that it is only with subsequent investigations that we could find definite aetiology in 10 cases. Perhaps prognosis is good and special investigations in a '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 era with rare causes of 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

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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gique des pancréatites cliniquement aiguës dans la région de Nice (résultats prelimi-

Screening for familial colorectal cancer

EDITOR,—Based upon their findings using an immunological fecal occult blood test, Crips and Heald (Gut 1996; 38: 421–5) make recommendations for screening of co-
rolectal cancer (CRC) on the basis of a positive family history. However, current knowledge is often targeted and scientifically founded approach.

Their recommendations are intended for subjects who do not have a family history suggestive of an autosomal dominant condi-
tion predisposing them to CRC. How is the distinc-
tion to be made between a ‘dominant pedi-
gree’ 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 (HNPPC) 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 demonstrate 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 cancers are verified with respect to location, age at onset, and histological type. The presence of DNA microsatellite instability is an important bio-
marker for HNPPC, particularly when found within early onset cancers,1,2 or more can-
cers from the same patient or in cancers from two or more members of the same family.3 Once classic FAP, attenuated FAP,4 and HNPPC have been excluded, what is left? Apart from various rare forms of precancerous polyposis the literature hists at least one addition of adenomatous carcinoma dominant syndrome. This has been described ‘late onset familial CRC’ or ‘adenoma families’.5 Still poorly understood are some features of 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.6 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 rela-
tives, perhaps representing an estimated 20% of the total population, be served through a conventional population based screening program? Obviously this would depend on correctly assigning high risk families to partic-
ular 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 inte-
gration of clinical, genetic, and pathological data and coordinate longerterm management strategies. One shudders at the prospect of local enthusiasts linked to unlicensed gene testing outlets.

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7 Boutron M-C, Favier J, Quipourt V, Senesse P, Michels C. Family history of colorectal tumours and implications for the adenoma-

8 Moran N. UK spurs proposed genetics regul-

Screening for colorectal cancer

EDITOR,—The article by Crips 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 addi-
tional data relevant to this subject.

Hobs and coworkers found significant dif-
cference in compliance among patients aged between 50–69 (683; 61–66%), 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 with the test (68.6% versus 78; 12–9%). More recently, they found that fatalism, in a study among 192 elderly African Americans, was the only sig-
nificant predictor of faecal occult blood testing in elderly persons, poverty, and education were controlled.2 Another study found significantly higher compli-
cance (72.8% versus 51.8%; p<0.01) among 153 patients when dietary restrictions were
Screening for familial colorectal cancer.

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