these symptoms inaccurately determined. The investigators also included patients with various organic diseases as controls. It is unclear, however, what symptoms led these patients to present, and it is likely that some of these patients also had irritable bowel syndrome (particularly the gall stone group who may have had incidental gall stones discovered during their evaluation). Such misclassification could have diluted the discriminant value of symptoms between patients with irritable bowel syndrome and the disease controls.

Our second concern relates to the statistical analyses. The authors seem to have relied largely on univariate analyses (χ^2 tests). As 78 tests of association were undertaken (13 symptoms times six comparison groups!), however, several spurious significant results could have been obtained just by chance alone.[3 4] One way to adjust for the number of tests undertaken would be to multiply each p value by the number of comparisons. When evaluated in this way, only p values less than 0·0006 would be significant at the 5% level. While the authors did use a multiple logistic regression analysis, it appears that gender and socioeconomic status (which are potential confounders) were not included in the model. Moreover, it would have been of interest to determine whether the same symptoms could distinguish irritable bowel syndrome from each of the control groups using logistic regression analysis; instead all the organic disease patients were lumped together (a criticism that the authors justifiably levy at past studies). In addition, their analysis is likely to have seriously overestimated the discriminatory ability of the symptoms identified; it is well recognised that estimates based on a single data set are typically biased towards optimism[5] and for this reason prospective samples need to be tested to confirm the discriminant value of any symptom model developed.

Finally, the authors' contention that multiplication of the relative risks can be used to estimate the 'overall risk' depends rather heavily on having the 'correct' model – for example, no other unobserved confounding variables and no interactions among the symptoms used in the model – but this was not documented in the article.

Dr Maxton and colleagues have provided some intriguing hypotheses, but based on the data presented the diagnostic value of 'non-colonic' features for irritable bowel syndrome remain, in our opinion, unclear.

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Reply

SIR,—Thank you for giving us the opportunity to reply to the letter from Dr Prather and colleagues. We have previously shown that the prevalence of the non-colonic symptoms referred to in the paper are very significantly more common in patients with irritable bowel syndrome than in normal controls and therefore felt it appropriate to repeat this work.[1] Each symptom was carefully defined before the project began and the exact wording used decided before patient recruitment. A single interviewer conducted the entire study to minimise possible differences of interpretation. Irritable bowel syndrome is a common disorder and, as Dr Prather suggests, could easily coexist with other organic diseases. In the absence of a diagnostic test for irritable bowel syndrome, there is no easy way of preventing or excluding this possibility. As Dr Prather rightly points out, however, this would reduce rather than increase the discriminant value of the symptoms in separating organic from functional bowel disease thus making our findings more reliable. The gall stone patients included in the study all had symptoms suggestive of this disorder at first clinic attendance which improved after cholecystectomy. None had gall bladder disease as an incidental finding.

The statistical problem of multiple comparisons is well known and the use of repeated statistical testing (without proper adjustment) to establish definitive relationships is inappropriate. In our study, however, univariate analysis was only used in the preliminary investigation of discriminant power. The main part of the study identified symptoms with a significant association with irritable bowel syndrome using multiple logistic regression, a technique which makes appropriate adjustment for the large number of symptoms under consideration. Although the numbers of subjects in each of the separate disease groups were large by the standards of previous publications, they were insufficient to allow discrimination from each control group using multiple regression analysis. As reported in the paper the effect of gender had little effect on the discriminant ability of the 'non-colonic' symptoms of irritable bowel syndrome. Socioeconomic group also did not differ significantly between irritable bowel syndrome and organic disease groups. The description of relative risk estimation was included only as an illustration of how the risks can be combined. No claim is made for the accuracy of these combined estimates (the associated confidence intervals would be wide) but they give some indication of the order of magnitude of the relative risks. We agree it would certainly be of value to test our symptom model on other groups of patients. We are continuing to do so and hope others will follow.

The answer to the question posed by Dr Prather and colleagues in the title of their letter is, yes, non-colonic symptomatology is indeed of discriminant value in separating irritable bowel syndrome from a large number of organic gastrointestinal diseases.

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1 Whorwell PJ, McCallum M, Creed FH, Roberts CT. Non-colonic features of irritable bowel syndrome. *Gut* 1986; **27**: 37–40.

BOOK REVIEW

Inflammatory bowel disease – diagnosis and treatment. By G Gitnick. (Pp 541; illustrated; £91.) New York: Igaku Shoin, 1991.

This is a multichapter (37), multiauthor (58), multinational (though predominantly US) textbook on ulcerative colitis and Crohn's disease. The editor's aim was to produce an up to date and concise clinical overview of these inflammatory bowel diseases, their diagnosis, and treatment. It is laid out in seven sections – aetiology and epidemiology, clinical features, diagnosis, prognosis, medical and surgical management, and management problems.

The book is extraordinarily uneven. Contrast the first section, 40 pages on genetics and probably the best chapter in the book, with the five pages on aetiology or seven pages on inflammatory mediators. The sections on clinical features and diagnosis encompass six chapters, which between them duplicate or triplicate the routine assessment of the two conditions. Some excellent endoscopic pictures are mixed with out of focus histopathology, but histopathological appearances are presumably regarded as so recherché that the section on dysplasia is not illustrated. A number of full, worthwhile, and extensively referenced sections – for example, those on the use of corticosteroids or on the natural history of these diseases, based not on anecdote but on the placebo arms of published studies – contrast strongly with, for example, the Tennessee experience on T cell apheresis. This is an uncontrolled study on 63 patients (from the reference list, I cannot see that it has appeared in any peer reviewed form) 'in which the chances of . . . undergoing spontaneous remission was statistically zero'. Few people who have experience of inflammatory bowel disease will recognise such a group.

There are better books on inflammatory bowel diseases, from both the scientific and the clinical view point.

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