hospitalization with duodenal or gastric ulcer, respectively, anytime between 1965 and 2003, until the date of death, emigration, partial or total gastrectomy, vagotomy, cancer diagnosis, or the study end point. Compared with cancer rates for the general population (matched for calendar period, age and sex), there was a 70% greater risk of esophageal adenocarcinoma in patients with duodenal ulcer, but no greater risk in patients with gastric ulcer. By contrast, there was an insignificant but slight excess risk of esophageal squamous cell carcinoma in patients with duodenal ulcer but an 80% greater risk in patients with gastric ulcer. The authors acknowledge that a lack of information on confounding factors (e.g. smoking and medication) and actual *H. pylori* status might limit the explanation of the associations observed. Nevertheless, they suggest that their results support two hypotheses. First, in patients with gastric ulcer, *H. pylori*-induced atrophic gastritis and hypoacidity protect against the development of esophageal adenocarcinoma. Second, the development of esophageal squamous cell carcinoma might be influenced by the intragastric environment that results from corpus atrophy.

Original article Bahmanyar S *et al.* (2007) Risk of oesophageal cancer by histology among patients hospitalised for gastroduodenal ulcers. *Gut* **56:** 464–468

Nonmyeloablative stem cell therapy accelerates tissue repair in experimental mouse IBD

IBD is managed by the use of immunomodulatory and anti-inflammatory drugs, but repair to the damaged digestive tract by other means is still required. Adult stem cell therapy seems to contribute to tissue regeneration in inflammatory-related diseases. Khalil *et al.*, therefore, investigated the potential of treatment with nonmyeloablative stem cells ($CD34^-$ cells) to facilitate epithelial repair in a mouse model of IBD.

Moderate or severe colitis was induced in adult mice in two cycles, each comprising addition of dextran sulphate sodium (DSS) to drinking water at a concentration of either 3% ($n=30$) or 5% ($n=36$) for 7 days, followed by a 10-day recovery period. On day 8, 2.0×10^6 mouse $CD34^-$ cells (from an immortalized cell line) were injected into the tail vein of these mice and the mice from one of two control groups (each $n=9$) that received no DSS. Mice were observed during treatment for a period of 35 days. Stem cell therapy significantly reduced the histological grade and morphological signs of mouse colitis, and, in the 5% DSS group, significantly increased survival of mice with severe colitis. The injected $CD34^-$ cells were seen to home to areas of damaged colon, but not undamaged regions, and promote neovascularization. Differentiation of the injected stem cells into endothelial cells to improve mucosal perfusion was noted.

Stem cell therapy seemed to accelerate tissue repair, which might be related to improved perfusion supporting pre-existing intestinal stem cell function.

Original article Khalil PN *et al.* (2007) Nonmyeloablative stem cell therapy enhances microcirculation and tissue regeneration in murine inflammatory bowel disease. *Gastroenterology* **132:** 944–954

Poor agreement among different methods of measuring creatinine levels

Serum creatinine levels are used to help calculate the MELD (model for end-stage liver disease) score. Several laboratory methods for measuring serum creatinine levels have been devised to overcome the influence of bilirubin; as serum bilirubin levels increase, the lower the creatinine values become. Cholongitas and colleagues have assessed the degree of agreement among four creatinine assays, and the subsequent effect on MELD scores.

Serum creatinine levels were measured in 403 consecutive blood samples from 158 patients who had abnormal liver function. All samples were analyzed with each of the following methods: O'Leary modified Jaffe; compensated (rate blanked) kinetic Jaffe; enzymatic creatinine; and standard kinetic Jaffe. Serum bilirubin levels and international normalized ratio prothrombin time were also measured so the MELD score could be calculated.

There was poor agreement among the results of all four methods. The O'Leary modified Jaffe method showed the best correlation between creatinine and bilirubin levels. MELD scores calculated using the results of the O'Leary modified Jaffe assay were markedly different from the results obtained using the other three assays when serum bilirubin concentrations were