

the lymph node biopsy specimen showed large cell lymphoma and the bone marrow biopsy specimen showed paratrabecular aggregates of small lymphoid cells.

- 1 Brunning RD, Bloomfield C, McKenna RW, Peterson L. Bilateral bone marrow biopsies in lymphoma and other neoplastic diseases. *Ann Intern Med* 1975;82:35-66.
- 2 Collier BS, Chabner BA, Gralnick HR. Frequencies and patterns of bone marrow involvement in non-Hodgkin's lymphoma: Observations on the value of bilateral biopsies. *Am J Hematol* 1977;3:105-19.

### Rapid ELISA for detecting Epstein-Barr virus infection

We read with interest the assessment of the rapid ELISA test (Monolert—Ortho Diagnostic Systems, New Jersey, USA) for detecting Epstein-Barr virus infection<sup>1</sup> and report a case of parvovirus infection which also gave a false positive result with this test.

An 11 year old child presented with spontaneous bruising and had thrombocytopenia (platelets  $20 \times 10^9/l$ ). A bone marrow examination showed increased megakaryocytes, slight haemophagocytosis, and a total absence of red cell precursors. A monospot test was positive and the rapid ELISA test for EBV infection was positive for an acute infection. IgM antibody to parvovirus was present in high titre (greater than 40 units). No EBV IgM antibody was detected, but IgG antibody to EBNA was positive in low titre. Thus despite the positive monospot and Monolert results, there was no serological evidence for an acute EBV infection.

Matheson *et al* found false positive results with adenovirus, cytomegalovirus, and *Toxoplasma gondii* infections. Our case suggests that parvovirus infection may also cross-react and we agree that this test has limitations.

M BHAVNANI  
RB MCGÜCKEN  
J CRASKE  
Department of Haematology,  
Royal Albert Edward Infirmary,  
District Laboratory,  
Wigan Lane WN1 2NN

- 1 Matheson BA, Chisholm SM, Ho-Yen DO. Assessment of rapid ELISA test for detection of Epstein-Barr virus infection. *J Clin Pathol* 1990;43:691-3.

### Cerebral aspergillosis

Boon *et al*<sup>1</sup> have reported a seasonal variation in cerebral aspergillosis following liver transplantation, with most cases undergoing post-mortem examination between November and April. The authors state that, "no environmental source was identified," and it was suggested that the seasonal variation may simply reflect a higher concentration of spores outside the summer months. The highest aspergillosis counts, however, are usually found in the autumn<sup>2</sup>; most of the cases after liver transplantation occurred between December and March.

The possible role of hospital demolition and maintenance work in outbreaks of this condition has been suggested in two recent

papers—one of four patients on a single intensive care unit<sup>3</sup> and the other of three immunosuppressed patients on a medical ward.<sup>4</sup> We therefore wondered whether the apparent seasonal variation in liver transplantation might be related to hospital building work and renovations occurring on a "seasonal basis" rather than to external spore counts. This would have clear implications for the risks of infection in immunosuppressed patients and the planning of hospital rebuilding. It would avoid considering restricting liver transplantation to the summer months.

D CLEMENTS  
Llandough Hospital,  
Penarth,  
South Glamorgan CF6 1XX  
S LEADBEATTER  
Department of Pathology,  
Cardiff Royal Infirmary  
IM HARVEY  
Department of Community Medicine,  
University of Wales,  
College of Medicine

- 1 Boon AP, Adams DH, Buckels J, McMaster P. Cerebral aspergillosis in liver transplantation. *J Clin Pathol* 1990;43:114-8.
- 2 Herman LG. Aspergillus in patient care areas. *Ann NY Acad Sci* 1980;353:140-6.
- 3 Harvey IM, Leadbeatter S, Peters TJ, *et al*. An outbreak of disseminated aspergillosis associated with an intensive care unit. *Community Med* 1988;10:306-13.
- 4 Dewhurst AG, Cooper MJ, Khan SM, Pallett AP, Dathan JRE. Invasive aspergillosis in immunosuppressed patients: potential hazard of hospital building work. *Br Med J* 1990;301:802-4.

