Correspondence

Manpower is another influential factor of necropsy rate

We read with interest the article by Start *et al* "Analysis of necropsy request behaviour of clinicians".[1] The grade of clinician was not a significant factor in requesting necropsies in their study, although the success of necropsy requests is said to be influenced by the grade of clinician making the request.[2] We would like to comment on another influential factor of necropsy rate. We assumed that the necropsy rate differed according to the number of doctors who request necropsy. We reviewed the necropsy rate (number of necropsies/number of deaths) during 1994–96. During that period, 331 necropsies among 1147 deaths were performed (necropsy rate 29%). We further analysed the necropsy rate for each month and found that the rate was lowest in April (20%) (table 1).

Table 1 Necropsies in Kawasaki Medical School Hospital (1994–96)

	March	*April*	*May*	*Year*
Total number of deaths	100	90	107	1147
Total number of necropsies	35	18	34	331
Necropsy rate	35%	20%	32%	29%

We postulated that this was caused by a change in manpower: most of the chief residents and senior residents move to other hospitals in April. Second year residents also move to other referring hospitals in April as part of the scheme of postgraduate education. In addition, first year residents, who have newly graduated from medical school start their work in May; therefore, there is a decrease in the number of the residents in April (table 2).

Table 2 Average number of doctors in Kawasaki Medical School Hospital (1994–96)

	March	*April*	*May*
Total number of residents	191	150	197
Total number of staff	148	152	152
Total number of doctors	339	302	349

Substitution of doctors in charge seems to result in a lack of intimate doctor-patient relationship. In addition, the increased work load because of the decreased number of residents possibly contributes to less frequency of requesting necropsies. We would like to emphasise that manpower is another influential factor in necropsy rate.

M SHIMIZU
M HIROKAWA
T MATSUMOTO
K OHMOTO
Departments of Pathology and Medicine (Division of Gastroenterology), Kawasaki Medical School, 577 Matsushima, Kurashiki-city, Okayama 701–01, Japan
email: shimizur@med.kawasaki-m.ac.jp

1 Start RD, Brain SG, McCulloch TA, *et al*. Analysis of necropsy request behaviour of clinicians. *J Clin Pathol* 1996;**49**:29–33.
2 Chana A, Rhys-Maitland R, Hon P, *et al*. Who asks permission for an autopsy? *J R Coll Physicians Lond* 1990;**24**:185–8.

Lymphocytic gastritis and *Helicobacter pylori*: a Brazilian survey

We read with interest the report by Niemelä *et al* on the frequency of lymphocytic gastritis and its association with *Helicobacter pylori* in Finland.[1] Their results are interesting because they refer to a 10 year follow up of 96 patients with dyspepsia of whom nine had lymphocytic gastritis at first examination and 12 at the second examination. This frequency is very much higher than those reported in other studies in Europe and Africa, which range from 0.1% to 4.8%. Moreover most reports have pointed out an inconstant association of lymphocytic gastritis with the presence of *H pylori*. For instance, Dixon and colleagues[2] studied 382 patients with dyspepsia (without gastric ulcer or neoplasm) and found 17 cases with lymphocytic gastritis (4.5%) and only seven of these patients were *H pylori* positive in histological sections. They did find serological evidence of *H pylori* infection in all patients, even in cases where the bacterium was not detectable in biopsy specimens, and concluded that this microorganism may be a possible antigen related to lymphocytic gastritis.

