allowed to undertake all endoscopic procedures in the field of conventional endoscopy. A recent editorial from Dewitt[1] asks, ''Is there anything now we can't do?'' with double balloon enteroscopy. We would ask, ''Is there anything now we *can* do''?

We encountered the case in our early experience of double balloon enteroscopy, and only a diagnostic endoscope was available in Taiwan. Because of our limited experience in the use of double balloon enteroscopy and the lack of suitable haemostasis equipment, a discussion with the patient was held and it was determined that he preferred the option of surgical resection of the polyp.

At present, we are capable of diagnosing various small intestinal diseases with the use of double balloon enteroscopy. However, we still need more experience to improve our technique for treating the lesions found and managing the associated complications. More studies are required to show the effectiveness and safety of therapeutic double balloon enteroscopy compared with traditional methods.

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Remission and relapse of Crohn's disease following autologous haematopoietic stem cell transplantation for non-Hodgkin's lymphoma

Autologous haematopoietic stem cell transplantation (HSCT) for Crohn's disease has been described by Oyama and co-workers as producing remission in 11 of 12 patients with refractory Crohn's disease after a median follow up of 18.5 months.[1] They postulated that autologous HSCT was useful for refractory Crohn's disease. We report the case of a 32 year old woman with biopsy proven Crohn's disease diagnosed when she was 16 years old. Her mother also suffered from Crohn's disease. She developed a KI1 positive anaplastic non-Hodgkin's lymphoma (NHL) three years later, treated successfully with ablative CHOP chemotherapy and autologous HSCT, inducing remission for the past 12 years (and she remains in remission on no treatment). As a consequence of her autologous HSCT she also went into long term remission from her Crohn's disease, but relapsed after eight years. She was a smoker, but was on no maintenance treatment for Crohn's disease in this eight year period. She developed abdominal pain and bloody diarrhoea and underwent a colonoscopy, and a biopsy proved the recurrence of her colonic Crohn's disease. She was treated with corticosteroids and 5-aminosalicylates, which induced temporary remission. However, she subsequently had two relapses over the past two years, with diarrhoea, histologically proven erythema nodosum, and seronegative arthropathy. She was treated with reducing courses of corticosteroids with prompt resolution of her symptoms. There is no evidence of relapse from her NHL, and long term treatment with immunosuppressive agents or infliximab is under consideration but has been deferred because of a small increased risk of lymphoma associated with these agents (though it is not known whether this is attributable to Crohn's disease, immunosuppression alone, or a combination of the two[2]). Genotyping revealed no *CARD15* mutations (R702W, G908R, and L1007fs).

Our case is the longest reported ''follow up'' of autologous HSCT in Crohn's disease and raises some interesting questions with regard to the long term efficacy of autologous HSCT for Crohn's disease, as our patient appeared to be in complete long term remission and was apparently ''cured'' of her Crohn's disease for eight years before relapsing. We postulate that the autologous HSCT led to ablation of ''activated'' T cells for a prolonged period (resetting the immune system) but that her genetic predisposition (positive family history), allied to other environmental factors (smoking), subsequently led to her disease relapse. This is in keeping with the current hypothesis of the aetiopathogenesis of Crohn's disease and suggests that autologous HSCT will not provide a long term ''cure'' for this disorder. Allogenic HSCT may be more appropriate,[3] albeit at a much greater risk of treatment related complications such as graft versus host disease. This is further supported by the results of allogenic HSCT for Crohn's disease, where the only relapse occurred in a patient who developed a mixed donor–host haematopoietic chimerism following allogenic HSCT.[4] Furthermore, in the study by Oyama and co-workers, there were two relapses in the short term and only two patients achieved treatment-free remission for more than three years.[5]

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Activated innate immune system in irritable bowel syndrome?

The discovery of antimicrobial peptides has extended our knowledge of non-specific defence mechanisms. In addition to the physical barrier, the intestinal epithelium contributes to host defence by producing antimicrobial peptides to limit access to enteric bacteria and other microorganisms.[1] The production of inducible antimicrobial peptides offers an initial rapid defence response of epithelial cells against invading microbes.[2]

Human β-defensin-2 (HBD-2) was the first inducible human antimicrobial protein discovered.[3] It can be induced by probiotic microorganisms and pro-inflammatory cytokines.[4] Recent results suggest that HBD-2 is expressed in active intestinal inflammation, especially in ulcerative colitis.[5] Thus far, the expression of β-defensins has been quantified in the intestinal mucosa using the polymerase chain reaction procedure. Our aim was to evaluate faecal measurements of HBD-2 in patients with active ulcerative colitis, compared with irritable bowel syndrome as a non-inflammatory clinical control and with healthy controls. We expected that faecal HBD-2 levels would show a similar pattern to mucosal HBD-2 and thus be selectively increased in ulcerative colitis.

Faecal specimens were collected from 69 patients (22 with active ulcerative colitis, 24 with irritable bowel syndrome, and 23 healthy controls) (table 1). To avoid any influence of probiotics in our data, patients on probiotic drugs were excluded. Further exclusion criteria for the patients with irritable bowel syndrome were a raised C-reactive protein or leucocyte count and a history of infectious gastrointestinal disease or bacterial overgrowth over the previous six months. Disease status was addressed in all participating subjects by medical history and current symptoms. In addition, each patient with irritable bowel syndrome or ulcerative colitis underwent ileocolonoscopy with histopathology. Faecal HBD-2 was measured by enzyme linked immunosorbent assay (Immundiagnostik, Bensheim, Germany) and reported as ng/g faeces. HBD-2 concentrations (mean (SD)) were 104.9 (62.9) ng/g in the active ulcerative colitis group, 72.9 (52.9) ng/g in the irritable bowel syndrome group, and 31.0 (15.4) ng/g in the healthy controls ($p<0.0005$). Scheffé post hoc tests showed significant differences between healthy controls and the two patient groups (fig 1).

The results first imply that levels of HBD-2 are detectable in faeces and can be quantified. Second, as in mucosal specimens, HBD-2 levels are significantly raised in faeces in patients with ulcerative colitis compared with healthy controls. Finally, in contrast to our hypothesis,

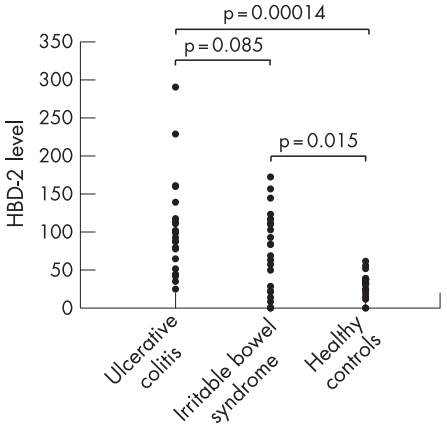

Figure 1 HBD-2 levels in faeces.