

Letters

Table Total amylase, P, and S type amylase activities in patients with liver disease

Group	Mean (SD)		
	Total amylase (IU/l)	P type isoamylase (IU/l)	S type isoamylase (IU/l)
A Normal volunteers (n = 30) reference range	199 (66) 77-317	110 (44) 25-184	89 (45) 1-184
B Patients without liver disease (n = 18)	194 (47)	96 (30)	98 (47)
C Patients with liver disease (n = 30)	234 (88)*	127 (69)	107 (79)
Primary biliary cirrhosis (n = 7)	210 (84)	92 (38)	118 (63)
Chronic active hepatitis (n = 9)	242 (74)	164 (71)**	78 (47)
Alcoholic cirrhosis (n = 9)	253 (121)	122 (87)	131 (116)

Patients v normal controls; *p < 0.1; **p < 0.05.

While evaluating the "Phadebas" isoamylase kit (Pharmacia) by comparing it with electrophoresis,¹² we applied the inhibition technique to study the serum isoamylases in patients with liver diseases. Sera were also obtained from normal volunteers and patients without liver diseases (Table). The diagnosis was based on clinical examination and laboratory findings. The distribution of men to women was similar in the three groups. We separated those who suffered from primary biliary cirrhosis, chronic active hepatitis, and alcoholic cirrhosis.

Our findings differed from those of Bhutta and Rahman² and agreed with those of others.^{1 6 13} Although MacGregor and Zakim¹ reported that hyperamylasaemia associated with chronic active hepatitis may be of salivary gland origin, as part of the spectrum of extrahepatic manifestations of the disease, we found that it was of pancreatic gland origin.

Finally, we found that three of our patients with alcoholic cirrhosis had S type hyperamylasaemia, whereas only one had P type hyperamylasaemia. The findings of this study suggest that in patients with liver disease and hyperamylasaemia the prominent type of isoenzyme is that of pancreatic origin, with the exception of patients with alcoholic cirrhosis where the prominent isoenzyme is that of S type. Further details of our study, including comparison of results between volunteers and patients and correlation with an electrophoretic technique for isoamylase separation, will be published elsewhere.

EB TSIANOS*
MT JALALI†
AH GOWENLOCK†
JM BRAGANZA*

University Departments of

*Gastroenterology and †Biochemistry,
Manchester Royal Infirmary, Manchester
M13 9WL, England

References

- MacGregor IL, Zakim D. A cause of hyperamylasaemia associated with chronic liver disease. *Gastroenterology* 1977;72: 519-23.
- Bhutta IH, Rahman MA. Serum amylase activity in liver disease. *Clin Chem* 1971; 17:1147-9.
- Meites S, Rogols S. Amylase isoenzymes. *CRC Crit Rev Clin Lab Sci* 1971;2:103-38.
- Karn RC, Wise RJ, Merritt AD. The issue origins of serum and urinary α -amylase. *Arch Biochem Biophys* 1976;175:144-52.
- Skude G. Sources of the serum isoamylases and their normal range of variation with age. *Scand J Gastroenterol* 1975;10:577-84.
- Fahrenkrug J, Staum-Olsen P, Magid E. Immunoreactive trypsin and pancreatic isoamylase activity in serum of patients with chronic renal failure or hepatic cirrhosis. *Clin Chem* 1981;27:1655-7.
- Kumar S, Gupta MC, Tyagi SP. A study of serum amylase in infective hepatitis. *J Indian Med Assoc* 1970;55:194-7.
- Lesser PB, Warshaw AL. Differentiation of pancreatitis from common bile duct obstruction with hyperamylasaemia. *Gastroenterology* 1975;68:636-41.
- Zeive L. Clinical value of determinations of various pancreatic enzymes in serum. *Gastroenterology* 1964;46:62-7.
- Warshaw AL, Bellini CA, Lee KH. Electrophoretic identification of an isoenzyme of amylase which increases in serum in liver diseases. *Gastroenterology* 1976;70:572-6.
- Nord HJ, Weis HJ, Colle H. Untersuchungen zum stoffwechsel der serumamylase. *Verh Dtsch Ges Inn Med* 1973;79:868-71.
- Tsianos EB, Jalali MT, Gowenlock AH, Braganza JM. Ethnic "hyperamylasaemia": clarification by isoamylase analysis. *Clin Chim Acta* 1982;124:13-21.
- Cummins AJ, Bockus HL. Abnormal serum pancreatic enzyme values in liver disease. *Gastroenterology* 1951;18:518-29.

