LETTERS TO THE EDITOR

Rectal motor activity

SIR,—We were interested to read that Prior *et al* (*Gut* 1991; **32**: 1360–3) have confirmed the existence of nocturnal periodic motor activity in the rectum, first reported from our laboratory[1] and subsequently confirmed at the Mayo Clinic.[2] We were also gratified to note that they have confirmed our findings,[3] published two years ago, that there is no temporal relationship between the rectal motor complex and the small intestinal migrating motor complex, but confess to surprise that our work is not cited.

We would concur with their conclusion that the rectal motor complex is a segmental rather than a propagated phenomenon. Our study was not designed to detect propagation of the rectal motor complex, because we were unaware of its existence when we designed our study. We could infer that the rectal motor complex was not propagated over any significant distance from the fact that it had not been previously detected in numerous previous studies of distal colonic motor activity, and that it is only apparent when sensors are carefully located in the rectum. They report greater variability in rectal motor complex incidence than has been found by others, but have not addressed the question of whether the difference might have been the result of the differences in recording technique. In our studies, we used a solid state recording system which enabled our subjects to go home, eat a meal of their own choice, and sleep in their own beds. Their subjects had to stay overnight in hospital; after the insertion of a rectal probe at 10 pm, and connection to a pneumohydraulic pump, they '. . . were allowed to fall asleep in a semidarkened room.' From our experience with the study of nocturnal small bowel motor activity, we know that the nocturnal pattern is most evident in subjects who are able to sleep normally at home,[4] and are therefore as little stressed as possible; the same may be true for the rectal motor complex.

We had assumed that the term 'rectal motor complex' had acquired a degree of ligitimacy as the term was first used in the title of a report[2] which was the very first paper to appear in another journal of which Professor Read is joint editor, but evidently he has had second thoughts on the matter. Prior *et al* doubt whether the rectal episodes deserve to be described as 'motor complexes'. They support their argument by suggesting that 'the contractions are similar to those observed by other workers in the human transverse, descending and sigmoid colon', we can find no report of such activity during sleep in the paper they cite,[5] but only a comment to the effect that '. . . sleep was nearly always associated with a sharp inhibition of motor activity'. In a study from our own laboratory[6] involving prolonged ambulant manometry in the unprepared human colon, we found the colon to be almost inert during the night, and observed nothing that resembled the rectal motor complex. We do not believe that migration or propagation is implied in the term 'complex', and certainly the phenomenon of disruption by food is irrelevant because this does not occur in the ruminant migrating motor complex. We contend that the rectal motor complex is a periodic biorhythm, and that such biorhythms seem to characterise neural command networks in the absence of sensory arousal. The use of the term 'complex' relates to the periodic stereotypy; for the small intestinal migrating motor complex, 'migrating' is added as a qualifying adjective.

Finally, the variability of the rectal motor complex leads Prior *et al* to speculate '. . . that monitoring of rectal activity is unlikely to be of value in the assessment of the intrinsic nervous system of patients with anorectal dysfunction'. Almost certainly this is true with regard to the periodicity of the event; similar hopes about monitoring human migrating motor complexes were dashed when the variability of the human migrating motor complex compared with the laboratory dog was shown.[7] This does not invalidate the case for monitoring human rectal motor activity, as it has been shown[8] that in patients with chronic intractable constipation, while the incidence of rectal motor complexes is not altered, the mean amplitude of rectal motor complex contractions is significantly ($p<0.001$) reduced from 42·4 (2·1) to 9·2 (0·7) mm Hg (means (SEM)).

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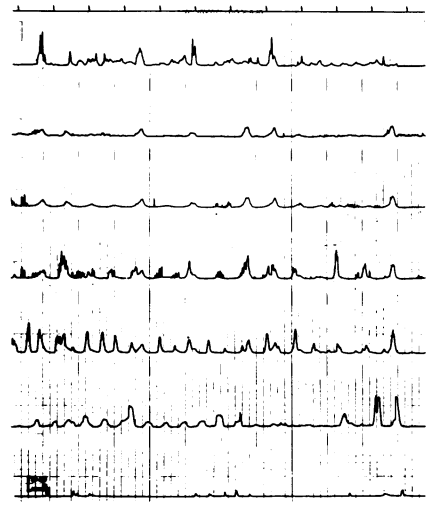
Rectal motor activity

SIR,—We read with interest the study of Prior *et al* (*Gut* 1991 **32**: 1360–3), in which they recorded rectal motor activity from multiple sites in healthy volunteers. We feel this investigation deserves some comments.

