sonable doubt. Personally, I find it hard to believe that the invasive potential of melanoma is reliably predicted by the width of dermal nests of melanocytes.²

Third, I realise that Dr Cook has not proposed a subdivision of MIN that also incorporates low grade changes.² To many histopathologists, however, this will be a natural temptation in view of similar usage in other types of intraepithelial neoplasia. The incidence of MIN would then increase dramatically and stand the chance of losing credibility by becoming a depository for many minor abnormalities.

Finally, as the epidemiological aspects of melanoma are of crucial public health importance, reports will have to contain both old and new terminology into the foreseeable future.

As emphasised by Professor Mooi, the CRC Panel's study is an important analysis of observer variation in the diagnosis of thin cutaneous melanoma.³ One wonders, however, what results would have been achieved by a control group of general histopathologists using traditional atypical (dysplastic) melanoma terminology and enforced reading of an acknowledged reference source, such as the Armed Forces Institute of Pathology, *Melanocytic tumours of the skin fascicle*.⁴

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- 1 Mooi WJ. Diagnosis of thin melanoma [editorial]. *J Clin Pathol* 1997;50:179-80.
- 2 Cook MG, Clarke TJ, Humphreys S, et al. The evaluation of diagnostic and prognostic criteria and the terminology of thin cutaneous malignant melanoma by the CRC Melanoma Pathology Panel. *Histopathology* 1996;28:497-512.
- 3 CRC Melanoma Pathology Panel. A nationwide survey of observer variation in the diagnosis of thin cutaneous malignant melanoma including the MIN terminology. *J Clin Pathol* 1997;50:202-5.
- 4 Elder DE, Murphy GF. *Atlas of tumour pathology. Melanocytic tumours of the skin*. 3rd series. Fascicle 2. Washington: Armed Forces Institute of Pathology, 1991.

Dr Cook and colleagues comment:

Dr Slater makes several points in his letter about our paper and the use of the term melanocytic intraepidermal neoplasia. It may be worthwhile reiterating the processes by which the CRC Melanoma Pathology Panel came to suggest MIN for consideration. The CRC study of melanoma in pigmented lesions identified a wide discrepancy between the centres participating in the diagnosis of thin melanomas. The CRC Panel tried to identify the causes and solutions to this problem but only succeeded in providing a modest improvement in concordance. The possible implication suggested that the difference between dysplastic melanocytic lesions and melanoma in situ was not sharp and that they were not biologically distinct. Furthermore, as they were not managed differently the panel considered the need to continue to recognise them as separate entities. A term such as "melanocytic dysplasia-melanoma in situ spectrum" might have been used but an alternative—MIN—was considered more usable, and analogies with CIN were emphasised. Professor Mooi pointed out that "intraepithelial" would be more precise than "intraepidermal" and we would not argue with that. He also considered the term neoplasia to be too inclusive but we would

argue that it can have a more specific meaning when used in the context as we have defined it.

Professor Mooi has indicated that he does not personally value MIN highly because he avoids the diagnostic dilemma by hardly ever making the diagnosis of melanoma in situ, and he therefore does not have a problem in distinguishing it from dysplastic lesions (personal communication, Professor Mooi, 1997). His idea that the origin of some melanomas may be from the dermal component is interesting but not in conflict with MIN. However, it does question the use of microinvasion as an appropriate term and clearly there is much work to be done in validating either theory.

As far as Dr Slater's comments are concerned, we would like to emphasise that we put forward the MIN terminology for consideration as a possible solution to a demonstrable problem. We have tested its acceptability with a national survey but accept that further discussion and agreement is necessary before it could be a formal recommendation.

Dr Slater is also broadening his aim to include the validity of the growth phase concept of Clark *et al.*¹ We agree that this interesting theory does need more published support before it can become firmly accepted. His suggestion of the inevitable subdivision of MIN is completely against the rationale for introducing the term and we cannot accept this argument.

