Letters to the Editor

Progesterone receptors in meningiomas: morphometric assessment of vascularity and cellularity on near facsimile cryostat sections

Recently, there has been much interest in progesterone receptors (PR) in meningiomas,[1] but although in breast cancer steroid hormone receptor status has been related to the cellularity of the neoplasm,[2] no comparable study has been performed on meningiomas.

Cryostat sections adjacent to sections assayed for PR from 45 meningiomas[3] were stained with haematoxylin and eosin. Using a MOP-Videoplan (Kontron Electronic Group) image analysis system with digitising tablet, images of the sections were projected at a standard magnification on a television monitor and analysed for cellularity with respect to the neoplastic cell content (expressed as a percentage of the section area occupied by neoplastic cells) and vascularity (expressed as the percentage area of the section occupied by blood vessel profiles).

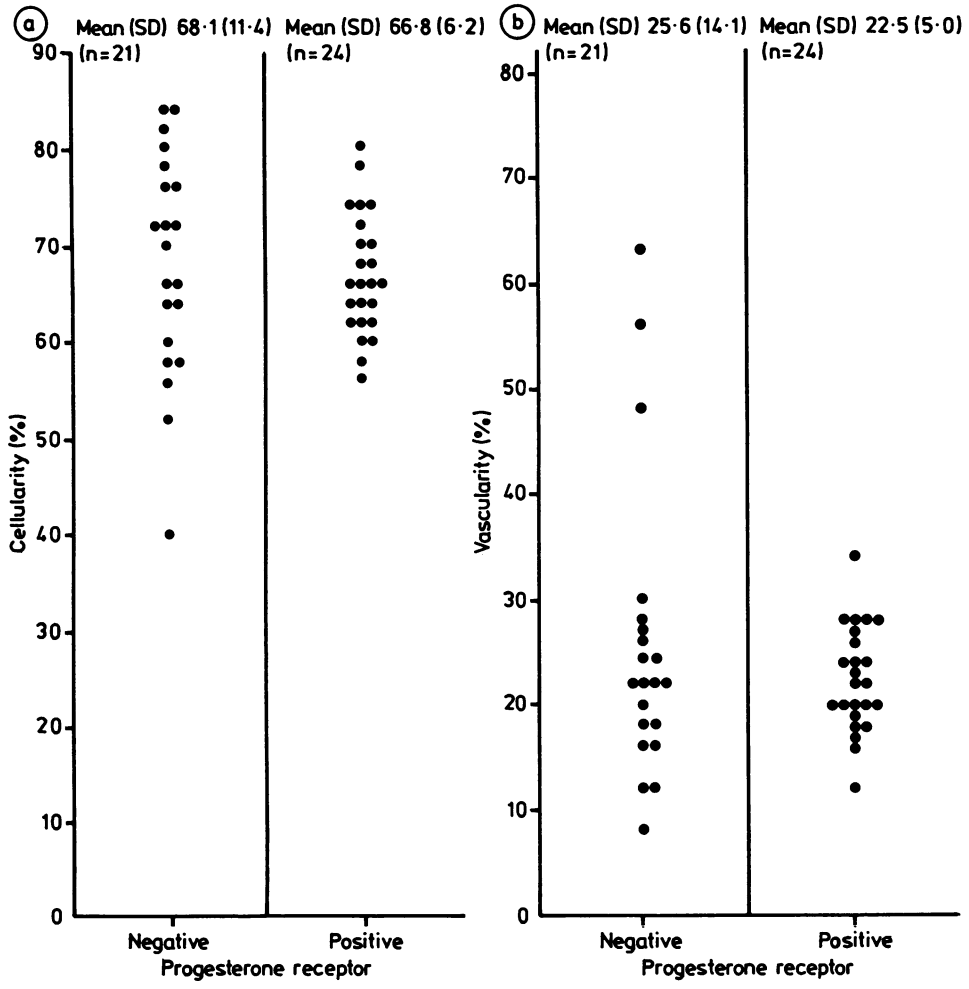
No significant differences were found with respect to cellularity (Fig. 1a) and vascularity (Fig. 1b) between PR negative and PR positive neoplasms ($p > 0.05$, unpaired t test), although the neoplasms with the greatest vascularity (one haemangioblastic and two angiomatous meningiomas) were all found to be PR negative. According to the method of Underwood *et al*[4] PR values were adjusted to compensate for differences in cellularity:

$$(PR)_c = \frac{(PR) \times 100}{C}$$

where $(PR)_c$ = compensated PR value; (PR) = assayed PR value on adjacent section; and C = per cent cellularity of neoplasm

This resulted in a higher range of PR values with one neoplasm changing from the PR negative group to the PR positive group (> 10 fmol/mg cytosol protein).

In a previous study[3] we found that PR values in meningiomas were unrelated to the age or sex of the patient and the site of the neoplasm. No association between the vas-



Correlation between PR values (positive = > 10 fmol/mg cytosol protein) and cellularity (a) and vascularity (b), as assessed by morphometry on adjacent cryostat sections of meningiomas.

cularity or cellularity of the neoplasms and PR values has been found. We are continuing our studies on a larger group of neoplasms to investigate the influence of histological subtype (WHO) on PR values.

