debris. It may, however, damage external surfaces and in particular the rubbers and plastics of the insertion tube so instrument manufacturers do not recommend immersion. Casemore and Blewett provide evidence that it is fully effective if cryptosporidial oocysts are exposed for 30 minutes. Flushing and shorter times may not be sufficient.

There are problems with the four other disinfectants listed and which are said to be partially effective. Exspor in preliminary tests against polio virus and herpes simplex virus has shown rather poor activity compared with glutaraldehyde (Ayliffe G. personal communication). One per cent ammonia is not suitable for bacterial disinfection, sodium hypochlorite damages endoscopes and sodium hydroxide may do also.

Although in immunocompetent individuals cryptosporidium causes a transient diarrhoea, the infection may be life threatening in the immunosuppressed. While there have been no reports of endoscopic transmission we would agree that a new disinfectant, active against cryptosporidium, is needed for use before endoscopy in immunosuppressed patients. At present we recommend thorough mechanical cleaning followed by immersion for one hour in 2% activated glutaraldehyde before and after endoscopy on immunocompromised patients. This is to ensure that atypical mycobacteria are not transmitted to immunosuppressed patients and that M tuberculosis is not transmitted from a symptomatic patient with HIV infection to an immunocompetent patient.

Casemore and Blewett provide evidence that 30 minutes in 2% glutaraldehyde is not effective for cryptosporidium. We stress in our report that thorough mechanical cleaning with detergent is the most important part of the disinfection procedure. It would seem that we have to rely on this until there is an alternative disinfectant. We hope that the work underway by Casemore et al will contribute to final recommendations.

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Epidemiological study of asymptomatic inflammatory bowel disease

Sir,—Dr Mayberry and colleagues (Gut 1989: 30: 481-3) used prevalence rates in the above article in a rather misleading way. For instance, comparisons in their Table 4 do not state which age groups the prevalence rates refer to in the various studies which they have compared. The use of age specific rates would have overcome this difficulty.

Correspondence

Dept of Community Medicine,
Lothian Health Board,
Edinburgh

Sir,—The paper by Mayberry et al is interesting and also a timely reminder of what significant pathology may exist undetected in our communities.

It would be interesting to know how the Nottingham group decided to manage these asymptomatic patients and how well their patients complied with any treatments suggested.

J A R SMITH
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Reply

Sir,—We would like to thank both correspondents for their comments. The use of 'age specific rates' for the previously published data reported in Table 4 would have been inappropriate as they refer to the prevalence of inflammatory bowel disease in whole populations. Unfortunately, as discussed in the article published, age specific prevalence rates for populations aged 50-74 are unavailable.

Both patients with Crohn's disease underwent surgical resection. This would not be our routine practise in asymptomatic patients but there was concern that the abnormalities detected on radiological examination could have been tumours. All patients with ulcerative colitis were treated with sulphasalazine and are now regularly followed in an inflammatory bowel disease clinic.

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Comparison of forceful dilatation and oesophagomyotomy in achalasia

Sir,—The paper in the March issue (Gut 1989; 30: 299–304) by Csendes and colleagues is of great interest. Pneumatic dilatation for the management of achalasia in their hands did not perform as well as surgical management though as they admit their results using the pneumatic method are not as good as those of others. We have now followed up a much larger group of patients than they refer to with the results remaining as good. Their disappointing results may be related to the much lower inflation pressures which they use.

My impression is that many physicians/gastroenterologists are managing achalasia non-surgically. I consider this a welcome development for the very
reasons which the authors state. I do hope that their results will not reverse this trend. It has been suggested that perhaps patients in the younger age group do not respond so well to this type of management but this has not been our experience or those of others using the same type of dilator. Csendes and colleagues state that the procedure is painful but this is not something our patients have complained of when the procedure is carried out under diazepam sedation which they do not use. The main danger of pneumatic dilatation, which the authors rightly emphasise, is perforation of the oesophagus. This complication can nearly always be managed conservatively but it must be emphasised that in this gastrointestinal procedure as in many others medicosurgical collaboration is important.

Lastly, the authors emphasise the problem of individuality of the different dilatation methods and the consequent difficulty of comparing these. This observation may be correct but I do not think it should be considered an impossible task. There are enough Units carrying out the procedure for such a project to at least be considered.

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References

Reply
SIR,—We are grateful to Dr Dellipiani for his interesting comments.

The inflationary pressures that we use vary from 12 to 15 pounds per square inches (250 to 300 mmHg metric) which we believe are high pressures.

We have not used diazepam sedation because the pain that is usually seen with pneumatic dilatation, could be masked. We always perform radiological control of dilatation, however, and could therefore use diazepam.

Cooperative randomised studies are needed, but it will not be an easy task.

A TTILA CSENDES

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Mediators of vasopressin induced natriuresis in cirrhosis—possible role of atrial natriuretic factor
SIR,—Lenz et al (Gut 1989; 30: 90-6) recently reported increased natriuresis and diuresis in patients with cirrhosis and ascites after vasopressin infusion. The authors suggested suppression of sympathetic nervous system activity as an important mediator of the beneficial effects of vasopressin. Neither this nor other mechanisms discussed, however, could satisfactorily explain the observed improvement of renal function.

For further elucidation of the interesting results reported by Lenz et al, investigation of the atrial natriuretic factor (ANF) might be helpful. The role of this first well defined natriuretic hormone in volume retention of cirrhosis is being controversially discussed,1 with some authors reporting a relative deficiency of ANF plasma concentrations or impairment of ANF release in patients with cirrhosis and ascites.2 In rats, infusion of vasopressin has been shown to increase ANF plasma concentrations and ANF-induced natriuresis was found potentiated by vasopressin administration.3 Observations of an inhibition of vasopressin release by ANF4 lend further support to the contention that both hormonal systems are closely related. Thus, determination of ANF plasma concentrations might reveal ANF as a mediator of the vasopressin induced natriuresis in patients with cirrhosis and ascites.

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References