colonic motility. This is also true for the subgroup of IBS sufferers who we have identified as having hypomotile colons.

With regard to clinical details about the patients, we have little to add to that which appears in the text. All the patients had been first seen in the outpatient clinics and the diagnosis of diverticular disease or irritable bowel syndrome made by an independent clinician. None of those with diverticular disease had peridiverticulitis or abscess either clinically or radiologically. All had had abdominal pain and alteration in bowel habit which had prompted referral to a specialist department and on subsequent investigation all had been found to have diverticular disease involving the sigmoid colon. It is a matter of debate as to whether the presenting symptoms and the finding of diverticular disease on barium enema examination are causally related, but it does little to enhance our knowledge of the mechanism of symptoms simply to say they are due to the irritable bowel syndrome.

Until a pathophysiological marker is found, the diagnosis of irritable bowel syndrome will remain one of exclusion. Stratifying patients according to symptoms is attractive but not always possible as the pattern of symptoms may be variable, definition of the symptoms imprecise and their elucidation highly subjective. The extent to which investigations are pursued to prove there is no 'organic' disease present is determined by many factors but we believe that the 20 patients we studied would have fulfilled rigorous criteria for the diagnosis of IBS. Moreover, in the several years of follow up since the studies were completed, there has been no reason to revise the diagnosis in any of the patients. Symptoms were recorded at structured interview at the time they were admitted to hospital and prior to colonoscopy. The patient whose bowel habit was considered normal was a 44 year old male with a six year history of left sided abdominal pain which was eased by defecation, whose general health remained otherwise good, and in whom investigation had been negative.

When the recordings of sigmoid IPs were analysed, we found no correlation between any symptom or group of symptoms and the pressure recording that had been made. Perversely, symptoms occurring during the period of study were extremely rare – a phenomenon frequently remarked on by others investigating intestinal motility in IBS. Only two of our subjects had their usual pain during the period of recording and, interestingly, this was accompanied by a reduction in the frequency and amplitude of pressure waves in both.

Finally, we would share Dr Thompson's sentiments that improved measurement techniques should be accompanied by 'more sophisticated definition of the subjects studied'. Unfortunately, we fear that defining subjects by symptoms alone is ingenuous and unlikely to lead to any useful new strategies for the diagnosis or management of the IBS sufferer in the everyday clinical environment.

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Colorectal cancer in UC

SIR,—Gyde *et al*[1] in their paper on the colorectal cancer risk in ulcerative colitis argue that patients tend to develop colorectal cancer at about 50 years of age, irrespective of their age at onset of colitis. They therefore suggest that screening patients aged less than 30 or more than 60 is unnecessary. This suggestion was based on 35 patients with carcinoma derived from a population which excluded those with onset of colitis before the age of 15, thus including a bias against carcinoma in younger age groups.

Of 100 patients treated at this hospital for carcinoma complicating ulcerative colitis, 11 were under 30 years of age, and 23 were over 60 years of age. If surveillance were limited to patients between 30 and 60 years of age, this would mean that a third of carcinomas developing would be missed, and one third of these would be in patients in their 20s. This is unacceptable. Results from this hospital show that the cancer risk in extensive colitis is related to the time from onset of the disease and all patients who have had their disease for 10 years or more are at increased risk. Surveillance should be offered to all such patients if other factors such as infirmity or old age do not prevent them from attending the hospital.

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Reference

1 Gyde SN, Prior P, Allan RN, *et al.* Colorectal cancer in ulcerative colitis: a cohort study of primary referrals from three centres. *Gut* 1988; **29:** 206–17.

Reply

SIR,—The main thrust of our paper was to provide the best estimate at present available of the colorectal cancer risk in ulcerative colitis. We did not wish to further muddy the already murky waters of screening for colorectal cancer in colitis.

In discussing our results we examined whether the

peak at 50 years for the development of colorectal cancer in colitis was a real effect or an artefact of patient selection and length of follow up. We had observed such a peak in our previously unselected series.[1] The peak was more clearly defined in the present analysis. Computer models of the rates of initiation of colorectal cancer in colitis are certainly suggestive of an association between early onset UC and onset of colorectal cancer with a peak around 50 years of age.[2,3]

Burch *et al* did not identify a separate childhood onset group but the fact that he modified the parameters of this model to accommodate rates for the very young suggest that there may be some differences in the development of ulcerative colitis in childhood and the risk of colorectal cancer.

We cannot comment specifically on the risk in childhood UC as our data for this group were incomplete. Among patients who were excluded on the basis of early onset colitis, however, six developed colorectal cancer between the ages of 26 and 29 years (mean 27 years), mean age at onset being 9·5 years (range 5-12 years).

We did not recommend any changes in the current screening practice and emphasised 'that more supportive evidence in relation to age at cancer in adult onset ulcerative colitis would need to be adduced from other studies before any changes in the basis of screening should be considered'.

The data from St Mark's Hospital suggest a wider spectrum of age at diagnosis of colorectal cancer than we observed in our study and it would be of interest to re-analyse those figures in relation to age at onset of ulcerative colitis.

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2 Burch PRJ, deDombal FT, Watkinson G. Aetiology of ulcerative colitis. II. A new hypothesis. *Gut* 1969; **10:** 277–84.
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Effects of meal temperature on intraluminal upper gastrointestinal temperature and motility

SIR,—In their paper studying the effects of meal temperature on gastric emptying rates, Sun *et al* (*Gut* 1988; **29:** 302–5) conclude that warm drink, taken at 50°C, and cold drink at 4°C emptied more slowly than a drink at body temperature. Only the cold drink showed a significant slowing in the initial period, and the difference in emptying rates when compared with the drink at the control temperature correlated with the difference in intragastric temperature. In their study the mean maximum intragastric temperature was 43°C and occurred 60 seconds after ingestion. The intraluminal temperature encountered in the upper gastrointestinal tract, however, may be higher than reported by this group even during normal daily life.

We have shown[1] that the preferred temperature for a hot drink varies considerably between individuals, from 45°C to over 70°C, and that patients with peptic disorders of the upper gastrointestinal tract chose to drink at higher temperatures than a group of matched asymptomatic controls (medians 56°C and 62°C respectively $p<0·001$, Mann Whitney U Test).

Furthermore, using a system we have developed[2] which records temperature at rates of up to 10 Hz in three oesophageal sites, two gastric sites and the duodenal bulb we have shown marked swings in intraluminal temperature within the oesophagus (7–63°C), stomach (19–49·5°C) and duodenum (25–42°C) of healthy subjects after normal eating and drinking. The time after ingestion at which temperature change starts to occur is also varied, such that after a cold drink intragastric temperature starts to fall four seconds and duodenal temperature 20 seconds after the first swallow. Ice cream, however, does not alter gastric or duodenal temperature at all, presumably being rewarmed in the oesophagus, after decreased peristalsis as a result of cold temperature.[3,4] That intraluminal duodenal temperature falls to 25°C and remains below 35°C for nearly six minutes after a cold drink would support Ritschel and Erni's[5] report that cold fluids leave the stomach faster than warm ones, in contrast with Sun *et al*'s results.

We would agree that the temperature of the diet may play an important part in motility patterns but would suggest that Sun and his colleagues have studied only a part of the range of temperatures at which patients frequently consume food and drink. A wider range of meal temperatures may have even more dramatic effects on upper gastrointestinal motility.

In the light of the findings of Sun *et al* and our own studies, investigators studying upper gastrointestinal motility with test meals should now report the temperature of ingestion of the meal, whether it be liquid or solid.

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