#### Dr Boon comments:

Dr Clements *et al* have, quite correctly, drawn attention to the possible role of hospital building and maintenance work in outbreaks of aspergillosis. Despite the current stringencies of NHS capital expenditure, such work still occasionally occurs, but was not a factor in our series.<sup>1</sup> In fact, two further cases of aspergillosis occurred in the liver unit at the Queen Elizabeth Hospital in the summer of 1989. This prompted a thorough investigation of possible sources of *Aspergillus* spores by our microbiologists. Heavy contamination of air shafts leading to the liver unit was discovered and the details of this excellent piece of detective work have been presented. (Elliot TSJ, Stone JW, Smith J. Abstract presented at Pathological Society of Great Britain and Ireland, January 1990.)

Clearly, where air contamination is very heavy, *Aspergillus* spores will lead to infection in susceptible patients, whatever the season. This does not conflict with our observations, which are consistent with a greater abundance of *A fumigatus* spores in winter,<sup>2</sup> wherever the organism might be lurking. One could argue, perhaps, about definitions of "autumn" or "winter," but I suspect there may be variations in the sporulation of *A Fumigatus* according to climate, and I note the North American source of the reference quoted by Clements *et al*.<sup>3</sup>

As others have recently testified,<sup>3-5</sup> aspergillosis is an important cause of morbidity and mortality in many groups of patients. It would, however, be quite as unrealistic to plan hospital rebuilding and maintenance work so as to avoid contamination of specific units at certain times of the year, as to restrict liver transplantation (or treatment of haematological malignancies) to the summer months! The correct approach must surely be effective prophylaxis, avoidance of high dose steroids except where absolutely essential,

early diagnosis, safer antifungal treatment and most importantly, a high clinical index of suspicion. I would also emphasise that without the data obtained from necropsy studies such as ours<sup>1</sup> the true extent of the problem posed by *Aspergillus* would not be apparent.

- 1 Boon AP, Adams DH, Buckels J, McMaster P. Cerebral aspergillosis in liver transplantation. *J Clin Pathol* 1990;43:114-8.
- 2 Seaton A, Robertson MD. Aspergillus, asthma and amoebae. *Lancet* 1989;i:893-4.
- 3 Herman LG. Aspergillus in patient care areas. *Ann NY Acad Sci* 1980;353:140-6.
- 4 Kelsey SM, Newland AC, Van der Walt J, Doran H. Pulmonary aspergillosis in patients with leukaemia. *J Clin Pathol* 1990;43:783.
- 5 Shields ML, Joyner M, Lee R. Invasive aspergillosis in immunosuppressed patients. *Br Med J* 1990;301:1046-7.
- 6 Boon AP, Shetty A. Aspergillosis: a 10 year autopsy review. *J Pathol* 1990;161:340A.

## BOOK REVIEWS

**Endocrine Pathology.** RV Lloyd. (Pp 260; 232 figs; DM 160.) Springer. 1990. ISBN 30540-97166-1

The stated aim of this textbook is to combine classic histological approaches to endocrine pathology with recent developments in immunohistochemistry and molecular biology. In attempting to achieve this aim in a single author textbook, covering the breadth of the endocrine system, Dr Lloyd has set himself a formidable task. He admits that certain areas have not been covered.

The text is variable. For example, there is a useful short, but comprehensive, discussion of the new classification of pituitary adenomas, based on immunohistochemistry and electron microscopy. In contrast, the problematic area of diagnosing malignancy in adrenocortical tumours is incompletely discussed and referenced. The book is extensively illustrated. There are very elegant colour plates of immunocytochemistry and non-isotopic in situ hybridisation, but some of the black and white photomicrographs are not as crisp as might be expected.

This volume must be compared with others based on a functional approach to the subject, which incorporate more of the clinical and biochemical aspects of endocrine disease. Perhaps to a greater extent than in any other area of pathology, histological diagnosis cannot stand alone. I feel, therefore, that this textbook will not be seriously competitive.

AM McNICOL

**The Renal Biopsy. Major Problems in Pathology. Vol 8.** 2nd ed. LJ Striker, JL Olson, GE Striker. (Pp 282; £40.) WB Saunders Company. 1990. ISBN 0 7216 3040 5.

The first edition of this text sits on my shelf but is rarely consulted, for useful information is obtained more easily elsewhere. Two of the authors have changed and this is now virtually a new work rather than just a new