In Belo Horizonte, Brazil, the occurrence of gastric infection with *H pylori* has been well established. The infection is very common in the general population (80%) with a high rate of infection among children.[3 4] The high frequency and early infection with *H pylori* are believed to be a predisposing factor to gastric atrophy, gastric cancer, and perhaps to other gastric disease such as lymphocytic gastritis.[5] Therefore, a possible association between *H pylori* and lymphocytic gastritis in populations with an early and high rate of infection would be an interesting point to study. We carried out a study of the frequency of lymphocytic gastritis in Belo Horizonte. We reviewed 800 consecutive patients with gastric biopsies of oxyntic and antral mucosa, without malignant neoplasm or gastric ulcer. The biopsies were fixed in formalin and stained with haematoxylin and eosin. The number of lymphocytes in 100 epithelial cells was graded in all specimens: grade 1 (≤ 15/100), grade 2 (16–29), and grade 3 (≥ 30/100). Grade 3 was considered to indicate lymphocytic gastritis, as generally accepted (table 1).

Nearly all patients (97.7%) had 0 to 15 lymphocytes/100 epithelial cells and only six patients (0.8%) had lymphocytic gastritis. Four of these six cases were *H pylori* positive histologically. Our data show that the frequency of lymphocytic gastritis in Brazil agrees with international reports but differs from the results of Niemelä *et al*.[1] Although different *H pylori* strains could be related to lymphocytic gastritis, as is apparently the case for gastric carcinoma and peptic ulcers, our results provide evidence that *H pylori* infection, based on epidemiological indexes, is not a predisposing factor to lymphocytic gastritis. Thus, the higher index (9–12.5%) found by Niemelä *et al* could be explained by other factors such as different kinds of antigens present in gastric lumen, geographic characteristics, and other specific factors related to a given restricted group of patients.

Table 1 Grading of number of intraepithelial lymphocytes/100 epithelial cells

	Grade		
Patients	*1*	*2*	*3*
800	782 (97.7%)	12 (1.5%)	6 (0.8%)

Grade 1 (≤ 15/100); grade 2 (16–29); grade 3 (≥ 30/100).

V L A A RIBEIRO
J S ANDRADE FILHO
A J A BARBOSA
Laboratory of Digestive and Neuroendocrine Pathology, Department of Pathology, School of Medicine, UFMG, Avenida Alfredo Balena, 190, 30130–100 Belo Horizonte, Brazil

1 Niemelä S, Karttunen T, Kerola AT, *et al*. Ten year follow up study of lymphocytic gastritis: further evidence on Helicobacter pylori as a cause of lymphocytic gastritis and corpus gastritis. *J Clin Pathol* 1995;**48**:1111–16.
2 Dixon MF, Wyatt JT, Burke DA, *et al*. Lymphocytic gastritis—relationship to Campylobacter pylori infection. *J Pathol* 1988;**154**: 125–32.
3 Coelho LGV, Das SS, Karin QN, *et al*. Campylobacter pylori in the upper gastrointestinal tract: a Brazilian study. *Arq Gastroenterol* 1987; **24**:5–9.
4 Carvalho AST, Queiroz DMM, Mendes EN, *et al*. Diagnosis and distribution of Helicobacter pylori in the gastric mucosa of symptomatic children. *Braz J Med Biol Res* 1991;**24**:163–6.
5 Parsonnet J, Friedman G, Vandersteen DP. *et al*. Helicobacter pylori infection and the risk of gastric carcinoma. *N Engl J Med* 1991;**325**: 1127–31.

Dr Niemelä and colleagues comment

We appreciate the interest of Ribeiro *et al*. The main messages of our study were the association of *H pylori* infection with lymphocytic gastritis and the progression of lymphocytic gastritis to atrophic corpus gastritis. The number of patients in our study was small, and although the material consisted of unselected patients, it is not fully representative of the population. Therefore, it may not be appropriate for the estimation of prevalence of lymphocytic gastritis, which has an unequal sex and age distribution. Lymphocytic gastritis is more prevalent in women,[1 2] and the reported mean age of diagnosis in adults is 47–49 years.[1 2] In our study, the patients were mostly middle aged or older (40–71 years) and there was a female predominance, both facts possibly contributing to the observed high prevalence of lymphocytic gastritis.

