results had absorbance values below 1·0. Thus, although raising the cut off point between positive and negative readings to a value of $\geq 1{\cdot}0$ provides a test which has an improved and acceptable specificity, it does, at the same time, produce one that has lamentable sensitivity. We sympathise with the desire and need to reduce false positive results in a population with a low chlamydial prevalence, but equally we cannot see that it is acceptable to analyse the results in such a way that sensitivity is so seriously jeopardised. Will our clinical colleagues be comforted by the knowledge that for every patient they can be assured has a chlamydial infection there are two who are also infected but go untreated because of a negative ELISA result? All of this indicates the urgent need for an ELISA which combines much greater specificity with sensitivity and not one that is made to have one of these attributes at the expense of the other.

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Reference

1 Taylor-Robinson D, Thomas BJ, Osborn MF. Evaluation of enzyme immunoassay (Chlamydiazyme) for detecting Chlamydia trachomatis in genital tract specimens. *J Clin Pathol* 1987;**40**:194–9.

Reporting colorectal cancer

Drs Jass and Morson argue persuasively the case for a new system for prognostic classification of colorectal cancer.[1] Having tried to follow their scheme, however, I find two difficulties and feel that comments on these might well be widely appreciated.

The authors seem not to have taken account of the findings of tumour breaching the peritoneum or being present at or close to the surgically dissected surface of the specimen or to the cut end. I presume that if any such features are present, the scheme would be invalid.

There is ambiguity in the definition of "lymphocytic infiltrate" as a favourable prognostic factor. In their fig 1 (identical with that in the earlier paper by Jass *et al*[2]) the legend reads "Conspicuous peritumoural lymphocytic infiltrate." Their text refers to an "inflammatory infiltrate", with other cells present, and to a resemblance to the normal lamina propria. Jass *et al*[2] also refer to these mixed cells being "scattered" rather than conspicuous.

A further problem arises from the patchy distribution of the inflammatory infiltrate. Jass advises assessment based on "the worst area"[3] but as prognosis is better with many cells, does the "worst area" have most cells or fewest?

I think that clarification of the lymphocytic infiltrate criterion is needed and would be much appreciated by those who wish to adopt this new scheme.

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References

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2 Jass JR, Love SB, Northover JMA. A new prognostic classification of rectal cancer. *Lancet* 1987;i:1303–6.
3 Jass JR. Lymphocytic infiltration and survival in rectal cancer. *J Clin Pathol* 1986;**39**:585–9.

Drs Jass and Morson comment:

We are grateful to Dr Penman for highlighting two difficulties he has experienced with the new prognostic classification.

Invasion by tumour beyond the confines of the serosa and involvement of the deep margin of surgical excision are likely to be associated with an adverse outcome. Regrettably, these variables have not been subjected to detailed study in the past and cases showing such features were not specifically excluded when the new classification was developed. The system was based on data derived from operative procedures that were considered by the surgeon to be curative. It is likely that some but not all specimens with serosal penetration by tumour or extensive spread to the deep excision margin would have been regarded as incurable. We would agree with Dr Penman that the system should not apply to growths extending to a surgical margin. Spread beyond the serosa, however, does not necessarily identify an operation as non-curative. For example, if an adjacent organ is fixed to the point of penetration and has been removed with the main specimen en bloc, then the procedure would be regarded as curative. Under these circumstances we would see no great objection to applying the new system but would again emphasise the need to undertake a detailed study of the prognostic importance of serosal disease in the future.

On the second point, there are always problems with applying subjective variables. On low power examination a conspicuous lymphocytic infiltrate manifests itself as a cellular lamina or cap at the invading margin of the tumour. It is not necessary to identify enormous numbers of lymphocytes, but the term "scattered" was perhaps unfortunate. "Distributed" might have been better. The worst area is that in which the lamina is least developed and lymphocytes are fewest in number. When there is any doubt, cases should not be regarded as showing conspicuous lymphocytic infiltration. It is always important to attempt to preserve the purity and prognostic value of a variable, even if the size of the resulting group is small.

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Book reviews

Venous Thrombosis. Causation and Prediction. D Ogston. (Pp 242; £26·50.) John Wiley. 1987. ISBN 0 471 91558 0.

Professor Ogston's book is a timely addition to the literature in the area of venous thromboembolism. Recently considerable advances have been made in the investigation of the pathogenesis of venous thrombosis, particularly in the recognition of familial deficiencies of natural anticoagulants such as protein C and protein S, and of abnormalities of the fibrinolytic system. These developments are of importance to all clinicians involved in the management of, and prophylaxis against, deep venous thrombosis, and they are exhaustively reviewed by professor Ogston. The opening chapters on haemostatic and thrombotic mechanisms are rather scantily illustrated and it is doubtful whether the non-specialist reader would find them as useful as was intended. More positive guidance, based on the author's experience, on the management of subjects with a congenital prothrombotic state and on screening for deficiencies could have been usefully included. In relation to the chapter