noted to the nearest tenth of a millimetre. Then the slide is moved to align the deepest part of the tumour to the same fixed point and a second reading taken. The tumour thickness can then be derived by subtracting one reading from the other (Figure).

This is a simple and reliable method. The measurement can be made using any combination of objective and eyepiece magnification and tube length. No conversion factors or calculations have to be used, thus eliminating further sources of error.⁶ The measurement is only made to the nearest tenth of a millimetre but in most cases the lesion can be easily assigned to one of the three prognostic categories (<0.76 mm, 0.76–1.50 mm and >1.50 mm) without measuring to the nearest hundredth of a millimetre. The only problem we find amongst our colleagues is uncertainty regarding the working of Vernier scales. These were invented by the Burgundian Pierre Vernier (c. 1580–1637) and described in 1631 in his book *La Construction, l'usage, et les propriétés du quadrant nouveau de mathématique*.⁷ Briefly, there is a short scale 9 mm long, divided into 10 equal 0.9 mm long parts, sliding on a scale graduated in millimetres. When one examines the short scale one of its 10 graduations will be exactly in line with a mark on the long scale. This then gives the decimal place value between 0.1 and 0.9 mm.

If one is unsure of the technique, or of the accuracy of the scales on a particular microscope, then a slide bearing a known standard scale can be employed, as one would in calibrating an ocular micrometer. We hope that histopathologists will now be encouraged to use this simple technique to report the thickness of melanomas in all cases.

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Routine bone marrow aspirations during maintenance treatment in acute lymphoblastic leukaemia do not improve survival

In the paper by Franklin¹ the value of routine bone marrow aspirations in terms of their contribution to prognosis has been questioned.

In 109 children with acute lymphoblastic leukaemia diagnosed in our department between January 1976 and June 1982 and treated with AIL-AIEOP (Italian Paediatric Cooperative Group for Therapy of Acute Leukaemia) 7601, 7602, 7901, 7902, and 7903,² 1308 routine bone marrow aspirations and 108 non-routine bone marrow aspirations were performed during maintenance treatment and after stopping treatment. Routine bone marrow aspirations were carried out at two to three monthly intervals; non-routine bone marrow aspirations were performed when haematological relapse was suggested by any of the following: peripheral blood count anomalies (circulating blast cells, haemoglobin concentration less than 10.5 g/dl, lymphocytosis more than 70%, platelet count less than $120 \times 10^9/l$), hepatomegaly (liver ≥ 3 cm) or splenomegaly (spleen ≥ 1 cm), or both, not attributable to infection, malaise, bone pains, or extramedullary relapse.

Nine out of 1308 routine bone marrow aspirations (0.7%) indicated relapse and 21 out of 108 non-routine bone marrow aspirations (19.5%) confirmed the suspicion of haematological relapse ($p < 0.001$). Apart from the presence of circulating blast cells, the predictive parameters for haematological relapse were: thrombocytopenia ($p < 0.003$), bone pains ($p < 0.004$), hepatomegaly ($p < 0.04$), and splenomegaly ($p < 0.02$). Of 30 relapsed patients, 27 died and three are surviving

(follow up to 31 December 1982) after 15, 12, and 24 months. (In the last two cases bone marrow transplantation was performed after the second remission.) The mean survival duration after haematological relapse was 8.7 ± 5.5 months in the routine bone marrow aspiration group and 9.5 ± 8.1 in the non-routine bone marrow aspiration group (NS).

Our study which was carried out on a considerable number of bone marrow aspirations confirms that routine bone marrow aspiration is not an effective diagnostic procedure for detecting haematological relapse. The duration of survival was the same both for patients whose relapse was detected by routine bone marrow aspiration and for those in whom relapse was suspected clinically.

In conclusion, as there is no evidence of any advantage from routine bone marrow aspirations, which are a stressful procedure for children, their periodical performance should be discontinued.

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Book reviews

Immunological Investigation of Lymphoid Neoplasms. Practical Methods in Clinical Immunology Series. Vol 6. GT Stevenson, JL Smith and TJ Hamblin. (Pp 91; £18). Churchill Livingstone. 1983.

There is a tendency amongst oncologists to categorise cases of non-Hodgkin's lymphoma according to their prognosis and to