Differences in the diagnostic methods might add to the observed variance of prevalence of lymphocytic gastritis. There seem to be different views about the way to estimate the number of intraepithelial lymphocytes (IEL). We and others[1] have used the ratio of IEL/100 epithelial cells (that is, 100 cells of epithelial origin). In another studies[3] the authors used the ratio of IEL/100 cells in the epithelium (apparently meaning both lymphoid and epithelial cells). It is obvious that a lower density of IELs is needed to reach the diagnosis with the first method. In addition, like Haot *et al*,[4] we selected the areas of maximal IEL concentration for counting. This probably increased the number of diagnoses compared with the evaluation made in random fields.[3] Finally, the rather extensive sampling (eight systematic biopsies) in our study potentially increased the number of diagnoses.

Ribeiro *et al* suggest that *H pylori* is not a predisposing factor for lymphocytic gastritis as the diagnosis was rare in their population with a high prevalence of *H pylori* infection and *H pylori* was absent from one third of the patients with lymphocytic gastritis. *H pylori* infection was, however, determined by haematoxylin and eosin stained sections with no

serological studies reported. Our study and other previous reports[1] show that the seropositivity for *H pylori* is common in lymphocytic gastritis, while histologically the bacteria may be present in very low numbers, or not be detectable. Histology is thus not a sensitive way to diagnose *H pylori* infection in these patients.

Association with coeliac disease[5] indicates that lymphocytic gastritis is not a disease with a single cause, but rather a reaction pattern associated with hereditary and environmental factors. We agree with Ribeiro *et al* that it is important to consider possible ethnic and environmental differences in the evolution of lymphocytic gastritis.

1 Dixon MF, Wyatt JI, Burke DA, *et al*. Lymphocytic gastritis—relationship to Campylobacter pylori infection. *J Pathol* 1988;**154**:125–32.
2 Jaskiewicz K, Price SK, Zak J, *et al*. Lymphocytic gastritis in nonulcer dyspepsia. *Dig Dis Sci* 1991;**36**:1079–83.
3 Rubio CA, Befritz R, Eriksson B, *et al*. The topographic distribution of lymphocytic gastritis in gastrectomy specimens. *APMIS* 1991;**99**: 815–19.
4 Haot J, Hamichi L, Wallez L, *et al*. Lymphocytic gastritis: a newly described entity: a retrospective endoscopic and histological study. *Gut* 1988;**29**:1258–64.
5 Karttunen T, Niemela S. Lymphocytic gastritis and coeliac disease [letter]. *J Clin Pathol* 1990; **43**:436–7.

Book reviews

Fungal Infection: Diagnosis and Management. MD Richardson, DW Warnock. (£19.95.) Blackwell Science, 1997. ISBN 0 8654 2724 0.

As an undergraduate and would be microbiologist at St Mary's in Paddington (London, UK), moulds played a very important and significant part in my training—historical, as in Alexander Fleming and then current with Dr Roland Davies.

So why is mycology more important today? The advent of HIV related disease, the ever increasing number of immunodeficient patients as a result of treatment (bone marrow, renal, liver, heart, and lung transplantation, and ever more heroic surgery: oesophagectomy, etc), and the wider availability of intensive care medicine have all contributed to bringing mycology to the fore of severe and life threatening microbiological problems for our patients.

This book, written by two leading UK mycologists, is divided into 27 chapters, together with a small but comprehensive select bibliography. The authors have attempted to blend the European practice of dealing with fungal infections, with those found in Australia and the USA.

One of the early chapters is devoted to laboratory diagnosis with help in how to obtain the best specimens. This is followed by 38 pages on antifungal treatments, including explanations on the ever increasing choice of amphotericin formulation. Six chapters are devoted to superficial mycoses and the remaining 17 to deep invasive disease—those experienced in Western hospitals managing immunocompromised patients, and those met within the tropics.

I found this book a joy to read; perhaps with my background that is to be expected. However, I believe this text will be of special interest to medical microbiologists, dermatologists, oncologists, intensive care specialists, and those caring for patients with HIV related disease. It should be on the shelf of all practitioners in these disciplines, somewhere close to hand and not just left to gather dust.

