we attempt 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 limitations imposed by attempting to glean 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's) 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 it is *cause* 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 we 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 coroners' cases, and thus the cases are not especially 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 inaccurate or excluded; 3 = acceptably inaccurate where the PCOD may be mistaken for the COD, and 4 = completely inaccurate. The results are shown in the table.

	Score				
	1	*2*	*3*	*4*	*Total*
1990	27 (28%)	43 (43%)	7 (7%)	22 (22%)	99
1991	23 (30%)	29 (38%)	9 (12%)	15 (20%)	76
1992	18 (29%)	27 (43%)	6 (10%)	11 (18%)	62