Vitamin K and metabolic bone disease

The report by Vermeer and colleagues indicated that vitamin K deficiency in rats reduces bone healing, and that vitamin K-2, assessed by high performance liquid chromatography, is lower in patients who spontaneously (traumatically) fracture their hips than in patients whose hip fracture is at the hands of an orthopaedic surgeon as an elective procedure. Further research into the interaction of these two vitamins may be indicated.

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Sudden infant death syndrome, long QT interval, and Helicobacter pylori

In response to the recent editorial by Addison,1 I would like to outline two potentially important theories on the aetiology of SIDS which, if true, may act in concert. First, long QT interval. Schwartz et al performed electrocardiography on 33 034 newborns at three to four days of age and subsequently found that of the 34 deaths in this group, 24 were due to SIDS.2 Twelve infants who died of SIDS, but none who died from other causes, had prolonged QT intervals (p 0.01). Long QT interval is known to be a marker of cardiac electrophysiology, and is therefore a predisposition to fatal cardiac arrhythmia.3 It is also known that the QT interval is increased from the second until the fourth month of life, returning to previous values by the sixth month.4 This is also the period of peak incidence of SIDS. Therefore, it is hypothesised that a sudden increase in sympathetic activity may be the trigger which leads to fatal arrhythmia in these cases.5

Second, neonatal Helicobacter pylori infection. As reviewed by Pattison et al,6 epidemiological aspects of SIDS and H pylori infection are similar; both are more common in males, preterm and low birth weight infants. The authors suggest that SIDS and H pylori infection are associated with growth retardation and familial clustering, and that H pylori infection has been documented in infants aged three to six months. In an H pylori infected neonate, vomiting or gastro-oesophageal reflux of gastric juice containing H pylori urease, combined with microaspiration may result in deposition of large amounts of urease in the alveolae, resulting in urea hydrolysis and ammonia supplied to the systemic circulation, with possible respiratory arrest due to ammonia toxicity. In addition, gastric inflammation due to H pylori infection results in synthesis of the cytokine interleukin-1 (IL-1), leading to fever, activation of the immune system, and increased deep sleep which, combined with a relatively minor additional infection, overwrapping, or prone sleep position, may lead to hyperthermia. These two theories are consistent with each other, in that IL-1 is known to activate the sympathetic nervous system in humans. Therefore, in neonates with cardiac electrical instability resulting from long QT interval
and who become infected with *H pylori*, IL-1 mediated activation of the sympathetic nervous system may provide the trigger for fatal arrhythmias leading to SIDS.

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Epithelial-myoepithelial carcinoma?

In the July edition of this journal, McCluggage et al describe a myoepithelial carcinoma arising in a pleomorphic adenoma.1 The report is interesting but raises the question as to whether the original tumour was in fact an epithelial-myoepithelial carcinoma, with the subsequent recurrences representing overgrowth of the malignant myoepithelial component.

In their description of the original tumour, McCluggage et al describe the epithelial element as being set in a hyalinised stroma. It is not clear from the illustration, but could the former be the sclerotic pattern of epithelial-myoepithelial carcinoma as described by Simpson et al?2 The encapsulated nature of the first tumour would not be against this diagnosis as this is an accepted feature of epithelial-myoepithelial carcinoma.1 The subsequent recurrences could then be interpreted as overgrowth of the myoepithelial element, again a well recognised feature of epithelial-myoepithelial carcinoma;2 this would be supported by the presence of ducts and tubules in the first recurrence. Although I would not dispute that malignant transformation occurs within pleomorphic adenomas the slightly atypical appearance of the original tumour in this case should perhaps raise the possibility of an epithelial-myoepithelial carcinoma.