The existing epidemiological data for melanoma is based on current terminology and is therefore flawed, hence the need for the CRC Pathology Panel study in the first place. It is accepted that clarification of that data will take many years but it will be necessary whatever terminology is adopted.

The most important results from the nationwide survey highlight the need for more reliability in the recognition of melanoma and related lesions. This problem needs to be urgently addressed, as the detection of thin melanomas and borderline lesions has been rising rapidly.

- 1 Clark WH Jr, Elder DE, Guerry D IV, Braitman LE, Tock BJ, Shultz D, et al. Model predicting survival in stage I melanoma based on tumor progression. *J Natl Cancer Inst* 1989;81:1893-904.

Serum CagA antibodies in asymptomatic subjects and patients with peptic ulcer

We read with great interest the paper by Graham *et al.* in which, although different methodologies were used, the authors failed to find a significant association between CagA seropositivity and peptic ulcer disease or increased antral inflammation in a North American population.

We would briefly like to mention our previous experience about the patterns of antibody response to *Helicobacter pylori* infection in patients with chronic renal failure.² A group of 51 patients with different degrees of chronic renal failure was studied: histology of gastric specimens was carried out according to the Sydney's system, and serum antibodies to CagA were investigated by western blotting. The prevalence of infection was 76.4% and patients infected by *H pylori* had a greater activity of gastritis than non-infected patients ($p < 0.0001$). However, patients infected by CagA positive strains of *H pylori* did not have

a greater activity of gastritis than those infected with CagA negative strains ($p = 0.540$). No significant correlations were found with other histological features such as gastric atrophy, chronic inflammation, and intestinal metaplasia.

These results raise some questions about the pathogenetic mechanism of *H pylori* infection. Finding a specific or a unique virulence factor of infection should lead us to select patients for more specific treatment and, among the virulence factors considered, CagA has been suggested as one of the most important.³

However, the work of Graham *et al.* seems to exclude this, at least for the North American population. Moreover, our findings, obtained in patients with chronic renal failure, seem to confirm that CagA seropositivity is not necessarily related to increased antral inflammation, suggesting that the gastric microenvironment present in the stomach of these patients could interfere with the pathogenetic mechanism of *H pylori* infection.

These findings underline that clinical infectious disease usually represents an interplay between the bacteria and the host and that the "variable host" should receive more attention. We believe that further studies in a well defined population could help clarify the exact pathogenetic mechanism of *H pylori*.

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- 1 Graham DY, Genta RM, Graham DP, Crabtree JE. Serum CagA antibodies in asymptomatic subjects and patients with peptic ulcer: lack of correlation of IgG antibody in patients with peptic ulcer or asymptomatic *Helicobacter pylori* gastritis. *J Clin Pathol* 1996;49:829-32.
- 2 Rapisarda S, Figura N, Granata A, Vasquez E, Fraggetta F, Randone S, et al. Degrees of gastritis and patterns of antibody response to *H. pylori* infection in patients with chronic renal failure [abstract]. *Gut* 1996;38(suppl 2):A53.
- 3 Peek RM, Miller GG, Tham KT, Perez-Perez GI, Zhao X, Atherton JC, et al. Heightened inflammatory response and cytokine expression in vivo to cagA+ *Helicobacter pylori* strains. *Lab Invest* 1995;73:760-70.

Inflammatory pseudotumour and Rosai-Dorfman disease of soft tissue

We read the recent article by Govender and Chetty¹ with great interest. The authors report the presence of a soft tissue lesion showing combined histological and immunophenotypical features of Rosai-Dorfman disease (RDD), and an inflammatory pseudotumour. They conclude that there may be a continuum between the two entities, with RDD representing the early histiocytic lesion and inflammatory pseudotumour the later (myo-) fibroblastic lesion. The alternative hypothesis is that aberrant cytokine expression in an inflammatory pseudotumour results in transformation of histiocytes.