Three recent studies have reported the presence of recurrent bursts of contractions occurring in the rectum,[1–3] and two of them proposed, in analogy with the phase III of the small intestinal migrating motor complex, the term rectal motor complex to define this kind of activity.[2 3] We agree with Prior *et al*, however, that there is no experimental evidence supporting the formalisation of this term, as none of the studies aimed at correlating small bowel and recurrent rectal activity was successful.[1 3 4]



Colonic manometric tracing showing sporadic contractions (tracings 1–3), and non-propagated bursts of contractions (tracings 4–6). Recording points are (from the top) in the proximal and distal transverse, the proximal and distal descending, the mid sigmoid colon, the rectosigmoid junction and the rectum. The event marker showing one minute recording every little bar.

Although the recording period was on average about six hours, the study of Prior *et al* confirmed our previous experience with 24 hour recordings,[5 6] showing that even in its very distal segments the colon does not display any cyclic activity, and contractile activity resembles that seen in more proximal portions (Fig). The physiological function of the bursts of contractions sporadically seen in the rectum is unknown. We have recently shown that in women with slow transit constipation such activity is heavily decreased during 24 hour recordings,[7] together with an extremely blunted rectal response to eating. Similar findings have also been reported by Waldron *et al*,[8] and a rectal neuropathic process has been suggested as the pathophysiological ground for these abnormalities.

The fact that Prior *et al* were not able to identify high amplitude propagated contractions, the manometric equivalent of mass movements,[5 9 10] is not surprising. In fact, apart from the relatively brief recording period of their study, high amplitude propagated contractions are rarely, if ever, seen below the rectosigmoid junction, as this motor phenomenon is subject to a physiological 'fading' in the distal colonic segments.[5 9] Moreover, high amplitude propagated contractions are more likely to occur after specific events such as awakening in the morning and meals.

In conclusion, Prior *et al* are to be congratulated on their interesting study, which adds another piece to the mysterious puzzle of human colonic motility. Further work is, however, necessary to highlight the still hidden secrets of the motor aspects of this interesting viscus.

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Increased intestinal permeability in ankylosing spondylitis

SIR,—We read with great interest the excellent paper by Morris *et al* (*Gut* 1991; **32**: 1470–2) on intestinal permeability in ankylosing spondylitis but we do not agree, however, with their conclusions.

Using the ^{51}Cr-EDTA resorption test[1 2] we recently studied gut permeability in inflammatory rheumatic disorders. Intake of non-steroidal anti-inflammatory drugs (NSAIDs) significantly increases gut permeability irrespective of the underlying disease. Patients with ankylosing spondylitis and with other spondylarthropathies not taking NSAIDs also presented a significant increase of gut permeability compared with controls. This indicates that the disturbance is disease related. Gut permeability was not significantly increased in patients with histological gut lesions on ileocolonoscopy, or in patients with a normal ileum, although patients with ankylosing spondylitis and chronic gut lesions (resembling Crohn's disease) showed a significant increase in gut permeability compared with patients with ankylosing spondylitis and acute gut lesions.

There are several explanations for the absence of a relationship between increased gut permeability and ileocolonoscopic evidence of gut inflammation. On ileocolonoscopy only the terminal aspect of the ileum, which is only a very small part of the small bowel, can be examined. Moreover, the distribution of the observed lesions was patchy. Intake of NSAIDs causes such major disturbances in gut permeability that minor and local inflammation of the ileum would not influence the results of the ^{51}Cr–EDTA resorption test.

