

# PostScript

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### Establishing a contract for bench training of specialist registrars in medical microbiology

In common with other medical microbiology laboratories in the UK, the biomedical scientist staff (BMS) at Cambridge have faced an increasing burden of specialist registrar (SpR) training from our own, and other specialties, in the face of falling BMS numbers as a result of efficiency savings and an increasing diagnostic workload. This has led to periods of several months in each year in which SpR training by the BMS has had to be abandoned locally in favour of services with formal laboratory contracts.

Developing a longterm strategy to deal with this problem necessitated the preparation of a business case for SpR training by the BMS, which required an estimation of the local BMS SpR training load. In the first two years, SpRs receive the same basic training in diagnostic methodologies provided for trainee BMS. SpRs spend four months annually in full time bench training, apprenticed to qualified BMS, who spend approximately half their working time on direct supervision of the trainee, equivalent to four months BMS time for each SpR over the first two years. In our own laboratory, we have nine trainees in bacteriology and virology, representing 36 months of BMS time over a notional five year period of SpR training. This calculation assumes that all trainees achieve their annual milestones without delay. In practice, half the trainees sitting the practical component of the examination for membership of the Royal College of Pathologists have required a further six or 12 months of bench training in preparation for a re-sit of the examination. Assuming that half of all the trainees require a nine month extension, this represents an increase in training load at this stage of 27 months, equivalent to an additional 13.5 months of dedicated BMS time, resulting in a total BMS training time of 42.75 months. From their third year of training, SpRs receive three months of advanced training annually, in focused preparation for the

RCPATH practical examination. The BMS provide and supervise the processing of simulated clinical samples, to be processed independently by the trainee, on a programme that runs continuously throughout the year. The BMS time required for this programme amounts to one day each week. This is equivalent to 2.4 months of BMS time, annually, in years 3, 4, and 5—that is, 7.2 months over the five year period, in both bacteriology and virology, amounting to 14.4 months in total. Therefore, the total of BMS time required for SpR training is 57.15 months—11.43 months of working time each year. Allowing for six weeks of leave entitlement, this is equivalent to one full time BMS. The experience and skills are of those at grade 2 of the BMS pay spine, for which the salary, with on costs, is £26 663 to £33 737 per annum.

Having established a business case for a full time training BMS (grade 2), we sought a source of funding from those with an interest in training. None was prepared to fund this comparatively large recurrent sum, but three local stakeholders were prepared to fund one third each: the deanery, with its responsibility for the delivery of SpR training; the workforce development directorate (the training and development arm of the Strategic Health Authority), with its overarching responsibilities to training laboratory staff; and the local primary care trust, which has an interest in preserving and developing the laboratory's services.

The core duty of the training BMS (grade 2) is to preserve SpR training. Whenever BMS numbers are inadequate to allow SpR training, this function is assumed in full by the training BMS. At other times, the training of SpRs continues to be shared between all the BMS, and the training BMS engages in related educational activities, which have been tailored to the sources of funding. These duties are, first, supporting and developing the education, training, and research of SpRs in medical microbiology, as directed by the deanery's programme director in medical microbiology. Second, duties supporting the laboratory training manager in the provision of training of SpRs, BMS, medical laboratory assistants, and visitors to the laboratory. Third, maintaining the training BMS's own professional status and microbiological skills by participating in the rota for provision of the laboratory's routine diagnostic service, under the direction of the laboratory manager. Because the job description contains three major components with different line managers, a clear division of the working week is necessary to ensure realistic expectations and harmonious working relations. The time allocated to the first, second, and third duties are three, one, and one day each week, respectively.

We resolved our local crisis in the provision of SpR technical training by negotiating a training contract, analogous to the laboratory's service contract, with local parties with a training interest. We recommend this model, which we believe to be unique in the UK, to laboratories experiencing difficulties similar to our own.