R C SPENCER

The Thyroid: Fine Needle Biopsy and Cytological Diagnosis of Thyroid Lesions. Monographs in Clinical Cytology. Vol 14. (US$97.50.) S Rorell, H Philips. Karger, 1997. ISBN 3 8055 6383 3.

The book continues the series *Monographs in clinical cytology* and is written by cytopathologists with great experience in the field of thyroid fine needle biopsy. The format is reasonably conventional, initial chapters providing succinct descriptions of thyroid anatomy, needle biopsy technique, and reporting guidelines including the assessment of specimen adequacy. The review of diagnostic accuracy and limitations of thyroid fine needle aspiration is particularly helpful.

Following this usual preamble are two main chapters that describe the cytomorphological features of thyroid disease. The first of these details the cellular and non-cellular components of the smear and includes the only colour illustrations in the book. These are somewhat disappointing being in multiplate format and often rather small. The second provides a more conventional description of thyroid lesions including goitre, thyroiditis, and neoplasia. The text is concise and clear and the black and white illustrations are of high quality. Each section ends with a very useful summary of diagnostic features and caveats, although the number of the latter might make one wary of ever attempting a diagnosis in some instances.

The final chapter briefly reviews diagnostic pitfalls. As acknowledged by the authors, the style of the book inevitably leads to some repetition in these chapters. The references are helpfully grouped at the end of the book and are impressively updated.

In summary, I believe this book has much merit and will provide a useful addition to the bookshelves of any department reporting thyroid cytology specimens. The presence of folded page corners in my own copy attests to its value as a bench book.

A M McNICOL

Laboratory Diagnosis of Group A Streptococcal Infections. DR Johnson, EL Kaplan, J Sramek, R Bicova, J Havlicek, H Havlickova, J Motlova, P Kriz. (Sw. fr. 32.) World Health Organisation, 1997. ISBN 9 2415 4495 3.

In view of the current interest in the changing epidemiology of group A streptococcal (GAS) infections and indeed the profound increase in the severity of streptococcal diseases reported in many countries, it is essential to have accurate microbiological and epidemiological surveillance for GAS in each country. The World Health Organisation has established a worldwide network of collaborating centres to assist in the diagnosis and understanding of haemolytic streptococcal infections. International Reference Centres have greatly contributed to the understanding and control of these infections including the education and training of laboratory personnel; they serve as reference laboratories to other research and service facilities globally and contribute greatly to basic applied and epidemiological research.

Hence, the preparation and publication of this manual, which is the definitive reference laboratory's "bible" for all microbiological and serological techniques concerning the laboratory diagnosis of streptococcal infections, notably Lancefield group A streptococci (*Streptococcus pyogenes*).

The manual has been written by global experts within this field from two WHO Collaborating Centres on Streptococci, namely the centres in Minneapolis and Prague. It is intended for wide use in many different countries, therefore, methodologies that may not be applicable—for example, in developing countries, are also included with the aim of promoting and developing laboratory technologies within the international network of Streptococcal Reference Centres and beyond. The protocols have been in use for decades within these centres; there is a comprehensive reference list for both conventional and new molecular typing methods that will enable the trained microbiologist to use these tests and establish a reference facility for streptococci (providing resources are available) almost anywhere.

The manual describes key methods that have disseminated from reference and research centres in Atlanta, London, and New York. The first Streptococcus Reference Laboratory was established within the Public Health Laboratory Service in 1946 by Dr Winston Maxted and Dr Fred Griffith with guidance from Dr Rebecca Lancefield (Rockefeller University, New York, USA). From these people has evolved this unique manual comprising decades of science and innovation. It is essential to anybody embarking on group A streptococcal reference or research.

A EFSTRATIOU

TNM Classification of Malignant Tumours. 5th edition. (£24.95.) UICC International Union Against Cancer. Wiley-Liss, 1997. ISBN 0 4711 8486 1.

