that it is only with subsequent investigations that we could find definite aetiology in 10 cases. Perhaps prognosis is good and special investigations unnecessary in 'true idiopathic pancreatitis' but how to know if it is a 'true idiopathic pancreatitis' without doing investigations? In the study of Ballinger *et al*, the pancreatitis was labelled idiopathic retrospectively and patients with rare causes of acute pancreatitis were excluded from the study. We think that prospectively, it is often difficult to distinguish between 'true idiopathic' and rare causes of acute pancreatitis at the time of admission and that specialised investigations are often needed to separate them. 'Idiopathic pancreatitis' is a rare diagnosis that can be accepted only after specialised investigations. We outline that three of our patients had acute pancreatitis revealing a carcinoma and that hyperparathyroidism is sometimes caused by a cancer.[2]

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1 Maes B, Hastier P, Caroli-Bosc F, Conio M, Dumas R, Demarquay JF, *et al.* Etude étiologique des pancréatites cliniquement aigues dans la région de Nice (résultats préliminaires). *Gastroenterol Clin Biol* 1995; **19:** 1043.
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Reply

EDITOR,—We have read the comments of Dr Maes and co-authors and also referred to their article that has been published in abstract form.[1] They have reported on 121 consecutive cases of acute pancreatitis and the abstract includes the first 62 of these patients. In the 19 patients in whom no cause of acute pancreatitis was found on initial investigation, the authors do not state if these were first or recurrent episodes of pancreatitis. This is obviously important if comparisons are to be made with our study in which we clearly state that we have only studied patients with a first attack of acute idiopathic pancreatitis and thus our conclusions are only applicable to this group of patients. As Maes *et al* recruited consecutive patients with acute pancreatitis it is probable that a proportion were presenting with recurrent episodes.

On subsequent investigation a possible cause for acute pancreatitis was found in 10 of 19 patients in the authors' series. We are surprised that some of these aetiological factors were not identified on the first hospital admission; for instance, we would expect an antral carcinoma invading the pancreatic duct to be easily seen on the abdominal computed tomogram. The authors do not state which drug was implicated in causation of acute pancreatitis but a clinical history taken on the first admission should have identified this as a possible causative factor.

Finally, the authors state that there was a 20% recurrence rate during the follow up period but only one recurrence after the correct diagnosis was made. We are not told what specific treatment, if any, those patients with a diagnosis received and therefore it is impossible to determine if the treatment changed the natural history. It is probably only worth searching for a cause if the treatment changes the longterm outcome.

In summary, the data presented in the authors' letter and their original abstract do not change our conclusions and recommendations for the treatment of first attacks of acute idiopathic pancreatitis.

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Screening for familial colorectal cancer

EDITOR,—Based upon their findings using an immunological faecal occult blood test, Cripps and Heald (*Gut* 1996; **38:** 421–5) make recommendations for screening of colorectal cancer (CRC) on the basis of a positive family history. However, current knowledge calls for a more targeted and scientifically founded approach.

Their recommendations are intended for subjects who do not have a family history suggestive of an autosomal dominant condition predisposing to CRC. How is the distinction to be made between a 'dominant pedigree' and a less than dominant pedigree? For example, one of their patients was found to have familial adenomatous polyposis (FAP). This patient was originally assigned with a low lifetime risk by virtue of the single affected first degree relative, yet the patient's true original risk was 1:2 not 1:17. Single case hereditary non-polyposis colorectal cancer (HNPCC) families have now been identified through the demonstration of germline mutations in a DNA mismatch repair gene. The affected subjects were ascertained exclusively on the basis of young age at onset of CRC.[1] These examples illustrate the inadequacy of attributing lifetime risk on the basis of family history alone.[2] Indeed such estimates are both crude and misleading.