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### Bilateral breast lumps in a patient after sex mismatched allogeneic transplantation for aplastic anaemia

We report an unusual diagnostic problem in a 35 year old woman with bilateral breast lumps. The patient first presented in August 1998 with a two week history of fever, tooth infection, and easy bruising. Her full blood count showed severe pancytopenia (total white blood cell count,  $1.0 \times 10^9$ /litre; neutrophil count,  $0.1 \times 10^9$ /litre; haemoglobin, 66 g/litre; platelet count,  $22 \times 10^9$ /litre). Her past medical history was unremarkable. She had no causative medications, chemical exposure, or viral symptoms. Flow cytometry for paroxysmal nocturnal haemoglobinuria was negative. A bone marrow biopsy showed a hypoplastic marrow consistent with aplastic anaemia. She had no response to standard doses of antithymocyte globulin, methylprednisone, and cyclosporine and underwent a sex mismatched sibling allogeneic transplant. She had essentially no graft versus host disease, and five months after transplantation she did not return for follow up when all drugs were stopped.

In April 2004, she presented with bilateral breast lumps, nearly six years after transplantation. She had received a course of antibiotics six weeks previously for an indurated lesion in the right inframammary region. On examination, there were erythematous changes over both breasts, and in the left upper outer quadrant there was a hard mass. Enlarged nodes were palpable in both axillae. The subcutaneous mass in the right inframammary region was indurated, hyperpigmented, and ulcerated towards one edge.

Diagnostic investigations were carried out in the following sequence:

- (1) Fine needle aspiration cytology of the breast lesion showed a monomorphic population of neoplastic cells suspicious of a haemopoietic malignancy. Flow cytometry on this aspirate showed the expression of CD34, CD33, CD117, and CD4 on these cells.
- (2) The biopsy of the subcutaneous lesion (right inframammary region) showed a poorly differentiated malignancy expressing CD45, CD34, CD117, and CD4 (immunohistochemistry).
- (3) At presentation the blood counts were normal (haemoglobin, 160 g/litre; total white cell count,  $8.6 \times 10^9$ /litre; platelet count,  $192 \times 10^9$ /litre). The peripheral smear was unremarkable. The bone marrow was hypocellular with 16% blasts. These blasts were difficult to characterise by morphology or standard cytochemistry. Two weeks after admission, thrombocytopenia developed

(haemoglobin, 150 g/litre; total white cell count,  $9.9 \times 10^9$ /litre; platelet count,  $130 \times 10^9$ /litre) and the blood film showed circulating blasts. A repeat bone marrow was hypercellular with 85% blasts. Flow cytometry confirmed a myeloid leukaemia with aberrant expression of CD4 as suggested by analysis of the breast aspirate. Fluorescent in situ hybridisation analysis showed that the tumour cells were XX (female), but with 15% residual male cells. Classic cytogenetics revealed a 46 XX, add(5)(p15) karyotype. A World Health Organisation classification of acute myeloid leukaemia (AML), minimally differentiated, was ascribed to this tumour.

The bilateral breast lesions in this patient raised the possibility of a primary breast carcinoma or a lymphoma in the post transplant setting. The diagnosis of acute leukaemia involving the breasts, soft tissue, and subsequently evident in the bone marrow required multiple diagnostic tissue sampling and techniques. Although the first bone marrow suggested an evolving acute leukaemia with 16% blasts, the World Health Organisation criteria for acute leukaemia were not fulfilled. A second bone marrow taken two weeks later confirmed the diagnosis of AML.

Several studies have shown that longterm survivors of acquired aplastic anaemia may be at high risk for malignant disease. The overall 15 year cumulative incidence for any cancer was found to be 10.9% in an analysis of longterm outcome after allogeneic transplant for aplastic anaemia.<sup>1</sup> In another study, the incidence of myelodysplastic syndrome or leukaemias/lymphomas was higher in patients receiving immunosuppressive treatment compared with those undergoing allogeneic bone marrow transplantation, with solid tumours being common in these last patients.<sup>2</sup> AML presenting as a breast mass (chloroma, granulocytic sarcoma) is rare.<sup>3</sup> There are only a few case reports of AML in the breast antedating AML in the bone marrow.<sup>4</sup> However, to the best of our knowledge, there are no reports of such an occurrence in a post transplant patient.