Inflammatory gut lesions were not found in patients with rheumatoid arthritis[3] taking high doses of NSAIDs for prolonged periods, while such lesions were present in more than 50 patients with spondyloarthropathies[4] who had not taken antiinflammatory drugs. This suggests that primary lesion in the ileocaecal region is associated with the spondyloarthropathies, while intake of NSAIDs probably induces more extensive and diffuse functional disturbances of the entire small bowel.

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Reply

SIR,—We do not feel that the evidence presented by Mielants *et al* in this letter convincingly shows that there is a primary pathology of the terminal ileum in patients with spondylarthropathies. Although patients with spondylarthropathies not taking non-steroidal antiinflammatory drugs (NSAIDs) did have significantly increased permeability compared with the controls, this was, by the authors' own admission, a very small group in whom no details of gut histology are given. Chronic lesions, resembling subclinical Crohns' disease, as described by the authors, may have been present and thus affected the results by increasing permeability in some of those patients.[1 2]

The argument that local or patchy inflammation of the ileum may not affect results of ^{51}Cr–EDTA absorption does not seem valid when previous studies have shown that ^{51}Cr–EDTA excretion increases towards the end of a nought to six hours collection consistent with increased absorption more distally in the small bowel.[3] Local inflammation in the ileum would be expected to have a relatively greater effect on ^{51}Cr–EDTA results and the lack of correlation between ileocolonoscopic findings and permeability (by Mielants *et al*) requires further explanation.

The fact that no inflammation was observed in rheumatoid arthritis patients at ileocolonoscopy should be considered in the context of more proximal inflammation and ulceration observed by our group using small bowel enteroscopy.[4] It may be that NSAID treatment is affecting different areas of the gut preferentially in ankylosing spondylitis and rheumatoid arthritis, thus explaining the increased permeability in rheumatoid arthritis patients on NSAIDs without ileocolonoscopic evidence of inflammation observed by Mielants and colleagues. Overall we find that the evidence for NSAID small bowel damage is more compelling than for a primary abnormality in spondylarthropathy.

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Crohn's disease after ileocolic resection

SIR,—Olaison, Smedh and Sjödahl (*Gut* 1992; **33**: 331–5) have provided endoscopic evidence that in many cases of Crohn's disease renewed ileal ulceration occurs soon after surgical resection; 22 of 30 examined at three months and proportionately more at 12 months. The authors consider that their data support views held by many that the bowel is permanently affected in Crohn's disease and is therefore liable to frequent clinical relapse even after apparent radical resection. Yet some follow up studies have also shown that as many as 25% of patients remain free of clinical symptoms for many years or even indefinitely. Nevertheless, with a relapse or recurrence rate as high as it is, it is clearly the responsibility of every physician and surgeon to do all that is possible to stave off renewed activity of the disease. Olaison *et al*'s report suggests that this needs to be done if possible before the onset of clinical symptoms when the disease process will have progressed to extensive ulceration and/or strictures.

Most clinicians at present monitor progress of these patients by regular checks for symptoms and signs of recurrence and test for anaemia, a rise in sedimentation rate and muramidases. Others, influenced by reports such as this one, may be inclined to prescribe maintenance doses of drugs such as aminosalicylates, immunosuppressives or corticosteroids. There is, however, evidence that such measures alone are not always enough. That evidence concerns the adverse affect of definitive emotional stress in this disorder which has either been forgotten or overlooked, or disbelieved and therefore ignored. The case both for and against it has been reviewed in the section on Crohn's disease in a recent book.[1] Appropriate psychological management of such cases is well within the competence of a non-psychiatrically trained physician once he or she has become aware of what is needed to help these sensitive and vulnerable people to change their previously damaging coping mechanisms in dealing with abrasive interpersonal strife in their immediate environment. How to do this is described with case histories and transcripts.[1] What so often happens now, however, is that patients, many of them very young, are returned without psychological help to the same abrasive domestic or social environment which immediately preceded the onset or relapse of their disease. Case histories and the few outcome studies available illustrate the value of such intervention in cutting short relapses when domestic strife escalates and patients find themselves caught in the middle. Before treatment they lack the ability to cope or escape. Many such patients managed in this way remain free of disease for many years or suffer only minor relapses. The authors of the article from a Department of Surgery may be unaware that the first reports on psychosomatic