PBMT-treated than PBAT-treated patients (65.5% versus 16.0%, $P<0.001$).

The authors speculated that PBAT performed poorly compared with PBMT because the bactericidal effect of amoxicillin–clavulanate was hampered by the bacteriostatic effect of tetracycline. PBMT therapy performed poorly in patients infected with metronidazole-resistant *H. pylori*; Cheon and colleagues recommend further investigation, to determine which antibiotic combinations are suitable substitutes for metronidazole and tetracycline in second-line eradication therapy.

Original article Cheon JH *et al.* (2006) Combinations containing amoxicillin–clavulanate and tetracycline are inappropriate for *Helicobacter pylori* eradication despite high *in vitro* susceptibility. *J Gastroenterol Hepatol* **21:** 1590–1595

Donor mortality after living donor liver transplantation

Living donor liver transplantation (LDLT) confers a small risk of donor death; however, the worldwide actual mortality of patients who undergo donor hepatectomy is unknown. Trotter *et al.*, therefore, searched PubMed for articles published from 1989 to February 2006 for data on donor outcomes following LDLT. Nonmedical literature was also searched. Deaths were recorded as 'definitely', 'possibly' or 'unlikely' to be donor-hepatectomy-related.

An estimated 4,598 LDLTs were performed in the US and Europe during the study period; the corresponding mortality definitely caused by donor hepatectomy was estimated at 0.15%, and mortality that was definitely or possibly related to donor hepatectomy was estimated at 0.20%. The authors identified 13 deaths worldwide (and 1 donor in an irreversible coma) that were definitely caused by donor hepatectomy. Two deaths (both suicides) were considered possibly related to donor hepatectomy, and four were probably unrelated. The most common cause of death definitely related to donor hepatectomy was sepsis (five donors), followed by liver failure and unknown (two donors each), myocardial infarction, cerebral hemorrhage, pulmonary embolus and peptic ulcer disease (one donor each); the mean time to death was 37.9 days (median 11 days).

The authors acknowledge their study might not include all donor deaths, partly because it is not mandatory worldwide to report donor outcomes after LDLT. They call for known, unreported donor deaths to be documented, to improve mortality estimates.

Original article Trotter JF *et al.* (2006) Documented deaths of hepatic lobe donors for living donor liver transplantation. *Liver Transpl* **12:** 1485–1488

Clonidine speeds up patient responses to standard diuretic therapy

In patients with cirrhosis, the mechanisms that lead to renal sodium retention are not fully known; however, elevated sympathetic nervous system (SNS) activity (indicated by high plasma norepinephrine concentrations) is a known contributory factor. Lenaerts *et al.* examined the effect of the adrenergic agonist, clonidine, on the outcomes of patients with cirrhosis and increased SNS activity who also received standard diuretic therapy.

The authors enrolled 64 cirrhotic patients with ascites and a plasma norepinephrine level >300 pg/ml. The patients were randomly allocated to receive either placebo ($n=32$) or 0.075 mg clonidine ($n=32$), twice daily for 3 months. After 8 days, both groups initiated diuretic therapy with 200 mg spironolactone daily for 10 days (subsequently, spironolactone dosage was gradually increased and another diuretic, furosemide, was added if required).

Patients were discharged when their daily body mass loss was >200 mg and their daily urinary sodium was >50 mmol/l. Mean time to discharge was shorter for clonidine-treated than placebo-treated patients (23.06 versus 31.97 days). Mean diuretic requirement was higher in placebo-treated than in clonidine-treated patients (spironolactone 350.00 mg versus 260.16 mg daily; furosemide 25.99 mg versus 3.13 mg daily). Diuretic complications (hyperkalemia and renal impairment) were markedly reduced in clonidine-treated patients compared with placebo-treated patients. Clonidine induced a sustained decrease in SNS activity, and inhibited the increased activity of the renin–aldosterone axis that is a consequence of diuretic therapy.

The authors conclude that clonidine plus standard diuretic therapy was more effective than standard diuretic therapy alone for