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The patient gave her consent for this report to be published.

**References**

- Ades L, Mary JY, Robin M, et al. Long-term outcome after bone marrow transplantation for severe aplastic anemia. *Blood* 2004;103:2490-7.
- Socie G, Henry-Amar M, Bacigalupo A, et al, for the European Bone Marrow Transplantation-Severe Aplastic Anemia Working Party. Malignant tumors occurring after treatment of aplastic anemia. *N Engl J Med* 1993;329:1152-7.
- Shea B, Reddy V, Abbott P, et al. Granulocytic sarcoma (chloroma) of the breast: a diagnostic dilemma and review of literature. *Breast J* 2004;10:48-53.

4 **Dutta Roy S, Stafford JS, Seally J, et al.** Granulocytic sarcoma of the breast antedating acute myelogenous leukemia. *Breast* 2004;13:242-6.

**Audit of the histological definition of cervical transformation zone**

We have noticed in our routine practice that there is some variation in the histological definition of the transformation zone of the uterine cervix. We decided to carry out an audit of other pathologists in the UK to see whether there is any variation.

Letters with five different possible definitions of the cervical transformation zone were sent out to members of the National Gynaecological External Quality Assessment scheme, asking them to tick the response they considered the most appropriate definition.

The five options in the questionnaire were:

- (A) Surface squamous epithelium in continuity with surface columnar epithelium (squamo-columnar junction) only.
- (B) Surface squamous epithelium with surface columnar epithelium or stromal gland/crypt, or both.
- (C) Surface squamous epithelium only.
- (D) Surface columnar epithelium only.
- (E) Surface columnar epithelium with squamous (metaplastic) epithelium in gland/crypt.

One hundred and seventeen questionnaires were sent out and responses were received from 82 histopathologists (70% response rate). Tables 1 and 2 summarise the results.

These results confirm our initial impression that there is confusion in the definition of the transformation zone.

The cervical transformation zone is a dynamic entity formed during puberty and, histologically, is the area where the glandular epithelium is being replaced by squamous epithelium.<sup>1</sup> The junction between the two types of epithelium is the squamo-columnar junction.<sup>2,3</sup> The transformation zone is not the same as the squamo-columnar junction but the squamo-columnar junction is part of the transformation zone.

The presence of squamous and columnar epithelium (be it on the surface or comprising a gland) will ideally represent the transformation zone histologically, but if a biopsy contains squamous epithelium only, it could still represent the transformation zone. If there is extensive squamous metaplasia in the transformation zone, histology cannot

**Table 1** Questionnaire results

Option/s	Responses	%
A	13	15.9
B	46	56.1
C	0	0
D	0	0
E	2	2.4
All	1	1.2
None	5	6.1
Combinations*	15	18.3
Total	82	100

\*See table 2.

**Table 2** Combinations

Options*	Responses
A+B	3
B+E	2
A+B+E	8
A+E	1
A+B+C+E	1
Total	15

\*See table 1.

confirm sampling of the transformation zone because of the absence of glandular epithelium.

The fact that some of the respondents in this audit chose multiple options probably reflects the difficulty of defining this dynamic zone. We would cautiously recommend that the transformation zone should be defined by the presence of squamous and columnar epithelium in continuity and/or the presence of squamous epithelium with underlying glands.

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**References**

- Hendrickson MR, Kempson RL. Normal histology of the uterus and fallopian tubes. In: Sternberg S, ed. *Histology for pathologists*, 2nd ed. New York: Lippincott-Raven, 1997:889-92.
- Singer A. Anatomy of the cervix and physiological changes in cervical epithelium. In: Fox H, Wells M, eds. *Haines and Taylor obstetrical and gynaecological pathology*, Vol. 1, 4th ed. Edinburgh: Churchill Livingstone, 1995:225-48.
- Ferenczy A, Wright TC. Anatomy and histology of the cervix. In: Kurman RJ, ed. *Blaustein's pathology of the female genital tract*, 4th ed. New York: Springer-Verlag, 1994:185-201.