The first complete edition of the *TNM classification of malignant tumours* by the International Union Against Cancer (UICC) was published in 1968, although individual site classifications had been available earlier. This system of staging tumours by their anatomical extent has found widespread if not universal acceptance as a means of categorising disease, assisting management decisions, predicting patient outcome, and enabling comparisons between different treatment protocols.

The fourth edition was published in 1987 and revised in 1992. This new fifth edition remains pocket-sized and inexpensive. The introduction includes a brief history of the system and the variously named UICC committee responsible for the classifications, a listing of members of which reveals a paucity of pathologists. Substantial differences from the last edition, and new classifications of previously unclassified tumours, are helpfully marked by a vertical bar adjacent to the relevant text and, while there is still no index, a table of contents is provided. Changes have been made in the classifications of nasopharyngeal, urological, fallopian tube, and brain neoplasms, and paediatric tumours have been deleted. Serum marker concentrations can now be used in staging testicular and gestational

trophoblastic tumours. For gynaecological cancers, comparisons with the relevant FIGO stages are tabulated and the entire UICC classification—criteria, notation, and stage grouping—is identical to that of the American Joint Commission on Cancer in their 1997 *Cancer staging manual*. This is convenient but does raise the question of why two separate publications are considered necessary.

As with previous versions, specialist interest groups will, no doubt, analyse and possibly refute some of the changes, and each pathologist will continue to apply the classifications and staging systems demanded by individual clinical colleagues. However, for all who diagnose tumours and work with clinical oncologists, this is an essential reference.

C FISHER

Progress in Pathology. Vol 3. (Pp 238; £35.00.) Edited by N Kirkham, NR Lemoine. Churchill Livingstone, 1997. ISBN 0 4430 5583 1.

This is an intriguing book. Its stated intention is to review the growing edge of pathology and it is certainly both enlightening and thought provoking. The sections addressing basic scientific issues, particularly those relating to molecular genetics, are not however for the faint hearted, and for many this introduction into phenomena such as genomic imprinting and microsatellite instability will challenge the imagination, as will the chapter emphasising the important role played by the stromal elements in neoplasia. There are also bold glimpses into the future, particularly with regard to information technology telepathology and the restructuring of research activity.

In contrast are excellent chapters that deal with vexing pathological problems such as trophoblastic disease, dermatoses, chronic hepatitis, and synovial fluid analysis—all of which are very much concerned with day to day diagnostic matters. There is also an interesting chapter on the spleen that should help to revive interest in this much maligned organ. Hypertension is another troublesome area in which the traditional views are critically assessed and it is obvious that most of us will have to revise our thoughts about this topic. On the other hand a valuable plea is made for retaining the necropsy, a tradition that should certainly be retained despite the alleged accuracy of imaging techniques. In general this book illustrates the important point that although pathology quite correctly retains time honoured techniques, it should always welcome new ideas, particularly if it is to retain its crucial role in medical education. It is therefore highly recommended not just to pathologists who perhaps have become a bit too set in their ways but also to the generation that will be taking the specialty into the third millennium.

F D LEE

Manual of Use and Interpretation of Pathology Tests, 2nd edn. Jean McPherson, ed. The Royal College of Pathologists of Australasia, 1997. ISBN 0 9593 3552 8.

The Royal College of Pathologists of Australasia has a distinguished record in educating the users of pathology services in the correct selection and interpretation of laboratory tests. This book is the second (and much expanded) edition of a manual first published in 1990. There is no questioning its authority—more than 50 pathologists have contributed to it and an independent, more senior group, were involved in reviewing the text.

