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Postsplenectomy sepsis: the need for lifelong prophylaxis

Dr Evans's report of three cases of late severe postsplenectomy sepsis¹ rightly draws attention to the fact that such sepsis is often rapidly fatal and can occur as long as 45 years after splenectomy.

I feel, however, that the opportunity to discuss the aetiology of this phenomenon was missed. This is of fundamental importance because successful prophylaxis of overwhelming postsplenectomy sepsis depends on rationally based treatment, and it can be argued that Dr Evans's recommendations were inadequate.

Overwhelming postsplenectomy sepsis is almost always associated with bacteria such as *Pneumococcus meningococcus* and haemophilus. These bacteria have a polysaccharide capsule, which evades ingestion by most macrophages and enhances their pathogenicity.² Specialised dendritic macrophages however, have evolved within the marginal zone of the spleen, and these have the ability to take up and present carbohydrate antigens,³ which are commonly of the T cell independent type.

Evidence from studies on animals shows that a specialised subset of B cells, capable of responding to carbohydrate, exists in close proximity to the marginal zone macrophages.⁴ This subset of B cells is non-circulating and resides in the spleen. Gaps in the adjacent endothelium allow direct contact between these specialised immune cells and the blood. Thus it can be seen that the spleen is optimally developed to police the blood for the presence of dangerous encapsulated pathogens.

This explains why the response to infections with encapsulated bacteria or to vaccination with pneumococcal vaccines is inadequate after splenectomy as the response by lymph node macrophages is insufficient. Unless a splenunculus is present the patient is permanently vulnerable to such encapsulated pathogens.

It is well known that postsplenectomy sepsis can be fatal in as little as 12 hours from the onset of symptoms: medical attention, however well informed, may arrive too late. It is therefore my belief that it is the responsibility of the physician or surgeon who recommends splenectomy to discuss the implications with the patient, explaining that lifelong prophylaxis with penicillin will be needed and that pneumococcal vaccine will be given before the operation, although the vaccine may only be beneficial for five years. The family doctor should also be alerted.

I have found that, provided the risks and benefits of splenectomy are clearly explained, patients comply very carefully with prophylaxis with penicillin. For those allergic to penicillin, alternatives are available.

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Dr Evans replies as follows:

Dr Kay's comments are apposite and mainly true. It was a desire for brevity which restricted my report. I am more cynical than he. He believes, however, that it is the responsibility of the doctor recommending or performing the operation to explain that lifelong penicillin will be needed. This may be true; but I do not believe that patients, in the main, will take

drugs lifelong, or that general practitioners or specialists will prescribe them. These patients all have an abdominal scar: the evidence of splenectomy is there for everyone to see. What is needed is recognition of the risk, not only by doctors but also by the patients themselves.

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Glycosylated haemoglobin

The excellent review by Dr Ian Peacock contains some biochemical information which, in my opinion, is not completely correct.

The author rightly refers to HbA_{1c} as

but its formation is expressed as:

A non-enzymatic reaction should lead to the production of a monoglycosylated compound and, to a much lesser degree, of a diglycosylated one. HbA_{1c} is really diglycosylated, while the monoglycosylated haemoglobin, when present, is in low concentration.^{2,3} Thus the above scheme is an oversimplification of the reaction and an equilibrium, producing symmetrical haemoglobin, should be postulated:

It is unclear whether it plays an important part within the erythrocyte or it derives from in vitro manipulation of the sample. A similar equilibrium is well known for some haemoglobin variants, such as Hb_S, and generally for haemoglobins which are modified near the NH₂ terminus of the α chain.

For the reasons given above the heterogeneous glycosylation on some late α lysines should provide principally monoglycosylated products. In this case the equilibrium can not be postulated and these haemoglobin components should be collectively indicated as:

These observations are based principally on theoretical considerations and sur-

ported by some experimental work.^{2,3}

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Fluids, Electrolytes, Nutrition. Allan Engquist. (Pp 320; paperback D.kr. 135.00.) Munksgaard. 1985.

Dr Enquist is the Director of a department of critical care medicine in Copenhagen, and in this book he provides a concise guide for the metabolic management of critically ill patients. The text includes short sections on the underlying physiological principles which enable the user to understand the bases of practical procedures. Fluid and electrolyte balance in numerous clinical situations are considered and there are useful practical details on enteral and parenteral feeding. Paediatric as well as adult problems are considered in detail.

