at the University Hospital Rangueil during the period 2004–2006; anti-HEV IgG was found in 14.5% of kidney recipients and in 10.4% of liver recipients. Unexplained short-term elevations of serum liver-enzyme levels occurred in 217 patients; screening for serum HEV RNA revealed that 14 (6.5%) of these patients (including 9 kidney transplant recipients and 2 combined kidney–pancreas transplant recipients) had acute HEV infection. Hepatitis was asymptomatic in seven patients but manifested as arthralgia, myalgia, and fatigue in the other seven patients. HEV infection resolved within 6 months in six patients. The other eight patients developed chronic hepatitis; serum levels of liver enzymes remained elevated and the patients had detectable serum or stool HEV RNA at the last follow-up (range 10–24 months). Hepatitis developed earlier after transplantation in patients with chronic than in those with acute disease (median 37.5 vs 78.5 months), and patients who developed chronic disease had significantly lower leukocyte, lymphocyte and platelet counts, and serum creatinine levels, at diagnosis.

The authors conclude that HEV should be considered as a possible cause of acute and chronic hepatitis in kidney and other organ transplant recipients.

Original article Kamar N *et al.* (2008) Hepatitis E virus and chronic hepatitis in organ-transplant recipients. *N Engl J Med* **358:** 811–817

Banff classification of renal allograft pathology—2007 update

Solez *et al.* have reported the conclusions of the 9th Banff Conference on Renal Allograft Pathology, held in La Coruña, Spain in June 2007. At this conference, the Banff criteria for renal allograft rejection were updated to include peritubular capillaritis scoring, complement degradation component C4d scoring, the reporting of C4d deposition in the absence of morphological evidence of active allograft rejection, and application of the criteria to biopsy samples taken at the time of transplantation. Other additions to the classification, the use of which remains optional pending 2 years of testing, are the scoring of total interstitial inflammation ('ti') and an alternative scoring system for hyaline arteriolar thickening. All existing scoring categories are unchanged.

In accordance with the updated classification, peritubular capillaritis should be scored from ptc 0 to ptc 3, on the basis of the percentage of inflamed capillaries in the sample and the number of inflammatory cells present. The composition of the inflammatory cell population (monocytes vs neutrophils) and the extent of capillaritis (focal 10–50% vs diffuse >50%) should be noted. C4d staining is scored from C4d0 (negative) to C4d3 (diffuse >50% of the sample). The current system for scoring arteriolar hyaline thickening ('ah') was reported to have very poor reproducibility ($\kappa = 0.18$); therefore, an alternative system ('aah'), in which the severity of arteriolopathy is quantified by the presence of circular or noncircular involvement and the number of affected arterioles, will be tested. Improved reproducibility with the new system ($\kappa = 0.67$) has already been reported.

Finally, working parties have been set up to explore the potential role of new gene-expression technologies in the diagnosis of graft rejection.

Original article Solez K *et al.* (2008) Banff 07 classification of renal allograft pathology: updates and future directions. *Am J Transplant* **8:** 753–760