The alternative approach is to offer targeted screening on the basis of the underlying genetic disorder. An approach to the correct diagnosis is achieved through the ascertainment of detailed and extended family pedigrees for which all cancer diagnoses are verified with respect to location, age at onset, and histological type. The presence of DNA microsatellite instability is an important biomarker for HNPCC, particularly when found within early onset cancers,[1] two or more cancers from the same patient or in cancers from two or more members of the same family.[3] Once classic FAP, attenuated FAP,[4] and HNPCC have been excluded, what is left? Apart from various rare forms of precancerous polyposis the literature hints at at least one additional important autosomal dominant disorder. This has been described 'late onset familial CRC'[5] or 'adenoma families'.[6] Still poorly understood, this syndrome features cancers of the left colon and rectum, a modest increase in the number of adenomas, and an increased tendency for adenomas to become large and villous.[5 7] No reliable marker for this putative autosomal dominant syndrome exists at this time.

A weak family history of colorectal cancer with no distinguishing clinical, pathological or molecular features is likely to be a chance event, associated with a low lifetime risk for family members. CRC is common and affected subjects are likely to have multiple first degree relatives. Might not these relatives, perhaps representing an estimated 20% of the total population, be served through a conventional population based screening approach? Obviously this would depend on correctly assigning high risk families to particular autosomal dominant disorders. Although we currently lack full diagnostic capability in this respect, the way forward is to establish cancer family clinics that would facilitate integration of clinical, genetic, and pathological data and coordinate longterm management strategies. One shudders at the prospect of local enthusiasms linked to unlicensed gene testing outfits.[8]

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Screening for colorectal cancer

EDITOR,—The article by Cripps and Heald made for interesting and informative reading (*Gut* 1996; **38:** 421–5). Their compliance rate of 60% was indeed impressive, comparing it with most other screening studies for detecting colorectal cancer. In this context, we would like to draw attention to some additional data relevant to this subject.

Hobbs and coworkers found significant difference in compliance among patients aged between 50–69 (683; 61·6%), 70 or over (343; 54·3%), and 40–49 (204; 43·8%), ($p<0·001$). They report that patients from the inner city practice were less likely to comply (55; 3·5% versus 78; 12·5%; $p<0·001$).[1] Powe found that fatalism, in a study among 192 elderly African Americans, was the only significant predictor of faecal occult blood testing (FOBT), even when factors such as age, poverty, and education were controlled.[2] Another study found significantly higher compliance (72·8% versus 51·8%; $p<0·01$) among 153 patients when dietary restrictions were

not imposed.[3] Thomas and colleagues report a significantly higher rate of screen compliance among participants living with other participants, while those who had a diagnostic colorectal examination with negative results had significantly lower odds of complying.[4]

Another study showed that compliance in first degree relatives of patients with colorectal cancer was significantly higher than in spouses (69% versus 47%, $p<0.01$), as was among those whose relatives died recently from colorectal cancer.[5] However they found that time since diagnosis in the index case had no effect on the compliance rate, in contrast with the findings by the authors. Finally, Neilson and coworker report that compliers are found to be of higher socioeconomic classes than persistent non-compliers, to have more personal and family experience of illness, and to visit their dentists more regularly.[6]

These data, in conjunction with that presented by the authors, helps us better understand the factors affecting compliance, while screening for colorectal cancer.

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Reply

EDITOR,—Thank you for the opportunity to reply to the letters commenting on our publication (*Gut* 1996; **38:** 421–5). Compliance with screening, although a complex issue, has a major impact on the ability of a programme to detect significant colorectal pathology. The additional data provided by Anand and colleagues are of clear interest.

Although we were pleased with overall uptake of screening (64·9%), our report shows – by demonstrating that some subjects were originally interested but then declined the offer of screening – that compliance is not an all or nothing phenomenon – that is, people may respond to an invitation but drop out when the process of screening is made clear. Obviously, the simpler the screening protocol the more likely it is to maximise uptake. This point is again made by Robinson *et al* in a recent report comparing one and three day HemeSelect testing who found compliance to be significantly better for the shortest test protocol.[1] A further example of this phenomenon is shown by our screened subject who refused further intervention despite being shown to have multiple left sided polyps at an uncomfortable colonoscopy.

The implication of these findings is that compliance can yet be improved but will peak at a level at which people are simply not willing to be screened despite experiencing a high personal risk of colorectal cancer. We have recently been very disappointed to discover a 32 year old patient who was offered (and declined) screening in 1992, only to present with an obstructing, Dukes's stage C caecal carcinoma in 1996. This patient is a member of an HNPCC family whose other family members are examined at St Marks' Hospital Family Cancer Clinic and who did not wish to be examined at any cost.