Cryptosporidium sp a "new" human pathogen

We were delighted to see our paper in print

and thank you for doing such an excellent job of the presentation. Unfortunately, on reading the published version, two errors were apparent, which we feel are of sufficient importance to require an addendum. The first is an editorial correction in the final proof copy. This had to be dealt with in great haste, and the nature of your correction was not realised. The error is on page 1323, right hand column, in which the word "not" has been incorrectly inserted. The point may be a pedantic one, but it is widely agreed that although extracytoplasmic, the fact that the parasite is enveloped by the outer cell membranes makes it intracellular. The word "not" should, therefore, be deleted from the text.

Secondly, in Table 2, page 1327, right hand column. I have been asked to point out that Liverpool did give a figure for total positives (41), although the denominator figure had not been given.¹

DP CASEMORE
Public Health Laboratory,
Ysbyty Glan Clwyd,
Bodelwyddan,
Rhyl,
Clwyd LL18 5UJ

Reference

- Baxby D, Hart CA. Cryptosporidiosis. *Br Med J* 1984;289:1148.

Book reviews

Cellular and Molecular Aspects of Aging: The Red Cell as a Model. Progress in Clinical and Biological Research. Vol 195. Ed JW Eaton, Diane K Konzen, JG White. (Pp 464; £52.) Alan R Liss Inc. 1985.

Because of its readily measured biological processes and its finite life span, the red blood cell is a good model for investigating the cellular and molecular aspects of aging. This was the theme of a conference held in Minneapolis in September 1984 with some 80 participants. The 30 papers presented at that conference, together with a verbatim record of the discussions are here presented in the *Progress in Clinical and Biological Research* series. Is aging controlled by 2, 3-DPG and determined by the DPG gene, as suggested by Brewer, or is the process determined by a programmed mechanism of progressive loss of genetic information? The problem of determining which is cause and which is effect was well illustrated in a study on the role of raised red cell calcium concentration: innocent bystander or kiss of death? In the end the validity of using the relatively

simple anucleate red cell as a model for the complexity of the whole organism remains questionable: it would be remarkably convenient if the red cell life span in any one subject were a guide to his ultimate survival, but as pointed out by JM Rifkind and colleagues, although an 80 year old donor does not have 80 year old red cells, his red cells have been produced by an 80 year old erythropoietic system.

Proceedings of conferences tend to have a limited life span, and relatively few merit publication. This is a rare exception, a fascinating subject, with some excellent reviews as well as original data by eminent workers: recommended reading for all haematologists.

SM LEWIS

Techniques in Immunocytochemistry. Vol 3. Ed Gillian R Bullock, Peter Petrusz. (Pp 241; £37.50.) Academic Press Inc. 1985.

This book is the third volume in a series devoted to immunocytochemistry techniques. There are some very useful contributions: the discussion of the effects of fixation on immunocytochemistry in chapter 1, although not applicable to much routine immunocytochemistry, should be studied carefully by all immunocytochemists when assessing a new antibody. Chapters on specific techniques, such as the APAAP immunoalkaline phosphatase method, immunoblotting, and microinjection are carefully written and allow the reader confidently to try these new methods out. The last three chapters on the use of metallic ions are particularly useful and I would highlight in particular, the chapter on the enzyme and gold technique, which, although not strictly immunocytochemistry, seems to open up whole new fields of study. This book should join its companion volumes in all immunocytochemistry laboratories.

PG ISAACSON

Mucosal Biopsy of the Gastrointestinal Tract. Vol 3 in the series Major Problems in Pathology. 3rd ed. R Whitehead. (Pp 320; £39.50.) Saunders. 1985.