Fluid and electrolyte balance is well served on the shelves of booksellers and this book has to compete in a very tough market. Those revising for examinations or those wanting a handy pocket sized electrolyte "prompt" supported by nutritional feeding, however, would be well advised to consider this useful book.

BRENDA SLAVIN

The Essentials of Forensic Medicine, 4th ed. CJ Polson, DJ Gee, and B Knight. (Pp 734; £95.) Pergamon Press. 1985.

It is twelve years since the last edition of this popular text book was published. This new volume benefits enormously from the contributions of Professor Bernard Knight whose writing has transformed Part II of the book dealing with the law relating to the practice of medicine, which is written with authority and style.

The first part of the book dealing with forensic pathology is regrettably very much the same and an opportunity to alter the emphasis on various topics has been missed. For example, there is still far too much on firearm and electrical injuries but no separate chapter relating to road traffic accidents. Some of the material is frankly dated—for example, the chapter on criminal abortion.

The book itself is almost the same number of pages and most of the photographs are identical with only very few additions. The chapter on cruelty to children suffers very much from a lack of photographs to illustrate the text. It is a beautifully produced volume but the price has risen from £12 in 1973 to £95, and some may find it compares less than favourably with the new edition of Taylor's Principles and Practice of Medical Juris-

prudence which covers much of the same ground, and it must be said in some areas, to better effect, for only £32. So regrettably, only a qualified welcome for the new Polson, Gee and Knight.

HRM JOHNSON

Models, Mechanisms and Etiology of Tumour Promotion. Ed M Börzsönyi, NE Day, K Lapis, and H Yamasaki. IARC Scientific Publications no 56. (Pp 532; £30.) Oxford University Press. 1985.

Biological approaches to carcinogenesis are particularly fruitful as long as the molecular mechanisms are not understood. The concept of two stage carcinogenesis, initiation, and promotion remains a valuable source of ideas, even more so in terms of phenomena to be investigated.

This book is the proceedings of a conference in 1983, organised by the IARC, on promotion—that is, the series of at least three steps between electrophilic attack on DNA (initiation) and the appearance of frank tumours. There is much display of recent biochemical knowledge about phorbol esters, protein kinase C and their secondary effects on cells, several accounts of a variety of changes in cells in vivo and in vitro, an intriguing epidemiological attempt to suggest that the process of promotion is important in man. Like all sets of conference papers it is both worthy and repetitious, but it is also a unique and worthwhile point of entry into an area of pathobiology that is little understood and probably very important.

AD DAYAN

Hemostasis and Thrombosis. Ed EJ Walter Bowie and AA Sharp. (Pp 341; £45.) Butterworths. 1985.

It is a great pleasure to review this book edited by two such well known haematologists. Walter Bowie has generously dedicated the book to the memory of Alan Sharp, who died during its preparation. We will always remember him, however, for his friendship and personal qualities.

In the rapidly growing fields of haemostasis and thrombosis the book readily updates our knowledge. There are excellent introductory chapters on platelets, coagulation, and fibrinolysis by leaders in their individual fields. I particularly enjoyed the chapter on "Hereditary and acquired bleeding disorders" by Rizza and

Book reviews

Atlas of Endometrial Histopathology. G Dallenbach-Hellweg and H Poulsen. (Pp 229; D kr 470.00.) Munksgaard. 1985.

The authors set out, according to their Introduction, to provide a book for pathologists and clinicians to help them recognise and classify the histopathology of endometrial disease. Their first objective is well served by the selection and quality of the photomicrographs. These are almost all excellent, with good choice of material, colour balance, and focus. The text of the book for the most part is adequate and is intended to be secondary to the illustrations, but suffers on several points. There are spelling mistakes, the terminology used in some of the descriptive passages is idiosyncratic (for example, moderate adenomatous hyperplasia of endometrium is equated with atypical hyperplasia), and there are solecisms such as undifferentiated adenocarcinoma. Large amounts of space are wasted, with only four lines of text on some pages. Nevertheless, the illustrations in the book might usefully accompany a text book of gynaecological pathology in departmental libraries.

D LOWE