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BOOK REVIEWS

Comparative Physiology of the Digestive System of Vertebrates. 2nd ed. Edited by C E Stevens, I Hume. (Pp 400; illustrated; £55 ($79.95)). Cambridge: Cambridge University Press, 1996. ISBN 0-521-444187-7.

For those who want to compare the colon lengths of the short nosed bandicoot and the koala bear, but have neither the fare, nor the agility to catch the beasts for themselves – this is very definitely the book to buy and a terrific saving. However, these days a book must appeal to a less specialised market.

As the authors make clear, there is value in the study of an organ system from a non-anthropocentric viewpoint: – it's all very well for you blighters who live off the fat of the land, with your well cooked digested meals, to gloat about how much more streamlined your guts are – but what about the rest of us raw fibre eaters, or shell eaters? – We'd like to see how long you survived on eucalyptus leaves, or how far you could fly on a diet of beetles!

On the one hoof, some of us fermenters have to retain our digesta a lot longer than you to derive any benefit from it. Furthermore, some of us are very large and have to eat a heck of a lot of 'indigestible' stuff to keep swanning around the savannahs. A neat way of reducing the bulkiness of our digesta is by having a faster throughput of fermentable particulates than of fluid. However, selective retention of small particulates over fluid entails specialised gastrointestinal structures behaving effectively as filter beds. These structures are variously present in our fore, mid or hindguts and very thoroughly described in this book, with excellent diagrams.

On the other hoof – predatory birds, like hawks, although eating only the juiciest morsels, obviously cannot afford to carry around excess baggage, or they will find themselves in the relegation zone. So they fluidise, digest, and ferment their prey in their gizzards prior to absorption in a comparatively short small intestine and vestigial hindgut; non-digestible particulates are jettisoned in this species prior to absorption.

And on the third hoof – (the additional advantage of non-anthropocentrism), the humming bird, weighing in at three grams – has an enormously high metabolic rate and therefore a continuous need for a rapidly available energy supply, has no room to accomodate either for a fermentation or digestive chamber; so it has neither caecum nor crop and lives exclusively on a fast food diet of liquid sugar.

So, we can learn a lot about animal adaptation to varying nutrition from simple macroscopic examination of the gastrointestinal tract in relation to body size. This book is very good on these aspects of comparative physiology.

In this second edition, the scope of the book has been broadened to include interesting and useful chapters on digest transit and retention, which includes a really useful discussion on digestive strategies in omnivores and herbivores; motor activity and a chapter on the evolution of the digestive system. There is a rather sparse chapter on the comparative biochemistry of digestive processes and a better one on bacterial fermentation in the gastrointestinal tract.

My main criticism of the book is its relative lack of attention to microscopic anatomy, or structure-functional correlates at the microscopic level. Perhaps this requires another book. My overall view is that this is a useful and stimulating book, well worth reading and I look forward to an enlarged third edition.

RICHARD NAFTALIN

The Kidney in Liver Disease. 4th ed. Edited by M Epstein. (Pp 561; illustrated; £95). Philadelphia: Hanley and Belfus, 1996. ISBN 1-56053-166-5.

This is the fourth edition of Epstein's 'The Kidney in Liver Disease', the first having been published in 1978. The format of each is similar with multiple authorship. The book is largely devoted to problems of alcoholic cirrhosis, in particular sodium retention and renal failure. Minimal attention is given to other types of cirrhosis or to the important condition of fulminant hepatic failure. Alcoholic hepatitis, a sometimes reversible condition that may be complicated by profound renal and electrolyte disorders is not specifically mentioned.

The balance of authors leaves something to be desired – the editor is the sole author of eight of the 27 chapters and only three are written by hepatologists. The lack of hepatological input is constantly apparent throughout much of the text. Is it really appropriate for the chapters on diuretic therapy, peritoneovenous shunting or extracorporeal techniques to have been written by nephrologists? Anyone from a specialist liver unit with an interest in these subjects would have far more