**BOOK REVIEWS**

**WHO Classification of Tumours: Pathology and Genetics of Tumours of the Breast and Female Genital Organs**

F A Tavassoli, P Devillee. World Health Organisation, 2003, \$75.00 (paperback). ISBN 9 28322 412 4

Pathology and Genetics of Tumours of the Breast and Female Genital Organs is the fifth volume of the series by the World Health Organisation on the Classification of Tumours. This book fills a gap in the availability of a ready reference manual for tumours of the breast and the female genital tract. It provides a well written, concise, and comprehensive reference book for pathologists and oncologists involved in these disciplines.

The authors use a very well researched approach to the topics included in the manual and the reader friendly layout of the material provides clarity to complex issues in diagnostic pathology. I found that the contents of the "blue boxes" were most

informative and provided helpful diagnostic hints. One of the chapters on breast pathology shows an individual author's bias on the subject, with the inclusion of different terminologies in the already confused subject of intraepithelial proliferative lesions of the breast. Fortunately, this trend does not persist in the remaining chapters of the book.

The immunological and molecular profiles of different tumours is exhaustive, well presented, and provides a valuable diagnostic aid to pathologists in practice, as well as a ready reckoner to research oriented clinicians.

The inclusion of the final section on inherited tumour syndromes is a valuable source of information. A final word of praise on the excellent images of tumours, which will be a delight to the picture matching trainee. I highly recommend this book and consider it an indispensable reference volume for both pathologists and oncologists.

R Vajpeyi

### Colour Atlas of Anatomical Pathology, 3rd Edition

R A Cooke, B Stewart, eds. Elsevier, 2003, £39.99 (paperback), pp 290. ISBN 0 44307 360 0

This atlas has formed an essential part of the training of both new anatomical pathology registrars and pathology undergraduate students. It is probably more essential now given the declining rate of necropsies and the slight ambivalence and aversion of new trainees towards necropsies in general. Knowledge of gross pathology is a fundamental component of anatomical pathology and this atlas is the next best thing to seeing fresh, wet macroscopic pathology.

The illustrations in the book are of a uniform high quality and portray the pertinent pathological features. I have no real criticisms of this compendium of gross photos and would recommend that it be on the shelves of training departments and medical school libraries.

R Chetty

### Bloody Easy: Blood Transfusions, Blood Alternatives and Transfusion Reactions: a Guide to Transfusion Medicine

Edited by J L Callum, P H Pinkerton. 2003 (paperback): Published by Sunnybrook and Women's College Health Sciences Centre, pp 115. ISBN 0 96813 4424

This is a useful book directed at medical staff who are responsible for the transfusion of blood, blood components, and blood products to patients. It is compact and would fit easily into a pocket. The approach is very practical and will provide clinical staff with the information necessary to answer patients' questions. The style is didactic but important

statements are supported by references provided in small print in an appendix. This is a sensible approach because the uncluttered pages are easy to read and can be referred to quickly, whereas the reader who wants to know more is not denied the necessary evidence base. The book links to a useful website, which is designed for those seeking a greater depth of knowledge.

The detailed information given is specifically applicable to Canada. Much, but not all, is readily transferable to other countries. Anyone wishing to provide this book for the use of clinical staff might wish to have an insert giving details of local differences. Two blank pages at the back of the book would permit this to be pasted in.

On a personal note, I do not like the title—it would make me less likely to recommend this book.

