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 "Cr-EDTA resorption test," 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 increased gut permeability in patients with ankylosing spondylitis is a systemic phenomenon and not limited to the ileum.

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 is examined, 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 disturbance of gut permeability that the patchy and local inflammation of the ileum would not influence the results of the "Cr-EDTA resorption test".

Inflammatory gut lesions were not found in patients with ankylosing spondylitis or arthritis taking high doses of NSAIDs for prolonged periods, while such lesions were present in more than 50 patients with spondylarthropathies who had not taken anti-inflammatory drugs. This suggests that a gut lesion in the ileocolonic region is associated with the spondylarthropathies, while intake of NSAIDs probably induces more extensive and diffuse disturbances of the entire small bowel.


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 the renewed ileal ulceration occurs soon after surgical resection. We believe that there are important points these authors may have disbelieved and therefore ignored.

What follows is a presentation of a case of ileocolonic Crohn's disease in which we believe that the preoperative severe symptoms were due to a relapse rather than an initial ileocolonic Crohn's disease.

A 32-year-old woman developed severe chronic diarrhoea and increased/perianal rectal pain. She was found to have perianal erythema and tenderness but no evidence of inflammatory bowel disease. She had a history of anorectal disease. I.


M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,
Royal Infirmary,
Glasgow G31 2ER

M. J. Morris
R. I. Russell
Gastroenterology Unit,