inflammatory cytokines such as interleukin 1, tumour necrosis factor, and gamma interferon,⁴ T cell activation,⁵ conversion T of lymphocytes from naive to memory cells⁶ and viral infection.⁷ One or more of these factors may be responsible for causing the increased expression of ICAM-1 that occurs during liver allograft rejection. The factors regulating ICAM-1 expression in normal hepatocytes remain uncertain.

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- 1 Smith MEF, Thomas JA. Cellular expression of lymphocyte function associated antigens and the intercellular adhesion molecule-1 in normal tissue. *J Clin Pathol* 1990;43: 893-900.
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Drs Smith and Thomas comment:

We thank Hubscher and Adams for clarifying the ICAM-1 expression they found in normal liver and agree that the factors they mention may be responsible for the upregulation of ICAM-1 they detected in the rejecting liver allografts.

Thrombocytosis and follicular thyroid carcinoma

We report a rare case of thrombocytosis associated with thyroid follicular carcinoma.

Thrombocytosis associated with malignant disease has been described in lung carcinoma, pleural mesothelioma, gastrointestinal tract carcinoma, lymphoma and acute leukaemia, hepatocarcinoma, neuroblastomas, histiocytosis and other epithelial cell origin tumours; it has not been described in association with thyroid carcinoma.¹⁻⁴

A 72 year old woman was diagnosed as having a follicular thyroid carcinoma because of progressive enlarging goitre. A fine needle aspiration biopsy specimen was consistent with follicular lymphoma, the diagnosis of which was confirmed when the excised thyroid was examined microscopically. Pre-

operative tests had shown increased numbers of platelets ($746 \times 10^9/l$ and $731 \times 10^9/l$, respectively). The haemoglobin concentration was 14.4 g/l, the haematocrit 41.8%, while red blood cell count, white cell count, mean corpuscular volume, mean corpuscular haemoglobin concentration, and serum ferritin and serum iron concentrations were normal. Haemostatic evaluation showed normal prothrombin and partial thromboplastin times. FDP concentrations were within the normal range. Two months after surgical resection the platelet count returned to normal.

Most cases of thrombocytosis secondary to malignant disease have a platelet count from 4 to $6 \times 10^9/l$. Iron deficiency may also contribute to an increased platelet count.^{2,3} In other cases slow activation of clotting and disseminated intravascular coagulation have occurred and FDP was detectable.^{3,5} It was not specified, however, whether these cases also had thrombocytosis. Although haemorrhagic and thrombotic episodes are characteristic of primary thrombocytosis,^{2,4} in these patients platelet production probably occurs in the tumour itself or in bone marrow by a protein produced by the neoplasm (thrombocyte stimulating factor) (TSF).^{2,3} The occurrence of unexplained thrombocytosis precludes malignancy.⁴

We suggest that clinicians should be aware of this rare sign in malignant diseases.

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- 1 Spiegel SC, Mooney LR. Extreme thrombocytosis associated with malignancy. *Cancer* 1977;39:339-41.
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Malignant melanoma and stage of disease

The paper by Calder, Campbell, and Plastow addresses an important practical problem that is faced by surgical pathologists when examining cases of malignant melanoma.¹ Although it does not detract from the main theme of the publication, however, the first sentence is incorrect. The single most important prognostic factor in determining the outcome of any malignant melanoma is the stage of the disease at the time of diagnosis. The tumour thickness is probably the second most important prognostic indicator and is

the best indicator of metastatic potential in stage I melanomas.² Unfortunately, this simple but very important fact about the role of the stage of the disease is often forgotten amid the plethora of articles, meetings, and examination questions which emphasise Breslow's thickness measurements or Clark's level of invasion.

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- 1 Calder CJ, Campbell AP, Plastow FR. Measurement techniques for melanoma: A statistical comparison. *J Clin Pathol* 1990;43:922-3.
- 2 Rosai J. Malignant melanoma. In: *Ackerman's surgical pathology*. New York: CV Mosby Co, 1989:135.

