

TITLE: RENAL HEMODYNAMICS DURING LIVER TRANSPLANTATION UTILIZING VENO-VENOUS BYPASS**AUTHORS:** TC Gunning, MR Brown, TH Swygert, AW Paulsen, TA Gonwa, MAE Ramsay**AFFILIATION:** Departments of Anesthesiology, Baylor University Medical Center, Dallas, TX 75246; University of Texas Southwestern Medical Center, Dallas, TX 75235

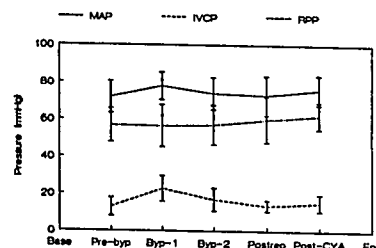
Introduction. Patients undergoing orthotopic liver transplantation (OLT) often experience a decrease in renal function postoperatively. Factors implicated in this development include cyclosporine nephrotoxicity, pre-renal azotemia, and intraoperative renal hypoperfusion. Of special concern during surgery is the anhepatic period, when IVC flow is interrupted and venous congestion of infrahepatic structures can occur. Although veno-venous bypass is often utilized to reduce congestion and maintain hemodynamic stability, its effects on renal hemodynamics have not been well-described.

Methods. We prospectively studied 13 patients with normal renal function who underwent OLT utilizing veno-venous bypass (VVB) during the anhepatic stage. Intraoperative measurements were made after induction of anesthesia, pre-bypass, twice during bypass, and twice following reperfusion of the graft liver. Variables included MAP, CI, SVR, PCWP, CVP, urine output (UOP), infrahepatic IVC pressure (via a femoral

catheter placed at the level of the renal veins), and renal perfusion pressure (RPP=MAP-IVCP). Renal function was determined by measuring GFR pre-op and 24 hours post-op using the Glofil method.

Results. The combination of IVC clamping and VVB resulted in a significant increase in IVCP which persisted throughout the anhepatic phase. MAP actually increased slightly and RPP was therefore not significantly altered. UOP during the dissection, anhepatic, and neohepatic phases was not different (2.2 ml/kg/hr). GFR decreased slightly post-op, but not to a significant degree (144±81 vs. 114±60 ml/min).

Discussion. We conclude that the use of VVB during IVC occlusion in OLT's preserves renal perfusion pressure and UOP, and does not result in perioperative renal dysfunction.



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TITLE: SYSTEMIC AND HEPATIC HEMODYNAMICS ASSOCIATED WITH FULMINANT HEPATIC FAILURE**AUTHORS:** TC Gunning, AW Paulsen, D Brajtford**AFFILIATION:** Departments of Anesthesiology, Baylor University Medical Center, Dallas, TX 75246; The University of Texas Southwestern Medical Center, Dallas, TX 75235

Introduction. The purpose of this study was to compare the circulatory changes that accompany fulminant hepatic failure (FHF), defined as complete liver failure occurring within 7 days, with the more common circulatory changes of end-stage liver disease at the time of orthotopic liver transplantation (OLT).

Methods. A retrospective study of 273 consecutive patients undergoing OLT was performed. Five patients with fulminant hepatic failure were identified. Portal and hepatic vein pressures [PVP, HVP] in the native liver were measured by direct puncture using a 27 gauge needle. Hepatic artery and portal vein flows [HAF, PVF] were measured by electromagnetic flowmetry. Routine hemodynamic measurements [MAP, CVP, CO] were acquired at the time of flow and pressure measurements. Measurements were obtained from the native liver approximately 30 minutes prior to removal. Hepatic artery and portal vein resistances were calculated from flow and pressure data [HAR, PoVR]. Systemic vascular resistance [SVR] was determined along with mesenteric, portal and hepatic artery perfu-

sion pressures [MPP, PPP, HAPF]. Statistical significance was computed using ANOVA and paired Student's t test.

Results. Relevant results (mean±SEM) are presented in the table below. There was a significant difference in SVR between groups, although arterial pressures were not dissimilar. PVP, PPP, and PoVR were all significantly lower in the FHF patients. CVP, HVP, HAF, and PVF were not significantly different.

Discussion. Patients with FHF represent a subgroup of liver transplant patients with distinctly different hemodynamics: lack of portal hypertension, lower systemic and portal vascular resistances, and a more elevated CO. The acuity of onset suggests these changes are humorally, not structurally, mediated. A lower PoVR corresponds to lack of cirrhotic changes in the liver.

	Chronic	FHF	sig.
MAP (mmHg)	74.9±1.0	67.6±3.8	NS
CO (l/min)	8.4±0.3	11.3±4.5	p<0.05
PVP (mmHg)	24.8±0.5	10.2±1.4	p<0.01
PPP (mmHg)	15.1±0.5	8.8±1.2	p<0.02
SVR (dyne-sec-cm ⁻⁵)	706±30	441±55	p<0.05
PoVR (dyne-sec-cm ⁻⁵)	1863±219	443±73	p<0.001