There are two main sections: one lists conditions and their causes, together with appropriate investigations for diagnosis, monitoring, etc; the second lists individual tests (drawn from all pathology disciplines) and describes their uses and limitations. There is a short introduction describing specimen collection, reference ranges, and predictive values (though not likelihood ratios) but sensitivities and specificities are not quoted for individual tests. An opportunity has perhaps been missed to discuss the concept of critical difference—the extent of change in the result of a test that may be of clinical significance rather than be due to natural variation—a concept with which even experienced clinicians are often not familiar. Appendices include reference intervals and a list of artefactual causes of erroneous results but these are also included in the main body of text.

The introduction does not make it clear whether the book is written primarily for hospital or primary care doctors. In the UK, ACB Venture Publications has recently published a book on *Laboratory Medicine and Primary Care* and the two books share many features, although the Australian product is more detailed. Indeed, it struck me as being inappropriately detailed for doctors in primary care though no doubt, given the huge effort that has gone into producing it, the Australian College has done its market research and been encouraged by the response to the first edition.

The book is easy to use and lies open flat without the need for weights or an elbow. It will be of value to clinicians who do not have easy access to direct consultation with a clinical pathologist but should not be used as a substitute for expert advice when this is readily available.

WILLIAM J MARSHALL

Ophthalmic Pathology—An Atlas and Textbook. (4th edn, 4 vols). Spencer S. (£299.00.) WB Saunders, 1996. ISBN 0 7216 4908 4.

The previous edition of this tome was large . . . the latest is even larger and the authors have divided it into four volumes. Fortunately, there is no requirement on reviewers to read it from cover to cover in two weeks, and in reality it is not meant to be read like a novel. This is a comprehensive bench book of the old-fashioned variety, the sort pathologists turn to in desperation when the diagnosis eludes everyone in the department. It has well written chapters on the eye and its adnexae, as well as any condition that has or might conceivably have affected the eye. I found the chapters on conjunctival melanoma particularly helpful, and congenital abnormalities of the anterior chamber angle are covered well. The index is excellent—a necessity for any bench book.

This is a book for serious eye pathologists who expect to spend one or two hours gleaning information from any globe that rolls into their sight. It is a pity that the illustrations are mainly black and white, and even more that the colour illustrations are collected into plates at the end of each chapter. However, this does not really detract from the overall usefulness of the book. In brief, if you are serious about reporting eye pathology, this book must be on your shelf.

I A CREE

Notices

UK NEQAS meetings

Octagon Centre, University of Sheffield, South Yorkshire, UK

17 March 1998

One day meeting of the UK National External Quality Assessment Schemes for leucocyte immunophenotyping.

For further details please contact Dr D Barnett, Manager, UK NEQAS Leucocyte Immunophenotyping Schemes, PO Box 996, Sheffield S10 2YD, UK (tel +44 (0)114 271 1736; fax: +44 (0)114 271 1737).

18 March 1998

One day meeting of the UK National External Quality Assessment Schemes for blood coagulation.

For further details please contact Mr T A L Woods, Scheme Manager, UK NEQAS for Blood Coagulation, 305 Western Bank, Sheffield S10 2TJ, UK (tel +44 (0)114 270 0862; fax: +44 (0)114 275 8989; email: neqas@coageqa.demon.co.uk).

Application has been made to the Royal College of Pathologists for Continuing Medical Education approval and for accreditation in the Institute of Biomedical Sciences Continuing Professional Development scheme for both meetings.

Postgraduate course in gynaecological and obstetric pathology

Four Seasons Hotel, Boston, Massachusetts, USA

23-27 March 1998

A five day course primarily for pathologists and pathology residents as well as for gynaecologists with an interest in pathology will be presented by the Departments of Pathology, Massachusetts General Hospital and Brigham and Women's Hospital, Harvard Medical School. The course has category 1 accreditation for approximately 36 hours CME credit by the American Medical Association. Course fee is US$850 (residents and fellows $650).

For further details please contact Department of Continuing Education, Harvard Medical School, PO Box 825, Boston, MA 02117-0825, USA (tel: +1 617 432 1525; fax: +1 617 432 1562).