Drs Calder, Campbell and Plastow comment:

We read with interest the comments on our recent paper and agree that the stage of the disease is of prime importance. Our paper was addressing the microscopic measurement of the Breslow thickness of excised melanomas where the stage of the disease might not necessarily be known—a situation commonly encountered by surgical pathologists.

BOOK REVIEWS

Breast Pathology: benign proliferations atypias and in situ carcinomas. RE Fechner, SE Mills. (Pp 190; book \$170.00; slides \$170.) Raven Press. 1990. ISBN (Book) 0-89189-298-2. ISBN [Slides] 0-89189-307-5.

This book comes at a time when British pathologists are beginning to come across the types of lesions, such as in situ carcinoma and atypical hyperplasia, that are more common in screening for breast cancer than in the general symptomatic workload. The illustrations are, for the most part, good, and many of the rarer lesions which can cause diagnostic difficulties, if unrecognised, are included. In contrast to a number of other texts, the lesions are in general recognisable as the entity they are stated to portray, no mean feat in an area as controversial as atypical hyperplasia.

A refreshing finding is the adoption of nomenclature similar to that of the National Screening Programme and hence the avoidance of confusing terminology such as the British "epitheliosis" and the American "papillomatosis". These lesions are now correctly termed "hyperplasia" and qualified by the terms "typical" or "atypical".

If criticism has to be found in this work one can only say that some of the more modern oncogene work has not been mentioned, the typing of in situ ductal carcinoma of comedo-type is slightly suspect, being primarily dependent on necrosis; some of the references at the chapter end are not referenced in the text; and the book is a little expensive, but these are minor criticisms in a useful diagnostic text.

The accompanying optional set of slides

are also of good quality with the exception of some of the low power shots and I would not be surprised if some of them occasionally find their way into lectures. On the whole, therefore, this book is highly recommended as a reference atlas and text for the diagnostic histopathologist faced with difficult proliferations in the duct system of the breast.

CLIVE WELLS

Histophysiology of the Circulating Platelet. Advances in Anatomy Embryology and Cell Biology. Vol 120. Ed LJ Wurzinger. (Pp 96; soft cover DM 68.00.) Springer. 1990. ISBN 3-540-52258-1.

This is a single author short monograph reviewing the basic pathophysiology of the circulating platelet; it also gives the personal views of the author with respect to the role of platelets in rheology and blood flow. As usual with these publishers, this is a beautifully produced monograph with about 40 excellent black and white photomicrographs of platelets in various forms of activation. The text is aimed primarily at pre-clinical scientists, particularly physiologists and anatomists concerned with the basic concepts of platelet activation. Overall, it is a very readable and well referenced review but does not relate specifically to congenital or acquired disease states. In the last chapter the author discusses his own particular research interest which is the role of platelets in the development of thrombosis under different flow and shear conditions. This text is not specific enough for the general pathologist or haematologist interested in platelets but may primarily be of help to the platelet research worker who is involved in electron microscopy for studying platelet function and also for rheologists interested in the varying concepts of atheroma.

SJ MACHIN

Familial Adenomatous Polyposis. Ed L Herrera. (Pp 402; \$129.50.) Alan R Liss Inc. 1989. ISBN 0-471-56225-4.

Familial adenomatous polyposis (FAP) is the term recommended by the Leeds Castle International Polyposis Group for a condition which serves as an important model for the study of cancer and cancer genetics in general. It is fitting that the person instrumental in stimulating the recent great leap in research activity and enlightenment in the field of polyposis should edit this book, the most important on FAP since Dr HJR Bussey's monograph of 1975.