B J Bain

### Forensic Pathology Reviews: Volume 1

Edited by M Tsokos. Published by Humana Press, 2004, £53.23 (hardback), pp 384. ISBN 1 58829 4145

As a rule, new forensic pathology books do not sprout original or unique information that is not already present in the print medium. It was with a sense of excitement that I agreed to review this book, which according to the cover spoke of an impressive list of international collaborators. The selection of contributors is excellent; an example would be the chapters on neonaticide written by the prolific pen of Roger Byard, who was also responsible for coauthoring the chapter on sudden infant death syndrome (SIDS) with Henry Krous.

The book is not presented in a standard "A to Z" form but instead has 15 chapters dealing with disparate issues, varying from common topics such as SIDS to unusual subjects such as ileopsoas haemorrhage. These chapters are placed into the subheadings of: death from environmental conditions, trauma, neurotraumatology, forensic neuropathology (separating these last two topics is not warranted in my opinion), sudden death from natural causes, child abuse, neglect and infanticide, SIDS, infectious diseases, death scene investigation, maternal death in pregnancy, iatrogenic injury, toxicology, and forensic differential diagnosis. The major advantage of such a format with short precise individual chapters is that one can take random "dips" into the book looking at topics that may catch your eye at a specific moment in time.

The text is current and contains a useful sprinkling of hints and pearls that would be of use in death investigation. A good example is the practical approach to sudden cardiac death in chapter 5.

A shortcoming, in my opinion, is the lack of colour illustrations. I know that this would increase the costs but perhaps the editor should consider a separate companion

CD that could have additional text and illustrations.

It is a great compliment to the editor that the book flows seamlessly from one chapter to another, despite the diversity of the contributors. This is a well compiled book, which is a refreshing addition to the forensic pathology genre. I would recommend this book to anyone in the medico-legal arena who has an interest in forensic pathology. The book has something to offer to both the novice and the expert. It would make a useful reference in any departmental or institutional library. I look forward to volume 2.

M A Dada

## RETRACTION

Pai SA, Kini D, Shetty K, *et al.* Psammomatous carcinoid of the rectum. *J Clin Pathol* 2003;**56**:978.

At the time of writing, the authors were not aware that the patient had undergone a panhysterectomy for a possible ovarian tumour six years previously. Details of surgery and pathology are still not available. In view of this, and the negative cytokeratin 7 and positive cytokeratin 20 results (kindly performed by Dr J Aidan Carney, Mayo Clinic, Rochester, Minnesota, USA), we realise that the neoplasm is not a psammomatous carcinoid.

## CALENDAR OF EVENTS

Full details of events to be included should be sent to Maggie Butler, Technical Editor JCP, The Cedars, 36 Queen Street, Castle Hedingham, Essex CO9 3HA, UK; email: [maggie.butler2@btopenworld.com](mailto:maggie.butler2@btopenworld.com)

### Practical Pulmonary Pathology

26–29 July 2005, Royal Brompton Hospital, London, UK  
Further details: Professor B Corrin, Brompton Hospital, London SW3 6NP, UK. (Fax +44 (0)20 7 351 8293; e-mail [b.corrin@ic.ac.uk](mailto:b.corrin@ic.ac.uk))

### Association of Clinical Pathologists' National Scientific Meeting

16–17 June 2005, Royal College of Physicians, London, UK  
Further details: ACP Central Office, 189 Dyke Road, Hove BN3 1TL, UK. (Tel +44 (0)1273 775700; e-mail [info@pathologists.org.uk](mailto:info@pathologists.org.uk))

### Breast Diagnostic Histopathology Update

22–23 September 2005, Hammersmith Hospital and Imperial College, London, UK  
Further details: Wolfson Conference Centre, Hammersmith Hospital, Du Cane Road, London W12 0NN, UK. (Tel +44 (0)20 8383 3117/3227/3245; Fax +44 (0)20 8383 2428; e-mail [wcc@ic.ac.uk](mailto:wcc@ic.ac.uk))



## Bilateral breast lumps in a patient after sex mismatched allogeneic transplantation for aplastic anaemia

A K Enjeti, M Seldon and S Braye

*J Clin Pathol* 2005 58: 670-671

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