The third edition of Professor Whitehead's book *Mucosal Biopsy of the Gastrointestinal Tract* appears six years after the second. The editions continue to improve. The third is now 320 pages long, some 80 pages longer than its predecessor. The biggest change is the welcome inclusion of a section devoted to the oesophagus which covers the first 30 pages. Another newcomer is the ileal biopsy,

although with only a small "walk on part" of one page.

The format of this edition is similar to the second, although it is larger and produced with more pleasing page quality and type set. As the photomicrographs were of such good quality it is not surprising that most have been retained with a few changes, and some gaps have been plugged; presumably, as the rarer conditions finally present in the Flinders catchment area. Thus in this edition biopsy photographs of Waldenstrom's macroglobulinaemia and A-beta-lipoproteinaemia are included. As one would expect the background discussion to many of the sections has been improved, expanded, and brought up to date, as have the selection of excellent references.

The book has always been one of the best for the diagnostic pathologist coping with gastrointestinal material, and the third edition preserves and improves its reputation. Such is the pace of gastrointestinal research that there are already topics lining up for the fourth edition, such as *Campylobacter pyloridis* and coeliac T cell lymphomas.

AB PRICE

Muir's Textbook of Pathology. 12th ed. Ed JR Anderson. (£29.50.) Edward Arnold. 1985.

Sixty one years have passed since the appearance of the first edition of "Muir." It is a pleasure to be able to report that this old friend, now in its twelfth edition, is not only alive but flourishing under the sympathetic guidance of Professor John Anderson.

With the rapid expansion of knowledge, not least in the fields of immunology and molecular biology, the editor has increased the number of contributors; the degree of uniformity of style and presentation is a tribute to his editorial skills. There has also been a very considerable and, in my view, welcome increase in the use of diagrams to illustrate both factual data and conceptual material. At the same time the standard of reproduction of the many photographs is high, many of them showing a degree of crispness and clarity that is very pleasing. The improvements both in the text and in presentation have been accompanied by an increase in bulk (and, alas, in price), and the very wide range of knowledge encompassed in this book has resulted in extensive cross referencing which can be a little tedious for the reader. These are trifling criticisms, however, and this new edition can be strongly recommended both to undergraduates and to pathologists in training. It is a credit to its editor, contributors, and publishers.

NEVILLE WOOLF

The Logic of Laboratory Medicine. Principles for Use of the Clinical Laboratory. Dennis A Noe. (Pp 373; paperback £19.95.) Pitman Publishing Ltd. 1985.

This is an unusual textbook of pathology. The author opens with a discussion of units and standardised presentation of data and moves on to the sources of variation in laboratory measurements. There is a full presentation of the evaluation of test performance in terms of diagnostic efficiency as well as the use of multivariate analyses including discriminant functions and Bayesian analysis. The author then discusses the disturbance of physiological systems by disease and how a structure of appropriate investigation can be built. The mathematical approach to diagnosis based on laboratory investigation is well presented, but a student reading the book may not appreciate the limited practicability of such techniques at present, because most of the tests we use have low specificity. Nevertheless, a knowledge of the principles would benefit medical students and trainees studying for higher qualifications in pathology. There are short chapters on morphological diagnosis, the use of some aspects of medical microbiology, and therapeutic drug monitoring. These tend to upset the balance of the book. One of the most apt quotations heading the chapters is from David Hume: "all knowledge resolves itself into probability."

MG RINSLER

Atlas of Oral Pathology. Current Histopathology. Vol 8. RB Lucas, JW Eveson. (Pp 152; £75.) MTP Press Limited. 1985.

The slim volume of 152 pages is aimed at the general diagnostic pathologist with the hope that it will serve as a bench book. In eleven chapters it covers the oral tissues, including bone and salivary glands with colour illustrations of high quality. These are accompanied by a succinct text, which, for the most part, presents a sound account of oral pathology, although in a number of areas this is not fully up to date. This is also reflected in the brief lists of references which follow each chapter.

The longest chapter is the one covering non-odontogenic tumours and includes an unhappy mixture of neoplastic and non-neoplastic swellings. This is the poorest part of the book including several conditions which might have been better placed in other chapters. There is an adequate index.

The authors have very largely achieved their aims in writing this volume, but at