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 unsselected series. 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.\(^1\) 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.

**PAT PRIOR, SYLVIA GYDE, AND ROBERT ALLAN**

The General Hospital, Steelhouse Lane, Birmingham B4 6NH.

**References**


**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 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 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.\(^4\) 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 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.

**R C PEARSON AND R F MCCLOY**

The University Department of Surgery, Manchester Royal Infirmary, Oxford Road, Manchester M13 9W.
meals used to study upper gastrointestinal motility, especially when using fatty meals with higher thermal inertia.

L A HOUGHTON AND W M SUN
Sub-Department of Human Gastrointestinal Physiology and Nutrition, University of Sheffield, Sheffield S10 2JF.

Cimetidine, carbenoxolone and gastric mucus
SIR,—Reading the paper by Ene et al. I was rather surprised to find that someone took the trouble to try to reproduce my eight to 10 year old studies on the effects of cimetidine and carbenoxolone on gastric mucus, a topic which seems a little outdated in 1988. Nevertheless, having been quoted and criticised so much by these authors, I cannot refrain from replying to some of their issues.

First, perusing Glass’ papers I did not have the impression, as Ene and colleagues seem to think, that he considered acid mucoproteins as the component of mucus responsible for mucosal protection. Of course this great pioneer in mucus researches is beyond the possibility of entering the debate. In my opinion, however, the viscous and protective properties of mucus are more related to neutral than to acid mucoproteins. This should be apparent by some of my papers quoted by Ene et al., where in fact I was using as a parameter of mucosal protection the ratio of neutral to total mucoproteins (so-called Mucoprotective Index). What we actually found at that time was that cimetidine treatment increases acid mucosubstances and induces a marked, significant decrease of neutral mucoproteins. Carbenoxolone, in contrast, promotes a rise of both mucin components, the effect being more dramatic on neutral mucoproteins, with a consequent increase in the values of Mucoprotective Index. The key point is that we undertook our studies in peptic ulcer patients treated for four weeks with either cimetidine 1 g daily or carbenoxolone 150 mg daily (300 mg the first week). Ene’s experiment included normal subjects who received either drug for only two weeks and in lower doses. Thus it is hard to compare Ene’s results with ours.

Furthermore I am not quite sure that the method used by the authors, sophisticated as it may be from a biochemical point of view, provides a better insight of the effects of drugs on mucus secretion. On the other hand the adverse influence of cimetidine on gastric mucus has been also observed by others. As for the mucogenic activity of carbenoxolone, this was reported, by means of various techniques, by different investigators before and after the appearance of my