TITLE: HEMODYNAMIC CHANGES IN LIVER TRANSPLANT RECIPIENTS DURING REPERFUSION OF THE HOMOGRAFT

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Introduction. The postreperfusion syndrome is a transient cardiovascular collapse occurring upon reperfusion of the graft in 30% of adult liver recipients[1]. This study focuses on hemodynamic changes during graft reperfusion of liver recipients not supported with veno-venous bypass.

Methods. 26 liver recipients; 8 males, 18 females (68 ± 3 kgs, mean ± SEM, 41 ± 2 years, 1.8 ± 0.04 m^2 bsa, primary biliary cirrhosis = 5, sclerosing cholangitis = 8, hepatitis = 5 and others = 8, UNOS class 5 to 9) were anesthetized and transplanted as previously reported[2]. Temperature (° C), serum K+ (mEq/L), pH (units), heart rate (HR, beats/min), and superior vena cava (SVCP), inferior vena cava (IVCP), mean pulmonary artery pressure (MPAP), pulmonary artery wedge pressure (PAWP), mean aortic pressure (MAP, mmHg), cardiac (CI, L/min/m^2 bsa), stroke volume index (SVI, ml/beat/m^2), pulmonary vascular resistance index (PVRI), systemic vascular resistance index (SVRI) (dynes/cm/sec^{-5}/m^2), IVCP-SVCP gradients ($\Delta G_{IVCP-SVCP}$, mmHg) and SVI/PAWP ratio were measured or calculated using standard equations before (Phase I, samples a and b), during (Phase II, samples c-e) and after (Phase III, samples f-i) the anhepatic phase. Analysis of variance, two-tailed Student's t test and linear regression, were used to establish significance and correlation.

Results. Following IVC clamping Phase I hemodynamics of liver recipients (HR = 93 ± 3, MAP 81 ± 2, MPAP 19 ± 1, PAWP 13 ± 1, IVCP 14 ± 1, SVCP 10 ± 1, CI 5 ± .5, SVI 61 ± 4, SVRI 1156 ± 114, PVRI 94 ± 9, SVI/PAWP ratio = 4.8, $\Delta G_{IVCP-SVCP}$ = 3 ± 1, Temp 36 ± .2, K^+ 2.9 ± .1, pH 7.40 ± .1) were changed except for SVCP (9 ± 1). Phase I $\Delta G_{IVCP-SVCP}$ rose 12 fold with clamping ($p < .0001$). By transfusing 3461 ± 422 ml of blood during 55 ± 2 min, Phase II SVCP was maintained, while PAWP fell 62% ($p < .001$). At lower preload, CI and SVI decreased 50 and 44% ($p < .001$), respectively. MAP during Phase II fell only by 13% from Phase I values ($p < .001$) because of a rise in central adrenergic drive and exogenous dopamine support (10 mcg/kg/min), which caused a maximum rise of 27%, 106% and 42% in HR, SVRI and PVRI from Phase I values ($p < .001$), respectively. SVCP, IVCP, MPAP and PAWP and the $\Delta G_{IVCP-SVCP}$ remained unchanged until caval unclamping. Then $\Delta G_{IVCP-SVCP}$ decreased to values (9 ±1) higher than those of Phase I ($p < .001$). Upon caval unclamping, improved venous return brought SVCP, MPAP and PAWP to values transiently higher than those immediately before unclamping (sample e, 14 ± 1, 12 ± 1, 9 ±1, $p < .001$, respectively) and MAP decreased to its lowest Phase III values (83 ± 3, $p < .009$). In 1 of 26 patients (4%) MAP fell to 54 (HR 98) upon reperfusion. Afterwards, Phase III MAP, MPAP, SVCP, and PAWP stablized at values not different from Phase I. CI, SVI and PVRI also increased and returned to Phase I values. However, SVI/PAWP ratio, which fell by 42% during Phase II, rose upon unclamping to only 4.1 remaining so for 5-10 minutes and returning to Phase I values at the end of surgery. Upon caval clamping (Fig) CI was a linear function of PAWP ($y = -1.5 + 0.2X$, $r = 0.58$, $p < .003$). Upon unclamping, this relationship disappeared, ($y = 3.5 + 0.05X$, $r = 0.14$, $p < 0.56$) suggesting myocardial depression. Upon unclamping, HR and SVRI decreased transiently to (119 ± 2, $p < .03$; 981 ± 80, $p < .01$), 4 and 60% respectively, from preunclamping Phase II values and then stablized, HR at values 22% above and SVRI at values 22% below Phase I in 5 minutes ($p < .001$). Following unclamping, there was a .2% fall in core Temp from 35 ± .1, a 25% rise in serum K^+ from 2.8 ± .1, a 0.03 unit fall in pH from 7.41 ± 0.1 and six-fold rise in urine output which fell following IVC clamping to 2 ± .5 ml/kg/hr.

Discussion. This data shows that upon caval unclamping all liver recipients not supported with veno-venous bypass experience a postreperfusion syndrome characterized by deceleration of HR, vasodilation (decreased SVRI), myocardial dysfunction (inability to adequately increase CI after a rise in PAWP, and a fall in SVI/PAWP ratio). This transient response caused significant hypotension (MAP < 60) in only 4% of recipients. This syndrome may be triggered by a sudden rise in filling presures and/or by circulating chemicals, loading or depolarizing, respectively cardiac baroreceptors (Bezold reflex).

References.

1. Aggarwal S, et. al.: Postreperfusion syndrome: cardiovascular collapse following hepatic reperfusion during liver transplantation. Trans Proc XIX(4)S3 54-55, 1987

2. Estrin, JA, et. al.: Massive transfusion during adult and peditric liver transplants I: transfusion techniques. Anesth Analg 65:S50, 1987