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Platelet aggregation in Raynaud's phenomenon

Biondi and Marasini recently reported that patients with Raynaud's phenomenon showed increased platelet aggregation induced by serotonine and adenosine diphosphate (low doses), and normal platelet aggregation induced by adrenalin.[1]

We also investigated adrenaline (5 μg/ml) induced platelet aggregation in 20 healthy volunteers, 27 patients with primary Raynaud's phenomenon, and 25 patients with obliterative atherosclerosis. We registered the time to the start of aggregation rather than its intensity. The mean (SD) figures were 34·2 (5·57) seconds in Raynaud's phenomenon and 37·8 (5·54) seconds in obliterative atherosclerosis. The time registered to the start of aggregation was significantly shorter in Raynaud's phenomenon compared with that in normal adults (46·3 (4·37) seconds ($p = 0·01$) and even with that in atherosclerotic patients ($p = 0·05$)).

It is interesting to note that both the time to the start of aggregation and its intensity are abnormal in patients with Raynaud's phenomenon. The observation of both variables may be useful in such patients.

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Immature lymphocytes in transient erythroblastopenia of childhood

The report by Foot *et al* on bone marrow lymphocytes in transient erythroblastopenia of childhood (TEC)[1] is important because it redirects our attention to the patterns of immature lymphocytes which may be found in children's bone marrows. Such cells were once called haematogones. A recent study by Longacre *et al* described detailed studies of these cells in 12 children with a variety of malignant and non-malignant disorders, among which were three cases of red cell aplasia.[2] They showed a complex pattern of phenotypic and morphological appearances of these lymphoid cells. These observations highlight what should now be axiomatic for haematologists: cell marker studies should not be used to make a diagnosis of leukaemia, but, once such a diagnosis has been made by the usual methods, may give an indication of what sort of leukaemia it is.

Foot *et al* also wonder why bone marrow lymphocytosis should occur in TEC. Among a range of possibilities is the fact that *normal* children of this age may have up to, or more than, 40% lymphocytes in their marrow. Removal of the erythroblast population, say 20%, could result in the lymphocytes reaching 50% of the total nucleated cell population without any apparent reduction in the cellularity of the sample, and without an absolute increase in the number of lymphocytes. "Lymphocytosis" in the bone marrow is of course relative. Nevertheless, the increased proportion of early lymphoid cells in the mononuclear cell population obtained by density separation does suggest that it may be "a consequence of an outpouring of immature lymphocytes," unless a corresponding decrease in the absolute number of mature lymphocytes has occurred. Perhaps all three of these processes contribute to the increased proportion of immature lymphoid cells in the bone marrow of those with TEC.

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Immunoalkaline phosphatase technique in renal pathology

It was a pleasure to read the article by Jackson *et al* regarding the immunoalkaline phosphatase technique on formalin fixed renal biopsy specimens. We are writing merely to comment on two problems outlined by the authors in their article.

The problem of weak or negative staining encountered in cases of anti-glomerular basement membrane disease (anti-GBM) may result from the fixative used; buffered formalin has a stronger effect on the antigenicity than acid formalin and also requires a greater digestion time to unmask the epitopes. By using formol saline, we have much shorter digestion times in trypsin and the staining of complement is usually stronger. We find C3 of more diagnostic value than IgG in cases of anti-GBM disease probably because of the lower background staining.

The other problem of spurious staining of plasma in capillary loops can be reduced or even stopped by washing the specimen in physiological saline for around one hour before fixation.

We use immunoperoxidase routinely on renal biopsy specimens as well as immunofluorescence performed in another department. Having read the article by Jackson *et al*[1] we will be assessing the immunoalkaline phosphatase technique.

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BOOK REVIEWS

Diagnostic Seminars in Pathology. Vol 1. Ed E Grundmann. (Pp 318; 45 tables; soft cover DM 89.). Gustav Fischer. 1990. ISBN 3 437 11336 4.

This new series "summarises articles previously published in pathology—research and practice". Volume I contains nine articles on neuroendocrine tumours, and single articles on prostatic carcinoma, cytological diagnosis of lung cancer, myositis, viral encephalitides, storage disorders, electron microscopy of large cell undifferentiated and giant cell tumours, Niemann-Pick diseases, chronic renal failure, and the use of lectins in histopathology. With such diverse subject matter this book may not immediately appeal to pathologists as "an up to date reference source", but I must confess to finding several of the articles most informative and helpful. The chapter on neuroendocrine tumours of the gastrointestinal tract is a gem; if you are not quite clear about enterochromaffin-like (ECL) hyperplasias and neoplasias of the stomach in relation to various stimuli then this chapter will sort things out. It concludes with a most useful and erudite discussion of the terminology of gut neuroendocrine tumours and the use of the term "carcinoid". The chapter on phaeochromocytomas and paragangliomas begins with very clear definitions of these tumours. The chapter on thymic neuroendocrine neoplasms is especially useful in its discussion of the differential diagnosis of such tumours. This is certainly a book that candidates for final MRCPath would be well advised to dip into.

DA LEVISON

Macro Techniques in Diagnostic Histopathology. DG Lowe, IM Jeffrey. (Pp 144; £40.) Wolfe. 1990. ISBN 0 7234 0945 5.

Many histopathologists learn to deal with specimens in an apprenticeship of varying length, collecting tips haphazardly from older colleagues whose skill was similarly acquired. Even in maturity our reports may not always make it clear to clinicians or reviewing pathologists precisely what we found.

Following the advice of this attractive, highly practical guide to specimen examination, description and block selection, should result in consistent high quality macroscopical reports and proper blocks.

Fourteen short chapters on different systems are written in an easy, carefully edited style with excellent closely matched photographs, tables, and diagrams. The 100 specimens illustrated well represent the daily work of the average histopathology laboratory. Procedures suggested are consistent and reasonable, although some might baulk at the number of blocks advocated. Clarification of why certain blocks are taken might have been desirable. None the less I wish I had this volume when I started.

AM MACKAY

Progress in Reproductive and Urinary Tract Pathology. Vol 1. Ed I Damjanov, AH Cohen, SE Mills, RH Young. (Pp 217; 49.) John Wiley. 1990. ISBN 0 938607 13 8

The editors have embarked on a new series of books containing "review articles . . . written