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References

[1] Martuza RL, Miller DC, MacLoughlin DT. Estrogen and progestin binding by cytosolic and nuclear fractions of human meningiomas. *J Neurosurg* 1985;**62**:750–6.
[2] Van Netten JP, Algard FT, Coy P, *et al.* Estrogen receptor assay on breast cancer microsamples. Implications of percent carcinoma estimation. *Cancer* 1982;**49**:2383–8.
[3] Ironside JW, Battersby RDE, Dangerfield VJM, Parsons MA, Timperley WR, Underwood JCE. Cryostat section assay of oestrogen and progesterone receptors in meningiomas: a clinicopathological study. *J Clin Pathol* 1986; **39**:44–50.
[4] Underwood JCE, Dangerfield VJM, Parsons MA. Oestrogen receptor assay of cryostat sections of human breast carcinomas with simultaneous quantitative histology. *J Clin Pathol* 1983;**36**:399–405.

Identifying "high risk" laboratory specimen's

Dr Whale questions the value of "high risk" labelling in her letter.[1] It is inevitable that specimens containing hepatitis B or HTLV-III viruses, or both, will be submitted to laboratories without the sender or recipient of the specimen being aware of the hazard. This fact has continually (and correctly) been used as an argument that laboratory practice should always be of a standard that should prevent laboratory infection. The argument that "high risk" labelling should therefore be abandoned overlooks another aspect of such identification of specimens that is related to laboratory accidents. These may fall into two groups: where the worker is at risk of infection by gross splashing or needle stick injury; where the specimen is damaged in transit, and a decision has to be taken regarding its retrieval or disposal. In the first instance if the specimen is known to be hepatitis B positive immune globulin can be used as a prophylactic where appropriate. With regards to an HTLV-III infected specimen, an acute serum can be taken from the worker who is then followed up to see the outcome of the accident and who can be reassured or counselled as appropriate. In the second situation it can be argued that it is much safer, under carefully controlled laboratory conditions, for a senior member of the laboratory staff to salvage damaged specimens than it is for somebody to go and venepuncture the patient. Of course, we are still in the same situation when it comes to the unrecognised specimen, but to abandon the labelling of specimens where there is a known risk would certainly be unhelpful in the case of accident.

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Reference

[1] Whale K. Is it time to rethink "high risk" labelling? *J Clin Pathol* 1986;**39**:114.

Dr Whale replies as follows:

I appreciate Dr Fallon's concern regarding unlabelled specimens in the two situations cited.

In the first I would propose that we extend the same service to our laboratory staff that most of us, in our role of control of infection officer, offer to other health care personnel—that is, inquiry regarding risk factors in the individual patient, if known, coupled with testing of a specimen of blood for hepatitis B antigen, followed by appropriate action, or, if HTLV-III infection is a possibility, counselling and reassurance, with collection of blood at intervals, with the informed agreement of the member of staff concerned, and, if necessary, regular follow up in the occupational health or other appropriate department.

With regard to the salvage of damaged specimens, clearly, action would depend on the nature of the specimen and on the expertise of the venepuncturist, but the risk from venepuncture seems to be minimal, and I venture to suggest that laboratory staff would far rather not have to deal with a leaking blood specimen, whatever the source, if another specimen could be obtained without causing too much distress to the patient.

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Necrotising lymphadenitis without granulocytic infiltration (Kikuchi's disease)

I read with interest the article by Ali and Horton describing four cases of Kikuchi's disease in the United Kingdom[1]; I was astonished, however, by the authors' failure to cite and discuss our report of 30 cases in 1983.[2] With the exception of one patient admitted to Stanford University Hospital, these cases had been submitted to me in consultation and included 21 residents of the United States. We emphasised the remarkable predilection of this disorder for the cervical lymph nodes of young women and confirmed the paucity of granulocytes and plasma cells in affected lymph nodes. Similar observations had previously been made by Kikuchi[3] and others[4 5] in Japan and subsequently by Pileri *et al*[6] in West Germany.

Prior to this report Dr Haruki Wakasa and I had presented the results of clinicopathological studies on 140 Japanese and 30 American cases, respectively, as part of the proceedings of the United States and Japan seminar on lymphoproliferative diseases, held in Seattle, Washington, in 1982.[7]

Since the publication of these reports I have received many more cases in consultation, and these now total 77. Of these, 62 are women and 15 men (a ratio of 4:1). The mean age of these patients is 29 years (range 11–75).

In none of these patients did we identify any evidence of an evolution to a malignant disorder. In most cases lymphadenopathy resolved spontaneously. Two patients (both young women) developed recurrent lymphadenopathy, biopsy specimens of which showed the characteristic morphological features of Kikuchi's disease.

We used the Leder method for showing esterase activity[8] (the naphthol-ASD-chloroacetate method, which identifies only mast cells and myeloid cells in paraffin embedded material), in an effort to evaluate the paucity of granulocytes in Kikuchi's disease. To my surprise some of the karyorrhectic debris stained positively, suggesting the phenomenon of leucocytoclasis. This may support the concept that Kikuchi's dis-