The authors did not mention whether investigations for mycobacteria were performed. We have recently observed the

concurrence of RDD in a lymph node alternating with areas of atypical mycobacterial infection in an HIV negative patient. The areas typical of RDD comprised a population of S100 positive histiocytes showing emperipolesis, while the other area showed the presence of sheets of S100 negative histiocytes, with areas of necrosis and suppuration, admixed with lymphocytes and plasma cells. Atypical mycobacteria were identified, in this latter area, but not in the area of RDD with appropriate special stains.

Although necrosis has been demonstrated in RDD,² the coexistence of mycobacteria and RDD has not been previously documented. Mycobacteria have, however, been cultured from RDD in bone (Miettinen, Philadelphia, USA, personal communication). Mycobacterial infection may also, uncommonly, manifest as a mycobacterial spindle cell pseudotumour.³ This may be mistaken clinically and histologically for a neoplasm.

Thus it is feasible that both RDD and mycobacterial spindle cell pseudotumour may be linked by a common aetiological process—mycobacterial infection. Clearly this possible pathogenetic link warrants further investigation, with special stains and molecular techniques.

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- 1 Govender D, Chetty R. Inflammatory pseudotumour and Rosai-Dorfman disease of soft tissue: a histological continuum? *J Clin Pathol* 1997;50:79–81.
- 2 Hayes MMM, Daya H, Uys CJ, Samson RI. Sinus histiocytosis with massive lymphadenopathy. *Arch Pathol* 1985;109:151–2.
- 3 Chen KTK. Mycobacterial spindle cell pseudotumour of lymph nodes. *Am J Surg Pathol* 1992;16:276–81.

Drs Govender and Chetty comment:

We thank Dr Nayler and Professor Cooper for their interest in our paper. In the South African context it is mandatory to perform a Ziehl Neelsen stain in almost every lesion that has an inflammatory appearance. The breast lesion that we described was no exception and the stain was, in fact, done three times. We regret not mentioning that we sought acid fast bacilli in our article. Since publication, we have encountered two additional cases of inflammatory pseudotumour of the breast.¹ Neither of these cases contained Rosai-Dorfman areas and were Ziehl Neelsen negative. While we concede that the spindle cells in an inflammatory pseudotumour may morphologically resemble the mycobacterial induced spindle cells, the two are histogenetically discrete: one being of myofibroblastic origin and the other being of the mononuclear phagocytic system. In addition, the case of Nayler and Cooper had areas of acute inflammation and necrosis that harboured atypical mycobacteria. No spindle cell areas were described and no mention is made of acid fast bacilli in spindle cells.

We feel that Rosai-Dorfman-like areas represent an inflammatory response to several immunological triggers (including mycobacteria). It is not a histological tocsin of any particular antigen. Lastly, polymerase chain reaction evidence of mycobacterial infection must be interpreted cautiously, especially in

laboratories where tuberculosis is rampant, as is the case in South Africa.

- 1 Chetty R, Govender D. Inflammatory pseudotumour of the breast. *Pathology*. [In press.]

Book reviews

Haines and Taylor Obstetrical and Gynaecological Pathology. 4th edn. Fox H, Wells M, eds. (2 volumes, £330.00.) Churchill Livingstone. 1996. ISBN 0 4430 4537 2.

The first edition of this book was published under the title *Gynaecological Pathology* by Magnus Haines and Claude Taylor in 1963. Now a two volume, multiauthor work *Obstetrical and Gynaecological Pathology* has expanded in just about every dimension with an exponential growth in the number of pages from 507 to the current 1851 (parabolic regression $r = 0.99$).

This, the fourth edition includes nine additional chapters that clarify our understanding of the morphology of premalignant and malignant glandular lesions of the cervix, metastatic tumours of the cervix and endometrium, and the pathology of the peritoneum and secondary Müllerian system. A chapter on the pathology of the secondary yolk sac, an organ that I suspect few pathologists in this country encounter in routine practice, is also welcome. Inevitably, I expect that rapid scientific advance will ensure that the chapters on quantitative pathology, molecular biology, and immunohistochemistry will become rapidly dated and doomed to obscurity long before a fifth edition of this work will be available.

