Standardisation of breast tissue fixation procedures

The Royal College of Pathologists Working Group have provided comprehensive guidelines for the reporting of histopathological specimens within the National Breast Screening Programme. We are concerned, however, that tissue fixation procedures are not to be standardised among participating laboratories.

Most fixatives penetrate tissues slowly; for example, 4% formaldehyde solution penetrates by 3.8 mm in 24 hours, so the centre of small unsliced breast biopsy specimens may not have been fixed within that period. The guidelines provide detailed instructions in relation to mastectomy specimens, but overnight fixation is sanctioned for unsliced breast biopsy specimens.

We have recently shown that a delay in fixation of 24 hours produces a reduction in the number of observable mitotic figures in breast carcinomas of about 75%. In some tumours the decreased count after just six hours can result in a change in the overall modified Bloom and Richardson tumour grade and hence prognostic group.

These findings have important implications for the accuracy and validity of any multi-centre study comparing breast carcinomas on the basis of a histological grading system that depends on the mitotic count. The inevitable variation in fixation delays of surgical specimens may be yet another factor which has contributed to the widely reported difficulties in consistency and reproducibility in breast carcinoma grading among centres. We believe that the failure to observe a defined system of tissue fixation may undermine any proposed pathological audit within the current National Breast Cancer Screening Programme.

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On behalf of RLP Advisory Group on Quality Assurance in Breast Screening Dr Sloane et al comment:

We note with interest the comments made by Drs Start, Cross, and Smith on the procedure for fixing breast biopsy specimens recommen-

Gastric histamine concentration and IgE in Helicobacter pylori infection

Queiroz et al recently observed a significantly lower gastric histamine concentration in Helicobacter pylori positive patients than in H. pylori negative subjects and hypothesised that this might be due to increased histamine release with a subsequent depletion of stores. We evaluated the immune response involving IgE in patients with chronic gastritis who were infected with H. pylori. Of 26 patients with H. pylori infection, 22 (84%) tested positive for basophil bound specific IgE (determined by the histamine release test) and 18 (69%) for serum specific IgE (determined by an enzyme linked immunosorbent assay). In contrast, among 17 subjects in whom the bacterium was not detected, only one, with histologically confirmed gastritis, had cell bound and serum specific IgE. In four subjects who tested positive by the histamine release test without detectable specific circulating IgE, acid elution experiments showed that histamine release from basophils occurred through an immunological mechanism. To demonstrate further the class specificity of the response, the H. pylori antigen was used to challenge normal basophils passively sensitised with serum from IgE positive patients. No significant histamine release obtained on exposure to antigen excluded interference with antibodies other than IgE directed against the same antigen. Inhibition experiments with bacteria obtained by Drs Start showed that the IgE antibody was specifically directed against this organism.

Our data strongly suggest that an immunological response involving IgE is present in patients infected with H. pylori. It is therefore conceivable that IgE has a role in inducing histamine release in the stomach through the activation of mast cells. In fact, these cells, which store histamine in human gastric mucosa, express specific surface receptors with high affinity for IgE. On antigenic stimulation mast cells are activated, undergo degranulation, and release their mediators by a secretory, energy requiring process. Not only histamine, but also other proteins, such as newly generated mediators released by mast cells, may be implicated in tissue damage.

The ability of H. pylori to induce a specific IgE immune response could answer key questions regarding the mechanisms of gastroduodenal inflammation.

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Dr Queiros comments:

We have shown that H. pylori positive patients, both children and adults, have lower gastric histamine concentrations than H. pylori negative subjects. This might be secondary to an increased release of histamine from mast cells which store histamine in human gastric mucosa; and induced by some H. pylori by-product; or by gastric acid which is increased in H. pylori positive patients.

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We think that histamine released by these or by other mechanisms could also be involved in gastric acid hypersecretion in these patients.