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Authors’ response

We thank Dr Cavill for his letter concerning our recent case report of myoepithelial carcinoma arising in a pleomorphic adenoma. We are confident that the original diagnosis of a pleomorphic adenoma (although with some atypical features) was correct and that this neoplasm does not represent an epithelial-myoepithelial carcinoma. Epithelial-myoepithelial carcinoma is characterised by tubules with a double layer of inner epithelial and outer myoepithelial cells. The ducts and tubules in the original tumour and in the first recurrence in the case we describe had a single cell lining. These cells were morphologically and immunohistochemically (AE1/AE3, EMA, and CEA positive; S-100 protein, α smooth muscle actin, and vimentin negative) characteristic of epithelial cells. We agree that from the illustration (fig 1A) it is not entirely clear whether the tubules have a single or double cell layer but we consider that the impression of more than one cell layer is due to plane of section artefact.

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Authors’ response

We thank Dr Cavill for his letter, which we read with a wry grin. The data used for serum ferritin were reproduced as they appeared in the text we referred to in our paper. We agree we should have pointed out the inaccuracy of the units used in the original source material but somehow, in our enthusiasm to make our point, we slipped. Sorry!

Dr Cavill’s slightly tongue-in-cheek letter does not, however, mask his disappointment at the shrinking laboratory role of many haematologists. We share that disappointment. We agree that on appointment most haematologists undertake both a clinical and a laboratory role. It is one of the consequences of, for example, the reduction in junior doctors’ hours that haematologists have now had to dedicate more of their time to clinical activities at the expense of the laboratory work. The 1997 haematology report from the Clinical Benchmarking Company3 showed that the majority of haematologists spent 45% of their time in activities related to laboratory work. For those laboratories who par-ticipated in both the 1996 and 1997 clinical benchmarking study, there was a reduction in consultants’ laboratory sessions which equated to a 17.5% drop in consultant input into laboratories. This probably reflects the increased commitment haematologists have to clinical duties.

It is clear from our own experience that clinicians who use laboratory services may not interpret the results of diagnostic tests in the same way that an experienced haematologist would. This can result in additional testing and unnecessary referral. The haematologist is, of course, ideally placed to aid clinicians’ interpretation of diagnostic tests in haematology. If likelihood ratios are to be used as ways of aiding clinicians, it is difficult to see how anyone other than haematologists can lead this process. We recognise that current manpower limitations might impede changes, even if we do wish to move in this direction. Perhaps this is an area which the British Society for Haematology, the Royal College of Pathologists, and the Association of Clinical Pathologists may wish to review.

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Department of Haematology, Royal Victoria Infirmary, Newcastle upon Tyne

CD-ROM

Peripheral Blood—Tutor (CD-ROM).
University of Washington. (£450.00.)

The advent of computer graphics has led to an explosion in material available for teaching, and this is another potential candidate for the list. How does this CD-ROM fare? Does it fulfill its promise of helping you make the right call when looking at peripheral smears? It is aimed mainly at the laboratory technical staff and possibly junior doctors. Illustrations and concise relevant text give a good overall flavour of morphology but it is not of sufficient depth to be a particularly useful aid to higher specialist training. An accompanying atlas is available but was not reviewed.

A general introduction covers sampling and film preparation, with useful examples of preparation artefacts. Sections on cell morphology and disease associations follow. It is clear from our own experience that clinicians who use laboratory services may not interpret the results of diagnostic tests in the same way that an experienced haematologist would. This can result in additional testing and unnecessary referral. The haematologist is, of course, ideally placed to aid clinicians’ interpretation of diagnostic tests in haematology. If likelihood ratios are to be used as ways of aiding clinicians, it is difficult to see how anyone other than haematologists can lead this process. We recognise that current manpower limitations might impede changes, even if we do wish to move in this direction. Perhaps this is an area which the British Society for Haematology, the Royal College of Pathologists, and the Association of Clinical Pathologists may wish to review.