The standard of photography is somewhat variable but this is probably a common problem in multiauthor texts, as is the delay between the author completing the chapter and publication—the most recent reference I could find was from 1994. These were the most obvious flaws in what is set to be a standard bench book, essential in every diagnostic anatomic pathology laboratory dealing with gynaecological specimens.

M B HEATLEY

Principle and Practice of Medical Laboratory Science. Volume 1. Leong A S-Y. (Pp 172; £25.00.) Churchill Livingstone. 1996. ISBN 0 443 05369 3.

The expected chapters for a book entitled “Principle and Practice of Medical Laboratory Science” are all here and include section preparation and staining, as well as microscopy. There is a helpful exposition on specimen cut-up procedures, which should appeal to junior pathologists and medical laboratory scientists alike. The overall flavour of the book is one of practical guidance, linked to diagnostic usefulness. Confusingly, the most substantial chapter entitled “Preparation of Tissue Sections” contains the staining methods. These also give the principles of the various reactions, but for some obscure reason this laudable precept seems to have been abandoned in the section on “Miscellaneous Stains”. Equally perplexing is why some solutions appear with the appropriate

method, while others are shown only in the Appendix.

Particularly useful chapters are those covering light and electron microscopy, and diagnostic immunocytochemistry. The “Laboratory Safety” chapter addresses the topic helpfully and succinctly, but it is unfortunate that it fails to make the vital point that chlorite disinfectants are unsuitable for tuberculous material.

It is a rare text that is without error and this book is no exception—for example, on page 58 the reference for Harris’s haematoxylin would, by my calculations, result in Dr Harris being 115 when he concocted his well-known nuclear stain; and on page 65 an incorrect formula for the standard alcian blue solution would result in a too strong a stain by a factor of 10.

The book is soft-back with a reasonably large page size making it easy to use on the bench. A bold typeface is used for the main text but, irritatingly, a smaller, less distinct typeface is employed for legends to figures and technique detail. This tends to make for arduous reading and was exemplified by the algorithm on page 116 (fig 6.6) that was virtually incomprehensible.

In summary, the book is well written in an authoritative style that engenders a feeling of confidence that the author knows what he is about. The price is a relatively modest one. On the other hand, its appeal is always going to be a limited one—given its restricted subject coverage. Therefore, it is unlikely to serve as a reference book, either in laboratory or college terms. As a bench book its value is readily apparent but it is not quite comprehensive enough to be used alone. The main appeal will undoubtedly be to newcomers to histology who require a straightforward, reliable, and above all comprehensible guide to its mysteries. The trouble is that this type of publication tends to be passed on by the possessor to oncoming neophytes, as opposed to their purchasing a new copy; a scenario guaranteed to engender melancholia in publishers.

H COOK

Clinical Practice of Transfusion Medicine. 3rd edn. Petz LD, Swisher SN, Kleinman S, Spence RK, Strauss RG, eds. (Pp 1115; US\$159.95.) Churchill Livingstone. 1996. ISBN 0 443 08981 7.

The first two editions of “Petz and Swisher” firmly established this text as a standard for the field of transfusion medicine. This third edition encompasses three additional editors, many more chapters, and a complete revision in its approach to the readership.

The emphasis, even more firmly than in the first two editions, is on clinical practice, with the result that some of the traditional topics have virtually disappeared—including those that pertain to the development of the laboratory aspects of transfusion that no longer need appear in a treatise dealing with clinical practice, prime examples being lectins and polyagglutinability. These, together with the traditional description of erythrocyte metabolism and its relationship to blood preservation, have disappeared between the second and third editions, but in return we have a much more structured approach to the subject.

After an introduction dealing briefly with history and giving a general overview, specific sections deal with biological principles such as blood groups and the HLA system, practical and organisational aspects, transfusion in spe-