GLUCOSE-CONTAINING ORGAN PRESERVATION SOLUTIONS AND INTRACELLULAR ACIDOSIS

We read with interest the excellent review by Belzer and Southard (1) on the principles of organ preservation by cold storage following vascular flushing. The UW solution developed by the authors holds the promise that improved preservation will be achieved, especially for organs other than the kidney, both in duration of safe storage and in quality of function in the early postreimplantation phase. One concept that we would like to question, however, is the view that glucose present in the preservation solution (as in EuroCollins') can contribute to the developing acidosis during hypothermia, particularly in the liver. We have been undertaking \(^3\)P NMR spectroscopy studies on the response of liver to vascular flush and cold storage, using the conventional hypertonic citrate solution (which contains no glucose) and Collins' solution (a phosphate-based solution with high glucose content) (2, 3). Using the chemical shift of the inorganic phosphate signal, we have followed the pH changes in the livers with time at hypothermia. In these experimental studies on the rat it is apparent that the pH fall in both solutions is identical (reaching by 8 hr pH \(6.85\pm0.04\) in citrate-flushed organs, and \(6.86\pm0.03\) in Collins'-flushed organs). Factors other than lactate accumulation can influence intracellular pH, including the hydrolysis of ATP, which liberates protons (5)—and from our studies we have no reason to implicate glucose contained in the storage solution as a major contributing factor in the hepatic acidosis. Indeed, in other studies (to be reported) when we used a phosphate-based preservation solution in which the glucose was completely replaced by mannitol, the intrahepatic pH fall was again identical to that seen in the glucose-containing medium. The metabolic pathways in play during hypothermia of mammalian cells still remain largely to be identified, and the interesting combinations of solutes in the solution developed by Belzer and Southard open up exciting new possibilities for organ preservation.

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REFERENCES
HEPATITIS IN LIVING-DONOR RENAL ALLOGRAFT RECIPIENTS

The effect of liver disease on the survival of renal allograft recipients is still controversial (1-5). We have therefore reviewed our experience with hepatitis in such patients.

All 447 patients who had living-donor kidney transplantation at our center from 1976 to 1985 were studied. Donor selection, immunosuppression regimen, and transfusion protocol have been reported earlier (6-8). Liver function tests and screening for hepatitis B surface antigen (HBsAg) were done at monthly intervals, or more frequently if necessary, up to 6 months after transplantation, and at frequent intervals thereafter. Serum was examined for HBsAg by the reverse passive hemagglutination test. Liver biopsies were performed when indicated.

Hepatitis was defined as a two-fold elevation of serum aspartate aminotransferase or serum alanine aminotransferase, or both, with or without an increase in serum bilirubin. Chronicity was defined as persistence of hepatitis for more than 6 months.

A total of 66 (14.8%) of the patients had hepatitis. All had received azathioprine, and 11 also received potentially hepatotoxic drugs such as rifampicin, isoniazid, pyrazinamide, and alpha methyldopa. The mean follow-up was 17 months (range 6-74 months), during that period 35 of the 66 patients (53%) had chronic hepatitis and the rest transient hepatitis.

Of the 35 with chronic hepatitis, 16 had persistent HBs antigenemia, 6 transient antigenemia, and the rest were always negative on HBsAg testing. Four patients with chronic hepatitis who had severe disease had liver biopsies that showed chronic active hepatitis in 2, micronodular cirrhosis in 1, and hemosiderosis in 1. Five patients with chronic hepatitis and persistent antigenemia died of hepatocellular failure, and another died of gram-negative bacterial septicemia. One patient with chronic hepatitis and transient antigenemia died of hepatocellular failure, 2 of gram-negative bacterial septicemia, 2 of severe bacterial respiratory infection, and 2 of disseminated tuberculosis.

Of the 31 with transient hepatitis, 6 had persistent HBs antigenemia, 16 transient antigenemia, and the rest were always negative on HBsAg testing. Liver biopsy of two patients...

REFERENCES