Bjarnason and coworkers are slight and can principally be ascribed to differences in the populations studied. We believe both the $^{51}$Cr-EDTA test and sugar permeability test have a role in the investigation of intestinal permeability and the techniques are complimentary rather than mutually exclusive.

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References

2 Catt SD, Menzies IS, Segal MB. The effect of poorly absorbed solute on human intestinal absorption. Proc Physiol Soc 1983; 78P.
8 Ukaham SO, Clamp JR, Cooper BT. Is small intestinal permeability increased or decreased in untreated coeliac disease? Clin Sci 1981; 61: 39P.

Reply

Sir,—In their first letter (August 1984; 909–10) criticising our paper (February 1984; 145–50) on the in vitro determination of mucosal permeability in coeliac disease Hamilton et al attempted to perpetuate the misconception that in coeliac disease permeability is decreased to small hydrophilic molecules. In our reply (August 1984; 110–11) we pointed out that this fallacy was because of their confusion between the terms absorption and permeability which are not necessarily synonymous when the villous architecture is grossly altered as in patients with untreated coeliac disease.

In their present letter they persist in their claims for the value of the combined cellobiose/mannitol/sucrose/lactose absorption test. These sugars clearly show differing tissue hydrolysis rates, variable absorption and differential endogenous production rates among themselves and between patients with normal and diseased intestines. Clearly interpretation of results with this test are fraught with difficulty although it may have some role as a screening procedure. The plethora of various sugars used to assess permeability, and the large range of osmotic fillers, including glycerol, used by different groups indicates a lack of conformity in this field so far.

Hamilton and colleagues claim that toxicity of gastrointestinal contents does not affect transit. Unfortunately their reference (I C MD Thesis) is unavailable to us but their claims are at variance with the results of others.1 2

Hamilton et al comments on the single report (letter) purporting to show that the $^{51}$CrEDTA test is valueless. They fail, however, to report the subsequent correspondence3 4 and other independent reports,5–8 confirming the value of the $^{51}$CrEDTA in both physiological and pathophysiological studies.

In seeking to perpetuate the cellobiose/mannitol/sucrose/lactose test, Hamilton and colleagues attempt to deny the clinical gastroenterologist a cheap, simple, rapid screening procedure for small intestinal damage, as well as burdening the hardened chemical chemist with difficult and demanding analytical procedures.

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References

5 O’Morain C, Chervu LR, Milsten DM, Das KM.


Books


This is a new series of Clinics which was introduced in 1982. It follows the familiar format and this particular volume is devoted to reviewing the mechanisms of natural immunity. Triggering of immunological effector mechanisms is usually considered to result from exposure to antigen. These mechanisms may be cellular, mediated by sensitised T cells, or humoral, mediated by sensitised B cells. Certain immunological reactivity, however, can be detected in individuals which have not been intentionally immunised – this is termed 'natural immunity'.

The first two chapters review the evidence for naturally occurring antibody to oncogenic viruses and tumour cells, both in animals and man. The possible origin of these antibodies and their role in controlling viral infections and in tumour surveillance is extensively discussed. These chapters are followed by a concise account of the complement system and the clinical syndromes resulting from deficiencies of the complement components. The role of the gastrointestinal tract in preventing antigen-entry into the body is covered by R J Levinsky. The rest of the volume deals with natural killer cell activity and its role in tumour immunity, the production of interferon by natural killer cells, and the role of macrophages in cancer surveillance and in controlling infection.

This volume is full of information and is, in general, well presented. It is certainly not intended for the general reader but those with some immunological knowledge will find some excellent reviews here.

D P JEWELL


The Italians have taken a keen interest in ultrasound for many years, and with sales of ultrasound equipment almost three times as great as in the United Kingdom. The authors are all very experienced in ultrasound but are gastroenterologists rather than radiologists.

The book is an instructional text for those performing and interpreting abdominal ultrasound. There is very little clinical material or clinical discussion. This is surprising considering the background of the authors and I think that few of my clinical colleagues would be willing to trawl through the text to find the material relevant to them.

From the viewpoint of the radiologist the first 100 pages is unnecessary. This covers background physics and techniques which are more thoroughly covered in other textbooks. Of the three guest chapters only that on guided puncture procedures by Otto is good. Dr Magnos's chapter on endoscopic ultrasonography fails to show the potential of this important new method, despite the author's eminence in the field.

The core of the book, on abdominal scanning is well illustrated and well referenced and is up to date in methodology. It is a useful read for those involved in scanning but not worth the price of £48.

W R LEES

Books received


News

2nd International Symposium on Inflammatory Bowel Diseases

This symposium will take place in Jerusalem, Israel, from 8–11 September 1985. Further details from the Secretariat, PO Box 50006, Tel Aviv 61500, Israel.
Reply

I Bjarnason and T J Peters

*Gut* 1985 26: 323-324
doi: 10.1136/gut.26.3.323

Updated information and services can be found at:
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