Repair of fetal bodies after dissection

We have, for the past year, used a method similar to that described by Gau and her colleagues to repair both fresh and formalin fixed fetal bodies after dissection, and occasionally to completely reconstruct early second trimester fetuses received in fragmentations after genetic termination of pregnancy by suction or other evacuation methods. We have found that large skin defects in the small fetus can be repaired using a patch of amnion attached with adhesive. Whether a single or double layered patch of amnion or a sheet of amniocorial membrane is used depends on the fetal skin colour, thickness, and texture in the region to be repaired. Moreover, we have found that colourless cyano acrylate adhesive, usually referred to as Super glue and commonly used to assemble plastic models and for domestic repairs, is entirely satisfactory and fully acceptable to funeral directors and parents. Even in single drop dispensing tubes this is between 10 and 20 times cheaper than surgical adhesive and is cheaper still in larger tubes.

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Experience of invasive aspergillosis in Hong Kong

We read with interest the article by Boon et al., and would like to share our experience in Hong Kong.

We reviewed the records of the 2462 necropsies performed between 1987 and 1990 by the Department of Pathology, Queen Mary Hospital, Hong Kong. Using the same criteria as Boon et al., 11 cases of invasive aspergillosis were identified (eight males and three females with a mean age of 46.4 years, ranging from 12 to 81 years). Eight of these were considered to be high risk for aspergillosis: they had haematological malignancies, four with acute myeloid leukaemia, two with chronic myeloid leukaemia in blastic transformation, one with acute lymphoblastic leukaemia and one with polycythaemia rubra vera). One patient was receiving long term parenteral nutrition after extensive gut surgery. Another was a 78 year old man with an incidental finding of a 1 cm hepatocellular carcinoma at necropsy. The last case was a 12 year old girl with apparent good health who suddenly developed bronchopneumonia and died four days later; necropsy showed invasive aspergillosis in both lungs. The commonest site of infection is the lung, evident in all our cases, and other sites include the liver, spleen, kidneys and the heart. Invasive aspergillosis was not diagnosed until necropsy in all our 11 cases, although fungal infection was strongly suspected in three of them. Repeated cultures for aspergillus were negative and transbronchial biopsy specimens were taken in the three cases, but none was considered diagnostic of aspergillosis.

Boon et al commented that invasive aspergillosis is not exclusive to the classic "high risk" group and included three in their report. There were also three such cases in our series, although two of the patients were debilitated by total parenteral nutrition or solid malignancy while the young girl was apparently healthy before death. Other authors have also reported similar experiences. While postmortem diagnosis of invasive aspergillosis has been refined by new investigatory techniques, it is still more often diagnosed at necropsy, and we reaffirm the value of necropsy in clinical audit.

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The question is "who pays?"—that is, for the testing, introduction, and regular use of an efficient system. The comment is that it is now vital that we pathologists give the same care and attention to obtaining and checking financial data about our laboratories, as most of us have for many years, to information about the number and variety of tests carried out and the quality of our results. In this department, however, the staff are very hard pressed to keep up with what, until now, has been regarded as their regular duties as clinical biochemists to provide results and comments to clinical colleagues, and it is hard to find the resources (staff) to assess, introduce, and apply simple computer packages for ordering, stock control, and noting expenditure without which accurate and precise budget statements can neither be obtained nor those from management validated.

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BOOK REVIEWS

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This short book, extracted from Diagnostic Surgical Pathology, covers the whole female genital tract, placenta, gestational trophoblastic disease and peritoneum in only eight chapters. It is not as detailed as the major diagnostic gynaecological text books, but the chapters are authoritative and comprehensively referenced up to 1990. The text is clear with excellent monochrome and colour illustrations. Much of the content is familiar and represents a concise overview and updating of work presented in other textbooks. Hendrickson and Kempson's chapter on uterine pathology includes many of the charts and lists from their textbook. The three chapters which Young, Clement, and Scully have written on the ovary, fallopian tube, and peritoneum are excellent summaries and
Gastric histamine concentration and IgE in Helicobacter pylori infection.

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