plan treatment accordingly. While this pragmatic approach has its merits, advances in our understanding of this group of diseases and in the evaluation of therapy can only be achieved using a scientific basis for their classification. This small volume will greatly assist clinicians and pathologists alike to this effect and is thus warmly recommended to all who deal with this complex group of diseases. The book clearly explains the basis of the immunological investigation of lymphoreticular neoplasms and usefully guides the clinician in the selection of tests. There are one or two minor, yet irritating, errors which should be corrected in the forthcoming editions. Amongst these are the use of the term "centrocytic/centroblastic" rather than the converse "centroblastic/centrocytic", the suggestion that 2 mm cubes of tissue are desirable for cryostat sections, whereas in fact these sections should be much larger, and the designation of Mediterranean lymphoma as a plasmacytic neoplasm. The long section on the preparation and use of anti-idiotypic antibodies is somewhat ahead of its time, whereas, in some other ways, the book is already slightly out of date. For example distinction between poorly differentiated carcinoma and lymphoma is now easily achieved with the use of appropriate monoclonal antibodies which can also be used to phenotype cells directly on the slide in cytocentrifuge or smear preparations. This is a reflection of the rapid development of this subject rather than a criticism of the authors. In recommending this book, I am conscious of the fact that the laboratory resources so elegantly displayed are not available to many oncologists and pathologists dealing with lymphoreticular disease. One would hope that this book would act as a catalyst for the expansion of immunology services throughout the country.

PG ISAACSON

giving details of five unusual cases of amyloid disease, and a variety of therapeutic regimens are presented. The contributors are mainly British, North American, Scandinavian, Dutch, and Israeli.

The emphasis is heavily on amyloid associated with other disease at the expense of amyloid of immunoglobulin or endocrine origin. The treatment reported is on the whole disappointing and it is sometimes difficult to separate the effect on the primary disease from the effect on the amyloid.

The editors give a short comment on the clinical section. A similar review of the other sections would have increased the value of the book for the general reader.

JS KENNEDY

Genetic Errors of Glycoprotein Metabolism. Ed Paolo Durand and John S O'Brien. (Pp 220; DM 84; US\$34.70.) Springer. 1983.

This book gives an up to date account of the four inborn errors of metabolism (fucosidosis, mannosidosis, sialidosis, and aspartylglycosaminuria) which can be described as glycoproteinoses on the basis of the specific enzyme deficiencies which cause them. Accounts of GM₁ and GM₂ gangliosidosis are also included on the grounds that, although these are disorders of sphingolipid catabolism, glycopeptides and oligosaccharides accumulate along with the gangliosides. Mucopolipidosis II (I-cell disease) and mucopolipidosis III (pseudoHurler polydystrophy) in which there is failure of the normal mechanism for the reuptake of secreted lysosomal enzymes also feature. There is a chapter on the recently delineated Salla disease in which an increased urinary excretion of free sialic acid is the biochemical marker for a neurological syndrome comprising severe mental handicap, ataxia, dysarthria, and disorders of muscle tone.

Both the clinical and biochemical aspects are well covered and up to date. The subject matter is well illustrated with diagrams, clinical photographs, x-rays and reproductions of electron micrographs.

This book will be a very valuable reference work for clinical geneticists and biochemists whose interest is in the inborn errors of metabolism, as well as to anyone trying to develop a research programme in this or related areas of human biochemical genetics.

It is also to be hoped that paediatricians and specialists in mental handicap will pay Durand and O'Brien's book more than passing attention because it is in these areas that a high level of clinical awareness of the individually rare inherited metabolic diseases is particularly needed. The experienced clinician's "index of suspicion" remains our most valuable screening test in many areas. Precise diagnosis which implies enzymological confirmation is necessary for prognosis and genetic counselling. Exact definition at the current attainable diagnostic level is a basis for studies of genetic and clinical heterogeneity from which further refinements may come in diagnosis, prognosis, and eventually in treatment. This work defines the present position for the glycoprotein storage diseases admirably.

RWE WATTS

Germ Cell Tumours. Clinics in Oncology. Vol 2, No. 1. (Pp 284; £11.75.) WB Saunders Company Limited. 1983.

This elegantly produced book is a full account of the pathology, clinical presentation, and management of germ cell tumours: It embodies the vast experience of the Charing Cross Hospital which is world renowned for its contribution to the subject. The discussion of pathology includes the role of immunocytochemistry and studies on experimental teratomas. Management is dealt with in general, but there are also useful chapters on intracranial germ cell tumours, and the management of these tumours when they are present in the mediastinum and retroperitoneum. Although understandably the Charing Cross experience is fully represented, the discussion is not parochial, and all the chapters refer widely to the international literature.

This book contains what one needs to know about the present state of the art in this branch of cancer medicine. The editors are to be particularly congratulated on the way repetition has been avoided in the text, and the high standard of the figures and their legends.

JS MALPAS

Pathology of Drug-Induced and Toxic Disease. Ed Robert H Riddell. (Pp 682; illustrated; £55.) Churchill Livingstone. 1982.

There is no hesitation in recommending the

Amyloidosis E.A.R.S. Proceedings of the First European Amyloidosis Research Symposium. Ed Colin R Tribe and Paul A Bacon. (Pp 215; paperback £25.) John Wright & Sons Ltd. 1983.

This book reports research into amyloid disease over the period 1979-1981 as the proceedings of the Symposium held in Bristol in 1981. The chief topics are the biochemistry of AA and SAA including dequation of SAA, the P component, and some experimental models and mechanisms. There is also a clinical section