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Briçon Congress of Dermatology
1–5 February 1999
Vars, France

The Briçon Congress of Dermatology is now established as a forum for pathologists who have an interest in dermatopathology, to meet, present cases of their own, and to participate in daily slide seminars. The 1999 seminars will be presented by Dr Philip LeBoit (USA), Dr Lorenzo Cerroni (Austria), Dr Janine Wechsler (France), Dr Nigel Kirkham (UK), and others. The meeting is recognised by the RCP for 20 CME points. For further details, contact Dr N Kirkham, Histopathology, Royal Sussex County Hospital, Brighton BN2 5BE, UK, tel +44 (0)1273 664501; fax +44 (0)1273 481012; email: nigeli@pavilion.co.uk

Cytopathology for Histopathologists
1–8 February 1999
Northwick Park Hospital, Harrow

An intensive course in basic cytopathology suitable for candidates preparing for the MRCPath examination in histopathology, and for established histopathologists requiring revision. It is given by the Department of Cellular Pathology, Northwick Park Hospital (Dr Ketan A Shah). Limited to 25 participants. Approved for 30 CME credits. Course fee £350.

Further details from: Debbie Booth, Postgraduate Courses Coordinator, Room 6VO17, Medical Education, Northwick Park hospital, Harrow, Middlesex HA1 3UJ; tel 0181 869 2254

Practical Pulmonary Pathology
Imperial College School of Medicine, London
14–15 April 1999

Further details from: Professor B Corrin, Brompton Hospital, London SW3 6NP, UK. Fax +44 (0)171 351 8293; email: b.corrin@ic.ac.uk

Histological Typing of Kidney Tumours.

This book was widely recognised as one of the most comprehensive and useful accounts of renal tumour pathology available, becoming the reference book for many pathologists. The new book corrects some of the previous deficiencies in the classification of benign and malignant tumours. This book is illustrated throughout in colour. The new figures are well chosen and make their points clearly and well. The text has been brought up to date. It is organised into sections that deal with each of the major tumour groups and tumour-like lesions of the renal parenchyma and pelvis. Several new entities unrecognised in 1981 have been discussed, including metanephric adenoma, chromophobe carcinoma, and renal medullary carcinoma. Renal adenomas have been presented in more detail. More extensive discussion of nephroblastic lesions is given, including mesoblastic nephroma and cystic nephroma. Two new entities are included: clear cell sarcoma and rhabdoid tumour. A list of miscellaneous tumours is also added. Various new tumour-like lesions have been included. The definitions and explanatory notes are very clear and easy to understand by pathologists not familiar with the English language.

The second part of the book includes 145 colour photomicrographs that cover most of the morphological spectrum of the lesions described in the first part. These are of good quality and useful for a quick reference and consultation in routine work. I have had this book by my microscope for several weeks and can confirm its value in day to day practice. Anyone who works in renal tumour pathology will want to get a copy without delay. In addition to this, I have used this book as a teaching tool for renal pathology for the undergraduate medical students. They have found it very useful because of the clarity of the definitions and explanations and, above all, for the wealth of images.

R MONTIRONI

Notices

The Brighton Histopathology Course
(Histopathology for the MRCPath)
17–22 January 1999
Oak Hotel, Brighton

Intensive residential course aimed at comprehensive preparation for the MRCPath part 1 and part 2 examinations in histopathology. The programme includes microscopy sessions, discussions, and formal presentations. The course in also suitable for consultants who want to update their knowledge. It is recognised for 25 CME credits. For further details and application form, contact Dr N Kirkham, Histopathology, Royal Sussex County Hospital, Brighton BN2 5BE; tel 01273 664501; fax 01273 481012; email: nigeli@pavilion.co.uk

K P DINGEMANS
Cellular and Molecular Pathology Update
University of Liverpool, UK
16–19 June 1999

Further details from: Professor C S Foster, Department of Cellular and Molecular Pathology, University of Liverpool, Duncan Building, Daulby St, Liverpool L69 3GA. Tel +44 (0)151 706 4480; fax +44 (0)151 706 5883; email: christopher.foster@liv.ac.uk