This is an encyclopedia, with each chapter written by an authority on the topic in question. Forty seven chapters cover almost every conceivable aspect of FAP, often including information on wider but related issues. Of interest to pathologists are chapter 4 (Watne and Sohrabi) on all the various inherited colonic polyposis syndromes and chapter 5 (Hamilton) on the pathology of adenomas and malignant polyps. These are generally excellent, although I differ with the statement that, "angio-lymphatic involvement is an indication for surgical resection" after carcinoma is found in a colonoscopic

polypectomy specimen. The later chapters cover specific matters of importance in the counselling of patients, the organisation of polyposis registries, and surgical and laser therapeutic methods. There are chapters on the important topics of extra-colonic manifestations (Bulow), comparative epidemiology with Japanese data (Utsunomiya), and retinal lesions, with beautiful colour illustrations. The only omission is specific coverage of the management of adenoma and carcinoma of the ampulla of Vater, although these are alluded to in general terms.

The stature of the contributors has ensured that this is a reference work of the highest order. It should not be regarded as simply a book for the super-specialist; the later chapters are up to the minute (1990) reviews of a wide range of topics of interest and value to pathologists, including the molecular genetics, genesis and biology of neoplasia (such as mucins, lectins, oncogenes, protein kinases and, tissue culture technology). It is a book that libraries, larger departments, and interested persons should buy.

IAN C TALBOT

Platelet Heterogeneity. Biology and Pathology. Ed J Martin, A Trowbridge. (Pp 270; 78 figs; DM228.00.) Springer. 1990. ISBN 3-540-19602-1.

Like any biological population, whether of cells or of whole organisms, the blood platelets are notably heterogeneous with regard to size, density, functional capacity, biochemical composition and survival. Platelet experts have argued for many years over the extent to which this heterogeneity is "congenital" or "acquired". Is it predominantly due to influences on their production from megakaryocytes or to stimuli which they encounter during their lifespan in the circulation? This argument formed the basis of a workshop held under the auspices of the European Society of Clinical Investigation in Graz in April 1988. The present volume contains papers written for publication after the meeting by the chief participants (and also by two groups who were unable to be present at the meeting itself), together with edited discussions. It might be thought that this editorial approach would result in a loss of immediacy, but in fact much of the cut and thrust of the debate seems to have been retained. The inclusion of technical questions and answers at the end of most of the papers contributes to this, and also serves to emphasise the influence of such variables as handling artefacts and species differences in muddying the waters.

Although two and a half years have passed between the meeting and the publication of these proceedings, progress in this complex field has not been so rapid as to deprive them of much of their interest. The book provides much food for thought for all those interested in platelet production and ageing in health and in haemorrhagic and thrombotic diseases. Among the many theoretical concepts advanced here which are still in need of experimental testing, perhaps the most challenging is that of Trowbridge, that platelets are shed from megakaryocytes, not in the bone marrow, as has been generally believed for the past 80 years, but in the pulmonary circulation.

RM HARDISTY

NOTICES

16th European Symposium on Hormones and Cell Regulation

September 23-26, 1991

Mont Ste Odile, Alsace, France

Topics include: nitric oxide and cyclic nucleotides; factors in the development of the nervous system; hormonal factors regulating embryonic development; GTP-binding proteins; receptors and channels; role of protein kinases and phosphoprotein phosphatases in signal transduction

Further information from: Dr B Hamprecht, Physiologisch-chemisches Institut der Universität, Hoppe-Seyler-Str. 4, D-7400 Tübingen, Germany
Telefax: 49-7071-293361.

12th Annual Conference of Australian Dermatopathology Society

Interface Dermatopathology

St Vincent's Hospital,
Melbourne, Australia
Friday, 27 September—Sunday,
29 September, 1991

Guest Speaker: Professor AB Ackerman, New York University Medical School. The Conference will highlight clinical and laboratory contributions to diagnosis and will include five minute presentations and a poster display. Participation is invited.

For further information, please contact:
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Australia. Telephone: +61 3 347 9633
Fax: +61 3 347 4547.

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